



Prospective associations between smoking abstinence and anhedonia among people who seek smoking cessation treatment

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ABSTRACT

Introduction: Depression has been broadly studied in association with tobacco smoking. However, little is known about the prospective association between anhedonia, one of the core symptoms of depression, and smoking outcomes after smoking cessation treatment. The goal of this study was to examine the bidirectional and longitudinal relation between anhedonia and smoking abstinence after smoking cessation intervention and up to a 12-month follow-up.

Methods: The sample was composed of 685 participants (Mage = 45.51; 62 % female) that were enrolled in three cognitive-behavioural smoking cessation interventions. Anhedonia and smoking abstinence were measured at post-treatment, and at 3-, 6- and 12-month follow-ups. The bidirectional and longitudinal association was analysed applying a Random Intercept Cross-Lagged Panel Model (RI-CLPM), and results were controlled for the effects of sex and differing studies.

Results: The RI-CLPM showed that smoking abstinence in one timepoint predicted lower anhedonia at the following timepoint for each assessment. Although greater anhedonia was related to lower smoking abstinence cross-sectionally at post-treatment, 6- and 12-month follow-ups, precedent anhedonia did not predict consequent smoking abstinence at any follow-up. The results were consistent when controlling potential confounding variables.

Conclusions: The bidirectional analysis indicated that smoking abstinence predicted lower anhedonia after a smoking cessation intervention during the course of 12 months, but anhedonia did not significantly predict smoking abstinence at any consequent timepoint. Findings have relevant implications as may serve to motivate healthcare professionals to apply tailored smoking cessation interventions, and people who smoke to quit due to the potential impact on mood.

1. Introduction

Smoking cigarettes is one of the leading causes of morbidity and mortality worldwide (US Department of Health and Human Services [USDHHS], 2020) and contributes to mental health issues such as depression and anxiety (Royal College of Physicians & Royal College of Psychiatrists, 2013). Smoking cigarettes increases the risk of depression (Wu et al., 2023), and people with depression are more likely to smoke (Fluharty et al., 2017) and experience greater nicotine dependence levels (Dierker et al., 2015). In the context of smoking cessation,

experiencing higher depressive symptoms increases withdrawal (Weinberger et al., 2010), reduces the likelihood of quitting (Ranjit et al., 2020), and increases the likelihood of relapse (Zvolensky et al., 2015). Although the relationship between depressive symptoms and smoking is well established, little is known about the longitudinal and bidirectional relationship between both constructs.

Depression is a very heterogeneous construct that can present itself with a highly variable course for a given person, and with widely differing combinations of symptoms for different people (van Eeden et al., 2019; Fried & Nesse, 2015). Such a complex phenomenon has

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driven the literature to examine the different symptoms that compose depression in greater depth. Specifically, one of the symptoms that most attention has been receiving is anhedonia, which is a core symptom of depression (Wang et al., 2021).

The definition of anhedonia has developed from being the incapability of experiencing pleasure or interest to a more complex construct that includes a variety of deficiencies in the processing of rewards (Thomsen, 2015). These deficiencies include the motivation, interest and effort that is put into pursuing a reward, the valuation that is given to the reward, the anticipation of pleasure from the reward, the learning process, the actual pleasure that is felt from rewards and the process of satiation (Serretti, 2023).

When anhedonia is analysed in the context of substance use, previous research has shown that people with substance use disorders experience higher levels of anhedonia than people without such disorders (Destoop et al., 2019), and the presence of anhedonia may also trigger earlier initiation of substance use and greater consumption severity (Sussman & Leventhal, 2014). Furthermore, greater levels of anhedonia have been associated with an increased likelihood of relapse across different substance use disorders (Hatzigiakoumis et al., 2011).

In particular, the association between anhedonia and cigarette smoking has been explored from different perspectives. On the one hand, greater levels of anhedonia lead to a greater risk for smoking onset in adolescence (Stone et al., 2017), a greater difficulty to quit smoking (Leventhal et al., 2014), and higher likelihood of smoking relapse (Leventhal & Zvolensky, 2015). Additionally, higher levels of anhedonia have been related to higher rates of past quit attempts that resulted in rapid relapse and to higher smoking urges related to the intention and desire to smoke (Leventhal et al., 2009). Conversely, smoking predicts higher levels of anhedonia in general population (Ameringer & Leventhal, 2010). Additionally, smokers with depression experience greater levels of anhedonia compared to non-smoking individuals with depression (Liverant et al., 2014). Other studies have also observed a rapid decrease in positive affect after a short period of smoking abstinence among people with higher levels of anhedonia, resulting in a direct impact on increased cigarette craving (Cook et al., 2004). While these studies have primarily focused on the unidirectional relationship between anhedonia and smoking, the bidirectional relationship remains underexplored.

Although cross-lagged research points toward a bidirectional relationship between smoking cigarettes and depressive symptoms within the general population (Sánchez-Villegas et al., 2024; Tjora et al., 2014), others suggest that depressive symptoms lead to consequent smoking rather than the reverse (Fluharty et al., 2017; Wang et al., 2022). Yet, no studies have examined the within-person bidirectional association between anhedonia and smoking among treatment-seeking smokers while accounting for the influence of time-invariant confounders, such as genetic (Barkhuizen et al., 2021; Ma et al., 2023) or personality factors (Hakulinen et al., 2015a,b). Additionally, while some studies suggest that depressive symptoms significantly improve after smoking cessation (Moss-Alonso et al., 2024; Rodríguez-Cano et al., 2016; Taylor et al., 2021), the change or stability of anhedonia in relation to smoking abstinence remains largely unexamined. Investigating whether levels of anhedonia increase with smoking abstinence, whether smoking cessation reduces anhedonia, or whether a bidirectional relationship exists between these variables on the long term would be of significant clinical interest.

Thus, using a state-of-the-art longitudinal modelling technique such as the Random Intercept Cross-Lagged Panel Model, we aim to analyse longitudinal and bidirectional associations between anhedonia and smoking abstinence among people who seek treatment for smoking cessation and during a 12-month follow-up period, while considering the time invariant variables related to anhedonia and smoking abstinence. Understanding these relationships could contribute to valuable insights for clinical interventions targeting both smoking cessation and anhedonia.

2. Methods

2.1. Participants

The total sample consisted of 685 participants who sought intervention for smoking cessation in the Smoking Cessation and Addictive Disorders Unit at the University of Santiago de Compostela (Spain), between the years 2015 and 2022.

The inclusion criteria were: (1) being 18 years old or above; (2) smoking at least 6 cigarettes a day; (3) consenting to participate in a psychological intervention for smoking cessation.

The exclusion criteria in this study were the following: (1) smoking any other tobacco product different from cigarettes; (2) presenting a concomitant substance use disorder (cannabis, cocaine, alcohol and/or opioids); (3) being diagnosed of a severe mental health disorder (specifically bipolar disorder and/or psychotic disorder); (4) having received an effective smoking cessation intervention in the previous year.

We conducted a binary logistic regression in order to identify whether pre-intervention anhedonia had an impact on participant attrition at follow-up, after adjusting for sex, age and baseline cigarettes per day. The analysis revealed that baseline anhedonia was not significantly associated with attrition at post-treatment (OR = 2.09; $p = 0.06$), nor at 3-month (OR = 1.02; $p = 0.94$), 6-month (OR = 1.29; $p = 0.49$) or 12-month follow-ups (OR = 1.34; $p = 0.42$).

2.2. Measures

Previous to the smoking cessation intervention all participants were assessed using a semi-structured interview and a set of questionnaires. Sociodemographic data and information on smoking-related variables such as age of smoking initiation or cigarettes smoked per day (CPD) was collected using the Smoking Habit Questionnaire (SHQ; Becona, 1994).

Anhedonia was assessed with the Beck Depression Inventory II (BDI-II; Beck et al., 2006; Sanz et al., 2003) using the 4-item model (Cogan et al., 2024). The 4-item model includes items 4, 12, 15 and 21, from the BDI-II. The items evaluate loss of ability to experience pleasure, loss of interest, energy or effort and loss of sexual interest respectively. This self-report questionnaire was administered pre-intervention, at the end of the smoking cessation treatment and at the 3-, 6- and 12-month follow-ups (Cronbach's alpha ranged from 0.77 to 0.85).

Smoking abstinence was assessed at the end of the cognitive-behavioural intervention for smoking cessation and at the 3-, 6- and 12-month follow-ups. Participants were considered abstinent at the end of the intervention if they self-reported not having smoked even one-puff at least 24 h before the last intervention session. At the 3-, 6- and 12-month follow-ups a 7-day point prevalence criterion was used (Piper et al., 2020).

Biochemical validation was obtained via carbon monoxide (CO) measurements in exhaled air using Bedfont's Micro + Smokerlyzer (Bedfont Scientific Ltd., Maidstone, Kent, UK). CO levels were obtained at in-person visits. The CO cutpoint used in the present study was 5 particles per million (ppm) in exhaled air (Benowitz et al., 2020). Participants with CO measurement under 5 ppm were deemed to be abstinent. Due to the social distancing measures established during the COVID-19 pandemic in 2020, we biochemically validated abstinence for 37.5 % of the sample, relying on self-report measures for the rest of participants.

2.3. Procedure

All participants received a cognitive-behavioural intervention for smoking cessation which was applied by clinical or general health psychologists specifically trained in the administration of this intervention. In Spain clinical and general health psychologists are professionals that are qualified to conduct psychological treatments and interventions.

The smoking cessation treatment consisted of 8 weekly group

sessions, with a duration of one hour and composed of 6 – 8 people per group. Some of the techniques used throughout the intervention are the following: nicotine fading, stimulus control, problem solving, anger and stress management, or relapse prevention (Becona, 2007). The present research is a secondary analysis that includes participants from 3 different studies (Study 1: (Martínez-Vispo et al., 2019); Study 2: (Barroso-Hurtado et al., 2024); Study 3: (López-Durán et al., 2024)).

The present study followed all ethical principles and was approved by the Bioethics Committee of the University of Santiago de Compostela. Informed consent was obtained from all participants in this study.

2.4. Analytical strategy

First, we analysed the descriptive statistics and zero-order correlations between anhedonia and smoking abstinence at post-treatment, and at 3-month, 6-month, and 12-month follow-ups after the smoking cessation treatment. Second, we applied a Random Intercept Cross-Lagged Panel Model (RI-CLPM) for categorical variables to identify within-person longitudinal associations between smoking abstinence and anhedonia (Asparouhov & Muthén, 2023; Hamaker et al., 2015; Muthén et al., 2024). RI-CLPM differentiates between within-person and between-person variations by creating random intercepts for both anhedonia and abstinence. The random intercepts capture the stable between-person components of anhedonia and abstinence, while the cross-lagged paths represent how variations in anhedonia and abstinence within individuals are longitudinally related. A significant advantage of these models is that the estimation of cross-lagged effects is free from the influence of all time-invariant confounding factors (e.g., genetic factors), because it is captured in the between person variation.

Previous studies suggested differences by sex in abstinence (Smith et al., 2016) and anhedonia (Adewuya et al., 2018); thus, we ran a multigroup analysis to test whether the bidirectional relationships might be moderated by sex (Mulder & Hamaker, 2020). Likewise, we ran a multigroup analyses to control whether participation in study 1, 2 or 3 moderates our analyses. We considered a fit acceptable for the models when the comparative fit index (CFI) was ≥ 0.95 , the Tucker–Lewis index (TLI) was ≥ 0.95 , the root-mean-square error of approximation (RMSEA) was ≤ 0.08 , and the standardised root-mean-square residual (SRMR) was ≤ 0.10 (Schermelleh-Engel et al., 2003). To address categorical variables and missing data, we used a robust estimator, Weighted Least Squares Mean and Variance adjusted (WLSMV) (Muthén et al., 2024; Muthén & Muthén, 1998). For data preparation, we used R version 4.3.1 (R Core Team, 2023) with the tidyverse 2.0.0 (Wickham et al., 2019) and psych 2.3.9 packages (Revelle, 2023), and for the RI-CLPM, we used Mplus version 8.11 (Muthén & Muthén, 1998–2024).

We set the threshold for statistical significance at $p < 0.05$ for all analyses.

3. Results

3.1. Descriptive statistics

A description of sociodemographic variables and other psychological and smoking related variables measured at baseline can be found in Table 1. Descriptive statistics and correlations are displayed in Table 2.

For anhedonia, the intra-class correlation (ICC) was 0.56, indicating that 56 % of the variance in anhedonia was due to variation between individuals, while 44 % was due to within-person variation across timepoints. The ICC for abstinence was 0.74, indicating that 26 % of the variance was due to within-person variation over time, and 74 % was due to variation between individuals.

3.2. Prospective relationship between anhedonia and smoking abstinence

First, we ran an RI-CLPM model to evaluate the bidirectional prospective relationship between anhedonia and abstinence. The model

Table 1

Sociodemographic variables and smoking related and psychological variables measured at baseline.

	Total sample (N = 685)	
	n	%
Sex		
Female	425	62
Male	260	38
Marital status		
Unmarried (single, divorced, or widowed)	347	50.7
Married/ lives with partner	338	49.3
Educational level		
Basic	114	16.6
Middle	233	34
Higher	338	49.3
Employment		
Employed	433	63.2
Unemployed/Sick leave/Student/Retired	252	36.8
	M	SD
Age	45.51	10.81
CPD ^a	19.09	8.49
BDI-II ^b at baseline	10.61	9.13
Anhedonia at baseline	2.77	2.33

^a CPD: Cigarettes per day.

^b BDI-II: Beck Depression Inventory II.

indicated adequate fit as shown by χ^2 (12.206), CFI = 1.00, TLI = 0.99, RMSEA = 0.023, and SRMR = 0.013. At the between-person level, the correlation was not significant ($r = -0.043$, $p = 0.901$), indicating that the stable components of both variables were not significantly related. At the within-person level, being abstinent in a previous timepoint predicted lower anhedonia at all later timepoints (see Fig. 1). This indicates that within-person deviations in early abstinence predicts within-person variations in consequent anhedonia. On the contrary, anhedonia did not predict abstinence at any timepoint (see Fig. 1).

Due to the RI-CLPM revealing a predictive power of abstinence on anhedonia, and for greater clarity, mean anhedonia according to smoking status at each point in time reporting the number of participants that provided anhedonia scores is depicted in Fig. 2. Moreover, a table that describes mean values of anhedonia across time has been included in [Supplementary materials](#).

3.2.1. Sex differences

We evaluated a multigroup RI-CLPM to analyse if the bidirectional relationship between abstinence and anhedonia differed by sex. First, we compared the unconstrained model against the model resulted from constraining the autoregressive, cross-lag, and correlational relationships to be equal within each sex. For parsimony reasons, we selected the constrained model for both sexes as it was indicated by the non-significant chi square tests. Then, we ran the two best models in the multigroup RI-CLPM. Table 4 depicts the differences between the unconstrained model and the model with all paths constrained to be equal within sexes and the cross-lag paths to be equal between sexes and by time, showing that the latter model had a similar fit to the constrained model ($\Delta\chi^2$ (10) = 11.168, $p = 0.35$). This model suggests that the relationship between being abstinent at a previous timepoint and lower anhedonia at a later timepoint was similar across all follow-up points and did not differ by sex (see Table 4).

3.2.2. Study differences

Similarly to sex, for the included studies we first set a series of models to determine the best-fitted model within each study before analysing

Table 2
Descriptives and correlations (N = 685).

Variable	M/ %	SD/ n	1	2	3	4	5	6	7	8
1. Abstinence post-treatment	64	441								
2. Abstinence 3-month follow-up	40	272	0.53***							
3. Abstinence 6-month follow-up	35	239	0.45***	0.79***						
4. Abstinence 12-month follow-up	34	234	0.41***	0.67***	0.78***					
5. Anhedonia at baseline	2.77	2.33	-0.12***	-0.06	-0.11**	-0.07				
6. Anhedonia post-treatment	1.43	1.91	-0.26***	-0.12**	-0.13**	-0.13**	0.46***			
7. Anhedonia 3-month follow-up	1.75	2.31	-0.29***	-0.25***	-0.24***	-0.20***	0.51***	0.51***		
8. Anhedonia 6-month follow-up	2.00	2.32	-0.26***	-0.30***	-0.33***	-0.31***	0.48***	0.42***	0.62***	
9. Anhedonia 12-month follow-up	1.93	2.44	-0.24***	-0.25***	-0.30***	-0.38***	0.49***	0.42***	0.57***	0.74***

Note. M = Mean; SD = Standard deviation. Abstinence variables are represented by % and sample size.

** p < 0.01.
*** p < 0.001.

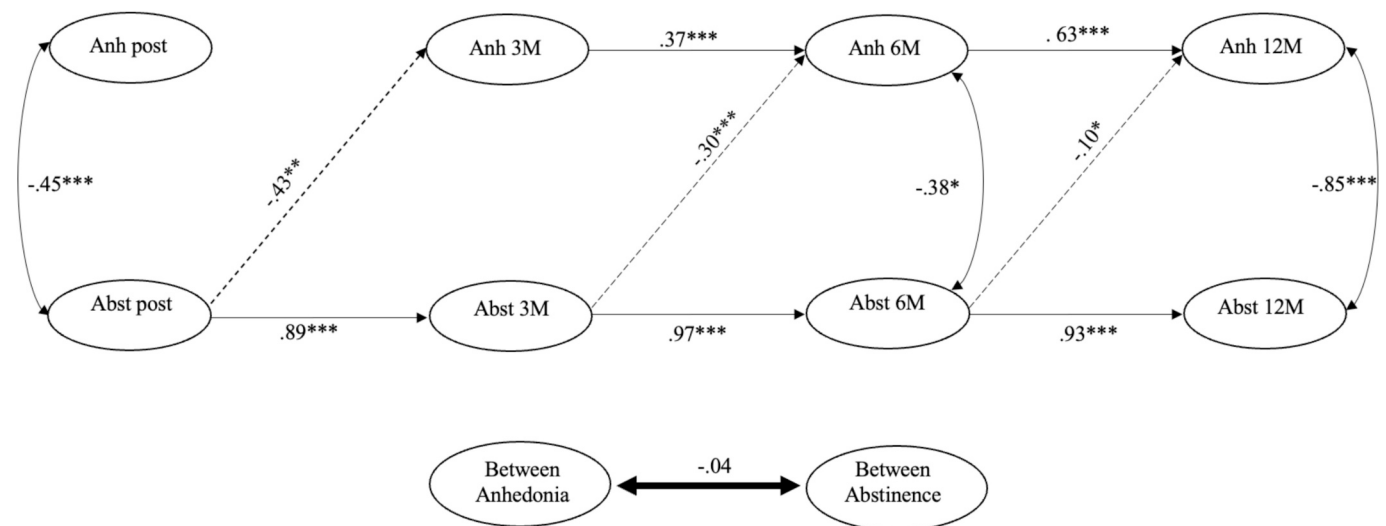


Fig. 1. RI-CLPM examining the association between anhedonia (Anh) and smoking abstinence (Abst) after receiving a smoking cessation treatment with the full sample. Standardised coefficients are depicted. * p < 0.05, ** p < 0.01, *** p < 0.001.

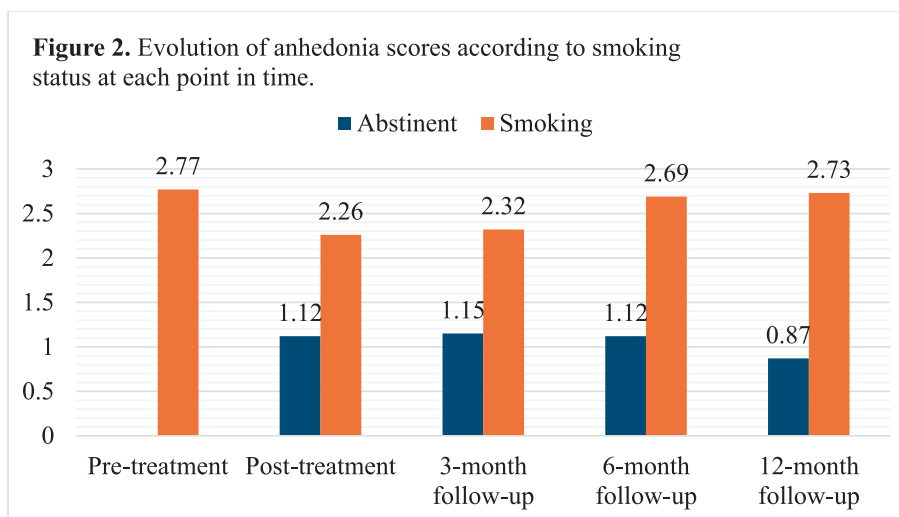


Fig. 2. Evolution of anhedonia scores according to smoking status at each point in time. Note. Participants that provided anhedonia scores at each point in time n (%): Pre-treatment 685 (100 %); Post-treatment 593 (86.6 %); 3-month follow-up 518 (75.6 %); 6-month follow-up 509 (74.3 %); 12-month follow-up 486 (70.9 %).

the multigroup RI-CLPM. The best models for the three studies constrained the autoregressive, cross-lag, and correlational relationships to be equal in time within each study. For study 3, the only difference was that the cross-lag path between anhedonia at 6 months and abstinence at

12 months was freely estimated. The baseline multigroup model included these three models.

Table 3 shows the selection of the models for the multigroup RI-CLPM analyses. The final selected model included equal constraints

Table 3
Multigroups RI-CLPM: model selection by sex and study.

Multigroup RI-CLPM	χ^2	df	CFI	TLI	RMSEA (90 % CI)	SRMR	Diff test		
							$\Delta\chi^2$	Δdf	p-value
Sex									
Unconstrained model	19.208	20	1.00	1.00	0.00 (0–0.045)	0.017			
Constrained cross lag to be equal among groups	28.518	30	1.00	1.00	0.00 (0–0.038)	0.023	11.168	10	0.35
Studies									
Unconstrained model	83.932	60	0.997	0.996	0.042 (0.016–0.062)	0.049			
Constrained cross lag to be equal among groups	NO CONVERGENCE. NUMBER OF ITERATIONS EXCEEDED.								
Constrained cross lag to be equal among groups, but Study 1 and Study 2 equal rest of the estimators	108.143	69	0.995	0.994	0.050 (0.031–0.067)	0.059	22.978	9	0.01
Constrained all path to be equal, but Study 3 crosslags 6–12 unconstrained and add regression from abstinence 3 m instead of correlation only in Study 3	102.079	71	0.996	0.996	0.044 (0.022–0.062)	0.055	19.277	11	0.06

Note. df = Degrees of freedom; CFI = Comparative fit index; TLI = Tucker-Lewis index; RMSEA = Root-mean-square error of approximation; CI = Confidence interval; SRMR = Standardised root-mean-square residual; Diff test = χ^2 differential testing; Study 1 (Martínez-Vispo et al., 2019); Study 2 (Barroso-Hurtado et al., 2024); Study 3 (López-Durán et al., 2024).

Table 4
RI-CLPM examining the association between anhedonia and smoking abstinence after receiving a smoking cessation treatment by multigroup analyses on sex and smoking cessation study pertinence.

	Multigroup by sex		Multigroup by study		
	Male	Female	Study 1	Study 2	Study 3
Abstinence → Anhedonia					
Post-treatment → 3-month follow-up	-0.374***	-0.458***	-0.103***	-0.112***	-0.135***
3-month follow-up → 6-month follow-up	-0.852***	-0.234***	-0.209***	-0.176***	-0.307***
6-month follow-up → 12-month follow-up	-0.934***	-0.272***	-0.323***	-0.300***	-0.229***
Anhedonia → Abstinence					
Post-treatment → 3-month follow-up	0.029	0.128	0.168	0.165	0.084
3-month follow-up → 6-month follow-up	0.040	0.130	0.113	0.103	0.076
6-month follow-up → 12-month follow-up	0.036	0.080	0.062	0.075	0.039
RI abstinence <-> RI anhedonia	-0.260*	-0.161	0.215	-0.540	-0.142

Note. RI-CLPM = Random intercept cross-lagged panel models; Abstinence = ITT smoking abstinence; Anhedonia = 4-items subscale of the Beck Depression Inventory-II; Study 1: (Martínez-Vispo et al., 2019); Study 2: (Barroso-Hurtado et al., 2024); Study 3: (López-Durán et al., 2024); RI = Random Intercept.

* p > 0.05.

*** p > 0,001.

between studies and by time except for a free estimation of the cross-lags between 6 and 12 months for anhedonia and abstinence, with the removal of the cross-sectional correlation between anhedonia and abstinence at timepoint 3 in favour of a prediction from abstinence to

anhedonia at 3-month follow-up ($\Delta\chi^2(11) = 19.277, p = 0.06$). The final model suggested no significant differences between studies in the prediction of being abstinent and lower anhedonia from post-treatment to 3 months and from 3 to 6 months. For the relationship between 6- to 12-month follow-ups, Study 3 differed from the other two models, but the direction of the relationship was still negative, though with a lesser magnitude ($\beta_{Study1} = -0.32, \beta_{Study2} = -0.30, \beta_{Study3} = -0.23$, all $p < 0.001$). This indicates that the prediction of being abstinent on lower anhedonia was stable throughout time and studies.

An additional RI-CLPM model controlling for the effect of pre-treatment anhedonia can be found in [Supplementary materials](#).

4. Discussion

The aim of the present study was to analyse the longitudinal and bidirectional association between anhedonia and smoking abstinence during a 12-month follow-up period among 685 participants who sought treatment to quit smoking.

Based on the RI-CLPM analyses, we found that being abstinent at each assessment point (end of treatment, 3-month, and 6-month follow-ups) predicted lower levels of anhedonia at subsequent timepoints (3-month, 6-month, and 12-month follow-ups). These findings align with previous research by Snuggs & Hajek (2013), which shows that continuous abstinence during the first 4 weeks after smoking cessation treatment seems to reduce anhedonia. Conversely, studies examining the effects of smoking abstinence on withdrawal-related negative affect following smoking abstinence showed that negative affect did not resolve over a period ranging from 31 to 67 days (Gilbert et al., 2002; Gilbert et al., 2019). These discrepancies may be due to the differing methodologies employed in these studies. Besides, the long interval between follow-ups in the present study prevent any causal inferences due to the impossibility to detect potentially rapid fluctuating levels of anhedonia which may be due to alternative circumstances other than smoking abstinence. Additionally, rapid unassessed increases in anhedonia between follow-ups may result in higher attrition among our sample potentially influencing results by losing participants higher in anhedonia at some follow-ups. Likewise, the possible effect of downward drift due to the repeated assessment of any negative affect related variable, such as anhedonia, should be considered as a possible contributor to the obtained result. Downward drift may also be impacting attrition and consequently the obtained results. Further research is needed to deeply explore the relationship between quitting and anhedonia.

However, precedent levels of anhedonia were not associated with

smoking abstinence at any timepoint in our study. The long 3-month interval between follow-ups in the present study should be considered as a possible reason behind the lack of this prospective association yet again due to the difficulty to capture the potentially fluctuating levels of anhedonia. In this vein, previous research has found that relapse is predicted by rapid increases in negative affect during the exact day the relapse occurred, rather than slower increases during a longer period of time (Shiffman & Waters, 2004). Moreover, as previously mentioned, such rapid increases in negative affect, or in anhedonia in this case, may impact study attrition which may explain the lack of an association in this direction.

Even though our results did not appear to show that precedent levels of anhedonia were related to smoking abstinence, when analysing the cross-sectional relations in the RI-CLPM model, there was a negative association between abstinence and anhedonia at the end of treatment and at 6- and 12- month follow-ups. These results indicate that anhedonia seemed to be associated with lower abstinence and suggest that anhedonia may be related to relapse across time. Similarly, Cook et al. (2015) found that higher levels of post-quit anhedonia, assessed through ecological momentary assessment during an 8-week follow-up, were associated with an increased likelihood of smoking relapse. In this line, Leventhal et al., (2014) suggested that lifetime anhedonia increases the likelihood of relapse at 8 weeks and 6 months after smoking cessation treatment. In contrast, no such cross-sectional association was observed at the 3-month follow-up in the present study. This discrepancy may be explained by differential methods used in the mentioned studies such as ecological momentary assessment, that has the ability to capture smaller fluctuations in affect, and to the use of different models to assess anhedonia. Additionally, literature suggests that smoking relapse most frequently occurs in the first few months for individuals receiving smoking cessation treatment (Lee et al., 2021; Martínez et al., 2016). In this context, anhedonia might manifest more as a symptom of tobacco withdrawal (e.g., Cook et al., 2015). It is essential to consider in future studies the impact of withdrawal symptoms on smoking relapse, since affective symptoms have been found to endure up to and over two months after smoking cessation (Gilbert et al., 2019) and to directly increase the likelihood of successive relapse (Robinson et al., 2019).

Our study contributes to the literature by examining within-person changes in anhedonia following smoking abstinence, while accounting for the bidirectional effects of anhedonia and abstinence at each timepoint and controlling for time-invariant covariates. Moreover, our findings extend previous research by providing further information on smoking abstinence potentially reducing anhedonia after a smoking cessation treatment when assessed using long intervals throughout a 12-month period. These results align with existing literature suggesting that smoking cessation leads to improvements in depressive symptoms among treatment-seeking smokers (Rodríguez-Cano et al., 2016), including those with a history of major depression (Moss-Alonso et al., 2024) and other mental health disorders (Taylor et al., 2021). Additionally, the cross-sectional finding that anhedonia is related to lower smoking abstinence at post-treatment and at 6- and 12-month follow-ups continues to extend previous literature on the impact of depressive symptoms on smoking cessation outcomes, while supplying a longer follow-up period.

Regarding the differences by sex, our results suggested that sex did not moderate the bidirectional association between abstinence and anhedonia. This result is in line with the study by Powers et al. (2017) that did not find differences according to sex, when measuring anhedonia previous to a smoking cessation intervention and assessing smoking abstinence 8 weeks after treatment. Our results differ from other previous studies suggesting that smoking behaviour (Smith et al., 2016) and depressive symptoms (Salk et al., 2017) vary by sex at between level, as we specifically analyse the bidirectional relationship between anhedonia and smoking abstinence while distinguishing within-person variation in both variables.

The present research has some limitations. First, although we have

captured the smoking cessation process during a 12-month period after the smoking cessation treatment, the long intervals between follow-ups may limit the observation of rapidly short-term fluctuating levels of anhedonia, which prevents the extraction of causal inferences from the obtained results. Second, biochemical verification of abstinence was limited for our sample. Research suggests that the use of self-report without biochemical verification in tobacco research may lead to higher rates of misreported abstinence (Thrul et al., 2023). However, discrepancies between self-report and biochemical validation have been suggested to be greater among populations in which smoking is especially frowned upon such as pregnant women or patients with cardiac or respiratory disease (Dietz et al., 2011; Scheuermann et al., 2017), which is not the case of the sample at hand. Moreover, self-reported abstinence has been deemed reliable when in-person contact is not viable (Benowitz et al., 2020). In this vein, interventions in which biochemical validation is not possible, such as telemedicine interventions, have been found to achieve a 90 % concordance between self-reported abstinence and CO measures (Cinciripini et al., 2019; Rodríguez-Cano et al., 2024). Third, we used self-report questionnaires to measure study variables, which may introduce social desirability and recall bias (Althubaiti, 2016). Moreover, the repeated administration of questionnaires may have affected the results due to changes in self-perception of negative mood states (Sharpe & Gilbert, 1998), which could impact the reliability of anhedonia symptom trajectories. Fourth, the sample was limited to participants who actively sought smoking cessation treatment, who did not have any co-occurring substance use disorders, nor severe mental health disorders and that did not smoke any tobacco product different from cigarettes, making the results non-generalisable to other populations of people who smoke. Furthermore, it is essential to consider these results in light of the potential impact of participants with greater tendency to negative affect or that have been exposed to adverse events during the course of the study dropping out at higher rates than the rest (Gilbert et al., 2019; Shiffman et al., 2004). Although in the present study baseline anhedonia did not explain the probability of participants not completing any follow-up ($\beta = 0.011$; $SE = 0.062$; $p = 0.861$), selective attrition should be monitored in future research. Finally, RI-CLPM is a strong modelling method, but its complexity may reduce study replicability. The use of constrained paths, even though these demonstrated a similar fit to the unconstrained ones, may be concealing underlying explanatory mechanisms; and the non-convergence of some models in the multigroup analysis which can be explained by the small subgroup sample sizes, and while having been addressed by simplifying the model and resulting in a convergent final model, may lead to instability in our findings.

This study also has several strengths. To our knowledge, no previous research has analysed the bidirectional association between smoking abstinence and anhedonia, distinguishing between- and within-person variation. The use of RI-CLPM allows to examine these variances while controlling for time-invariant confounders like genetics. Moreover, no study has analysed this association over a 12-month follow-up, which contributes to a further understanding of the bidirectional association between smoking and anhedonia. These findings may help us to develop more effective interventions for smoking cessation considering specific symptoms like anhedonia. This could ultimately motivate people with depression to quit smoking due to the positive impact of abstinence (Campion et al., 2023; Taylor et al., 2021; Sawyer et al., 2023).

As future research directions we believe it might be of interest to analyse the bidirectional association presented here among clinical populations of people with major depressive disorder or with other mental health disorders that course with anhedonia such as schizophrenia or post-traumatic stress disorder (Da Silva et al., 2017). Tobacco smoking is highly prevalent among people with these kinds of disorders (Dickerson et al., 2018; Pericot-Valverde et al., 2018). Furthermore, as this study is focused on people who seek smoking cessation treatment it may be of interest to test our research objective with non-treatment

seeking populations. Therefore, disentangling this relationship may also help improve smoking treatment in these populations.

An additional interesting direction for future research may be to analyse the prospective and bidirectional relationships between smoking and anhedonia using shorter intervals between assessments and using methods that are more fine-grain, exhaustive and sensitive to rapid fluctuations in affect and mood, such as ecological momentary assessment. This method could allow to delve deeper into the mechanisms underlying the association between smoking abstinence and anhedonia. Future research could also benefit from exploring further the underlying mechanisms that may have impacted the absence of prospective findings in the influence of anhedonia on future smoking abstinence.

Furthermore, it could be compelling for future studies to use other scales that specifically measure anhedonia, such as the Snaith-Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995), in order to gain a better and more comprehensive understanding of the complex relation between anhedonia and smoking abstinence.

The present study has considered the potential impact of sex and differing cohorts on the results. However, it would be of interest to also explore the effect of other variables such as socioeconomic status, life stressors, psychiatric history and nicotine dependence as they could play significant roles in both smoking cessation and the experience of anhedonia.

For future studies, we also recommend ensuring adequate subgroup sizes when conducting multigroup RI-CLPMs and considering alternative modelling strategies (e.g., latent change score models or simplified longitudinal frameworks) when sample size or outcome characteristics impose constraints.

Finally, and due to the importance of studying the mechanisms underlying relapse in the context of smoking cessation, it may be an essential future direction to further study the impact of anhedonia on smoking relapse and to expand this research to other populations such as those who do not seek treatment, or who have psychiatric disorders or medical issues.

5. Conclusions

In conclusion, smoking cessation is associated with a reduction in anhedonia when assessed after a smoking cessation intervention throughout the course of a year and the presence of anhedonia prospectively seemed to not have an impact on subsequent smoking abstinence. However, anhedonia seems to be associated with lower abstinence cross-sectionally at the end of the treatment and at 6- and 12-month follow-ups. These findings reveal the complex nature of the association between smoking cessation and anhedonia and may have implications for clinical practice. For instance, healthcare professionals could use more tailored smoking cessation interventions addressing anhedonia in order to increase cessation success but also encourage quitting smoking due to the potential impact on mood improvement.

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CRedit authorship contribution statement

Elizabeth Moss-Alonso: Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Rubén Rodríguez-Cano:** Writing – review & editing,

Visualization, Validation, Methodology, Formal analysis, Data curation, Conceptualization. **Carmela Martínez-Vispo:** Writing – review & editing, Visualization, Validation, Methodology, Investigation, Conceptualization. **Ana López-Durán:** Writing – review & editing, Visualization, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization. **Elisardo Becoña:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.addbeh.2025.108439>.

Data availability

Data will be made available on request.

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