

The Effects of a Psychological Stressor on Cigarette Smoking and Subsequent Behavioral and Physiological Responses

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ABSTRACT

Stressful stimuli have been reported to trigger increased smoking. The present experiment involved induction of differing levels of performance anxiety in 10 male smokers. In the high anxiety condition, subjects performed mental arithmetic with competitive pressure; in the low anxiety control condition, no competitive pressure was applied. Both sessions included smoking and sham smoking trials. Scores on the Profile of Mood States showed significantly more anxiety on the day of the high anxiety session than on the day of the low anxiety session, confirming the effectiveness of anxiety-induction procedures. Nicotine intake as inferred from topographical measures was significantly greater under conditions of high anxiety. Greater post-smoking decreases in anxiety in the high-anxiety condition, as compared to the low-anxiety condition and the two sham-smoking conditions, were also observed. Both smoking and anxiety level significantly increased heart rate; lack of statistical interaction suggests additive rather than potentiating effects. Peripheral skin temperature rose in response to mental arithmetic, a trend counteracted by smoking. No significant smoking-related changes in performance were found.

DESCRIPTORS: Anxiety, Heart rate, Mental arithmetic, Reactivity, Smoking, Stress.

Chronic or repeated overstimulation of the sympathetic adrenomedullary system is believed to be associated with cardiopathology (Herd, 1981). Studies on both humans and infrahumans suggest that potentially cardiopathic physiological states can be evoked by a variety of stressful stimuli. Exposure to psychological stressors like mental arithmetic, video games, an aversive reaction time task, harassment, and threat of shock, for example, elicit substantial elevations in heart rate, blood pressure, and circulating catecholamines (Frankenhaeuser, 1971, 1983; Henry, 1983; Herd, 1981; Langer et al., 1985; Obrist et al., 1978; Turner & Carroll, 1985).

Smoking, like stress, is believed to increase the risk of coronary heart disease (Su, 1982), a phenomenon attributable at least in part to the pronounced sympathomimetic effects of nicotine (Voll & Koelle, 1975). Stimulation of sympathetic neurons and release of adrenomedullary catechol-

amines by the drug (Kershbaum, Pappajohn, Bellet, Hirabayashi, & Shafiiha, 1968; Westfall & Watts, 1964) have been shown to produce metabolically-exaggerated increases in diastolic and systolic blood pressure, cardiac rate, myocardial oxygen demand, and vasoconstriction (Cryer, Haymond, Santiago, & Shah, 1976; Domino, 1973; Herxheimer, Griffiths, Hamilton, & Wakefield, 1967; Jarvik, 1979; Trap-Jensen, Carlsen, Svendsen, & Cristensen, 1979).

The combined effects of the two putative cardiopathogens, psychological stress and smoking, have not been examined extensively. Recent studies by Dembroski, MacDougall, and colleagues (Dembroski, MacDougall, Cardozo, Ireland, & Krug-Fite, 1985; MacDougall, Dembroski, Slaats, Herd, & Eliot, 1983), however, demonstrated that the acute cardiovascular effects of smoking followed by application of a mild stressor (playing a video game) are additive in the case of heart rate; the combination may have a potentiating effect upon blood pressure.

The possible interaction of stress and smoking is of more than theoretical interest because psychological stressors may in fact trigger increased smoking. Analysis of the circumstances surrounding recidivism (Pomerleau, Adkins, & Pertschuk,

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1978; Shiffman, 1982) as well as retrospective examination of factors associated with craving (Myrsten, Elgerot, & Edgren, 1977) suggest that dysphoric states, particularly anxiety, frequently precede smoking. Significant increases in smoking have been reported in response to a variety of laboratory stressors, including shock (Schachter et al., 1977), public speaking (Dobbs, Strickler, & Maxwell, 1981; Rose, Ananda, & Jarvik, 1983), and aversive white noise (Golding & Mangan, 1982).

Investigations of the relationship between stress and smoking tend to be marred by a failure to assess adequately the subjective and physiological concomitants of situational manipulations, and in particular to differentiate among levels of anxiety. For example, whether a particular task, as observed either in the field or in the laboratory, is experienced as stressful cannot be determined by fiat. Nicotine is capable of producing both stimulation and sedation, depending on dose and rise time in plasma, and it has been shown that smokers can regulate intake to produce the pharmacological response most appropriate to the needs of the situation (Ashton & Stepney, 1982; Pomerleau & Pomerleau, 1984); different levels of stress, then, would be expected to produce different patterns of smoking. If so, it is clearly important to identify the elements of a given situation to which a smoker is responding.

The present experiment, using a within-subjects factorial design, attempted to manipulate anxiety level in order to tease out the differential effects of induced anxiety on smoking and subsequent physiological and subjective measures. On the first day of the experiment, the high anxiety condition, each subject was asked to carry out a mental arithmetic task (serial 13 subtractions) with the understanding that he was competing for a series of graduated bonuses based on his performance. On the second day, the low anxiety control condition, the importance of performance was downplayed and attempts were made to minimize anxiety without decreasing alertness. Mental arithmetic tasks with and without competitive pressure were deemed representative of differentially stressful work-like situations encountered on a daily basis in our culture. During each of the two sessions, subjects engaged in both a smoking and a sham smoking trial. Smoking topography (total puff volume, number of puffs, and mean puff volume) was measured to determine whether conditions designed to maximize anxiety elicited different patterns of smoking and, inferentially, of nicotine intake, compared with conditions intended to minimize anxiety. Subjective anxiety was assessed immediately before and after smoking, and heart rate was monitored as an index

of cardiovascular reactivity. Peripheral skin temperature, a measure of sympathetic activity known to be affected by nicotine, which produces vasoconstriction, was also recorded.

Method

Subjects

Subjects were 10 male smokers with a mean age of 27.9 ± 2.1 (SEM) recruited from the local community by newspaper advertisement. They reported having smoked for 13.5 ± 2.3 yrs, with a mean of 27.8 ± 3.0 cigarettes per day. Mean level of plasma cotinine, a nicotine metabolite with a half-life of thirty hours, was 372.9 ± 56.6 ng/ml, characterizing them as heavy smokers (Pomerleau, Fertig, & Shanahan, 1983). Subjects were paid \$25 for completing the two sessions.

Apparatus

Subjects were seated in an easy chair about 30 cm from a console that served to signal by light and sound the beginning and end of each condition in a trial. The console was also equipped with a potentiometer calibrated on a scale of 0 to 10 and appropriately labeled to permit ongoing assessment of anxiety/relaxation. A small cassette recorder was placed directly in front of the subject. Temperature was maintained at 23°C ($\pm 4^\circ\text{C}$) and humidity at 50% ($\pm 10\%$). Experimental sequences and data acquisition were fully automated, using a 16,000-word computer (Alpha 16). Subjects were observed through a one-way mirror, and no interaction between subject and experimenter took place once a trial was underway.

Assessment of Subjective State

The Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971) was administered prior to each session to determine the success of the anxiety-induction manipulation as well as to provide assessment of the overall subjective state along the following dimensions: Composed/Anxious, Agreeable/Hostile, Elated/Depressed, Confident/Unsure, Energetic/Tired, and Clearheaded/Confused. After sessions for 3 subjects were completed, we introduced an additional measure of anxiety that could be collected immediately before and after smoking without disrupting the experimental sequence. Accordingly, the remaining 7 of the 10 subjects were asked to assess anxiety level at these times by means of an "anxiety dial" calibrated on an 11-point scale (0 = completely relaxed, 10 = intense anxiety).

Smoking Topography

In the smoking condition, the subject was asked to smoke a cigarette of his own brand through a holder designed to collect topographical data. (Use of own-brand cigarettes was felt to permit accurate self-dosing on the part of the subject, as well as to maximize verisimilitude in the collection of topographical data.) In the "sham smoking" activity control condition, the subject was asked to "puff" on a cigarette through the

holder without actually lighting up. This procedure provided a control for motor activity and for the non-pharmacological effects of smoking.

Smoking topography—puff frequency, duration, and intensity—was measured using a gauge pressure transducer (LX 160-46; National Semiconductor). Pressure changes produced by inhaling through a modified cigarette holder were transmitted to the pressure sensor via flexible plastic tubing, where they were converted to digital electric signals. Puff intensity \times puff duration, measured in arbitrary units and summed for all puffs, provided a total puff volume score, the volume of smoke reaching the mouth over a 5-min period. It should be noted that in the absence of measurements of plasma nicotine, no firm conclusions can be drawn about actual nicotine intake; nor can between-subjects variability be meaningfully interpreted, since different commercial brands have different ventilation (flow-through) characteristics. Differences between puff volume for a given subject across the two arousal conditions, however, can be regarded as a valid indicator of relative nicotine intake. This contention is supported by the findings of Herning, Jones, Benowitz, and Mines (1983), who reported significant increases in the predictability of blood nicotine levels when topographical data were added to an assessment of intake based on the nicotine content of commercial cigarettes.

Physiological and Biochemical Measures

Heart rate was measured continuously using three Dyna/trace pregelled silver/silver chloride electrodes placed on the left and right ribcage and upper left arm (ground), with Med Associates physiological modules. Peripheral (digit) temperature was obtained using a Yellow Springs temperature sensor taped to the left index finger. Blood samples for cotinine were withdrawn from the left median antecubital vein and centrifuged at 2500 rpm for 15 min at 4°C. Aliquots of plasma were maintained at -70°C. Frozen samples were subsequently packed in dry ice and shipped to the American Health Foundation (Valhalla, NY) for radioimmunoassay. Inter-assay and intra-assay coefficients of variation were 6 percent, and the lower limit of detectability was 0.37 ng/ml (Haley, Axelrod, & Tilton, 1983; Hill, Haley, & Wynder, 1983).

Procedure

Subjects were scheduled for two sessions one or two days apart. Shortly before his first scheduled session, each subject was called to confirm his appointment. At that time, the mental arithmetic task (subtracting 13's successively from a randomly chosen 4-digit number) was explained to the subject, and he was briefly tested to ensure that he understood the instructions. He was asked to practice the task and informed that on the first day he would be competing for bonuses of \$50, \$40, \$30, \$20, and \$10, to be awarded to the 5 highest scorers. (Score consisted of the total number of answers minus the total number of wrong answers. A "derailment" counted only once as an error; subsequent answers were accepted if they were 13 less than the previous answer.) He was further requested to ab-

stain from coffee after lunch (to avoid contamination by the stimulant effects of caffeine) and to smoke just before arrival (to ensure a standard 30-minute deprivation at the start of the session).

All sessions were run between 1500 and 1800 hours, a time when resting cortisol is low and stable and when subjects are unlikely to be in a state of nicotine or caffeine withdrawal. In the high anxiety session (always run on the first day to take advantage of the anxiety-provoking effects of unfamiliarity), each subject was asked to sit in the experimental chair, and the requirements of the experiment were explained in detail, in a manner designed to maximize anxiety (i.e., the experimenter adopted a formal and distant manner). The subject was then asked to sign an informed consent form, following which EKG electrodes and temperature sensor were attached, a blood sample was withdrawn for analysis of plasma cotinine, and the POMS was administered. The experimental sequence was then reviewed briefly, and the tape recorder was activated.

The session consisted of a smoking and a sham smoking trial, counterbalanced across subjects, with competitive pressure applied. Each trial included 5 min of mental arithmetic, followed by 5 min of cigarette manipulation (smoking or sham smoking), followed by 5 more min of mental arithmetic. An intertrial interval of 25 min was maintained in order to ensure half an hour's nicotine deprivation if the first trial involved smoking.

The second (low anxiety) session proceeded in identical fashion, except that the blood drawing and informed consent procedures were eliminated, the order of trials (sham vs. smoking) was reversed, a less aloof manner was adopted by the experimenter, and no competitive pressure involving monetary bonuses was applied. In contrast to the Anxiety session, the subject was supplied with neutral reading material (*National Geographic*) before the session and during the intertrial interval.

Results

The primary data analyses were conducted using a 2×2 repeated measures ANOVA (BMDP2V; Dixon, 1983). Anxiety (two levels, high and low) and Cigarette (two levels, smoking and sham smoking) were within-subject factors; where appropriate, time, pre/post, and task/no task were used as additional within-subject factors. All analyses included an initial evaluation for order effects, using Counterbalancing Sequence as a between-groups variable; some analyses were repeated omitting the between-groups variable in instances where the order effect was too small to reduce error variance. Directional (one-tailed) hypotheses were tested when supported by the literature cited and unless otherwise noted are reflected in the statistics that follow.

Pre-session POMS were analyzed to confirm that attempts to differentiate and operationalize the two arousal conditions had been successful. Mean scores

on all subscales of the POMS were lower (denoting greater negative affect) during the high anxiety condition than during the low anxiety condition; for four of the six subscales, these differences were significant using paired *t*-tests (Composed/Anxious: $t(9)=3.58$, $p<.005$; Confident/Unsure: $t(9)=3.32$, $p<.005$; Energetic/Tired: $t(9)=2.64$, $p<.05$; Clear-headed/Confused: $t(9)=3.44$, $p<.005$). It is worth noting that means for the second day actually suggest less subjective distress on all subscales than experienced during an *ad lib* take-home administration of the tests.

To assess the possible impact of practice or order effects, anxiety dial and heart rate data for the pre-smoking/sham smoking segments of the first trials of the two days (combined), regardless of whether the subject was to smoke or sham smoke, were compared with the corresponding segments of the second trials, using paired *t*-tests. Mean anxiety dial readings taken just before smoking or sham smoking on the first trial of the two days ($6.1 \pm .4$) did not differ significantly from corresponding readings for the second trial ($6.2 \pm .6$), whereas means for the two trials on the high-anxiety day ($6.7 \pm .5$) were significantly higher than those for the low-anxiety day ($5.6 \pm .4$; $t(6)=1.95$, $p<.05$). Similarly, mean heart rate over the first 5-min mental arithmetic segments for the first trials of both days (85.8 ± 3.2) did not differ significantly from mean heart rate for the corresponding segments of the second trials (83.8 ± 2.9). By contrast, mean heart rate during the first 5-min segments of the two trials on the high-anxiety day (86.7 ± 3.1) was significantly higher than the mean for the corresponding segments on the low-anxiety day (82.9 ± 3.1 ; $t(9)=2.39$, $p<.05$).

Topography data for the high and low anxiety conditions, smoking trials only (shown in Figure 1), were subjected to a paired *t*-test. Total puff vol-

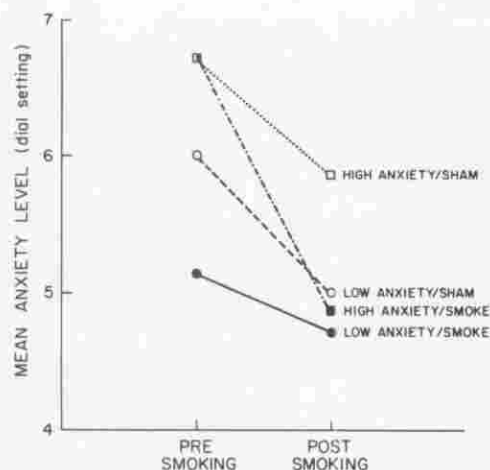


Figure 2. Mean anxiety dial readings pre- and post-smoking over 4 trials: high anxiety/sham, high anxiety/smoke, low anxiety/sham, and low anxiety/smoke; $N=7$.

ume in the High Anxiety condition was significantly higher than in the Low Anxiety condition ($t(9)=2.82$, $p<.01$). Puff frequency, though slightly lower during the low anxiety condition, did not differ significantly across the two conditions, suggesting that differences in puff volume were accounted for by greater intake per puff in the high anxiety condition rather than by higher puff frequency.

Pre- and post-smoking anxiety dial data for 7 subjects are shown in Figure 2. Post-smoking scores for 7 subjects were subjected to a 2(high vs. low anxiety) \times 2(smoking vs. sham smoking) ANCOVA, covarying pre-smoking values in order to focus on the subjective change produced by smoking. Adjusted cell means for the low anxiety condition showed a strong trend toward lower values than means for the high anxiety condition ($F(1/5)=3.64$, $p<.06$); adjusted cell means for smoking were significantly lower than for sham smoking ($F(1/5)=14.24$, $p<.01$).

As shown in Table 1, cell means for post-Cigarette performance increased slightly over pre-Cigarette scores except during the smoking trial in the high anxiety condition, in which the post-Cigarette score showed a small decrement. Data were highly variable, however, and no significant smoking-re-

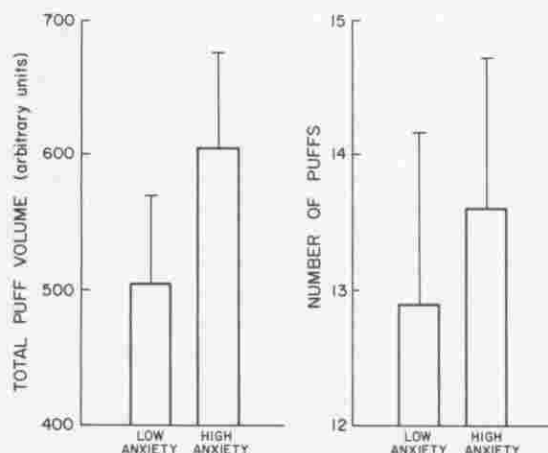


Figure 1. Smoking topography for high and low anxiety conditions: Total puff volume (arbitrary units) and number of puffs; mean scores and SEMs; $N=10$.

Table 1

Mean scores and standard errors for performance on mental arithmetic trials (number of correct subtractions)

Anxiety Conditions	Mean Mental Arithmetic Performance Scores (SEMs in Parentheses) (N=10)			
	Sham Smoke		Smoke	
	Pre	Post	Pre	Post
High	37.6 (7.4)	39.5 (8.1)	38.3 (8.3)	38.1 (8.7)
Low	47.4 (9.5)	48.3 (9.0)	45.4 (8.9)	46.3 (9.5)

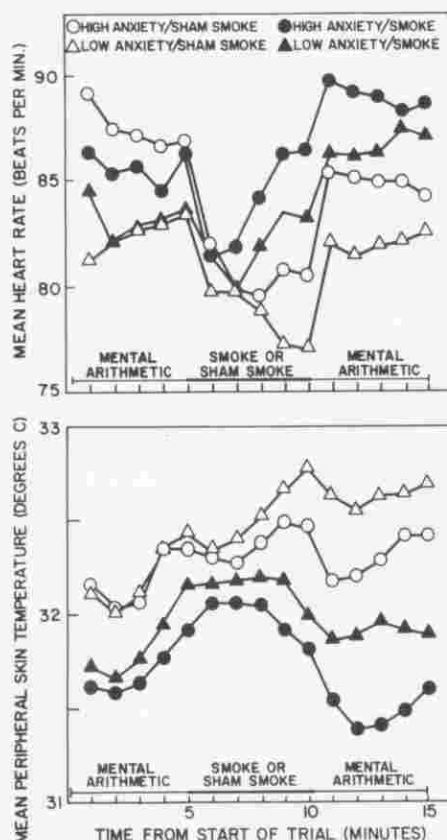


Figure 3. Mean heart rate and mean peripheral (digit) temperature as a function of time over 4 trials: high anxiety/sham, high anxiety/smoke, low anxiety/sham, and low anxiety/smoke; $N=10$.

lated changes were observed. Overall performance, independent of both changes between pre- and post-scores and Cigarette condition, was significantly better during the low anxiety condition than during the high anxiety condition ($F(1/8)=19.29$, $p<.01$, two-tailed).

The upper panel of Figure 3 presents mean heart rate data for all 4 trials. In order to focus clearly upon the several components under investigation, minutes 10 (last minute of smoking interval) and 11 (first minute of post-smoking mental arithmetic interval) were subjected to a $2(\text{low vs. high anxiety}) \times 2(\text{smoking vs. sham smoking}) \times 2(\text{task vs. no task})$, providing an index of the inherent reactivity of the mental arithmetic task in and of itself analysis. Significant main effects were detected for each of these variables (anxiety: $F(1/8)=4.88$, $p<.05$; smoking: $F(1/8)=16.49$, $p<.005$; task: $F(1/8)=19.00$, $p<.005$). No interactions were detected, indicating a lack of potentiating effects for the combination of stress and smoking.

Data for mean digit temperature appear in the lower panel of Figure 3. Analysis of minutes 6

through 15 revealed a strong Cigarette \times Time interaction ($F(9/72)=4.49$, $p<.0001$, two-tailed) with a significant linear trend ($F(1/8)=5.99$, $p<.05$, two-tailed). (Time course analysis was felt to be best suited to these data because the effects of the stress manipulations appeared to be expressed in a damped and gradual fashion.)

Discussion

Effects of the Experimental Manipulations of Anxiety

The experimental manipulations were associated with clearly differential levels of psychological distress, as shown by the POMS. In particular, anxiety (on the Composed/Anxious subscale) was higher preceding the high anxiety manipulation than preceding the low anxiety (control) manipulation. Since the high anxiety session was always run on the first day, the possibility that practice or habituation, rather than the experimental manipulation, was responsible for the observed effects cannot entirely be ruled out. If order effects were a factor, however, one would expect to find a significant decrement in self-reported anxiety and heart rate on the pre-smoking/sham smoking portions of the second trial of the day, regardless of which smoking condition came first. The fact that no significant differences within days were detected, although there were clear differences between days for both variables, increases our confidence in the experimental manipulations.

Effects of a Psychological Stressor Upon Smoking

The topography data confirm and extend previous observations of increased smoking in the presence of stressors. A marked difference in both pattern and volume of smoke and nicotine intake for the two sessions can be inferred (Herning et al., 1983), with total puff volume for the high anxiety condition exceeding that for the low anxiety condition. Lack of significant differences in puff frequency between the two anxiety conditions suggests that subjects accomplished this greater nicotine intake by taking longer, deeper drags rather than by taking more puffs.

There are several possible explanations for the observed stress-induced increase in smoking. According to the nicotine-addiction theory, smokers regulate intake to maintain levels of plasma nicotine within a certain range. Schachter (1978), observing a decrease in urinary pH in response to psychological stress, hypothesized that this pH change increased nicotine excretion, causing nicotine withdrawal and thus prompting smoking. Since

phasic plasma nicotine levels are only minimally affected by urinary pH (Rosenberg, Benowitz, Jacob, & Wilson, 1980), however, a more likely explanation lies in the direct anxiolytic effects of nicotine. These effects have been previously demonstrated in this laboratory (Pomerleau, Turk, & Fertig, 1984) and tend to be supported by the anxiety diagraph data collected in the present experiment, in which self-rated anxiety was significantly lower following smoking. Slight, non-significant decrements in performance following smoking during the high anxiety session suggest that the calming effects of nicotine may be sought even at the possible expense of performance under high levels of stress. A different performance measure, or possibly a larger N, might provide more definitive evidence for the hypothesis that nicotine stimulates in small doses but sedates in larger doses. (It cannot be determined whether the unlooked-for improvement in overall performance in the low anxiety session was due to differences in anxiety *per se* or to a practice or consolidation effect; the finding indicates, however, that mental arithmetic performance can change considerably in response to situational factors, even though it appears to be minimally affected by smoking.)

Physiological Responses to Stress-Induced Increases in Smoking

The elevation of heart rate observed in response to smoking is consistent with the known effects of nicotine (Gilbert, 1979). In addition, heart rate rose as stress level increased: Heart rate during performance demand exceeded that during the no-task (smoking/sham smoking) interval, and heart rate during the high anxiety condition was higher than during the low anxiety condition. The finding of additive effects of smoking and psychological stress upon heart rate is in accord with data presented by MacDougall et al. (1983); although potentiation of stress and smoking effects was not found in the present experiment, such effects cannot be ruled out. Thus, when heart rate is elevated by a stressor, nicotine from smoking seems to place an additional burden upon the cardiovascular system by stimulating further increases in heart rate. These effects appear to be attributable to the pharmacological

action of nicotine, since increases in pulse rate are not observed after inhalation of nicotine-free cigarette smoke (Herxheimer et al., 1967).

Nicotine is also known to cause vasoconstriction, with consequent reduction in peripheral temperature (Jarvik, 1979; Stephens, 1977); indeed, smoking exacerbates Raynaud's syndrome (Mangan & Golding, 1984). The current findings are noteworthy, however, in that they suggest a system in which an upward trend in peripheral temperature, reflecting vasodilation, is reversed by nicotine. The net effect may be potentiation by smoking of stress-induced blood pressure increases. Determination of mechanisms and the cardiodynamic significance of these findings will probably require concurrent measurement of blood pressure and circulating catecholamines as well as peripheral skin temperature.

The dose-response relationship between nicotine and the changes observed during stress cannot be specified using a self-dosing procedure, since nicotine intake is not under experimental control; this procedure, however, is representative of smoking as it occurs in the ordinary environment and is the only paradigm that permits an evaluation of self-administration in response to various environmental demands. The results of this experiment lend further support to the accumulating body of evidence indicating that psychological stress enhances sympathetic tone and cardiovascular reactivity and also provokes increased smoking. Even more critically, from the perspective of cardiopathology, when smokers do smoke in response to psychological stress, there is augmentation of cardiovascular reactions. These reactions may exacerbate detrimental health consequences associated with chronic or excessive stimulation of the sympathetic adrenomedullary system in susceptible or hyperreactive persons. Thus, the paradoxical stimulation by nicotine of simultaneous sympathetic arousal and subjective calming, which has been previously reviewed by the authors at some length (Pomerleau & Pomerleau, 1984), renders nicotine, to the extent that it is used as chronic "self-medication" for anxiety, a dangerous drug from the point of view of cardiovascular health.

REFERENCES

- Ashton, H., & Stepney, R. (1982). *Smoking: Psychology and pharmacology*. London: Tavistock Publications.
- Cryer, P.E., Haymond, M.W., Santiago, J.V., & Shah, S.D. (1976). Norepinephrine and epinephrine release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. *The New England Journal of Medicine*, 295, 573-577.
- Dembroski, T.M., MacDougall, J.M., Cardozo, S.R., Ireland, S.K., & Krug-Fite, J. (1985). Selective cardiovascular effects of stress and cigarette smoking in young women. *Health Psychology*, 4, 153-167.
- Dixon, W.J. (1983). *BMDP statistical software*. Berkeley, CA: University of California Press.
- Dobbs, S.D., Strickler, D.P., & Maxwell, W.E. (1981). The effects of stress and relaxation in the presence of stress on urinary pH and smoking behavior. *Addictive Behaviors*, 6, 345-353.
- Domino, E.F. (1973). Neuropsychopharmacology of nic-

- otine and tobacco smoking. In W.L. Dunn (Ed.), *Smoking behavior: Motives and incentives* (pp. 5-31). Washington, DC: Winston.
- Frankenhaeuser, M. (1971). Behavior and circulating catecholamines. *Brain Research*, 31, 241-262.
- Frankenhaeuser, M. (1983). The sympathetic-adrenal and pituitary-adrenal response to challenge: Comparison between the sexes. In T.M. Dembroski, T.H. Schmidt, & G. Blümchen (Eds.), *Biobehavioral bases of coronary heart disease* (pp. 91-104). Basel: S. Karger.
- Gilbert, D.G. (1979). Paradoxical tranquilizing and emotion-reducing effects of nicotine. *Psychological Bulletin*, 86, 643-661.
- Golding, J., & Mangan, G.L. (1982). Arousing and de-arousing effects of cigarette smoking under conditions of stress and mild sensory isolation. *Psychophysiology*, 19, 449-456.
- Haley, N.J., Axelrod, C.M., & Tilton, K.A. (1983). Validation of self-reported smoking behavior: Biochemical analyses of cotinine and thiocyanate. *American Journal of Public Health*, 73, 1204-1207.
- Herd, J.A. (1981). Behavioral factors in the etiology and pathogenesis of cardiovascular diseases: Sudden death, hypertension, and myocardial infarction. In S.M. Weiss, J.A. Herd, & B.H. Fox (Eds.), *Perspectives on behavioral medicine* (pp. 55-65). New York: Academic Press.
- Henry, J.P. (1983). Coronary heart disease and arousal of the adrenal cortical axis. In T.M. Dembroski, T.H. Schmidt, & G. Blümchen (Eds.), *Biobehavioral bases of coronary heart disease* (pp. 365-381). Basel: S. Karger.
- Herning, R.I., Jones, R.T., Benowitz, N.L., & Mines, A.H. (1983). How a cigarette is smoked determines nicotine blood levels. *Clinical Pharmacology & Therapeutics*, 33, 84-90.
- Herxheimer, A., Griffiths, R.L., Hamilton, B., & Wakefield, M. (1967). Circulatory effects of nicotine aerosol inhalations and cigarette smoking in man. *Lancet*, 2, 754-755.
- Hill, R., Haley, N., & Wynder, E.L. (1983). Cigarette smoking as a risk for cardiovascular disease: Biochemical analyses, carboxyhemoglobin, plasma nicotine, cotinine, and thiocyanate versus self-report smoking data. *Journal of Chronic Disease*, 36, 439-449.
- Kershbaum, A., Pappajohn, D.J., Bellet, S., Hirabayashi, M., & Shafiqi, H. (1968). Effect of smoking and nicotine on adrenocortical secretion. *Journal of the American Medical Association*, 203, 113-116.
- Jarvik, M. (1979). Biological influences on cigarette smoking. In N. Krasnegor (Ed.), *The behavioral aspects of smoking* (NIDA Monograph 26, pp. 7-45). Rockville, MD: National Institute on Drug Abuse.
- Langer, A.W., McCubbin, J.A., Stoney, C.M., Hutcheson, J.S., Charlton, J.D., & Obrist, P.A. (1985). Cardiopulmonary adjustments during exercise and an aversive reaction time task: Effects of beta-adrenoceptor blockade. *Psychophysiology*, 22, 59-68.
- MacDougall, J.M., Dembroski, T.M., Slaats, S., Herd, J.A., & Eliot, R.S. (1983). Selective cardiovascular effects of stress and cigarette smoking. *Journal of Human Stress*, 9, 13-21.
- Mangan, G.L., & Golding, J.F. (1984). *The psychopharmacology of smoking*. Cambridge: Cambridge University Press.
- McNair, D.M., Lorr, M., & Droppleman, L.F. (1971). *Profile of Mood States*. San Diego, CA: Educational and Industrial Testing Service.
- Myrsten, A.L., Elgerot, A., & Edgren, B. (1977). Effects of abstinence from tobacco smoking on physiological and psychological arousal levels in habitual smokers. *Psychosomatic Medicine*, 39, 25-38.
- Obrist, P.A., Gaebelein, C.J., Teller, E.S., Langer, A.W., Light, K.C., & McCubbin, J.A. (1978). The relationship among heart rate, carotid dP/dt, and blood pressure in humans as a function of the type of stress. *Psychophysiology*, 15, 102-115.
- Pomerleau, O.F., Adkins, D., & Pertschuk, M. (1978). Predictors of outcome and recidivism in smoking cessation treatment. *Addictive Behaviors*, 3, 65-70.
- Pomerleau, O.F., Fertig, J., & Shanahan, S.O. (1983). Nicotine dependence in cigarette smoking: An empirically-based, multivariate model. *Pharmacology, Biochemistry & Behavior*, 19, 291-299.
- Pomerleau, O.F., & Pomerleau, C.S. (1984). Neuroregulators and the reinforcement of smoking: Towards a biobehavioral explanation. *Neuroscience and Biobehavioral Reviews*, 8, 503-513.
- Pomerleau, O.F., Turk, D., & Fertig, J. (1984). The effects of cigarette smoking on pain and anxiety. *Addictive Behaviors*, 9, 265-271.
- Rose, J.E., Ananda, S., & Jarvik, M.E. (1983). Cigarette smoking during anxiety-provoking and monotonous tasks. *Addictive Behaviors*, 8, 353-359.
- Rosenberg, J., Benowitz, N.L., Jacob, D., & Wilson, K.M. (1980). Disposition kinetics and effects of intravenous nicotine. *Clinical Pharmacology and Therapeutics*, 28, 517-522.
- Schachter, S.B. (1978). Pharmacological and psychological determinants of smoking. *Annals of Internal Medicine*, 88, 104-114.
- Schachter, S.B., Silverstein, B., Kozlowski, L.T., Perlick, D., Herman, C.P., & Liebling, B. (1977). Studies of the interaction of psychological and pharmacological determinants of smoking. *Journal of Experimental Psychology*, 106, 3-40.
- Shiffman, S. (1982). Relapse following smoking cessation: A situational analysis. *Journal of Consulting and Clinical Psychology*, 50, 71-86.
- Stephens, R. (1977). Psychophysiological variables in cigarette smoking and reinforcing effects of nicotine. *Addictive Behaviors*, 2, 1-7.
- Su, C. (1982). Actions of nicotine and smoking on circulating catecholamines. *Pharmaceutical Therapeutics*, 17, 129-141.
- Trap-Jensen, J., Carlsen, J.E., Svendsen, T.L., & Christensen, N.J. (1979). Cardiovascular and adrenergic effects of cigarette smoking during immediate non-selective and selective beta-adrenergic blockade in humans. *European Journal of Clinical Investigation*, 9, 181-183.
- Turner, J.R., & Carroll, D. (1985). Heart rate and oxygen consumption during mental arithmetic, a video game,

- and graded exercise: Further evidence of metabolically-exaggerated cardiac adjustments? *Psychophysiology*, 22, 261-267.
- Volle, R.L., & Koelle, G.B. (1975). Ganglionic stimulating and blocking agents. In L.S. Goodman & A. Gilman (Eds.), *The pharmacological basis of therapeutics* (5th edition; pp. 565-574). New York: Macmillan Publishing Co.
- Westfall, T.C., & Watts, D.T. (1964). Catecholamine excretion in smokers and nonsmokers. *Journal of Applied Physiology*, 19, 40-42.

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Announcements

Twenty-Seventh Annual Meeting Society For Psychophysiological Research

From October 15th through 18th, 1987, the Twenty-Seventh Annual Meeting of the Society for Psychophysiological Research will be held at the Okura Hotel in Amsterdam, the Netherlands.

The deadline for submission of papers is May 15, 1987. All submissions should be sent to the Program Chair: Connie C. Duncan, Laboratory of Psychology and Psychopathology, National Institute of Mental Health, NIH, Building 10, Room 4C110, Bethesda, MD 20892, USA.

Registration information may be obtained either from the Convention Manager (Joanne Fetzner, Society for Psychophysiological Research, 2380 Lisa Lane, Madison, WI 53711, USA, 608/271-1500) or the European Registration Liaisons (A.J.W. Boulhouwer and C.H.M. Brunia, Department of Psychology, Tilburg University, Tilburg 5000 LE, the Netherlands, 31/13-669111).

Membership in the Society for Psychophysiological Research

Information about membership in the Society for Psychophysiological Research and application forms may be obtained from either of the Society's membership chairmen: Alan W. Langer, Behavioral Physiology Lab, 101 Stevens Place, Syracuse University, Syracuse, NY 13210; or Kenneth Hugdahl, Department of Somatic Psychology, University of Bergen, Årstadveien 21-5000 Bergen, Norway.

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