



Review

The Role of Cognitive Control in the Self-Regulation and Reinforcement of Smoking Behavior

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Abstract

Cognitive control (CC)—the ability to regulate attention and memory—plays an important role in a variety of health behaviors, including smoking behavior. In this theoretical review of the literature, we propose a CC and smoking behavior framework that includes (1) the positive influence of CC on the self-regulation of smoking, (2) nicotine-induced improvements in CC that may indirectly reinforce smoking (including withdrawal reversal effects), and (3) the long-term effects of smoking on the brain that may result in reduced CC. Integration of these literatures suggests that CC contributes to both self-regulation (ie, brake pedal) and nicotine-related reinforcement (ie, gas pedal) amid the catastrophic effects of long-term smoking, which may reduce self-regulatory control over smoking while also enhancing indirect reinforcement. Supportive evidence and limitations of this approach will be presented, as well as ideas for future research directions that may fully examine this multifaceted modeling of CC in relation to smoking behavior.

Implications: There is substantial evidence that CC contributes to self-regulation (ie, brake pedal) and reinforcement (ie, gas pedal) of smoking behavior as well as evidence that long-term smoking may cause reduced CC. The proposed model delineates how these opposing influences of CC may mask the unique contribution of self-regulation and reinforcement in maintaining smoking behavior. Targeting CC for treating nicotine dependence will require more nuanced approaches that consider the independent and combined effects of self-regulation and reinforcement to improve smoking cessation success rates.

Cognitive Control

There is growing evidence that cognitive control (CC)—the ability to control attention and memory—is associated with cigarette smoking behavior in multiple respects, including self-regulation,^{1,2} nicotine-related reinforcement,² and long-term reduction of CC.³ CC is a general construct that encapsulates multiple cognitive processes that are relevant to the performance of daily activities that involve effort, including working memory, target detection, response inhibition, response

preparation, and attentional shifting.⁴ The “CC” and “executive function”⁵ labels are often used interchangeably, but the latter is generally viewed more broadly and includes the self-regulation of behavior in addition to CC processes.^{6,7} With respect to the current model, we utilize the label CC to distinguish between CC processes and the self-regulation of behavior in specific contexts (eg, smoking behavior).

The framing of cognitive processes that comprise CC as a general construct remains challenging, as it is difficult to isolate processes that overlap both empirically and conceptually, especially as

these processes operate amid whole brain activity. For example, the majority of CC measures require the capacity to sustain attention, regardless of the more specific CC process (eg, response inhibition, working memory) that are highlighted in the literature. The ability to sustain attention over time might therefore influence multiple CC facets. Similarly, measures of working memory are highly dependent on attentional control.⁸ Thus, if working memory is the construct of interest then attentional control is unavoidably involved as well. Consistent with this notion, different CC measures activate similar brain regions including the anterior cingulate cortex (ACC) and dorsolateral cortex (DLPFC), suggesting these are core neural substrates recruited by common CC resources.^{9–11} However, in spite of this apparent overlap among CC processes, measures of CC are often modestly correlated, or even uncorrelated.¹² It is important to note that even amid the noted frequent absence of substantive correlations among CC measures, the cognitive processes underlying these measures may additively and/or interactively contribute to the self-regulation of smoking and other CC–smoking links. As described below, there are several reasons to focus on CC as a general construct, as opposed to targeting a single process or subset of processes.

Studies examining the association between CC and smoking involve multiple domains, including response inhibition,^{13,14} working memory,^{15–17} multiple aspects of attention,^{2,3,17} and other processes.^{18,19} For example, meta-analyses found that nicotine acutely enhances a range of CC processes, including attention, episodic memory, and working memory. Moreover, nicotine may have beneficial effects on indices of response time and accuracy.²⁰ These findings indicate that inclusion of a single CC domain or index of CC functioning may not fully explain the effects of nicotine and tobacco use on CC. While response time as a measure of processing speed may cut across measures, more research is necessary to evaluate whether response time during a working memory task represents the same underlying process as response time during attentional set shifting. There is also compelling evidence that multiple domains of CC are implicated in other CC–smoking links, including the influence of long-term smoking on CC functioning,^{3,21,22} and the association between CC and the self-regulation of smoking (ie, achieving abstinence).^{13,15,16}

Ultimately, individual studies must rely on a limited set of measures due to time constraints and to avoid participant fatigue, which may ultimately lead to focusing on more specific measures of CC. However, the inclusion of well-validated, standardized measures with normative data (eg, NIH Toolbox) will provide additional rigor and result in reduced variability in CC measurement across studies. Given the above issues relevant to CC and smoking behavior, it is paramount that we examine CC more inclusively, as opposed to focusing more exclusively on a limited range of specific domains. In this theoretical review, we build a model of CC and smoking behavior that involves connecting multiple literatures, some of which are more developed than others. For example, research on the relationship between nicotine and CC is captured in large part by previous reviews and meta-analyses. Thus, our goal is to establish plausible links among self-regulation and both state and trait CC and highlight less developed areas that may inform future research and ultimately bridge gaps in our knowledge.

Self-Regulation and CC

The “CC” and “executive function” labels often seem to be used somewhat interchangeably. In this theoretical review, we discuss CC and self-regulation as distinct components of executive function because it is essential to demonstrate whether a given CC measure

is empirically associated with self-regulation (eg, capacity to resist smoking). William James proposed that willpower is dependent on basic attentional processes, noting that “The essential achievement of the will, in short, when it is most ‘voluntary,’ is to attend to a difficult object and hold it fast before the mind.”²³ CC may reflect the machinery by which self-regulation is made possible, whereas the execution of self-regulation involves motivations emanating from the “self” in addition to CC processes (eg, the personal desire to resist smoking). The evidence suggests attention (or more generally CC) underlies resources that seem to be used during self-regulation.²⁴

Self-regulation’s dependence on CC has been demonstrated in numerous studies and across behaviors.^{24–26} Neuroimaging studies reveal that CC (eg, working memory and inhibitory control) and self-regulation may have common neural substrates, including the ACC and DLPFC.^{11,27,28} Well-validated tasks that measure CC and require sustained, effortful attention acutely reduce self-regulation; similarly, tasks that require self-regulation acutely impair CC.²⁹ The importance of CC in self-regulation extends to various behaviors. For instance, performance on CC measures of working memory (measured by the operation span task) and inhibitory control (measured by the stop signal task) are positively associated with the self-regulation of eating behavior.³⁰ The connection between CC and self-regulation has been well-studied and has served as the foundation for several theories, including Baumeister’s self-control depletion theory,³¹ theories of behavioral inhibition in attention-deficit hyperactivity disorder (ADHD),³² theories of CC development,²⁶ as well as more general theories of self-regulation and CC.³³ Ultimately, the influence of CC processes on self-regulation may impact the ability to resist smoking.^{34,35} For example, Posner and Rothbart found an association between CC development and the early ability to control distress. Furthermore, performance on CC tasks among children correlated with parent-report of self-regulation amid daily life.³⁶ The converging evidence suggests that CC contributes to goal-directed behaviors, such as maintaining an exercise regimen, focusing on a singular task, or maintaining smoking abstinence.^{24,25} It also makes intuitive sense (ie, face validity) that various aspects of CC contribute to goal planning and execution, and therefore to smoking abstinence. That is, the ability to shift attention away from smoking-related thoughts and cues while remaining focused on alternative activities is fundamental to maintaining abstinence. We now address research that links CC with the capacity to resist smoking, presumably via improved self-regulation as described earlier.

CC and the Capacity to Resist Smoking

The research linking CC with self-regulation^{24,25} suggests that CC should be positively associated with the capacity to resist smoking. Indeed, there is evidence showing working memory,^{15,16} response inhibition,¹³ and other CC processes³⁷ as predictive factors. In general, these studies reveal that smokers with lower baseline trait CC or who experience greater CC deficits during withdrawal are the most vulnerable to relapse. However, the effect size of CC predicting smoking behavior tends to be small, indicating a need for more sensitive CC indices. Neuroimaging techniques such as functional magnetic resonance imaging (fMRI) have provided insight into the underlying neural mechanisms between CC and smoking behavior. fMRI blood-oxygen-level dependent responses in corticothalamic circuits (eg, right inferior frontal gyrus), which are important for inhibitory control processes, are associated with the ability to resist smoking.³⁸ Specifically, the brain regions activated when exerting CC have been associated with the ability to quit smoking. Similarly, deficits

in neural substrates resulting from nicotine withdrawal (ie, reduced working memory-related activity in the left DLPFC and increased activity in the posterior cingulate cortex) during abstinence predicted relapse.¹⁶ Several other studies also support a connection between CC and reward-related brain regions with smoking relapse.^{39,40}

While there is some support for the role of CC in smoking outcomes, CC must be considered relative to other factors that influence smoking behavior, such as cue-elicited craving and stress-precipitated smoking. For example, reduced CC may increase susceptibility to cue reactivity, and the exposure to cues may drain CC resources.⁴¹ Dual process theories, which involve fluctuations between automatic/implicit processes that encourage drug use (eg, smoking cues) and controlled/explicit processes (ie, CC), may provide insight into how to inhibit automatic processes.⁴² Tobacco use may be more likely to occur if automatic motivational-emotional processes (eg, craving) override CC-based efforts to resist smoking. While this theoretical review focuses primarily on CC and smoking, the complexities between CC, smoking outcomes, and factors independent of CC all require further analysis. Ultimately, an improved understanding of these interactions will place the field in a better position to develop more targeted treatment strategies.

Nicotine and CC

We have thus far suggested that because CC contributes to generalized self-regulatory capacity, CC may play a prominent role in the regulation of smoking behavior. We will now address the effects of nicotine on CC, and its role in the reinforcement of smoking behavior. The proposed model of CC in relation to smoking behavior is complex due to the opposing roles of self-regulation (ie, both nicotine-related and independent of nicotine) and nicotine-related reinforcement. While CC's role in self-regulation may actually enable inhibition of smoking behavior, nicotine-induced enhancement of CC may reinforce (ie, maintain) smoking behavior.

In addition to nicotine's primary reinforcing effects via activating the mesolimbic dopaminergic reward system,⁴³ it has CC-enhancing effects² which may act as an indirect reinforcer. That is, enhanced CC may positively affect emotional regulation⁴⁴ and the ability to efficiently complete daily tasks,² which are likely to be reinforcing. Nicotine's effects on CC support a self-medication hypothesis, whereby smokers with decrements in CC (due to either withdrawal or lower baseline CC) may find smoking reinforcing due to nicotine's effects on cognitive functioning.^{2,17} Beyond nicotine's enhancing effects on CC, evidence suggests the degree to which nicotine deprivation impairs CC varies as a function of one's baseline CC (ie, trait CC). Smokers with low trait CC, as measured by electroencephalogram and ERP neural substrates, exhibit greater reductions in CC following nicotine deprivation,¹⁹ and nonsmokers demonstrate greater enhancement of CC following nicotine administration.⁴⁵ Trait and state influences on CC may interact synergistically to produce greater deficits, meaning those with lower baseline CC are potentially doubly challenged and at heightened risk for relapse. More research is needed in this area, including the extent to which this synergy may predict difficulty in maintaining smoking abstinence.

Deleterious Long-Term Effects of Smoking on CC

Research suggests that tobacco use causes declines in CC processes throughout the lifespan. First, chronic smoking via nicotine may

disrupt the development of brain neurotransmitter processes that contribute to CC function during adolescence and early adulthood.³ An abundance of animal studies have shown that prenatal exposure to nicotine causes a range of CC deficits, including attention, learning, and memory.^{46,47} These findings may be explained by interruption of the normal development of prefrontal cortical areas that play important roles in the development of attention and other CC processes that continue into early adulthood.^{48,49} Evidence also suggests that middle aged smokers show greater decline in CC across a 5-year period compared with nonsmokers.⁵⁰ Smokers also show age-related declines in brain areas associated with Alzheimer's disease²¹, and relative to nonsmokers, smokers perform worse on CC tasks.²² The role of nicotine versus constituents in cigarettes independent of nicotine is often difficult to separate, but the preponderance of evidence nevertheless suggests that long-term smoking causes declines in CC processes across the lifespan.

CC and Smoking Status

The role that CC plays in smoking behavior may be especially relevant among contemporary smokers. The prevalence of smoking among adults in the United States has steadily declined across the past several decades. In 1964 (ie, year of Surgeon General's seminal report on smoking), the rate of current smokers was 43%.⁵¹ As of 2015, the smoking rate had declined to 15%.⁵² Despite this overall substantial decline in smoking prevalence, the rate of smoking among individuals with psychological disorders⁵³ and lower socioeconomic status (SES)⁵⁴ has remained high. Individuals diagnosed with psychological disorders are over twice as likely to smoke versus individuals without disorders.⁵⁵ Individuals diagnosed with ADHD, a disorder characterized by deficits in CC and self-regulation, are more likely to smoke and have more difficulty quitting.⁵⁶ However, evidence is mixed regarding whether individuals with ADHD experience greater withdrawal-related changes in CC, with some studies showing ADHD symptoms may exacerbate CC deficits during withdrawal⁵⁷ while others do not.⁵⁸ Among individuals with schizophrenia spectrum disorders, which are also characterized by low CC, the smoking prevalence ranges from 64% to 79%, and existing treatments are less effective in this population.⁵⁹ Given that more effective treatments are necessary for smokers with comorbid conditions, individuals with these CC-related disorders may garner greater benefit from treatment approaches that target CC. Lower SES (ie, lower income and/or lower educational attainment) is also associated with substantially higher smoking rates.⁶⁰ Smokers with psychological disorders and/or lower SES also exhibit substantially reduced capacity to successfully quit.^{61,62} Notably, schizophrenia, mood and anxiety disorders,⁶³ and lower SES^{60,64} are also associated with compromised CC. The links between psychopathology and SES with elevated smoking rates and lower CC highlight important health disparities that exist among the current population of smokers. To better treat individuals within clinical and low SES populations, it is essential to study the specific factors interacting between CC and smoking behavior more comprehensively.

Proposed Model of CC and Smoking Behavior

Based on the literature discussed thus far, the proposed model includes three key components. First, the model suggests that CC plays a role in the ability to sustain smoking abstinence due to its effect on self-regulation. That is, all else being equal (eg, motivation

to quit, nicotine dependence), the individual with stronger CC would be more likely to successfully maintain smoking abstinence. Current CC functioning may vary according to state factors (eg, disruption during nicotine withdrawal) as well as more chronic, trait-like factors (eg, comorbid psychiatric disorders), which in turn may additively impact self-regulation. For example, evidence that negative affect impairs CC⁶⁵ may contribute to a further reduction in self-regulation and enhance the reinforcement derived from smoking.² Indeed, reduced CC has been observed in depression and other negative affect-related disorders,⁶⁶ which may partially explain high smoking rates among those with psychiatric disorders.⁶⁷ The second component involves the capacity for nicotine to enhance CC (ie, including nicotine withdrawal reversal). We propose that nicotine's positive effects on CC provide indirect reinforcement of smoking behavior (ie, self-medication) because CC enhancement may enhance emotion regulation and performance of daily tasks that rely on CC. The model suggests that CC influences smoking in terms of both self-regulation (ie, brakes) and reinforcement (ie, gas pedal). These dual influences may make it difficult to disentangle the role of self-regulation versus the role of reinforcement without carefully designed research that aims to tease these variables apart. The third component involves the potential degrading effects of long-term smoking on CC, which are relevant to understanding CC in the context of the first two components (ie, CC-related self-regulation and indirect nicotine reinforcement). That is, because self-regulation over smoking behavior requires CC, a smoker with fewer CC resources as a consequence of years of chronic smoking may have more difficulty regulating smoking behavior. Additionally, nicotine effects on CC may be reinforcing as difficulty with self-regulation emerges. As shown in [Figure 1](#), the long-term effects of smoking on CC may create a negative feedback loop whereby CC relevant to daily functioning is reduced. As a result, the ability to self-regulate decreases, which in turn reduces the ability to maintain smoking abstinence. Thus, CC-related self-regulation, indirect smoking reinforcement, and consequences of long-term smoking represent three distinct factors that influence the reinforcement and self-regulation of smoking. In tandem with self-regulation, the model suggests that improvements in CC will improve self-regulation and thus the capacity to resist smoking—an assumption we discuss in detail in the next section.

Additionally, the dual process theory suggests that drug use-related decision making is influenced by competing motivational approach (eg, smoking) and self-regulatory CC systems.^{68–70} This is consistent with our model which proposes that CC may be an underlying mechanism of self-regulation of smoking behavior. Indeed, dual process theories have been applied to smoking behavior as well.⁷¹ An important distinction between our model and the dual process model is the inclusion of CC-related, nicotine-induced indirect reinforcement (ie, gas pedal) as well as the negative feedback loop associated with long-term effects of smoking. The motivational approach activation in our model may be an indirect consequence of the beneficial effects of nicotine on CC. In addition, the model posits the possibility of indirect smoking reinforcement through nicotine's effects on CC via emotion regulation and task efficiency (including reversal of withdrawal-related deficits). The proposed model and the dual process model also differ in the approach to treatment. The cognitive bias modification approach⁷² to treatment is focused on the motivational–emotional aspect of traditional dual process modeling. Specifically, the dual process model targets the motivational approach element, whereas interventions described in the next section are intended to enhance self-regulation through improved CC.

CC-Enhancing Therapies as a Strategy to Enhance Smoking Cessation

Interventions that target CC processes may represent novel strategies for smoking cessation^{73,74} by utilizing behavioral approaches (eg, cognitive training, exercise, mindfulness training), pharmacotherapy (eg, cognitive-enhancing medications), and more recently, noninvasive brain stimulation (NIBS) techniques. It is also important to note that certain subgroups of smokers who may be more vulnerable to CC deficits (eg, people diagnosed with ADHD,⁷⁵ schizophrenia,⁷⁶ and other disorders that may involve CC deficits⁷⁷) may benefit the most from interventions that improve CC.

In the following paragraphs, we provide an overview of studies that have evaluated these strategies as interventions for smoking cessation. Consistent with our broad approach to CC, our examination of interventions intended to improve CC (eg, exercise, mindfulness) encompasses a range of strategies. Overall, these treatment approaches may impact multiple facets of CC. Computerized cognitive training may be an exception in that a single module reflecting a more specific CC process may be targeted (eg, response inhibition; see below).

With respect to behavioral strategies, cognitive training seems like a natural choice in light of evidence showing computerized cognitive training enhances CC in healthy adults.⁷⁸ Multiple randomized controlled trials (RCTs) show cognitive training's positive influence on various facets of CC, including information processing speed,⁷⁹ attention,⁸⁰ inhibitory control,⁸¹ and working memory.⁸² These effects have been observed across diverse populations such as older adults⁸³ and traumatic brain injury patients.⁸⁰ Cognitive training has also resulted in beneficial effects on important health outcomes, including depression symptoms,⁸⁴ schizophrenia symptoms,⁸⁵ and problem drinking.⁸⁶ Furthermore, working memory training has been shown to reduce impulsivity (measured via delay discounting),⁸⁷ suggesting CC enhancements through cognitive training may improve self-regulation and thus enhance the ability to quit smoking. However, it is also important to note that critics have raised valid concerns, including issues related to task validity, inclusion of no-contact control groups, subjective measurements of cognitive changes, lack of consistent compelling evidence of skill transfer, and a paucity of true replications.⁸⁸ In one of the largest RCTs to date testing this hypothesis, Loughhead and colleagues randomized 213 smokers to either computerized cognitive training or a computerized relaxation control group.⁸⁹ Working memory, attention, and response inhibition training modules were included in the training. Beneficial effects of cognitive training on CC tasks were observed, but these effects did not transfer to higher quit rates among the cognitive training group. While this first set of results does not support cognitive training (ie, null findings) as an efficacious treatment for nicotine dependence, the approach may be more effective for some smokers than others (eg, those with lower baseline CC) and/or combined with additional cognitive-enhancing strategies, such as pharmacotherapy or brain stimulation techniques. Additionally, there were multiple training modules, including working memory, attention, and response inhibition along with other measures not directly related to CC. It is possible that more intensive training in a single domain of CC would yield better training effects (ie, more opportunity for specific training to take hold).

Exercise may also show promise for improving smoking cessation through behavioral CC enhancement. Only one smaller study that we know of has examined exercise and cognitive function in the context of smoking behavior, but this study did not demonstrate

that smoking cessation rates were improved by beneficial effects of exercise on CC.⁹⁰ Additional studies are needed to directly test whether CC mediates the effects of exercise on smoking cessation and whether this varies by type of exercise (eg, aerobic, resistance training, yoga).

The cognitive science literature provides evidence for mindfulness and related meditation practices enhancing neural and behavioral indices of CC functioning^{91,92} (eg, response inhibition,⁹³ P300⁹⁴ and error-related negativity⁹⁵ event-related potential (ERP) neural substrates of CC, and working memory performance⁹⁶). However, this burgeoning literature is tempered by a lack of precise replications (eg, replication of evidence that mindfulness improves the same outcome measured by a single task). Although more research examining the effects of mindfulness training on smoking cessation is needed, this is nevertheless a potentially promising treatment approach to improving CC processes and consequently improving smoking cessation efforts. No studies have tested whether enhanced CC mediates the effects of mindfulness on smoking cessation. Examining whether individuals with lower baseline CC derive greater benefit from mindfulness practice as a smoking cessation aid may provide insight for developing more targeted treatment strategies.

Despite promising evidence from studies involving potential pharmacotherapy effects on CC and smoking-related behaviors (eg, craving and number of cigarettes smoked), there is little evidence that CC mediates the improvements in smoking cessation. For instance, varenicline, an effective smoking cessation medication, and an $\alpha 4\beta 2^*$ nicotinic receptor (nAChR) partial agonist and full agonist at $\alpha 7$ nAChRs, has produced positive effects on multiple domains of CC.^{97,98} However, none of these studies examined whether varenicline's effects on cognitive performance accounted for its effects on smoking cessation. Acetylcholinesterase inhibitors (AChEIs), which are FDA-approved medications for Alzheimer's disease, have been proposed as plausible treatment aids. AChEIs increase levels of acetylcholine in the brain by inhibiting the catabolic enzyme responsible for metabolizing acetylcholine.⁹⁹ AChEIs have been shown to enhance cognition in smokers¹⁰⁰ and serve as a substitute for the discriminative stimulus properties of nicotine in humans.¹⁰¹ Specifically, the AChEI galantamine reduces smoking rate, craving, smoking satisfaction, and perceived reward from smoking.^{101,102} Similar to varenicline, no studies of AChEIs have demonstrated that enhanced CC mediates the effects of pharmacotherapy on smoking cessation success. Additionally, findings for the effects of AChEIs on smoking cessation are mixed.¹⁰³

Modafinil, which is FDA approved to treat narcolepsy and other sleep conditions,¹⁰⁴ is known to enhance dopamine and

norepinephrine activity and is another drug that has been found to enhance CC processing.¹⁰⁵ A clinical trial that examined modafinil as a smoking cessation treatment was suspended before completion because the drug appeared to be actually increasing smoking behavior in comparison with the placebo condition.¹⁰⁶ Similarly, a preclinical experimental study found that modafinil alone or in combination with nicotine replacement did not reduce nicotine withdrawal.¹⁰⁷

Transcranial magnetic stimulation (TMS; magnetic fields that evoke electrical current stimulation) and transcranial direct current stimulation (tDCS; soaked surface sponge electrodes on the scalp that deliver electrical current) are well-established forms of NIBS methods that increase/decrease brain activity. Both methods have been shown to increase various facets of CC, including attention, learning, and working memory.¹⁰⁸ Recent discoveries present the interesting notion of using NIBS to promote CC as a means to treat substance abuse, including smoking cessation, with the most commonly targeted region being the DLPFC.¹⁰⁹ A recent meta-analysis found tDCS targeting the left DLPFC enhanced the working memory.¹¹⁰ Additionally, several reviews of NIBS studies show stimulating the DLPFC may attenuate craving in the context of substance abuse (including nicotine) and highly palatable food,¹¹¹ making it a promising approach to treat addiction (including smoking cessation) via improved CC.¹¹² Both TMS¹¹³ and tDCS¹¹⁴ have been shown to improve cessation rates and enhance the ability to resist smoking in a laboratory paradigm; thus, the NIBS literature supports utilizing these methods as a potentially promising means to promote smoking cessation via improved CC.

We view the above treatment approaches as potential avenues to enhance self-regulation in the context of smoking cessation by enhancing CC. Independent of smoking behavior, interventions to improve CC is a challenging area of research. Only two aforementioned clinical trials have examined therapies intended to improve CC as a means to improve smoking cessation efforts (ie, cognitive training⁸⁹ and modafinil trials¹⁰⁶), and neither of these studies positively impacted smoking cessation. However, we have noted a number of preclinical findings suggestive of CC treatment effects that have translational potential for impacting indices of smoking behavior in humans as well as promising pilot studies indicating a signal for intervention efficacy. While we suggest that this area of research holds promise, the field might be best characterized as being in the earlier stages of development. Future research needs to (1) emphasize basic research that identifies well-validated CC measures as markers of the capacity to quit, and (2) conduct research that shows these CC markers of smoking abstinence can be improved via treatment approaches that improve CC. Ultimately, smoking cessation clinical

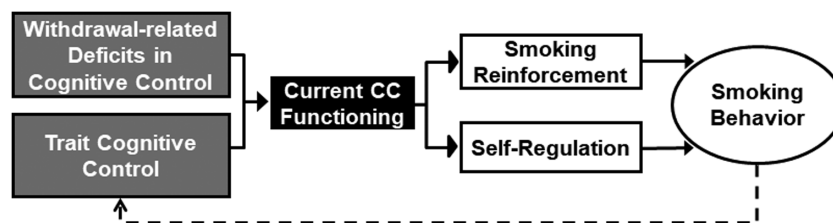


Figure 1. Model of cognitive control (CC) in relation to smoking behavior. Our proposed model suggests both nicotine withdrawal-related disruption of CC as a state factor and trait CC (gray rectangles) impact current CC functioning, which in turn impacts self-regulation and smoking reinforcement (white rectangles). Depending on whether these factors increase or decrease self-regulation (ie, brake pedal) and/or smoking reinforcement (ie, gas pedal), this will lead to a decision point: smoke or not smoke. This decision influences whether smoking behavior or smoking abstinence is maintained. Finally, long-term smoking may have detrimental effects on CC, thus creating a negative feedback loop (dashed line), which may also impact CC-related indirect reinforcement of smoking behavior.

trials may then be in a better position to examine these CC markers in the context of a therapeutic approach to improve CC.

Finally, it is important to note that individuals who possess fewer CC resources may profit the most from CC-enhancing treatments that are implemented to improve the capacity to remain abstinent.¹¹⁵ Individuals with greater decrements in CC may lack sufficient CC resources to both resist smoking and cope with daily activities and stressors that compete for these limited resources. Thus, research may examine individual differences in CC in an effort to test this hypothesis, which in turn may lead to tailored and/or matching of CC-enhancing treatments based on individual smoker CC characteristics.

Concluding Remarks

We assert that research involving CC via self-regulation on the capacity to resist smoking is still in its formative stage. Empirical work is needed to (1) identify and validate CC measures as predictors of the capacity to resist smoking, (2) evaluate whether treatments that enhance CC improve self-regulation and whether these effects translate into the capacity to remain abstinent, (3) examine whether the effect of CC-enhancing treatments on smoking cessation is mediated by beneficial effects on CC, and (4) identify state and trait factors that may predict for whom and in what manner this approach to smoking cessation might be most appropriate. As shown in Figure 1, we propose a model in which trait CC and nicotine both impact current CC functioning, which in turn impacts self-regulation and indirect reinforcement. Changes in CC and concomitant self-regulation occur amid a variety of states, including nicotine withdrawal, acute stress, and affective changes. The negative impact of long-term smoking on CC is also important to consider, as reduced CC from smoking across time may reduce self-regulatory capacity over smoking, thereby enhancing indirect nicotine-related smoking reinforcement. Because we are connecting diverse literatures to address gaps in the literature, empirical research is needed to test and validate the proposed model. In developing this model, we have attempted to incorporate multiple disciplines that involve varying levels of empirical support ranging from robust (eg, nicotine's impact on CC processes) to sparse (eg, evidence demonstrating that nicotine's positive effects on CC actually impact smoking behavior). Investigating interactions between CC, cue reactivity, and stress responses, as well as research to evaluate the efficacy of treatments designed to enhance CC to enhance smoking cessation, will also be important as the field evolves.

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Declaration of Interests

None declared.

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