Behaviour change techniques associated with smoking cessation in intervention and comparator groups of randomized controlled trials: a systematic review and meta-regression

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ABSTRACT

Aims   To estimate the strengths of associations between use of behaviour change techniques (BCTs) and clusters of BCTs in behavioural smoking cessation interventions and comparators with smoking cessation rates. **Method**   Systematic review and meta-regression of biochemically verified smoking cessation rates on BCTs in interventions and comparators in randomized controlled trials, adjusting for a priori-defined potential confounding variables, together with moderation analyses. Studies were drawn from the Cochrane Tobacco Addiction Group Specialised Register. Data were extracted from published and unpublished (i.e. obtained from study authors) study materials by two independent coders. Adequately described intervention ($k = 143$) and comparator ($k = 92$) groups were included in the analyses ($n = 43\,992$ participants). Using bivariate mixed-effects meta-regressions, while controlling for key a priori confounders, we regressed smoking cessation on (a) three BCT groupings consistent with dual-process theory (i.e. associative, reflective motivational and self-regulatory), (b) 17 expert-derived BCT groupings (i.e. BCT taxonomy version 1 clusters) and (c) individual BCTs from the BCT taxonomy version 1. **Results**   Among person-delivered interventions, higher smoking cessation rates were predicted by BCTs targeting associative and self-regulatory processes ($B = 0.034, 0.041, P < 0.05$), and by three individual BCTs (prompting commitment, social reward, identity associated with changed behaviour). Among written interventions, BCTs targeting taxonomy cluster 10a (rewards) predicted higher smoking cessation ($B = 0.394, P < 0.05$). Moderation effects were observed for nicotine dependence, mental health status and mode of delivery. **Conclusions**   Among person-delivered behavioural smoking cessation interventions, specific behaviour change techniques and clusters of techniques are associated with higher success rates.

Keywords   Behaviour change technique, control group, dual-process theory, meta-analysis, meta-regression, smoking cessation, systematic review.

INTRODUCTION

Tobacco use is one of the leading risk factors contributing to the global burden of disease, with an estimated annual 7.1 million deaths attributable to tobacco smoking, smokeless tobacco and exposure to second-hand smoke [1]. Systematic reviews show that behavioural interventions can effectively increase rates of smoking cessation, but with substantial heterogeneity in the strengths of effects (e.g. [2–5]). Reviews already tend to focus on

interventions by specific modes of intervention delivery (e.g. [2–6]) or for specific populations (e.g. [7–9]), which suggests that other intervention characteristics may also vary between trials and drive intervention effectiveness. In this review, we examined whether variability in the potential active content (i.e. behaviour change techniques; BCTs) of the smoking cessation interventions and comparators can account for heterogeneity in intervention effects. Moreover, we developed and implemented several potentially important methodological advances that aimed to enhance the accuracy of the results obtained (namely, retrieving intervention and comparator details from authors, addressing comparator group variability, including only well-described studies, including multiple outcome time-points in single models, pre-registering analyses, reducing multiple testing, controlling for confounders and statistically testing potential moderators).

The potential active ingredients of behavioural interventions can be described using the BCT taxonomy version 1 (BCTTv1) [10]. This 93-item taxonomy was developed through a systematic review of existing BCT classification systems (identification of BCTs), followed by a rigorous and iterative process of Delphi procedures with international behaviour change experts and input from an international advisory board (refinement of BCTs) [10,11]. It is commonly used in literature syntheses to describe the active content of the reviewed behavioural interventions (e.g. [6,7,12–24]), as it has consistently demonstrated reliability of coding BCT content. Examples of BCTs are: 'advise the person to identify and compare reasons for wanting (pros) and not wanting (cons) to change the behaviour' (pros and cons) and 'prompt detailed planning of performance of the behaviour' (action planning). Through open sort and consensus procedures, the 93 BCTs have been organized into 16 clusters according to each BCT's presumed mechanism of action [10]. In this study, we use the BCTTv1 for coding and analysing the included smoking cessation interventions.

Within smoking cessation research, four previous systematic reviews have extracted BCTs from intervention descriptions and examined which BCTs were associated with smoking cessation rates [6–9]. Among these reviews, there is very little concordance between which BCTs are found to be associated with smoking cessation. This could indicate that different BCTs are effective among the different populations examined in these reviews. However, there are also other probable explanations related instead to study methodology, such as potential low power (i.e. usually fewer than 20 included trials, infrequent BCTs use), using different BCT taxonomies, primarily relying on the incomplete intervention and comparator descriptions available in published articles (65 and 74% of intervention and comparator BCTs in smoking cessation trials are not

reported) [25], not accounting for variability in comparator interventions (which contain between 0–45 BCTs, and many also receive smoking cessation medication) [26], multiple testing (e.g. testing each BCT univariately in separate models) [27] and confounding (e.g. not controlling for sample characteristics or BCT co-occurrence) [27]. There is a need for a well-powered examination of potential active ingredients of smoking cessation interventions that accounts for these methodological issues, and that also allows for formal tests of whether effects of BCTs differ for different populations, settings and modes of intervention delivery.

Theory, evidence and expert opinion to inform BCT analyses

Theories offer a useful way of organizing the large number of identified BCTs (93) into clusters, which can reduce the number of statistical tests conducted and also facilitate tests of the theory itself. In the BCTTv1, BCTs are clustered according to the mechanism by which experts agree they are affecting behaviour—resulting in 16 clusters [10]. Dual-process models introduce a high-level ordering comprised of three clusters. First, behaviour can be enacted through processes that are relatively more associative and those that are relatively more reflective (e.g. [28–30]). Associative processes are those that occur relatively automatically through encountering cues that prompt behaviour. Reflective processes are those that are relatively more conscious and effortful and drive behaviour through decisional processes. These can be thought of as two types of subprocesses: those that give direction to behaviour (reflective motivational processes) and those that control whether a person is able to enact the behaviour (self-regulatory processes). In this review, we used these three groupings of higher-order processes and the 16 BCTTv1 clusters to provide structure to the analyses, reduce the number of statistical tests conducted and generate findings that might inform theory as well as practice.

Potential moderators of BCT effectiveness

Globally, the prevalence of smoking is in decline [31]; however, rates of smoking remain substantial among specific population groups, including people with substance use disorders [32] or other mental health disorders [33], those who are highly dependent on nicotine [34] and those of low socio-economic status (SES) [35]. If effective BCTs were to be identified in this study, it would be relevant to know whether these are also associated with better outcomes for these populations. In a previous study from the current review, we found that the total number of BCTs delivered only predicted higher cessation rates in comparator groups

when delivered by a person, not in writing [26]. Other potentially important moderators are intervention mode of delivery (e.g. group versus individually) and whether or not it matters if the person is a behaviour change specialist (e.g. a psychologist versus a doctor). These analyses could provide important guidance on what type of behavioural support can best be provided to which people, how and by whom, and were therefore included as moderators in the current study.

The current study

In summary, the aims of this systematic review were (1) to identify theory- and expert-informed BCT clusters as well as individual BCTs associated with smoking cessation rates in intervention and comparator groups of smoking cessation trials; and (2) to determine whether BCT effectiveness varies among population and intervention delivery characteristics. The current study included a large sample of studies, but only analysed data from groups for which comprehensive intervention descriptions were obtained after having contacted authors to obtain any missing information. It capitalized on the information available for both the intervention and comparator groups, control for a priori-identified potential confounding variables and attempt to minimize the number of tests by clustering BCTs.

METHOD

Design

This study was part of a larger, ongoing review of smoking cessation trials ('Intervention and Comparison group support provided in SMOKing cEssation', IC-SMOKE; PROSPERO registration number CRD42015025251 [36]). The completed Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist [36] is included in Supporting information, Appendix A. The Cochrane Tobacco Addiction Group Specialised Register was searched on 1 November 2015 for randomized controlled trials assessing the impact of behavioural interventions (with or without pharmacological support) on biochemically verified smoking cessation at 6 months or longer. Trials were excluded if they were published before 1996, were not reported in English or in peer-reviewed journals or if any of the participants were aged under 18 years. Trials published before 1996 were excluded to manage work-load, because older trials of behavioural interventions are less relevant in a continually changing social and policy environment and because preliminary work indicated it was very difficult to retrieve the required materials from authors of trials published beyond 20 years earlier.

Procedure

The search and screening led to the inclusion of 142 trials containing 346 intervention and comparator groups (see Fig. 1 for the PRISMA flow diagram and Supporting information, Appendix B for the list of studies included in the review). After searching for additional published materials on included studies (e.g. supplements, protocols, intervention development papers), we contacted all study authors to send us any unpublished materials (e.g. practitioner manuals, training manuals) describing the interventions and comparators. Additionally, as authors often do not have detailed written descriptions of comparators, a purpose-built comparator group checklist was developed (https://osf.io/e834t/) and authors were requested to complete this for their comparator groups, as in de Bruin *et al.* [37]. Additional materials, whether published or unpublished, were retrieved for 288 of 346 (83%) groups.

As described previously [38], BCTs targeting quitting and abstinence were reliably extracted from all materials by two trained, independent coders using the BCTTv1 [10], with one BCT added, one BCT removed and smoking cessation examples added (see Supporting information, Appendix A for the full taxonomy used). BCTs were also extracted automatically (using a syntax) from author responses to the comparator group checklist. If the corresponding intervention group in a trial received the comparator plus additional support, then the comparator group checklist BCTs were assumed to be present in both the comparator and intervention groups. If a BCT was present and targeted either quitting smoking (making the initial attempt) or maintaining abstinence (sustaining their quit success), it was scored 1 (present); if not, it was scored 0 (absent).

We sought to group individual BCTs to reduce the issue of multiple testing and to provide results that might inform or support behaviour change theory. The BCTs have previously been organized into 16 clusters through an open sort task with behaviour change experts [10]. Through team discussion, we organized the 16 clusters of BCTs from BCTTv1 into three higher-order, dual-process theory processes (namely, associative, reflective motivational and self-regulatory) (e.g. [28–30]). This involved splitting one cluster into two, for a total of 17 clusters organized under three processes (see Supporting information, Fig. A1 for the groupings). Sum scores of the number of BCTs targeting the three processes and 17 clusters were computed.

We agreed that these analyses should be based only on comprehensive intervention descriptions. Interventions were labelled as well-described if two independent coders judged the materials to be of sufficient detail and clarity to identify all or almost all the BCTs that were delivered to that group (see Supporting information, Fig. A2 for
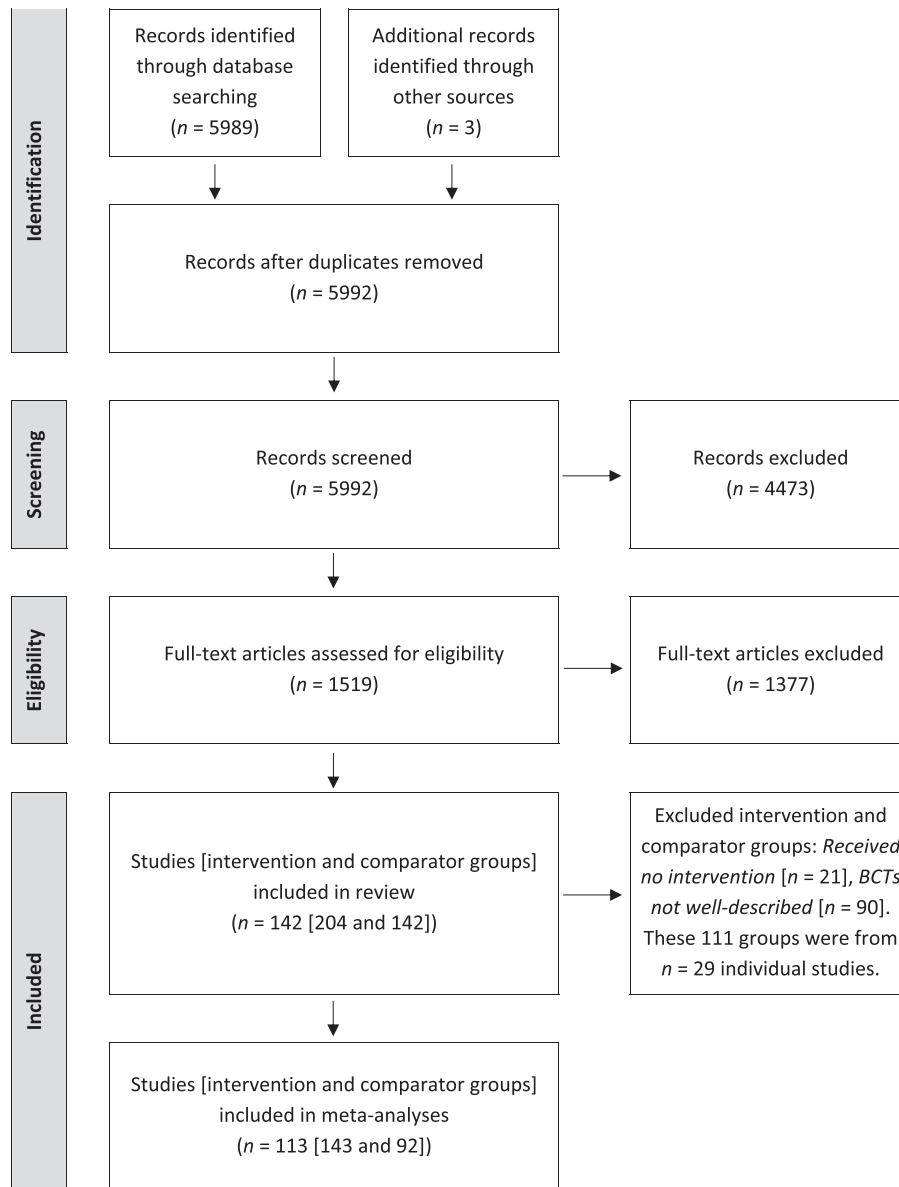
Figure 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram. BCTs = behaviour change techniques

decision tree used). This coding was reliable [prevalence and bias adjusted kappa (PABAK) = 0.79; cf. Altman's guidelines that values of 0.61–0.80 indicate good reliability [39]). A total of 256 of 346 (74%) intervention and comparator groups were rated as 'well-described', of which 206 received the intervention/comparator from a person, 29 received the intervention/comparator in writing (i.e. in print and/or digitally) and 21 did not receive an intervention (i.e. were passive). All (206 + 29 =) 235 well-described, active intervention (k=143) and comparator (k=92) groups were included in the current analyses. This includes $n = 43\,992$ participants from 113 trials and 393

smoking cessation outcomes (all biochemically verified assessments at 6 months or longer).

Analyses

Overview

The outcome of interest was the logit-transformed smoking cessation rate within groups (transformed to approximate a normal sampling distribution), with missing participants considered to be smoking [40]. Outcome time-points were all those at 6 months[a] post-randomization or later (i.e.

[a]To allow for practical variation between studies in the exact time at which follow-up assessments were conducted, we also included those studies for which the '6-month' follow-up occurred slightly earlier—anywhere from 5 months.

multiple time-points per study were permitted). Analyses were performed using mixed-effects (multi-level) meta-regression models, with random effects for trials (to account for between-trial heterogeneity), random effects for groups within trials (to account for between-group heterogeneity within trials), correlated random effects for multiple outcomes (i.e. logit rates) within groups using a continuous-time autoregressive structure (to account for heterogeneity across time within groups) and correlated sampling errors for multiple outcomes within studies (to account for the dependency between multiple observations corresponding to the same group). For the sampling errors, we conservatively assumed an autocorrelation coefficient of $\rho = 0.9$ for a lag of 1 month. In essence, we combined a multi-level model (with random effects for studies and groups) [41] with a model that accounts for the dependencies in multiple observed cessation rates for the same group (when there are multiple follow-ups) using an autoregressive structure [42,43], except that these authors proposed the use of an AR(1) structure with fixed time-points. Instead, we used a continuous-time autoregressive structure, which is more appropriate here, as the months of follow-up are not evenly spaced in the present case. Analyses were conducted using the metafor package in R [44] and the analysis plan was published on Open Science Framework before conducting the analyses (https://osf.io/m5vea/). The analysis script and data (https://osf.io/tfb2p/) are also available on Open Science Framework.

Predictor variables (BCTs, clusters and groupings)

Step 1   *(planned)*. At the highest grouping, the independent variables were the number of BCTs used to target associative, reflective motivational and self-regulatory processes. These groupings were tested in three separated models due to high intercorrelations (and therefore potential multi-collinearity; range of correlations: 0.65–0.90, all $P < 0.001$; full correlation tables are provided in the Supporting information, Appendix B). Models were run separately for interventions and comparators primarily delivered by a person and those delivered in writing (i.e. print and/or digital), as per our expectation that primarily those BCTs delivered by a person would be effective. Print and digitally delivered interventions and comparators were not considered separately due to the low numbers of these types.

The primary models assume linearity. As a check, among the person-delivered interventions and comparators, non-linear relationships between the associative, reflective motivational and self-regulatory processes and cessation were also examined using restricted cubic splines [45]. Five knots were used at 0, 3, 6, 9 and 12 BCTs, yielding one coefficient for the linear association and three coefficients allowing for non-linear relationships. A joint test of the latter coefficients was conducted to examine if there is evidence of non-linearity.

Step 2   *(planned)*. Next, the independent variables used were the number of BCTs targeting each of the 17 BCT clusters. These were also tested in separate models to avoid multi-collinearity and run separately for person-delivered and written interventions and comparators.

Step 3   *(planned)*. Whenever any of the 17 BCTTv1 clusters was predictive of smoking cessation at $\alpha = 0.1$, we explored which specific BCTs were driving the association by entering all BCTs from that cluster as predictors in one model. This approach was favoured over univariate BCT analyses, as it substantially reduced the number of tests conducted—thereby probably reducing the Type 1 error rate—and controlled for confounding of the use of different BCTs from the same cluster. If BCTs to be entered into the same model were highly correlated, we combined these into a single predictor. This occurred in one case (BCTs 12.1, 12.2 and 12.3 were highly correlated). In all analyses, independent variables were only tested if they contained reasonable variability (e.g. use and non-use in at least 10 groups for individual BCTs).

Step 4   *(unplanned; suggested by reviewer)*. Next, we repeated steps 1–3 while controlling for the total remaining amount of support (number of other BCTs) delivered. These analyses determine whether associations seen between the above predictors and smoking cessation are due to unique effects of those predictors or to their correlations with the total number of BCTs delivered.

Step 5   *(in the original review protocol [46] and added to this paper following reviewer suggestion)*. The above steps describe a theoretical approach to identifying effective components. The final step was an empirical approach. We conducted principal components analysis on all BCTs delivered at least 10 times in the person-delivered interventions and comparators. Principal components analysis was chosen over factor analysis, as the BCTs within a given factor were viewed to define that factor rather than being a result of an immeasurable underlying factor [47]. Direct oblimin rotation was used to permit correlation between factors [48]. All factors above

the break in the scree plot were extracted [48]. The resultant factor scores were used as predictor variables.

Moderator and control variables

Moderator variables were selected based on discussion with our project advisory board, literature review and team discussions. Identified variables were population characteristics and intervention/comparator delivery characteristics that might moderate the BCT–cessation relationships (see Table 1). Whenever there was reasonable variability in a moderator variable, its interaction with those independent variables described in step 2 (above) were tested. Moderation analyses were conducted among the 206 person-delivered interventions and comparators, as there were very few (29) written interventions and comparators. Sample sizes for these moderator analyses were 180–206 groups, due to missing data on some of the moderators.

Our analyses involved predicting smoking cessation rates (i.e. at the group level), rather than the traditionally used effect sizes of difference/ratios between groups. Given this, through a literature review we identified control variables that may vary between trials and impact smoking cessation (see p. 4 of our analysis plan: https://osf.io/m5vea/). These variables (in Table 1) were included in all analyses.

Table 1 Variables used as moderator and/or control variables in analyses.

Variable	Description	Coding	Type
Mode of intervention delivery	The primary mode of delivery of the person-delivered interventions	1 = group, 0 = individual	Moderator
Provider behaviour change expertise	Was the intervention provider a behaviour-change 'expert'? We considered experts to be those with primary training in this area (e.g. psychologists, counsellors, health educators) versus those with primary training in other areas (e.g. physicians, nurses). Note that while some nurses are experts in behaviour change, this is not the norm	1 = yes, 0 = no	Moderator
Nicotine dependence	Level of nicotine dependence as assessed on the Fagerström Test for Nicotine Dependence [49]. Missing values were imputed based on cigarettes per day scores where available	0–10. Higher = greater nicotine dependence	Moderator
Intention to quit smoking	Was it explicit in the trial inclusion criteria that participants had to want to quit smoking in order to participate?	1 = yes, 0 = no	Moderator
Low socioeconomic status	Were most (≥ 50%) of the sample of low socio-economic status? Determined based on author report (e.g. if authors characterized their sample as 'disadvantaged', 'deprived', 'low income', etc.) and on the income and/or education levels reported	1 = yes (low), 0 = no (medium–high)	Moderator
Physical health condition	Did most (≥ 50%) of the sample have an ongoing physical health condition?	1 = yes, 0 = no	Moderator
Mental health condition	Did most (≥ 50%) of the sample have a mental health condition?	1 = yes, 0 = no	Moderator and control
Acute stop-smoking trigger	Did most (≥ 50%) of the sample experience an acute trigger that could prompt them to quit smoking (e.g. pregnancy, hospitalization)?	1 = yes, 0 = no	Moderator and control
Length of follow-up	The length of time post-randomization at which abstinence was assessed	Log-transformed	Control
Cotinine verification	Was cotinine verification used to assess abstinence? Note: all studies used some form of biochemical verification. Those scoring 'no' on this variable typically used CO	1 = yes, 0 = no	Control
Type of abstinence assessed	Whether sustained or point prevalence abstinence was used	1 = sustained abstinence, 0 = point prevalence abstinence	Control
Pharmacological support	Did participants receive any stop-smoking medication (e.g. NRT, varenicline)?	1 = yes, 0 = no	Control
Adjuvant interventions	Did participants receive any adjuvant interventions (e.g. hypnosis, support for alcohol consumption, diet, exercise) that was hypothesized by the authors to increase smoking cessation but that was otherwise not captured by the coded behaviour change techniques?	1 = yes, 0 = no	Control

CO = carbon monoxide; NRT = nicotine replacement therapy.

RESULTS

Descriptive statistics

Study characteristics

The intervention and comparator group samples included in the current analyses were, on average, aged 42.52 years [standard deviation (SD) = 9.03)], 48.8% (SD = 25.4%) female and moderately dependent on nicotine (mean$_{\text{FTND}}$ = 4.96, SD = 0. 94). Of the 235 well-described groups, most were from North America (k = 180, 77%; primarily United States), followed by Europe (k = 37, 16%; primarily United Kingdom), Asia (k = 12, 5%) and Oceania (k = 6, 3%). Most (k = 134, 57%) received smoking cessation medication. The minority of groups receiving these interventions and comparators were of low SES (k = 56, 24%), were experiencing a physical (k = 32, 14%) or mental health problem (k = 34, 14%) or had experienced an acute stop-smoking trigger (e.g. pregnancy, heart attack) (k = 28, 12%). For participants in most groups (k = 145, 62%), intention to quit was not reported to be a prerequisite for entry into the trial (i.e. participants were permitted to participate regardless of whether or not they intended to quit smoking).

Intervention and comparators characteristics

Table 2 (left side) shows the descriptive statistics for the number of BCTs used to target the 3 and 17 BCT clusters. Associative, reflective motivational and self-regulatory processes tended to be targeted using similar numbers of BCTs, on average. The most frequently used clusters were 12 (antecedents), 5 (natural consequences) and 1 (goals and planning), whereas the least frequently used were 16 (covert learning), 10b (incentives), 14 (scheduled consequences) and 3 (social support).

Associations between intervention and comparators content and smoking cessation

Associative, reflective motivational and self-regulatory BCTs

When delivered by a person, each of associative, reflective motivational and self-regulatory process BCTs were associated with higher smoking cessation; however, when controlling for total BCTs, only associative and self-regulatory BCTs remained as significant predictors (Table 2, right side). Results of the restricted cubic spline models indicated no significant departure from linearity (P = 0.10, P = 0.64 and P = 0.26, respectively). When delivered in writing, the associative, reflective motivational or self-regulatory process BCTs were not significantly associated with smoking cessation.

BCT taxonomy clusters

Among person-delivered interventions and comparators, the numbers of BCTs in 12 of 16 tested BCT clusters were associated with higher smoking cessation rates; however, none of these remained significant when controlling for total BCTs (Table 2, right). In fact, cluster 10b predicted lower smoking cessation, when controlling for total BCTs. Among interventions and comparators delivered in writing, none of the 13 tested BCT clusters were associated with higher smoking cessation rates, except when controlling for total BCTs. Cluster 10a predicted higher smoking cessation when controlling for total BCTs.

Individual BCTs

In the BCT clusters associated with smoking cessation at $P < 0.1$, we tested all individual BCTs that had a frequency of (use and non-use of) at least 10. When controlling for the other BCTs in the clusters, 17 individual BCTs were associated with increased smoking cessation when delivered by a person (see Table 3 and Supporting information, Appendix B; all $P < 0.05$). When controlling for total BCTs, four individual BCTs predicted higher smoking cessation and two predicted lower smoking cessation (Table 3). No individual BCTs predicted increased smoking cessation when delivered in writing. Supporting information, Appendix B presents examples of applications of those BCTs that were significantly associated with increased smoking cessation.

The principal components analysis of individual BCTs yielded three factors: 23 individual BCTs strongly (> 0.5) loaded on factor 1, 7 on factor 2 and 6 on factor 3 (Supporting information, Appendix B). When these three factor scores were entered into a meta-regression model together, factors 1 and 3 significantly predicted higher smoking cessation (Supporting information, Appendix B). Table 3 shows the BCTs that strongly loaded on factors 1 and 3.

Moderators: population and intervention delivery characteristics

Of all tested interaction effects, only three were significant ($P < 0.05$). Cluster 9 (comparison of outcomes) was more strongly associated with smoking cessation among those with higher (versus lower) baseline nicotine dependence [B = 0.194 (0.053, 0.335), P = 0.007] and when delivered in a group (versus individual) setting [B = 0.324 (0.049, 0.599), P = 0.021]. Cluster 11 (regulation) was more strongly associated with smoking cessation among those without (versus with) a mental health condition [B = −0.310 (−0.619, −0.001), P = 0.049]. All model outputs are provided in the Supporting information.

Table 2 Mean (SD) and range of behaviour change techniques used targeting three theoretical processes and 17 clusters (left) and regression coefficients (*B*, 95% CI) for the prediction of (logit-transformed) smoking cessation rates from number of behaviour change techniques targeting three theoretical processes and 17 clusters (right), by mode of delivery.

	Descriptive statistics			Associations (B, 95% CI) with smoking cessation rates	
	Mean (SD)	Range	Theoretical range	Not controlling for total BCTs	Controlling for total BCTs
Interpersonal					
Associative processes	5.23 (3.99)	0–15	0–39	**0.052 (0.034, 0.071)*****	**0.034 (0.004, 0.065)***
7. Associations	0.32 (0.57)	0–4	0–8	**0.210 (0.086, 0.334)*****	0.068 (−0.061, 0.197)
8. Repetition and substitution	1.15 (1.21)	0–5	0–6	**0.118 (0.052, 0.185)*****	−0.017 (−0.099, 0.066)
10a. Rewards	1.05 (1.02)	0–4	0–6	**0.209 (0.126, 0.292)*****	0.077 (−0.040, 0.195)
12. Antecedents	2.53 (1.76)	0–5	0–6	**0.111 (0.070, 0.151)*****	0.047 (−0.020, 0.115)
14. Scheduled consequences	0.16 (0.49)	0–3	0–10	0.088 (−0.084, 0.261)	−0.040 (−0.186, 0.106)
16. Covert learning	0.03 (0.17)	0–1	0–3	–	–
Reflective motivational processes	5.89 (3.48)	0–14	0–26	**0.055 (0.029, 0.080)*****	−0.004 (−0.041, 0.034)
5. Natural consequences	2.30 (1.38)	0–5	0–6	**0.108 (0.046, 0.170)*****	0.006 (−0.064, 0.076)
6. Comparison of behaviour	1.08 (0.72)	0–3	0–3	0.101 (−0.023, 0.224)	−0.071 (−0.196, 0.054)
9. Comparison of outcomes	0.83 (0.68)	0–2	0–3	**0.163 (0.034, 0.292)*ᵃ**	0.018 (−0.112, 0.149)
10b. Incentives	0.08 (0.27)	0–1	0–5	−0.180 (−0.564, 0.205)	**−0.402 (−0.749, −0.054)***
13. Identity	0.88 (0.98)	0–3	0–5	**0.182 (0.095, 0.268)*****	0.030 (−0.082, 0.141)
15. Self-belief	0.73 (0.82)	0–4	0–4	**0.151 (0.030, 0.272)***	−0.022 (−0.150, 0.105)
Self-regulatory processes	6.07 (3.55)	0–15	0–27	**0.060 (0.041, 0.078)*****	**0.041 (0.009, 0.073)***
1. Goals and planning	2.34 (1.49)	0–6	0–9	**0.117 (0.073, 0.161)*****	0.050 (−0.020, 0.119)
2. Feedback and monitoring	1.03 (1.05)	0–5	0–7	**0.187 (0.099, 0.275)*****	0.074 (−0.028, 0.176)
3. Social support	0.17 (0.43)	0–2	0–2	0.188 (−0.013, 0.389)†	−0.096 (−0.309, 0.118)
4. Shaping knowledge	0.93 (0.80)	0–3	0–5	**0.156 (0.050, 0.262)****	−0.063 (−0.195, 0.070)
11. Regulation	1.60 (1.02)	0–3	0–4	**0.199 (0.108, 0.290)***ᵃ**	0.081 (−0.019, 0.181)
Written					
Associative processes	4.76 (3.00)	0–13	0–39	−0.025 (−0.111, 0.061)	0.146 (−0.037, 0.330)
7. Associations	0.55 (0.63)	0–2	0–8	−0.187 (−0.591, 0.217)	0.008 (−0.656, 0.672)
8. Repetition and substitution	0.93 (0.92)	0–3	0–6	−0.014 (−0.356, 0.328)	0.266 (−0.201, 0.734)
10a. Rewards	0.55 (0.69)	0–3	0–6	0.131 (−0.207, 0.469)	**0.394 (0.024, 0.764)***
12. Antecedents	2.48 (1.50)	0–5	0–6	−0.127 (−0.305, 0.051)	−0.130 (−0.389, 0.129)
14. Scheduled consequences	0.10 (0.31)	0–1	0–10	–	–
16. Covert learning	0.14 (0.35)	0–1	0–3	–	–
Reflective motivational processes	5.62 (3.18)	1–13	0–26	−0.074 (−0.165, 0.017)	−0.129 (−0.318, 0.060)
5. Natural consequences	2.28 (1.36)	0–4	0–6	−0.279 (−0.566, 0.008)†	−0.270 (−0.600, 0.060)
6. Comparison of behaviour	1.17 (0.97)	0–3	0–3	−0.085 (−0.334, 0.164)	−0.008 (−0.306, 0.291)
9. Comparison of outcomes	0.79 (0.90)	0–3	0–3	−0.250 (−0.633, 0.133)	−0.142 (−0.693, 0.408)
10b. Incentives	0.03 (0.19)	0–1	0–5	–	–
13. Identity	0.76 (0.79)	0–3	0–5	−0.226 (−0.567, 0.114)	−0.153 (−0.773, 0.467)
15. Self-belief	0.59 (0.78)	0–2	0–4	0.085 (−0.396, 0.566)	0.407 (−0.074, 0.887)†
Self-regulatory processes	5.52 (2.98)	0–14	0–27	−0.061 (−0.151, 0.029)	−0.040 (−0.262, 0.182)
1. Goals and planning	1.97 (1.24)	0–6	0–9	−0.038 (−0.240, 0.163)	0.237 (−0.129, 0.602)
2. Feedback and monitoring	0.86 (0.92)	0–3	0–7	−0.070 (−0.431, 0.291)	0.282 (−0.287, 0.851)
3. Social support	0.10 (0.31)	0–1	0–2	–	–
4. Shaping knowledge	1.14 (0.69)	0–2	0–5	−0.320 (−0.714, 0.075)	−0.281 (−0.735, 0.172)
11. Regulation	1.45 (1.15)	0–3	0–4	−0.225 (−0.505, 0.056)	−0.198 (−0.521, 0.125)

Significant (*p* < .05) associations are presented in bold. ᵃAn interaction effect occurred for this variable. Please see Supporting information, Appendix B for the coefficient at different levels of the moderator. Sample size for meta-regression analyses: interpersonal: 206 groups with 344 outcomes, written: 29 groups with 49 outcomes. Cells with '–' indicate that variability was too low in the predictor to test this relationship (as determined a priori). All models were controlled for provision of medication, interventions targeting additional behaviours, length of follow-up, cotinine verification, abstinence type, mental health conditions and health triggers (as per the a priori-defined analysis plan: https://osf.io/m5vea/). SD = standard deviation; CI = confidence interval; BCT = behaviour change technique. ***P < 0.001; **P < 0.01; *P < 0.05; †*P* < 0.1.

DISCUSSION

Overview of findings

This systematic review examined associations between BCTs and smoking cessation, by applying and extending recommended methods for enhancing confidence in BCT–outcomes associations identified through meta-regression analyses in systematic reviews [27]. Among 235 intervention and comparator groups from 113 trials throughout a wide range of populations, modes

Table 3 Individual behaviour change techniques shown to be associated with smoking cessation (when delivered interpersonally) in at least one analysis.

		Individual BCTs within each BCT Taxonomy cluster that predicted smoking cessation		BCTs strongly loading on factor scores that predicted smoking cessation
		Not controlling for total BCTs	Controlling for total BCTs	
1.1	Goal setting (behaviour)	NS	NS	+
1.2	Problem solving	NS	NS	+
1.5	Review behaviour goal(s)	NS	NS	+
1.9	Commitment	+	+	+
2.2	Feedback on behaviour	NS	+	NS
2.6	Biofeedback	NS	NS	+
2.7	Feedback on outcome(s) of behaviour	+	NS	NT
3.2	Social support (practical)	+	NS	+
4.1	Instruction on how to perform the behaviour	+	NS	+
5.2	Salience of consequences	+	NS	+
5.3	Information about social/environmental consequences	+	NS	+
5.6	Information about emotional consequences	NS	−	+
7.3	Reduce prompts/cues	+	NS	NS
8.1	Behavioural practice/rehearsal	+	NS	+
8.2	Behaviour substitution	NS	NS	+
8.6	Generalization of target behaviour	NS	NS	+
9.1	Credible source	NS	NS	+
9.2	Pros and cons	+	NS	+
10.3	Non-specific reward	NS	NS	+
10.4	Social reward	+	+	+
10.9	Self-reward	NS	NS	+
10.10	Reward (outcome)	+	NS	NT
11.2	Reduce negative emotions	+	NS	+
11.3	Conserving mental resources	NS	NS	+
12.1	Restructuring the physical environment	+	NS	+
12.2	Restructuring the social environment	+	NS	+
12.3	Avoidance/reducing exposure to cues for the behaviour	+	NS	+
12.4	Distraction	NS	NS	+
13.1	Identification of self as role model	NS	NS	+
13.2	Framing/reframing	NS	−	+
13.5	Identity associated with changed behaviour	+	+	+
15.3	Focus on past success	NS	NS	+
15.4	Self-talk	+	NS	+

BCT = behaviour change technique; + = indicates the BCT predicts higher smoking cessation; − = indicates the BCT predicts lower smoking cessation; NS = non-significant; NT = not tested. Odds ratios (95% confidence interval) for those BCTs significantly predictive of smoking cessation when controlling for total support are as follows: 1.9: 1.30 (95% CI = 1.02, 1.67); 2.2: 1.30 (95% CI = 1.02, 1.67); 5.6: 0.78 (95% CI = 0.65, 0.94); 10.4: 1.22 (95% CI = 1.02, 1.47); 13.2: 0.79 (95% CI = 0.63, 0.99); 13.5: 1.34 (95% CI = 1.08, 1.67). All models were controlled for provision of medication, interventions targeting additional behaviours, length of follow-up, cotinine verification, abstinence type, mental health conditions and health triggers (as per the a priori-defined analysis plan: https://osf.io/m5vea/).

of delivery and providers, more extensive use of theoretically effective BCTs targeting associative and self-regulatory processes predicted higher smoking cessation rates. Examining the BCTs targeting the BCTTv1 clusters—which represent different presumed mechanisms of action—we found that, when delivered by a person, none predicted higher smoking cessation rates while controlling for the total support delivered. When delivered in writing, cluster 10a (rewards) predicted higher smoking cessation

rates. We also identified three individual BCTs that consistently predicted higher smoking cessation rates across analyses and while controlling for potential confounders (viz. total support delivered and those control variables listed in Table 1). Notably, these BCTs were only associated with smoking cessation when delivered by a person (individually, group, telephone), not when delivered in writing (printed materials, web-based interventions and comparators). Examining other potential moderators, we found

little evidence that effects of BCTs vary for the different populations, settings and delivery characteristics examined. Hence, this expansive meta-analysis has identified that smoking cessation interventions and comparators that more extensively target (with more BCTs) pathways to behaviour change consistent with theory are more effective than those that target these pathways less extensively (with fewer BCTs), and has yielded three individual BCTs that might be particularly effective for person-delivered interventions across populations and settings.

Key interpretations

Theories (e.g. [28–30]) suggest that associative, reflective motivational and self-regulatory processes are relevant to successful behaviour change. The current meta-analyses provide strong support for these building blocks of smoking cessation programmes. In particular, associative and self-regulatory processes appeared to be the primary predictors, as these remained significant when controlling for the total support delivered. This is in line with theorizing about maintenance of behaviour change, which broadly suggests that associative and self-regulatory processes, rather than reflective processes, are particularly important for long-term behaviour change such as abstinence at 6 months and longer [50].

Regarding individual BCTs, to maximize confidence in our findings, we used both theory- and data-informed approaches to identifying those that predict higher smoking cessation. Results identified 29 individual BCTs as potentially important predictors of smoking cessation in at least one analysis. Of these, three consistently predicted higher smoking cessation rates (prompting commitment, social reward and identity associated with changed behaviour). Further, the factor analytical approach yielded three sets of BCTs, two of which independently predicted higher smoking cessation. These two specific combinations of individual BCTs (listed in Supporting information, Appendix B) might show particular promise in interventions to increase smoking cessation. Together, these findings run counter to the notion that it is exclusively non-specific elements of therapeutic relationships (e.g. warmth, empathy and genuineness) that drive effectiveness. Future trials could examine whether adding any of the BCTs identified here to existing interventions increases smoking cessation rates.

While we found extensive support for the relevance of person-delivered BCTs, we found little evidence that BCTs delivered in writing were associated with higher smoking cessation. The exception to this was taxonomy cluster 10a (rewards), which predicted higher smoking cessation rates when delivered in writing. This finding contributes to a mixed literature on the associations between the number of BCTs delivered digitally and behavioural outcomes, with some studies finding positive effects [7,51] and others

finding no effects [12,13,52]. Previous discordant findings might be explained by the use of specific effective BCTs in trials in some, but not other, reviews (i.e. perhaps those reviews finding an association included trials that used specific effective BCTs, whereas those that found no association included trials that primarily used specific ineffective BCTs); or by differences in any of the methodological issues that we tried to overcome in this review (e.g. incomplete intervention and comparator reporting, not addressing confounding). Nonetheless, it might also be that the current analyses of predictors among written interventions were underpowered to detect true associations or that it is more difficult (and therefore less reliable) to identify BCTs in written materials. Further, low adherence to written interventions might have lessened the observed BCT–cessation relationships. Given that written smoking cessation interventions have been shown to increase smoking cessation in some randomized controlled trials [4,5,53], it would be useful to investigate more in-depth what makes these written interventions effective, potentially first focusing on rewards and/or tailoring [53].

Regarding moderation effects, in most cases there was no evidence that the associations of clusters of BCTs with smoking cessation varied depending on the population to whom they were delivered, the provider who delivered the content or whether they were delivered in a group versus individual setting. It is possible that some of these analyses were underpowered to detect true interaction effects, although we attempted to mitigate this by pre-specifying which interaction effects to test, based on judgements of sufficient distributions of the variables to be tested (p. 9, https://osf.io/m5vea/). Certainly, the (near-)absence of significant interaction effects should not be taken as evidence that BCT–smoking cessation associations are consistent among population and intervention characteristics. Rather, results simply do not lend strong support for modifying the types of BCTs delivered depending on the population, provider or group versus individual setting.

Strengths and limitations

Particular strengths of this study are the large number and diverse range of studies included, the restriction to objectively verified outcomes, which should protect against multiple sources of bias [54,55], the inclusion of comparator interventions, addressing incomplete reporting by successfully retrieving an extensive amount of additional information from study authors, the inclusion of only well-described intervention and comparators in the analyses and the a priori-specified statistically advanced analysis plan that included the use of multivariate models to minimize the risk of confounding.

The main study limitations are that, first, the analyses are still correlational, despite the careful multivariate

approach. Additionally, the attempt to minimize the number of tests and make the analyses theoretically informative required grouping BCTs under three processes. This was based on theory and team discussion, rather than on a formal expert consensus procedure. Other teams may have ended up with other clusters. Thirdly, the external validity of our findings is restricted in some ways, given that most trials were conducted in high-income, western countries with participants who were not required to intend to quit smoking in order to participate. Fourthly, while our models and analyses were quite elaborate, they cannot fully account for the complexity of interventions that are delivered. Machine learning approaches (see www.humanbehaviourchange.org) and a stronger evidence-base for developing a priori hypotheses about synergistic, antagonistic or ordering effects between BCTs might partially overcome these limitations in future research. Fifthly, only randomized controlled trials were included. A more complete analysis might also include observational studies. Lastly, there are other potentially important moderators or control variables, such as intervention fidelity and therapist relationship, that should be considered, but were not included in the models due to infrequent reporting.

CONCLUSIONS

This systematic review of smoking cessation interventions and comparators examined associations between BCTs and smoking cessation, while applying and extending methods for enhancing confidence in the validity of BCT–outcomes associations in systematic reviews of behavioural interventions. The analyses provide support for behavioural interventions and comparators delivered by a person for smoking cessation and for an association between the number of BCTs targeting associative and self-regulatory processes and higher smoking cessation rates. The analyses also identified three individual, promising BCTs that might be particularly effective in promoting smoking cessation.

Registration

PROSPERO (CRD42015025251) and the Open Science Framework (analysis plan: https://osf.io/m5vea/).

Declaration of interests

R.W. undertakes research and consultancy for companies that develop and manufacture smoking cessation medications (Pfizer, J&J and GSK). R.W. is an unpaid advisor to the UK's National Centre for Smoking Cessation and Training. N.B. and M.C.E.'s salaries were funded by Cancer Research UK. All other authors declare that they have no conflicts of interest.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Data S1 Supporting Information.