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# Implicit and explicit reward learning in chronic nicotine use

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ABSTRACT

*Background:* Chronic tobacco use is related to specific neurobiological alterations in the dopaminergic brain reward system that can be termed "reward deficiency syndrome" in dependent nicotine consumers. The close linkage of dopaminergic activity and reward learning led us to expect implicit and explicit reward learning deficits in dependent compared to non-smokers. Smokers who maintain a less regular, occasional use may also, to a lesser extent, show implicit reward learning deficits. The purpose of our study was to examine the behavioral effects of the neurobiological alterations on reward related learning. We also tested whether any deficits observed in an abstinent state are also present in a satiated state.
*Methods:* In two studies, we examined implicit and explicit reward learning in smokers. Participants were administered a probabilistic implicit reward learning task, and an explicit reward- and punishment-based trial-and-error learning task. In Study 1, we compared dependent, occasional, and non-smokers, and in Study 2 satiated and abstinent smokers.
*Results:* In Study 1, chronic and occasional smokers showed impairments in both, implicit and explicit reward learning tasks. In Study 2, satiated smokers did not perform better than abstinent smokers.
*Conclusions:* The results support the hypothesis of reward learning deficits. These deficits are not limited to explicit but extend to implicit reward learning and cannot be explained by tobacco withdrawal.


## 1. Introduction

Nicotine is a highly addictive substance that leads to dependence faster and more often than many other drugs (O'Brien, 2001). One of the main causes for the high addictive potential of nicotine is its legality; another is its fast and indirect stimulation of the dopaminergic brain reward system (BRS). Particularly relevant BRS structures are the nucleus accumbens (NAc) and the ventral tegmental area (VTA; Di Chiara, 1992, 2002; Mao and McGhee, 2010). The incentive salience of nicotine and all its associated cues are boosted after the strong dopaminergic stimulation. Tobacco-associated cues alone begin to reward or rather reinforce, and are therefore learned faster and are more intense (Balfour et al., 2000). At the same time, the incentive salience of alternative reinforcers decreases (Robinson and Berridge, 1993; Volkow et al., 2003), and learning with non-drug reinforcing stimuli is impaired (Bühler et al., 2010). The memory traces of nicotine-associated cues are often resistant to extinction and are recoverable even after many years of abstinence (Chiamulera et al., 1996).

### 1.1. Reward learning in addiction

Reward learning is not only relevant for nicotine dependence, but addiction in general. The Incentive Sensitization Theory by Robinson and Berridge (2000) emphasizes the role of different associative learning processes in addiction. Addiction emerges after drug-induced alterations in BRS circuitry and associated changes in motivational processes and associative learning. In their corresponding model of reward, Berridge and Robinson (2003) distinguish three components of reward: liking, wanting, and learning. Liking is the emotional component, whereas wanting is the incentive motivational component. The learning component purveys the ability to predict reward, and hence forms the basis of wanting. Berridge et al. (2009) further distinguish between different associative learning processes that can be classified as explicit vs. implicit.

Based on these models, we expect that implicit and explicit reward learning processes play distinct roles during development and maintenance of addiction. With regard to their neuropsychology, implicit and explicit reward learning involve distinct neural circuits (Frank and Claus, 2006). Frank and Claus (2006) proposed that the dopaminergic basal ganglia (BG) system underlies implicit, context-dependent response initiation based on the relative probability of positive or negative outcomes, hence implicit reward-dependent learning. The dopaminergic activity in the BG determines whether a response is executed or inhibited, according to the contingencies of the response. This is a slow,

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implicit associative learning process, where a positive outcome promotes a behavior and a negative outcome inhibits a behavior. Explicit response selection based on anticipated rewards, however, requires a top-down control of the dopaminergic activity in the BG by the orbitofrontal cortex (OFC), which provides estimates of reinforcement magnitudes activated in working memory. This explicit system is successful at estimating the true expected value of reward-related decisions and is fast in switching behavior while changing reinforcement contingencies. Due to the nicotine-induced alterations in the BRS, including BG and OFC (Dagher et al., 2001; Volkow et al., 2002a,b; Fehr et al., 2008), implicit and explicit reward-related learning may be altered in nicotine addiction.

### 1.2. Previous research

To our knowledge, there are no behavioral studies that separately examined implicit and explicit reward learning in nicotine addiction. A hint for alterations in implicit reward learning comes from animal studies. Using a Pavlovian discriminative approach, Olausson et al. (2003) found that repeated nicotine administration temporarily improves implicit reward related learning in rats. Besheer and Bevins (2003) demonstrated that abstinence phases during chronic nicotine administration lead to deficits in the conditioning of place preference, a classic implicit reward learning paradigm.

There are a few studies on explicit reward processing in humans. In an imaging study, Martin-Soelch et al. (2001) compared satiated smokers and non-smokers in a delayed pattern recognition task with or without monetary feedback. In both groups, monetary reward led to activations in the occipital, frontal and orbitofrontal cortex, cingulate gyrus, cerebellum and midbrain. Reward related activations in the typical dopaminergic regions such as the striatum were only found in non-smokers, i.e., smokers showed a reduced processing of non-drug rewarding stimuli. These results were replicated in a further study by Martin-Soelch et al. (2003). Using the delayed pattern recognition task, the authors varied the amount of monetary reward. Smokers and non-smokers showed an involvement of a cortico-subcortical loop, including the dorsolateral prefrontal cortex, the orbitofrontal cortex, the cingulate gyrus and the thalamus in processing increasing monetary reward. Again, reward related activations in the striatum were only found in non-smokers. Furthermore, smokers showed no significant mood changes in response to the different monetary rewards.

Further support for deficits in explicit reward learning in smokers comes from studies using reward-related decision-making paradigms. Chronic tobacco users exhibit abnormal reactivity to reinforcers (Bickel and Madden, 1999), a reduced subjective value of delayed drug and non-drug rewards in a delay discounting paradigm (Bickel et al., 1999), and deficits in the anticipation of reward in a behavioral choice task (Mitchell, 1999).

### 1.3. Present research

The aim of the present study was to examine alterations in reward learning in smokers. In addition to distinguishing explicit and implicit learning, we also want to consider two other relevant factors, frequency of use and satiation. To point out the relevance of frequency, we need to review the developmental stages of addiction. The initial nicotine dose during tobacco smoking leads to a dopaminergic overflow in the VTA and the NAc shell and is experienced as rewarding (Koob, 2006). With occasional, repeated use DA release is reduced and the rewarding effects are diminished. Chronic tobacco use in addition has an inhibitory effect on DA releasing neurons in the mesolimbic system due to desensitization of the nicotinic acetylcholine-receptors (nAChR) (Koob, 2000), further decreasing the rewarding effects of nicotine. These neuroadaptive changes affect the tonic and phasic DA signals in the BRS, which are important for implicit reward learning (Di Chiara, 1999; Schultz, 2002). Hence we expect deficits in implicit reward learning in chronic, dependent smokers. However, as nicotine associated DA release is reduced already after occasional use, deficits in implicit reward learning should also occur in occasional, non-dependent smokers. We expect no deficits in explicit reward learning for occasional smokers, as orbitofrontal control is intact, as evidenced by the apparent control over nicotine use and no heightened nicotine cue reactivity (Haight et al., 2012) until chronic use orbitofrontal control is impaired, similar to other drugs as alcohol, cocaine, and methylphenidate abuse (Volkow et al., 2002a,b). Hence we expect deficits in explicit learning in dependent smokers only.

The second relevant factor we want to consider in our present study is satiation. In dependent smokers, nicotine withdrawal further reduces the NAc DA release by 25% (Hildebrand et al., 1998). This leads to a reduced responsiveness of the BRS to other rewarding stimuli (Volkow et al., 2003), which, in turn, is associated with reduced appetency and decreased interest in reward (Robinson and Berridge, 1993). Gutkin et al. (2006) termed this behavioral effect a hypohedonic state that could be countered by actual nicotine consumption or associated cues. With respect to our hypotheses, we expect that any reward-learning deficits in dependent smokers are also compensated by acute nicotine consumption and, hence, are not observable in a satiated state.

### 1.4. Predictions

The close linkage of dopaminergic activity and reward-dependent response selection led us to expect reward learning deficits in dependent tobacco smokers: the reduced number of dopamine-D2 receptors and the dampened dopamine neurotransmission impair the dopaminergic BG system, thereby causing a deficit in implicit reward learning. A dampened dopamine neurotransmission would also impair the OFC regulated response selection, causing a deficit in explicit reward learning. To test our assumptions, we conducted two behavioral studies. In Study 1 we examined reward learning in dependent, occasional, and non-smokers. In Study 2 we compared the performance of dependent smokers in a satiated and abstinent state.

We expected impaired performance in implicit and explicit reward learning for dependent smokers in comparison to non-smokers. We further expected that any such deficits in dependent smokers are only observable in an abstinent but not in a satiated state.

With respect to occasional, repeated but not dependent smoking we expected reduced implicit reward learning, but no deficits in explicit reward learning, compared to non-smokers.

## 2. Study 1

### 2.1. Method

*2.1.1. Participants.* Subjects were students of the Martin-Luther-University Halle-Wittenberg, as well as their relatives and acquaintances. All participants signed informed consent before participating. The study was accomplished in compliance with the declaration of Helsinki. Of the overall group of $n = 75$ subjects, one non-smoker with a comorbid depression, one smoker with a comorbid bulimia nervosa, and one non-smoker with epilepsy were excluded from the analysis.

The sample consisted of three groups: 27 dependent smokers (seven males; fulfilling criteria of tobacco dependence of DSM-IV, daily consumption, and at least four withdrawal symptoms),