Environmental Influences on Craving and the Physiological and Cognitive Effects of Cigarette Smoking

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A novel environment was used to examine whether arbitrary environmental stimuli could come to elicit conditioned compensatory responses from cigarette smokers. It was hypothesised that: 1) Craving for cigarettes would be linked to environmental stimuli, and 2) these stimuli would elicit physiological and cognitive conditioned compensatory responses. Fourteen participants aged between 19 and 51 were exposed to 10 conditioning sessions in a novel environment followed by 2 experimental sessions in the same environment. Half of the participants smoked during the conditioning sessions while the other half mock smoked. During the two experimental sessions, the participants smoked in one session and mock smoked in the other. The participants' heart rate, cognitive and craving responses were recorded. The craving hypothesis was supported, however, there was no little statistically significant support for the second hypothesis. The benefits of using the current design are discussed, along with the importance of the results for smoking cessation programs.

Cigarette smoking is a prolific problem in New Zealand Society. Statistics indicate that approximately 26% of the population are regular smokers and smoking directly and indirectly results in thousands of deaths each year (Ministry of Health, 2000). Reducing the rates of smoking is a major aim of health groups within New Zealand and around the world.

Cigarette smokers experience more than a physiological addiction. While nicotine is a highly addictive drug, cigarette smoking also involves a behavioural component. Cigarette smokers often follow ritualistic patterns before, during and after a cigarette (Le Houezec, 1998). A substantial body of research has focused on cigarette smoking as a learnt addiction and it has become clear that behavioural techniques could play an important role in any treatment strategy aimed at establishing long-term cigarette smoking cessation (O'Brien, Childress, Ehrman, & Robbins, 1998).

The effects of nicotine are important when examining smoking from a behavioural perspective, as these effects have the potential to lead to conditioned responses. Nicotine is a potent stimulant (Gottlieb, 1992) and its consumption is accompanied by marked increases in plasma norepinephrine and epinephrine (Cryer, Haymond, Santiago & Shad 1976). The influx of these agents has a profound and almost instantaneous effect on the body's sympathetic nervous system. Two of the most frequently reported physiological changes resulting from nicotine administration are increases in heart rate (e.g., Fagerstrom, 1978) and blood pressure (e.g., Grassi, Seravalle, Calhoun, Bolla, Giannattasio, Marabini, Bo, & Mancia, 1994; Hashimoto, 1993). Studies have also found that nicotine can improve performance on many different cognitive tasks such as short term memory tasks (Kerr, Sherwood & Hindmarch, 1991; Sherwood, Kerr & Hindmarch, 1992; West & Hack, 1991), choice reaction time tasks (Kerr et al., 1991; Sherwood et al, 1992), compensatory tracking tasks (Kerr et al., 1991; Sherwood et al., 1992), and continuous performance tasks (Pritchard, Robertson & Guy, 1992).

Cue reactivity refers to classically conditioned responses to environmental cues that consistently coincide with drug administration. These responses may be cognitive, behavioural or physiological in nature (Payne, Etscheidt, & Corrigan, 1990). Studies measuring craving for nicotine have found that the presentation of smoking-related stimuli result in increased desire for a cigarette (e.g., Tiffany & Hakeneworth, 1991; Tiffany & Drobes, 1990). Studies have also revealed that classically conditioned physiological responses to nicotine have tended to be compensatory in nature, that is, opposite to the effects of the drug (e.g., Niaura, Abrams, DeMuth, Pinto & Monti, 1989). No published studies have examined the influence of

environmental cues on the cognitive effects of nicotine. However, due to their association with the ingestion of nicotine, cognitive improvements also have the potential to become conditioned (like the physiological responses).

Studies that test for a relationship between smoking-related cues and motivation to smoke, generally follow a similar format – smokers' responses (self report, physiological and/or behavioural) are measured in response to smoking related, negative and/or neutral cues (see Brandon, Piasecki, Quinn & Baker 1995 for a summary of the research in this area). Cue reactivity designs are frequently criticized because of the way smoking related cues are selected for the studies. Studies recruit participants who already smoke cigarettes on a regular basis. Hence, the experimenter has no control over the conditioning of environmental stimuli to the cigarette smoking behaviour and the cues chosen are only assumed to be related to the intact of nicotine. Thus, while the studies provide evidence to support the notion that environmental cues can elicit responses from participants, they cannot conclude that these changes are due to learning processes rather than a response to the environment itself.

Robbins and Ehrman (1992) have argued that to provide evidence for the case of classical conditioning, it is crucial for the experimenter to control the conditioning trials rather than to rely on 'opportunistic designs' that utilize pre-existing environmental cues assumed to be conditioned to cigarette smoking. One way to achieve this level of control would be to obtain a sample of smokers and condition them to smoking in a particular environment. To date, only one published paper has attempted such a study. Using a single subject design, Payne et al. (1990) used 20 conditioning trials in two different environments to attempt to condition a cigarette smoker to an arbitrary set of environmental stimuli. The participant was exposed to two different environments and smoked in one and mock smoked in the other. Following the conditioning trials, the conditions were reversed with the participant smoking in the mock smoking environment and mock smoking in the smoking environment and their physiological responses to this manipulation were measured. While the results showed no conclusive conditioning effect, heart rate and skin temperature trends indicated possible conditioned physiological responses.

The strength of Payne et al.'s (1990) design is that it allows for control over the conditioning process and hence allows researchers to determine whether or not the response to the environmental cues is a learnt phenomenon. However, the design still uses two environments and as such it cannot be determined whether the observed changes are not simply artefacts of the different testing environments.

The current study used a design similar to that used by Payne et al. (1990), however only one testing environment was used. This single environment was used to test the influence of environmental cues on both the cognitive and physiological effects of nicotine as well as on craving. The study was broken into two phases – a conditioning and an experimental phase. The participants were divided into two groups – a smoking group and a mock smoking group. During the conditioning phase, distinct environmental stimuli were paired with either smoking or mock smoking. In this way, specific environmental cues were associated with cigarette smoking for one group of participants and with not smoking for the other group. 'Mock smoking' as opposed to 'not smoking' was used to attempt to extinguish the cues associated with preparing to smoke a cigarette (e.g., taking out a cigarette, igniting the lighter etc.) in the novel environment.

During the experimental phase of the study participants completed physiological, cognitive and craving tests in two sessions – first when they were in their original condition (e.g., the smoking group participants smoking in the novel environment); and then a second time when the conditions were reversed (e.g., the smoking group participants mock smoking in the novel environment). Using this design, the influence of environmental cues was tested without the confounds of having two different testing environments (as in the Payne et al, 1990 design), using a comparison group of non-smokers, or assuming which environmental cues are important for individual smokers.

It was expected that the novel environment would come to elicit compensatory conditioned responses in the smoking group but not in the mock smoking group. This would be apparent in the following observations during experimental sessions: first, smoking group participants would report higher levels of craving for cigarettes following a period of abstinence than mock smoking group participants (Hypothesis 1); second, heart rate of smoking group participants would be lower than that of mock smoking group participants, both before and after smoking a cigarette (Hypothesis 2); and third, smoking group participants would perform less well on a cognitive task than mock smoking group participants, after both smoking and mock smoking a cigarette (Hypothesis 3).

Method

Participants

Fourteen participants (eleven females, three males) took part in this study. The mean age of participants was 30.1 years (SD = 9.1, range 19 years to 51 years). The participants had been smoking for an average of 13.0 years (SD=9.5, range 3.5 years to 32 years) and smoked an average of 17.8 cigarettes per day (SD=12.7, range 10 to 60). The average strength of the cigarettes smoked was 8.8mg of nicotine (SD=4.5, range 1 mg to 16 mg). The participants reported a median of 'one to three' attempts to quit smoking over the course of their addictions. The smoking and the mock smoking groups did not differ significantly on any of the these measures.

Initial screening of the applicants was conducted during recruitment. Interested persons were asked not to apply if they were: a) pregnant; b) expecting to quit smoking within the next month; c) regular users of recreational drugs (other than nicotine or coffee) or alcohol (greater than three standard drinks a night, more then three times a week for more than a month); d) currently diagnosed as having a mental illness; or e) under 18 years of age. Applicants

must also have reported as being regular cigarette smokers (greater than 10 cigarettes per day for the majority of days of the week).

Prior to their participation, applicants were provided with two information pamphlets – one on the health risks associated with cigarette smoking and the other on the effects of passive smoking (an ethics committee requirement). The participants were then assigned to one of two groups - the smoking group or the mock smoking group – by placing every second participant in the same group. While not a truly random assignment, there is no evidence to suggest that the practice lead to any systematic differences between the groups and was conducted to maintain equal group sizes.

Participants were required to abstain from smoking cigarettes for at least three hours (but preferably 12 hours) prior to both experimental sessions. The three-hour option was provided so as not to place participants under any undue discomfort. Participants were also required to abstain from alcohol, caffeine and other mood altering drugs for 12 hours prior to both experimental sessions. Compliance with these abstinence conditions was checked by way of participant self-report. Participants brought their own cigarettes to all sessions. Informed consent was obtained in writing from all participants. This study received ethical approval from the University of Canberra Ethics Committee.

Apparatus

A novel environment was created by partially enclosing a portion of a second floor balcony. The 'walls' of this enclosure were built by draping multicoloured sheets of plastic over ropes that were stretched between pillars on the balcony. This 'room' (approximately 4.5m x 1.5m) contained two lounge chairs, a sandbox, scientific equipment and a lap top computer. Posters were arranged around the inside of the 'room' and a spotlight was used to light the enclosure. To create a distinct smell, 'Vanilla Coconut Black Diamond' incense (Jungle Juice) was burnt. Finally, as well as being used to run the cognitive tasks and collect data, the computer was used to play music: Emma Paki's "Oxygen of Love" (Virgin Records, 1996). The music was always started at the same point for each session and was turned off at the beginning of the cognitive task. A manipulation check confirmed that the participants rated the environment as being novel, with the median response on the scale indicating that the environment was rated as "Quite Dissimilar" to any other environments they might encounter.

Heart rate was measured using a biofeedback system (Bioview, Series V) that was attached to each participant's ear using a small clip. The biofeedback system measured heart rate every 0.5s and recorded an average for each minute.

Questionnaire and cognitive task

Craving measure

Craving for cigarettes was measured using the Drug Related States Questionnaire (Droungas et al., 1995). This scale comprised three items tapping a participant's craving for a cigarette, desire to smoke and feelings of withdrawal. Specifically, the questionnaire asks the participants: a) whether they crave a cigarette right now (craving), b) whether they would have a cigarette now if they had the chance (desire to smoke), and c) whether they feel 'edgy', as if they had not had a cigarette in a while (withdrawal). Each item was answered on a scale from 0 – 10 (0 = not at all and 10 = very much). The total summed scores ranged from 0 – 30 with a higher score indicating a higher level of craving for a cigarette. The scale exhibited a high degree of internal consistency with a Cronbach alpha of 0.87.

Continuous performance task

The continuous performance task was based on a similar task described by Pritchard et al. (1992). The task required the participants to determine whether a digit (0 – 9) was a target digit or not. The target digit (0) appeared randomly on 19% of the trials with the remaining 'non-target' digits (1-9) appearing on 9% each. The digits appeared in the centre of the computer monitor for 0.1s before being covered by a 'mask' (an asterisk) that prevented the digit from being seen. Each digit was displayed in white text with a black background and had a vertical height of 5 mm. Following each presentation, the participants had 1.5s in which to decide if the digit had been the target digit or not. Participants were instructed to push 'V' if they believed the digit was the target digit, and 'N' if they believed that it was not. Both buttons were labelled ('YES' and 'NO' respectively) to make key identification easier. Participants were notified of incorrect responses with the word "Incorrect" appearing in the top left hand corner of the monitor and the sounding of a brief, 100 Hz tone. Correct responses were signalled by the word "Correct!" appearing briefly in the top left hand corner of the monitor. Failure to respond within the 1.5s was treated as an incorrect response. Data were collected on both the speed and accuracy of participants' responses. Each session consisted of 500 trials (taking approximately 12 minutes) and was preceded by a practice run of 10 trials. Practice trials were not included in the data analysis.

The measures reported were part of a larger battery administered.

Procedure

The study was broken into two phases – the Conditioning Phase and the Experimental Phase. In the conditioning phase, each participant completed ten 10-minute sessions in the novel environment over the course of a three-week period. In the experimental phase, each participant completed two sessions, each lasting approximately one hour. The experimental sessions were completed on consecutive days within a week of the completion of the final conditioning session. All sessions were conducted individually for each participant. The experimenter remained in the novel environment with the participants throughout the sessions, leaving only during the smoking/mock smoking component and during the cognitive task. The experimenter did not interact with the participants except to explain the procedures.

During the conditioning sessions, the two groups of

participants - Smoking Group and Mock Smoking Group - were exposed to the same novel environment however, the smoking group smoked in the novel environment whilst the mock smoking group mock smoked. When mock smoking, participants were asked to complete all the actions normally associated with smoking a cigarette (for example inhaling through the cigarette and 'ashing' the cigarette) using an unlit cigarette.

Conditioning sessions began with the experimenter attaching the heart rate monitor. The participant's heart rate was then monitored for the first six minutes of the session. Approximately two minutes after the start of the session, the participant either smoked or mock-smoked a cigarette (depending on the group they had been assigned to). The heart rate monitor was then detached, the music turned off, and the participant completed a short-term memory task. Heart rate recordings were taken so as to keep the conditioning and experimental sessions as similar as possible, however none of these data were analysed.

Following completion of the final conditioning session, participants entered the experimental phase. During one of the experimental sessions, participants were asked to smoke a cigarette and in the other they were asked to mock smoke a cigarette. The order of these two sessions was determined by the group that the participant was in, with the first experimental session always being the same as the participants conditioning sessions. This was done so as not to contaminate any conditioned cues that were present in the environment. It was felt that the advantages of maintaining the integrity of the environmental cues (the consistency of the pairing of the environmental cues with the smoking/ mock smoking behaviour) out-weighed controlling for potential order effects. Participants were not informed of whether they would be smoking or mock smoking until they were required to do so.

Each experimental session began with the participant being connected to the heart rate monitor. The participant's heart rate was then monitored for the first 20 minutes of the session. The first 10 minutes of both experimental sessions was used as an 'acclimatization period', during which the participant completed a biographical questionnaire. After the acclimatization period, the participant completed the 'Craving Measure' and then either smoked or mock smoked a cigarette (depending on which session they were in). The participant then completed the continuous performance task. At the end of each participant's second experimental session, they completed the manipulation check and were debriefed.

Results

The mean number of days taken to complete the conditioning sessions was 20 days (SD = 4.8) for the smoking group and 21 days (SD = 5.3) for the mock smoking group. The means and standard deviations of the time abstained from smoking prior to both experimental sessions for the smoking and mock smoking groups can be found in Table 1. For the first experimental session, the smoking group abstained for a shorter time-period on average than the mock smoking group. However, this difference was not significant, $t(12) = 1.05$, $p = .315$, two-tailed. Analysis revealed no significant correlations between the number of hours abstained prior to the experimental session and a participant's reported level of craving for either the first, $r(14) = -.31$, $p = .275$, or the second, $r(14) = .29$, $p = .373$, experimental session.

Craving Data

Figure 1 shows the mean craving scores for the smoking group and mock smoking group in both experimental sessions and the means and standard deviations can be found in Table 2. The mean craving scores for the first experimental session were compared and no significant

Figure 1. Mean level of craving reported by participants in the smoking and mock smoking groups in each experimental session. Standard error bars are shown.

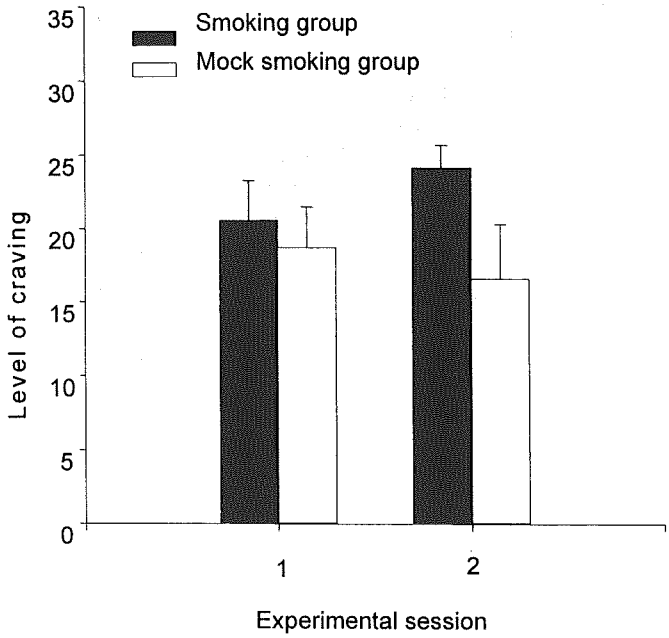


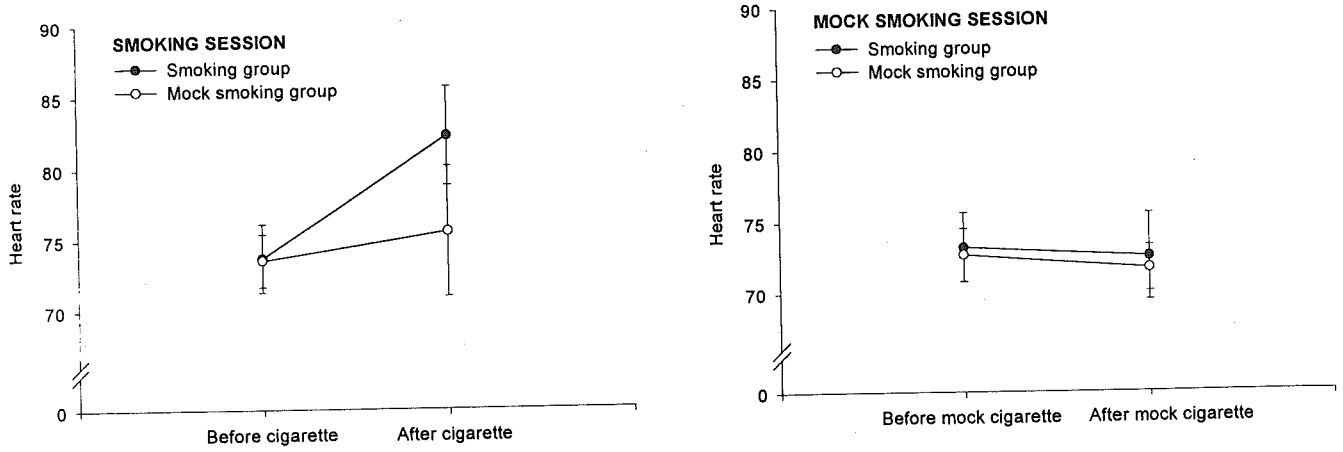
Table 1. Means and Standard Deviations for the Number of Hours Abstained from Smoking Prior to the First and Second Experimental Sessions for Smoking and Mock Smoking Groups

		Experimental Sessions	
Group		Session 1	Session 2
Smoking	Mean	8.71	10.86
	SD	5.47	3.29
Mock Smoking	Mean	11.29	10.93
	SD	3.49	3.52

Table 2. Means and Standard Deviations of Craving Scores for the Smoking and Mock Smoking Groups in both Experimental Sessions

		Experimental Sessions	
Group		Session 1	Session 2
Smoking	Mean	20.57	24.14
	SD	7.14	4.02
Mock Smoking	Mean	18.71	16.57
	SD	7.30	9.81

Figure 2. Mean heart rate of participants in each group before and after having a real/mock cigarette in the experimental sessions. Standard error bars are shown.



difference was found between the smoking (20.57) and mock smoking (18.71) groups, $t(12) = 0.48$, $p = .319$, one-tailed. In the second experimental session the smoking group (24.14) was found to be craving significantly more than the mock smoking group (16.57), $t(8) = 1.89$, $p = .048$, one-tailed.

Physiological Data

Figure 2 shows the mean heart rate of participants in each group before and after the smoking and mock smoking components of the experimental sessions. While heart rate increased on average after a real cigarette (in both groups), a two-way split plot analysis of variance (SPANOVA) showed this effect did not reach statistical significance, $F(1,12)=3.9$, $p=.070$. The SPANOVA also showed, no effect of group, $F(1,12)=0.8$, $p=.380$, and no interaction between smoking and group membership, $F(1,12)=1.5$, $p=.242$. It can be seen in Figure 2 that mean heart rate increased in the group who had smoked previously in the experimental environment, more so then it did for the group who had not.

A second two-way SPANOVA assessed the effects of mock smoking on heart rate. As expected, there was no effect of mock smoking, $F(1,12)=1.7$, $p=.209$, no effect of group, $F(1,12)=0.1$, $p=.836$, and no interaction between mock smoking and group, $F(1,12)=0.5$, $p=.819$. The means and standard deviations for the heart rate data for the two groups for both experimental sessions can be found in Table 3.

Cognitive Data

Average accuracy (log d) and response time (ms) for the smoking and mock smoking groups on the continuous performance task during the experimental sessions are shown in Figure 3. A two-way SPANOVA revealed that participants were more accurate after smoking a cigarette than after mock smoking, $F(1,12)=9.5$, $p=.009$, no effect of group, $F(1,12)=0.1$, $p=.910$, but an interaction between these two factors, $F(1,12)=7.1$, $p=.020$. As Figure 3 shows, accuracy was affected in participants smocking in the experimental environment for the first time (mock smoking group) but not in those who had smoked there previously (the smoking group).

A two-way SPANOVA on the response time data showed participants were slower after smoking a cigarette than after mock smoking, $F(1,12)=5.0$, $p=.045$, no effect of group, $F(1,12)=0.7$, $p=.432$, but an interaction between these two factors, $F(1,12)=8.4$, $p=.014$. As Figure 3 shows, response times were affected in the smoking group but not in the mock smoking group. The means and standard deviations for the accuracy and response times for both groups in the two experimental sessions can be found in Table 4.

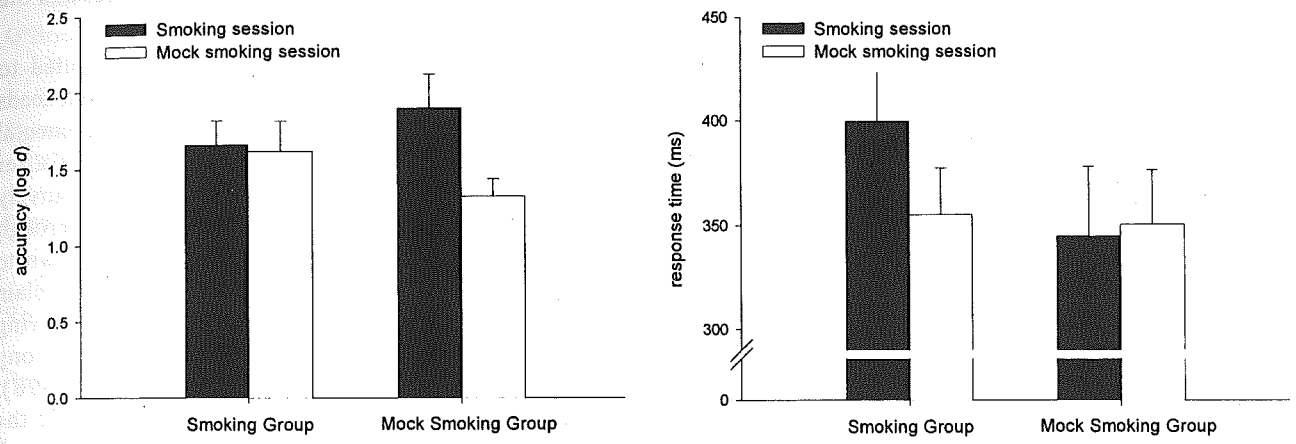
Qualitative Observations

During the experimental sessions, participants commented informally on their feelings. A participant from the smoking group told the experimenter - "*I didn't really want a cigarette*

Table 3. Means and Standard Deviations for Heart Rate (bpm) for the Smoking and Mock Smoking Groups Before and After the Smoking Component of the Experimental Sessions

		Experimental Session			
		Smoking		Mock Smoking	
Group		Before	After	Before	After
Smoking	Mean	73.70	82.19	73.21	72.51
	SD	6.39	9.05	6.41	8.09
Mock Smoking	Mean	73.54	75.54	72.69	71.69
	SD	5.92	9.11	5.94	8.28

Figure 3. Mean accuracy and median response times of participants in each group after smoking / mock smoking in experimental sessions. Standard error bars are shown.



(during the abstinence period)– *I was doing really well throughout the 12 hours – but as soon as I came in here* (the novel environment), *I really started to want a cigarette*". Three participants from the mock smoking group reported 'head-spins' following their cigarette in the second experimental session. Another participant from the mock smoking group reported that the cigarette they were smoking (during the final experimental session) made them feel nauseous.

Discussion

The results provided support for the craving hypothesis (Hypothesis 1), with significantly higher levels of craving reported by the smoking group in the second experimental session. Together with the qualitative data, these results support the findings of previous research on cue induced craving for nicotine (e.g., Niaura et al., 1989; Tiffany & Hakeneworth, 1991; Tiffany & Drobes, 1990). The heart rate hypothesis (Hypothesis 2) however, was not supported by the data, and little statistically significant support was obtained for the cognitive performance hypothesis (Hypothesis 3).

The smoking group reported higher levels of craving than the mock smoking group in both experimental sessions, however only in the second session was this difference significant. A possible reason for the greater difference in craving scores observed during the second experimental session might be because participants had experienced an extra session in the novel environment and hence the association between the environmental cues and smoking/ mock smoking behaviour was stronger.

The heart rate data provided little or no evidence to support the second hypothesis. Smoking a real cigarette seemed to lead to a greater increase in mean heart rate in the smoking group than in the mock smoking group (although this interaction was not statistically significant). This finding differs from previous studies (e.g. Niaura et al., 1989) that have found evidence of conditioned compensatory physiological responses following the presentation of smoking related cues. If anything, data from this current study this would suggest a conditioned 'drug like' response rather than a compensatory (drug opposite) conditioned response as hypothesised. However, given the lack of statistically significant differences and the large amount of variance in the data no conclusions can be drawn from the data.

There was no overall difference in accuracy or response times on the continuous performance task between the smoking and mock smoking groups (Hypothesis 3). However, these two groups appeared to react differently (in terms of these measures) to smoking a cigarette in the novel environment. The mock smoking group showed higher accuracy after smoking a real cigarette than after mock smoking, however their response times remained unaffected. The smoking group showed slower response times after smoking a real cigarette than after mock smoking,

Table 4. Means and Standard Deviations for Accuracy and Response Time (RT) (ms) for the Smoking and Mock Smoking Groups on the Continuous Performance Task (CPT)

		Experimental Session			
		Smoking		Mock Smoking	
Group		Before	After	Before	After
Smoking	Mean	1.65	399.64	1.60	355.00
	SD	0.42	61.95	0.50	59.08
Mock Smoking	Mean	1.88	344.71	1.31	71.69
	SD	0.58	88.30	0.30	68.88

however their accuracy was unaffected. These trends are not antithetical to some sort of compensatory conditioned response in operation. Given the lack of statistical power of this current study, these trends warrant further investigation.

Finally, the qualitative data provide additional support for the notion of cue reactivity. These data suggest that the presentation of smoking related cues led to a noticeable increase in craving for at least one of the participants. Furthermore, these data also suggest that tolerance for nicotine is mediated by environmental cues. This can been drawn from the observation that the intake of nicotine, when it was not proceeded by smoking related cues, resulted in feelings of nausea and 'head spins' for some participants.

This study supported the notion that craving for nicotine can be mediated through environmental cues. Furthermore, the study also found limited support for the notion that physiological and cognitive responses to nicotine might also have the capacity to become conditioned responses. Whilst cue reactivity is not a new concept, this study demonstrated the phenomenon using a design that eliminated the confounds that traditionally plague this type of research. Environmentally mediated craving was demonstrated without the confounds of having two different testing environments, using a comparison group of non-smokers, or assuming which environmental cues are important for individual smokers. As such, it provides clear evidence that environmentally mediated craving is a learnt association – not merely an artefact of the testing environments.

Although this study's design is an improvement on previous cue reactivity studies, the basic framework could still be improved. Future research should aim to recruit a larger sample of participants and to conduct a greater number of conditioning sessions. The greater number of conditioning sessions would help to strengthen any learnt associations whilst the larger sample size would both increase the statistical power of the study and strengthen the conclusions that can be drawn from any findings. Furthermore, although it is obviously unethical to recruit non-smokers, the recruitment of less experienced smokers might make any behavioural trends easier to observe because their smoking behaviour would be less likely to already be strongly associated with alternate environmental cues.

Given the results both from this study and from the convergence of results from previous cue reactivity studies, it is clear that environmental cues can mediate craving for cigarettes. This finding is important from a treatment perspective as craving is the main reason given by smokers for failed cessation attempts (Russell, 1988). The finding poses challenges for smoking cessation program and policy developers both within New Zealand and abroad as it suggests that programs aimed at promoting smoking cessation need to consider including behavioural treatment options along with the more traditional pharmaceutical based treatments. At present, no mainstream New Zealand smoking cessation program attempts to directly address the behavioural component of the cigarette smoking addiction. The finding that environmental cues can mediate craving for cigarettes suggests that policy developers need to consider implementing behavioural treatment options that attempt to break the association between the cues and the smoking behaviour.

Before behavioural techniques can be applied to cigarette smoking cessation however, future research needs to explore whether conditioned responses to environmental cues remain active for extended periods of time after smoking has ceased. This study showed that after a relatively short period of abstinence, craving could be induced through environmental cues. If this relationship can be shown to continue over a longer time frame it would help to explain why ex-cigarette smokers often report feelings of craving when exposed to smoking related cues even after long periods of cessation (Juliano & Brandon, 1998). Furthermore, research also needs to establish if the conditioned responses can be successfully extinguished and, if so, the most effective way of achieving this goal. The answers to these questions are crucial steps to establishing how best to apply behavioural techniques to drug addiction.

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