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Cigarette Smoking and Depression Comorbidity: Systematic Review & Proposed Theoretical Model

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Abstract

Background and Aims—Despite decades of research on co-occurring smoking and depression, cessation rates remain consistently lower for depressed smokers than for smokers in the general population, highlighting the need for theory-driven models of smoking and depression. This paper provides a systematic review with a particular focus on psychological states that disproportionately motivate smoking in depression, and frame an incentive learning theory account of smoking-depression co-occurrence.

Methods—We searched PubMed, Scopus, PsychINFO, and CINAHL through December 2014, which yielded 852 articles. Using pre-established eligibility criteria, we identified papers focused on clinical issues and motivational mechanisms underlying smoking in established, adult smokers (i.e., maintenance, quit attempts, and cessation/relapse) with elevated symptoms of depression. Two reviewers independently determined whether articles met review criteria. We included 297 articles in qualitative synthesis.

Results—Our review identified three primary mechanisms that underlie persistent smoking among depressed smokers: low positive affect, high negative affect, and cognitive impairment. We propose a novel application of incentive learning theory which posits that depressed smokers experience greater increases in the expected value of smoking in the face of these three motivational states, which promotes goal-directed choice of smoking behavior over alternative actions.

Conclusions—The incentive learning theory accounts for current evidence on how depression primes smoking behavior and provides a unique framework for conceptualizing psychological

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mechanisms of smoking maintenance among depressed smokers. Treatment should focus on correcting adverse internal states, and beliefs about the high value of smoking in those states, to improve cessation outcomes for depressed smokers.

Keywords

Smoking; nicotine dependence; depression; review

Introduction

Individuals with depression are more likely to smoke, smoke more cigarettes per day, and are less likely to successfully quit smoking than individuals without depression, and this health disparity is an important clinical and public health concern. Recent epidemiological data show the smoking rate for clinically depressed individuals is about twice the rate in the general population (1–6). Further, smokers with depression report greater nicotine withdrawal symptoms (7, 8), likely due in part to greater nicotine dependence among depressed versus nondepressed smokers (9–12). Though depressed smokers endorse levels of motivation to quit that are similar to, or even higher than, smokers in the general population (13–15) and attempt to quit at similar rates (16), odds of successful abstinence at one-month are 30–50% lower for those with current depression and elevated depressive symptoms (16).

As smoking prevalence continues to decline in the general population, those with mental illness are increasingly overrepresented among smokers and constitute an important tobacco use disparity group (17). Those with mental illness die up to 25 years earlier than those in the general population, largely due to chronic illnesses associated with smoking (e.g., cardiovascular disease; (18–20)). Given that smoking cessation improves both mental (21–26) and physical health outcomes (27), even among smokers with chronic health conditions, development of smoking cessation interventions targeted to those with various forms of mental illness (i.e., mood and anxiety disorders, substance use disorders, serious mental illness) are greatly needed. Herein, we focus our review on continuous depressive symptomatology (28–30), ranging from subsyndromal levels of depression through clinical disorders (i.e., current or past diagnosis of major depression).

Although promising cessation treatments exist (e.g.,(31), no treatment has been shown to fully attenuate the elevated relapse risk associated with depression, and further innovations in treatment development are needed. One major limitation to advances in treatment of comorbid smoking-depression has been a lack of comprehensive theories of the psychology of nicotine dependence and depression. Identifying the psychological mechanisms that underlie smoking persistence among those with elevated depressive symptoms is critical for 1) a comprehensive characterization of the causal connection between depression and smoking, and 2) developing innovative, theory-based, and effective treatment strategies which specifically target these psychological mechanisms in depressed smokers.

This systematic review provides an update of the current state of evidence on the topic of co-occurring smoking and depression and proposes a novel application of incentive learning theory to conceptualize smoking persistence among depressed smokers. As detailed below,

the incentive learning account has gained considerable empirical support in the study of drug dependence (32–40), and offers a unique lens through which to view the clinical problem of smoking and depression. In particular, incentive learning theory proposes that specific states drive goal-directed selection of smoking behavior over alternate choices for depressed smokers. We believe the incentive learning theory accounts for current evidence on how depression primes smoking and identifies novel psychological targets to optimize smoking cessation treatment for depressed smokers.

Materials and Methods

Data sources and searches

We searched the PubMed, Scopus, PsycINFO, and CINAHL databases through December 2014, using Medical Subject Headings or Major Concept search terms when available. We combined smoking-related (i.e., smoking, tobacco, nicotine, smoking cessation, tobacco use, tobacco use disorder, tobacco use cessation) and depression-related terms (i.e., depression, depressive, major depression). Search delimiters were ‘adult’, ‘human’, and ‘English language’. We also performed manual searches of reference lists of pertinent articles.

Study eligibility

Two authors independently reviewed abstracts and full-text publications. Inclusion criteria stipulated that studies examine clinical issues relevant to established, adult smokers (age ≥ 18 years), such as causes and correlates of smoking maintenance, willingness to quit, and cessation/relapse among established smokers, rather than smoking initiation or progression to regular smoking. The review included both current/past major depression and elevated depressive symptoms. Studies not assessing depression through clinical diagnostic instrument or validated, continuous scale (i.e., by self-reported diagnosis or single item only) were excluded. Studies of special populations of smokers (e.g., psychiatric, medical) without major depression were excluded. We included qualitative/quantitative reviews and clinical trial, human lab-based, observational, and epidemiological study designs. Non-peer reviewed publications and case studies were excluded.

We excluded publications that both reviewers agreed did not meet eligibility criteria. Authors resolved disagreements by consensus.

Results

Results of literature searches

Our searches of databases identified 852 unique citations (Fig. 1). We screened 852 records by abstract and 453 full-text articles. Of these, 297 articles were included in the qualitative synthesis. Publications incorporated in the narrative review were those which contributed most directly to our theoretical framework.

Psychological mechanisms promoting smoking maintenance and relapse in depressed smokers

Affective processes—While depression is characterized by a wide range of affective, cognitive, behavioral, and somatic features (41), evidence is mounting that the affective disturbances in depression may play a qualitatively unique role in depression-smoking comorbidity over and above most non-affective depressive symptoms (15, 42, 43). Indeed, symptom-level analyses have shown that high negative affect (NA; experience of subjective distress) and low positive affect (PA; low engagement with the environment (44, 45)) are uniquely associated with nicotine dependence severity (46, 47), smoking heaviness (48), and smoking relapse risk (42, 49–53). Moreover, low PA accounts for independent variance in relapse risk, above and beyond high NA (42, 49), suggesting that these two processes reflect unique etiological influences on smoking.

The notion that two distinct affective processes promote smoking is consistent with multi-dimensional models of psychopathology and personality which purport that low PA and high NA are empirically-distinct overarching psychological dimensions that have unique psychosocial and biological etiologies (54–56). The low PA dimension includes constructs related to appetitive emotion including diminished pleasure, interest, expectancy, motivation, and reinforcement learning (54, 57)), whereas the NA dimension includes constructs related to aversive emotion including sadness, irritability, anxiety, low distress tolerance and neuroticism (54). In the proposed theoretical model, low PA and high NA represent distinct states that each worsen more rapidly following abstinence for depressed versus non-depressed smokers (58, 59) and serve to augment smoking motivation.

First, as nicotine dependence increases, low PA signals the greater reinforcement value of smoking. In the overall population of smokers and non-smokers, nicotine moderately improves PA and reward responsivity (60). Crucially, smokers with prominent symptoms of anhedonia (i.e., the decreased capacity to experience pleasure) experience frequent bouts of low PA and endorse greater nicotine-induced increases in PA and reward responsivity following smoking (61). While smoking itself is more reinforcing in the anhedonic state, a greater experience of reward may also be derived from other non-nicotine reinforcers, such as music, and contribute to the greater net value of smoking in the anhedonic state (62). Following acute withdrawal, regular smokers show moderate-sized reductions in PA and reward responsivity (51, 59, 63), while anhedonic smokers exhibit greater reductions in PA, reward responsivity, and urge to smoke to enhance PA (64–67). Potentially, pre-existing deficits in hedonic experience among depressed smokers may be unmasked and exacerbated by nicotine withdrawal, producing strong motivation to resume smoking to end a temporary ‘time out’ in reward experience and obtain smoking’s positive reinforcing effects (57). Thus, low PA is established as a signal for the increased reinforcement value of smoking, particularly among depressed smokers with prominent symptoms of anhedonia.

Second, elevated NA also serves as a motivational state driving smoking maintenance among depressed smokers. Although escape or avoidance of NA is thought to be the prepotent motive for addictive drug use (68), the relationship between smoking and NA is complex. Nicotine is thought to have antidepressant properties (69–72) and experience of NA is shown

to reliably decrease latency to smoke and increase number of puffs consumed (73). However, while smoking reverses withdrawal-induced NA (74–77), there is little evidence that it alleviates experimentally-induced NA (77–82).

Depressed smokers likely fail to distinguish NA provoked by withdrawal versus other sources (i.e., environmental stressors), and thus seek out smoking in response to both states. Nicotine withdrawal is characterized by a marked increase in NA among smokers in the general population (83, 84). Depression-prone smokers experience even greater deprivation-induced increases in NA (85–87), which could reflect an unmasking of a propensity towards negative affective states (57). Accordingly, depressed smokers (88) and individuals prone to depression (89) are especially likely to smoke for affect regulation motives. NA thus provides a reliable signal for the greater reinforcement value of smoking and becomes established as a motivational state which primes smoking behavior, particularly in depressed smokers.

Cognitive impairment—In addition to affective disturbances, depression is characterized by nonspecific cognitive deficits that span domains of attention, memory, processing speed, and all aspects of executive functioning (90–96). Although the aggregated effect size estimates of cognitive deficits in major depression are small to medium (93, 97), these deficits are clinically significant (93, 97), functionally impairing (98–100), and may persist even after an acute depressive episode has abated (90). Crucially, cognitive impairment associated with depression is similar to that observed during nicotine withdrawal, particularly in domains of executive functioning and attention (101–103). In a general population of smokers, smoking abstinence is associated with impaired sustained attention, working memory, and response inhibition (104), and biases cognition toward the perceived salience of smoking-related stimuli ((105, 106), but also see (107)). Depressed smokers are shown to experience even greater withdrawal-induced deficits in cognitive performance (108), which may reflect the overlap (or perhaps additive effect) of cognitive deficits in depression with cognitive deficits induced by smoking abstinence. Accordingly, depressed smokers may maintain smoking behavior in order to attenuate cognitive deficits, especially when engaged in effortful or cognitively demanding tasks (109, 110). Importantly, cognitive deficits are prospectively associated with smoking relapse for both smokers in the general population (111, 112) and depression-prone smokers (113). Thus, background cognitive deficits in depression may summate with acute withdrawal- related cognitive deficits to prime smoking (108).

Our theoretical model posits that cognitive impairment comes to signal the additional reinforcement of smoking produced by the resulting improvement in cognitive functioning. However, far fewer studies have examined cognitive vs. affective processes underlying smoking maintenance in depressed smokers, limiting the definitive conclusions that can be drawn. Preliminary evidence suggests that depressed smokers endorse smoking in part to manage negative intrusive thoughts and facilitate concentration and problem-solving (114). Additionally, depressed versus nondepressed smokers showed improved reaction time performance after smoking, suggesting they experienced greater cognitive enhancement compared to baseline (115). Further studies are needed to test whether depressed versus

nondepressed smokers experience greater reinforcement value of smoking in the face of cognitive impairment, as suggested by an incentive learning theory account.

Proposed incentive learning model of smoking and depression and distinction from negative reinforcement-based models

Negative reinforcement models may be divided into three categories based upon the proposed mechanisms by which adverse states promote drug use. All three categories make the common prediction that depressed smokers experience frequent bouts of high NA, low PA, and cognitive impairment, which intensify rapidly during acute abstinence and function to promote smoking maintenance and relapse (80–83). The distinction between the three categories of negative reinforcement accounts lies in their depiction of the learning processes by which these adverse states acquire control over smoking behavior. The early negative reinforcement accounts referred to the adverse states as internal instrumental *discriminative stimuli* (or S^Ds for short) because they 'set the occasion' or provide the context (116) in which smoking is more reinforcing. But exactly how these internal S^Ds prime smoking behavior was not fully specified (117, 118).

The later wave of negative reinforcement theories (119), allostasis theories (120, 121) and more recent incentive habit theories (122) have sought to specify how adverse internal states prime smoking behavior. These theories follow Hull's (123) stimulus-response/reinforcement view of instrumental discrimination learning, according to which, adverse internal stimuli (S) form a direct association with smoking response sequences (R) as a result of reinforcement from smoking. The S-R association between adverse states and smoking responses is especially strong because smoking is more reinforcing in the adverse states, which strengthens the S-R association. This learning enables adverse states to elicit smoking behavior directly, without retrieving knowledge of the consequences of the behavior. Accordingly, S-R based control of smoking has been called automatic, unconscious, preconscious, habitual and compulsive. Such S-R accounts are attractive because they can explain how adverse internal states could promote smoking behavior bypassing the individual's intentions to remain abstinent, which could account for depressed smokers' elevated rates of smoking cessation failure, despite high motivation to quit. These accounts ascribe to internal stimuli the same form of direct control over response selection that 'standard' habit theories of addiction have ascribed to external drug-related cues (124–126).

However, S-R accounts of action control by internal states have been challenged on logical grounds by researchers from the behavior-analyst tradition (127–130). The grounds for this challenge are that the behavioral sequences needed to yield nicotine reinforcement differ vastly depending on the context (e.g., purchasing cigarettes from a store vs. from a machine; smoking during a break vs. in a bar). Internal states on their own are not sufficiently discriminating to elicit the response sequence that is required to produce nicotine reinforcement in these various external contexts via simple S-R/reinforcement learning (127–130). Instead, from the behavior-analyst perspective, adverse internal states function as *motivating operations* (or MOs) because they predict that smoking has greater reinforcement value independently of the external context and the response currently required to produce

that reinforcer. MOs are thought to motivate behavior by interacting with the external S^Ds. Specifically, by virtue of signaling that a smoking reinforcement has a greater value, MOs lower the threshold of activation required for external S^Ds to elicit the specific response sequences required to produce smoking reinforcement in the external context. On this account, adverse states do not elicit smoking directly (as predicted by S-R theories), but rather, modulate the ability of external discriminative stimuli to evoke smoking behavior through S-R learning. The main weakness of this account is that it does not fully explain the nature of the interaction which allows adverse states (MOs) to modulate control by external stimuli over response selection.

Incentive learning theory addresses this limitation by suggesting that adverse states promote smoking behavior through a conjunction of explicit desire and goal-directed instrumental knowledge (32–34); see also, (35). On this view, adverse states function as *motivational states* (like MOs) which, by virtue of predicting the greater reinforcement value of smoking, come to elicit an expectation of the greater reinforcement value of smoking (i.e., greater subjective desire to smoke). Smokers also acquire explicit goal-directed instrumental knowledge of the specific response sequences that yield nicotine reinforcement in different external contexts. These two forms of knowledge are then synthesized in a cognitive inference. The experience of an adverse state generates an expectation of the greater reinforcement value of smoking which is combined with goal-directed instrumental knowledge of the response sequence required to produce the desired smoking outcome given the specific external context (36–39). The incentive learning account is unique amongst the categories of negative reinforcement theory in predicting that adverse internal states prime smoking behavior via a conjunction of subjective desire to smoke and instrumental knowledge of the responses required to produce that outcome in the current context.

Proposed theory

As shown in Figure 1, we propose a novel application of the incentive learning account to co-occurring smoking and depression. Panel A illustrates the acute smoking phase in which depressed smokers develop an expectation of greater reinforcement value of smoking in three specific motivational states: NA, low PA, and cognitive impairment. As smoking persists, Panel B displays the role of chronic smoking in worsening NA, low PA, and cognitive impairment over time due to frequent bouts of nicotine withdrawal. Belief about the high reward value of smoking in these motivational states, particularly during abstinence, is conceptualized as the primary trigger for smoking maintenance and relapse following a quit attempt in depressed smokers.

Although this theoretical framework is relevant to smokers in the general population, we believe the motivational states are especially effective in depressed smokers. First, those with depression experience more intense adverse states, and greater reinforcement value of smoking in these states, so these states become more powerful motivators of smoking. Second, depression is associated with a more severe withdrawal syndrome, adding further intensity to the adverse states as motivators of smoking behavior. Lastly, depression is thought to counter acute satiety, so depressed smokers sustain a greater expected value of smoking even immediately after smoking.

The unique prediction of the incentive learning account (compared to the S-R account) is that subjective desire to smoke prompted by adverse states, and goal-directed instrumental knowledge of the effective smoking response in each context, are integrated to drive smoking behavior. One source of support for this claim is the finding that experimental induction of stress or NA provoke increases in both smoking desire and smoking behavior (79, 82, 131–139). Although the mood-induced increase in smoking desire may cause the smoking behavior, as anticipated by incentive learning theory, S-R based negative reinforcement accounts would argue that the increase in smoking desire is actually epiphenomenal in relation to smoking behavior. The causal status of subjective desire in controlling behavior remains an unresolved scientific question (34, 140).

We recently reported direct empirical evidence supporting the incentive learning account over the S-R account of how negative mood provokes smoking (40). A unique prediction of the incentive learning account is that induced negative mood should be able to augment a novel tobacco-seeking response in an extinction test through integration of 1) expected high value of tobacco, and 2) knowledge of the instrumental response required to produce that outcome in the context. By contrast, the S-R account predicts that an induced negative mood should not be able to augment a novel tobacco-seeking response in an extinction test. This is because the S-R account requires direct experience of the greater value of tobacco in the induced mood state to strengthen the association between that state and the novel tobacco-seeking response. Such S-R learning is not possible because the tobacco reinforcer is omitted from the extinction test. The results of our study (40) showed that inducing a negative mood state in smokers augmented tobacco-seeking in an extinction test, confirming that NA primes tobacco-seeking through an inference of knowledge about the greater value of tobacco in the mood state and knowledge of which response produces tobacco in the current context.

We generalize this finding to suggest that when depressed individuals experience an adverse state (NA, low PA, or cognitive impairment), which is heightened during abstinence, this state provokes an expectation of the current high value of smoking, which is integrated with knowledge of the response sequence required to smoke in the context, to drive selection of that behavior over alternatives (141). Below, we review implications of our proposed theory for pharmacological and behavioral interventions for smoking cessation in depressed smokers.

Discussion

Treatment implications of the incentive learning-based theoretical model: Guiding principles

The foremost prediction made by the incentive learning account is that adverse internal states are the primary drivers of smoking behavior in depressed individuals. Accordingly, experimental treatment approaches which seek to modify reactivity to external smoking cues (e.g., via cognitive bias modification (142)) are unlikely to be effective in depressed smokers. Rather, treatments should identify the adverse motivational states that are the primary drivers of smoking in each individual and personalize treatment to target those states. Further, effective intervention strategies should address smokers' beliefs about the

high reward value of smoking in these motivational states. In sum, our theory suggests that treatments which 1) attenuate adverse internal states, and 2) reverse positive expectancies associated with smoking, are most likely to achieve cessation among depressed smokers.

Targeted pharmacological interventions

Consistent with our theory, pharmacological interventions may be particularly effective in depressed smokers by ameliorating the motivational states which raise the expected value of smoking: high NA, low PA, and cognitive impairment. In particular, varenicline has cognitive-enhancing effects in both treatment-motivated smokers (143) and non-smokers (144), and reduces the cognitive impairment that accompanies withdrawal (145). Bupropion is also shown to be an effective pharmacological intervention among depressed smokers (146, 147), possibly through its effects on affective functioning (88, 148). Further, given that multiple withdrawal-related motivational states may interact or summate to motivate smoking among depressed smokers, combination pharmacotherapy (i.e., combined varenicline and bupropion or combination nicotine replacement therapy; (149–151) may be particularly warranted for this population to optimally address each state. As studies of pharmacological agents have largely focused on cessation outcomes rather than mediating variables, future research testing the effects of medications on the motivational states identified in this review could help guide pharmacological treatment for depressed smokers.

Targeted behavioral interventions

To date, NA has been the primary component of depression characteristically targeted by behavioral interventions in smokers with comorbid depression (152–155). Expanding behavioral interventions to also explicitly target low PA could improve smoking outcomes for smokers with depression. Behavioral activation — an efficacious treatment for major depressive disorder that increases engagement in reinforcing activities congruent with a person's goals (156) — may counteract withdrawal-related anhedonia and increase quit rates by increasing exposure to alternative sources of non-smoking reinforcement (157).

Our model further states that relations between motivational states and goal-directed smoking behavior are mediated by expectancies that smoking will improve these states. Behavioral interventions which address these expectancies (i.e., by challenging maladaptive beliefs about the long-term mood regulating effects of nicotine) could help break the link between cognitive/affective deficits and smoking maintenance among depressed smokers. Although smokers with depression commonly believe smoking helps regulate their negative mood states (158, 159), evidence suggests that smoking actually leads to worsening of mental health symptoms over time and, conversely, quitting leads to improved mental health status (160). Thus, behavioral interventions should emphasize the distinction between withdrawal reversal and true mood improvement, to allow smokers to learn that the benefits of smoking are short- but not long-term, and restricted to the withdrawal syndrome and not environmental stressors. Novel strategies incorporating self-monitoring and personalized feedback could help to improve the salience and credibility of this message, particularly among depressed smokers.

Conclusions

Our proposed theory posits that smoking is maintained among depressed smokers through the expectation that smoking will alleviate specific states, in integration with the goal-directed choice of smoking over alternate behaviors. Drawing from a systematic review of the smoking-depression literature, we propose high NA, low PA, and cognitive impairment as the three motivational states that drive this process. While we have chosen a narrative review format in order to frame our conceptual model, this approach limited our ability to systematically assess for risk of bias and strength of evidence across studies. As the treatment literature on smoking and depression expands, it will be increasingly important for future reviews of this topic to include both qualitative and quantitative methods of analysis.

Our review identifies several directions for future research. First, it remains unclear how awareness of cognitive deficits interacts with awareness of affective states (i.e., low PA and high NA) to provide the motivational signal promoting goal-directed smoking behavior. Studies that characterize each of these withdrawal-related states among depressed smokers, as well as their unique and shared contribution to tobacco-seeking behavior, are needed. Second, although our theory holds that expectancies play a causal role in smoking maintenance, it has not been fully specified how knowledge of reward values and context-dependent response requirements translate into actual action selection. Third, cognitive processes among depressed smokers have received relatively little research attention, but we believe cognitive impairment represents a key motivational state maintaining smoking behavior in this population (161). Future research should further elucidate the extent to which depressed smokers smoke for cognitive enhancement motives, differentially benefit from smoking in terms of functional cognitive outcomes, and respond to pro-cognitive agents as part of a smoking cessation intervention. Lastly, as depression and smoking commonly co-occur with other mental health concerns (i.e., substance use disorders, anxiety disorders) (162–164), identifying the unique versus shared incentive states underlying each of these conditions in order to guide integrated treatment is an important priority.

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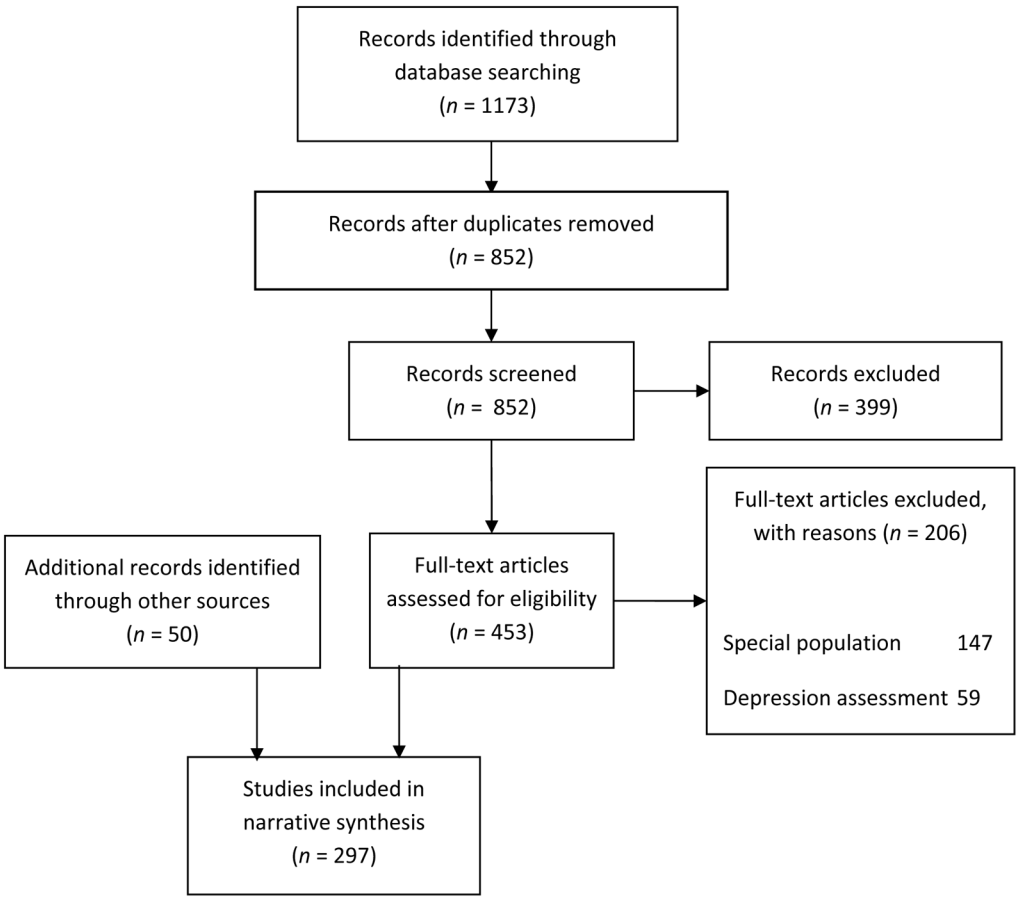
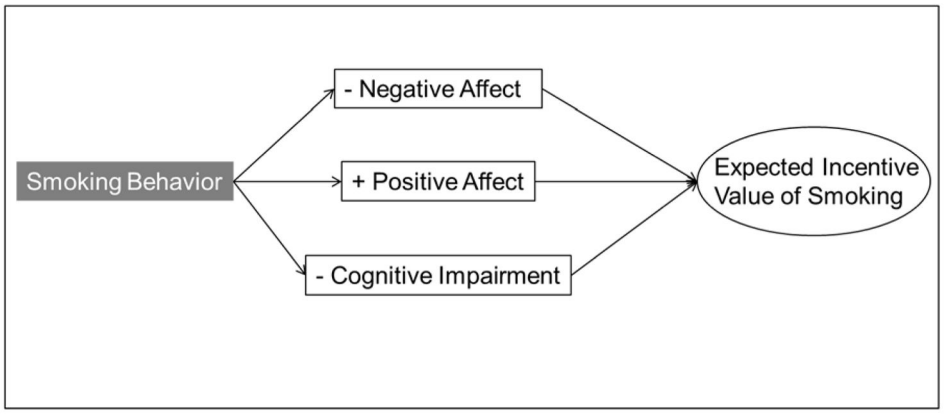


Figure 1.
Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) diagram: Summary of search and selection

A) Acute Effect of Smoking in Depressed Smokers



B) Chronic Effect of Smoking in Depressed Smokers

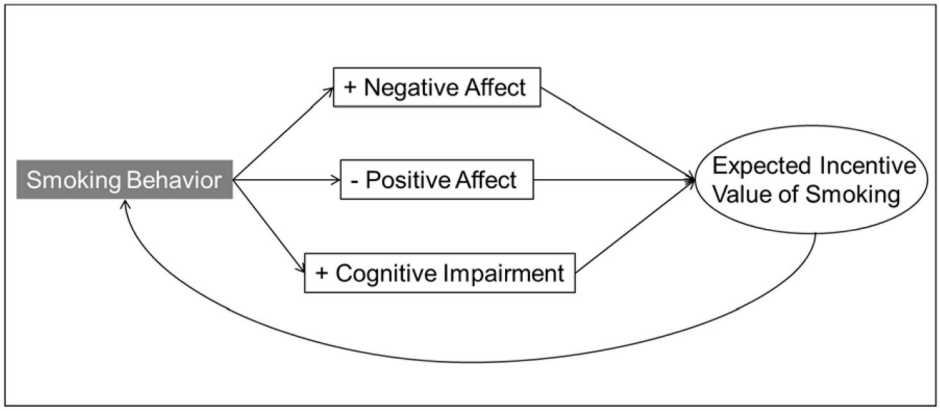


Figure 2.

Note. A) Incentive learning processes by which specific states (i.e., negative affect, low positive affect, and cognitive impairment) are acutely alleviated by smoking, and thus signal the enhanced incentive value of smoking. B) Enhanced incentive reward value fosters persistence of smoking though goal-directed selection of this behavioral choice. Chronically, smoking engages allostatic processes such that the aversive states are augmented. These aversive states in turn signal the higher incentive value of acute smoking, thus completing the vicious cycle.