

A family history of smoking predicts heightened levels of stress-induced cigarette craving

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ABSTRACT

Aims Individuals with histories of smoking in first-degree relatives are significantly more likely to be persistent smokers themselves. The mechanisms underlying this relationship are unknown. Considerable research has demonstrated that smokers display heightened levels of cigarette craving after being exposed to stressful situations, and the magnitude of these craving responses is thought to be predictive of later cessation failure. Based on this research, we tested experimentally the hypothesis that smokers with two or more first-degree relatives who smoked (FH+) would exhibit stronger craving reactions following stressful stimuli than smokers without such family histories (FH-).

Participants We recruited 83 smokers by advertisement (mean age = 41.2 years, 57% female, 41% completed some college, 59% African American).

Setting The study was conducted in an interview room in an urban medical center.

Design Participants were exposed to a neutral situation (changing a lightbulb) and a stressful situation (dental work) using script-guided imagery.

Measurements Participants completed background measures of demographics, distress and smoking behavior. In addition, participants completed cigarette craving and anxiety questionnaires immediately before and after each condition.

Findings Supporting the study hypothesis, FH+ smokers ($n = 39$) selectively displayed stronger craving reactions to dental imagery ($P < 0.03$) than did FH- smokers ($n = 44$).

Conclusion The higher levels of stress-induced cigarette craving demonstrated experimentally for individuals with family histories of smoking suggest one mechanism for their poorer cessation success.

KEYWORDS Craving, family history, smoking, stress.

INTRODUCTION

Tobacco smoking is the single most preventable cause of morbidity and mortality in the United States (Public Health Service 2000). Indeed, smoking has been implicated in at least 30% of all cancer deaths in the United States and an estimated three million deaths per year world-wide (American Cancer Society 2001). Despite the

clear negative consequences of smoking, a significant subset of the population continues to smoke (Public Health Service 2000). Moreover, although most smokers express a strong interest in quitting, only about 15% are successful at maintaining abstinence over a 1-year period, even after employing some type of smoking cessation aid (e.g. nicotine replacement, self-help, counseling) (Zhu *et al.* 2000). Persistent smoking behavior (i.e. failure

or difficulty in remaining abstinent) has continued to be a major public health concern. These alarming data have stimulated widespread interest in better understanding of the predictors of persistent smoking and identifying groups at risk for persistent smoking.

Numerous studies over the past two decades have demonstrated that individuals with histories of cigarette smoking in first-degree relatives are even more likely to be persistent smokers than those without such histories (Heath & Martin 1993; Chassin *et al.* 1994, 1996; Kendler *et al.* 1999). Although shared environmental influences are clearly a factor, several studies have demonstrated that persistent smoking behavior has a substantial heritable component as well. For example, Heath & Martin (1993) reported heritability estimates of 0.53, while Kendler *et al.* (1999) reported heritability estimates as high as 0.72. These studies, while underscoring the importance of both environmental and genetic factors in contributing to persistent smoking, did not elucidate mechanisms that underlie the relations between family history and persistence. A better understanding of such mechanisms might provide new targets for interventions to enhance smoking cessation attempts.

It is now well established that stress plays a major role in persistent substance use and relapse (see Sinha 2001 for a review). Indeed, clinical reports indicate that stress is frequently cited as a reason for relapse to substance use (McKay *et al.* 1995; Sinha 2001). Empirical data in the tobacco literature have demonstrated that an acute laboratory stressor can trigger powerful craving reactions (e.g. Perkins & Grobe 1992) which may, in turn, be related to persistent smoking and relapse (e.g. Cohen & Lichtenstein 1990). Findings have been well corroborated in the animal literature, establishing causal links between both acute (e.g. footshock; Buczek *et al.* 1999) and chronic stressors (e.g. reared without mothers; Fahlke *et al.* 2000) and increased vulnerability to persistent substance use, including nicotine (Shaham *et al.* 1998; Buczek *et al.* 1999). Whether or not individuals at risk for persistent substance use differ in stress-induced craving has to date, not been examined.

In this study, we explored the hypothesis that smokers with histories of smoking in first-degree relatives (FH+), who are at increased risk of persistent smoking, would exhibit more powerful craving reactions to stressors than smokers without such family histories (FH-). To that end, we explored first the possibility that smokers with and without family histories differ on a variety of personal smoking variables, including their age at initiation, for how long they have been smoking, how many cigarettes per day they smoke, the strength of their addiction and the severity of their withdrawal symptoms. We then exposed them to an acute stressor and predicted that FH+

smokers would exhibit greater craving reactions than FH- smokers.

METHOD

Overview

To address the study hypotheses, we employed a classic imaginal exposure paradigm (Maude-Griffin & Tiffany 1996), in which 83 smokers were exposed to script-guided imagery of neutral and stressful subject matter, separated by a 5-minute rest. Assessments of cigarette craving were taken immediately before and after each imaginal exposure. Changes in craving and acute anxiety as a function of stressful imagery were measured, controlling for changes as a function of neutral imagery.

Participants

Eighty-three smokers recruited by advertisements placed in and around a major medical center participated in the study. To minimize participation biases, we did not oversample specifically for FH+ smokers. All participants qualified for a current DSM-IV diagnosis of nicotine dependence (American Psychiatric Association 1994) ascertained during initial telephone contact with a trained interviewer and re-ascertained in person upon arrival. In addition, to qualify as a smoker, only participants who reported smoking at least 10 cigarettes per day for at least 5 years were included in the study. To minimize sources of sample heterogeneity, we excluded participants if they reported: (1) currently being treated (or in a program) for smoking cessation, (2) having had a history of other substance abuse, (3) having had a history of hospitalization for mental illness or (4) having had a history of smoking-related illness (e.g. cancer, emphysema, chronic obstructive pulmonary disease). Mean age of the sample was 41.2 years (± 0.9 ; range 23–62). Of the participants, 56.6% were female and 55% reported earning below \$20 000.00 annually. Twenty-seven per cent of participants reported not completing high school education, 32% reported completing high school, and 41% reported some education beyond high school. Consistent with the demographics of our catchment area, ethnic background of the sample was diverse: 59% of the sample reported African-American ethnicity, 29% reported Hispanic ethnicity, 10% reported Caucasian ethnicity and 2% reported Asian/Pacific Islander ethnicity. Forty-six per cent of the sample reported never being married, 15% were currently married, 39% were separated, divorced or widowed. Participants reported smoking an average of 21.3 (± 1.1) cigarettes per day for an average of 21.5 (± 1.0) years, with an average age at initiation of 15.9 (± 0.4), a Fagerstrom score of 5.7 (± 0.3).

Measures

Demographic and smoking questionnaire

Participants completed questionnaires assessing basic demographic information (e.g. age, gender, education, income, ethnicity, marital status) and personal smoking history (e.g. age at initiation, cigarettes per day, years smoked). They also completed a self-report familial smoking pedigree which included items assessing cigarettes per day and years smoked, in reference to first-degree relatives (e.g. parent, child, sibling). Investigators were blind to participants' family history status. A positive smoking history in a family member was defined in the same fashion as for the participant (minimum 10 cigarettes per day for 5 years). None of the participants reported not knowing family members' smoking status, although their accuracy could not be verified independently.

Fagerstrom Test of Nicotine Dependence (FTND)

A refinement of the well-known Fagerstrom Tolerance Questionnaire (Fagerstrom 1978), this six-item instrument (Heatherton *et al.* 1991) was developed to improve upon some of the psychometric properties of the original scale. The FTND was used to assess the strength of participants' addiction. The instrument has good reliability and validity (Heatherton *et al.* 1991).

Minnesota Nicotine Withdrawal Questionnaire (MNWQ)

This eight-item instrument was used to assess withdrawal symptoms over the last 24 hours. The MNWQ is considered the withdrawal questionnaire of choice, according to a recent review (Patten & Martin 1996), and has good reliability and validity (Hughes & Hatsukami 1986; Hughes *et al.* 1991).

Profile of Mood States—Short Version (POMS-SV)

This 47-item instrument measures transient levels of distress. This short version (Shacham 1983) of the classic mood adjective checklist (McNair *et al.* 1971) assesses six affective dimensions and provides a total distress score. Test–retest reliability for each of the subscales have been found to be consistent with the long version, and have been demonstrated to have excellent criterion validity (DiLorenzo *et al.* 1999).

Brief Symptom Inventory (BSI)

This 53-item scale assesses general psychological distress on nine separate symptom dimensions (somatization, obsessive compulsive, interpersonal sensitivity, depres-

sion, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism) and also provides a global index of distress. The reliability of the scale has been demonstrated by internal consistency and test–retest reliability (Derogatis & Spencer 1982). Participants were asked to endorse symptoms they may have had over the past month.

Bett's Vividness of Mental Imagery Questionnaire—Shortened form (BVMIQ)

The 35-item BVMIQ was used to assess vividness of mental imagery as a potential covariate for imagery-induced stress. The questionnaire has been used extensively in imagery research, and has been shown to have good psychometric properties (Sheehan 1967).

Acute anxiety

To rapidly assess acute anxiety responses to stress exposures, we employed a visual analog scale (Bond & Lader 1974; Cella & Perry 1986) consisting of a 100-mm line anchored on the left by 'Not at all anxious' and on the right by 'As anxious as can be'. Participants responded to the question 'How anxious are you feeling right now?' and 'How anxious did you feel during the scene?' (pre- and postimagery, respectively) by striking a line across the continuum. VAS measures have been found to be good measures of subjective feelings (Bond & Lader 1974), and have been used extensively in other studies measuring changes in anxiety (e.g. Krystal *et al.* 1993).

Cigarette Craving Questionnaire

Improving on the use of single-item craving assessments, and following the recommendations of Kozlowski and colleagues (Kozlowski & Wilkinson 1987; Kozlowski *et al.* 1989) that craving be assessed using multiple descriptors (e.g. crave, urge, desire), this brief, five-item, 0–100 instrument is designed specifically to make rapid assessments of craving during experimental manipulations. The instrument is adapted from the 'desire to smoke' subscale of the Questionnaire on Smoking Urges (Tiffany & Drobes 1990), and has been used as an outcome measure in previous studies (e.g. Shadel *et al.* 2001). As above, pre- and postimagery versions of the instrument assessed craving 'right now' and 'during the scene', respectively. The instrument evidenced high levels of internal consistency ($\alpha = 0.97$) at all four time-points (pre–post neutral and stress imagery).

Procedures

Potential participants who responded to advertisements were screened via telephone to determine eligibility. The

study was described over the telephone and, if eligible, participants were scheduled for the study. In sum, two-thirds of those who responded were eligible, and 100% of those were interested in participating. Three people who were scheduled did not show up for the study. To avoid variation in craving associated with time of day, participants were scheduled to arrive between 8 and 10 a.m. for the study session. To avoid floor effects in cigarette craving, they were instructed not to smoke that morning until after completing the study. Upon arriving at the study site, participants provided written informed consent. To ascertain overnight abstinence, participants were given a CO breath test using a MicroCO monitor (MicroDirect, Lewiston, ME, USA), and to be conservative, CO levels were considered as a potential covariate in the analyses (see below). None of the participants was excluded on the basis of their CO levels. Participants then completed the demographic, smoking history and familial pedigree questionnaires, the FTND, MNWQ, BSI, POMS and the BVMIQ. The experimenter was blind to participants' FH status. Following the methodology of Tiffany & Drobes (1990), we then described the classic script-guided imagery task: participants were to listen to a 60-second script and try to imagine the scene as vividly as possible, followed by a 30-second silent period, during which they were to continue imagining the scene, drawing on personal experiences if they wished. Before beginning the exposures, participants were read a practice script (a trip to the grocery store) to familiarize them with the task. They then completed the pre-imagery craving and anxiety measures and were exposed to a neutral imagery scene involving the changing of a lightbulb. Craving and anxiety were assessed again immediately after the scene. To minimize the possibility of carryover effects, we included a 5-minute recovery period, during which time participants listened to classical music. After the recovery period, participants completed the pre-imagery craving and anxiety scales, listened to the stress script, which described a trip to the dentist (Maude-Griffin & Tiffany 1996) and completed the postimagery craving and anxiety scales. To minimize further the possibility of carryover effects, the stress imagery was always administered after the neutral imagery. The procedures took approximately 2 hours to complete. Upon completion of the study participants were thanked for their participation, offered referrals for smoking cessation interventions and paid an honorarium of \$75.00 for their time.

Data analysis

To address the study hypothesis, we first categorized participants on the basis of their family histories. To take a conservative approach to family history (e.g. Conrod *et al.* 1998), only participants with at least two first-degree rel-

atives who smoked (i.e. parent, child, sibling) were categorized as having a family history of smoking (FH+; $n = 39$) and all others as FH- ($n = 44$). Participants with only one first-degree relative ($n = 18$), more likely to indicate a 'sporadic' case of family history, were considered part of the FH- group. Preliminary analyses comparing these subgroups within the FH- group yielded no significant stress-induced craving effects ($P < 0.61$). We used the same criteria for familial smoking history (at least 10 cigarettes/day for at least 5 years) that we used for personal smoking history.

FH+ and FH- groups were compared on demographic variables to identify relevant covariates to include in the primary analyses. Next, the two groups were tested for possible differences on personal smoking variables. To address the primary study hypothesis, we then performed a classic mixed design group (FH+/FH-) \times time (pre-stress/post-stress) ANOVA on distress levels and craving associated with the stress imagery, including [baseline-adjusted] change scores of craving associated with the neutral imagery as a covariate. We performed planned pairwise comparisons using covariate-adjusted means, and we corrected these P -values for Type I error using the Bonferroni method.

RESULTS

Background variables

FH+ and FH- smokers did not differ on age; $t(82) = 0.34$, $P < 0.74$, gender; $\chi^2(1) = 0.85$, $P < 0.35$, education; $\chi^2(1) = 0.14$, $P < 0.71$, income; $\chi^2(1) = 0.16$, $P < 0.68$, ethnic background; $\chi^2(2) = 0.21$, $P < 0.90$; or marital status; $\chi^2(2) = 1.14$, $P < 0.56$. In addition, to address the possibility that the two groups would differ in background levels of psychological distress that might impact on responses to an acute stressor, we compared FH+ and FH- smokers on the BSI and POMS. Findings indicated that there were no significant group differences on the BSI total score; $t(82) = 1.20$, $P < 0.23$, or the POMS total score; $t(82) = 0.15$, $P < 0.88$. Finally, to rule out the possibility that groups would differ on imagery skill, we tested for differences on the BVMIQ, and found no significant difference; $t(82) = 0.53$, $P < 0.60$. As a result, none of these variables was included as a covariate in the primary analyses.

Personal smoking variables

To explore the possibility that the FH+ and FH- groups differed on variables related to their smoking behavior, we ran a series of t -tests. Findings indicated that FH+ and FH- smokers began smoking at comparable ages;

$t(82) = 0.91$, $P < 0.37$, smoked a comparable number of cigarettes per day; $t(82) = 0.50$, $P < 0.62$, and had been smoking for a comparable number of years; $t(82) = 0.71$, $P < 0.48$. In addition, the two groups did not differ on the FTND, a measure of habit strength; $t(82) = 0.94$, $P < 0.35$, nor did they differ on the MNWQ, a measure of the strength of withdrawal symptoms; $t(82) = 1.00$, $P < 0.32$. Finally, FH+ and FH- smokers did not differ on expired CO at the time of the study, an objective indicator of recent smoking; $t(82) = 0.92$, $P < 0.36$.

Manipulation check: acute anxiety

As expected, the group \times time ANOVA for the imaginal stress scene yielded a significant main effect of time (pre-post stress imagery) on acute anxiety, confirming that exposure to the stressful imagery increased anxiety $F_{1,81} = 45.26$, $P < 0.0001$. The mean anxiety rating pre-imagery was 29.3 (± 4.2), compared to 57.6 (± 4.3) post-imagery, for a mean increase of 28.3 units. In addition, there was no group \times time interaction; $F_{1,81} = 1.68$, $P < 0.20$, indicating that FH+ and FH- smokers were not differentially distressed by the imaginal stressor.

Cigarette craving

Consistent with previous literature, there was a significant main effect of time (pre-post-stress imagery) on cigarette craving, such that exposure to the stress imagery increased craving; $F_{1,81} = 7.17$, $P < 0.009$, even after controlling for variability in response to neutral imagery. Consistent with the study hypothesis, we found a significant group \times time interaction; $F_{1,81} = 4.81$, $P < 0.03$, such that FH+ smokers exhibited increases in craving as a result of stressful imagery, while FH- smokers did not (see

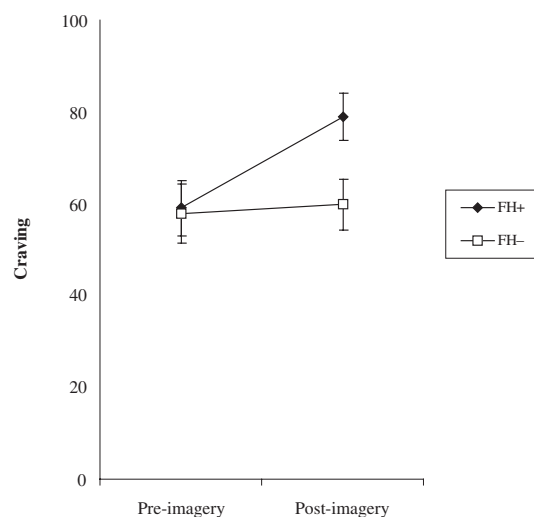


Figure 1 FH- and FH+ smokers' stress-induced craving responses

Fig. 1). Planned pairwise comparisons confirmed that, as a group, FH+ smokers had a mean 20.8 (± 5.4)-point increase in craving; $t(82) = 3.75$, $P < 0.0006$, compared to only a 1.6 (± 6.0)-point increase; $t(82) = 0.27$, $P < 0.78$ among FH- smokers. As expected based on these results, anxiety and craving reactions (baseline-adjusted change scores) were only modestly correlated ($r = 0.34$, $P < 0.05$).

DISCUSSION

This study aimed to identify differences between smokers with and without histories of smoking in first-degree relatives that might explain their differential risks of persistent smoking. In this study, we found that while FH+ and FH- smokers displayed comparable levels of anxiety after a laboratory stressor, only FH+ smokers exhibited significant craving reactions in response to the stressor. Findings are particularly intriguing, because the FH+ and FH- groups were statistically indistinguishable in the number of cigarettes smoked daily, number of years as a smoker, age at initiation, strength of addiction or strength of withdrawal symptoms. This dissociation suggests that FH+ and FH- smokers' differential levels of stress-induced craving are not simply secondary to differential smoking histories.

Findings are consistent with previous literature that has documented increased craving in response to laboratory stressors (Perkins & Grobe 1992), but add to this literature the possibility that the phenomenon is particularly (if not exclusively) salient among FH+ smokers, who made up nearly half of our unselected sample. It is possible that previous literature demonstrating stress-induced craving may have yielded different effects if FH+ and FH- subsets were examined separately. Our findings suggest an area for future research to better understanding of the pathway from family history to persistent smoking. The findings also raise the possibility that interventions revolving around stress management may be particularly appropriate for FH+ smokers. We should also note that, to our knowledge, this is the first study to replicate stress-induced craving in a predominantly minority sample, suggesting that the phenomenon is not limited to Caucasian smokers. The possibility that ethnic differences moderate the FH relations observed in the present study needs to be explored further.

While additional research is clearly needed to formally examine the mechanism(s) underlying relations between family history and stress-induced craving, we suggest several possible explanations. First, it is possible that FH+ smokers may have learned from family members at an early age that lighting-up is a method of managing stress. Individuals not exposed to this strategy, on the other

hand, may be less likely to use cigarettes to manage their stress.

Secondly, it is possible that stressors become a conditioned stimulus eliciting craving, a conditioned response. Indeed, it is well established in the literature that cues (e.g. the sight of one's preferred brand of cigarettes, walking by a preferred smoking spot) associated with smoking can become conditioned stimuli that elicit craving (see Stewart, deWit & Eikelboom 1984). Whether or not stress operates as a conditioned stimulus in a similar fashion, and whether or not FH+ smokers are particularly susceptible to this type of conditioning remains an intriguing possibility. This possibility is particularly noteworthy, as accumulating evidence points to the fact that biochemical substrates subserving the processes of cue-elicited craving are similar to the processes subserving stress-induced craving (Self 1998; Everitt *et al.* 1999; Weiss *et al.* 2001). Finally, it is possible that FH+ smokers are more reactive to stress in general, and that our self-report measure of anxiety and/or our use of only one type of stressor was not sufficiently powerful to detect group differences. The use of alternative stressors (e.g. speech task) and response measures (e.g. physiological indices) in future research would address this possibility.

The present results do not address whether FH+/FH- differences in stress-induced craving have a genetic, environmental or combined etiology; formal behavioral genetic studies of this process are necessary to tease apart these factors. The possibility of a genetic contribution is particularly intriguing in light of emerging data in the animal literature demonstrating that genetic variations in stress hormone levels (corticotropin-releasing hormone; CRH) contribute to stress-induced persistent alcohol consumption (Sillaber *et al.* 2002). In addition, recent data have linked persistent smoking to genetic factors associated with dopamine, a neurotransmitter thought to subserve, among other functions, stress-induced craving responses (see Self 1998 for a review). Blum *et al.* 2000) has argued that genetic differences in the opioid system may also play a role in craving. To the extent that familial risk for persistent smoking is driven by genetic factors, the roles of dopamine and CRH may implicate stress-induced craving as a potential mechanism. This, of course, is in addition to other genetic and environmental factors that may contribute to FH+ smokers' increased levels of persistence.

In the present study, smokers were not followed longitudinally to assess any changes in smoking behavior. As a result, we are unable to assert formally that the observed findings mediate relations between family history and smoking behavior. Follow-up longitudinal studies are still needed to evaluate the utility of observed differences in stress-induced craving in understanding variability in smoking behavior. In addition, because we did not coun-

terbalance the presentation of neutral and stress imagery scenes, we cannot rule out the possibility of order effects. It is highly unlikely, however, that order of presentation would have affected FH+ and FH- smokers differentially.

We should also note that the present findings are based on a relatively small number of participants, which precludes further dissection of subsets of interest (e.g. FH- with one first-degree relative smoker, FH+ whose relatives quit during the participants' childhood). In addition, we employed only one type of stressor (dentist imagery) in this study, assessed only one outcome (self-reported craving), defined family history in a very specific fashion and relied on self-report to establish family history. A larger study employing different types of well-established laboratory stressors (e.g. mental arithmetic), multiple outcomes (e.g. psychophysiological reactivity), other definitions of family history (e.g. accounting for first- and second-degree relatives, using measures of family history density, assessing the degree of contact between probands and family members) and independent confirmation of family history status would be necessary to confirm definitively applicability of the present results more broadly. It should also be noted that we chose to take a conservative approach, including as FH+ smokers those participants whose first-degree relatives had smoked, without excluding those whose family members' quit successfully. It is possible that using this stricter definition of family history would have yielded even greater group differences.

In addition, we relied on self-report methodology to exclude individuals with histories of other drug use, which is likely to underestimate true levels in the sample. In addition, we did not assess family histories of alcoholism in the sample. Given the accumulating evidence for the common etiology of abuse for various substances (e.g. Blum *et al.* 2000), we cannot rule out the possibility that observed effects are due to familial risk for other substances. This intriguing possibility warrants further attention, as knowledge about mechanisms underlying persistent use of one substance may become highly relevant in understanding use of others.

Finally, although we demonstrated that FH+ smokers exhibit greater stress-induced craving than FH- smokers in an experimental setting, we have not yet demonstrated that stress experienced in the 'real world' causes particularly high craving in this population, nor have we demonstrated that the phenomenon predicts persistent smoking. It should be noted, however, that the connection between 'real world' stressors, craving and persistent smoking has been reported in the literature by some investigators (e.g. Cummings *et al.* 1985), although additional research is warranted to fully document the pathway from family history to persistent smoking. It is also important to note that there may be subsets of FH-

smokers who demonstrate high levels of stress-induced craving as well, a phenomenon not detected by our analysis of group means. Further research is warranted to identify factors that may further moderate relations between stress and craving.

In summary, the present study found that FH+ smokers exhibited greater stress-induced craving responses to a laboratory stressor than did FH- smokers, even though they did not differ on key smoking-related variables such as the number of cigarettes smoked per day and the strength of their addiction. To the extent that these findings reflect a mechanism through which FH+ smokers are at increased risk of persistent smoking, stress management-related interventions may be particularly appropriate for this sizeable subset of smokers. Future research might profitably focus on better understanding the source(s) of differential stress-induced craving so that interventions can be further refined and targeted.

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REFERENCES

- American Cancer Society (2001) *Cancer Facts and Figures: 2001*. New York: American Cancer Society.
- American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders, IV (DSM-IV)*. Washington, DC: American Psychiatric Association.
- Blum, K., Braverman, E., Holder, J., Lubar, J. F., Monastra, V., Miller, D., Lubar, J. O., Chen, T. & Comings, D. (2000) Reward deficiency syndrome: a biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive disorders. *Journal of Psychoactive Drugs*, **32**, 1–112.
- Bond, A. & Lader, M. (1974) The use of analogue scales in rating subjective feelings. *British Journal of Medical Psychology*, **47**, 211–218.
- Buczek, Y., Le, A., Stewart, J. & Shaham, Y. (1999) Stress reinstates nicotine seeking but not sucrose solution seeking in rats. *Psychopharmacology*, **144**, 183–188.
- Cella, D. & Perry, S. (1986) Reliability and concurrent validity of three visual analogue mood scales. *Psychological Reports*, **59**, 827–833.
- Chassin, L., Presson, C., Rose, J. & Sherman, S. (1996) The natural history of cigarette smoking from adolescence to adulthood: demographic predictors of continuity and change. *Health Psychology*, **15**, 478–484.
- Chassin, L., Presson, C., Sherman, S. & Mulvenon, S. (1994) Family history of smoking and young adult smoking behavior. *Psychology of Addictive Behaviors*, **8**, 102–110.
- Cohen, S. & Lichtenstein, E. (1990) Perceived stress, quitting smoking and smoking relapse. *Health Psychology*, **9**, 466–478.
- Conrod, P., Pihl, R. & Vassileva, J. (1998) Differential sensitivity to alcohol reinforcement in groups of men at risk for distinct alcoholism subtypes. *Alcoholism: Clinical and Experimental Research*, **22**, 585–597.
- Cummings, K., Jaen, C. & Giovino, G. (1985) Circumstances surrounding relapse in a group of recent exsmokers. *Preventive Medicine*, **14**, 195–202.
- Derogatis, L. & Spencer, P. (1982) *The Brief Symptom Inventory (BSI) Administration Scoring and Procedures Manual—I*. Riderwood, MD: Clinical Psychometric Research.
- DiLorenzo, T., Bovbjerg, D., Montgomery, G., Valdimarsdottir, H. & Jacobsen, P. (1999) The application of a shortened version of the profile of mood states in a sample of breast cancer chemotherapy patients. *British Journal of Health Psychology*, **4**, 315–325.
- Everitt, B., Parkinson, J., Olmstead, M., Arroyo, M., Robledo, P. & Robbins, T. (1999) Associative processes in addiction and reward. The role of amygdala-ventral striatal subsystems. *Annals of the New York Academy of Sciences*, **877**, 412–438.
- Fagerstrom, K. (1978) Measuring degree of physical dependence to tobacco smoking with reference to individualization of treatment. *Addictive Behaviors*, **3**, 235–241.
- Fahlke, C., Lorenz, J., Long, J., Champoux, M., Suomi, S. & Higley, J. (2000) Rearing experiences and stress-induced plasma cortisol as early risk factors for excessive alcohol consumption in non-human primates. *Alcoholism: Clinical and Experimental Research*, **24**, 644–650.
- Heath, A. & Martin, N. (1993) Genetic models for the natural history of smoking: evidence for a genetic influence on smoking persistence. *Addictive Behaviors*, **18**, 19–34.
- Heatherston, T., Kozlowski, L., Frecker, R. & Fagerstrom, K. (1991) The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction*, **86**, 1119–1127.
- Hughes, J., Gust, S., Skoog, K., Keenan, R. & Fenwick, J. (1991) Symptoms of tobacco withdrawal. A replication and extension. *Archives of General Psychiatry*, **48**, 52–59.
- Kendler, K., Neale, M., Sullivan, P., Corey, L. & Gardner, C. (1999) A population-based twin study in women of smoking initiation and nicotine dependence. *Psychological Medicine*, **29**, 299–308.
- Hughes, J. & Hatsukami, D. (1986) Signs and symptoms of tobacco withdrawal. *Archives of General Psychiatry*, **43**, 289–294.
- Kozlowski, L., Mann, R., Wilkinson, D. & Poulos, C. (1989) 'Cravings' are ambiguous: ask about urges or desires. *Addictive Behaviors*, **14**, 443–445.
- Kozlowski, L. & Wilkinson, D. (1987) Use and misuse of the concept by alcohol, tobacco, and drug researchers. *British Journal of Addiction*, **82**, 31–36.
- Krystal, J., Seibyl, J., Price, L., Woods, S., Heninger, G., Aghajanian, G. & Charney, D. (1993) Chlorophenylpiperazine effects in neuroleptic-free schizophrenic patients. Evidence implicating serotonergic systems in the positive symptoms of schizophrenia. *Archives of General Psychiatry*, **50**, 624–625.
- Maude-Griffin, P. & Tiffany, S. (1996) The production of smoking urges through imagery: The impact of affect and smoking abstinence. *Experimental and Clinical Psychopharmacology*, **4**, 198–208.

- McKay, J., Rutherford, M., Alterman, A., Cacciola, J. & Kaplan, M. (1995) An examination of the cocaine relapse process. *Drug and Alcohol Dependence*, **38**, 35–43.
- McNair, D., Lorr, M. & Droppelman, L. (1971) *Manual: Profile of Mood States*. San Diego, CA: EDITS/Educational and Industrial Testing Service, Inc.
- Patten, C. & Martin, J. (1996) Measuring tobacco withdrawal: a review of self-report questionnaires. *Journal of Substance Abuse*, **8**, 93–113.
- Perkins, K. & Grobe, J. (1992) Increased desire to smoke during acute stress. *British Journal of Addiction*, **87**, 1037–1040.
- Public Health Service (2000) *Reducing Tobacco Use*. A report of the Surgeon General. Washington, DC: Department of Health and Human Services.
- Self, D. (1998) Neural substrates of drug craving and relapse in drug addiction. *Annals of Medicine*, **30**, 379–389.
- Shacham, N. (1983) A shortened version of the profile of mood states. *Journal of Personality Assessment*, **47**, 305–306.
- Shadel, W., Niaura, R. & Abrams, D. (2001) Does completing a craving questionnaire promote increased smoking craving? An experimental investigation. *Psychology of Addictive Behaviors*, **15**, 265–267.
- Shaham, Y., Erb, S., Leung, S., Buczek, Y. & Stewart, J. (1998) CP-154,526, a selective, non-peptide antagonist of the corticotrophin-releasing factor receptor attenuates stress-induced relapse to drug seeking in cocaine and heroin-trained rats. *Psychopharmacology*, **137**, 184–190.
- Sheehan, P. (1967) A shortened form of Bett's questionnaire upon mental imagery. *Journal of Clinical Psychology*, **23**, 386–389.
- Sillaber, I., Rammes, G., Zimmerman, S., Mahal, B., Zieglansberger, W., Wurst, W., Holsboer, F. & Spanagel, R. (2002) Enhanced and delayed stress-induced alcohol drinking in mice lacking functional CRH1 receptors. *Science*, **296**, 931–933.
- Sinha, R. (2001) How does stress increase risk of drug abuse and relapse? *Psychopharmacology*, **158**, 343–359.
- Stewart, J., de Wit, H. & Eikelboom, R. (1984) Role of unconditioned and conditioned drug effects in the self-administration of opiates and stimulants. *Psychological Review*, **91**, 251–268.
- Tiffany, S. & Drobes, D. (1990) Imagery and smoking urges: the manipulation of affective content. *Addictive Behaviors*, **15**, 531–539.
- Weiss, F., Ciccocioppo, R., Parsons, L., Katner, S., Liu, X., Zorrilla, E., Valdez, G., Ben-Shahar, O., Angeletti, S. & Richter, R. (2001) Compulsive drug-seeking behavior and relapse. Neuroadaptation, stress, and conditioning factors. *Annals of the New York Academy of Sciences*, **937**, 1–26.
- Zhu, S., Melcer, T., Sun, J., Rosbrook, B. & Pierce, J. (2000) Smoking cessation with and without assistance: a population-based analysis. *American Journal of Preventive Medicine*, **18**, 305–311.