

**Relationship between delay discounting and depression in cigarette smokers and non-smokers**

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## **1. Introduction**

Delay discounting (DD) provides an operational measure of intertemporal choice that describes the devaluation of reinforcers when delayed (Bickel & Marsch, 2001). An example of this is how some subjects have an excessive preference for the immediate acquisition or consumption of a commodity (e.g., smoking a cigarette) despite long-term negative outcomes (Bickel, Jarmolowicz, Mueller, & Gatchalian, 2011). High DD rates are associated with a variety of problematic behaviors, including drug use and pathological gambling (Reynolds, 2006). Thus, DD is relevant for the study of drug use initiation, maintenance, and relapse (Madden & Bickel, 2010).

With regard to tobacco, greater DD is associated with a higher severity of nicotine dependence (ND) (Amlung & MacKillop, 2014; Ohmura, Takahashi, & Kitamura, 2005; Rezvanfard, Ekhtiari, Mokri, Djavid, & Kaviani, 2010; Sweitzer, Donny, Dierker, Flory, & Manuck, 2008), lower abstinence rates after receiving treatment (Sheffer et al., 2012), and an increased risk of smoking relapse (Krishnan-Sarin et al., 2007; Muench & Juliano, 2017; Sheffer et al., 2014; Yoon et al., 2007). Additionally, smokers are twice as likely to be depressed than the general population (Lasser et al., 2000). Smokers with current or past depression are more likely to have high ND and to relapse than those who are not depressed (Glassman, Covey, Stetner, & Rivelli, 2001; Minami et al., 2014; Niaura et al., 2001; Redner, White, Harder, & Higgins, 2014; Tsoh, Humfleet, & Mu, 2000).

Beyond the field of addictions, several studies have focused on studying DD as a transdiagnostic process (Amlung et al., 2019; Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Bickel, Quisenberry, Moody, & Wilson, 2015). For instance, Amlung et al. (2019) conducted a meta-analysis showing that individuals with a psychopathological disorder (i.e. major depressive disorder, MDD) had higher rates of DD than healthy controls. Focusing on this relationship, some studies have found a positive association between DD and depressive symptomatology (Acheson, Vincent, Sorocco, & Lovallo, 2011; Jarmolowicz et al., 2014; Olson, Kaiser, Pizzagalli, Rauch, & Rosso, 2018; Szuhany, MacKenzie Jr, & Otto, 2018), MDD (Engelmann, Maciuba, Vaughan, Paulus, & Dunlop, 2013; Pulcu et al., 2014; Takahashi et al., 2011), suicidal ideation (Caceda et al., 2014), and past depression in pregnant woman (Yoon et al., 2007). On the other hand, the DD rates of people in remission of MDD are equivalent to the healthy controls (Pulcu et al., 2014). Accordingly, the treatment of depression could help reduce high levels of DD (Engelmann et al., 2013).

Given that there is a high co-occurrence of ND and depression (between 33-41% of individuals with mood disorders are smokers, which is double the prevalence of smoking in the general population; Cook et al., 2014; Goodwin et al., 2017; Martins & Gorelick, 2011; Parker, Sigmon, & Villanti, 2019), and the fact that these comorbidities are strongly associated with high DD, it is essential to assess their combined effects on impulsivity rates. To our knowledge, only two studies have addressed this issue. Imhoff, Harris, Weiser, and Reynolds (2014) found that DD rates exhibited by adolescent smokers (both depressed and non-depressed) and depressed adolescent non-smokers were similar to each other, and all of them discounted more than non-smokers who were not depressed. Similarly, Weidberg, García-Rodríguez, Yoon, and Secades-Villa (2015) did not observe differences in DD rates between smokers with and without depressive symptoms. Despite being valuable, these

previous results are limited by the use of small sample sizes (Imhoff et al., 2014) and unbalanced groups (Weidberg et al., 2015). Of note is that none of the abovementioned studies included clinical samples of depressed smokers. Moreover, only one of them was conducted with adults (Weidberg et al., 2015), but it used a secondary analysis to explore the impact of depression on DD, which jeopardized the study's generalizability.

The present study sets out to address some of the pitfalls of previous research, with the goal of comparing the performance on a DD task across five groups of adults: smokers without depressive symptoms, smokers with moderate depressive symptoms, smokers with severe depressive symptoms, non-smokers with moderate depressive symptoms, and controls.

## **2. Materials and methods**

### **2.1 Participants**

Participants in the present study were 200 smokers and non-smokers with and without depressive symptoms. Smokers (with and without depression) were recruited from two randomized controlled trials (RCT) aimed at treating cigarette smoking at the Addictive Behaviors Clinic of the University of Oviedo (Spain). The participants completed the assessment before they started the smoking treatment. All smokers intended to quit smoking. Non-smokers with moderate depressive symptoms were recruited through the health system of the Principality of Asturias. Controls were recruited through advertisements and flyers posted in the community and by word of mouth.

Common inclusion criteria for all participants were: being over 18 years of age and not meeting diagnostic criteria for substance use disorder (SUD) or severe mental disorder (excluding MDD in depressive participants). Inclusion criteria for smokers (regardless of their depression) were: smoking  $\geq 10$  cigarettes per day for the last twelve months and

meeting the criteria for ND (4th ed., text rev.; DSM–IV–TR) (American Psychiatric Association, 2002). The inclusion criterion for non-smokers (regardless of their depression) was not having smoked during the last year.

Participants were divided into groups according to their Beck Depression Inventory-II (BDI-II; Beck et al., 1996) score: A BDI-II score of less than 14 was used to categorize both smokers without depressive symptoms and controls. A score between 14 and 28 was used to determine the group of smokers with moderate depressive symptoms. A score higher than 28 was used to categorize smokers with severe depressive symptoms. Finally, a score higher than 13 on the BDI-II was used to establish the group of non-smokers with moderate depressive symptoms.

All participants provided informed consent prior to study initiation and the study was approved by the Research Ethics Committee of the Principality of Asturias (n°124/15) and the Institutional Review Board of the University of Oviedo.

## 2.2 Procedure

The application procedure for the DD task is described elsewhere (García-Rodríguez, Secades-Villa, Weidberg, & Yoon, 2013). The DD task was presented to participants through a laptop. In short, participants were instructed on how to interact with the task. They were instructed to respond as if the decisions were real, understanding that they would not obtain any money. Participants had to choose between \$1,000 available after a fixed delay, versus different amounts of money available now (ranging from \$5 to \$995) using an adjusting-amounts procedure (Holt, Green, & Myerson, 2012). Due to the above, the number of trials that were presented to the participants, in order to find their indifference point, was variable. There were seven delay values (one day, one week, one month, six months, one year, five

years, and twenty-five years), presented in escalating order. The indifference point, which is the subjective value at which the immediate reward is equivalent to the delayed reward, was estimated for each delay.

### 2.3 Instruments and variables

During the intake session, participants' clinical history was obtained in order to collect data on sociodemographic (gender, income, marital status, age, and level of education) and smoking-related characteristics (cigarettes per day, age of smoking onset, and years of regular smoking).

ND was assessed through the SCID-I (4th ed., text rev.; DSM-IV-TR) (American Psychiatric Association, 2002), and the Fagerström Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991).

Depressive symptoms were assessed through the Beck Depression Inventory (BDI-II; Beck et al., 1996). Scores less than 13 represent minimal depression, those between 14 and 19 indicate mild depression, between 20 and 28 suggest moderate depression, and scores higher than 29 indicate severe depression. The internal consistency of the BDI-II was high in our sample ( $\alpha = 0.935$ ).

A Pico Smokerlyzer (Bedfont Scientific Ltd., Rochester, UK) was used in order to measure carbon monoxide (CO) in expired air. A BS-120 chemistry analyzer (Shenzhen Mindray Bio-medical Electronics Co. Ltd., Shenzhen, P. R. China) was used to determine quantitative urine cotinine levels.

### 2.4 Data analysis

We summarized each indifference point using the area under the curve (AUC) proposed by Myerson, Green, and Warusawitharana (2001) in order to avoid assumptions of any particular discounting model. AUC values range between 1 (no discounting) and 0 (maximum discounting). According to Johnson and Bickel (2008), four cases were identified as non-systematic (i.e., non-discounting and/or bounce). The results of the analysis did not vary with or without non-systematic data, so it was decided to include these data in the analysis.

Analyses of variance (continuous variables) and  $\chi^2$  tests (categorical variables) were used to examine group differences in baseline demographics. Pearson correlations were run to explore the association between AUC and BDI-II. A one-way analysis of variance (ANOVA) and one-way analysis of covariance (ANCOVA) were used to assess differences in AUC across the five groups (smokers, smokers with moderate depressive symptoms, smokers with severe depressive symptoms, non-smokers with depressive symptoms, and controls). Covariates included in the ANCOVA were those sociodemographic variables that showed significant differences across groups (i.e. age). The effect size was calculated through  $\eta^2$ . Cohen (1988) suggested that small, medium, and large effects would be reflected in  $\eta^2$  values equal to 0.01, 0.06 and 0.11, respectively. The statistical software used was SPSS (v20, Chicago, IL). In accordance with previous literature (Reynolds & Schiffbauer, 2004), significance for all statistical comparisons was defined at  $p \leq .05$ .

### **3. Results**

Table 1 shows a comparison of sociodemographic and smoking-related characteristics between groups. There were significant differences across groups in sex, age, years of education, FTND, years of regular smoking, carbon monoxide (CO), and BDI-II.

Figure 1 provides summary statistics of DD for each of the study groups. Overall, there was a significant correlation between AUC and BDI-II ( $r_{xy} = .226, p = .001$ ). Dividing the sample into smokers and non-smokers, a linear relationship between AUC and BDI-II emerged from non-smokers ( $r_{xy} = .336, p = .002$ ) but dissipated in smokers ( $r_{xy} = -.102, p = .270$ ). The one-way ANOVA showed significant differences in AUC between groups (smokers without depressive symptoms, smokers with moderate depressive symptoms, smokers with severe depressive symptoms, non-smokers with moderate depressive symptoms, and controls)  $F_{AUC}(4, 195) = 5.29, p > .001, \eta^2 = .098$ . Specifically, in the post hoc comparisons, controls (AUC = .38) were less impulsive than smokers with moderate depressive symptoms (AUC = .21,  $p > .028$ ), smokers with severe depressive symptoms (AUC = .18,  $p > .002$ ), smokers without depressive symptoms (AUC = .21,  $p = .034$ ), and non-smokers with moderate depressive symptoms (AUC = .20,  $p = .01$ ). There were no significant differences between smokers with different depressive symptoms, smokers without depression, and non-smokers with depressive symptoms (all  $p$  values  $\geq .916$ ). Group differences maintained after adjusting for age, sex, and years of education ( $F_{logk}(4, 192) = 3.14, p = .016, \eta^2 = .061$ ). Only age ( $F_{logk}(1, 192) = 4.2, p = .003$ ) was significantly related to the dependent variable.

#### **4. Discussion**

The present study advances knowledge regarding the association between DD and depression by comparing DD rates between smokers, smokers with different depressive symptoms, non-smokers with depressive symptoms, and controls. We highlight two findings. Firstly, DD and depressive symptoms showed a significant association only in non-smokers. Secondly, smokers (regardless of depressive symptoms) and non-smokers with depressive symptoms showed higher DD rates than controls.

The results of this study show that DD rates and depressive symptoms are not linearly associated in smokers, although they are in non-smoking subjects. Previous literature on this topic is heterogeneous. While some studies show a relationship between DD and depression (Acheson et al., 2011; Engelmann et al., 2013; Imhoff et al., 2014; Jarmolowicz et al., 2014; Olson et al., 2018; Pulcu et al., 2014; Szuhany et al., 2018; Takahashi et al., 2011), others do not find such an association (Brown, Hart, Snapper, Roffman, & Perlis, 2018; Dennhardt & Murphy, 2011; Dombrowski et al., 2011; Gonzalez, Reynolds, & Skewes, 2011). Nonetheless, inconsistencies in this literature could be related to method variance (Farris, Aston, Abrantes, & Zvolensky, 2017) and the variety of populations included. Specifically, it seems that studies excluding participants with drug use other than nicotine (Acheson et al., 2011; Engelmann et al., 2013; Imhoff et al., 2014; Jarmolowicz et al., 2014; Olson et al., 2018; Pulcu et al., 2014; Szuhany et al., 2018; Takahashi et al., 2011), find a relationship between depressive symptoms and DD rates, while the studies including this population do not find a significant relationship (Brown et al., 2018; Dennhardt & Murphy, 2011; Dombrowski et al., 2011). The only exception was Gonzalez et al. (2011), who did not find a relationship between DD and depression in a sample that did not report any problematic drug use. These data suggest that smoking (and drug use) may have a moderating effect on the association between DD and depressive symptomatology.

DD was steeper in clinical groups in comparison with controls. Among them, there were no differences in DD rates among smokers without depressive symptoms, smokers with depressive symptoms, and depressed non-smokers. This result is consistent with previous research conducted among adolescent (Imhoff et al., 2014) and adult smokers (Weidberg et al., 2015), and in individuals with SUD (Moody, Franck, & Bickel, 2016). There are three plausible explanations for these results. Firstly, men's delay discounting rates are more

affected by mood, while negative affect does not predict women's discounting (Koff & Lucas, 2011). The fact that in this study 74.2% of the individuals with depressive symptoms were women might account for the similar discounting rates observed across groups. Secondly, it is possible that there is no additive effect of depression and smoking over DD, since other variables that are not considered in this study, such as anxiety, may serve as a partially protective factor for reduced DD (Engelmann et al., 2013). Thirdly, it could be that psychopathological comorbidity (beyond the comorbidity explored in this study, i.e. smoking and depression) does not have a clear cumulative effect on DD. Some studies find an additive effect (Moallem & Ray, 2012; Moody et al., 2016a; Moody, Franck, Hatz, & Bickel, 2016), while the vast majority do not find such an effect (Albein-Urios, Martinez-González, Lozano, & Verdejo-Garcia, 2014; Businelle, McVay, Kendzor, & Copeland, 2010; Farris et al., 2017; García-Rodríguez et al., 2013; Gowin, Sloan, Swan, Momenan, & Ramchandani, 2019; MacKillop & Tidey, 2011; Wing, Moss, Rabin, & George, 2012). Perhaps in general this additive effect is not found because the DD itself functions as a transdiagnostic process that underlies these disorders (Bickel et al., 2012), in which there is a ceiling effect (Gowin et al., 2019) when there is a co-occurrence of several diagnostic categories simultaneously. Our finding could therefore reinforce the idea of DD as a trans-diagnostic process (Amlung et al., 2019) and it could also help explain the high comorbidity among psychopathological disorders (Koffarnus, Jarmolowicz, Mueller, & Bickel, 2013).

There is one aspect that merits consideration for future research. Several studies show that smoking cessation interventions reduce DD rates in smokers without depression who achieve long-term abstinence (Secades-Villa, Weidberg, Garcia-Rodriguez, Fernandez-Hermida, & Yoon, 2014). Nevertheless, there is previous evidence showing that DD reductions are not only conditioned by abstinence but also by depressive symptoms in

smokers from the general population (Weidberg et al., 2015). Therefore, future studies should assess whether interventions that reduce DD in smokers from the general population can also reduce DD levels in smokers with depression.

Several limitations of the present study should be noted. First, depressed groups were predominantly composed of women, which limits the generalizability of the present findings. Nevertheless, sex was entered as a covariate in the performed analyses. Second, the use of medication was not controlled in this study and antidepressants can affect DD rates (Homberg, 2012). Third, this study has a cross-sectional design, which does not allow us to know the causality relationships between the variables studied. Fourth, continuous variables have been categorized to form the groups (i.e. depression), which may limit the statistical power of our analysis. Fifth, the results obtained are not fully generalizable for all smokers with and without depression, since the smokers in our study had to meet certain special criteria (DSM-IV criteria for nicotine dependence) and all were treatment-seeking smokers. This last aspect is very relevant especially in depressive smokers, since inactivity is a factor associated with depression (González-Roz, Secades-Villa, & Muñoz, 2018), and these depressive smokers had the initiative to participate in the study. Sixth, an outdated version of the DSM diagnostic criteria for ND was used in this study. Despite this, the DSM-IV demonstrated a better concurrent validity in ND than the DSM-V (Chung, Martin, Maisto, Cornelius, & Clark, 2011), so this fact is not expected to have a significant impact on the present findings.

#### 4.1 Conclusions

This study shows that depression and smoking affect DD rates. However, there is no additive effect on intertemporal choice when the two conditions appear together. Given the

scarcity of studies assessing DD rates in this specific population, further exploration will be needed to confirm the present findings.

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Figure 1. AUC values of each study group. Box-and-whisker plot of AUC values, higher values of AUC indicate lower DD rates.

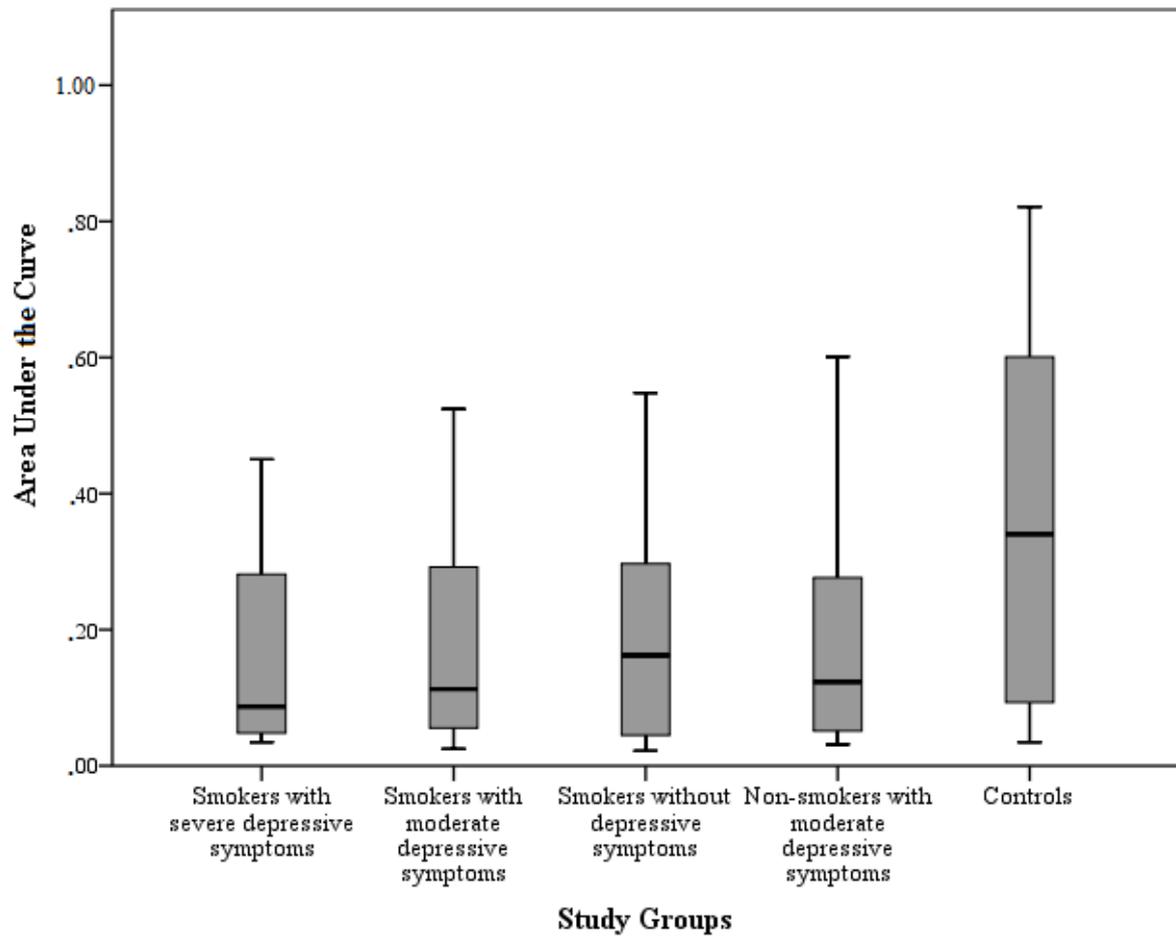


Table 1

*Comparison of sociodemographic and smoking-related characteristics across groups*

Characteristic	Smokers without depressive symptoms (n = 40)	Smokers with moderate depressive symptoms (n = 40)	Smokers with severe depressive symptoms (n = 40)	Non-smokers with moderate depressive symptoms (n = 40)	Controls (n = 40)	Statistical test (df)	p
Age (years) <sup>a</sup>	47.4±11.8	50.1±11.7	52.1±7.6	54.0±12.0	45.2±15.1	F <sub>(4, 195)</sub> = 3.51	.009
Sex (% women)	60.0	72.5	70.0	80.0	50.0	χ <sup>2</sup> <sub>(4)</sub> = 9.79	.044
Marital status (% married)	42.5	55.0	60.0	62.5	65.0	χ <sup>2</sup> <sub>(4)</sub> = 5.18	.269
Years of education (%)						χ <sup>2</sup> <sub>(8)</sub> = 19.83	.011
10 or less	20.0	20.0	25.0	25.0	20.0		
11 to 15	32.5	50.0	57.5	52.5	25.0		
16 or more	47.5	30.0	17.5	22.5	55.0		
Monthly income (%)						χ <sup>2</sup> <sub>(15)</sub> = 18.48	.556
Less than \$665	15.4	28.9	31.5	5.0	17.5		
\$666 to \$1,000	12.9	15.8	23.7	22.5	15.0		
\$1,001 to \$1,330	15.4	15.8	5.3	15.0	12.5		
\$1,331 to \$1,660	12.8	10.5	7.9	15.0	7.5		
\$1,661 to \$2,220	25.6	18.4	18.4	27.5	25.0		
\$2,221 or more	17.9	10.6	13.2	15.0	22.5		
FTND total score <sup>a</sup>	5.7±1.9	6.2±1.8	6.7±1.7	-	-	F <sub>(2, 117)</sub> = 3.21	.044
Cigarettes per day <sup>a</sup>	20.0±7.7	21.6±8.2	23.4±10.1	-	-	F <sub>(2, 117)</sub> = 1.57	.213
Years of regular smoking <sup>a</sup>	27.7±11.7	29.9±12.1	34.0±8.1	-	-	F <sub>(2, 117)</sub> = 3.48	.034
CO (ppm) <sup>a</sup>	16.0±9.0	27.2±19.8	24.8±13.6	-	-	F <sub>(2, 117)</sub> = 6.40	.002
Cotinine (ng/ml) <sup>a</sup>	2599.8±1514.1	2398.8±1228.5	2897.5±2887.9	-	-	F <sub>(2, 117)</sub> = .60	.549
BDI-II <sup>a</sup>	6.3±3.7	20.5±4.2	38.5±6.5	25.7±6.9	5.2±3.5	F <sub>(4, 195)</sub> = 293.77	< .001

*Note.* FTND = Fagerström Test of Nicotine Dependence; CO = Carbon monoxide; BDI-II = Beck Depression Inventory.

<sup>a</sup> = Means ± standard deviation