Long-term changes in delay discounting following a smoking cessation treatment for patients with depression

Ángel García-Pérez[1], Guillermo Vallejo-Seco[1], Sara Weidberg[1], Alba González-Roz[1] and Roberto Secades-Villa[1]

[1]Department of Psychology. University of Oviedo, Plaza Feijoo, s/n, 33003 Oviedo, Spain.

Corresponding author:

Ángel García-Pérez

Facultad de Psicología – Universidad de Oviedo

Plaza Feijoo s/n 33003 – Oviedo – Spain

Phone: +34-98-5104189

Email: garciaperangel@uniovi.es

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Abstract

Background: Delay discounting (DD) has been identified as a trans-disorder process underlying addictive behaviors, including smoking. Previous studies have evaluated how different treatments for drug dependence have affected DD, showing mixed results. Furthermore, no study has examined the effects of changes in depression on DD rates. The aim of this study was to evaluate the impact of treatment type: cognitive behavioral treatment (CBT), CBT + behavioral activation (BA) or CBT + BA + contingency management (CM), and changes in smoking status and depression on DD rates in long-term follow-up among a sample of treatment-seeking smokers with depression. **Methods**: Participants were 180 treatment-seeking smokers with depression who were randomly assigned to one of the following treatment conditions: CBT (n = 60), CBT + BA (n = 60), and CBT + BA + CM (n = 60). Depressive symptomatology and major depression diagnosis were evaluated through the BDI-II and the SCID-I of the DSM-IV-TR. DD rates were assessed using the DD task with hypothetical monetary rewards. Smoking status, DD, and depressive symptomatology were collected at baseline, at end-of-treatment and at one-, two-, three- and six-month follow-ups. **Results**: CM for smoking cessation reduces DD rates (p = .0094). Smoking abstinence (p = .0024) and reduction in depressive symptoms (p = .0437) were associated with decreases in DD rates in long-term follow-up. **Conclusions**: CM interventions for smoking cessation, smoking abstinence, and the improvement of depression contribute to reductions in DD over time.

Keywords: Smoking, Delay discounting, Depression, Contingency management,

1. Introduction

Delay discounting (DD) has been identified as a trans-disorder process underlying addictive behaviors, including smoking (Amlung et al., 2019; Bickel et al., 2019). For instance, the preference for smaller rewards now instead of larger rewards in the future (i.e. high DD) has been systematically related to the onset of smoking (Audrain-McGovern et al., 2009), greater severity of nicotine dependence (ND) (Amlung et al., 2017), lower smoking cessation rates (Loree et al., 2015), and a higher probability of relapse once abstinence has been achieved (González-Roz et al., 2019). Most of the evidence accumulated on DD is related to the predictive validity of DD, suggesting that DD has an etiological role both in the development of smoking (Audrain-McGovern et al., 2009), and in the clinical course of addiction (Bickel et al., 2014). This fact, together with the temporal stability of DD (Beck and Triplett, 2009; Kirby, 2009; Martínez-Loredo et al., 2017; Weafer et al., 2013), led some authors to conclude that DD could behave more as a causal variable (Audrain-McGovern et al., 2009; De Wilde et al., 2013) than as a state variable. Nevertheless, other studies have found how different contextual variables (e.g., gambling contexts, nicotine deprivation, alcohol intoxication, etc.) can affect DD rates (Dixon et al., 2006; Mitchell, 2004; Ortner et al., 2003; Scholten et al., 2019; Sofis et al., 2017), highlighting the modifiability of this impulsivity variable that does not always behave as a trait. These findings alongside the conceptualization of DD as a trans-disorder process elevate the importance of understanding and developing treatments that reduce DD rates (Felton et al., 2019; Koffarnus et al., 2013). Since DD is associated with important clinical phenomena such as worse treatment outcomes and relapse (Bickel et al., 2014),

examining whether certain treatments are effective in reducing DD is relevant, as these reductions could ultimately enhance enduring drug abstinence rates.

Previous studies that assessed DD changes caused by interventions in individuals with substance use disorders (SUD) have found mixed results (most studies focused on smokers). Specifically, studies that examined the impact of a 12-step program combined with other techniques (Aklin et al., 2009; Littlefield et al., 2015), nicotine replacement therapy (Dallery and Raiff, 2007), cognitive behavioral treatment (CBT) plus motivational interviewing (MI) (De Wilde et al., 2013), brief MI intervention (Dennhardt et al., 2015), and physical exercise (Kurti and Dallery, 2014), did not find changes in DD rates. In contrast, all interventions using episodic future thinking (EFT) found a significant reduction in impulsive decision making (Chiou and Wu, 2016; Snider et al., 2016; Stein et al., 2018; Stein et al., 2016). Two other studies found a decrease in DD after receiving money management interventions (Black and Rosen, 2011) and CBT (Secades-Villa et al., 2014). Finally, most of the studies that included contingency management (CM) found lower rates of DD after receiving the intervention (Hughes et al., 2017; Landes et al., 2012; Weidberg et al., 2015a; Weidberg et al., 2015b; Yi et al., 2008), although other studies found no change (Peters, et al., 2013; Yoon et al., 2009; Yoon et al., 2007). It should also be noted that the only type of intervention that has obtained better outcomes in reducing DD is EFT, which is the only approach designed to affect DD rates directly, rather than focusing on DD modifications through behavioral or symptom change.

An important limitation of these studies is that the vast majority did not include results beyond post-treatment. In fact, only four studies examined the long-term effect of the intervention at six (Weidberg et al., 2015a; Weidberg et al., 2015b; Yoon et al.,

2007) and twelve months (Secades-Villa et al., 2014). On the other hand, only four studies examined the impact of abstinence on DD. Three of them found that abstinent individuals showed a reduction in DD (Hughes et al., 2017; Secades-Villa et al., 2014; Weidberg et al., 2015a), while Landes et al. (2012) found a trend in the same direction, but it was not statically significant in decreasing DD.

Of the previous studies, there was only one investigation that included individuals with psychopathological comorbidity. Specifically, this study found that abstinent individuals with depressive symptoms reduced their DD more than smokers did (Weidberg et al., 2015a). However, the aforementioned study did not evaluate how changes in depression over time could affect DD rates and it did not include participants diagnosed with major depressive disorder (MDD) or any other psychopathological disorder. Studying these types of comorbidities is truly important, since psychopathological disorders in general, and depression in particular, are closely linked to substance use (Grant et al., 2004; Lai et al., 2015). Moreover, individuals with MDD and other psychopathologies also have higher rates of DD than individuals without such disorders (Amlung et al., 2019; Engelman et al., 2013; García-Pérez et al., 2019; Pulcu et al., 2014), which could help explain the poor cessation outcomes found among individuals presenting these comorbidities (Bakken et al., 2007; Pettinati et al., 2015; Tidey and Miller, 2015). To date, only one study has explored how depression treatment impacts DD, showing no post-treatment effects on DD (Teti-Mayer et al., 2019). Although there is scarce evidence in this regard, Pulcu et al. (2014) found that DD rates of individuals with remitted MDD were equivalent to healthy controls, so the treatment of this psychopathology may help to reduce DD in individuals with MDD.

The current study aims to overcome some of the limitations of previous studies by assessing the impact of the type of treatment (CBT, CBT + behavioral activation–BA–or CBT + BA + CM), changes in smoking status, and changes in depression on DD rates among a sample of treatment-seeking smokers with depression.

2. Material and methods

2.1 Participants

This secondary analysis is derived from a randomized controlled trial [redacted] of the treatment of smoking in depressed individuals ([redacted]), which was approved by the research ethics committee of [redacted]

Participants in this study were 180 treatment-seeking smokers at the [redacted]. Participants were recruited through advertisements in the local media and flyers posted in the community and by word of mouth. The inclusion criteria were: 1) smoking at least 10 cigarettes daily during the last year, 2) meeting the criteria for ND according to the DSM-IV-TR (American Psychiatric Association, 2002), 3) and having a score of 14 or higher on the Beck Depression Inventory-II (BDI-II). Participants were excluded if they: 1) met diagnostic criteria for severe mental disorder (i.e., bipolar disorder and schizophrenia), 2) met current diagnostic criteria for SUD (except ND), or 3) received any psychological or pharmacological treatment for smoking cessation at the study onset.

Participants provided informed consent and were randomly assigned to each of the following treatment conditions: CBT (n = 60), CBT + BA (n = 60), and CBT + BA

+ CM (n = 60). There were no significant differences between conditions in any sociodemographic or clinical variable at intake (see Table 1).

2.2 Assessment

At baseline, sociodemographic information was collected from participants (e.g. age, sex, income, etc.) as well as variables of clinical interest related to smoking (e.g. number of cigarettes per day, years of regular smoking, etc.).

The Fagerström Test for Nicotine Dependence (FTND) (Heatherton, Kozlowski, Frecker, and Fagerstrom, 1991) was used to evaluate ND. The FTND differentiates among low (FTND ≤ 3), medium (FTND between 4 and 6), and high (FTND ≥ 7) ND. The evaluation of the diagnosis of depression was carried out through the Structured Clinical Interview of the DSM-IV-TR (Spitzer et al., 1996). Depressive symptomatology was evaluated through the BDI-II (Beck et al., 1996). The BDI-II has the following cut-off scores: 0-14 minimal depression, 14-19 mild depression, 20-28 moderate depression, and 29-63 severe depression.

The DD task was used to evaluate impulsive decision making. Participants had to choose between an amount of money available now (between €5 and €995) and a fixed amount of money (€1,000) available later. The main objective of the presentation of choices was to identify the indifference point (the point at which the delayed reward has an equivalent subjective value to the immediate reward) of each subject in each of the seven delays contained in the task (1 day, 1 week, 1 month, 6 months, 1 year, 5 years, and 25 years). A quantity adjustment procedure proposed by Holt et al. (2012) was used for this purpose. The subjects were informed that they would not receive any of the amounts presented in the task and that they should respond as realistically as possible. This DD task has demonstrated good psychometric properties when it has been

used in other populations (Martínez-Loredo et al., 2017), and it has been used frequently in previous DD studies (see Amlung et al., 2017 for a meta-analysis).

The smoking abstinence criteria was defined as providing a self-report of smoking abstinence (not even a puff) for the past 24 hours at end-of-treatment and for the last seven days at the follow-ups, as well as presenting a breath CO level ≤4 ppm (Cropsey et al., 2014) and cotinine levels ≤ 80 ng/ml (Nondahl et al., 2005). A piCO Smokerlyzer (Bedfont Scientific Ldt, Rochester, UK) and the BS-120 analyzer (Shenzhen Mindray Bio-medical Electronics Co. Ltd., Shenzhen, China) were used to assess breath CO and urine cotinine respectively.

Smoking status, DD, and depressive symptomatology were collected at baseline, at end-of-treatment and at one-, two-, three-, and six-month follow-ups.

2.3 Treatment conditions

All interventions were implemented by master and doctoral level psychologists who had previous experience in the treatment of smoking. The treatments were carried out twice a week for eight consecutive weeks. The intervention was conducted in a group-based format, with a maximum number of four people per group. In addition, the activity of all therapists was reviewed weekly by the principal investigator through audio records. The principal investigator provided feedback on significant deviations from treatment protocols.

2.3.1 CBT

The CBT protocol has been described in more detail in [redacted]. The most important components of this protocol were: gradual fading of nicotine consumption (30% per week), psychoeducation on tobacco use, self-

registration and graphic representation of tobacco consumption, stimulus control, techniques for managing withdrawal symptoms, problem solving, and relapse prevention. The reduction of nicotine consumption was carried out between the first and the fourth session of the treatment, with the participants being asked to start abstinence 48 hours before the start of the fifth session.

2.3.2 CBT + BA

In this treatment condition, a BA module for mood management based on previously published protocols (Lejuez et al., 2011; MacPherson et al., 2016) was added to the CBT treatment. The main objective of BA was to increase the number of pleasant activities of the participants, taking into account their baseline level of activity, their interests, and their difficulties. The main components developed were: treatment rationale, psychoeducation on the relationship between smoking and depression, identification of life values and goals, planning of pleasant activities, weekly monitoring of activity and mood, and development of the participants' social support.

2.3.3 CBT + BA + CM

Participants allocated to this condition received the interventions previously described in combination with a CM protocol for reinforcing abstinence. The reinforcers used were vouchers that could be exchanged for a variety of goods and services in the community (e.g. gift cards, access to the spa, movie tickets, etc.). From the fifth week onwards, if the participants were biochemically abstinent (CO $\leq$ 4ppm and cotinine $\leq$ 80 ng/ml), they would earn vouchers with a value of €10 that would be increased by €5 at each successive abstinent session. In the case of identifying a participant as a smoker after being abstinent, a reset procedure described previously in Secades-Villa et al.

(2019) was followed. During follow-ups, if participants met the abstinence criteria, they were reinforced with €45 and €50 in the one- and three-month follow-ups. Thus, the total amount they could earn if they were totally abstinent was €175 for the duration of treatment or €270 including follow-ups.

2.4 Data analysis

The area under the curve (AUC) proposed by Myerson et al. (2001) was calculated for the purpose of analyzing and summarizing the indifference points of each participant. AUC values ranged between 0 (maximum discounting) and 1 (minimum discounting).

In order to analyze differences between the groups at baseline and in retention, continuous, and categorical variables, analysis of variance (ANOVA) and $\chi 2$ tests were used respectively. Participants who did not attend follow-up visits were considered smokers following the intent-to-treat approach. Mixed-effects model repeated measures (MMRM) analysis was used to explore the DD changes as a function of treatment, smoking abstinence, and depression over time. This analysis included an unstructured modelling of frequencies at each visit and a within-subject error correlation structure. Outcomes of both depressive symptoms and self-reported smoking status were treated as time-varying covariates. At the same time, baseline DD and baseline depression were treated as time-invariant variables. Likelihood based methods (e.g., the covariance pattern regression model and the linear mixed-effects model) are the most popular solution for dealing with incomplete longitudinal Gaussian data due to their validity when data are missing at random (Vallejo et al., 2011). In this study, frequencies at each visit were considered as a classification rather than a continuous variable. In the absence of a theory providing contrasting data, we used a data-driven strategy to move toward a

simpler structure by eliminating predictors or (co)variances that did not appear to be related to the outcome variable. In order to explore the changes across time, a linear combination of means was estimated and compared using the LSMEANS statement of the Proc MIXED.

A covariance pattern regression model with heterogeneous variances was conducted to assess the ability of the treatment condition to predict DD at end-of-treatment, one-, two-, three-, and six-month follow-ups. The AUC data were analyzed using MMRM with maximum likelihood (ML) estimation as implemented in SAS Version 14.3 (SAS Institute, 2018) Proc MIXED. To control the family-wise error rate for all possible pairwise comparisons, the Hochberg (1988) step-up Bonferroni inequality was applied using the ESTIMATE statement in SAS PROC MIXED and the HOC option in SAS PROC MULTTEST. The statistical software used in other analyses was SPSS (v20, Chicago, IL). Significance for all statistical comparisons was defined as $p \leq .05$.

3. Results

Figure 1 shows that the dropout rates of the participants were similar in all the groups, although retention was higher at the statistical level in the CM group at one- (p = .029) and six- month (p = .030) follow-ups.

The top panel of Table 2 shows the MMRM results of the fixed and random effects in the final model fit by comparing deviance statistics (see central panel of Table 2). There was a statistically significant effect of treatment [$F(2, 183) = 4.81$, p = .0094] and time [$F(4, 145) = 3.19$, p = .0132] variables on DD. As indicated in the bottom panel of Table 2, Hochberg's post hoc tests revealed that the CBT + BA + CM group

was more effective in reducing DD than the other treatments, although there were only statistically significant differences with the CBT + BA group [t(183) = -3.00; p = .0093]. In contrast, there was no significant interaction effect between the two main effects, which indicates that the effect of the treatment conditions on DD was stable over time.

On the other hand, we can see from the results reported in the top panel of Table 2 that the longitudinal covariate depressive symptoms is effective as a principal effect [F(1, 418) = 4.09, p = .0437]. Although the sign of the relationship between the BDI-II covariate and the AUC is not included in Table 2, it was negative. In other words, decreases in depressive symptomatology were associated with reductions in DD rates. Table 2 also reveals a significant interaction between the treatment and the BDI-II [F(2, 271) = 5.01, p = .0073], but there was no difference in their trends across time. To visualize the moderating effect of this covariate, we classified patients into two groups (above and below 13 points) based on their average BDI-II score in all follow-ups. Taken together, there is no evidence that the three treatment groups differ in terms of their AUC scores in patients with depression, whereas the treatment groups did differ in terms of their DD rates in patients without depression. This occurred in such a way that the CBT + BA + CM group helped to reduce DD rates more than the other treatment groups. Figure 2 represents these results graphically.

Results also indicate that the longitudinal covariate smoking status is effective as a principal effect [F(1, 365) = 14.77, p < .0001] and as a secondary effect [F(4, 162) = 4.31, p = .0024] (see top panel of Table 2). Specifically, smoking status has an initial effect that is significant (p < .0001), indicating that smoking abstinence is associated with a greater overall decrease in DD, and this positive effect of smoking abstinence

becomes more pronounced over time (p = .0024). Figure 3 helps to illustrate the conclusions of the analysis. Table 3 shows the differences at the statistical level between smokers and abstainers across time, controlling for BDI-II effects. Inspecting the least-squares mean estimates and their test statistics in Table 3 confirms that the means of smokers and abstainers are significantly different at end-of-treatment and for the all the follow-ups, except the one-month follow-up.

4. Discussion

To our knowledge, this is the first longitudinal study that explores the effect of smoking intervention, depressive symptoms, and smoking abstinence on DD among smokers with depression. There were three main findings: 1) receiving smoking cessation treatment, especially CM, reduces DD rates; 2) smoking abstinence (regardless of treatment condition) is related to a greater reduction in DD in long-term follow-up; and 3) decreases in depressive symptoms are associated with a reduction in DD rates.

Contrary to some previous studies (Dallery and Raiff, 2007; Kurti and Dallery, 2014; Yoon et al., 2009; Yoon et al., 2007) but in accordance with most of the scientific evidence (Chiou and Wu, 2016; Hughes et al., 2017; Secades-Villa et al., 2014; Stein et al., 2018; Stein et al., 2016; Weidberg et al., 2015a; Weidberg et al., 2015b; Yi et al., 2008), receiving an intervention for smoking cessation in general, and CM in particular, was associated with a decrease in DD rates across time. Several factors may contribute to the positive effect of a smoking cessation treatment, and CM in particular, on DD rates. First, problem solving, a shared component in all three groups, could help patients to improve the decision-making process (Marsiske and Margrett, 2006; Torrance et al., 1976) and therefore to decrease their DD rates (Jarmolowicz et al., 2016; Wittmann et

al., 2007; Wittmann and Paulus, 2008). Second, several studies have found that psychological treatments, and particularly CM, promotes engagement in health-related behaviors, such as regular exercise (Higgins et al., 2007; Irons, 2013; Stonerock and Blumenthal, 2017), and these changes may result in decreased DD (Bradford, 2010; Melanko and Larkin, 2013; Sofis et al., 2017; Tate et al., 2015). Third, decreases in DD could be due at least in part to the extended smoking abstinence rates achieved for patients in all groups, and especially participants in the CM condition. In addition, both CBT and CBT + BA produced similar DD decreases. This is in line with the findings of Jacobson et al. (1996) on reducing depression, in that the fact that CBT is based on the same process of change as BA (both treatments intend to involve patients in non-smoking alternative activities promoting the individual's contact with sources of reward) may explain why CBT + BA produced similar reductions in DD compared to CBT alone.

Our second finding supports the preceding explanation, in that, consistent with previous studies (Hughes et al., 2017; Secades-Villa et al., 2014; Weidberg et al., 2015a), smoking abstinence had a positive impact on DD rates, such that non-smokers reduced their DD more than smokers, and this reduction was more pronounced in subsequent follow-ups. Several mechanisms could account for this result. Firstly, addictive behaviors are characterized by persistent engagement in immediately reinforced behavior (e.g., smoking) without taking into account future negative health outcomes (Stein et al., 2016). Quitting smoking involves a behavioral change that represents an increase in the value of future consequences, since it implies being willing to experience the discomfort and difficulties associated with withdrawal symptoms (Aguirre et al., 2015; Frandsen et al., 2017) in order to obtain a health benefit in the

medium and long term (Gratziou, 2009; Jha et al., 2013; Taylor et al., 2014). Similarly, stopping smoking is associated with lifestyle changes, increasing the likelihood of engaging in healthy behaviors (Higashibata et al., 2016; Manczuk et al., 2019; Nagaya et al., 2007; Strine et al., 2005). Finally, quitting smoking can produce neuroadaptations (Froeliger et al., 2010; Sweitzer et al., 2016; Wang et al., 2017) that may lead to reductions in the activation of the impulsive decision system (e.g. striatum, hippocampus, cingulate cortex), and increases in the activation of the executive system (e.g. prefrontal) (Bickel et al., 2019; Koffarnus et al., 2013) which could result in a reduction in DD rates.

Third, in accord with previous research (Pulcu et al., 2014; Teti-Mayer et al., 2019), a decrease in depressive symptomatology causes decreases in DD. There are several mechanisms by which changes in depression may result in a reduction in DD rates. MDD is characterized by a reduced ability to modulate behavior as a function of rewards, particularly in the presence of anhedonia (Pizzagalli et al., 2008; Vrieze et al., 2013). Moreover, depressive mood tends to generate fewer approach goals and plans (Dickson and MacLeod, 2004), and to reduce the salience of future goals in favor of current temptations (Szuhany et al., 2018). On a related note, fading of depressive symptoms could also improve the EFT process (Hallford et al., 2018), which results in reduced DD (Rung and Madden, 2018; Scholten et al., 2019). Additionally, both high DD (Amlung et al., 2019) and MDD (Brody et al., 2001; Loonen and Ivanova, 2016; Lorenzetti et al., 2009) are related to excessive limbic circuit activation. Finally, in the same way as with smoking abstinence, remission of depressive disorder can encourage individuals to immerse themselves in new, healthier lifestyles (Hiles et al., 2017) that in turn are related to lower DD.

Taken together, our results provide further support of DD as a state-like and transdiagnostic process, given its modifiability and relationship with several psychopathological disorders (smoking and depression). Furthermore, this study shows how quitting smoking and the improvement of depressive symptomatology contribute to reduce DD in the long term. This twofold mechanism adds evidence in favor of concurrent interventions of psychopathological comorbidity, which may produce better treatment outcomes. Finally, our findings support adding CM procedures to conventional interventions, to promote not only abstinence but also reductions in DD rates.

Our results must be taken with some caution because of the study limitations. The first one is that DD was treated as a dependent variable, assuming that changes in smoking and depression would affect DD in a single direction. Nevertheless, it is reasonable to think that there may be a bidirectional relationship among smoking, depression, and DD (Chaiton et al., 2009; Yi et al., 2008). Secondly, these results may not be fully generalizable to men, who represent a minority in the total sample. Nevertheless, this underrepresentation seems to be congruent with the available epidemiological data on depressed smokers (Goodwin et al., 2017). Thirdly, this study used an outdated version of the DSM, which could compromise the capability to extrapolate or generalize the study findings. Fourthly, intervention fidelity was not monitored using a formal assessment tool to determine the degree to which treatment components were implemented as intended.

5. Conclusions

This study enhances the available knowledge about factors related to a decrease in DD rates. CM for smoking cessation, smoking abstinence, and reductions in depressive symptomatology contribute to decreased DD rates over time. Future studies should explore whether other interventions that are aimed at DD, such as EFT, are able to improve smoking cessation treatments outcomes.

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Table 1. Comparison of baseline variables across intervention groups

Characteristic	CBT (n = 40)	CBT+BA (n = 40)	CBT+BA+CM (n = 40)	Statistical test (*df*)	*p*
Age (years)[a]	53.7±10.0	50.6±10.2	52.7±8.9	$F_{(2, 177)}$ =1.60	.204
Sex (% women)	79.3	65.0	76.7	$\chi^2_{(2)}$ = 3.55	.170
Years of education (%)				$\chi^2_{(4)}$ = 9.21	.056
10 or less	37.9	18.3	23.3		
11 to 15	48.3	50.0	46.7		
16 or more	13.8	31.7	30.0		
Monthly income (%)				$\chi^2_{(8)}$ = 9.62	.293
Less than 600€	31.6	18.9	34.5		
601€ to 900€	19.3	20.8	23.6		
901€ to 1,500€	22.8	26.4	27.3		
1,501€ to 2,000€	15.8	26.4	7.3		
2,001€ or more	10.5	7.5	7.3		
MDD diagnosis (%)				$\chi^2_{(4)}$ = 3.71	.447
Single episode	16.7	25.0	18.0		
Recurrent episode	39.6	50.0	48.0		
Chronic disorder	43.7	25.0	34.0		
FTND total score[a]	6.4±1.7	6.9±1.8	6.2±1.9	$F_{(2, 176)}$ = 2.35	.098
Cigarettes per day[a]	22.5±10.3	22.9±8.2	20.7±6.7	$F_{(2, 176)}$ = 1.22	.298
Years of regular smoking[a]	33.5±9.8	31.7±11.5	32.2±9.1	$F_{(2, 177)}$ = .46	.629
CO (ppm)[a]	25.9±20.7	25.7±15.2	23.1±13.6	$F_{(2, 176)}$ = .53	.591
Cotinine (ng/ml)[a]	2454±2591	2509±1252	2380±1077	$F_{(2, 170)}$ = .08	.921
BDI-II[a]	30.9±7.5	27.1±9.7	29.5±8.8	$F_{(2, 179)}$ = 2.90	.058
AUC[a]	0.19±0.21	0.18±0.17	0.17±0.18	$F_{(2, 177)}$ = .17	.848

Note. CBT = Cognitive-behavioral therapy; BA = Behavioral activation; CM = Contingency management; MDD = Major depressive disorder; FTND = Fagerström Test of Nicotine Dependence; CO = Carbon monoxide; BDI-II = Beck Depression Inventory; AUC = Area under the curve.

[a] = Means ± standard deviation

Table 2. Results of fitting taxonomy of MMRM models to the AUC (top panel) and differences among means for all pairwise contrasts (bottom panel).

	Model A				Model B				Model C[1]			
Fixed Effect	df_N	df_D	F	Pr > F	df_N	df_D	F	Pr > F	df_N	df_D	F	Pr > F
AUC_BL	1	156	172.64	<.001	1	152	169.46	<.001	1	153	167.66	<.001
Grp	2	113	1.10	.3351	2	195	3.12	.0465	2	183	4.81	.0094
Time	4	138	1.03	.3951	4	167	2.12	.0802	4	145	3.19	.0132
Grp×Time	8	111	0.59	.7811	8	128	1.07	.3883				
BDI-II_BL					1	141	0.01	.9306				
BDI-II					1	456	4.17	.0417	1	418	4.09	.0437
BDI-II×Grp					2	293	4.30	.0144	2	271	5.01	.0073
BDI-II×Time					4	172	1.57	.1835				
BDI-II×Grp×Time					8	131	0.97	.4636				
Stat					1	416	17.66	<.001	1	365	14.77	<.001
Stat×Grp					2	284	0.13	.8740				
Stat×Time					4	183	2.90	.0231	4	162	4.31	.0024
Stat×Grp×Time					8	140	0.71	.6800				
Goodness-of-fit (Deviance/AIC/BIC/Parms)												
	-1016.6/-894.6/-700.5/60				-1074.5/-890.5/-604.8/91				-1062.7/-940.7/-751.7/60			
Hochberg Post Hoc Pairwise Contrasts												
Treatment			Estimate	Standard errors		df_N	t-Value	Pr > |t|	Hoc_p		*d*	
CBT+ BA *vs* CBT+BA+CM			-.0844	.0281		183	-3.00	.0030	.0092		.44	
CBT *vs* CBT+BA			.0539	.0289		162	1.87	.0636	.1272		.27	
CBT *vs* CBT+BA+CM			-.0305	.0316		179	-0.97	.3353	.3353		.14	

Note. AUC = Area under the curve; AUC_BL= Baseline AUC; BDI_BL = Baseline depression assessed using the Beck Depression Inventory (BDI-II); Grp = Treatment group; Stat = Smoking status; CBT = Cognitive-behavioral therapy; BA = Behavioral activation; CM = Contingency management; df_N = Numerator degrees of freedom; df_D = Denominator degrees of freedom.

[1] Both likelihood ratio tests and information criteria (i.e., AIC and BIC) allow us to conclude that Model C provides a better fit than Models A and B.

Table 3. Simple Effect Comparisons of Status × Time Least-Squares Means for AUC (Model C).

Time	Status	Status	*Estimate*	SE	DF	t Value	p > |t|	*d*
EOT	Non-smokers	Smokers	.0624	.0187	113.1	3.33	.0012	.63
1FU	Non-smokers	Smokers	.0160	.0197	139.1	0.81	.4183	-
2FU	Non-smokers	Smokers	.0686	.0175	175.9	3.92	.0001	.59
3FU	Non-smokers	Smokers	.0791	.0182	154.1	4.35	<.0001	.70
6FU	Non-smokers	Smokers	.0309	.0147	149.6	2.11	.0356	.35

Note. AUC = Area under the curve; EOT = End-of-treatment; FU = Follow-up. Time-varying standardized effect sizes have been computed using a similar approach to that described by Vallejo et al. (2019). The *d* values for the significant contrasts ranged from .35 to .70, a moderate effect according to Cohen's guidelines.

Figure 1. Flow diagram of study participants' survival.

Figure 2. Interaction between depression and treatment group in AUC means. Treatment groups were CBT, CBT + BA, and CBT + BA + CM, whereas depression groups were divided into non-depressed (BDI < 14) and depressed participants (BDI > 13).

Figure 3. Interaction between smoking status and time in AUC across time.