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Do social stimuli increase cue-reactivity? Visual event-related potentials in smokers

(Verstärkt der soziale Bezug von substanzassoziierten Bildern Effekte der suchtspezifischen Reaktivität? Ereigniskorrelierte Potenziale bei Rauchern)

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Abstract

Background: In various studies of addictions, event-related brain potentials (ERP) have been used to demonstrate reactivity to substance-associated stimuli (cue-reactivity). In smokers there is evidence for cue-reactivity effects for the ERP components N300 and P300, as well as in slow positive wave (SPW).

Objective: The study at hand attempted to confirm ERP cue-reactivity effects in smokers and to investigate whether the social reference of stimuli would increase these effects.

Methodology: Following a stressor, ERP were evoked in 24 smokers and 19 non-smokers by pictures of objects and persons both with and without association to smoking.

Results: No significant cue-reactivity was found for smokers' N300 or P300 components. At the medial and posterior scalp some statistical analyses found significant cue-reactivity for slow positive wave 500 to 850 ms after stimulus-onset. Smoking stimuli evoked higher positivity than non-smoking stimuli in smokers. For non-smokers the stimulus differences were substantially smaller. Post-experiment ratings of experimental stimuli revealed higher craving after smoking stimuli than after non-smoking stimuli in smokers. This difference was higher in objects stimuli than in persons stimuli. Smokers perceived smoking stimuli as more pleasant than non-smoking stimuli while the ratings were reversed for non-smokers.

Conclusions: This study casts doubt on smoking cue-reactivity in ERPs' components N300 and P300 but suggests that cue-reactivity might rather be observed in the domain of SPW. This result is consistent with other studies demonstrating SPW cue-reactivity in different types of addictions. However, SPW cue-reactivity has only limited statistical significance. SPW cue-reactivity in smokers could reflect increased allocation of processing resources for smoking-related stimuli. SPW cue-reactivity seems to be independent from the social reference of stimuli (persons, objects). However, social reference seems to modulate cue-reactivity in self-report measures like craving ratings in an attenuating way. The effects of social reference are discussed in terms of distraction from smoking-association and inflation of the semantic space.

Keywords: event-related potentials, N300, P300, slow positive wave, smoking cue-reactivity, social cues, social stimuli, tobacco addiction, smoking

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1. Introduction

Stimuli associated with the consumption of a drug or psychotropic substance have been shown to elicit various reactions in addicts. Symbolic-expressive reactions, like reports of an increased "conscious experience of a desire to take a drug" (*craving*, Drummond, 2001, p. 35), have been found as well as behavioral (e.g. amount of a drug consumed, latency to consume a drug, cigarette puff frequency), autonomic (e.g. heart rate, skin conductance), and central physiological reactions (e.g. event-related brain potentials, fMRI blood oxygenation level-dependent responses) (Carter & Tiffany, 1999; Drummond, Cooper, & Glautier, 1990; Franken, Stam, Hendriks, & van den Brink, 2003; Niaura et al., 1988; for a review of neuroimaging studies: Wilson, Sayette, & Fiez, 2004). The specific reactions provoked by substance-associated stimuli in addicted subjects (*cue-reactivity*, Niaura et al., 1988) are regarded to be an important factor in the pathogenesis and maintenance of addiction disorders including smoking as well as in relapse after drug use cessation (Abrams, Monti, Carey, Pinto, & Jacobus, 1988; Chiamulera, 2005; Niaura, Abrams, Demuth, Pinto, & Monti, 1989; Niaura et al., 1988; Rohsenow, Childress, Monti, Niaura, & Abrams, 1991; Tiffany, 1990; Waters et al., 2003).

In recent years, researchers began to use event-related brain potentials (ERP) of the electroencephalogram (EEG) to investigate cue-reactivity in addicted subjects: in alcoholics (Genkina & Shostakovich, 1986; Herrmann et al., 2000a; Namkoong, Lee, Lee, Lee, & An, 2004), social drinkers (Herrmann, Weijers, Wiesbeck, Böning, & Fallgatter, 2001b), heavy cannabis users (Wölfling, Flor, & Grüsser, 2008), heroin addicts (Franken et al., 2003; Lubman, Allen, Peters, & Deakin, 2008; Lubman, Allen, Peters, & Deakin, 2007), cocaine addicts (Franken, Hulstijn, Stam, Hendriks, & van den Brink, 2004; van de Laar, Licht, Franken, & Hendriks, 2004), and smokers (Jang, Lee, Yang, & Lee, 2007; Littel & Franken, 2007; McDonough & Warren, 2001; Parker & Gilbert, 2008; Warren & McDonough, 1999). Irrespective of the very early and methodologically variational Russian study of Genkina and Shostakovich (1986), Warren and McDonough (1999) were the first to use the ERP approach for the measurement of cue-reactivity in addiction. Warren and McDonough evoked visual ERP by presenting randomly mixed smoking-related and neutral pictures of single persons or people to non-

smokers and light-moderate smokers after the completion of a stressor task. Subjects were only required to watch the stimuli attentively, no further task was given. In smokers, Warren and McDonough found greater amplitudes of a frontally dominant, negative component of the ERP labeled N300 for pictures without smoking content than for smoking-related pictures. Additionally, a subsequent parietally dominant, positive component identified as P300 was greater in smokers when they were confronted with smoking-related stimuli than when confronted with neutral stimuli. P300 cue-reactivity was correlated with smokers' ratings for stimulus valence. Stimulus differences in non-smokers' ERPs were either not significant (N300) or smaller (P300) than in smokers ERPs. In a replication of their experiment, McDonough and Warren (2001) used the same paradigm with non-smokers, non-deprived smokers, and twelve hour tobacco-deprived smokers, finding greater N300 cue-reactivity in deprived smokers than in non-deprived smokers. P300 cue-reactivity, however, was not affected by deprivation and there were also some stimulus differences in non-smokers' P300 amplitudes, suggesting that this component might be less specific to nicotine-addiction. Both of Warren and McDonough's studies used stimuli depicting people. This begs the question about the necessity and precise effects of the social stimulus reference in cue-reactivity experiments and led to the study at hand (see section 1.5.).

1.1. Excursus: Concepts of the relationship between drug-associated stimuli and provoked reactions in addicts - theories of cue-reactivity

Cue-reactivity ERP studies in smokers like those conducted by Warren and McDonough (1999; McDonough et al., 2001) must be differentiated from non-cue-reactivity studies. Non-cue-reactivity ERP studies might explore the effects of smoking or of acute nicotine application/deprivation on ERP latencies and amplitudes, and they might use non-nicotine-associated stimuli to investigate information processing (e.g. Anokhin et al., 2000; Edwards, Wesnes, & Warburton, 1985; Gilbert et al., 2004; Hasenfratz, Michel, Niel, & Bättig, 1989; Houlihan, Pritchard, & Robinson, 1996; Knott, 1986, 1990; Knott, Bosman, Mahoney, Ilivitsky, & Quirt, 1999; Knott & De Lugt, 1991; Knott & Venables, 1978; Le Houezec et al., 1994; Neuhaus et al., 2006; Pineda, Herrera, Kang, & Sandler, 1998; Stough, Bates, Mangam, & Pellett, 1995). However, non-cue-reactivity ERP studies do not investigate the *effects of nicotine-associated stimuli in relation to neutral stimuli*.

The cue-reactivity paradigm refers to an empirical approach in which addicted subjects are confronted with stimuli having a close association to the addiction-specific substance or its consumption in order to explore the provoked reactions. This research approach is sometimes referred to as *cue-exposure* even though the same term is also used for a certain intervention approach in addiction treatment (Drummond, Tiffany, Glautier, & Remington, 1995). For reasons of clarity, the term "*cue-reactivity*" is used in this publication to label the research approach and "*cue-exposure*" for the treatment approach. Cue-reactivity research does not deal with direct substance effects but investigates the effects of substance-associated stimuli. According to Glautier and Tiffany (1995, p. 77), "the attempt to identify (a) differences in responses elicited by drug-related and neutral stimuli, and/or (b) differences in responses elicited by drug-related stimuli amongst different groups of subjects" is central to studies of cue-reactivity. Furthermore, "a core assumption of cue-reactivity investigations is that stimuli previously associated with drug taking will elicit distinctive patterns of responses because of the drug user's history of experiences with those stimuli."

Various theoretical concepts have been formulated to describe and explain the relationship between drug-associated stimuli and the reactions to them. These cue-reactivity theories try to describe simply the relationship between stimulus and reaction and usually neglect other factors contributing to variance in results like unconditioned stimulus characteristics and conditioned stimulus characteristics (see Glautier & Remington, 1995 for an overview). Further semantic factors like "overlap with pharmacological effect" (Mucha, Geier, & Pauli, 1999) and social reference (Niaura, Abrams, Pedraza, Monti, & Rohsenow, 1992) seem to also contribute to the diversity of results in cue-reactivity research (see section 1.2.) as well as modulating situational factors like stress and substance availability (see section 1.3.).

Several influential theories describe the stimulus-reaction relationship in terms of classical conditioning. Formerly neutral stimuli acquire the ability to elicit conditioned responses through repeated association with the consumption of a drug and/or its pharmacological effects. The relationship between these conditioned responses to drug-associated stimuli and the original unconditioned responses to the drug itself is described differently in the various conditioning theories which assume different characters of the conditioned responses (withdrawal-like, compensatory/drug-opposite, drug-

like; Drummond et al., 1995). According to these theories, operant mechanisms following the classical conditioning processes can account for the perpetuation of drug-use behaviors. Although most of the conditioning theories are formulated for a specific drug, they are applied to other drug types as well, including nicotine dependence and smoking.

In his influential theory, Wikler (1948, alcohol: Ludwig & Wikler, 1974) argues that during morphine addiction several stimuli acquire the ability to evoke conditioned responses that resemble withdrawal symptoms. These withdrawal-like conditioned responses are aversive, but can be relieved by drug intake. The resulting negative reinforcement of drug consumption could be an important factor in the perpetuation of addiction or relapse after interim abstinence.

In Siegel's model (1975), conditioned responses to drug-associated stimuli are likewise assumed to have an aversive quality, but not to resemble withdrawal symptoms. Rather, conditioned responses are regarded to be opposed to the direct effects of a drug (see also Koob & Le Moal, 2001). Siegel supposes that the direct effects of a drug are accompanied by opposed *compensatory responses* to maintain or reconstitute a condition of equilibrium. The conditioned drug-associated stimuli have acquired their properties by an association with these compensatory responses. By means of this association, the conditioned stimuli can evoke drug-opposed conditioned compensatory responses that are comparable with the original compensatory responses. The result, then, of the classical conditioning process is a conditioned tolerance for the pharmacodynamical effects of the drug. However, if conditioned compensatory responses are evoked by drug-associated stimuli that are not followed by an actual drug intake, an aversive state of physiological disequilibrium is generated. According to Siegel (1975), drug intake during this aversive state has palliating effects motivating the subject to perpetuate the drug consumption in the long run.

While the models of Wikler and Siegel postulate aversive conditioned responses, Stewart, de Witt, and Eikelboom (1984) propose that conditioned responses evoked by drug-associated stimuli are positive and pleasant in nature. The conditioned stimulus acquires positive affective properties by the association with the positive direct effects of the drug. In this model, the conditioned responses resemble the direct effects of the drug, but are less intensive. The conditioned responses therefore have strong appetitive effects and activate motivational processes that lead to the consumption of a drug to

intensify the positive sensations. In this model, positive reinforcement leads to the perpetuation of drug consumption.

Unlike the conditioning models mentioned above, Tiffany (1990, 1997) proposes cognitive processes and structures as the basic principles of cue-reactivity instead of conditioning. Tiffany hypothesizes that drug use is initiated and coordinated by automatized action schemata that are shaped through the repetition of uniform drug-use behaviors. According to Tiffany, these schemata are usually activated by specific stimulus constellations or situations and are performed with little cognitive effort. The processes involved in the development of craving are seen as separate and independent from the automatized action schemata, although they are commonly activated simultaneously. Craving or urges can occur if non-automatic cognitive and motor processes are activated to either support or impede the execution of the drug-use schemata. If drug use is suppressed or if a drug is used against external impediments, physiological reactions, overt behaviors, and verbal reports are triggered. In this model, craving is not a necessary preceding factor for the initiation and perpetuation of automatized drug-use behavior.

While Drummond (2001) reviews several other cue-reactivity theories in addition to the ones described above, even this short overview gives an impression of the diversity of the ideas in this field. The evidence does not support one single theory: no single theory can account for all empiric findings, results are complex and often contradictory, and the exact relationship between craving, cue-reactivity, and relapse is still unsettled (Drummond, 2001). Other possible explanations for the effects of exposure to cues can be claimed, as is done for example by the social learning theory. Older concepts, like the original two-process theories of the interaction between operant and classical conditioning, have been succeeded by the concept of central emotional states as mediators of instrumental behavior (Rescorla & Solomon, 1967).

In their meta-analysis of cue-reactivity research, Carter et al. (1999) found a pattern of physiologic reactions in smokers that was compatible with the appetitive model (e.g. Stewart, de Witt, & Eikelboom, 1984). Smoking-associated stimuli evoked physiological reactions that were comparable to the reactions induced by nicotine itself such as increases in heart rate and sweat gland activity, although decreases in skin temperature were not significant. Nonetheless, this is not sufficient evidence for the appetitive model as other explanations of this reaction pattern, like Tiffany's (1990, 1997), are still cogit-

able and results for other drugs do not consistently favor the appetitive model. Instead of reflecting conditioned responses, cue-reactivity could represent other processes such as an orienting response, general arousal, or frustration (e.g. Drummond et al., 1990).

To account for a wider range of findings, more integrative models have been developed. They can not, however, cover all phenomena. Baker, Morse, and Sherman (1986) propose a dual affect model to integrate empirical findings of increased relapse and craving during both positive and negative affective states, and concomitant with drug-agonistic, as well as drug-antagonistic/withdrawal responses. They hold that drug cravings are processed in neural affective systems and suggest that drug taking behaviors are regulated by these affective processing systems. Two types of neural craving networks are postulated: first, a "*positive-affect*" *network* that is activated by appetitive stimuli (e.g. priming doses) in an associative or non-associative manner and that is linked to brain reward systems; second, a "*negative-affect*" *network* which can be also activated associatively and non-associatively by inappetitive events like stress, frustration, punishment, and withdrawal as well as by stimuli associated to these events. While the activation of the negative-affect network should be accompanied by unpleasant affects and withdrawal symptoms, the activation of the positive-affect network should result in pleasant affects. According to Baker, Morse, and Sherman (1986), the activation of one system should inhibit the activation of the other system.

In their three-pathway psychobiological model of craving for alcohol, Verheul, van den Brink, and Geerlings (1999) integrate a wide array of empirical findings and propose "*reward craving*" as a desire for the rewarding and stimulating effects of alcohol, "*relief craving*" as a desire for the reduction of aversive states, and "*obsessive craving*", as a lack of control over intrusive thoughts about drinking. The first two pathways can be seen as analogous to the positive and negative affect craving networks postulated by Baker, Morse, and Sherman (1986).

The older conditioning models (Siegel, 1975; Stewart et al., 1984; Wikler, 1948) dealt mainly with the emotional valence of the classically conditioned response of substance-associated stimuli and assumed that operant mechanisms made this emotional state a trigger for relapse. In the more integrative models of Baker, Morse, and Sherman (1986) and Verheul, van den Brink, and Geerlings (1999) emotional states are seen as crucial for relapse as well; however, they are not necessarily seen as conditioned responses to substance-associated stimuli but can have been caused by a variety of factors.

These theories show the importance of considering and manipulating emotional states in cue-reactivity experiments. This was accounted for in this study by using a stressor task to evoke negative affect craving (see also section 1.3.). In their model, Verheul et al. (1999) describe evidence for the neurobiological basis of the different pathways and postulate that for alcoholics reward craving might arise from a dopaminergic dysregulation. The release of dopamine in the nucleus accumbens is related to the positive reinforcing effects of alcohol and the mesocorticolimbic dopaminergic system might be important for incentive motivational learning. Similarly, Robinson and Berridge (1993; Berridge & Robinson, 1995) underscore the role of dopamine in their incentive-sensitization theory of addiction. They hold that repeated drug use results in a sensitization to dopamine transmission in the striatum, increasing the incentive motivational properties of drugs and drug-associated stimuli. This process can be described as "*incentive salience,*" a process by which stimuli become especially attractive "wanted" stimuli. The repeated use of a drug, then, causes drug-associated stimuli to acquire incentive value. The drug use caused sensitization and the resulting hypersensitivity of these structures might be an irreversible neuroadaptive alteration leading to an excessive "*wanting*" of the drug that might even persist after abstinence. Drug cues that have acquired excessive incentive salience can act as potent perpetuators of further drug-consuming behaviors. The processes of wanting are seen as independent from processes of withdrawal and the experience of pleasant drug effects ("*liking*"), which are assumed to be mediated by different neural structures. According to Robinson and Berridge, sensitization of incentive salience can induce drug taking behavior even when the state of withdrawal or expectations of drug pleasure (liking) are lacking. These assumptions are picked up by Franken (2003) in a similar theoretical account of addiction. In his model, he assumes that attentional bias emerges from the increased dopaminergic activity during repeated drug use (see Robinson & Berridge, 1993: incentive salience, sensitization, formation of liking): drug-associated stimuli become extremely attractive and attention grabbing, and drug users are hyperattentive to these stimuli. Attentional bias, then, activates the feeling of craving that, reciprocally, enhances the attentional bias since attentional bias and craving are seen to modulate each other. Franken argues that attentional bias contributes to drug use behaviors by facilitating the formation of further drug cues that can be associated to conditioned responses that precede relapse or continued drug use. Also, drug cues are assumed to lead to a preoccupation with the drug, to memory bias, and intrusive thoughts making it difficult to draw attention away from these stimu-

li, resulting in increased craving. Finally, the absorption of cognitive resources with drug-associated stimuli can result in a consecutive malfunction in the processing of alternative stimuli, resulting in an attentional narrowing towards drug cues.

A processing bias for substance-associated stimuli has been shown for various addictions and several methods. Performance in the emotional Stroop task and visual probe tasks are usually interpreted in terms of attentional bias. Several studies found effects for different groups of addicted subjects; however the processing biases were not always positively related to indices of drug taking behavior, an indication that this relationship is far from clear. Reaction time paradigms were used to investigate processing biases in smokers (Ehrman et al., 2002; Fehr, Wiedenmann, & Herrmann, 2006; Gross, Jarvik, & Rosenblatt, 1993; Hogarth, Mogg, Bradley, Duka, & Dickinson, 2003; Hogarth, Dickinson, & Duka, 2005; Johnson, Thayer, Laberg, & Asbjornsen, 1997; Mogg, Field, & Bradley, 2005; Rusted, Caulfield, King, & Goode, 2000; Waters & Feyerabend, 2000; Waters, Shiffman, Bradley, & Mogg, 2003; Waters et al., 2003; Wertz & Sayette, 2001b; Zack, Belsito, Scher, Eissenberg, & Corrigall, 2001), alcoholics/social drinkers (Bruce & Jones, 2004; Cox, Hogan, Kristian, & Race, 2002;Duka & Townshend, 2004; Field, Mogg, Zetteler, & Bradley, 2004; Pothos & Cox, 2002; Stormark, Field, Hugdahl, & Horowitz, 1997; Townshend & Duka, 2001), cocaine addicts (Franken, Kroon, & Hendriks, 2000), heroin addicts (Franken, Kroon, Wiers, & Jansen, 2000; Lubman, Peters, Mogg, Bradley, & Deakin, 2000; Marissen et al., 2006), and cannabis users (Field, Mogg, & Bradley, 2004). In alcoholics even a memory bias was found (Franken, Rosso, & van Honk, 2003). Recent research has begun to use ERP to investigate processing biases in addicted subjects (see sections 1. and 1.4.), a trend followed in this publication. In comparison with reaction times, ERP offer a more direct opportunity to explore information processing, offering the possibility to examine effects on different stages of information processing and attention more easily. Additionally, in ERP no overt reactions are needed, since it provides an analysis of covert processing irrespective the subject's awareness. ERP offer temporal precision of analysis and distinguish between the rapid perceptual and cognitive processes occurring during stimulus processing (Luck, 2005; Luck, Woodman, & Vogel, 2000).

Although the different theories of cue-reactivity can not account satisfyingly for all data and phenomena and although a plethora of specific factors contribute to variance in cue-reactivity research (see sections 1.2. and 1.3.), the theories described in this section

help to clarify the concept of cue-reactivity and enable a rough comprehension of this phenomenon. Finally, cue-reactivity theories point to the importance of processing biases for the understanding of addiction. Information processing and processing biases for substance-associated stimuli are the core research problems of this dissertation.

1.2. Neglected factors contributing to empirical variance in cue-reactivity research: "Overlap with pharmacological effect" and "social reference"

A problem in cue-reactivity theories is the diversity of results for the relationship between drug-associated stimuli and evoked reactions. In the terminology of classical conditioning, it can be stated that the conditioned reaction (CR) can take a range of different forms depending on several factors. According to Glautier et al. (1995) both the characteristics of the unconditioned stimulus (US) and the conditioned stimulus (CS) are important determinants of the CR. However, if we go beyond the scope whether the reaction to drug-associated stimuli is withdrawal-like (Wikler, 1948), compensatory/drug-opposite (Siegel, 1975), or drug-like (Stewart et al., 1984), if physiological reactions are just activated concomitant to the execution of drug-use schemata (Tiffany, 1990), or what else and if we ask which stimuli are predominantly potent triggers of cue-reactivity and drug-craving, some additional factors have to be taken in account. These additional factors can be described as attributes of the drug-associated stimuli but are mainly semantic in nature. Little research has been conducted to clarify the significance of these factors, but several specific factors could account for the empirical variance in cue-reactivity research and must be regarded in the planning of an experimental investigation.

Mucha, Geier, and Pauli (1999) present evidence for the importance of semantic stimulus characteristics for cue-reactivity effects. Pictures of different phases of smoking were presented to smokers and never-smokers. While pictures of the preparation for smoking and pictures of actual smoking provoked high ratings of craving in smokers, this was not the case with stimuli depicting the end of smoking (disposal of smoking material, cigarette stubs, or ash). However, these different types of stimuli might not only represent different phases of smoking or different "*overlap with the pharmacological effect*" of smoking, which should directly affect the process of classical conditioning, but might be confounded by the negatively appraised "trashiness" in the end of

smoking stimulus category. Regardless of the overlap of pharmacological effects, trashiness, or other interpretations, these results call attention to the effects of stimulus characteristics. To maximize cue-reactivity effects an experiment should use stimuli depicting the actual smoking process or the preparation of smoking material, not the end of smoking. In the study at hand, pictures of the preparation and execution of smoking were used for the smoking related stimulus categories (smoking objects and smoking persons stimuli, see section 2.2.).

Mucha, Geier, and Pauli (1999) used mainly pictures of objects with and without reference to smoking. "*Social reference*", however, might be an important stimulus characteristic unregarded so far. In two recent studies *smoking* cue-reactivity was found in the domain of event-related brain potentials of the electroencephalogram (McDonough et al., 2001; Warren et al., 1999). In both experiments Warren and McDonough used neutral and smoking-associated visual stimuli depicting a person or people collected mainly from magazines and "taking care to avoid well-known public or entertainment personalities" (McDonough et al., 2001, p. 283). They did not, however, systematically explore the effects of social reference. Following Niaura, Abrams, Pedraza, Monti, and Rohsenow (1992), Warren and McDonough (1999, p. 1571) "assumed that social cues may be necessary for smoking cue-reactivity to be observed at its strongest extent." Niaura et al. (1992) had found cardiovascular cue-reactivity to social modeling of smoking but not to physical cues presented in isolation. Since social influence and social factors are important in the development of cigarette addiction and relapse (Niaura et al., 1989) and social factors such as peer socialization and peer affiliation are potent predictors of smoking initiation (Oetting & Donnermeyer, 1998), social smoking-associated stimuli might be more effective in creating cue-reactivity than stimuli without social reference.

To our knowledge no other publications exist on the effects of social cues on smoking cue-reactivity. The goal of the present study was to explicitly explore the modulating effects of social reference on smokers' ERP to photographic stimuli. A paradigm similar to Warren and McDonough was to be used.

1.3. Effects of situational/contextual factors on cue-reactivity and drug craving: Stress and drug availability

Along with semantic stimulus properties, other factors can have influence on cue-reactivity effects and contribute to variance in results. As predicted by the dual affect model (Baker, Morse, & Sherman, 1986, negative-affect craving), stress can increase

drug craving and cue-reactivity (Perkins & Grobe, 1992; Sinha, Fuse, Aubin, & O´Malley, 2000). Perkins and Grobe (1992) confronted smokers who either were permitted to smoke in the experiment or sham-smoke an unlit cigarette with a stressful computerized memory task and a non-stress task. For sham-smokers the desire to smoke was greater in the stress condition than in the non-stress condition, for smoking smokers there was a tendency in the same direction. Sinha, Fuse, Aubin, and O'Malley (2000) found increases in cocaine and alcohol craving in cocaine dependent subjects during stress imagery and drug cues imagery, but not during relaxing imagery. Additional support for the relationship between stress and craving was found for heroin addicts (Hyman, Fox, Hong, Doebrick, & Sinha, 2007), cocaine addicts (Duncan et al., 2007), recently abstinent alcohol-dependent subjects (Fox, Bergquist, Hong, & Sinha, 2007), and smokers (Erblich, Boyarsky, Spring, Niaura, & Bovbjerg, 2003). Beyond laboratory experiments, research has demonstrated that psychosocial stressors can increase smoking and the risk for smoking relapse (Baer & Lichtenstein, 1988; Bliss, Garvey, Heinold, & Hitchcock, 1989; Kassel, Stroud, & Paronis, 2003 give a review; Siahpush & Carlin, 2006).

Along with stress, *perceived drug use opportunity* (*drug availability*) can increase craving, cue-reactivity, and withdrawal symptoms in smokers (Dols, van den Hout, Kindt, & Willems, 2002; Dols, Willems, van den Hout, & Bittoun, 2000; Droungas, Ehrman, Childress, & O´Brien, 1995; Juliano & Brandon, 1998; Sayette et al., 2003; Thewissen, van den Hout, Havermans, & Jansen, 2005; Wertz & Sayette, 2001a; Wertz et al., 2001b). Wertz and Sayette (2001b) demonstrate that attentional bias to smoking cues as explored with a modified Stoop task is influenced by the perceived opportunity to smoke. Wilson, Sayette, Delgado, and Fiez (2005) suggest that the effects of perceived drug use opportunity might even impact cerebral cue-reactivity and correlates of craving indicated by functional neuroimaging. In a prior review of neuroimaging studies of cue-elicited craving, Wilson et al. (2004) had related inconsistent results across studies to a different *treatment status* of the subjects. For the dorsolateral prefrontal cortex (DLPFC) and the orbitofrontal cortex (OFC), significant cue-reactivity effects are predominantly found in studies with actively drug consuming subjects currently not seeking treatment. In subjects currently undergoing a treatment, restricted effects were found only for the DLPFC and OFC. The activity of other craving-sensitive regions like the amygdala or the anterior cingulated cortex (ACC) seems to be less affected by treatment

status. Wilson et al. interpret treatment status in terms of perceived drug use opportunity, a contextual factor influencing the response to drug-associated stimuli.

Because stress and smoking availability influence cue-reactivity it is important to address these factors explicitly in an experimental design. In the study at hand we used a stressor task to maximize cue-reactivity and gave instructions about the smoking opportunity to homogenize the subjects' expectations.

1.4. Cue-reactivity in the domain of visual event-related brain potentials

As mentioned above (section 1.) ERP studies have examined the processing of drug-associated stimuli within a variety of different addicted or substance consuming populations such as smokers (Jang et al., 2007; Littel et al., 2007; McDonough et al., 2001; Warren et al., 1999), alcoholics (Genkina et al., 1986; Herrmann et al., 2000a; Namkoong et al., 2004), social drinkers (Herrmann et al., 2001b), heavy cannabis users (Wölfling et al., 2008), heroin addicts (Franken et al., 2003; Lubman et al., 2008; Lubman et al., 2007), and cocaine addicts (Franken et al., 2004; van de Laar et al., 2004). Three further ERP studies also investigated the processing of drug-associated stimuli but differ from the studies mentioned above due to their divergent methodological approaches: Fehr, Wiedenmann, and Herrmann (2006) evoked ERP during a classical and a modified Stroop task in smokers and non-smokers; Gilbert, Sugai, Zuo, Rabinovich, McClernon, and Froeliger (2007) assessed the effect of distraction by emotionally negative, positive, neutral, and smoking-related pictures on ERP to the immediately following target digits and the modulation of the distraction by nicotine; Parker and Gilbert (2008) used positive, negative, and smoking-associated pictures - each experimental trial persisted in a repeated display of the same picture - to evoke a brain wave pattern known as stimulus preceding negativity (SPN) in smokers and non-smokers. SPN is claimed to represent the anticipation of motivationally relevant events. In smokers significantly greater SPN amplitudes were found for the anticipation of smoking-associated stimuli than for the anticipation of neutral stimuli. Parker and Gilbert interpreted this result in terms of an increased incentive salience of smoking stimuli for smokers. Even if we omit the last three studies, it must be stated that ERP cue-reactivity studies are far from homogenous in methodology and results. These studies have limited comparability because of a variety of factors such as the use of different paradigms

(passive viewing, oddball, conditioning) with different subjects (type of addiction, non-deprived, substituted, deprived, abstinent, never addicted), different types of stimuli (pictures, words, different types of filler stimuli), differences in the inclusion of affective stimulus material like neutral, pleasant, and unpleasant pictures, a wide range of presentation times (12 – 7000 ms), and different parameters in the recording and processing of the EEG (reference, included electrode positions, filtering, different parameterization of ERP components). Some experimenters (Franken et al., 2003; Jang et al., 2007; Littel et al., 2007; Lubman et al., 2008; Wölfling et al., 2008) used a passive viewing task with pictorial stimuli similar to the paradigm from the experiments of Warren and McDonough (1999; McDonough et al., 2001), which are authoritative for the study at hand. In some additional studies (pictorial stimuli: Herrmann et al., 2001b; van de Laar et al., 2004; verbal stimuli: Herrmann et al., 2000a) the passive viewing task was combined with further intermittent tasks (e.g. delayed verbal repeating of the objects on the last stimulus or stimulus ratings) to avoid attention decrements that can occur during long lasting viewing sessions and might result in an abruption of eye contact with the stimuli. Warren and McDonough tried to avoid attentional deficits by telling the subjects that they would have to answer important questions about the stimuli after the session. Franken et al. (2004) combined the passive viewing task with intermittent startle trials beginning 3000 ms after stimulus onset. An oddball task has been used in two ERP cue-reactivity studies so far (Lubman et al., 2007; Namkoong et al., 2004). Namkoong et al. used a blockwise presentation of drug-associated and neutral target stimuli while Lubman et al. used a random presentation. The very early study by Genkina and Shostakovich (1986) employed a conditioning paradigm in alcoholics to associate images of straight lines with the subliminally presented word “vodka” or neutral words and measured ERP to line and word stimuli. Irrespective of the conditioning results, Genkina and Shostakovich found higher P300 amplitudes to the word “vodka” than to neutral words in alcoholics. Table 1 shows the different EPR cue-reactivity experiments, the results for the different ERP components, and important details of the experimental designs. In their review Wilson et al. (2004) reported a contextual modulation of cue-reactivity in neuroimaging studies. Significant cue-reactivity in the DLPFC and OFC was found predominantly in studies with drug users currently not seeking treatment. However, cue-reactivity was found for DLPFC and OFC in only one of nine studies with drug users currently undergoing a treatment. Wilson et al. referred to this treatment status in terms of perceived drug use opportunity (see section 1.3.). As indi-

cated by table 1, however, the treatment status of subjects (see the subjects' column in the table) seems to be less influential for cue-reactivity in the domain of ERP.

ERP cue-reactivity studies provide heterogeneous results about the interrelations between electrophysiological cue-reactivity and self-reports of subjective craving states or craving ratings for stimuli. Only a minority of studies reported significant correlation between ERP cue-reactivity and a self-reported craving state (Franken et al., 2003 for SPW; Littel et al., 2007 for P300; Lubman et al., 2008 for P300). The correlation of N300 amplitudes with craving self-reports demonstrated by van de Laar et al. (2004) did not persist after corrections for multiple statistical testing, however other authors did not perform such corrections. Namkoong et al. (2004) found significant correlations between P300 amplitudes and visual analogue scale craving ratings of the alcohol stimuli. Other studies reported correlations between ERP cue-reactivity and emotional ratings of substance-associated stimuli (Herrmann et al., 2000a for a segment 86-172 ms; Herrmann et al., 2001b for N100; Warren et al., 1999 for P300), but not with craving ratings. None of the aforementioned correlations were detected by the remaining ERP cue-reactivity studies (Franken et al., 2004; Genkina et al., 1986; Jang et al., 2007; Lubman et al., 2007; McDonough et al., 2001; Wölfling et al., 2008).

Table 1: Overview of ERP cue-reactivity experiments

Population type	Reference	Paradigm/task	Subjects	Stimuli (t)	N1	P2	N2/ N300	P3/ P300	SPW	Comments
cigarette smoking	Jang et al. (2007)	passive viewing pictures	n; active smokers / non-smokers	smoking / antismoking / neutral (500ms)	a.n.r.	a.n.r.	a.n.r.	**n.s.**	a.n.r.	similarity of P300 waveforms for smoking and antismoking stimuli
	Littel et al. (2007)	passive viewing pictures	n; active smokers / ex-smokers / non-smokers	smoking / neutral (2000ms)	a.n.r.	a.n.r.	**n.s.**	*	*	no cue-reactivity in ex-smokers
	McDonough et al. (2001)	passive viewing pictures	n; deprived smokers / non-depr. smokers / non-smokers	smoking / neutral (150ms)	a.n.r.	a.n.r.	*	* (limited)	a.n.r.	N300 is modulated by deprivation, P300 is not modulated
	Warren et al. (1999)	passive viewing pictures	n; active smokers / non-smokers	smoking / neutral (150ms)	a.n.r.	a.n.r.	*	*	**n.s.**	
alcohol	Genkina et al. (1986)	associating lines with subthreshold words, counting lines	?; chronic alcoholics / controls	lines (100ms) / alcohol words / neutral words (12ms)	a.n.r.	a.n.r.	a.n.r.	*	a.n.r.	very deviant methodology from other reported studies
	Herrmann et al. (2000)	viewing with later verbal repetition words	t; detoxified alcoholics / controls	alcohol / neutral (500ms)	analysis of ERP segments at Pz: 86-172 (+), 176-305 (*), 309-500 (n.s.) ms					manner of data analysis impedes comparability to other experiments
	Herrmann et al. (2001)	intermittent naming of last object pictures	n; heavy social drinkers / light social drinkers	alcohol / neutral (500ms)	**significant (*)**, deviant nomenclature, see comments	n.s.	*	*	a.n.r.	P300 cue-reactivity significant at Fz only, significant cue-reactivity for N100 (Fz, Cz, Pz) and P100 (Cz), note: deviant nomenclature of N100 and P100
	Namkoong et al. (2004)	oddball pictures (targets) checkerboard (filler)	t; abstinent alcoholics / controls	alcohol / neutral / filler (300ms)	a.n.r.	a.n.r.	a.n.r.	*	a.n.r.	oddball paradigm with blockwise presentation
heroin	Franken et al. (2003)	passive viewing pictures	t; detoxified heroin addicts / controls	heroin / neutral / pleasant / unpleasant (6000ms)	**n.s.**	**n.s.**	a.n.r.	**n.s.**	*	results for pleasant and unpleasant stimuli were excluded from analysis/are not reported
	Lubman et al. (2008)	passive viewing pictures	t; detoxified or substituted heroin addicts / controls	opiate / neutral / affective (500ms)	a.n.r.	a.n.r.	a.n.r.	*	a.n.r.	opiate cue P300 > affective/neutral in addicts, not in controls; no typical P300 enhancement to affective cues in addicts
	Lubman et al. (2007)	oddball pictures (targets and fillers)	t; substituted heroin addicts / controls	opiate / neutral / opiate targets / neutral targets (500ms)	a.n.r.	a.n.r.	a.n.r.	* (+)	a.n.r.	significant P300 cue-reactivity for filler pictures, trend for target pictures
cocaine	Franken et al. (2004)	passive viewing combined with startle (from 3000ms on) pictures	t; high craving / low craving abstinent cocaine addicts	cocaine / neutral / pleasant / unpleasant (7000ms)	a.n.r.	a.n.r.	a.n.r.	**n.s.**	*	significant cue-reactivity (differentiation between groups) in sustained SPW, in SPW significant at contrast level only
	van de Laar et al. (2004)	passive viewing and intermittent ratings pictures	t; abstinent cocaine addicts / controls	cocaine / neutral (4000ms)	a.n.r.	a.n.r.	*	*	*	significant cue-reactivity in extended P300, late SPW) and sustained SPW, but not in early SPW
cannabis	Wölfling et al. (2008)	passive viewing pictures	n; active cannabis addicts / controls	cannabis / alcohol / neutral / pleasant/ unpleasant (6000ms)	a.n.r.	a.n.r.	a.n.r.	a.n.r.	*	

a.n.r.: analysis not reported, n.s. no significant cue-reactivity effect, * significant cue-reactivity effect reported, + trend for cue-reactivity effect; t = current treatment of substance using subjects reported, n = no treatment reported, ? = treatment status not explicit.

1.4.1. Inconsistent results for cue-reactivity in components earlier than N300

Only one study reported significant effects earlier than the N300. Herrmann et al. (2001b) found effects for components they called N100 and P100 with higher positivity for alcohol cues than neutral cues in heavy social drinkers compared to light social drinkers. While the N100 effects were found at all three midline electrode sites (Fz, Cz, Pz), P100 effects were significant at Cz only. Additionally, the same workgroup (Herrmann et al., 2000a) reported a non-significant trend at Pz for a very early segment of the ERP between 86 to 172 ms after stimulus onset. Similar early effects were not reported by any other workgroup. Also, the effects are limited to certain electrode sites and hard to interpret. Even the authors did not make concrete interpretative statements about the specific information processing represented by these early cue-reactivity effects.

1.4.2. Relatively rare evidence for N300 cue-reactivity

A minority of ERP cue-reactivity studies (McDonough et al., 2001; Warren et al., 1999; van de Laar et al., 2004) reported larger amplitudes for neutral stimuli than for drug-associated stimuli for a P300-preceding frontal dominating negative component (N300) in addicted subjects. McDonough et al. (2001) additionally demonstrated that this N300 cue-reactivity is modulated by smoking deprivation as the effect was greater for deprived than for non-deprived smokers.

Warren et al. (1999, p.1581) interpreted the N300 cue-reactivity effect in terms of "an incentive-motivational state deviance detection process which monitors incoming pictorial stimuli and detects those whose content is incongruent with memory attributes activated in tobacco-addicted states": the mismatch between the non-smoking theme of neutral stimuli and smokers' tobacco-need state results in higher amplitudes. Non-smokers do not have a craving state and perceive a lower or no mismatch between their motivational-emotional state and the non-smoking stimulus material.

In their further interpretation McDonough and Warren (2001) pointed out the similarity of their N300 with the one found in a semantic priming task by Barrett and Rugg (1990) concerning latency, topography, and relationship to experimental manipulations. Barrett and Rugg sequentially presented pairs of pictures, while the subjects had to make decisions if the pictures were semantically associated. When there was no seman-

tic interrelation between the pictures, the N300 to the second picture was more negative than when there was a semantic relation. Prior research had found a similar but later component in the semantic priming of *words* (Kutas & Hillyard, 1980; 1984): a centro-parietally dominant negative ERP component called N400 was increased for words that were incongruous with the preceding sentence stem. Later experimental works on the semantic processing of *pictures* found similar N400 components (Barrett, Rugg, & Perrett, 1988; Barrett & Rugg, 1989). Barrett and Rugg (1990) found – along with the N400 - a more frontally distributed N300 and speculated that it might be associated to a semantic processing system different to that activated by words. Further evidence for the image-specificity of the N300 came from a semantic priming experiment with pictures conducted by McPherson and Holcomb (1999). In this experiment subjects made relatedness judgments for objects in pictures that were highly, moderately, or unrelated to a preceding priming picture. N400 and N300 components were found, the latter with a more anterior distribution. Both the N400 and N300 in McPherson's and Holcomb's experiment differentiated between unrelated and related pictures. N300 amplitudes were more negative for unrelated pictures. The authors discussed - in line with Barrett and Rugg (1990) - that the N300 could represent image-specific semantic processing. Furthermore, McPherson and Holcomb described the two-process theory of priming (e.g. Neely, 1977) that holds that priming can result from two mechanisms, of which the first is described as *automatic*, *effortless*, making few demands on processing resources, and very rapid in on- and offset. This first mechanism is conceptualized as spreading activation (Collins & Loftus, 1975) within a semantic network elicited by a preceding verbal stimulus. Similar processes as in the *verbal* system are assumed in *object* recognition (Vanderwart, 1984). A second mechanism of priming is assumed based on *effortful* or *attentional processing* of the relationships between stimuli. If subjects attend to the relationship between a priming stimulus and a target stimulus and use this information consciously in the processing of the target, attentional priming can occur independently of direct links in semantic memory. Attentional priming is thought to be relatively slow and limited in capacity and while it might improve the processing of some related stimuli it could impede the processing of other events by the absorption of processing resources (Holcomb, 1988). Some evidence for these two modes of priming is reported by Neely (1977) and Holcomb (1988). McDonough and Warren (2001) refer to this findings on semantic priming to augment their primary mismatch interpretation of the N300. They hold that internal motivational states can prime stimuli similar to external stimuli

and thus influence the perceptive and attentional processes. The smoker's need state for nicotine and the concomitant smoking-related cognitions might have been a stimulus set that activated smoking-related parts of the semantic-affective network via internally evoked attentional priming. Smoking-related stimuli fit well with the activated parts of the semantic network and evoked smaller N300 amplitudes than neutral stimuli that did not match the activated semantic structures. Based on comparisons with non-smokers' reactions, Warren and McDonough argue that the stimulus difference in smokers' N300 amplitudes were primarily due to an increase of amplitudes to neutral stimuli rather than to a reduction of amplitudes to smoking stimuli. This would mean that the effect of attentional priming does not primarily enhance the processing of smoking-related stimuli but mainly results in an inhibition of the processing of out-of-context neutral stimuli. However, as no explicit priming was used in Warren and McDonough's experiment, the priming interpretation though plausible is challengeable.

More recent research suggests that the N300 might be a component specific to the semantic processing of non-verbal stimuli and that it is sensitive to semantic congruency but probably without linguistic mediation. West and Holcomb (2002; further discussion of this article by Kounios, 2002) presented simple stories in the form of picture sequences to their subjects. The final picture was either incongruous or congruous with the context of the anteceding pictures. In the N300 of the ERPs to the final pictures, larger negativity was found for incongruous pictures than for congruous pictures. The N300 was mainly distributed over central and frontal sites and distinct from a later more widespread centro-frontal N400 that was also sensitive for congruency but might be not specific for the processing of pictorial stimuli, as similar components with a slightly different distribution were found in experiments with verbal stimulus material (e.g. Kutas & Hillyard, 1984; for a review see Kutas & Federmeier, 2000). The N300 found by West and Holcomb has a comparable time course and distribution as the N300 in Warren and McDonough's experiments. West and Holcomb's results concerning the sensitivity to semantic congruency of this component support Warren and McDonough's conclusion that a smoker's increased N300 amplitude to non-smoking stimuli might reflect a conceptual discrepancy of these stimuli to his internal craving state.

1.4.3. Profound evidence for P300 cue-reactivity effects

In the majority of ERP cue-reactivity studies, effects for parietal dominating P300-like components were found with enhanced positivity for drug associated stimuli

(Genkina et al., 1986; Herrmann et al., 2001b; Littel et al., 2007; Lubman et al., 2008; Lubman et al., 2007; Namkoong et al., 2004; van de Laar et al., 2004; Warren et al., 1999). McDonough and Warren (2001) found limited cue-reactivity effects for the P300 only, and Herrmann et al. (2000a) report effects in an ERP segment from 176-305 ms at Pz, but their waveform did differ considerably from other studies and they did not identify the P300 peak exactly. Overall, P300 cue-reactivity effects are the most stable result over different paradigms and drug-use populations and in different workgroups. Anyhow it must be considered that P300 components from different paradigms (e.g. passive viewing, oddball) might represent slightly different, but overlapping processes especially as a different topography of the P300 was found by Warren and McDonough (1999) compared to visual targets in an oddball task (Alexander et al., 1995). However, oddball cue-reactivity experiments (Lubman et al., 2007; Namkoong et al., 2004) found a P300 with increasing amplitudes from frontal to posterior like Warren and McDonough did.

Warren and McDonough (1999) interpreted smokers' increased P300 amplitudes for smoking-associated stimuli in terms of a special motivational and emotional relevance of these stimuli for the subjects. The smokers' incentive-motivational state of tobacco craving might have resulted in an increased salience of smoking-associated stimuli. Increased P300 amplitudes might suggest that for smokers smoking stimuli have a higher motivational relevance than neutral stimuli and attracted more attention. Thus attentional processing resources are predominantly allocated for the perception of smoking stimuli as indicated by the P300. As they were unrelated to craving self-reports, Warren and McDonough speculated that smokers' increased P300 amplitudes might reflect the operation of an automatic processing system conceptualized by Tiffany (1990) that could be activated in response to drug-associated stimuli and consists of drug-use schemata. According to Tiffany, such an automatic processing system could be activated independently from craving processes. Because of the limitation of the P300 effect, however, the latter interpretation was not used by McDonough and Warren in a later experiment (2001).

Early research on the P300 component had been conducted mainly with the auditory oddball paradigm. In this early research primarily cognitive factors such as the "subjective probability of the evoking task-defined stimulus" were found to modulate the amplitude of the P300 (e.g. Donchin, 1981; Donchin & Coles, 1988; Duncan-Johnson & Donchin, 1977; Pritchard, 1981). The effects of affective factors were neglected in these early studies. Additional factors were demonstrated to have influence on the P300 am-

plitude such as the perceived task relevance of a stimulus or its usefulness in the context of the task (e.g. Duncan-Johnson et al., 1977). Based on this research, Donchin proposed that the P300 might be related to a process in which a subjective representation of the current environment is updated: the current model is modified through new sensory information ("*context updating*", Donchin, 1981; Donchin et al., 1988).

Despite a plethora of further research, there is no consensus about the exact meaning of the P300 and the precise cognitive or neural process it reflects. Luck (2005) proposed two reasons for this. First, the P300 can be found in nearly every experimental condition and every experiment; its ubiquity impedes the isolation of the exact underlying processes. Second, different cognitive processes and components might overlap in the time window of the P300 and are hard to isolate. Apparently the P300 represents a complex system with various elements (Johnson, Jr., 1986) that impede the attribution of single processes to the P300.

In addition to solely cognitive factors, motivational factors also influence the P300 amplitude, as e.g. indicated by ERP cue-reactivity research. Previously the influence of motivational factors on the ERP waveform/P300 had been demonstrated in non-addict populations. After early hints that the P300 reflects the subjective motivational properties of stimuli (Begleiter, Porjesz, Chou, & Aunon, 1983), Baldeweg, Ullsperger, Pietrowsky, Fehm, and Born (1993) reported that hunger and satiety modulate P300 amplitudes to verbal stimuli describing possible states of hunger, thirst, and tiredness. Johnston and Wang (1991) found that the P300 amplitudes of female subjects to photos of babies and male models in relation to normal people were highest when progesterone levels were high. Thus biological and motivational states like hunger or menstrual phase have proven to modulate reactions to motivationally relevant stimulus material. In line with these motivational modulations, Miltner et al. (2005) and Schienle, Schäfer, and Naumann (2008) demonstrated increased P300 amplitudes and slow positive wave to fear-relevant pictures in snake/spider phobics and interpreted this in terms of motivated attention in picture processing. Beside biologically and psychopathologically induced motivational states, induced emotional states may also influence the information processing represented by ERP/P300: Kliegel, Horn, and Zimmer (2003) found reduced P300 amplitudes on acoustic stimuli after a negative mood induction and interpreted this in terms of a reduced allocation of cognitive processing resources due to a preoccupation with the negative mood state that might get processing priority. Meinhardt and Pekrun (2003) reported similar emotional after-effects on the P300 for acoustic stimuli

after an induction of negative mood, but also in a positive mood condition compared to a neutral mood condition. In their review Polich and Kok (1995) documented in a very broad sense the sensitivity of P300 amplitudes to fluctuations in the arousal state of the subjects and tried to relate different natural (circadian, ultradian, seasonal, menstrual) and environmentally (exercise, fatigue, drugs) induced states to arousal.

Additionally, general emotional properties of the stimulus material itself have proven to affect the ERP waveform. While Polich and Kok (1995) mainly focused on effects of induced arousal states, other research proved the effects of arousing stimuli on P300 amplitudes. In a review of ERP findings on affective picture processing, Olofsson, Nordin, Sequeira, and Polich (2008) stated that arousal effects of stimuli were consistently obtained and generally occurred at longer latencies (including the P300) while valence effects were found only inconsistently in varying components. However, Conroy and Polich (2007) found that valence affected frontal P300 amplitudes independently of arousal. Earlier Begleiter, Gross, and Kissin (1967) had found that the conditioned affective valence of visual stimuli modulated the ERP-waveform. Waveforms of all three conditions (positive, negative, neutral) differed, but the methodology makes this experiment hard to compare with later studies. Cuthbert et al. (2000) also found valence effects of the stimulus material. They evoked ERP by the presentation of pleasant, neutral, and unpleasant pictures; additionally peripheral measures were conducted to access autonomic arousal. In the ERP waveform a differentiation between the stimulus categories began between 200 and 300 ms after stimulus-onset with more positivity for pleasant stimuli than for neutral stimuli. Between 300 and 400 ms (time range of P300) pleasant stimuli still evoked the highest positivity, significantly more than neutral and unpleasant stimuli. Pleasant pictures still evoked more positivity than neutral pictures in a widespread component from 400 to 700 ms, but here additionally unpleasant pictures produced more positivity than neutral pictures. Between 700 and 1000 ms there was no more difference between pleasant and unpleasant pictures, but both categories still evoked more pronounced positivity than neutral pictures. This latter difference persisted for the remaining five seconds of the stimulus presentation. The positivity between 700 and 1000 ms was higher for stimuli that gained high ratings for arousal and evoked pronounced physiologic activation.

Evidence for *valence* effects remains inconsistent but a majority of experiments on the processing of emotional stimuli suggest that P300 and other, mainly later components are sensitive to the emotional *arousal* value of pictorial stimuli (e.g. Cuthbert,

Schupp, Bradley, Birbaumer, & Lang, 2000; Delplanque, Silvert, Hot, & Sequeira, 2005; Gierych, Milner, & Michalski, 2005; Johnston, Miller, & Burleson, 1986; Keil et al., 2002; Mini, Palomba, Angrilli, & Bravi, 1996; Olofsson & Polich, 2007; Palomba, Angrilli, & Mini, 1997; Schupp et al., 2000; Schupp et al., 2007; see also section 1.4.4.). Highly arousing stimuli evoked a more pronounced positivity than neutral stimuli in a variety of components. Mini et al. (1996) and Johnston et al. (1986) reported higher P3(00) and P4 amplitudes for pleasant and unpleasant stimuli than for neutral stimuli. Palomba et al. (1997) found higher positivity for pleasant and unpleasant stimuli compared to stimuli in components labeled N2 and P3 and in later components. Schupp et al. (2000) demonstrated that a late positive potential (350-750 ms) was higher for pleasant and unpleasant stimuli than for neutral stimuli. Within the affective stimulus categories, pictures associated to high arousal evoked more positivity than less arousing pictures.

However, in some studies the effects of arousal were opposed to the frequently found increased positivity of the P300 and other ERP components. Experiments implementing diverse paradigms found an association between highly arousing stimulus material and a decline in positivity in ERP waveforms. Schupp, Cuthbert, Bradley, Birbaumer, and Lang (1997) demonstrated reduced positivity in the probe P300 of an acoustic startle experiment during the presentation of highly arousing pleasant and unpleasant pictures in relation to neutral pictures. During a high-speed presentation of emotional pictures (Junghöfer, Bradley, Elbert, & Lang, 2001) an arousal effect could be evoked. In an experimental condition with a presentation rate of 3 Hz, the differentiation of stimuli was observable from approximately 150 ms after stimulus onset on. In two components called P200 and N260, highly arousing stimuli produced less positivity than less arousing stimuli. In an earlier P100 no significant differences were found. With a higher presentation rate of 5 Hz, a similar differentiation with more negativity for arousing stimuli was found for a N60 only. The N60 was interpreted as a continuing processing of the preceding picture because of the very fast presentation. A later study with high-speed presentations replicated the increased posterior negativity for emotional stimuli compared to neutral stimuli (Flaisch, Junghöfer, Bradley, Schupp, & Lang, 2008). Because of their differing paradigms (acoustic startle, high-speed presentations) the latter three studies are not comparable to the foregoing ERP studies on affective picture processing. However, it can be stated that most research indicates that along with cognitive factors affective and motivational factors can increase the amplitude of P300

(Baldeweg, Ullsperger, Pietrowsky, Fehm, & Born, 1993; Begleiter et al., 1983; Cuthbert et al., 2000; Johnston et al., 1986; Johnston & Wang, 1991; Mini et al., 1996; Palomba et al., 1997; Polich & Kok, 1995) and influence other components (Begleiter, Gross, & Kissin, 1967; Cuthbert et al., 2000; Johnston et al., 1986; Palomba et al., 1997; Schupp, Mucha, & Pauli, 1996; Schupp et al., 2000).

These various cognitive, emotional, and motivational determinants of P300 are congruent with Johnson's (1986) triarchic model. In his model Johnson claimed that every variable that has influence on the P300 amplitude can be assigned to one of three dimensions. Johnson described these three dimensions as *subjective probability*, *stimulus meaning*, and *information transmission*. Johnson claimed that subjective probability and stimulus meaning might contribute additively and independently to the P300 amplitude, but the influence of these factors is only possible if there is sufficient information transmission that depends on attentional processes. Emotional and motivational factors can influence stimulus meaning and attention. Warren and McDonough applied this model to their P300 and supposed emotional and motivational processes as factors underlying the P300 cue-reactivity.

1.4.4. Evidence for SPW cue-reactivity effects

Some studies using extended stimulus presentation times (2000 ms minimum in Littel et al., 2007; 7000 ms maximum in Franken et al., 2004) found increased positivity for drug-associated stimuli in the domain of slow positive waves (SPW) in users of nicotine, cannabis, heroin, and cocaine. All of these SPW cue-reactivity effects were reported by authors of the workgroup around Franken (Franken et al., 2004; Franken et al., 2003; Littel et al., 2007; van de Laar et al., 2004) except the study by Wölfling et al. (2008). Other authors failed to find significant effects for the SPW (Warren et al., 1999) or did not report them.

In their interpretations of these SPW cue-reactivity effects, all the aforementioned authors hypothesized that augmented amplitudes in the domain of SPW reflect increased processing and processing/attentional bias for substance-associated stimuli in addicted populations. This enhanced processing could sustain for periods of up to six seconds (Franken et al., 2003; Hajcak & Olvet, 2008) or even longer. From ERP research in the emotional picture paradigm (see also section 1.4.3. about P3 for further results), it is well established that SPW is increased for highly arousing pictures compared to neutral pictures (Amrhein, Mühlberger, Pauli, & Wiedemann, 2004; Bradley,

Hamby, Low, & Lang, 2007; Codispoti, Ferrari, & Bradley, 2007b; Codispoti, Ferrari, & Bradley, 2006; Cuthbert et al., 2000; de Cesarei & Codispoti, 2006; Hajcak, Dunning, & Foti, 2007; Keil et al., 2002; Olofsson, Nordin, Sequeira, & Polich, 2008; Olofsson et al., 2007; Palomba et al., 1997; Pastor et al., 2008; Schupp et al., 2000; Schupp, Junghöfer, Weike, & Hamm, 2003; Schupp, Junghöfer, Weike, & Hamm, 2004). Cuthbert et al. (2000) suggested that increased SPW indicates a selective attentive processing of emotional stimuli, reflecting the activation of a cerebral motivational system. Schupp et al. (2000) also found evidence for the modulation of SPW with arousal and stated that SPW is modulated by the intrinsic motivational significance of stimuli. SPW might represent processes similar to the P300 component (Kok, 1997; Littel et al., 2007) but might also be more closely related to emotional processing and index increased stimulus relevance at higher-order stages of information processing (Schupp et al., 2003). SPW captures sustained attentional processes later than P300. Similar to P300 effects, increased SPW might reflect enhanced allocation of attention and effort during a variety of tasks (Codispoti et al., 2007b; Kok, 1997). Motivational relevant stimuli like emotional pictures attract attention and lead to increased SPW. In line with this view, augmented SPW to motivationally relevant stimuli was found in spider/snake phobics (Miltner et al., 2005; Schienle, Schäfer, & Naumann, 2008). It has been suggested that increased SPW reflects increased allocation of attentional resources to motivational relevant stimuli, a condition described as "*motivated attention*" (Schupp et al., 2000; Schupp et al., 2004). A processing bias for substance-related stimuli is consistent with the incentive-sensitization theory of Robinson and Berridge (1993; Berridge et al., 1995; see section 1.1.) who claim that drug stimuli acquire attention-grabbing properties – incentive salience - during the process of sensitization. Similar theoretical propositions about an attentional bias concerning drug-related cues were made by Franken (2003).

The modulation of SPW by the motivational significance of emotional stimuli seems to be independent from the difficulty of a concurrent task (mathematics), indicating the high priority and automaticity of this emotional processing (Hajcak et al., 2007). However, emotional processing as indicated by the SPW might be susceptible to intentional emotion modulation/top-down processing. Moser, Hajcak, Bukay, and Simons (2006) were able to show voluntary suppression of emotional responses to unpleasant stimuli.

Dolcos and Cabeza (2002) found higher positivity in a SPW associated time-domain for subsequently remembered pictures than for subsequently forgotten pictures. Pleasant

and unpleasant pictures evoked higher SPW and were remembered more often than neutral pictures (Palomba et al., 1997). Additional research also found links between memory and SPW (Deveney & Deldin, 2004) suggesting that SPW might be involved in memory formation. Ruchkin, Johnson, Jr., Canoune, and Ritter (1990) demonstrated that the amplitude of SPW was directly related to information load in a memory task and provided evidence that increased positivity of SPW was related to storing items into memory.

Stimulus composition features like complexity seem to be relatively unimportant for the modulation of the SPW; SPW seems to be primarily affected by motivational relevance (Bradley et al., 2007). However, picture size has an influence on SPW: larger pictures evoke higher positivity, but this effect seems not interact with the affective modulation of the SPW (de Cesarei et al., 2006) although picture size might modulate emotional reactions measured by skin conductance (Codispoti & de Cesarei, 2007a).

Until now source localizations of emotional SPW modulations did not give consistent results. Keil et al. (2002) used a passive viewing task for emotional pictures and found the usual modulation with higher P300 and SPW amplitudes for positive and negative pictures compared with neutral pictures. In a source space projection with a minimum norm procedure based on an average reference, sources for SPW modulation were located in occipital and posterior parietal cortex with a right-hemispheric dominance. Carretié, Hinojasa, Albert, and Mercado (2006) presented affective pictures during a non-affective discrimination task. For negative pictures they found an increase in the positivity of a late component (680 ms) of the ERP and located this effect in the left precentral gyrus by means of LORETA (low resolution tomography algorithm) source localization. These results lead Carretié et al. to speculate about a possible "motor-related bias" during the reaction to unpleasant stimuli. Sabatinelli, Lang, Keil, and Bradley (2007) assessed both ERP of the EEG and functional hemodynamic measures (fMRI) during the presentation of emotional pictures in separate sessions. SPW and blood oxygenation level-dependent (BOLD) responses were both modulated by pictorial arousal. Significant correlations were found between SPW amplitudes and BOLD intensity in lateral occipital, inferotemporal, and parietal visual areas across picture contents. However, regional estimated sources did not correlate significantly with regional BOLD reactions. The authors suggested that enhanced SPW during emotional picture

processing might represent activity in a system of visual cortical structures that is induced by the motivational relevance of stimuli.

1.5. Present study: Research question and hypotheses

The purposes of this study were to replicate the findings of Warren and McDonough (1999) for cue-reactivity in the N300 and P300 components of the ERP and to examine the effects of social or objective stimulus content on these effects. Additionally, SPW cue-reactivity effects were to be explored, since larger SPW is expected in smokers after smoking stimuli than after neutral stimuli, reflecting the higher motivational relevance of smoking stimuli (Littel et al., 2007).

Based on Niaura et al. (1992), Warren and McDonough (1999) had assumed that social stimuli might be necessary to evoke strong cue-reactivity but did not test this hypothesis. Warren and McDonough (1999; McDonough et al., 2001) used pictures from magazines as substance cues, leading to confoundations like recognition or familiarity effects. Following Niaura et al. (1992), it is hypothesized in the present study that social reference of stimuli would lead to higher cue-reactivity rather than distracting from smoking-association. Social factors are important for smoking initiation (Oetting et al., 1998) and could therefore be more effective in creating cue-reactivity than stimuli without social reference. To avoid confounders like recognition or stimulus familiarity and to allow stimuli sets to be as homogenous as possible, social stimuli with and without smoking-association were created specifically for this study and submitted to a pretest. The experimental stimuli were selected to maximize the difference in craving evoked by smoking and non-smoking stimuli but to minimize the difference in evoked arousal and valence as much as possible. Experimental stimuli were not taken from magazines. A confoundation of social and objective stimuli (like in Littel et al., 2007; McDonough et al., 2001; and Warren et al., 1999) was avoided.

Following Warren and McDonough, we used a stressor task (McCubbin, Cheung, Montgomery, Bulbulian, & Wilson, 1992) to evoke negative affect and increase smokers reactivity to smoking-associated stimuli (Baker et al., 1986). Additionally, bogus information was given to smokers to create and standardize perceived availability of cigarettes. To avoid uncontrolled oddball effects, the probability of smoking and non-smoking stimuli was kept equal and subjects were informed that an equal amount of both stimuli categories would be presented in the experiment. Between the experimental

blocks, a recognition task was applied to ensure sustained attention for the experimental stimuli during the EEG acquisition.

The present study tested the following hypotheses:

1) Based on the work of Warren and McDonough (1999), it is expected that cue-reactivity will be found for the N300 and P300 components of the visual ERPs to smoking and non-smoking stimuli. The N300 in smokers are expected to have more negative amplitudes for non-smoking stimuli than for smoking stimuli. In the P300 components smokers should produce more positivity after smoking stimuli than after non-smoking stimuli. Additionally, based on recent research (Franken et al., 2004; Franken et al., 2003; Littel et al., 2007; van de Laar et al., 2004; Wölfling et al., 2008) cue-reactivity is expected for the SPW domain of the ERP with smokers presenting more positivity after smoking stimuli than after non-smoking stimuli.
2) In his theoretical approach Franken (2003) assumes that attentional bias and feelings of craving reciprocally enhance each other. As ERP cue-reactivity is also seen as an indicator of attentional bias, it is expected to show correlations with self-report measures of pre-experimental state craving. Unfortunately, preliminary findings are inconsistent in showing this correlation, although some studies have demonstrated this phenomenon (Franken et al., 2003; Littel et al., 2007; Lubman et al., 2008).
3) Since ERP cue-reactivity is seen as a phenomenon of addiction and believed to require an "addicted learning history", it is possible that cue-reactivity in the domain of N300, P300, and SPW components is interrelated with other indices of addiction such as the sum score of the Fagerström test for nicotine dependence, reported number of cigarettes smoked per day, or elements of the smoking history like years smoked so far.
4) Previous research has found correlations between ERP cue-reactivity and ratings for the substance-associated experimental stimuli, connections we anticipate demonstrating. Namkoong et al. (2004) reported an interrelation with craving ratings while Herrmann et al. (2000), Herrmann et al. (2001), and Warren et al. (1999) found interrelations with emotional ratings.
5) Cue-reactivity is expected for post-experimental stimulus ratings. Smokers should give higher craving ratings to smoking stimuli than to non-smoking stimuli while non-smokers should not report any evoked craving at all. Smokers should perceive smoking stimuli more positively than non-smoking stimuli while non-smokers

should be more negative in their evaluation of smoking stimuli (Mucha et al., 1999; Warren et al., 1999).

6) Following Niaura et al. (1992) we hypothesized that the social reference in stimuli leads to higher cue-reactivity, instead of distracting from smoking-association. For stimuli showing persons, higher cue-reactivity is expected than for stimuli depicting objects only. Social reference should modulate cue-reactivity effects. This should be true for a) ERP cue-reactivity in the different components N300, P300, and SPW (see hypothesis 1) and for b) cue-reactivity in post-experimental stimulus ratings (see hypothesis 5).

2. Materials and methods

2.1. Subjects

This study was approved by the ethical committee of the medical faculty of the University of Münster before implementation.

24 smokers (14 female) and 19 non-smokers (13 female) were recruited through announcements in a local weekly journal and posters placed at communal locations and institutes of the University of Münster. Subjects were screened by telephone for study eligibility: smokers were suitable if they smoked at least 15 cigarettes per day for the last two years (22 subjects) or if they reached a score of five or higher (17 subjects) in the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991). No smoker was currently trying to quit smoking. Only subjects who reported to have smoked less than 25 cigarettes in their lives were assigned to the non-smoker group.

Persons reporting prior or actual mental/neurological health conditions or the use of psychotropic medications were excluded. All subjects had normal or corrected to normal vision and hearing and were right-handed according to the Edinburgh Inventory (EHI; Oldfield, 1971). Written consent was obtained from all subjects. Subjects were paid for participation.

The resulting samples were approximately balanced for education, gender, and age. Groups did not differ significantly in age (smokers: M = 24.04 years, SD = 3.46; non-smokers M = 23.11 years, SD = 3.65; ($t_{(41)}$ = 0.86, p = .394) or gender distribution ($\chi^2_{(1, N = 43)}$ = 0.46, p = .497). 17 non-smokers and 19 smokers were students. Smokers had been smoking regularly for M = 7.58 years (SD = 3.17), had attempted to quit a mean number of M = 1.58 times (SD = 0.65), smoked M = 18.58 cigarettes per day (SD = 3.72), and had an average FTND-score of M = 5.33 (SD = 1.27). Mean alveolar carbon monoxide-level of smokers was 14.08 ppm (SD = 7.32; non-smokers: M = 1.58 ppm, SD = 0.84). Table 2 summarizes the demographic subject characteristics, and table 3 gives an overview of smokers' addiction-related characteristics.

Table 2: Demographic characteristics for smokers, non-smokers, and total sample

	Smokers (n = 24)	Non-smokers (n = 19)	Total sample (N = 43)
Gender (n)			
Female : male	14 : 10	13 : 6	27 : 16
Age			
Mean in years (SD)	24.04 (3.46)	23.11 (3.65)	23.63 (3.53)
Nationality (n)			
German	22	18	40
Other	2	1	3
Marital status (n)			
Unmarried	22	19	41
Married, living together	1	0	1
Married, living apart	1	0	1
Education (highest degree) (n)			
Elementary/secondary school	1	0	1
Grammar school	3	0	3
University entrance qualification	19	15	34
University degree	1	4	5
Occupation (n)			
Clerk/civil servant	2	1	3
Worker/skilled worker	2	0	2
Pupil	1	1	2
Student	19	17	36 (83.7%)
Mean alveolar CO in ppm (SD)	14.08 (7.32)	1.58 (0.84)	8.56 (8.32)

CO: Carbon monoxide; ppm: parts per million.

Table 3: Characteristics of the smokers (n = 24)

	M	Range
Age of onset smoking (years) (*SD*)	15.88 (1.83)	13-19
Duration of regular smoking (years) (*SD*)	7.58 (3.17)	2-16
Cigarettes/day (*SD*)	18.58 (3.72)	11-25
Attempts to quit smoking (*SD*)	1.58 (0.65)	1-3
FTND sum-score (*SD*)	5.33 (1.27)	3-8

FTND = Fagerström Test for Nicotine Dependence.

2.2. Experimental stimuli

The stimuli for the elicitation of ERPs consisted of 72 different color pictures divided into four categories of 18 stimuli:

1) S*moking objects (SO):* stimuli depicting objects associated with the smoking process (smoking-relevant materials, cigarettes, burning cigarettes not yet finished). These stimuli were taken from a set arranged by Mucha et al. (1999).

2) *Non-smoking objects (NO):* stimuli depicting objects from everyday life without any reference to smoking. These were chosen from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999; pictures 6150, 7000, 7002, 7006, 7009, 7010, 7025, 7030, 7034, 7040, 7050, 7060, 7080, 7090, 7150, 7170, 7190, 7233). Most of these pictures had been used previously as control stimuli in studies of cue-reactivity (Geier, Mucha, & Pauli, 2000; Mucha et al., 1999). For this set, neutral pictures with medium valence and medium arousal value were selected according to the criterion reported by Mucha et al. (1999).

The SO and NO stimuli did not contain faces or persons. Stimuli of the other two categories displayed portraits of single persons with either smoking or non-smoking attributes:

3) S*moking persons (SP):* portraits of persons (nine female) smoking (inhaling, exhaling, holding a burning cigarette) or lighting a cigarette.

4) *Non-smoking persons (NP):* portraits of persons (nine female) without any reference to smoking. In four of these pictures, the portrayed person held either a glass of water or a pencil.

The SP and NP pictures were shot frontally or half-frontally portraying the upper part of the body from the middle of the upper arms upward, including the head. A dark blue cloth served as background. No person was depicted twice.

Stimuli categories with and without reference to smoking were held equiprobable to avoid confounding oddball effects.

All stimuli appeared on a color video monitor (CTX – 1785XE, 17") located at approximately eye level about 1.5 m in front of each subject's head. The size of the stimuli was 18 cm x 25 cm wide (visual angle: vertical 6.9°, horizontal 9.5°, constant for all stimuli as size effects on emotional reactions have been reported [Codispoti et al., 2007a]).

2.2.1. Construction, evaluation, and selection of the SP and NP stimuli: Pretest

To obtain the final sets of persons stimuli described above in section 2.2., 41 persons (age range 20-48 years) were photographed multiple times both smoking and not-smoking. A digital camera (Canon Powershot G2, resolution 1600 x 1200 pixels) was used. From the resulting 712 pictures, 100 pictures were selected for evaluation in a pretest and divided in two subsets of 50 pictures with about equal numbers of smoking and non-smoking persons (set 1: 24 pictures of smoking persons, 26 pictures of non-smoking persons; set 2: 28 pictures of smoking persons, 22 pictures of non-smoking persons). In each subset a person appeared either as a smoker or as a non-smoker. In both subsets the sequence of SP and NP pictures varied pseudo-randomly with no more than four pictures of the same category in succession. To reduce sequence effects, each subset was displayed in a forward and a backward sequence on different internet pages. 92 subjects rated the pictures in one of the sets for valence and for arousal by means of a computer-based version of the Self-Assessment Manikin (SAM; Bradley & Lang, 1994; Lang et al., 1999; nine-point rating scales). Additionally, craving was assessed, on a nine-point rating scale ("When I look at this picture, I sense … no – a medium – a very strong urge to smoke"). For an exact account of the subject distribution to the subsets see table A-1 in the appendix; tables A-2 to A-7 in the appendix report smokers' and non-smokers' pretest-ratings (valence, arousal, craving) for each picture. The subjects had been recruited by word-of-mouth advertising and by email announcements. Five book-coupons of 15 € each were raffled among subjects as an incentive for participation. Former smokers (n = 12) were excluded from the analysis, which left 32 smokers.

Because of a preponderance of non-smokers, 32 non-smokers were selected randomly to avoid disproportionate groups. However, due to data loss, the final sample included in the analysis consisted of 32 non-smokers (19 female) and 28 smokers (18 female). Smokers (M = 27.11 years, SD = 6.76) and non-smokers (M = 26.13 years, SD = 3.42) in the sample were comparable in age. Smokers had an average FTND sum-score of M = 2.79 (SD = 2.25) with a range of 0-6.

For the ERP experiment, the 18 SP and the 18 NP stimuli were selected based on the ratings given by the subjects in the final pretest-sample. Three criteria were applied for the selection of the stimuli. First, the SP stimuli were selected based on high average craving ratings from the smokers. For the NP stimuli we preferred pictures with low craving ratings from the smokers. Second, the SP and NP stimuli should be comparable in smokers and non-smokers in terms of the average ratings of valence and arousal. Third, SP and NP stimuli should be comparable regarding average ratings of valence and arousal. The second and the third criterion were applied to minimize confounding differences in valence and arousal as these are known to affect ERP-waveforms (e.g. Amrhein et al., 2004; Cuthbert et al., 2000). The goal was to investigate purely the effect of smoking-association.

However, as smoking-related stimuli will be evaluated differently by smokers and non-smokers (Mucha et al., 1999; Warren et al., 1999), these matching requirements could only be partly fulfilled with regard to the valence ratings. See table 4 for an overview of the average responses of smokers and non-smokers in the pretest-sample concerning the various ratings.

Complying with the requirements of the first selection criterion, the resulting SP stimuli provoked significantly higher ratings for craving in smokers than NP stimuli ($t_{(27)}$ = 5.91, $p < .000$, dependent samples) (smokers: SP: M = 3.30, SD = 1.86; NP: M = 1.87, SD = 0.83). Adherence to the selection criteria was also checked using repeated-measures analyses of variance (ANOVA) with “group” (smokers, non-smokers) as a between-subjects factor and “smoking content” of stimuli (smoking, non-smoking) as a within-subjects factor. ANOVAs were computed separately for ratings of valence and arousal. Ratings for craving were not analyzed further because of lacking variance in non-smokers (non-smokers: SP: M = 1.06, SD = 0.21; NP: M = 1.00, SD = 0.00). The analysis for arousal yielded significantly higher arousal ratings for SP (M = 3.87, SE =

0.23) than for NP stimuli (M = 3.33, SE = 0.21) ($F_{(1, 58)}$ = 15.79, p < .000) but no significant effect for "group" ($F_{(1, 58)}$ = 1.07, p = .306) and no significant interaction of both factors ($F_{(1, 58)}$ = 1.16, p = .285). With regard to valence there was a significant main effect for "group" ($F_{(1, 58)}$ = 15.16, p < .000) with lower average valence ratings in non-smokers (M = 4.47, SE = 0.12) than in smokers (M = 5.18, SE = 0.13). This was accompanied by a significant hybrid interaction (Leigh & Kinnear, 1980) of "group" x "smoking content" ($F_{(1, 58)}$ = 12.24, p = .001) with SP stimuli being more pleasant than NP stimuli in smokers and reverse in non-smokers (smokers: SP: M = 5.35, SD = 1.26; NP: M = 4.90, SD = 0.94; non-smokers: SP: M = 4.07, SD = 1.27; NP: M = 4.88, SD = 0.61). No main effect for "smoking content" of stimuli was found for valence ($F_{(1, 58)}$ = 1.88, p = .176). The detected differences for stimuli - especially for valence - can be seen as a phenomenon of cue-reactivity itself but must be taken into account in the interpretation of effects in ERP.

Table 4: Average pretest-ratings of smokers and non-smokers for the persons stimuli selected for the ERP experiment

Rating scale	Stimuli	Group in pretest	
		smokers	non-smokers
craving	smoking persons	3.30	1.06
		(1.86)	(0.21)
M (*SD*)	non-smoking persons	1.87	1.00
		(0.83)	(0.00)
valence	smoking persons	5.35	4.07
		(1.26)	(1.27)
M (*SD*)	non-smoking persons	4.90	4.88
		(0.94)	(0.61)
arousal	smoking persons	3.59	4.16
		(1.50)	(1.96)
M (*SD*)	non-smoking persons	3.19	3.47
		(1.44)	(1.73)

All nine-point rating scales; craving: higher values indicate higher craving; valence: values higher than five represent pleasant ratings, values lower than five represent unpleasant ratings; arousal: higher values indicate more arousal.

2.3. Self-report measures

Sociodemographic data were requested by six short questions (age, marital status, education, occupation, nationality; see "Soziographischer Fragebogen" in appendix B).

Ratings of craving

Subjective craving for cigarettes was assessed repeatedly using two items (see "Fragebogen zur subjektiven Einschätzung", in appendix B): "Would you like to smoke a cigarette now?" ranging from 0 ("no, not at all") to 10 ("yes, very willingly") (*craving item a*) and "To smoke a cigarette now would be …" ranging from -5 ("very unpleasant") via 0 ("neither unpleasant nor pleasant") to 5 ("very pleasant") (*craving item b*). Both items were realized as visual analog scales (VAS) consisting of 10-cm lines with grades every cm and at the endpoints. These grades were added to the VAS to prevent artificial variance due to imprecise perception of the middle and the ratios of the scales. Subjects were asked to make a mark on the line that represented their level of perceived actual craving.

Assessment of dexterity

The Edinburgh Inventory (EHI; Oldfield, 1971) was used to confirm dexterity. This 10-item-questionnaire enables a quantitative assessment of handedness and is simple to employ. From the results in this questionnaire a laterality quotient can be computed. The EHI had been translated into German for use in this study (see EHI in appendix B).

Tobacco dependence

The Fagerström Test for Nicotine Dependence (FTND; Heatherton et al., 1991; German version by Batra & Fagerström, 1997) was used to assess the subject's level of nicotine dependence. According to Batra and Fagerström (1997) the average score of smokers in the FTND is about 3; scores greater than 4 indicate the likely presence of nicotine dependence (maximum score 10). The FTND was accompanied by nine questions assessing the past smoking history (e.g. age at beginning of smoking, years of regular smoking, attempts to quit, see "Rauchanamnese" in appendix B). Six of these questions had been reported and validated by Schupp, Batra and Buchkremer (1997).

Assessment of mood

Mood was assessed several times during the testing procedure using the short form B of a mood questionnaire (Multidimensionaler Befindlichkeitsfragebogen; MDBF; Steyer, Schwenkmezger, Notz, & Eid, 1997). This instrument covers the dimensions good-bad mood, alertness-fatigue and calmness-agitation with four items each (with five-point scales) and possesses acceptable reliability. Two items of each dimension have reversed polarity. The minimum/maximum sum score of each dimension is -8/8. Higher values indicate better mood/higher alertness/more calmness.

Post-experimental stimulus ratings

After EEG recording subjects rated all experimental stimuli for arousal, valence, and craving. Ratings for arousal and valence were assessed with a computer-based version of the Self-Assessment Manikin (SAM; Bradley et al., 1994; Lang et al., 1999), ratings for craving were made on a computer-based nine-point rating scale ("When I look at this picture, I sense … no – medium – very strong urge to smoke").

2.4. Testing procedure

During the prior screening for study eligibility, subjects had been asked not to consume any alcohol the evening before the experiment, to sleep sufficiently, and to stop smoking one hour before the experiment (to cause mild nicotine-deprivation). Subjects were informed that abstinence would be confirmed by alveolar CO measurement. In the laboratory, subjects were first informed about the details of the study and completed consent forms. Alveolar carbon monoxide (CO) was gauged in all subjects using the EC50 MICRO III Smokerlizer (Neomed Medizintechnik, Bedfont Scientific Ltd, Köln, Kent). To make smokers believe that they could smoke a cigarette in the course of the experiment (availability), they were told that there was a 50% chance that they could smoke during the experiment, depending on a random generator. Subsequently, all subjects rated subjective craving for cigarettes using the craving items a and b (see section 2.3.). Subjects were asked if they had refrained from smoking during one hour prior the experiment and observed the other requirements imposed on the telephone. The subjects completed the pre-experiment questionnaires (Edinburgh Inventory, sociodemography, and additionally for smokers: Fagerström Test for Nicotine Dependence and questions about their past smoking history). Then an electrode cap was fitted to the subjects, elec-

trodes were attached, and subjects took a seat in a relaxation chair. The EEG recording was anteceded by a 10-minute distressing computer-controlled arithmetic task designed by McCubbin et al. (1992) that adjusted task difficulty based on the performance. In this task subjects had to summate two numbers as quickly and accurately as possible and type the result into the computer. Immediate acoustic feedback on the correctness of the answer was given. To verify the impact of the stressor task, it was preceded and succeeded by ratings for craving (items a and b) and the mood questionnaire MDBF. Following the stressor, smokers were told that after the recording of EEG they would have the chance to smoke depending on the result of the random generator. The smokers were asked to hand over their private cigarettes to the investigator so he could bring and light the cigarettes to enable smoking while seated and wired in the flue-equipped experimental chamber. After the EEG recording the "random generator" gave persistent negative results for smoking to prevent acute substance effects during the later rating-section. Due to the experimental preparations subjects had been deprived for about 2.5 h totally at the beginning of the EEG acquisition. Subjects were instructed that stimuli would appear in random order and that half of the stimuli would be smoking-related and half would not. Subjects were told to attend to all stimuli equally and saw three test stimuli for familiarization (pictures 1670, 2210, 7595 from the IAPS; Lang et al., 1999). They were informed that stimuli would appear repeatedly in four blocks and after each block they would see three stimuli and would have to decide for each stimulus whether it had been shown before (recognition task). For each correct recognition subjects won 0.5 € (maximum 6 €). During all three trials of the recognition task six stimuli not previously seen (neutral, non-smoking objects pictures 7100, 7175, 7705 from the IAPS; two non-smoking persons pictures; one smoking persons picture) and six pictures previously seen were presented. Previous seen pictures were selected randomly for the recognition task. Subjects received general instructions to avoid movements during the test, to keep the head still, not to chew, and to maintain eye-fixation on the center of the screen. Following the instructions EEG was recorded while the 72 different stimuli were presented in random order. Presentations were repeated three times for a total of 288 presentations in four blocks. A recognition task presenting three stimuli followed each block. EEG recording and recognition tasks took about half an hour altogether. During each experimental trial of the EEG acquisition, a fixation-cross was flashed on the center of the screen for 500 ms followed by a blank screen for 200 ms. After this a randomly chosen stimulus appeared for 150 ms (cf. Warren et al., 1999) succeeded by a blank

screen for a randomly variating interval of 3150 – 4150 ms until the delivery of the next fixation-cross. Experimental Run Time System software (ERTS, version 3.00; Berisoft, Germany) was used to present the stimuli and simultaneously emit pulses to a neighboring computer which acquired the EEG data. These pulses permitted identification of category and onset of each stimulus. The presenting and the acquiring computers were located in a room adjoining to the sound- and light-attenuated, electrically shielded, sound- and video-monitored experimental chamber.

After EEG recording cap and electrodes were removed and after a short break, the stimuli from the study were presented again to the subjects to obtain post-experimental stimulus ratings. Subjects viewed the pictures as long as they desired to rate them for valence, arousal, and craving. For these ratings, stimuli were presented randomly and self-paced. Finally subjects were debriefed and paid.

Parallel to the described procedure several other experimental modules of a more extensive project were conducted balanced in order but not reported here.

2.5. EEG recording

EEG was recorded from all 19 scalp sites of the international 10-20 system (Jasper, 1958; Klem, Luders, Jasper, & Elger, 1999) with additional electrodes AF3, AF4, FC5, FC6, FC1, FC2, CP5, CP6, CP1, CP2 according to the ten percent electrode system (Chatrian, Lettich, & Nelson, 1985; Chatrian, Lettich, & Nelson, 1988) using a forehead ground (AFz) and reference located at Cz. Two clip electrodes were attached to the earlobes (A1, A2). Four additional electrodes were placed to the outer canthi of both eyes and above and below the right eye (bipolar horizontal/vertical electrooculogram; EOG).

At all sites Marquette Hellige Ag/AgCl electrodes were used. Electrodes for the EEG recordings were mounted in an electrode cap (Easycap, Falk Minow Services, Germany). All channels were amplified with a gain of 1000, impedance values were kept below 5 kΩ. For recording, a Neuroscan amplifier (32 channels, SynAmps, Model 5083, Neuroscan, Inc.) and Acquire 4.0 acquisition software (Neuroscan, Inc.) were used. High- and low-pass were set to 0.05 and 70 Hz (12 dB/octave). All channels were sampled continuously at a rate of 500 Hz and data were stored for off-line analysis.

2.6. EEG processing and statistical analyses

EEG data were processed using Brain Vision Analyzer software (Brain Products, Germany). Due to an unknown technical reason, ear channels A1 and A2 had reversed polarity in 25 subjects of the experimental sample. Data sets affected by the polarity shift could be identified with certainty by the polarity of blink artifacts in channels A1 and A2 (see figures A-1 to A-4 in the appendix A). For the subjects affected by polarity change, channels A1 and A2 were retransformed to correct recording conventions before further processing. To ensure that reversing the polarity of a channel would not distort data and give a correct signal, it had been checked with a pulse generator giving a 100 µV, 10 Hz rectangle-signal.

EEG data of 8 additional subjects were acquired to explore the technical artifact under varying circumstances (blockwise stimulus presentation with different sequences); however the reason for the polarity change could not be identified. Data of additional subjects were not included in the experimental sample because of the deviant experimental procedure. Even though the reason for the polarity change artifact remains unknown, its impact on data was eliminated by the recomputation of affected channels A1 and A2.

For further processing, the ear channels A1 and A2 were combined to form a linked reference and all EEG-data were re-referenced to this offline. The implicit reference channel was reused (Cz). Data were digitally refiltered to a bandwidth from 0.05 to 40 Hz using shift-free Butterworth filters (24 db/octave slope). Sections with a difference less than 0.10 µV between the maximum and the minimum in an interval of 100 ms length were omitted. Continuous EEG was segmented to epochs of 3100 ms starting 100 ms prior to the onset of each visual stimulus. After ocular correction for blinks and eye movements using the regression method described by Gratton, Coles, and Donchin (1983) without raw average subtraction because of the possibility of time-locked eye movements, epochs with an EEG activity above/below $^{+}/_{-}$ 150 µV or a step of more than 50 µV per sampling point (Franken et al., 2004) were excluded from further analysis (234 of 12384 epochs = 1.89 % of all epochs).

Remaining epochs were corrected for baseline by subtraction of the 100 ms prestimulus interval and averaged separately for each stimulus category in each subject.

For the average subject *M* = 282.56 epochs were averaged for further analysis (*SD* = 13.44; *Mdn* = 287.00; range = 202 - 288). More detailed information about the number of averaged epochs can be found in table 5. Table 6 reports further information about the blink numbers.

Table 5: Number of averaged epochs after exclusion of artifact-afflicted trials as a function of group and stimulus type, N = 43

Group	Stimulus type	*M*	*SD*	Range
smokers [*n* = 24, (*n'* = 23)]	smoking objects	70.17 (70.96)	4.04 (1.19)	52-72 (68-72)
	non-smoking objects	69.96 (70.78)	4.32 (1.57)	51-72 (67-72)
	smoking persons	70.04 (71.00)	4.94 (1.57)	48-72 (67-72)
	non-smoking persons	70.42 (71.26)	4.25 (1.01)	51-72 (69-72)
non-smokers [*n* = 19]	smoking objects	71.37	.96	69-72
	non-smoking objects	71.21	1.65	66-72
	smoking persons	71.16	1.89	64-72
	non-smoking persons	71.32	1.46	66-72

Range: Number of epochs in the subject with the least/most epochs in the averaging. In round brackets: values of smokers after omission of one outlier, *n'* = 23; this outlier is cause of apparently different *SD* between the groups.

Table 6: Number blinks in the averaged epochs as a function of group and stimulus type, N = 43

Group	Stimulus type	*M*	*SD*	Range
smokers (*n* = 24)	smoking objects	77.21	34.35	18-158
	non-smoking objects	76.79	33.46	10-141
	smoking persons	73.63	35.99	8-166
	non-smoking persons	77.67	35.67	13-159
non-smokers (*n* = 19)	smoking objects	94.05	55.19	22-184
	non-smoking objects	95.89	55.28	25-194
	smoking persons	92.53	54.22	23-196
	non-smoking persons	92.95	55.50	20-192

Range: Number of blinks in the subject with the least/most blinks.

Separate repeated-measures ANOVAs for number of epochs and blinks with a "group" (smokers, non-smokers) x "stimulus type" (smoking objects, non-smoking ob-

jects, smoking persons, non-smoking persons) design were computed with "stimulus type" as a within-subjects factor. ANOVAs did not reveal any significant differences for "group", "stimulus type", or their interaction for the number of epochs ($F_{(1, 41)} = 1.18$, $p = .284$; $F_{(3, 123)} = 0.92$, $p = .432$; $F_{(3, 123)} = 0.30$, $p = .823$), or blinks ($F_{(1, 41)} = 1.64$, $p = .207$; $F_{(3, 123)} = 2.46$, $p = .066$; $F_{(3, 123)} = 1.02$, $p = .387$). A statistical trend observed for "stimulus type" corresponds to slightly lower numbers of blinks in trials with smoking persons stimuli than in the other stimulus categories (SP: $M = 81.98$, $SD = 45.38$; NP: $M = 84.42$, $SD = 45.56$; SO: $M = 84.65$, $SD = 44.98$; NO: $M = 85.23$, $SD = 44.89$).

The ERP components were identified initially from the super grand average formed across both groups, all four stimulus categories, and the three midline sites (see figure A-5 in the appendix A). Polarity and the latency at which the maximum of each component occurred relative to stimulus onset were used in naming the component. Thus the negative peak reaching its maximum at 118 ms was termed the N118; the positive peak occurring at 162 ms was termed the P162. A N246 and a P362 were also identified. In the super grand average time-windows for automatic individual peak amplitude measurements were defined by visual inspection: N118: 85-140 ms, P162: 145-185 ms, N246: 190-310 ms, P362: 280-450 ms. Peaks were defined as local maximums and voltage was gauged relative to the mean voltage of the 100 ms pre-stimulus baseline (base-to-peak amplitudes). Every peak was identified in a reference channel. In the remaining channels peak voltages were assigned at the same latency as in the reference channel (reference channels: N118: Fz; P162: Cz; N246: Fz; P362: Pz).

Slow positive wave activity (SPW) was captured with an area measurement (μV * ms) for an interval from 500-850 ms for the formation and maintenance of the SPW. The phase of abatement of SPW was assessed separately in an interval from 850-1300 ms. Intervals were chosen after visual inspection of the super grand average. Area measurement was used because of the absence of pronounced peaks. Because of a different SPW-time course than in some previous studies (e.g. Cuthbert et al., 2000; Franken et al., 2003), intervals were defined differently.

Cue-reactivity scores were computed for all area measures and peak voltage measurements. Following Herrmann et al. (2000a; 2001b), cue-reactivity was defined as ERP amplitude or area measure of smoking-related minus amplitude or area of neutral

stimuli. This computation was done for all component and area measures, separately for persons and objects stimuli. Note that following the hypotheses (see section 1.5.) for all components N300, P300, and SPW substantially higher/more positive cue-reactivity scores are expected for smokers than for non-smokers as a manifestation of cue-reactivity.

For all group analyses of cue-reactivity (section 3.5.), cue-reactivity scores were submitted to repeated-measures ANOVAs separately for each component and measure of area information looking for differences between smokers and non-smokers. Each ANOVA was performed with the factors “group” (smokers, non-smokers), “midline” (Fz, Cz, Pz), and “stimulus type” (persons, objects), with “midline” and “stimulus type” as within-subjects factors. Figure 1 presents the processed and corrected grand average ERP waveforms at midline sites of smokers and non-smokers in response to NO, SO, NP, and SP stimuli, the group analyses were based on this data.

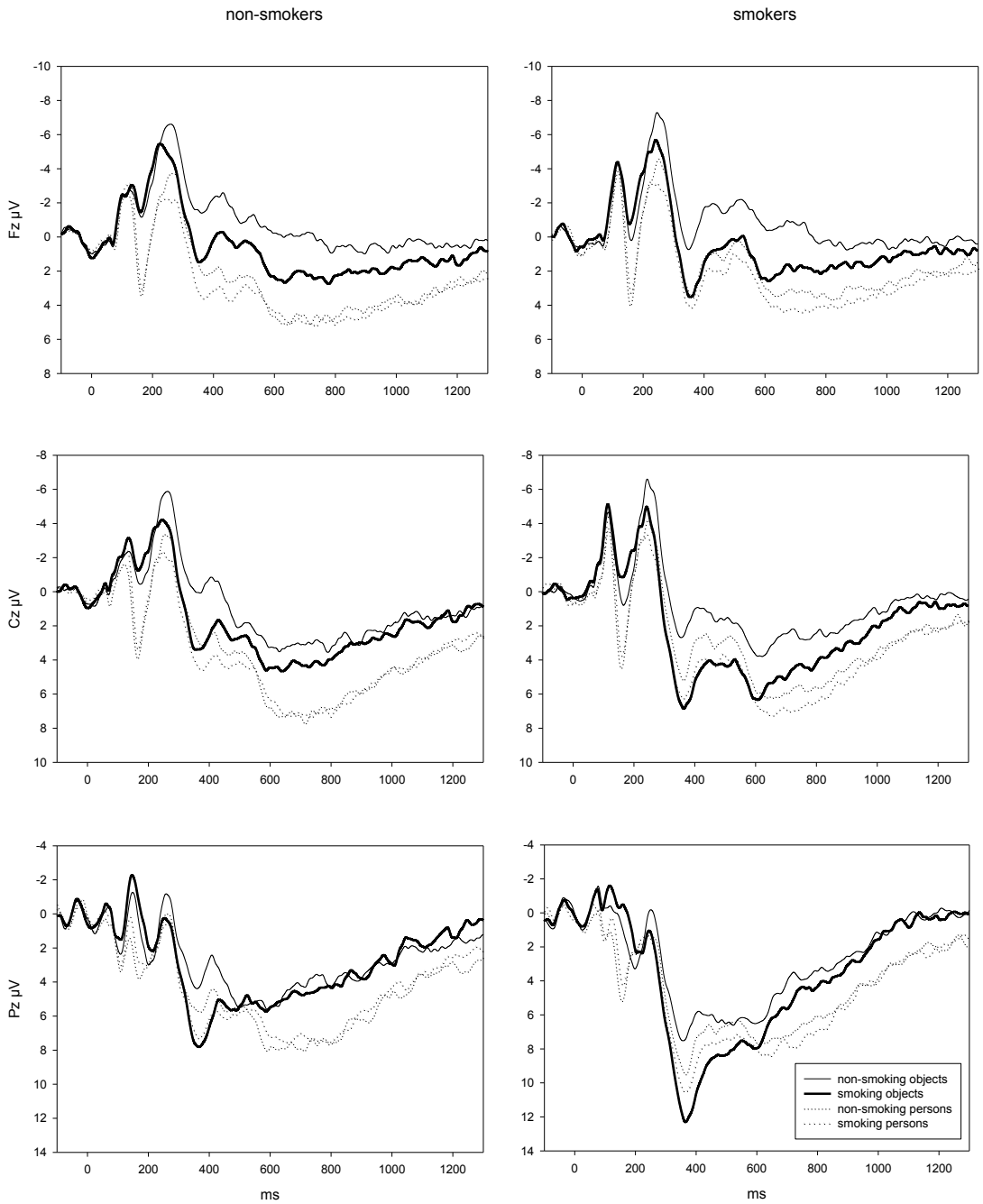

Figure 1: Non-smokers' (n = 19) and smokers' (n = 24) (columns) grand average ERP waveforms (µV, ms after stimulus-onset) at Fz, Cz, and Pz sites (rows) in response to four types of stimuli: non-smoking objects, smoking objects, non-smoking persons, and smoking persons.

3. Results

The Statistical Package for the Social Sciences (SPSS, Version 13.0.1, SPSS Inc., USA) was used for computations. For ANOVAs including within-subject factors, corrected degrees of freedom according to Greenhouse and Geisser (1959) are reported. Additional contrast analyses and MANOVAs with repeated measurements were computed with Statistica (Version 6.1, StatSoft, USA). An alpha level of .05 was used for all statistical tests.

3.1. Impact of stressor task on self-reported craving and mood

Craving

As revealed by a MANOVA (Statistica) of smokers' ratings on craving items a and b with repeated measurement (pre- vs. post-stressor), there was a trend for an increase in craving (Wilk's $\Lambda = .796$; $F_{(2, 22)} = 2.81$, $p = .082$). According to follow up analyses with matched pair *t*-tests for the pre- versus post-stressor scores, craving as assessed in item a increased significantly ($t_{(23)} = -1.91$, $p = .035$ one-tailed) (pre: $M = 6.84$, $SD = 2.54$; post: $M = 7.14$, $SD = 2.39$). No effect was found for craving item b ($t_{(23)} = -0.06$, $p = .477$ one-tailed) (pre: $M = 2.63$, $SD = 2.10$; post: $M = 2.65$, $SD = 1.95$). Craving ratings of non-smokers were not considered in these statistical analyses because of their minimal variance.

Mood

Stressor effects on mood were explored using a MANOVA (Statistica) with factors "group" (smokers, non-smokers) and repeated measures (pre-stressor, post-stressor) on the three MDBF subscales. There was a significant change in mood from pre- to post-stressor (Wilk's $\Lambda = .362$; $F_{(3, 39)} = 22.90$, $p < .000$) but no significant group effect or interaction in the multivariate statistics. For each subscale a follow-up analysis was computed using an ANOVA (SPSS) with a group-factor and repeated measures (pre-stressor, post-stressor) on the subscale. Analyses confirmed a significant drop in good mood ($F_{(1, 41)} = 19.63$, $p < .000$) (pre: $M = 3.38$, $SE = 0.33$; post: $M = 1.20$, $SE = 0.44$), an increase in agitation ($F_{(1, 41)} = 31.08$, $p < .000$) (pre: $M = 2.59$, $SE = 0.42$; post: $M = -0.30$, $SE = 0.51$), and an increase in alertness ($F_{(1, 41)} = 64.77$, $p < .000$) (pre: $M = -1.04$,

SE = 0.43; post: *M* = 3.17, *SE* = 0.38). There was an additional effect for "group" not found in the multivariate analysis indicating higher alertness in non-smokers (*M* = 1.71, *SE* = 0.46) than in smokers (*M* = 0.42, *SE* = 0.41) ($F_{(1, 41)} = 4.36$, $p = .043$).

3.2. Initial differences in mood between smokers and non-smokers

To check for initial differences in mood between smokers and non-smokers prior to the EEG recording, a MANOVA (SPSS) for group differences on the three MDBF subscales was conducted but did not yield significant multivariate group differences (Wilk's $\Lambda = .938$; $F_{(3, 39)} = 0.86$, $p = .470$). Follow-up analyses confirmed the absence of significant group effects on the three separate subscales of good-bad mood ($F_{(1, 41)} = 0.02$, $p = .895$), alertness-fatigue ($F_{(1, 41)} = 2.07$, $p = .158$), and calmness-agitation ($F_{(1, 41)} = 0.52$, $p = .475$).

3.3. Post-experiment ratings of the experimental stimuli for evoked craving, valence, and arousal

After EEG recording, 23 smokers and 18 non-smokers rated emotional valence, arousal, and craving evoked by each of the experimental stimuli.

Craving

A two-way ANOVA for smokers with "smoking content" (smoking, non-smoking) and "social content" (persons, objects) as within-subjects factors revealed significantly higher mean craving in response to smoking associated stimuli than to stimuli without association to smoking ($F_{(1, 22)} = 41.75$, $p < .000$). According to a significant interaction of "smoking content" and "social content" ($F_{(1, 22)} = 8.21$, $p = .009$), the difference in elicited craving between stimuli with and without smoking-association was larger for objects than for persons stimuli (see figure 2 and table A-8 in the appendix A). Non-smokers' craving ratings were not analyzed because of a lack of variance (non-smokers: NO: *M* = 1.06, *SD* = 0.15; SO: *M* = 1.09, *SD* = 0.21; NP: *M* = 1.06, *SD* = 0.21; SP: *M* = 1.08, *SD* = 0.24).

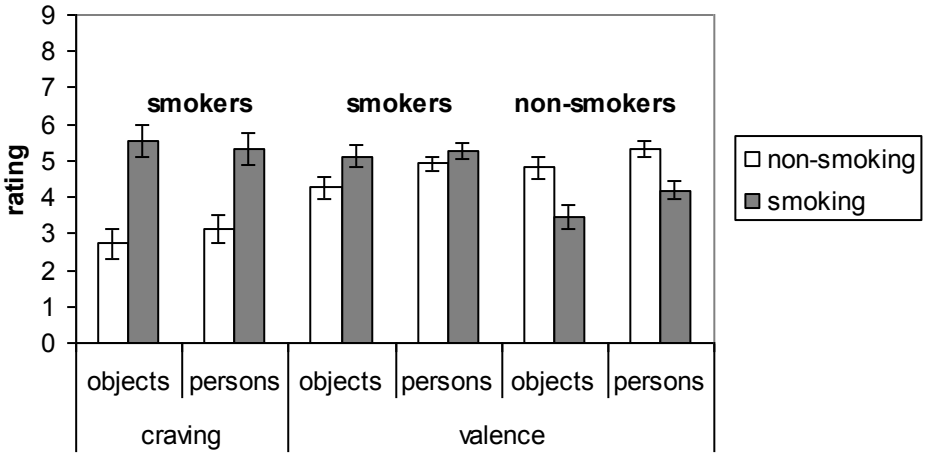

Figure 2: Estimated marginal means and standard errors of post-experiment ratings given by smokers (n = 23) and non-smokers (n = 18) for craving and valence of four types of experimental stimuli: non-smoking objects, smoking objects, non-smoking persons, smoking persons. Higher craving values indicate higher perceived craving. Valence ratings below five indicate unpleasant values, above five they are pleasant.

Ratings of smokers and non-smokers for emotional valence and arousal evoked by experimental stimuli were submitted to separate "group" (smokers, non-smokers) x "smoking content" of stimuli (smoking, non-smoking) x "social content" of stimuli (persons, objects) ANOVAs.

Valence

The analysis for valence yielded a trend for "smoking content" ($F_{(1, 39)} = 3.74$, $p = .060$) with more pleasant ratings for non-smoking stimuli ($M = 4.83$, $SE = 0.17$) than for smoking stimuli ($M = 4.51$, $SE = 0.18$). Yet the impact of "smoking content" was largely dependent on group affiliation ($F_{(1, 39)} = 31.11$, $p < .000$). While in non-smokers non-smoking stimuli evoked more positive emotions than smoking cues, this effect was reversed in smokers (non-smokers: non-smoking stimuli: $M = 5.07$, $SD = 0.26$, smoking stimuli: $M = 3.82$, $SD = 0.27$; smokers: non-smoking stimuli: $M = 4.59$, $SD = 0.23$, smoking stimuli: $M = 5.20$, $SD = 0.24$). Persons stimuli ($M = 4.93$, $SE = 0.14$) had significantly more pleasant ratings than objects stimuli ($M = 4.41$, $SE = 0.19$) ($F_{(1, 39)} = 14.63$, $p < .000$). In addition to the two-way interaction there was a significant "group" x "smoking content" x "social content" interaction ($F_{(1, 39)} = 5.46$, $p = .025$) (see figure

2, and also table A-9 in the appendix A) that was followed by separate ANOVAs for each group. In smokers, smoking-related stimuli received significantly more pleasant ratings than non-smoking stimuli ($F_{(1, 22)} = 6.64$, $p = .017$). This difference was significantly higher in objects stimuli than in persons stimuli (interaction of "smoking content" and "social content": $F_{(1, 22)} = 5.90$, $p = .024$). Moreover, there was a trend for persons stimuli: they tended to receive more pleasant ratings than objects stimuli ($F_{(1, 22)} = 3.70$, $p = .067$). In non-smokers, smoking-related stimuli received significantly less positive ratings than non-smoking stimuli ($F_{(1, 17)} = 30.61$, $p < .000$), and persons stimuli received more positive ratings than objects stimuli ($F_{(1, 17)} = 18.51$, $p < .000$).

Arousal

For arousal ratings neither group effects nor interactions were found. Smoking related stimuli ($M = 3.28$, $SE = 0.29$) evoked significantly higher arousal than non-smoking stimuli ($M = 2.57$, $SE = 0.24$) ($F_{(1, 39)} = 12.25$, $p = .001$). An effect for "social content" was marginally significant ($F_{(1, 39)} = 4.11$, $p = .050$) with persons stimuli ($M = 3.05$, $SE = 0.24$) more arousing than objects stimuli ($M = 2.80$, $SE = 0.26$). An interaction effect of "smoking content" and "social content" appeared as a trend, indicating again greater differences in arousal ratings for smoking and non-smoking objects stimuli than persons stimuli ($F_{(1, 39)} = 3.75$, $p = .060$), (NO: $M = 2.37$, $SD = 0.25$; SO: $M = 3.24$, $SD = 0.31$; NP: $M = 2.77$, $SD = 0.24$; SP: $M = 3.32$, $SD = 0.28$).

3.4. Comparison between smokers and non-smokers in event-related reactions to stimuli without smoking-association

To test for absolute differences of amplitudes in smokers and non-smokers for stimuli without association to smoking, repeated-measures ANOVAs were conducted using the between-subjects factor "group" (smokers, non-smokers) and the within-subjects factor "midline" (Fz, Cz, Pz). Because of restricted direct comparability, ANOVAs were done separately for NP and NO stimuli. Separate ANOVAs were computed for every component and every area measure. Corrected degrees of freedom (Greenhouse & Geisser, 1959) are reported for effects and interactions of the within-subjects factor.

For N118, N246, and SPW 500-850 there were neither significant effects nor trends for "group" nor "group" x "midline" interactions in both sets of stimuli. Trends for group differences in NP stimuli were found for P362 ($F_{(1, 41)} = 4.05$, $p = .051$) and SPW 850-1300 ($F_{(1, 41)} = 3.38$, $p = .073$). For NO stimuli there were also a trend for group

differences in P162 ($F_{(1, 41)}$ = 3.01, p = .090) and significant group differences in P362 ($F_{(1, 41)}$ = 5.33, p = .026) with higher amplitudes in smokers (see figure 1).

3.5. Group analyses of the several ERP components

ERP cue-reactivity scores were submitted to repeated-measures ANOVAs separately for each component's peak measure and both area measures looking for differences between smokers and non-smokers. Each ANOVA was done using a "group" (smokers, non-smokers) x "midline" (Fz, Cz, Pz) x "stimulus type" (persons, objects) design with "midline" and "stimulus type" being within-subject factors.

3.5.1. N118, P162, N246

ANOVAs for N118, P162, and N246 produced no significant effects or interactions. For P162, cue-reactivity scores tended to become more negative from anterior to posterior (Fz: M = -0.59, SE = 0.22; Cz: M = -0.78, SE = 0.20; Pz: M = -1.04, SE = 0.24) just missing significance ($F_{(1.23, 50.26)}$ = 2.99, p = .082).

3.5.2. P362

Analysis for this component yielded a significant effect for the electrode site on the midline ($F_{(1.31, 53.76)}$ = 8.43, p = .003) with increasing cue-reactivity scores from frontal to posterior (Fz: M = 2.05, SE = 0.3; Cz: M = 2.72, SE = 0.32; Pz: M = 2.73, SE = 0.33) (see also table 7 incorporating additional information for stimulus type). Effects of "stimulus type" reached significance ($F_{(1, 41)}$ = 17.84, p < .000) with higher cue-reactivity scores for objects stimuli (M = 3.31, SE = 0.38) than for persons stimuli (M = 1.69, SE = 0.33). These effects were complemented with a significant interaction of "midline" and "stimulus type" ($F_{(1.31, 53.79)}$ = 4.03, p = .039) of a hybrid type according to Leigh et al. (1980) leaving the main effect for "stimulus type" interpretable but delimiting the increase of cue-reactivity scores from frontal to central and posterior to objects stimuli. Cue-reactivity scores for persons stimuli reached maximum at Cz (see table 7). A trend effect for "group" x "stimulus type" with differences of cue-reactivity scores between persons and objects stimuli tending to be higher in smokers than in non-smokers (see table 8) missed significance ($F_{(1, 41)}$ = 3.40, p = .072). In analyses of contrasts conducted with Statistica, the difference between cue-reactivity scores of persons stimuli and objects stimuli was highly significant in smokers ($F_{(1, 41)}$ = 20.83, p < .000)

but not significant in non-smokers ($F_{(1, 41)}$ = 2.83, p = .119). However, for persons stimuli the difference between cue-reactivity scores in smokers and non-smokers ($F_{(1, 41)}$ = 0.77, p = .384) was not significant, as it was for objects stimuli ($F_{(1, 41)}$ = 1.24, p = .271).

Table 7: Estimated marginal means of P362 cue-reactivity scores as a function of "stimulus type" and "midline location"

Midline site	Stimulus type			
	persons		objects	
	M	*SE*	*M*	*SE*
Fz	1.49	0.38	2.62	0.36
Cz	1.91	0.35	3.53	0.42
Pz	1.66	0.39	3.79	0.40

Table 8: Estimated marginal means of P362 cue-reactivity scores as a function of "group" and "stimulus type"

Group	Stimulus type			
	persons		objects	
	M	*SE*	*M*	*SE*
smokers (n = 24)	1.40	0.44	3.73	0.50
non-smokers (n = 19)	1.98	0.49	2.90	0.56

3.5.3. Slow positive wave: 500-850 ms

This ANOVA revealed a significant main effect for "midline" ($F_{(1.4, 57.47)}$ = 12.54, p < .000) indicating decreasing cue-reactivity scores from frontal to posterior (Fz: M = 476.68, SE = 81.34; Cz: M = 365.81, SE = 80.24; Pz: M = 154.41, SE = 80.73). "Stimulus type" had a significant effect, too, ($F_{(1, 41)}$ = 11.98, p = .001) with higher cue-reactivity scores for objects stimuli (M = 528.23, SE = 90.91) than for persons stimuli (M = 136.37, SE = 91.33).

Main effects were accompanied by a significant interaction for "midline" and "stimulus type" ($F_{(1.44, 58.93)}$ = 8.39, p = .002) of ordinal type described by Leigh et al. (1980). Therefore, main effects can still be interpreted, when amended by the fact that differences of cue-reactivity scores between electrode positions are smaller for persons stimu-

li (see figure 3 and figure 1) than for objects stimuli. An effect for "group" barely missed significance ($F_{(1,\ 41)}$ = 3.86, p = .056; but note: p = .028 one-tailed). Cue-reactivity scores tended to be higher in smokers (M = 472.58, SE = 94.93) than in non-smokers (M = 192.02, SE = 106.69). Further analyses were conducted of this area measure to explore the marginal cue-reactivity effect as reported in section 3.6. (additionally, table A-10 in the appendix A gives the estimated marginal means of the SPW 500.850 ms cue-reactivity scores as a function of "stimulus type" and "midline location").

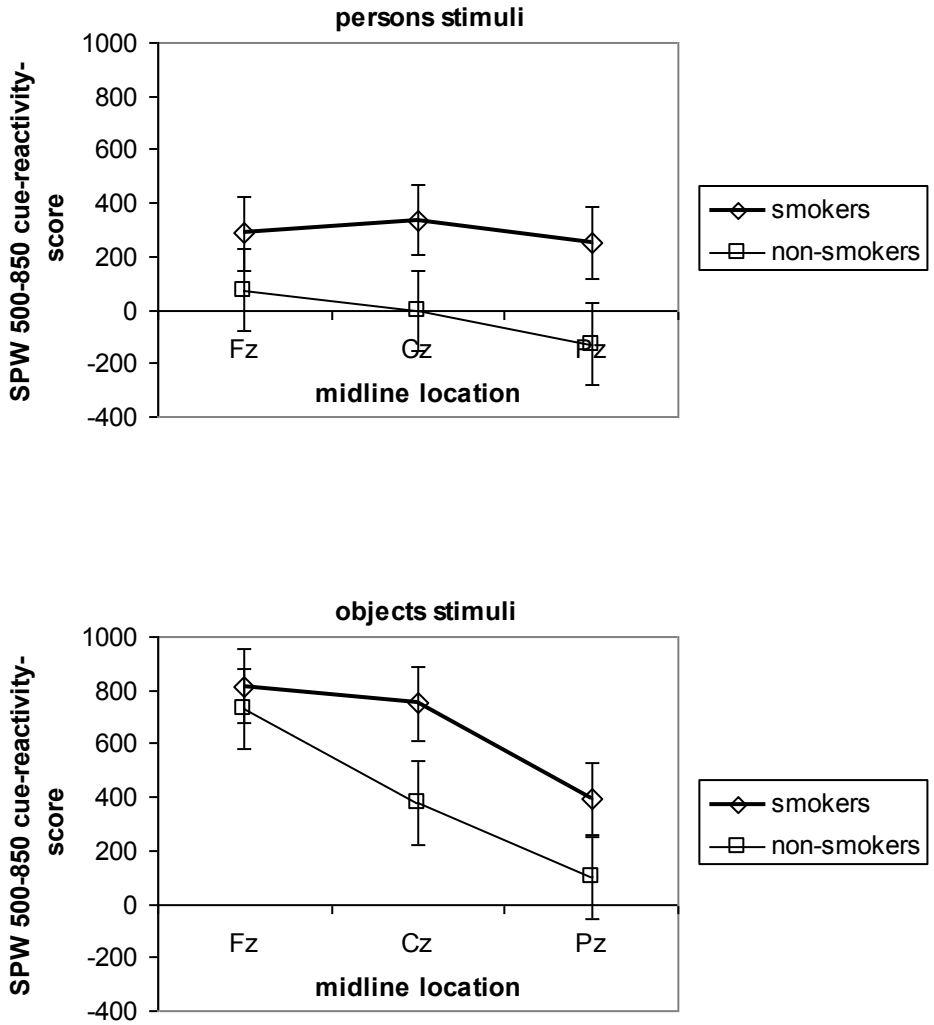

Figure 3: Slow positive wave (SPW) 500-850 ms: Estimated marginal means of cue-reactivity scores of smokers (n = 24) and non-smokers (n = 19) for persons (top) and objects stimuli (bottom) at midline locations Fz, Cz, and Pz.

3.5.4. Slow positive wave: 850-1300 ms

For this area measure only a significant effect for "midline" was found ($F_{(1.66, 68.01)}$ = 8.08, p = .001). Cue-reactivity scores decreased from frontal to posterior, finally becoming negative (Fz: M = 281.12, SE = 116.03; Cz: M = 130.55, SE = 122.46; Pz: M = -58.58, SE = 107.59). Additionally there was a trend for an interaction of "midline" x "stimulus type" ($F_{(1.54, 63.13)}$ = 2.51, p = .088) of ordinal type (Leigh et al., 1980). Differences of cue-reactivity scores between electrode positions on the midline tended to be smaller for persons stimuli than for objects stimuli (see table 9).

Table 9: Estimated marginal means of cue-reactivity scores of 850-1300 ms slow positive wave as a function of "stimulus type" and "midline location"

Midline site	Stimulus type			
	persons		objects	
	M	*SE*	*M*	*SE*
Fz	146.73	142.21	415.51	132.29
Cz	31.82	142.95	229.27	140.24
Pz	-76.20	137.68	-40.97	130.19

N = 43

3.6. Slow positive wave 500-850: Further analyses of cue-reactivity

To explore the effects of cue-reactivity further analyses were conducted. Non-midline sites were explicitly included because some studies, like van de Laar et al. (2004), failed to demonstrate cue-reactivity at midline sites. According to Warren et al. (1999), an analysis of non-midline sites was applied "using 12 symmetric and balanced, non-midline, topographic regions, using a 'group' x 'hemisphere' (left, right) x 'lateral-medial' (lateral, medial) x 'anterior-posterior' (frontal, central, posterior) x 'stimulus type'" (persons, objects) design (p. 1573) with cue-reactivity scores as dependent variable. The design "covered the left (F3, C3, P3) and right medial (F4, C4, P4) and left (F7, T3, T5) and right lateral (F8, T4, T6) sites. The anterior-posterior axis was balanced: frontal (F7, F3; F4, F8); central (T3, C3; C4, T4); and posterior (T5, P3; P4, T6)" (Warren et al., 1999, p. 1573). Significant effects involving the factor "group" were interactions for "group" x "lateral-medial" ($F_{(1, 41)}$ = 4.92, p = .032) and "group" x "hemisphere" x "lateral-medial" x "anterior-posterior" x "stimulus type" ($F_{(1.89, 41)}$ = 4.84, p

= .012). For the "group" x "lateral-medial" interaction the contrast analysis revealed no significant differences between the groups lateral ($F_{(1, 41)}$ = 0.47, p = .499) and medial ($F_{(1, 41)}$ = 2.82, p = .101) but significant differences between lateral and medial sites in smokers ($F_{(1, 41)}$ = 30.77, p < .000) and non-smokers ($F_{(1, 41)}$ = 3.86, p = .056) with the interaction indicating bigger differences in smokers than non-smokers (see table 10, ordinal interaction).

Table 10: Estimated marginal means of cue-reactivity scores for 500-850 ms SPW as a function of "group" and "lateral-medial" location

Group	Location lateral-medial	*M*	*SE*
smokers (n = 24)	lateral	146.35	50.37
	medial	400.41	80.06
non-smokers (n = 19)	lateral	94.66	56.62
	medial	195.81	91.1

Lateral: sites F7, T3, T5; F8, T4, T6, medial: sites F3, C3, P3; F4, C4, P4.

An analysis including the midline sites used a "group" x "anterior-posterior" (frontal, central, posterior) x "laterality" (left, midline, right) x "stimulus type" (persons, objects) design. The anterior-posterior axis was balanced: frontal (F3, Fz, F4), central (C3, Cz, C4), and posterior (P3, Pz, P4). Covered sites were left (F3, C3, P3), midline (Fz, Cz, Pz), and right (F4, C4, P4). The analysis still revealed a trend for "group" only ($F_{(1, 41)}$ = 3.23, p = .080; but note: p = .040 one-tailed). The factor "group" was not involved in any other significant interaction effects or trends.

Further analyses were done separately for each midline location (see Herrmann et al., 2000a) using a "group" (smokers, non-smokers) x "stimulus type" (persons, objects) design in ANOVAs according to the GLM with cue-reactivity scores as dependent variables. These analyses revealed significant cue-reactivity effects at Cz and Pz but not at Fz. Cue-reactivity did not depend on "stimulus type" (persons, objects) as no "group" x "stimulus type" interaction reached significance. For a summary of the analyses see table 11, figure 3 illustrates the results, additionally table A-11 in the appendix A reports the related descriptive statistics of figure 3.

Table 11: Summary of two-way analyses of variance (GLM) of cue-reactivity scores of 500-850 ms SPW for the factors "group" and "stimulus type" (persons, objects) computed separately for each midline electrode position, N = 43.

Midline site	Source	*F*	*p*
Fz	group	0.83	.368
	stimulus type	22.61	.000**
	group x stimulus type	0.25	.618
Cz	group	4.87	.033*
	stimulus type	9.25	.004**
	group x stimulus type	0.18	.894
Pz	group	4.41	.042*
	stimulus type	2.21	.145
	group x stimulus type	0.11	.747

For all analyses: $df = 1$, error $df = 41$; *$p < .05$, **$< .01$.

3.7. Correlations of 500 – 850 ms slow positive wave cue-reactivity scores with pre-experimental craving, characteristics of smoking history, and CO in exhaled air

In smokers cue-reactivity scores for persons and objects stimuli recorded at Fz, Cz, and Pz were not significantly related to self-reported craving immediately before ERP recording (craving item a and craving item b) with exception of a negative correlation between craving item b and cue-reactivity scores for objects stimuli at Cz ($r = -.45$, $p = .028$, two-tailed). Additionally there were no significant correlations of the cue-reactivity scores with the self-reported number of cigarettes smoked per day, the FTND-sum score, ppm CO in exhaled air at the beginning of the experiment, years smoked so far, or age at starting smoking.

3.8. Correlations of 500 – 850 ms slow positive wave cue-reactivity scores with post-experiment cue-reactivity of valence, arousal, and craving ratings

According to Warren et al. (1999), Pearson product-moment correlations were calculated for smokers ($n = 23$) between the mean of 500 – 850 ms SPW cue-reactivity scores for persons and objects stimuli at midline sites (Fz, Cz, Pz) and post-experiment

cue-reactivity scores of valence, arousal, and craving ratings (mean valence, arousal, or craving rating given to smoking related objects or persons stimuli minus the corresponding mean for neutral stimuli). Significant correlations were found sporadically only, mainly at Pz (see table 12), and these were negative. There were no correlations for craving ratings but for valence and arousal ratings.

Table 12: Pearson product-moment correlations for smokers (n = 23) between 500 – 850 ms SPW cue-reactivity scores for persons and objects stimuli at midline sites and post-experiment cue-reactivity scores for valence, arousal, and craving ratings of objects and persons stimuli

Cue-reactivity-scores for rating type/ stimulus type	*r*: 500 – 850 ms cue-reactivity scores at midline location/for stimulus type					
	Fz/objects	Fz/persons	Cz/objects	Cz/persons	Pz/objects	Pz/persons
valence/objects	-0.11	-0.43*	-0.18	-0.19	-0.43*	-0.24
	$p = .611$	$p = .039$	$p = .415$	$p = .379$	$p = .043$	$p = .273$
valence/persons	-0.26	-0.29	-0.35	-0.17	-0.60**	-0.16
	$p = .236$	$p = .179$	$p = .106$	$p = .437$	$p = .003$	$p = .477$
arousal/objects	-0.05	-0.21	-0.12	-0.42*	-0.29	-0.53**
	$p = .812$	$p = .345$	$p = .584$	$p = .044$	$p = .187$	$p = .009$
arousal/persons	-0.20	-0.11	-0.39^{+}	-0.25	-0.57**	-0.34
	$p = .372$	$p = .632$	$p = .068$	$p = .249$	$p = .005$	$p = .111$
craving/objects	-0.17	-0.18	-0.23	-0.29	-0.24	-0.21
	$p = .432$	$p = .417$	$p = .285$	$p = .178$	$p = .277$	$p = .336$
craving/persons	-0.13	-0.02	-0.22	-0.06	-0.28	0.03
	$p = .547$	$p = .932$	$p = .314$	$p = .789$	$p = .196$	$p = .892$

$^{+}$: $p < .10$, * $p < .05$, ** $p < .01$.

3.9. Principal component analyses

Several principal component analyses (PCAs) of covariance matrices (Rösler & Manzey, 1981) were applied to the voltages in epochs ranging from 0 to 3000 ms after stimulus onset. Extracted components were Varimax-rotated. All PCAs were done using the data of both groups together inserting the data of all 19 electrodes according to the 10-20 system (Jasper, 1958; Klem et al., 1999). PCAs with different numbers of factors

(2-9) were computed for persons and objects stimuli both separately and together. Finally, a four-factorial solution was selected because it included a special factor representing the P362 component. Such a factor did not exist in a three-factorial solution. Solutions having more than four factors yielded pronounced bipolar factors, which were to be avoided for interpretation reasons. As separate four-factorial solutions for persons and objects stimuli gave comparable factors, results of the PCA for persons and objects stimuli are reported together. All four factors accounted for 81.6% of total variance. The first extracted factor (44.5% of total variance) corresponded to early slow positive wave; the second factor (29.1%) represented the P362 component. Late slow positive wave was indicated by the third factor (13.3%). Earlier components N118 and N246 were mainly accounted for by the fourth factor (3.7%) and secondarily by the third factor. Factor loadings are reported in figure 4.

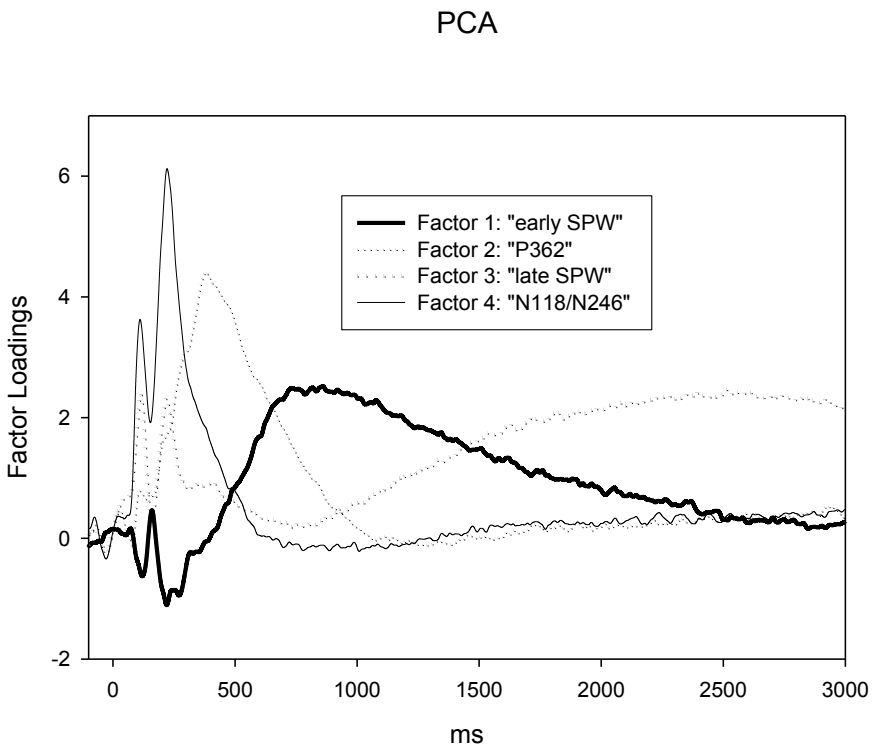

Figure 4: Factor loadings, emerging from the four-factorial principal components analysis together for smokers and non-smokers inserting data of all 19 sites according to the 10-20 system. The factors are interpreted as N118/N246, P362, early SPW, and late SPW.

Cue-reactivity scores were computed from the resulting factor scores from the PCA using the procedure described in section 2.6. and submitted to repeated-measures ANOVAs. ANOVAs were computed separately for each PCA-factor using a “group”

(smokers, non-smokers) x "midline" (Fz, Cz, Pz) x "stimulus type" (persons, objects) design with "midline" and "stimulus type" being within-subjects factors. However, no significant cue-reactivity effect was found in the ANOVAs for all four PCA factors. For the factor corresponding to P362, the analysis yielded a significant effect for "stimulus type" ($F_{(1, 41)}$ = 32.56, p < .000) indicating higher cue-reactivity scores derived from factor scores for objects stimuli (M = 0.69, SE = 0.07) than for persons stimuli (M = 0.26, SE = 0.06).

3.9.1. ANOVA for the first factor from PCA (corresponding to early SPW)

In this analysis of cue-reactivity scores derived from factor scores, a main effect for "midline" ($F_{(1.63, 66.85)}$ = 12.48, p < .000) and an interaction for "midline" x "stimulus type" ($F_{(1.89, 57.76)}$ = 8.81, p = .002) reached significance. Cue-reactivity scores derived from factor scores tended to become smaller and finally negative from frontal to posterior (Fz: M = 0.31, SE = 0.09; Cz: M = 0.14, SE = 0.1; Pz: M = -0.05, SE = 0.09). This effect was more pronounced for objects stimuli (see table 13).

Table 13: Estimated marginal means of cue-reactivity scores for the first factor from PCA as a function of "stimulus type" and "midline location"

Stimulus type	Midline site	*M*	*SE*
persons	Fz	0.12	0.11
	Cz	0.05	0.11
	Pz	-0.03	0.11
objects	Fz	0.51	0.12
	Cz	0.23	0.12
	Pz	-0.07	0.11

3.9.2. ANOVA for the second factor from PCA (corresponding to P362)

This analysis yielded a significant effect for "stimulus type" ($F_{(1, 41)}$ = 32.56, p < .000) indicating higher cue-reactivity scores derived from factor scores for objects stimuli (M = 0.69, SE = 0.07) than for persons stimuli (M = 0.26, SE = 0.06). Additionally, there were a trend for "midline" ($F_{(1.34, 54.80)}$ = 3.45, p = .057) (Fz: M = 0.43, SE = 0.06; Cz: M = 0.52, SE = 0.06; Pz: M = 0.47, SE = 0.06) and a trend for "midline" x "group" ($F_{(1.34, 41)}$ = 2.93, p = .081) (see table 14).

Table 14: Estimated marginal means of cue-reactivity scores for the second factor from PCA as a function of "group" and "midline location"

Group	Midline site	*M*	*SE*
smokers (n = 24)	Fz	0.46	0.08
	Cz	0.62	0.08
	Pz	0.57	0.08
non-smokers (n = 19)	Fz	0.40	0.09
	Cz	0.42	0.08
	Pz	0.36	0.08

3.9.3. ANOVA for the third factor from PCA (corresponding mainly to late SPW)

Here an interaction of "midline" x "stimulus type" x "group" ($F_{(1.69, 41)}$ = 7.75, p = .002) reached significance. This effect indicated that smokers had increasingly negative cue-reactivity scores from frontal to posterior for persons stimuli, while for objects stimuli negativity of cue-reactivity scores decreased. In non-smokers persons stimuli had a very slight increase in negativity of cue-reactivity scores from frontal to posterior, which was more pronounced for objects stimuli (see table 15).

3.9.4. ANOVA for the fourth factor from PCA (corresponding to N118 and N246)

For this factor there was a significant effect for "midline" ($F_{(1.48, 60.51)}$ = 3.51, p = .049) (Fz: M = 0.03, SE = 0.05; Cz: M = -0.02, SE = 0.05; Pz: M = -0.06, SE = 0.05) and a trend for an interaction of "midline" x "stimulus type" x "group" ($F_{(1.31, 41)}$ = 2.89, p = .085) (see table 16).

Table 15: Estimated marginal means of cue-reactivity scores for the third factor from PCA as a function of "group", "stimulus type", and "midline location"

Group	Stimulus type	Midline site	*M*	*SE*
smokers (n = 24)	persons	Fz	-0.02	0.19
		Cz	-0.36	0.19
		Pz	-0.41	0.18
	objects	Fz	-0.62	0.20
		Cz	-0.40	0.18
		Pz	-0.32	0.17
non-smokers (n = 19)	persons	Fz	-0.07	0.21
		Cz	-0.11	0.21
		Pz	-0.13	0.20
	objects	Fz	0.05	0.22
		Cz	-0.10	0.20
		Pz	-0.23	0.19

Table 16: Estimated marginal means of cue-reactivity scores for the fourth factor from PCA as a function of "group", "stimulus type", and "midline location"

Group	Stimulus type	Midline site	*M*	*SE*
smokers (n = 24)	persons	Fz	0.02	0.09
		Cz	0.05	0.09
		Pz	0.06	0.09
	objects	Fz	0.15	0.10
		Cz	-0.02	0.10
		Pz	-0.13	0.09
non-smokers (n = 19)	persons	Fz	0.03	0.10
		Cz	-0.03	0.10
		Pz	-0.05	0.10
	objects	Fz	-0.08	0.11
		Cz	-0.09	0.11
		Pz	-0.12	0.11

3.10. Peak-to-peak amplitudes

In the components P162, N246, and P362 it was possible to account for initial values for the formation of these components by subtracting the peak amplitude of the preceding component. Using these corrected values, cue-reactivity scores were computed separately for each component (P162, N246, and P362) by subtracting amplitudes of neutral stimuli from the amplitudes of smoking-related stimuli. Analyses using a "group" (smokers, non-smokers) x "midline" (Fz, Cz, Pz) x "stimulus type" (persons, objects) design were conducted separately for each component. For the P162 component no significant effect or interaction was found, for N246 there was a significant effect for "stimulus type" ($F_{(1, 41)} = 5.15$, $p = .029$) with higher cue-reactivity scores for objects stimuli than for persons stimuli (persons: $M = 1.42$, $SE = 0.32$; objects: $M = 2.33$; $SE = 0.33$). For the P362 component the analysis indicated effects for "midline" ($F_{(1.33,\ 54.43)} = 11.35$, $p = .001$) (Fz: $M = 0.80$, $SE = 0.28$; Cz: $M = 1.57$, $SE = 0.28$; *Pz*: $M = 1.92$, $SE = 0.31$), "stimulus type" ($F_{(1, 41)} = 6.07$, $p = .018$) (persons: $M = 0.88$, $SE = 0.34$; objects: $M = 1.97$, $SE = 0.34$) and a significant interaction of "midline" x "stimulus type": ($F_{(1.17,\ 47.77)} = 5.00$, $p = .025$) (see table 17). No significant effects or interactions for "group" were found.

Table 17: Estimated marginal means of cue-reactivity scores for peak-to-peak amplitudes of P362 as a function of "stimulus type" and "midline location"

Stimulus type	Midline site	*M*	*SE*
persons	Fz	0.54	0.37
	Cz	1.14	0.36
	Pz	0.97	0.42
objects	Fz	1.06	0.35
	Cz	2.00	0.39
	Pz	2.84	0.42

4. Discussion

The study at hand used a paradigm comparable to that applied by Warren and McDonough (1999; McDonough et al., 2001) but employed distinct stimuli: stimuli depicting objects and persons both with and without reference to smoking. It had been assumed that the social reference of stimuli would increase cue-reactivity effects. Stimuli were not taken from magazines. They were also parallelized with regard to evoked arousal and valence to avoid confoundations. Following Warren and McDonough a stressor task was used anteceding the EEG acquisition, but a distinct task was employed. The study at hand used an adaptive computer-controlled arithmetic task of ten minutes duration (McCubbin et al., 1992) while Warren and McDonough (1999; McDonough et al., 2001) had used a complex arithmetic rule learning task of about 30 minutes duration. Divergent from Warren and McDonough, specific bogus instructions were given to create perceived smoking opportunity. Additionally a recognition task was employed between the experimental blocks to ensure that subjects attended to the stimuli thoroughly and persistently.

In the present study no cue-reactivity effects were found for a frontal dominating N246 and a posterior dominating P362 component. However, in these time ranges Warren and McDonough (1999) had identified cue-reactivity for components called N268 (frontal dominating) and P412 (posterior dominating). In a later study (McDonough et al., 2001), cue-reactivity was mainly found for a N241 component, effects for a P358 were limited. McDonough and Warren (2001) attributed the differing peak latencies in the super grand averages of their experiments to different display software. The detected components N246 and P362 in the present study are comparable to the components reported in the experiments by Warren and McDonough regarding latency and topography. Moreover, similar underlying processes can be assumed as a comparable experimental paradigm was used. The N246 and P362 in this study correspond to the N241 and the P358 in the study of McDonough and Warren (2001) and can therefore be interpreted as N300 and P300. The absence of cue-reactivity for these N300 and P300 components in the present study partly falsifies hypothesis 1. Yet hypothesis 1 is partly confirmed by the study at hand as cue-reactivity effects were present for the SPW domain (effects for the interval between 500-850 ms at Cz and Pz, but not in the later interval),

although these effects were *not* significant at *all* midline sites and did not emerge in all analyses (see sections 3.5.3., 3.6., and 4.2.). This result for SPW cue-reactivity is in line with recent ERP cue-reactivity studies that explored different populations like smokers, heroin, cocaine, and cannabis addicts and also demonstrated SPW cue-reactivity (Franken et al., 2004; Franken et al., 2003; Littel et al., 2007; van de Laar et al., 2004; Wölfling et al., 2008). Unfortunately, the present SPW cue-reactivity was not positively correlated to self-reported craving in smokers immediately before EEG recording (hypothesis 2). Unexpectedly, a significant negative correlation was found between one item accessing craving and cue-reactivity scores for objects stimuli at Cz. Contradictory to hypothesis 3, no correlations between SPW cue-reactivity and other indices of addiction like FTND sum score, number of cigarettes smoked per day, or characteristics of the smoking history emerged. Correlations between SPW cue-reactivity scores and cue-reactivity scores formed from craving ratings for the experimental stimuli were not significant. Some isolated correlations with emotional ratings reached significance, but unexpectedly constituted negative interrelations (hypothesis 4).

Post-experiment ratings of experimental stimuli for craving expectedly revealed higher craving ratings for smoking stimuli than for non-smoking stimuli in smokers (hypothesis 5). Additionally, as hypothesized, smokers perceived smoking stimuli as more positive than non-smoking stimuli while the ratings were reversed in non-smokers. These results support hypothesis 5 and are in line with prior research (Mucha et al., 1999; Warren et al., 1999).

Actual results do not deliver support for hypothesis 6. Against expectations (see hypothesis 6a), SPW cue-reactivity scores (500-850 ms) were higher for objects stimuli than for persons stimuli, but this effect was independent from group (smokers, non-smokers). The social reference of stimuli did not increase cue-reactivity. However, results indicate that social reference might modulate cue-reactivity, but other than expected. For smokers' stimulus ratings for craving (hypothesis 6b), the social reference of stimuli also had an effect opposite to the expected. The differences in elicited craving between smoking stimuli and non-smoking stimuli were larger for objects stimuli than for persons stimuli. Smokers gave more positive ratings for smoking stimuli than for non-smoking stimuli with the difference being significantly higher for objects stimuli than for persons stimuli.

Prior to the acquisition of ERP no mood differences existed between smokers and non-smokers as indicated by the MDBF for the dimensions good-bad mood, alertness-fatigue, and calmness-agitation. Because of this, the impact of differential mood states on ERP (Kliegel, Horn, & Zimmer, 2003; Meinhardt & Pekrun, 2003) can be ruled out with high confidence.

Concordantly with the lack of initial mood differences, smokers and non-smokers showed comparable affective reactions to the stressor. However, the increase in smokers' cigarette cravings through the application of a stressor task expected according to Perkins et al. (1992) (see also Sinha et al., 2000) could be observed with regard to one of two craving items only. This might be due to a lack of the validity of the item, since a concept as broad and complex as craving is difficult to access in single items (Drummond, Litten, Lowman, & Hunt, 2000). The stressor used in the present study was of a shorter duration (10 minutes) than the stressor used by Warren and McDonough (1999; McDonough et al., 2001; 30 minutes). Perhaps a longer period of stress is needed to produce a more profound increase in craving that can be observed for both craving items. This constricted effect of stress-evoked craving might have limited ERP cue-reactivity effects in the present study. However, Littel et al. (2007) demonstrated P300 and SPW cue-reactivity effects in smokers without the employment of a anteceding stressor.

4.1. N300 (N246) and P300 (P362) components

While most previous studies conceptualized cue-reactivity as a "stimulus type" x "group" interaction, this study follows Herrmann et al. (2000a; 2001b) in computing cue-reactivity scores. Here, however objects and persons stimuli are analyzed separately to improve clarity. Cue-reactivity scores were also used because persons and objects stimuli were not directly comparable due to their different stimulus complexity. Higher cue-reactivity scores in smokers than in non-smokers were regarded as cue-reactivity.

Warren and McDonough (1999; McDonough et al., 2001) had found more negative N300 amplitudes for neutral stimuli than for smoking-associated stimuli in smokers and had interpreted this in terms of semantic priming. This study could neither replicate these results, nor find any other significant cue-reactivity effect for the N300 component. Consistently, analyses for the N300-corresponding factor scores from the PCA and

analyses of peak-to-peak amplitudes did not yield any cue-reactivity effects. Interestingly a differentiation of persons and objects stimuli was found in the analysis of N300 peak-to-peak amplitudes: cue-reactivity scores were higher for objects stimuli than for persons stimuli.

With regard to the P300, a differentiation between objects and persons stimuli with higher cue-reactivity scores for objects stimuli than for persons stimuli was found and confirmed in both the ANOVAs of PCA factor scores and peak-to-peak amplitudes. In the analysis of the original ERP values this differentiation tended to be greater in smokers, but missed significance. An effect of original cue-reactivity could be found neither for objects nor for persons stimuli. Likewise, additional analyses of PCA factor scores and peak-to-peak amplitudes did not detect any cue-reactivity effects. This result was again discrepant to Warren and McDonough who had demonstrated higher amplitudes for smoking-associated stimuli than for neutral stimuli in smokers but smaller stimulus differences in non-smokers and interpreted this in terms of allocation of attentional processes.

Explanatory notes about the lacking N300 and P300 cue-reactivity

The lack of N300 cue-reactivity in the present study – though inconsistent with Warren and McDonough (1999; McDonough et al., 2001) and a study by van de Laar et al. (2004) – is in accordance with the majority of ERP cue-reactivity research as most experiments using pictorial stimuli did not demonstrate effects in this component (Franken et al., 2004; Franken et al., 2003; Genkina et al., 1986; Herrmann et al., 2001b; Jang et al., 2007; Littel et al., 2007; Lubman et al., 2008; Lubman et al., 2007; Namkoong et al., 2004; Wölfling et al., 2008). Stimulus presentation times and recording parameter in the present study were comparable to those of Warren and McDonough, and a similar experimental paradigm was used. The main difference to the studies of Warren and McDonough lies in the experimental stimuli. Stimuli were not just divided into those with and without smoking association but were additionally divided into persons and objects types. This inflation of stimulus categories might have extended the semantic space of possible references in the stimulus processing. In the experiments of Warren and McDonough and van der Laar et al. stimuli created a simple distinction between smoking and non-smoking themes defining this simple semantic space for the perception and evaluation of stimuli. The incongruence of non-smoking stimuli to the tobacco-need

state of smokers was situated on one semantic dimension, enabling "mismatch" to easily arise. In the present study a second stimulus dimension – social reference – may have overlapped and concealed one-dimensional effects.

However, it is possible that N300 cue-reactivity effects in the sense of semantic priming have not been masked by the additional stimulus dimension but have just not emerged. Differing from the experiments of Warren and McDonough and all other cited ERP cue-reactivity experiments standardization and homogenization of stimuli were particularly emphasized in the experiment at hand. Especially the parallelization of social stimuli with regard to arousal and valence might have ruled out important differences for the creation of both N300 and P300 ERP cue-reactivity. During the process of stimulus generation stimulus differences in valence and arousal were minimized to create more precise stimuli and expunge confoundings, but possibly these differences usually accompanying smoking associated stimuli might be essential for the demonstration of more prominent ERP cue-reactivity. Cuthbert et al. (2000) demonstrated variation of ERP with the judged affective arousal of stimuli while Amrhein et al. (2004) found additional effects of valence. A mere selection of smoking stimuli evoking maximum craving and non-smoking stimuli evoking minimum craving in smokers might have been less artificial and more effective in creating ERP cue-reactivity, especially taking into account that stimulus differences could not be erased totally. Specific valence and arousal of smoking stimuli for smokers can be seen as cue-reactivity effects that should not be eliminated and are natural properties of smoking stimuli. But if ERP cue-reactivity in N300 and P300 are found for such stimuli only, it can not be determined if ERP cue-reactivity effects have a basis further than valence and arousal effects on ERP. Future research should try to find methods to solve this problem and explore the interrelationships between craving, valence, arousal, and ERP cue-reactivity.

In the comparisons of smokers' and non-smokers' ERPs to stimuli without smoking-association (see section 3.4.) there is evidence for increased absolute P300 amplitudes in smokers. Augmented P300 amplitudes in smokers might reflect an increased general activation of attentional resources extending even over stimuli without smoking-association during the time of the experiment. These increased attentional processes might have been evoked by the experimental situation associated to smoking. Generally augmented allocation of attentional resources might have reduced the opportunity for differentiation between smoking and non-smoking stimuli for the P300 component

("ceiling effect"). An increased P300 amplitude in smokers is especially noteworthy as there is some evidence from non-cue-reactivity experiments that smokers' P300 amplitudes are typically reduced (Anokhin et al., 2000; Neuhaus et al., 2006).

Another problem reducing the opportunity for special P300 cue-reactivity effects to arise are non-smokers' substantial P300 cue-reactivity scores for both objects and persons stimuli. It was expected that cue-reactivity scores should be higher in smokers than in non-smokers and ideally non-smokers' scores should be close to zero or even negative for a maximal separation effect. Unexpectedly, however, non-smokers' P300 cue-reactivity scores for persons stimuli – without regard of statistical significance - were even higher than those of smokers. For objects stimuli the difference was as anticipated (see section 3.5.2.). The explanation for the relatively high cue-reactivity scores remains unclear, however, at least for objects stimuli, it can be speculated that the non-smoking stimuli were so uninteresting and evoked such low arousal that attention even in non-smokers was drawn to smoking stimuli. Additionally, P300 cue-reactivity scores for persons stimuli in general were lower than for objects stimuli giving rise to the speculation that for social stimuli social aspects might have distracted from the smoking to non-smoking differentiation.

As no initial mood differences between smokers and non-smokers were found, there is no convincing evidence that the lack of N300 and P300 cue-reactivity effects might be attributable to such initial differences. Rather one could assume that such mood differences might contribute to cue-reactivity effects in less controlled studies. Mood differences might be correlated with extended smoking deprivation or confounding variables that promote cue-reactivity.

Possibly the stressor was not effective enough to produce sufficient craving and cue-reactivity. Warren and McDonough had used a stressor task of longer duration. However, other ERP cue-reactivity experiments demonstrated N300 or P300 cue-reactivity without the use of a prior stressor (Genkina et al., 1986; Herrmann et al., 2000a; Herrmann et al., 2001b; Littel et al., 2007; Lubman et al., 2008; Lubman et al., 2007; Namkoong et al., 2004; van de Laar et al., 2004). This creates doubt that the lack of N300 and P300 cue-reactivity is attributable to insufficient stress-induced craving in smokers.

Although the present study used a comparable paradigm (stimulus presentation time, presentation mode, stimulus size, screen-subject-distance, sample size) and comparably addicted smokers, the N300 and P300 cue-reactivity effects of Warren et al. (1999) and McDonough et al. (2001) could not be replicated. As outlined above, methodological aspects like stimulus characteristics, lack of initial mood differences, and stressor type might be more or less convincing reasons for the lack of N300 and P300 cue-reactivity in the experiment at hand. With regard to some of the aspects above, it is possible that the desire to eliminate methodological flaws might have led to the absence of cue-reactivity. On the other hand, the present study might be evidence that N300 and P300 cue-reactivity effects are not persistent phenomena in smokers/addicts.

ERP cue-reactivity research has so far demonstrated effects concerning un-uniform ERP components. In the research done with picture stimuli, N300 cue-reactivity effects were demonstrated by a minority of studies only (McDonough et al., 2001 [smokers]; van de Laar et al., 2004 [cocaine addicts]; Warren et al., 1999 [smokers]). However, a majority of ERP cue-reactivity experiments demonstrated P300 effects (Herrmann et al., 2001b [social drinkers]; Lubman et al., 2008 [heroin addicts]; Lubman et al., 2007 [heroin addicts]; McDonough et al., 2001 [smokers]; Namkoong et al., 2004 [alcoholics]; Littel et al., 2007 [smokers]; van de Laar et al., 2004 [cocaine addicts]; Warren et al., 1999 [smokers]). Yet it must be noted that a substantial fraction of cue-reactivity studies with pictorial stimuli demonstrated neither N300 nor P300 cue-reactivity effects (Franken et al., 2004 [cocaine addicts]; Franken et al., 2003 [heroin addicts]; Jang et al., 2007 [smokers]; Wölfling et al., 2008 [cannabis addicts]). This heterogeneity of results might also be an indication that N300 and P300 cue-reactivity are not stable psychological phenomena in addicted populations which can perhaps be accounted for by methodological aspects. The possibility that results are skewed by methodological aspects has to be taken in account for the ERP cue-reactivity research in general. The heterogeneity of cue-reactivity evidence for N300 and P300 might partly be accounted for by different types of addiction, treatment phases, and aspects as stimulus presentation times and stimulus type. However, it remains unclear exactly which premises are needed for N300 and P300 cue-reactivity and if it can be demonstrated consistently at all. Future research is needed for clarification.

4.2. Slow positive wave between 500-850 ms

For the SPW between 500-850 ms after stimulus-onset cue-reactivity scores were again higher for objects stimuli than for persons stimuli, and cue-reactivity scores decreased from frontal to posterior. Additionally, there were limited effects of original cue-reactivity with higher smoking-non-smoking stimulus differences in smokers than in non-smokers, indicating increased higher positivity for smoking stimuli than for non-smoking stimuli in smokers. In analyses of midline and midline plus surrounding sites this effect was significant if tested one-tailed, otherwise it narrowly missed significance. However, the described SPW 500-850 ms cue-reactivity effect could not be replicated in the ANOVA of the corresponding PCA factor scores.

In a separate analysis of non-midline sites, no SPW 500-850 ms cue-reactivity effect was observable, but separate analyses of each single midline site yielded significant cue-reactivity posterior and medial, but not frontal. These cue-reactivity effects were independent from stimulus type (persons, objects). Midline cue-reactivity scores for early SPW did not correlate with post-experiment cue-reactivity scores for craving ratings, but some sporadic correlations were found between scores - mainly at Pz - and ratings for valence and arousal (most significant correlations involved objects stimuli).

Based on former studies with affective pictures (e.g. Cuthbert et al., 2000, Schupp et al., 2000, see also section 1.4.4.) that found enhanced SPW for emotional stimuli compared to neutral stimuli, in the present study larger SPW for smoking stimuli in smokers can be interpreted as a correlate of increased processing and as an indicator of higher emotional relevance due to addiction. In his review, Kok (1997) relates SPW to the allocation of attention processes and the activation of processing resources that here appear to depend on the motivational significance of the stimuli in terms of a processing/attentional bias for smoking-associated stimuli in smokers. According to Cuthbert et al. (2000) this increased allocation of attentional resources might presumably reflect the involvement of cerebral motivational systems. As outlined by Codispoti at al. (2006), the modulatory processes underlying SPW might be mandatory and seem to be not affected by habituation since in that study affective modulation remained intact through stimulus repetitions although the ERP amplitudes declined to a certain degree.

SPW cue-reactivity effects might have been limited by the tight selection of stimuli, reducing group differences of stimulus valence and arousal values, as valence and arousal are known to influence SPW amplitudes (Cuthbert et al., 2000; Amrhein et al., 2004). The sporadic correlations of SPW cue-reactivity with valence and arousal cue-reactivity scores and the failed replication of N300 and P300 cue-reactivity suggest, that ERP cue-reactivity might highly depend on stimulus valence and arousal value. As the variation of stimulus smoking association did not succeed totally independent from valence and arousal values, it remains unclear whether ERP cue-reactivity can be evoked independently from valence and arousal differences; at least cue-reactivity effects evoked like this seem to be smaller. For replication reasons (Warren et al., 1999), the study at hand used shorter stimulus presentation times than other studies exploring SPW effects which might have reduced the occurrence of higher SPW cue-reactivity. Longer presentation times might have enhanced SPW effects. Further exploration of these inter-relationships and the creation of appropriate methods for this remain the challenges of future research.

However, the demonstrated SPW cue-reactivity is in line with previous ERP studies exploring the cognitive processing of drug-associated stimuli (Franken et al., 2004; Franken et al., 2003; Littel et al., 2007; van de Laar et al., 2004; Wölfling et al., 2008). All of these studies found increased SPW in addicted subjects when confronted with drug stimuli. SPW-amplitudes might be an index of the motivational relevance of these stimuli. Future studies should verify the behavioral pertinence of these effects.

4.3. Social reference of stimuli/post-experiment ratings

Contrary to our assumptions, the social reference of stimuli did not enhance the found cue-reactivity effects in ERP or post-experimental stimulus ratings. However, the ERP showed a significant discrimination of cue-reactivity scores elicited by persons and objects stimuli, with objects stimuli having higher P362 and SPW 500-850 ms cue-reactivity scores than persons stimuli over both groups. This means that the differences between smoking and non-smoking related stimuli were higher in objects stimuli than in persons stimuli.

Additional support for a differentiation between subjects and objects stimuli and modulation of the effects of smoking relatedness was found in the post-experiment ratings for craving, valence, and arousal. In smokers, the difference in elicited craving be-

tween stimuli with and without association to smoking was higher in objects stimuli than in persons stimuli. Here again, the hypothesis of increased cue-reactivity scores for persons stimuli was disapproved. Overall, in both groups, persons stimuli received more pleasant ratings than objects stimuli. Consistent with Mucha et al. (1999) and Warren et al. (1999), smoking related stimuli received less positive ratings than non-smoking stimuli in non-smokers, while this effect was reversed in smokers with smoking-related stimuli being rated more pleasant than non-smoking stimuli. The smoking-non-smoking difference in smokers' pleasantness-ratings was more accentuated in objects stimuli than in persons stimuli, indicating again less cue-reactivity in social stimuli. Effects of differentiation between persons and objects stimuli were less definite in the arousal ratings: Effects of persons stimuli were more arousing than objects stimuli and the difference between smoking and non-smoking stimuli being bigger in objects stimuli narrowly missed significance. Social stimuli as used in the actual study are not more effective in evoking cue-reactivity than pictures of objects, the reverse seems more likely. In summary, the results indicate that social reference seems to degrade the differentiation between smoking and non-smoking stimuli: For persons stimuli smaller smoking-non-smoking differences were found for P362 and SPW 500-850 ms components, cue-reactivity in post-experiment craving ratings is higher for objects stimuli than for persons stimuli, and there is evidence that differences in pleasantness-ratings are higher in objects stimuli. A trend with similar results was found for arousal ratings. These results might be interpreted in a way that social aspects "wash out" smoking-non-smoking differentiation by adding semantic complexity or distraction. However, in the ERP domains – especially for SPW - the diminished cue-reactivity scores in persons stimuli might also be attributable to ceiling effects since, due to higher complexity, there were higher amplitudes for persons stimuli than for objects stimuli. The relevance of ceiling effects though has to be relativized since broader research (Franken et al., 2004; Franken et al., 2003; Littel et al., 2007; van de Laar et al., 2004) has found differentiations in areas of similar potential positivity.

Since arousal, and to some extent also valence of stimuli, modulate P300 and SPW amplitudes (Olofsson et al., 2008), a reduction of valence and arousal differences between smoking and non-smoking persons stimuli by "social distraction" might have minimized cue-reactivity effects which are to some extent based on arousal and valence differences. In line with these propositions, it might be hypothesized that higher P362 and SPW 500-850 ms cue-reactivity scores were found for objects stimuli than for per-

sons stimuli because of the lower stimulus complexity in objects stimuli. Emotional features might have more impact on information processing and emotional modulation might take place in a more pronounced manner when emotional aspects of the stimuli dominate the stream of incoming sensory information. Evidence for this assumption was reported by Bradley et al. (2007) who used neutral, positive, and negative pictures of high and low complexity and found that the late positive potential (LPP) at centro-parietal sites – a complex of P300 and SPW-like waves – primarily reflects the motivational relevance of stimuli. Stimulus complexity affected earlier waveforms beginning 150 ms after stimulus onset; however, the later emotional modulation in the LPP seemed to be more enhanced for the more simple stimuli.

Additional research is needed to address the effects of stimulus properties as the operationalization of complexity by Bradley et al. (2007) can not be directly applied to the stimuli in the study at hand. Furthermore, it must be stated that the category of persons stimuli – despite the assumed higher complexity of the individual stimuli - was more homogeneous in terms of perceptual and semantic variability/broadness than the category of objects stimuli.

4.4. Valence and arousal differences: Confounders of smoking association or requirements for (ERP) cue-reactivity?

As reported in section 2.2.1., this study has attempted to minimize differences in valence and arousal in the construction and selection of experimental stimuli (persons stimuli) as these are known to affect ERP-waveforms (e.g. Amrhein et al., 2004; Cuthbert et al., 2000). Concurrently an attempt was made to select smoking-associated persons stimuli that were rated as evoking preferably high craving in smokers and neutral persons stimuli that evoke minimal craving. The intention was to parallelize smoking and non-smoking stimuli for evoked arousal and valence in order to investigate as pure effects of smoking-association as possible. However, the conceptualization of valence and arousal differences as confounders lead to problems as these confounders could not be ruled out totally and the attempts to reduce these differences might have impeded cue-reactivity, especially for persons stimuli.

Smokers tend to give positive ratings to stimuli associated with the consumption of cigarettes, while non-smokers rate smoking-related stimuli negatively (e.g. Mucha et al., 1999; Warren et al., 1999; post-experiment ratings in study at hand). These differences in the perceived valence of smoking stimuli are likely to be accompanied by differences

in evoked arousal as valence and arousal are not totally independent: pleasant and unpleasant materials tend to be associated with higher ratings for arousal relative to neutral material (e.g. Bradley, Greenwald, Petry, & Lang, 1992; Bradley et al., 1994; Lang et al., 1999). In the study at hand, arousal effects in post-experiment ratings were not dependent on group affiliation but smoking stimuli were more arousing than non-smoking stimuli and persons stimuli were marginally more arousing than objects stimuli. Additionally, there was a trend for greater smoking– non-smoking differences in arousal ratings for objects stimuli than for persons stimuli which might be a result of the stimulus selection strategy for persons stimuli. This is in-line with lower cue-reactivity scores for persons stimuli in the arousal-sensitive components P362 and SPW 500-850 ms.

In ERP studies differences in arousal and valence might place limitations on interpreting the causes of potential cue-reactivity effects: different ERP-waveforms might be caused by group-specific stimulus-evoked craving as well as by differences in arousal (e.g. Cuthbert et al., 2000) and valence (e.g. Amrhein et al., 2004). However, reducing valence and arousal differences might also reduce ERP cue-reactivity effects. Differences in the emotional quality of stimuli should not only be seen as limitations but also as an original cue-reactivity effect that should not be expunged. These differences in the emotional quality might be an important precondition of ERP waveform modulation. In this latter interpretation perceived craving would *necessarily*, or at least *commonly*, be accompanied by differences in evoked valence and arousal while watching the stimuli, and a mere selection based on evoked craving alone might have been more successful for the generation of ERP cue-reactivity. This is supported by conditioning models of cue-reactivity with the central assumption that substance-associated stimuli evoke reactions of a certain emotional quality – pleasant or unpleasant. Attempts to reduce or rule out valence and arousal effects and address pure "craving" effects on ERP modulation were factitious and not appropriate to "real-life" psychic functioning. Future research should avoid improper stimuli selection strategies.

4.5. Recapitulatory conclusions

Overall, the present study provides very limited support for the argument that ERP cue-reactivity effects are found in smokers. Expected cue-reactivity for N300 and P300 components could not be demonstrated; effects for SPW were confined to a time window between 500-850 ms and did not emerge in global ANOVA analyses but at Cz and

Pz at contrast level only or if tested one-tailed. Additionally, the limited SPW cue-reactivity effects were not interrelated or not interrelated in the expected way with self-report measures of pre-experimental craving, indices of addiction or characteristics of the smoking history, and stimulus ratings for craving or emotional dimensions. Against the background of prior research (Cuthbert et al., 2000; Franken et al., 2004; Franken et al., 2003; Littel et al., 2007; Schupp et al., 2000; van de Laar et al., 2004; Wölfling et al., 2008) these small and limited SPW cue-reactivity effects can be interpreted in terms of increased processing and higher motivational relevance of smoking stimuli for smokers: smokers might allocate increased attentional resources for the processing of these relevant stimuli (Kok, 1997) and information processing might be biased for these stimuli. Cerebral motivational systems might be involved in these processes (Cuthbert et al., 2000), however, as in most other ERP cue-reactivity studies (see end of section 1.4.), no convincing relationships of ERP cue-reactivity to self-report measures of cue-reactivity were found.

In the post-experiment ratings for experimental stimuli, results were as expected and present in the current literature (e.g. Mucha et al., 1999; Warren et al., 1999): smokers' ratings for craving were higher for smoking stimuli than for non-smoking stimuli, and smokers perceived smoking stimuli as more positive than non-smoking stimuli while ratings were reversed in non-smokers.

Social reference did not affect cue-reactivity in the domain of SPW. However, for post-experiment ratings, the social reference of stimuli did unexpectedly diminish smokers' cue-reactivity since the differences between smoking and non-smoking stimuli for craving and valence were lower in persons stimuli than in objects stimuli. The assumption that the social reference of stimuli – as operationalized in the present study - would increase cue-reactivity effects seems to be incorrect, rather opposite effects might be assumed based on the effects on post-experiment ratings. Different explanations for the effects of social reference were proposed like adding semantic complexity or distraction. However, different results might be possible in a more natural setting and social effects might be more relevant at a behavioral level: Social cues or affiliation might enhance drug taking behavior and undermine attempts at abstinence (Marlatt, 1996) as these different classes of behavior and reactions are not necessarily concordant (Tiffany, 1990).

Several limitations of this study have to be regarded. First, SPW 500-850 ms cue-reactivity effects did not reach full statistical significance in the overall analyses. This might be a result of low statistical power because of small sample sizes. Comparable experiments, however, demonstrated significant cue-reactivity effects with similar or smaller sample sizes. Nonetheless, the statistical power might have been insufficient to detect modulatory effects of social reference on the SPW cue-reactivity. Generally, for the evocation of SPW effects, longer stimulus presentations times might be helpful, shorter times were used in order to replicate McDonough's and Warren's experiments (2001; Warren et al., 1999).

Second, certain factors may have diminished potential ERP cue-reactivity effects in smokers. An insufficient impact of the stressor task was discussed although an anteceding stressor is not indispensable for ERP cue-reactivity. Though a simultaneous application of stress which according to Baker et al. (1986) induces negative-affect craving, and availability inducing pre-information, which might create positive-affect craving, could lead to a mutual inhibition of both craving systems. However, to our knowledge empirical support for an assumption that a parallel activation of perceived availability and stress reduces cue-reactivity in comparison to both conditions in isolation does not exist. Alternatively, perceived availability could be a potential signal or discriminative stimulus for relieving effects of drug consumption in a negative affective state. Further research is needed to clarify the exact impact of parallel stressor and drug availability inductions.

Special aspects in the construction and selection of experimental stimuli might also have undermined the occurrence of ERP cue-reactivity: the admission of social reference might have inflated the semantic space and distracted from smoking-association, the attempts to parallelize SP and NP stimuli for valence and arousal might have ruled out inevitable differences for cue-reactivity, and higher complexity of persons stimuli led to higher SPW amplitudes and ceiling effects might have reduced the opportunity for cue-reactivity to occur.

Third, certain unspecified factors resulted in substantial cue-reactivity scores in non-smokers and diminished differences to smokers. This might have been caused by the salience of the smoking theme even for non-smokers so that smoking stimuli might have been perceived as targets. Moreover, the extreme lack of stimulation by non-smoking stimuli -especially NO stimuli seemed to be exceptionally boring – might have led to a relative attractiveness of smoking-associated stimuli.

The outlined limitations and questions provide manifold opportunity and need for future research.

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Bedienungsanleitung des EC50 MICRO III Smokerlizer, Handmessgerät zur Bestimmung des Kohlenmonoxidgehaltes in der ausgeatmeten Luft.

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6. Appendix A: Tables and figures

Table A-1: Distribution of resulting pretest-sample to the pretest subsets and sequences

Name of set	*n*	Composition			
		Smoking status	Gender		Total
			male	female	
set 1 fwd	21	smokers	2	4	6
		non-smokers	6	9	15
set 1 bwd	19	smokers	7	4	11
		non-smokers	3	5	8
set 2 fwd	11	smokers	3	2	5
		non-smokers	2	4	6
set 2 bwd	9	smokers	1	5	6
		non-smokers	2	1	3

fwd = forwards, bwd = backwards.

Annotations for the tables A-2 to A-7 on the following pages: These tables contain the pretest ratings for social stimuli obtained from smokers (n = 28) and non-smokers (n = 32). Subjects (N = 60) rated stimuli for evoked craving, arousal, and valence (all nine-point rating scales, see section 2.2.1.). Higher rating values indicate higher craving/higher arousal; valence ratings below five indicate unpleasant values, above five they are pleasant. Ratings from the forward and the backward presentation order of each set were conflated. The notations of the pictures contain information about the picture set (A = set 1, B = set 2) that had been used, and the smoking reference of the pictures (S = picture with a smoking person, NS = picture with a person not smoking). Pictures selected from the pretest for the main-experiment are indicated by bold type.

Table A-2: Pretest ratings of pictures of smoking persons for craving

Craving rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_02S	3.24 (2.39)	1.00-9.00	1.00 (0.00)	1.00-1.00
A_03S	**3.47 (1.97)**	**1.00-6.00**	**1.09 (0.29)**	**1.00-2.00**
A_06S	**3.29 (2.23)**	**1.00-8.00**	**1.09 (0.29)**	**1.00-2.00**
A_07S	2.71 (1.45)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_10S	2.82 (1.88)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_11S	2.59 (1.84)	1.00-6.00	1.09 (0.29	1.00-2.00
A_14S	**3.06 (2.16)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
A_15S	**3.47 (2.35)**	**1.00-8.00**	**1.09 (0.29)**	**1.00-2.00**
A_18S	**3.41 (2.15)**	**1.00-6.00**	**1.09 (0.29)**	**1.00-2.00**
A_19S	**3.71 (2.02)**	**1.00-8.00**	**1.09 (0.29)**	**1.00-2.00**
A_22S	**3.88 (2.57)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
A_23S	**3.00 (1.73)**	**1.00-6.00**	**1.17 (0.58)**	**1.00-3.00**
A_26S	**3.24 (2.14)**	**1.00-7.00**	**1.00 (0.00)**	**1.00-1.00**
A_27S	**3.24 (2.28)**	**1.00-7.00**	**1.00 (0.00)**	**1.00-1.00**
A_30S	**2.88 (2.23)**	**1.00-7.00**	**1.17 (0.49)**	**1.00-3.00**
A_31S	3.24 (2.14)	1.00-9.00	1.00 (0.00)	1.00-1.00
A_34S	**3.71 (2.52)**	**1.00-8.00**	**1.17 (0.58)**	**1.00-3.00**
A_35S	3.12 (1.90)	1.00-7.00	1.00 (0.00)	1.00-1.00
A_38S	2.59 (1.62)	1.00-5.00	1.00 (0.00)	1.00-1.00
A_39S	1.47 (0.94)	1.00-4.00	1.09 (0.29)	1.00-2.00
A_41S	2.65 (1.62)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_43S	1.71 (1.21)	1.00-4.00	1.00 (0.00)	1.00-1.00
A_45S	2.88 (2.06)	1.00-8.00	1.00 (0.00)	1.00-1.00
A_50S	2.00 (1.66)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_02S	2.55 (1.37)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_03S	1.64 (1.50)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_06S	2.36 (1.96)	1.00-7.00	1.00 (0.00)	1.00-1.00
B_07S	2.09 (1.22)	1.00-5.00	1.00 (0.00)	1.00-1.00

Craving rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
B_09S	2.00 (1.34)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_10S	2.36 (1.91)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_11S	1.64 (1.12)	1.00-4.00	1.00 (0.00)	1.00-1.00
B_13S	1.91 (1.92)	1.00-7.00	1.00 (0.00)	1.00-1.00
B_14S	2.36 (1.75)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_16S	2.27 (1.68)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_17S	3.27 (2.10)	1.00-8.00	1.00 (0.00)	1.00-1.00
B_19S	2.82 (1.83)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_20S	**2.82 (2.23)**	**1.00-7.00**	**1.00 (0.00)**	**1.00-1.00**
B_22S	2.45 (1.37)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_24S	**3.09 (2.39)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
B_25S	2.64 (1.50)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_28S	2.64 (1.86)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_29S	**3.55 (2.07)**	**1.00-7.00**	**1.00 (0.00)**	**1.00-1.00**
B_32S	**3.36 (1.80)**	**1.00-7.00**	**1.00 (0.00)**	**1.00-1.00**
B_33S	**2.82 (2.27)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
B_36S	3.27 (2.15)	1.00-8.00	1.00 (0.00)	1.00-1.00
B_37S	1.64 (1.03)	1.00-4.00	1.00 (0.00)	1.00-1.00
B_40S	2.45 (2.25)	1.00-8.00	1.00 (0.00)	1.00-1.00
B_41S	2.36 (1.80)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_44S	2.73 (1.74)	1.00-7.00	1.00 (0.00)	1.00-1.00
B_45S	**3.09 (1.97)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
B_48S	3.18 (2.14)	1.00-8.00	1.00 (0.00)	1.00-1.00
B_49S	2.27 (2.15)	1.00-8.00	1.00 (0.00)	1.00-1.00

Table A-3: Pretest ratings of non-smoking persons pictures for craving

Craving rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_01NS	**2.00 (1.62)**	**1.00-6.00**	**1.00 (0.00)**	**1.00-1.00**
A_04NS	1.71 (1.05)	1.00-4.00	1.00 (0.00)	1.00-1.00
A_05NS	1.88 (1.54)	1.00-5.00	1.00 (0.00)	1.00-1.00
A_08NS	**2.06 (2.19)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
A_09NS	**2.00 (1.70)**	**1.00-6.00**	**1.09 (0.00)**	**1.00-1.00**
A_12NS	2.12 (1.83)	1.00-7.00	1.00 (0.29)	1.00-2.00
A_13NS	**1.65 (1.22)**	**1.00-6.00**	**1.00 (0.00)**	**1.00-1.00**
A_16NS	1.76 (1.44)	1.00-5.00	1.00 (0.00)	1.00-1.00
A_17NS	1.82 (1.38)	1.00-5.00	1.00 (0.00)	1.00-1.00
A_20NS	**1.71 (1.16)**	**1.00-4.00**	**1.00 (0.00)**	**1.00-1.00**
A_21NS	1.88 (1.54)	1.00-5.00	1.00 (0.00)	1.00-1.00
A_24NS	**1.71 (1.31)**	**1.00-5.00**	**1.00 (0.00)**	**1.00-1.00**
A_25NS	2.06 (1.82)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_28NS	1.88 (1.50)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_29NS	2.53 (1.62)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_32NS	**2.35 (1.41)**	**1.00-6.00**	**1.00 (0.00)**	**1.00-1.00**
A_33NS	**1.65 (1.22)**	**1.00-5.00**	**1.00 (0.00)**	**1.00-1.00**
A_36NS	**1.65 (1.06)**	**1.00-4.00**	**1.09 (0.00)**	**1.00-1.00**
A_37NS	3.12 (2.42)	1.00-8.00	1.00 (0.29)	1.00-2.00
A_40NS	2.41 (1.80)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_42NS	2.71 (1.99)	1.00-7.00	1.00 (0.00)	1.00-1.00
A_44NS	2.24 (1.48)	1.00-5.00	1.00 (0.00)	1.00-1.00
A_46NS	2.24 (1.86)	1.00-6.00	1.00 (0.00)	1.00-1.00
A_47NS	**1.88 (1.50)**	**1.00-6.00**	**1.00 (0.00)**	**1.00-1.00**
A_48NS	2.35 (1.90)	1.00-7.00	1.00 (0.00)	1.00-1.00
A_49NS	**2.59 (2.15)**	**1.00-8.00**	**1.00 (0.00)**	**1.00-1.00**
B_01NS	**1.64 (1.12)**	**1.00-4.00**	**1.00 (0.00)**	**1.00-1.00**
B_04NS	1.91 (1.30)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_05NS	2.00 (1.55)	1.00-6.00	1.00 (0.00)	1.00-1.00

Craving rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
B_08NS	2.00 (1.79)	1.00-7.00	1.00 (0.00)	1.00-1.00
B_12NS	1.73 (1.56)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_15NS	**1.64 (1.03)**	**1.00-4.00**	**1.00 (0.00)**	**1.00-1.00**
B_18NS	1.73 (1.01)	1.00-4.00	1.00 (0.00)	1.00-1.00
B_21NS	2.55 (2.11)	1.00-7.00	1.00 (0.00)	1.00-1.00
B_23NS	**1.64 (0.81)**	**1.00-3.00**	**1.00 (0.00)**	**1.00-1.00**
B_26NS	1.73 (1.27)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_27NS	2.18 (1.40)	1.005.00	1.00 (0.00)	1.00-1.00
B_30NS	**2.09 (1.22)**	**1.00-4.00**	**1.00 (0.00)**	**1.00-1.00**
B_31NS	**1.55 (0.82)**	**1.00-3.00**	**1.00 (0.00)**	**1.00-1.00**
B_34NS	2.00 (1.10)	1.00-4.00	1.00 (0.00)	1.00-1.00
B_35NS	2.27 (1.62)	1.00-6.00	1.00 (0.00)	1.00-1.00
B_38NS	**1.64 (1.03)**	**1.00-4.00**	**1.00 (0.00)**	**1.00-1.00**
B_39NS	2.18 (1.33)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_42NS	2.00 (1.41)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_43NS	1.73 (0.90)	1.00-3.00	1.00 (0.00)	1.00-1.00
B_46NS	1.73 (0.90)	1.00-3.00	1.00 (0.00)	1.00-1.00
B_47NS	2.00 (1.41)	1.00-5.00	1.00 (0.00)	1.00-1.00
B_50NS	**1.64 (0.81)**	**1.00-3.00**	**1.00 (0.00)**	**1.00-1.00**

Table A-4: Pretest ratings of pictures of smoking persons for arousal

Arousal rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_02S	6.53 (2.15)	2.00-9.00	5.65 (2.14)	1.00-9.00
A_03S	**6.59 (1.84)**	**3.00-9.00**	**5.52 (2.31)**	**1.00-9.00**
A_06S	**6.29 (2.31)**	**2.00-9.00**	**5.87 (2.18)**	**1.00-9.00**
A_07S	6.41 (1.54)	4.00-9.00	6.35 (2.23)	3.00-9.00

Arousal rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_10S	6.47 (1.84)	3.00-9.00	5.78 (2.47)	1.00-9.00
A_11S	6.82 (1.78)	3.00-9.00	5.96 (2.34)	2.00-9.00
A_14S	**6.47 (2.37)**	**2.00-9.00**	**4.87 (2.36)**	**1.00-9.00**
A_15S	**6.00 (1.94)**	**3.00-9.00**	**6.09 (2.13)**	**2.00-9.00**
A_18S	**5.94 (1.92)**	**2.00-9.00**	**5.61 (2.46)**	**1.00-9.00**
A_19S	**5.94 (1.85)**	**3.00-9.00**	**5.91 (2.33)**	**1.00-9.00**
A_22S	**6.41 (1.94)**	**3.00-9.00**	**6.13 (2.44)**	**2.00-9.00**
A_23S	**6.71 (1.57)**	**4.00-9.00**	**5.61 (2.08)**	**2.00-9.00**
A_26S	**6.41 (1.54)**	**4.00-9.00**	**5.65 (2.21)**	**2.00-9.00**
A_27S	**6.35 (2.03)**	**3.00-9.00**	**5.83 (2.17)**	**1.00-9.00**
A_30S	**5.94 (2.01)**	**2.00-9.00**	**6.57 (2.00)**	**2.00-9.00**
A_31S	6.94 (1.89)	3.00-9.00	5.83 (2.21)	2.00-9.00
A_34S	**6.41 (1.94)**	**3.00-9.00**	**6.17 (2.27)**	**2.00-9.00**
A_35S	6.35 (1.58)	3.00-9.00	5.78 (2.32)	1.00-9.00
A_38S	6.24 (1.92)	3.00-9.00	5.30 (2.30)	1.00-9.00
A_39S	6.35 (2.06)	3.00-9.00	6.78 (1.91)	3.00-9.00
A_41S	6.76 (1.79)	3.00-9.00	5.74 (2.45)	1.00-9.00
A_43S	6.76 (1.71)	3.00-9.00	6.61 (1.88)	3.00-9.00
A_45S	6.41 (1.94)	3.00-9.00	5.74 (2.16)	2.00-9.00
A_50S	7.06 (1.98)	2.00-9.00	6.39 (2.21)	2.00-9.00
B_02S	6.09 (1.30)	4.00-8.00	5.44 (2.19)	2.00-9.00
B_03S	6.27 (2.00)	3.00-9.00	5.44 (2.01)	3.00-9.00
B_06S	7.27 (1.27)	5.00-9.00	6.44 (1.74)	5.00-9.00
B_07S	6.64 (1.29)	5.00-9.00	5.78 (2.11)	3.00-9.00
B_09S	6.09 (1.81)	3.00-9.00	5.56 (1.81)	3.00-9.00
B_10S	6.64 (1.75)	3.00-9.00	6.00 (1.87)	4.00-9.00
B_11S	6.82 (1.78)	4.00-9.00	6.56 (1.74)	5.00-9.00
B_13S	6.09 (2.02)	3.00-9.00	4.89 (2.71)	1.00-9.00
B_14S	6.82 (1.83)	4.00-9.00	6.33 (1.80)	5.00-9.00
B_16S	6.55 (1.75)	4.00-9.00	6.11 (1.76)	5.00-9.00

Arousal rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
B_17S	6.45 (1.86)	4.00-9.00	5.56 (2.51)	2.00-9.00
B_19S	6.45 (2.54)	3.00-9.00	5.89 (1.76)	4.00-9.00
B_20S	**6.55 (1.51)**	**4.00-9.00**	**5.67 (2.24)**	**3.00-9.00**
B_22S	6.18 (1.66)	3.00-9.00	5.89 (1.96)	4.00-9.00
B_24S	**6.55 (1.75)**	**4.00-9.00**	**6.00 (2.06)**	**4.00-9.00**
B_25S	6.82 (1.72)	3.00-9.00	4.89 (2.76)	2.00-9.00
B_28S	7.27 (1.49)	4.00-9.00	6.00 (2.35)	3.00-9.00
B_29S	**6.45 (1.37)**	**4.00-9.00**	**5.78 (2.05)**	**4.00-9.00**
B_32S	**6.09 (1.51)**	**3.00-8.00**	**6.00 (2.06)**	**4.00-9.00**
B_33S	**7.18 (1.25)**	**5.00-9.00**	**5.89 (2.15)**	**4.00-9.00**
B_36S	6.36 (1.63)	4.00-9.00	5.78 (1.64)	4.00-9.00
B_37S	6.55 (1.75)	4.00-9.00	5.33 (2.74)	2.00-9.00
B_40S	7.36 (1.69)	4.00-9.00	6.00 (2.06)	4.00-9.00
B_41S	7.00 (1.67)	4.00-9.00	6.67 (1.73)	5.00-9.00
B_44S	5.91 (2.30)	2.00-9.00	6.22 (1.86)	5.00-9.00
B_45S	**6.73 (1.90)**	**4.00-9.00**	**6.00 (2.06)**	**4.00-9.00**
B_48S	6.27 (1.85)	3.00-8.00	5.67 (2.06)	3.00-9.00
B_49S	6.73 (1.49)	5.00-9.00	6.44 (1.74)	4.00-9.00

Table A-5: Pretest ratings of pictures of non-smoking persons for arousal

Arousal rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_01NS	**6.24 (2.05)**	**3.00-9.00**	**6.83 (1.72)**	**5.00-9.00**
A_04NS	6.94 (1.64)	5.00-9.00	6.83 (2.06)	3.00-9.00
A_05NS	6.94 (1.64)	4.00-9.00	6.96 (1.87)	4.00-9.00
A_08NS	**7.29 (1.57)**	**5.00-9.00**	**7.13 (1.84)**	**4.00-9.00**
A_09NS	**6.59 (1.70)**	**4.00-9.00**	**7.04 (1.89)**	**3.00-9.00**

Arousal rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_12NS	6.59 (1.77)	3.00-9.00	6.52 (2.33)	1.00-9.00
A_13NS	**6.59 (2.06)**	**3.00-9.00**	**7.00 (2.11)**	**3.00-9.00**
A_16NS	6.65 (1.84)	3.00-9.00	6.83 (1.70)	5.00-9.00
A_17NS	6.29 (2.17)	3.00-9.00	6.65 (1.87)	2.00-9.00
A_20NS	**6.88 (1.41)**	**5.00-9.00**	**6.87 (2.40)**	**1.00-9.00**
A_21NS	6.59 (1.66)	4.00-9.00	6.43 (2.17)	2.00-9.00
A_24NS	**6.29 (1.72)**	**4.00-9.00**	**6.96 (1.82)**	**3.00-9.00**
A_25NS	6.41 (1.80)	4.00-9.00	6.87 (2.14)	1.00-9.00
A_28NS	6.59 (2.00)	3.00-9.00	6.74 (2.16)	3.00-9.00
A_29NS	*6.71 (1.49)*	4.00-9.00	5.74 (2.36)	1.00-9.00
A_32NS	**6.94 (1.60)**	**5.00-9.00**	**6.17 (2.23)**	**1.00-9.00**
A_33NS	**7.18 (1.74)**	**5.00-9.00**	**7.04 (2.10)**	**2.00-9.00**
A_36NS	**6.94 (1.75)**	**5.00-9.00**	**7.00 (2.00)**	**3.00-9.00**
A_37NS	6.94 (1.56)	5.00-9.00	5.96 (2.03)	2.00-9.00
A_40NS	6.29 (2.02)	4.00-9.00	5.83 (2.17)	2.00-9.00
A_42NS	5.94 (2.14)	2.00-9.00	5.35 (2.10)	1.00-9.00
A_44NS	6.24 (1.95)	3.00-9.00	6.00 (2.13)	3.00-9.00
A_46NS	6.53 (2.29)	3.00-9.00	6.65 (1.94)	3.00-9.00
A_47NS	**6.41 (2.45)**	**1.00-9.00**	**6.35 (2.04)**	**2.00-9.00**
A_48NS	6.29 (2.05)	4.00-9.00	5.70 (2.58)	1.00-9.00
A_49NS	**6.00 (2.12)**	**1.00-9.00**	**5.57 (2.15)**	**1.00-9.00**
B_01NS	**6.91 (1.64)**	**5.00-9.00**	**6.00 (2.12)**	**3.00-9.00**
B_04NS	6.64 (2.06)	4.00-9.00	6.00 (2.29)	2.00-9.00
B_05NS	7.09 (1.38)	4.00-9.00	5.67 (2.06)	2.00-9.00
B_08NS	7.00 (1.90)	3.00-9.00	6.00 (1.94)	4.00-9.00
B_12NS	6.73 (1.74)	4.00-9.00	6.67 (2.00)	5.00-9.00
B_15NS	**7.64 (1.63)**	**4.00-9.00**	**6.11 (1.76)**	**5.00-9.00**
B_18NS	6.73 (2.15)	3.00-9.00	6.67 (2.00)	5.00-9.00
B_21NS	7.18 (1.60)	4.00-9.00	6.44 (1.94)	5.00-9.00
B_23NS	**6.73 (1.42)**	**4.00-9.00**	**5.78 (2.17)**	**3.00-9.00**

Arousal rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
B_26NS	7.55 (1.63)	5.00-9.00	6.22 (1.86)	5.00-9.00
B_27NS	7.18 (1.40)	5.00-9.00	6.67 (1.66)	5.00-9.00
B_30NS	**6.73 (1.85)**	**4.00-9.00**	**6.11 (2.09)**	**3.00-9.00**
B_31NS	**7.55 (1.63)**	**5.00-9.00**	**7.22 (1.92)**	**5.00-9.00**
B_34NS	6.73 (1.62)	4.00-9.00	6.44 (1.74)	5.00-9.00
B_35NS	6.73 (1.95)	3.00-9.00	7.00 (2.29)	3.00-9.00
B_38NS	**7.00 (1.95)**	**4.00-9.00**	**6.22 (1.72)**	**5.00-9.00**
B_39NS	7.18 (1.66)	4.00-9.00	5.89 (2.03)	4.00-9.00
B_42NS	6.82 (1.60)	4.00-9.00	7.22 (1.86)	5.00-9.00
B_43NS	7.09 (1.45)	5.00-9.00	5.89 (2.15)	4.00-9.00
B_46NS	7.00 (1.73)	3.00-9.00	5.89 (2.20)	2.00-9.00
B_47NS	7.45 (1.81)	4.00-9.00	6.33 (1.41)	5.00-9.00
B_50NS	**7.45 (1.37)**	**5.00-9.00**	**6.33 (1.80)**	**5.00-9.00**

Table A-6: Pretest ratings of pictures of smoking persons for valence

Valence rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_02S	5.29 (1.65)	2.00-7.00	4.91 (1.08)	3.00-8.00
A_03S	**5.29 (1.26)**	**3.00-7.00**	**4.17 (1.92)**	**3.00-9.00**
A_06S	**5.71 (1.61)**	**1.00-7.00**	**4.35 (1.40)**	**3.00-9.00**
A_07S	4.76 (1.20)	3.00-8.00	3.96 (1.22)	5.00-8.00
A_10S	3.65 (1.80)	3.00-9.00	3.70 (1.40)	5.00-9.00
A_11S	4.53 (1.66)	3.00-9.00	4.61 (1.64)	3.00-8.00
A_14S	**4.35 (1.93)**	**2.00-9.00**	**3.52 (2.27)**	**2.00-9.00**
A_15S	**5.94 (1.75)**	**2.00-8.00**	**5.04 (1.82)**	**1.00-8.00**
A_18S	**5.06 (1.60)**	**2.00-8.00**	**4.00 (1.65)**	**3.00-9.00**
A_19S	**5.24 (1.75)**	**2.00-8.00**	**4.09 (1.50)**	**4.00-9.00**

Valence rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_22S	**5.41 (1.70)**	**3.00-8.00**	**4.17 (1.47)**	**2.00-8.00**
A_23S	**5.12 (1.54)**	**3.00-7.00**	**3.87 (1.66)**	**3.00-9.00**
A_26S	**4.88 (1.65)**	**3.00-9.00**	**4.22 (1.35)**	**3.00-8.00**
A_27S	**5.41 (1.23)**	**3.00-7.00**	**3.91 (1.47)**	**4.00-9.00**
A_30S	**4.76 (1.60)**	**1.00-7.00**	**4.96 (1.40)**	**3.00-7.00**
A_31S	5.18 (1.67)	3.00-7.00	4.30 (1.29)	3.00-8.00
A_34S	**5.88 (1.69)**	**2.00-8.00**	**4.74 (1.63)**	**2.00-8.00**
A_35S	4.65 (1.66)	3.00-9.00	3.61 (1.64)	3.00-9.00
A_38S	3.94 (1.75)	3.00-9.00	3.43 (1.78)	3.00-9.00
A_39S	4.06 (1.75)	3.00-9.00	4.61 (1.31)	3.00-9.00
A_41S	4.65 (1.46)	3.00-8.00	4.22 (1.09)	5.00-8.00
A_43S	4.94 (1.98)	1.00-8.00	5.17 (1.44)	2.00-8.00
A_45S	4.00 (1.46)	3.00-8.00	4.09 (1.70)	3.00-9.00
A_50S	4.47 (1.55)	3.00-9.00	4.83 (1.61)	2.00-9.00
B_02S	5.73 (0.90)	3.00-6.00	5.56 (3.03)	2.00-7.00
B_03S	4.09 (1.51)	3.00-8.00	3.89 (5.11)	3.00-9.00
B_06S	4.55 (1.21)	3.00-7.00	3.78 (1.69)	5.00-8.00
B_07S	5.18 (0.87)	3.00-6.00	3.44 (3.78)	5.00-9.00
B_09S	5.55 (1.21)	3.00-7.00	4.44 (7.78)	2.00-9.00
B_10S	4.82 (0.98)	4.00-7.00	3.00 (2.75)	5.00-9.00
B_11S	4.45 (0.82)	5.00-7.00	4.00 (3.50)	3.00-8.00
B_13S	3.91 (1.64)	3.00-8.00	3.00 (3.25)	5.00-9.00
B_14S	5.82 (0.98)	2.00-5.00	4.44 (3.03)	3.00-8.00
B_16S	4.91 (1.38)	2.00-7.00	3.22 (1.69)	5.00-8.00
B_17S	5.73 (1.10)	3.00-7.00	5.00 (1.50)	4.00-8.00
B_19S	4.91 (1.45)	2.00-7.00	5.44 (5.78)	1.00-8.00
B_20S	**5.09 (0.54)**	**4.00-6.00**	**3.78 (3.44)**	**3.00-8.00**
B_22S	5.82 (1.17)	2.00-6.00	3.89 (2.11)	5.00-8.00
B_24S	**5.45 (0.93)**	**3.00-6.00**	**3.67 (3.50)**	**4.00-9.00**
B_25S	4.64 (0.92)	4.00-7.00	4.33 (4.50)	1.00-8.00

Valence rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
B_28S	5.09 (0.70)	3.00-6.00	3.67 (1.50)	5.00-8.00
B_29S	**6.00 (1.48)**	**2.00-7.00**	**3.22 (2.69)**	**5.00-9.00**
B_32S	**5.64 (1.21)**	**3.00-7.00**	**3.89 (2.86)**	**5.00-9.00**
B_33S	**5.18 (0.75)**	**3.00-6.00**	**3.56 (2.53)**	**5.00-9.00**
B_36S	5.82 (1.40)	2.00-7.00	5.11 (8.86)	1.00-9.00
B_37S	5.36 (1.50)	1.00-6.00	5.33 (2.50)	1.00-7.00
B_40S	4.82 (0.98)	4.00-7.00	4.33 (3.75)	4.00-9.00
B_41S	5.09 (0.94)	4.00-7.00	4.00 (0.75)	5.00-7.00
B_44S	5.05 (0.93)	3.00-6.00	4.56 (1.78)	3.00-7.00
B_45S	**5.73 (1.10)**	**3.00-6.00**	**3.89 (1.61)**	**5.00-8.00**
B_48S	5.91 (1.04)	2.00-5.00	4.33 (2.00)	4.00-7.00
B_49S	4.82 (0.98)	3.00-7.00	3.78 (1.94)	5.00-9.00

Table A-7: Pretest ratings of pictures of non-smoking persons for valence

Valence rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_01NS	**5.29 (1.65)**	**2.00-7.00**	**4.91 (1.08)**	**3.00-8.00**
A_04NS	4.18 (1.67)	3.00-9.00	4.78 (1.54)	2.00-8.00
A_05NS	4.76 (1.79)	2.00-7.00	4.83 (1.37)	2.00-7.00
A_08NS	**4.65 (1.41)**	**3.00-8.00**	**4.91 (1.08)**	**3.00-8.00**
A_09NS	**4.65 (1.69)**	**3.00-9.00**	**4.74 (1.14)**	**3.00-8.00**
A_12NS	5.65 (1.41)	1.00-7.00	6.00 (1.31)	1.00-6.00
A_13NS	**5.12 (2.09)**	**2.00-9.00**	**5.22 (1.13)**	**2.00-7.00**
A_16NS	5.12 (1.22)	3.00-7.00	5.26 (1.21)	1.00-7.00
A_17NS	5.29 (1.69)	3.00-9.00	4.91 (1.44)	2.00-8.00
A_20NS	**4.18 (1.33)**	**3.00-9.00**	**4.39 (1.20)**	**4.00-9.00**
A_21NS	4.65 (1.27)	3.00-7.00	5.78 (1.38)	1.00-6.00

Valence rating for picture	Smokers (n = 28)		Non-smokers (n = 32)	
	M (*SD*)	Range	*M* (*SD*)	Range
A_24NS	**4.18 (1.81)**	**3.00-9.00**	**4.70 (0.88)**	**4.00-7.00**
A_25NS	4.82 (1.29)	3.00-7.00	4.91 (1.38)	3.00-9.00
A_28NS	4.65 (1.62)	2.00-7.00	5.96 (1.49)	2.00-7.00
A_29NS	5.12 (1.90)	3.00-9.00	4.35 (1.30)	4.00-9.00
A_32NS	**4.41 (1.77)**	**2.00-9.00**	**3.78 (1.28)**	**4.00-9.00**
A_33NS	**4.35 (1.90)**	**3.00-9.00**	**4.35 (1.72)**	**2.00-9.00**
A_36NS	**4.35 (1.37)**	**3.00-8.00**	**4.78 (1.00)**	**2.00-7.00**
A_37NS	5.65 (1.27)	3.00-7.00	5.04 (1.55)	2.00-8.00
A_40NS	4.41 (1.77)	3.00-8.00	4.22 (1.00)	4.00-8.00
A_42NS	5.18 (1.78)	1.00-7.00	4.78 (2.07)	1.00-9.00
A_44NS	5.18 (1.55)	2.00-7.00	4.57 (1.38)	4.00-8.00
A_46NS	4.94 (1.71)	2.00-7.00	6.13 (1.66)	1.00-8.00
A_47NS	**4.59 (1.97)**	**1.00-9.00**	**4.91 (1.56)**	**3.00-9.00**
A_48NS	4.18 (1.85)	2.00-9.00	4.00 (1.62)	4.00-9.00
A_49NS	**5.29 (1.72)**	**1.00-7.00**	**4.22 (1.98)**	**1.00-9.00**
B_01NS	**4.64 (0.67)**	**5.00-7.00**	**5.00 (2.06)**	**1.00-8.00**
B_04NS	4.27 (1.35)	4.00-8.00	5.44 (1.42)	1.00-6.00
B_05NS	5.27 (1.19)	3.00-7.00	5.56 (1.94)	1.00-7.00
B_08NS	5.36 (1.12)	3.00-6.00	5.11 (1.17)	3.00-7.00
B_12NS	4.36 (0.92)	4.00-7.00	4.56 (1.01)	4.00-7.00
B_15NS	**5.18 (1.08)**	**2.00-6.00**	**4.78 (0.67)**	**5.00-7.00**
B_18NS	4.55 (1.04)	4.00-7.00	5.00 (1.41)	3.00-7.00
B_21NS	4.55 (1.13)	4.00-8.00	4.89 (0.60)	4.00-6.00
B_23NS	**5.55 (1.44)**	**3.00-8.00**	**6.11 (1.45)**	**2.00-7.00**
B_26NS	4.64 (0.50)	5.00-6.00	4.89 (0.33)	5.00-6.00
B_27NS	4.64 (1.03)	3.00-7.00	4.33 (1.41)	4.00-8.00
B_30NS	**5.73 (0.79)**	**3.00-5.00**	**5.44 (0.88)**	**3.00-6.00**
B_31NS	**4.64 (0.67)**	**5.00-7.00**	**4.78 (0.44)**	**5.00-6.00**
B_34NS	5.36 (0.67)	4.00-6.00	5.00 (0.87)	4.00-7.00
B_35NS	5.45 (1.44)	1.00-7.00	5.33 (0.71)	3.00-5.00

Valence rating for picture	**Smokers** (n = 28)		**Non-smokers** (n = 32)	
	M (SD)	Range	M (SD)	Range
B_38NS	**5.36 (1.50)**	**1.00-6.00**	**5.33 (1.58)**	**1.00-7.00**
B_39NS	5.82 (0.98)	3.00-5.00	6.44 (1.33)	1.00-5.00
B_42NS	5.09 (0.70)	3.00-6.00	5.33 (1.41)	3.00-7.00
B_43NS	5.00 (1.18)	3.00-7.00	5.33 (0.50)	4.00-5.00
B_46NS	5.27 (0.79)	3.00-6.00	5.11 (0.78)	3.00-6.00
B_47NS	4.82 (1.54)	2.00-7.00	3.78 (1.72)	3.00-8.00
B_50NS	**5.27 (1.10)**	**3.00-6.00**	**5.22 (0.44)**	**4.00-5.00**

Table A-8: Estimated marginal means of post-experiment craving ratings for experimental stimuli given by smokers (n = 23) as a function of "smoking content" and "social content"

Stimuli		M (SE)
Smoking content	Social content	
smoking	persons	5.30 (0.44)
	objects	5.55 (0.45)
non-smoking	persons	3.14 (0.38)
	objects	2.72 (0.41)

Table A-9: Estimated marginal means of post-experiment valence ratings for experimental stimuli as a function of "group" and "smoking content"/"social content" of experimental stimuli

Group	Smoking content	Social content	M	SE
smokers	smoking	persons	5.27	0.23
		objects	5.13	0.29
	non-smoking	persons	4.93	0.20
		objects	4.26	0.29
non-smokers	smoking	persons	4.19	0.26
		objects	3.44	0.33
	non-smoking	persons	5.33	0.23
		objects	4.81	0.32

See also figure 2 in section 3.3.

Table A-10: Estimated marginal means of SPW 500-850 ms cue-reactivity scores as a function of "stimulus type" and "midline site"

Midline site	Stimulus type	
	persons	objects
	M (*SE*)	*M* (*SE*)
Fz	180.03 (102.87)	773.33 (102.16)
Cz	168.14 (100.15)	563.47 (106.27)
Pz	60.95 (103.13)	247.87 (101.58)

Table A-11: Estimated marginal means of cue-reactivity scores of 500-850 ms SPW wave as a function of "midline site" and "group" and "stimulus type"

Midline site	Group	Stimulus type	*M*	*SE*
Fz	smokers	persons	285.53	136.76
		objects	816.05	135.82
	non-smokers	persons	74.54	153.70
		objects	730.62	152.65
Cz	smokers	persons	336.57	133.15
		objects	749.35	141.28
	non-smokers	persons	-0.29	149.64
		objects	377.59	158.78
Pz	smokers	persons	251.00	137.10
		objects	396.98	135.05
	non-smokers	persons	-129.10	154.09
		objects	98.77	151.78

Annotations for the figures A-1 to A-4 on the following pages: Figures A-1 to figure A-4 demonstrate that artifact affected data (reversed polarity in channels A1 and A2, see section 2.6.) can be identified with certainty by the polarity of blink artifacts in channels A1 and A2.

In the raw data *free* from the reversed polarity artifact, blinks are positive (downwards) in the VEOG while the corresponding physiological blink artifacts in the EEG channels A1 and A2 are negative (upwards) (see figures A-1 and A-2).

In raw data *affected* by the reversed polarity artifact blinks are still positive (downwards) in the VEOG while the corresponding physiological blink artifacts in the polarity shift artifact affected EEG channels A1 and A2 are also positive (downwards) (see figures A-3 and A-4). This aberration enables a reliable identification of polarity shift artifact affected data sets.

Figure A-1: VEOG raw data of non-smoker N01 (not affected by polarity shift artifact) showing ten seconds beginning from 2 minutes and 11 seconds in the data set, for facility of inspection reasons the EOG amplitudes in this figure are scaled smaller than the corresponding EEG data in figure A-2:

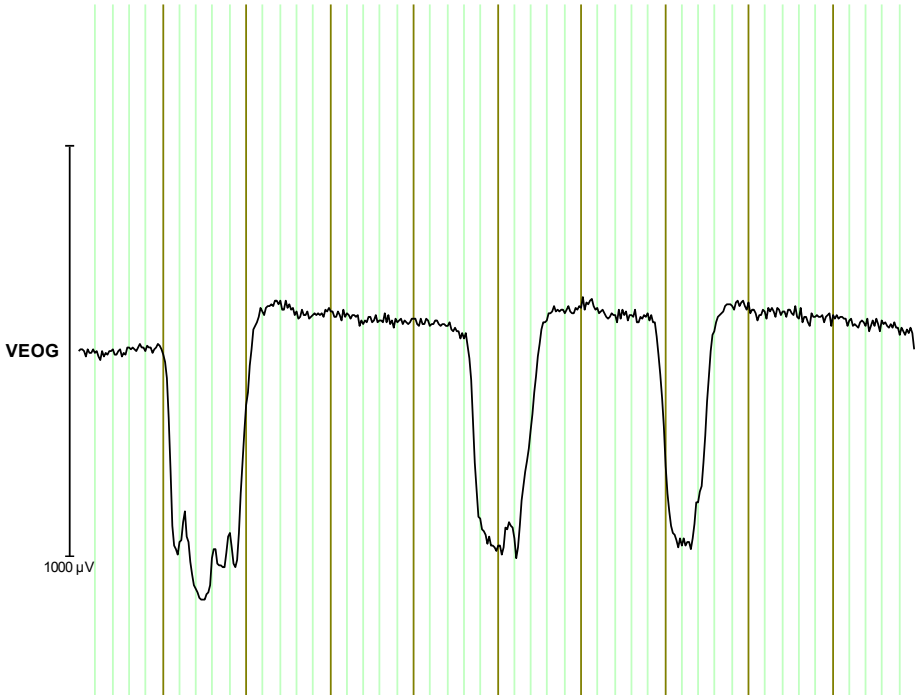


Figure A-2: EEG raw data of channels A1 and A2 of non-smoker N01 (not affected by polarity shift artifact) showing ten seconds beginning from 2 minutes and 11 seconds in the data set:

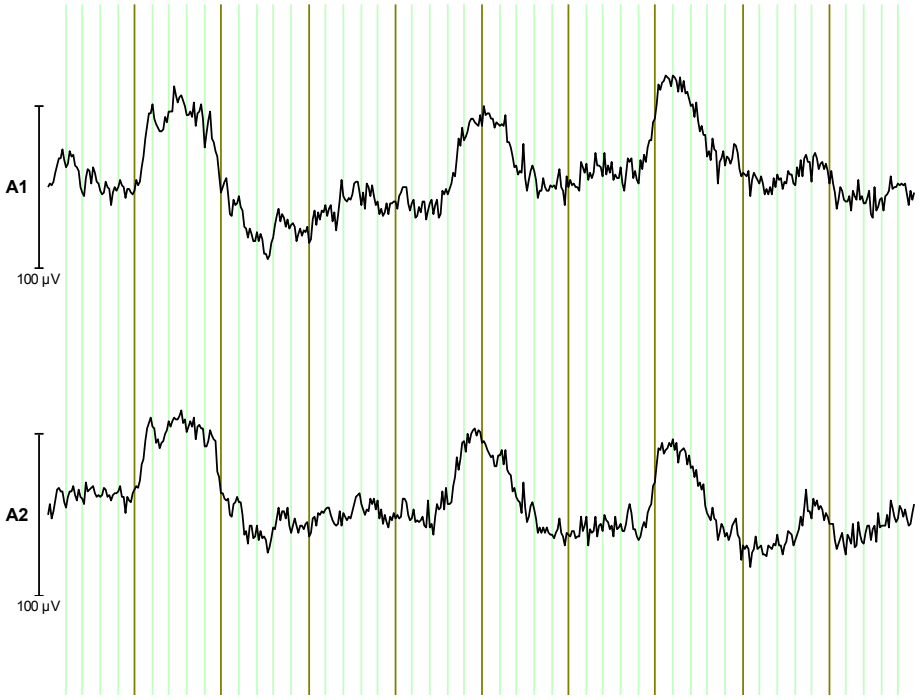


Figure A-3: VEOG raw data of non-smoker N07 (affected by polarity shift artifact) showing ten seconds beginning from 24 seconds in the data set, for facility of inspection reasons the EOG amplitudes in this figure are scaled smaller than the corresponding EEG data in figure A-4:

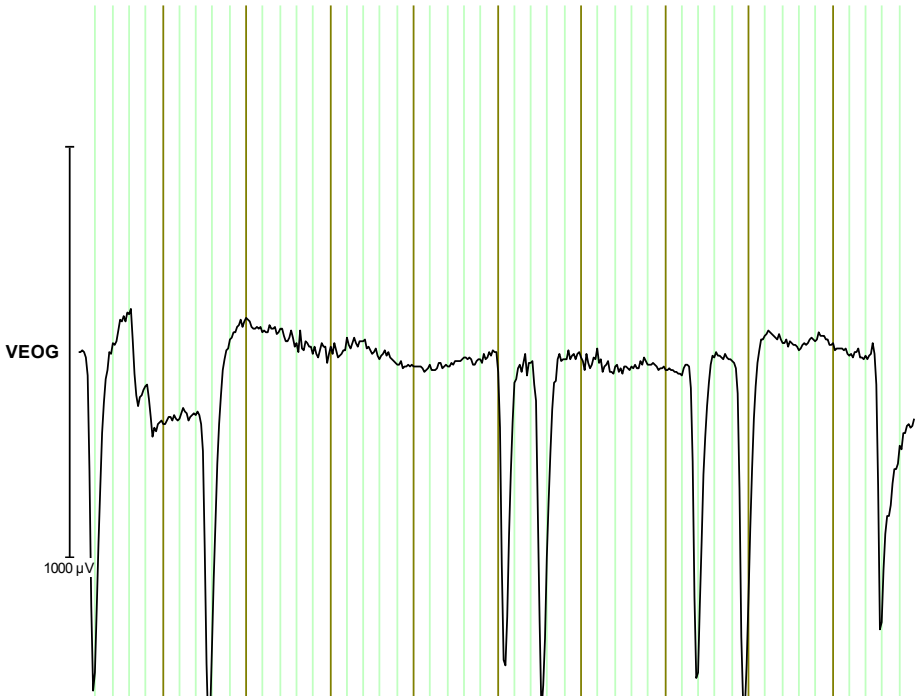


Figure A-4: EEG raw data of channels A1 and A2 of non-smoker N07 (affected by polarity shift artifact) showing ten seconds beginning from 24 seconds in the data set:

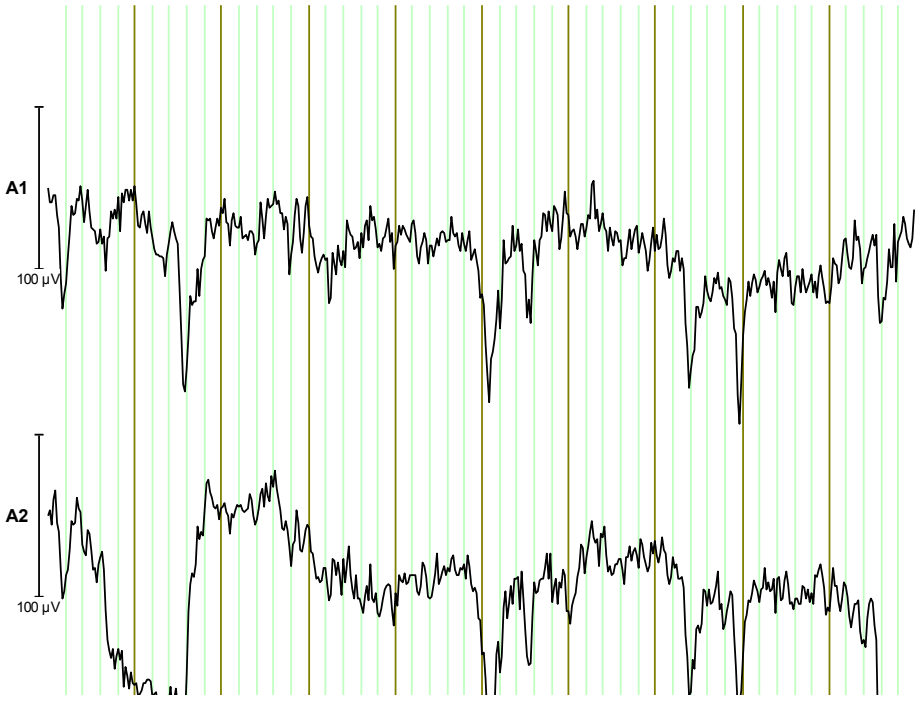
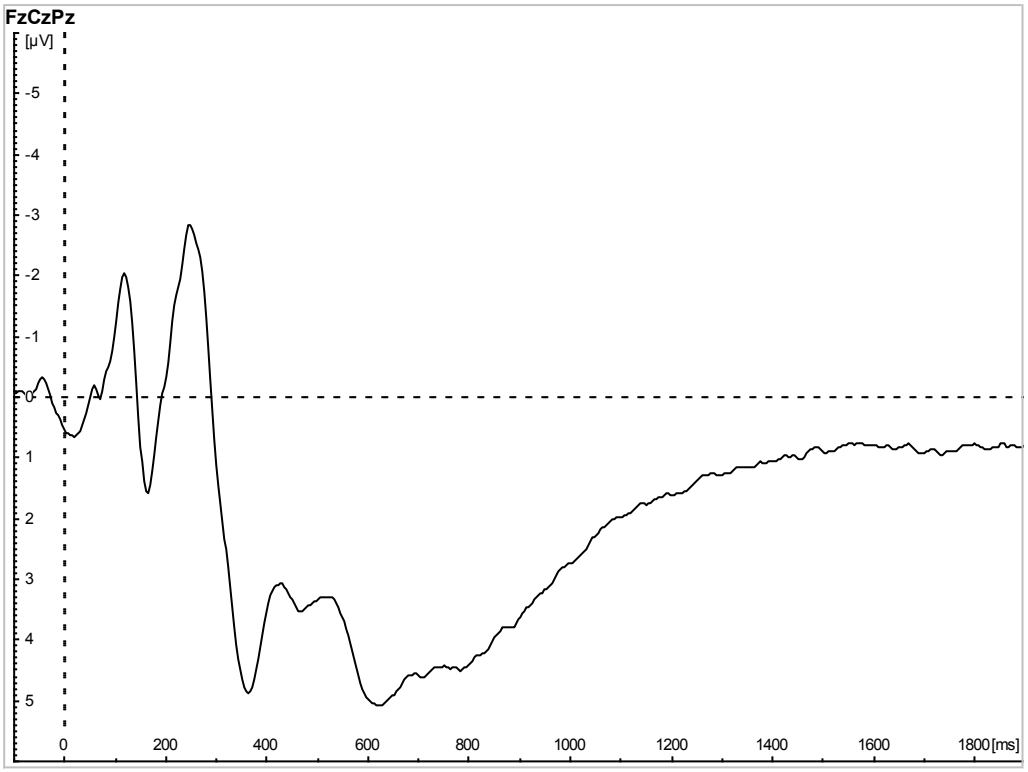


Figure A-5: Super grand average combining all subjects (both groups: smokers and non-smokers), all four stimulus categories (smoking objects, non-smoking objects, smoking persons, non-smoking persons), and the three midline sites Fz, Cz, and Pz (see section 2.6.):



7. Appendix B: Questionnaires

Soziographischer Fragebogen

Angaben zur Person:

VP Nr.: _____ Kontakt Nr.: _____ Datum: _____ (Bitte eintragen)

Alter: _____ Jahre (Bitte eintragen)

Familienstand:

1 ☐ ledig
2 ☐ verheiratet, zusammenlebend
3 ☐ verheiratet, getrennt lebend
4 ☐ geschieden
5 ☐ verwitwet

Höchster erreichter Schulabschluss:

1 ☐ kein Abschluss
2 ☐ Sonderschulabschluss
3 ☐ Hauptschul-/Volksschulabschluss
4 ☐ Realschulabschluss/Polytechnische Oberschule
5 ☐ (Fach-) Abitur
6 ☐ Hochschulabschluss
7 ☐ anderer Schulabschluss: _____ (Bitte eintragen)

Berufsausbildung:

1 ☐ keine/abgebrochen
2 ☐ Berufsausbildung/Lehre
3 ☐ Sonstige: _____ (Bitte eintragen)

Staatsangehörigkeit:

1 ☐ deutsch
2 ☐ andere:_____ (Bitte eintragen)

Erwerbstätigkeit:

1 ☐ Auszubildender
2 ☐ Angestellter, Beamter
3 ☐ Arbeiter/Facharbeiter
4 ☐ Selbständiger/Feiberufler
5 ☐ mithelfender Familienangehöriger
6 ☐ Arbeitsloser
7 ☐ Schüler
8 ☐ Student
9 ☐ Hausmann
10 ☐ Rentner
11 ☐ berufl. Rehabilitation
12 ☐ Sonstige: _____ (Bitte eintragen)

Falls Sie studieren, geben Sie bitte Studienfach/-fächer und Zahl der Fachsemester an:

Studienfach:_____ Fachsemester: _____ (Bitte eintragen)

Studienfach:_____ Fachsemester: _____ (Bitte eintragen)

Studienfach:_____ Fachsemester: _____ (Bitte eintragen)

weitere: _____

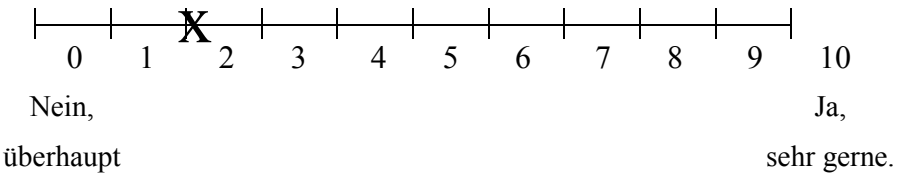
Datum:_____ Code:_____

Fragebogen zur subjektiven Einschätzung

Die folgenden Fragen beziehen sich auf Ihre momentane Befindlichkeit. Bitte beantworten Sie diese Fragen so, wie es Ihrem Empfinden jetzt im Moment entspricht. Antworten Sie spontan und machen bitte an der Stelle, die Ihrer augenblicklichen Befindlichkeit entspricht, ein Kreuz (s. Beispiele). Dabei ist es auch möglich, zwischen den Markierungen Kreuze zu setzen. Die folgenden Fragen werden in dieser Untersuchung mehrmals gestellt. Bitte prüfen Sie jedes Mal genau, wie Sie sich fühlen, bevor Sie antworten.

Beispiel 1:

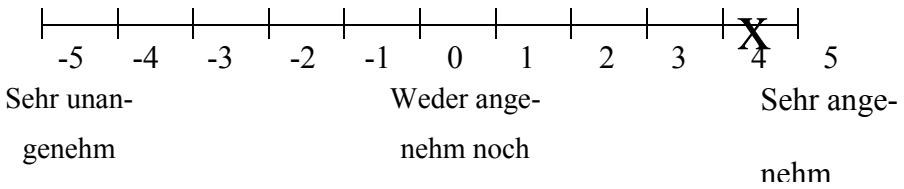
Würden Sie jetzt gerne eine Zigarette rauchen?



Diese Frage soll uns zeigen, wie gerne Sie im Moment eine Zigarette rauchen möchten. Die Skala reicht von „überhaupt nicht gerne“ auf der linken Seite bis zu „ja, sehr gerne“ auf der rechten Seite. Im mittleren Bereich sind Abstufungen zwischen den Extremen möglich. Bedenken Sie bitte, dass auch Angaben zwischen den Markierungen durch die Zahlen möglich sind. Im obigen Beispiel ist ein geringer Wunsch eine Zigarette zu rauchen dargestellt.

Beispiel 2:

Eine Zigarette zu rauchen wäre jetzt...



Diese Frage soll uns zeigen, wie angenehm es im Moment für Sie wäre, eine Zigarette zu rauchen. Die Skala reicht von „sehr unangenehm“ auf der linken Seite bis zu „sehr angenehm“ auf der rechten Seite. In der Mitte der Skala können Sie „weder angenehm noch unangenehm“ vermerken. Im restlichen Bereich sind Abstufungen zwischen den Extremen möglich. Bedenken Sie bitte, dass auch Angaben zwischen den Markierungen durch die Zahlen möglich sind. Im obigen Beispiel ist dargestellt, dass es im Moment „angenehm“ wäre zu rauchen.

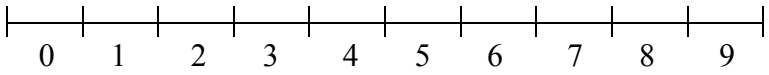
Bitte blättern Sie jetzt auf die nächste Seite, um Ihre eigenen Einschätzungen durchzuführen.

Datum:_____ Code:_____

Messung:_____ Uhrzeit:_____

Fragebogen zur subjektiven Einschätzung

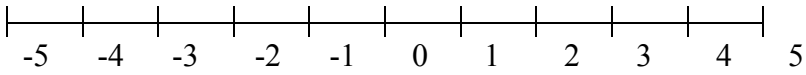
Würden Sie jetzt gerne eine Zigarette rauchen?



Nein, überhaupt

Ja, sehr gerne.

Eine Zigarette zu rauchen wäre jetzt...



Sehr unangenehm

Weder angenehm noch

Sehr angenehm

EHI

VP-Nr.:
Geburtsdatum:
Geschlecht:

Bitte geben Sie für die folgenden Aktivitäten an, welche Hand Sie dafür verwenden. Dafür schreiben Sie ein „+“ in die entsprechende Spalte. In Fällen, in denen die Präferenz so stark ist, dass Sie niemals die andere Hand verwenden würden, es sei denn, Sie wären dazu gezwungen, setzen Sie bitte “++”.
Sind Sie bei einer Tätigkeit unentschieden, so markieren Sie bitte beide Spalten mit einem “+”. Einige Tätigkeiten erfordern beide Hände. In diesen Fällen steht der Teil der Tätigkeit oder des Objekts, für den die Handpräferenz erfragt werden soll in Klammern.
Bitte versuchen Sie alle Fragen zu beantworten und lassen Sie nur solche Fragen aus, bei denen Sie überhaupt keine Erfahrung mit dem Objekt oder der Handlung haben.

		Links	Rechts
1	Schreiben		
2	Malen		
3	Werfen		
4	Schere		
5	Zahnbürste		
6	Messer (ohne Gabel)		
7	Löffel		
8	Besen (obere Hand)		
9	Streichholz anzünden (Streichholz)		
10	Schachtel öffnen (Deckel)		
I	Mit welchem Fuß ziehen Sie es vor zu treten?		
II	Welches Auge verwenden Sie, wenn Sie nur eines benutzen?		

Vom Versuchsleiter auszufüllen:

L. Q:	
Dezil	

Datum:_____ Code:_____ Geb._____

Beruf:_____

Rauchanamnese

Nun möchten wir Sie bitten, ein paar allgemeine Fragen zu Ihren Rauchgewohnheiten zu beantworten.

1. Wie alt sind Sie? ____ Jahre

2. Wie alt waren Sie, als Sie angefangen haben, Zigaretten *regelmäßig* zu rauchen? ____ Jahre

3. Rauchen Sie bis heute? ☐ ja ☐ nein

4 Wie viele Zigaretten rauchen Sie durchschnittlich am Tag? ____ Zig./Tag

5. Wie viele Jahre haben Sie bis jetzt *regelmäßig* geraucht? ____ Jahre

6. Haben Sie schon mal versucht, das Rauchen aufzugeben? ☐ ja ☐ nein
Falls "ja", wie oft? ____ Mal

7. Gibt es eine Zigarettenmarke, die Sie gewöhnlich rauchen? ☐ ja ☐ nein

Falls "ja", welche? _____
Ist es eine "light" Marke? ☐ normale Zigarette ☐ light Zigarette

Wie hoch ist der Nikotingehalt dieser Marke? _____mg/Zig.

8. Konsumieren Sie Nikotin in einer anderen Form als Zigaretten (z.B. Pfeife, Schnupftabak)? ☐ ja ☐ nein
Falls "ja", in welcher Form? _____

9. Raucht Ihr Umfeld? ☐ ja ☐ nein

8. German summary/Deutsche Zusammenfassung

Hintergrund: Bei verschiedenen Arten von Substanzabhängigkeit wurden ereigniskorrelierte Potenziale (EKP) wiederholt genutzt, um suchtspezifische Reaktivität von Konsumenten der verschiedenen Substanzen zu demonstrieren. Substanzabhängige und –konsumenten zeigten spezifische Reaktionen auf substanzassoziierte Reize, die in dieser Form nicht bei Kontrollpersonen auftraten und die sich von Reaktionen auf neutrale Reize unterschieden. Verschiedene Arbeiten fanden bei Rauchern suchtspezifische Reaktivität für die Komponenten N300 und P300 des ereigniskorrelierten Potenzials, neuere Hinweise gibt es auch auf Reaktivität im Bereich der langsamen positiven Wellen.

Fragestellung: Die vorliegende Arbeit hatte zum Ziel, Effekte von suchtspezifischer Reaktivität im Bereich der ereigniskorrelierten Potenziale zu replizieren und zu untersuchen, ob der soziale Bezug von rauchassoziierten Reizen diese Effekte verstärkt.

Methodik: Im Anschluss an die Durchführung anstrengender Rechenaufgaben zur Steigerung des Verlangens wurden 24 Rauchern und 19 Nichtrauchern in randomisierter Reihenfolge Bilder von Personen und Objekten/Gegenständen gezeigt, die mit dem Rauchen assoziiert waren oder nicht. Parallel dazu wurden die ereigniskorrelierten Potenziale abgeleitet.

Ergebnisse: Es wurde keine suchtspezifische Reaktivität für die N300 und die P300 Komponenten der EKP von Rauchern gefunden. In begrenztem Umfang zeigten einige statistische Analysen signifikante suchtspezifische Reaktivität im Bereich der langsamen positiven Wellen zwischen 500 und 850 ms nach Reizbeginn an medialen und posterioren Ableitungspositionen von Rauchern. Dabei lösten rauchassoziierte Reize bei Rauchern höhere Positivität aus als Reize ohne Bezug zum Rauchen, diese Unterschiede waren bei Nichtrauchern kleiner. In nach den EKP-Ableitungen erhobenen Bewertungen der experimentellen Reize erlebten die Raucher höheres Verlangen beim Betrachten von rauchassoziierten Bildern als nach Bildern ohne Rauchbezug. Dieser Unterschied war größer bei Bildern von Gegenständen als bei Bildern von Personen. Zudem bewerteten Raucher die rauchassoziierten Bilder als angenehmer als Bilder ohne Rauchbezug, während die Bewertungen der Nichtraucher diesbezüglich entgegengesetzt waren.

Schlussfolgerungen: Die vorliegende Arbeit kann in zuvor durchgeführten Arbeiten gefundene Effekte von suchtspezifischer Reaktivität in den N300 und P300 Komponenten der ereigniskorrlierten Potentiale auf bildhafte Reize nicht replizieren und bestätigen. Stattdessen gibt es Hinweise, dass suchtspezifische Reaktivität eher im Bereich der langsamen positiven Wellen zu finden ist. Die Hinweise für suchtspezifische Reaktivität im Bereich der langsamen positiven Wellen stehen in Übereinstimmung mit anderen Studien, die ebensolche Effekte bei verschiedenen Populationen von Abhängigen zeigen konnten. Allerdings erreicht die suchtspezifische Reaktivität im Bereich der langsamen positiven Wellen in der vorliegenden Studie nur sehr eingeschränkt statistische Signifikanz.

Die suchtspezifische Reaktivität im Bereich der langsamen positiven Wellen könnte die verstärkte Allokation von kognitiven Ressourcen und Aufmerksamkeitsressourcen für die Verarbeitung rauchbezogener Reize repräsentieren. Die Effekte suchtspezifischer Reaktivität im Bereich der langsamen positiven Wellen scheinen nicht durch den sozialen Bezug der Reize moduliert zu sein. Allerdings scheint der soziale Bezug des Reizmaterials die suchtspezifische Reaktivität in Selbstauskunftsmaßen wie dem wahrgenommenen Verlangen zu verringern. Als Erklärung dieser Effekte des sozialen Bezugs der Stimuli werden die Ablenkung vom Rauchbezug der Bilder und die Erweiterung des semantischen Raumes diskutiert.

9. Erklärung

Hiermit versichere ich,

a) dass ich Arbeit selbständig verfasst und keine unerlaubte fremde Hilfe in Anspruch genommen habe
b) dass ich die Arbeit noch in keinem anderen Prüfungsverfahren vorgelegt und keine anderen als die in der Dissertation aufgeführten Quellen benutzt habe
c) dass es sich bei dem eingereichten Exemplar um das Original handelt.

Münster, den _____ _____

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10. Lebenslauf