Psychopharmacology (1997) 129 : 382–389

ORIGINAL INVESTIGATION

David G. Gilbert · Steven L. Estes · Richard Welser

Does noise stress modulate effects of smoking/nicotine?

Mood, vigilance, and EEG responses

Received: 19 July 1996/Final version: 8 September 1996

Abstract Effects of smoking multiple cigarettes on EEG, vigilance, and subjective state were assessed in a repeated measures design where noise level (high versus minimal) was crossed with nicotine dose (quasi-ad lib own versus 1.0 mg FTC nicotine machine-delivered dose versus 0.05 mg FTC nicotine machine-delivered dose). Vigilance was increased by nicotine, but not by noise and there was no noise by dose interaction. Effects of nicotine on EEG varied as a function of dose, noise, hemisphere, time, and eyes-open versus eyes-closed condition. Smoking normal nicotine delivery (0.9–1.1 mg FTC-estimated) cigarettes resulted in decreases in percentages of delta and theta EEG magnitude and increased percentage beta-1 EEG magnitude across conditions and time. Changes in alpha and theta magnitude were dependent on eyes being open versus closed. Hemispheric asymmetries varied as a function of noise and time. Consistent with inverted "U" models, effects of nicotine on EEG were clearly stimulant during the quiet conditions while there were minimal to no differences between nicotine doses during the high-noise conditions. The failure of nicotine to modify mood is interpreted in terms of bioinformational models of nicotine's subjective effects.

Key words Arousal · Attention · EEG · Emotion · Nicotine · Stress · Smoking · Vigilance

Introduction

Nicotine's tranquilizing and negative-affect reducing effects have been interpreted by many as paradoxical, since most experimental studies have found nicotine to increase indices of physiological arousal, including heart rate, blood pressure, stress hormones, and electrocortical activiation (reviewed by Gilbert 1979, 1995). The question is how a drug with stimulant properties can produce emotionally tranquilizing and affect-reducing effects. Psychobiological mechanisms postulated to account for nicotine/smoking's affect-modulating and reinforcing effects include arousal state-dependent modulations of electrocortical activation (Eysenck 1973) and state-dependent lateralized cortical modulation of affect-related neural networks (Gilbert 1987; Gilbert et al. 1989).

The arousal-modulation model is based on evidence that nicotine's affect-modulating effects may be a function of its decreasing cortical activation when pre-nicotine activation is high, as it typically is during stressful situations (Mangan and Golding 1978; Eysenck and O'Connor 1979). This model suggests that nicotine has EEG activating effects during low-arousal situations (as during most experimental studies reporting such effects) but de-activating (sedative) effects during stressful and other high-arousal conditions. Nicotine-induced cortical de-activation during stressful conditions is seen as resulting in decreased negative affect and increased tranquilization. The lateralized arousal modulation (LAM) model of nicotine's negative-affect-reducing effects postulates that nicotine has greater cortical de-activating (or less activating) effects on the more activated hemisphere and greater activating (or less deactivating) effects on the less activated hemisphere (Gilbert and Welser 1989; Gilbert 1995). This state-dependent asymmetry of nicotine-induced cortical activation and deactivation is seen as important in the modulation of affective processes, since evidence indicates that negative affect is associated with activation of the right cortex and/or with underactivation of the left cortex (Davidson 1995). Thus, the LAM model predicts that during stressful and certain other negative-affect-related states nicotine increases the ratio of left-relative-to-right cortical activation and

D.G. Gilbert (✉) · S.L. Estes · R. Welser
Department of Psychology, Southern Illinois University, Carbondale, IL 62901, USA

thereby increases the ratio of positive to negative affect (Gilbert 1987, 1995; Gilbert et al. 1989).

Nicotine's effects on EEG activity (reviewed by Knott 1990) have received a good deal of attention over the last 2 decades. However, EEG and emotional responses to nicotine have rarely been assessed under stressful conditions. Those that have assessed stress-dependent effects of nicotine on EEG have frequently failed to assess the possibility that nicotine has lateralized effects. Thus, few studies have empirically tested either the arousal modulation model or the lateralized arousal modulation model of nicotine's tranquilizing and negative-affect reducing effects.

Thus, the primary purpose of the present experiment was to test the arousal modulation and lateralized arousal modulation models of nicotine's effects on affective states and cognitive performance. In an effort to assess the hypothesis of interactions between nicotine and stress, the present study characterized effects of smoking-delivered nicotine in quiet and high-noise conditions. Dependent variables were vigilance, mood, and EEG.

Materials and methods

Participants

Participants were 12 males, aged 21–35 years, who were habitual smokers of at least 15 cigarettes per day. They habitually smoked cigarettes ranging from 0.9 to 1.1 mg FTC-estimated nicotine delivery. All were right-handed as determined by the Edinburgh Inventory (Oldfield 1971). Most cigarettes smoked in the USA have FTC machine-estimated nicotine deliveries of between 0.5 and 1.2 mg (Maxwell Consumers Report 1990). EEG data from four participants were dropped due to excessive EEG artifact.

Feeling state questionnaire (FSQ)

Mood states were assessed with the FSQ (Gilbert et al. 1992), which uses a series of 11-point (0 = none to 10 = extreme) Likert scales previously demonstrated to be sensitive to stress- and smoking-induced changes in moods and other subjective states (Gilbert et al. 1994).

Equipment and data collection

A quantified smoke delivery system (QSDS) developed by Gilbert et al. (1988) was used to administer standard-sized doses of smoke/nicotine on the experimental days. This system takes 2-s duration, 35 cc, sinusoidal-shaped puffs from a cigarette drawn by a motor-driven syringe. The syringe ejects the puff into the smoker's mouth over a period of 0.5 s. The smoker then immediately inhales the smoke deeply and holds it until a signal light goes out, 5 s after inhalation.

EEG was recorded using tin electrodes arranged in the standard 10/20 configuration in a commercial cap. A tin electrode on the nose tip served as the reference and an electrode at the canthus served to monitor eye artifacts. Electrode impedances were kept below 3 Kohm. EKG signals were obtained from electrodes on the rib cage. Electrophysiological signals were recorded using Grass preamplifiers with 0.3 Hz high-pass and 75 Hz low-pass settings. EEG amplifier outputs were passed through 48 dB/octave low-pass filters (Frequency Devices, model 790, -3 dB at 40 Hz) prior to being digitized at 250 Hz by a BenchTop signal processing unit interfaced with a Macintosh Plus computer. Heart rate was assessed using EKG signals obtained from axial leads on Grass polygraph paper tracings.

Blood samples and analysis

Blood samples for plasma nicotine and other measures were drawn from the median cubital vein of the participant's left arm using an indwelling catheter. Sampling and analysis procedures were similar to those reported elsewhere (Gilbert et al. 1992).

Rapid information processing (RIP) vigilance task

The rapid information processing (RIP) task (Warburton and Wesnes 1984) required that participants monitor single digits (1–9) presented in a pseudo-random order at a rate of 116/min and press a bar whenever the last three digits were either even or odd. There were a total of 160 targets during the 16-min duration task. In order to attain a stable performance level, each participant practiced this task a total of 6–8 times on three or four different occasions prior to initiating the series of six experimental sessions.

Data analysis

For each 55-epoch collection period, as many artifact-free EEG 1.024-s duration epochs as possible (minimum = 25/period) were submitted to a Fast Fourier Transform (FFT) using a Hanning window (Cooley and Tukey 1967). The raw power spectra were then converted to magnitude spectra by taking the square root of each ordinate to provide better representation of the weaker portions of the spectrum and to normalize the distribution (Lykken et al. 1974). Artifact rejection was accomplished by visual inspection of EEG channels, where eye blinks showed up as large spikes at frontal loci and electromyographic (EMG) activity was typically identified as high-frequency spiking, most often at temporal loci. Recordings were obtained from F3, F4, P3, and P4 (International system). Magnitudes were calculated for each of the following bands: delta (0.95–3.91 Hz), theta (3.91–7.81 Hz), alpha (7.81–13.67), $beta_1$ (13.67–20.51), and $beta_2$ (20.51–29.30). Relative power was assessed for each of the five EEG bands by expressing the magnitude of each band as a percentage of the total magnitude of all five bands. Heart rate (HR) was determined from ECG polygraph recordings obtained during EEG collection periods.

Procedure

During a late-afternoon orientation session participants practiced smoking both a regular tobacco cigarette (Camel Filter) and a denicotinized cigarette (Next) by means of the QSDS. Participants were instructed to eat breakfast as usual on each of the following 6 test days, and were also required to abstain from smoking cigarettes or using any tobacco products after midnight on each of the test days.

Smokers participated in six test sessions lasting approximately 4 h each, beginning at 8:00 a.m. During each session, a total of five cigarettes were smoked, the beginning of each separated by 30-min intervals. The experimental design was Noise (high versus minimal) × Dose (ad lib own versus 1.0 mg-QSDS versus 0.05 mg QSDS low-nicotine control). Thus, during two sessions (one no-noise and one high-noise) each individual smoked ad lib as much as desired

of his accustomed brand during each of five 10-min smoking breaks between experimental tasks. During each of the other four sessions individuals smoked a 1.0 mg- or 0.05-mg nicotine delivery cigarette via the QSDS in a high-noise or quiet environment. QSDS cigarettes were smoked to a point 3 mm from the filter overwrap. During QSDS smoking sessions 35 cc puffs of smoke were delivered into the smoker's mouth and inhaled at 30-s intervals, following a protocol similar to that used in earlier work demonstrating the reliability and validity of the system (Gilbert et al. 1988; Gilbert and Meliska 1992). All smokers participated in all six conditions and the order of testing was counterbalanced. Subjective state was assessed before smoking and immediately after each EEG recording. Fifty-five 1.024-s duration epochs of eyes-open EEG followed by 55 eyes-closed epochs were recorded, sampling every fourth second over a 3-min period. During the eyes-open recordings participants were instructed to inhibit eye blinks and movements and to look straight ahead at the intersection of a cross used for visual fixation. During eyes-closed conditions participants were asked to inhibit eye movements by imagining looking at the cross. EEG, HR, blood, and subjective measures were obtained before smoking (baseline), after the second cigarette, prior to the fifth cigarette, and immediately after the fifth cigarette.

Noise manipulation

The noise manipulation consisted of delivery or not of two types of sound stimuli. One type of stimuli was a series of high-intensity (95–104 dB), complex sounds presented at pseudo-random (24–53 s) intervals while participants smoked (seven stimuli/cigarette) and performed the RIP task (eight stimuli/task). These stimuli included aircraft, train, siren, machine gun, and animal sounds, and numerous other noises, ranging from 2.7- to 4.1-s duration. The second set of sound stimuli was interspersed with EEG collection epochs. Eight white noise bursts (104 dB, 1-s duration, 5 ms rise time) were presented pseudo-randomly during each 8-min period while participants had their EEG recorded. Thus, EEG was collected for a number of seconds, followed by a noise burst, followed by additional EEG. All auditory stimuli were presented via a speaker placed one meter above the center of the participant's head.

Results

Effects of experimental manipulations were assessed on change scores from baseline with Dose (ad lib versus 1.0 mg QSDS versus 0.05 mg QSDS) × Noise (high versus minimal) × Time (post-second cigarette versus pre-fifth cigarette versus post fifth cigarette) ANOVAs. ANOVAs were performed on change scores from baseline values on the given experimental day (with the exception of the vigilance task for which there was no baseline). For EEG analyses, Eyes (Open versus Closed) × Location (Frontal versus Parietal) × Hemisphere (Left versus Right) were additional factors. All ANOVAs used Greenhouse-Geisser (1959) correction for sphericity of repeated measures. This correction results in attenuated degrees of freedom. Follow-up analyses of simple effects were routinely performed only on significant interactions. Ad-lib, 1.0-mg QSDS, and 0.05-mg QSDS nicotine conditions did not differ significantly prior to smoking for any of the dependent variables reported below. Hormonal and glucose blood measures obtained in the present study will be reported in a forthcoming publication (Gilbert, et al. 1997).

Plasma nicotine

Plasma nicotine change scores showed the expected main effect of Dose [$F(1.23,13.55) = 71.06$, $P < 0.001$]. Mean plasma nicotine concentrations were elevated to approximately the same degree whether subjects smoked the regular (1.0 mg nicotine Camel Filter) cigarette via the QSDS or the own ad-lib procedure after two, four, and five nicotine cigarettes. Mean plasma nicotine boosts across the three nicotine conditions after two, four and five cigarettes were generally in excess of 20 times greater in the ad lib and 1.0 mg QSDS conditions than in the 0.05 mg QSDS condition and ranged from 11–25 ng/ml after the second through the fifth cigarettes, in contrast with 1 ng/ml or less for the control cigarette. The 1.0 mg QSDS condition elevated plasma nicotine significantly above the 0.05 mg condition after two [$F(1,11) = 167.33$, $P < 0.001$]; four [$F(1,11) = 383.31$, $P < 0.001$]; and five cigarettes [$F(1,11) = 71.06$, $P < 0.001$]. The ad-lib condition elevated plasma nicotine significantly above the low-nicotine cigarette after two [$F(1,11) = 77.34$, $P < 0.001$]; four [$F(1,11) = 69.25$, $P < 0.001$]; and five cigarettes [$F(1,11) = 72.84$, $P < 0.001$]. A significant main effect was found for Noise [$F(1,11) = 6.64$, $P = 0.026$], with the high-intensity condition having larger increases in nicotine than the quiet condition (11.36 versus 10.71 ng/ml).

Heart rate (HR) change

There was a main effect for Dose [$F(1.44,15.89) = 68.31$, $P < 0.001$], but no main effect for Noise [$F(1,11) = 0.63$, $P = 0.443$] and no Dose × Noise interaction, [$F(1.29,14.17) = 0.89$, $P = 0.389$]. Change scores from baseline showed HR boosts associated with 1.0 mg QSDS and ad-lib smoking did not differ at any time; however, they were consistently greater (8–10 bpm) than low-nicotine conditions (all Ps < 0.001).

Subjective change

As seen in Fig. 1, Noise had a significant effect on FSQ-assessed negative affect (tension + worry + fear + anger + sad + unpleasant), increasing negative affect in the high-noise condition relative to the quiet condition [$F(1,11) = 5.42$, $P = 0.040$]. There was no effect for nicotine Dose [$F(1.76,19.37) = 0.41$, $P = 0.645$] or Dose × Noise interaction on negative affect [$F(1.98,21.79) = 0.03$, $P = 0.975$]. Cognitive arousal (Alert - Drowsy) also was greater in the noise relative to the quiet condition [$F(1,11) = 5.35$,

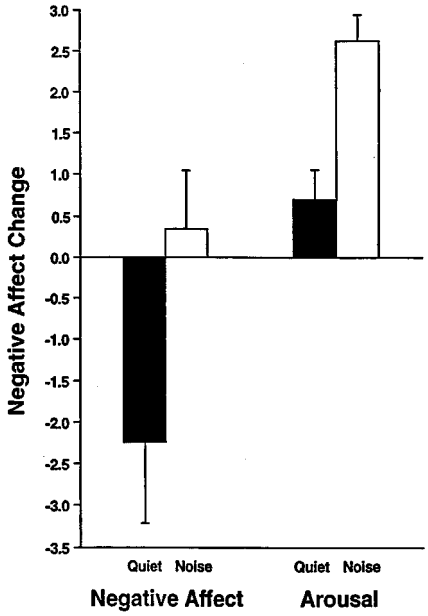

Fig. 1 Mean and standard error of negative affect and arousal change as a function of noise

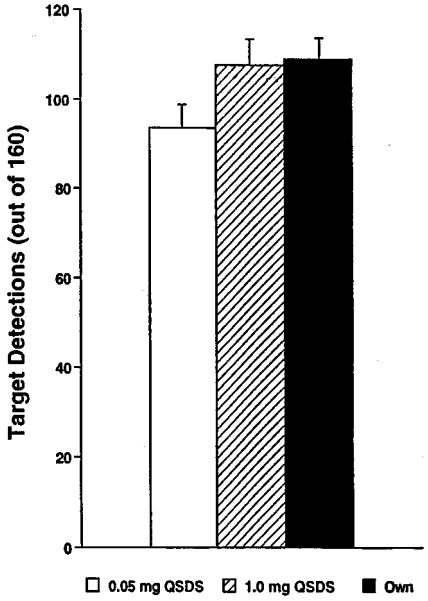

Fig. 2 Mean and standard error of target detections during vigilance task as a function of dose

$P = 0.041$], but there was no effect for nicotine Dose [$F(1.63,17.93) = 1.61$, $P = 0.228$] or Dose × Noise interaction [$F(1.74, 19.19) = 0.68$, $P = 0.498$]. Other subscales of the FSQ (including pleasantness, happiness and relaxation) failed to show significant effects of Dose.

Vigilance performance during rapid information processing

Vigilance detection (Fig. 2) was significantly better in own ad-lib and QSDS 1.0 mg conditions relative to the 0.05 mg nicotine condition [$F(1.92,21.12) = 21.90$, $P < 0.0001$], but was not influenced by Noise [$F(1,11) = 0.36$, $P = 0.560$] or by the interaction of Dose with Noise [$F(1,38) = 0.16$, $P = 0.773$]. Commission errors were not influenced by Dose, Noise, or their interaction.

Percentage delta magnitude (Δ)

As seen in Fig. 3, there was a significant main effect for Dose [$F(1.82,12.72) = 27.7$, $P < 0.001$], such that in the own ad-lib condition smoking resulted in a lower percentage of Δ relative to baseline than the QSDS 0.05 mg condition [$F(1,7) = 12.04$, $P = 0.010$], while the QSDS 1.0 and 0.05 mg conditions did not differ significantly [$F(1,7) = 2.07$, $P = 0.193$]. In addition, there was a significant Dose × Eye interaction (Fig. 4) [$F(1.97,13.82) = 4.45$, $P = 0.033$] that reflected the ad-lib condition having a significantly greater decrease in Δ than the 0.05 mg nicotine condition both with eyes

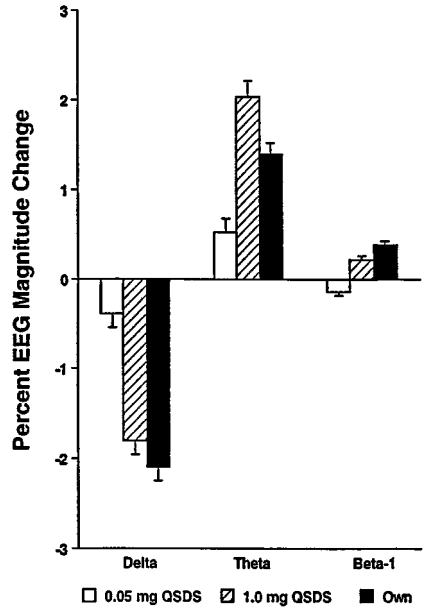

Fig. 3 Mean and standard error of percent delta, theta and beta-1 EEG magnitude changes as a function of dose

open [$F(1,7) = 6.81$, $P = 0.0349$] and eyes closed [$F(1,7) = 51.45$, $P < 0.001$], while the 1.0-mg QSDS condition was significantly different from the 0.05 mg condition only with eyes closed, [$F(1,7) = 40.60$, $P < 0.001$]. There was also a significant Dose × Noise × Time × Hemisphere interaction (Fig. 5) [$F(3.51,24.59) = 3.24$, $P = 0.034$]. This interaction appears to reflect larger reductions in left- than right-hemisphere Δ after cigarette 2 in the 1.0 mg relative to the 0.05-mg QSDS during the quiet condition. This is

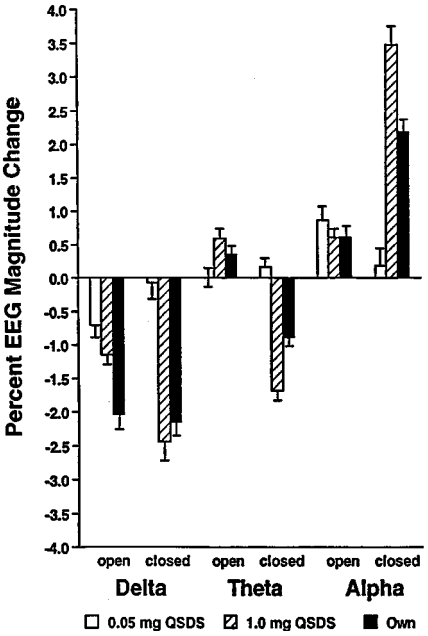

Fig. 4 Mean and standard error of percent delta, theta and alpha EEG magnitude changes as a function of dose and eyes open vs eyes closed condition

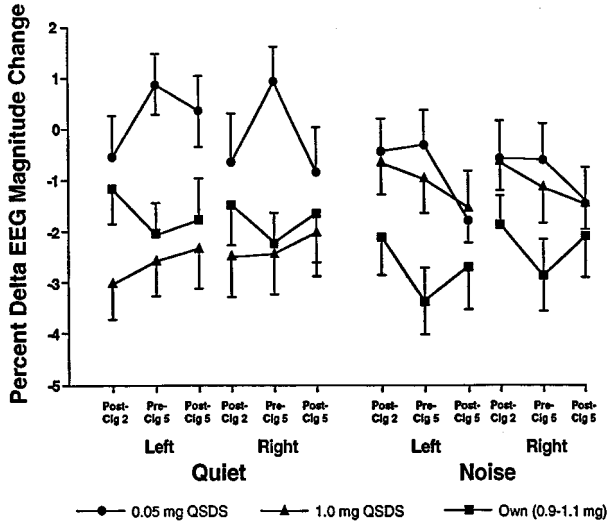

Fig. 5 Mean and standard error of percent delta EEG magnitude change as a function of dose, noise, time, hemisphere and EEG frequency band

in contrast with the noise condition, where there were no 1.0-mg QSDS hemispheric differences but where there were larger left than right reductions in the own ad-lib condition. This interaction also reflects the fact that during quiet conditions 1.0-mg QSDS smoking resulted in as large or larger decrements in Δ than own ad-lib and 0.05-mg QSDS smoking, but during noise, own ad-lib smoking produced larger Δ reductions while QSDS 1.0 mg and QSDS 0.05 mg did not differ significantly. An ANOVA using only the two QSDS doses (0.05 mg versus 1.0 mg) revealed a significant Dose $\times$ Noise interaction [$F(1,7) = 8.28$, $P = 0.024$]. Follow-up contrasts showed that during the quiet condition the 1.0 mg QSDS delivery cigarette resulted in less Δ than the 0.05 mg cigarette [$F(1,7) = 24.24$, $P = 0.002$], but that during the stress condition there was no significant difference between nicotine doses [$F(1,7) = 0.65$, $P = 0.438$].

Percentage theta magnitude (θ)

There was no significant main effect for Dose [$F(1.32,9.22) = 2.12$, $P = 0.179$] or Noise [$F(1,7) = 0.12$, $P = 0.742$] and no Dose $\times$ Noise interaction, [$F(1.78,12.48) = 0.05$, $P = 0.941$]. However, there was a significant Dose $\times$ Eye interaction (Fig. 4), [$F(1.75,12.24) = 8.50$, $P = 0.006$] that was due to a slight increase in θ in the 1.0-mg QSDS condition with eyes open, and a decrease in θ with eyes closed, resulting in a significant difference from each other, [$F(1,7) = 21.35$, $P = 0.002$]; changes in the own ad-lib condition followed a pattern similar to the 1.0-mg QSDS, but fell somewhat short of significance. The 0.05-mg nicotine condition was not associated with post-smoking changes in eyes-open versus eyes-closed conditions.

Percentage alpha magnitude (α)

There was a significant main effect for Dose (Fig. 3) [$F(1.88,13.17) = 6.89$, $P = 0.010$], as well as a Dose $\times$ Time interaction [$F(3.01,21.04) = 5.27$, $P = 0.007$]. Analysis of this interaction demonstrated that smokers in the three Dose conditions did not differ significantly prior to smoking [$F(1.08,7.53) = 0.34$, $P = 0.595$], but did marginally differ after two cigarettes [$F(1.79,12.50) = 3.88$, $P = 0.053$] and significantly differ after four cigarettes [$F(1.88, 13.18) = 15.13$, $P < 0.001$], such that α increased for both QSDS 1.0 mg [$F(1,7) = 16.91$, $P = 0.005$] and own ad-lib [$F(1,7) = 25.50$, $P = 0.002$] relative to the 0.05-mg nicotine control. There was no significant difference between the levels of Dose after the fifth cigarette [$F(1.69, 11.83) = 1.25$, $P = 0.316$]. There was also a significant Dose $\times$ Eye interaction (Fig. 4) which resulted from significantly greater increases of α eyes closed relative to eyes open in the 1.0-mg QSDS [$F(1,7) = 19.09$, $P = 0.003$] and the ad-lib [$F(1,7) = 7.81$, $P = 0.027$] conditions, while no such difference was present for the low-nicotine condition. There were no other significant interactions and the main effect of Noise was not significant [$F(1,7) = 0.07$, $P = 0.797$].

Percentage beta$_1$ magnitude (β_1)

There was a significant main effect for Dose (Fig. 3) [$F(1.9,13.32) = 6.21$, $P = 0.013$]. In the own ad-lib

condition β_1 magnitude increased significantly [$F(1,7) = 13.16$, $P = 0.008$] relative to the 0.05 mg condition, and the 1.0-mg QSDS also tended to increase relative to the 0.05 mg condition [$F(1,7) = 4.32$, $P = 0.076$]. The 1.0-mg QSDS and the own ad-lib did not differ [$F(1,7) = 1.57$, $P = 0.251$].

Percentage beta$_2$ magnitude (β_2)

There was no significant main effect for Dose [$F(1.60,11.23) = 2.93$, $P = 0.102$]; Noise, [$F(1,7) = 0.01$, $P = 0.968$]; or Dose × Noise interaction [$F(1.48, 10.38) = 1.35$, $P = 0.290$].

EEG activation ratio: $(\alpha + \beta_1 + \beta_2)/(\Delta + \theta)$

A significant main effect for nicotine was found [$F(1.54, 10.78) = 13.30$, $P = 0.002$], indicating greater arousal increases (more $\alpha + \beta_1 + \beta_2$ relative to $\Delta + \theta$) in the ad-lib and 1.0-mg QSDS nicotine conditions relative to the 0.05-mg nicotine QSDS condition. There was also a significant Dose × Noise × Hemisphere × Time interaction (Fig. 6) [$F(2.41,16.85) = 3.45$, $P = 0.045$], mostly reflecting less of a stimulant effect in the right than left hemisphere at pre-cigarette five in the noise relative to the quiet condition. Follow-up contrasts showed that during the quiet condition the 1.0 mg QSDS delivery cigarette resulted in a significantly larger activation ratio than the 0.05 mg cigarette [$F(1,7) = 21.24$, $P = 0.003$], but that during the stress condition there was no significant difference between nicotine doses, [$F(1,7) = 3.27$, $P = 0.114$].

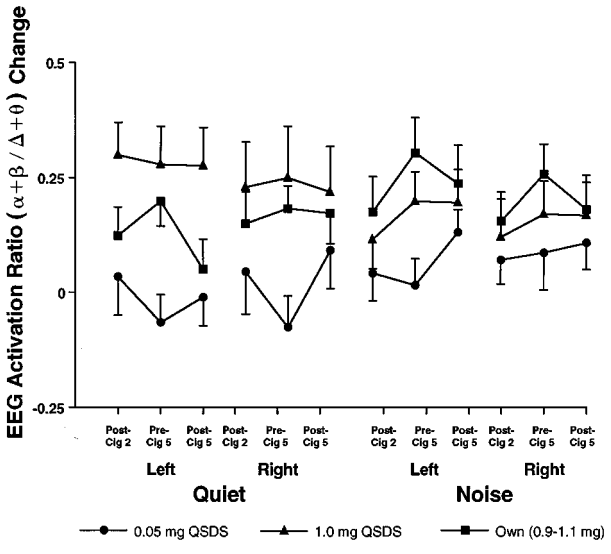

Fig. 6 Mean and standard error of EEG activation ratio change as a function of dose, noise, time and hemisphere

Discussion

Noise modulated the effects of nicotine on EEG, but not on affective state, vigilance or heart rate. The failure of affect and vigilance to exhibit the same curvilinear effects as cortical activation suggests partial independence of electrocortical state from affect and vigilance state. This lack of coherence across measures is inconsistent with the curvilinear arousal modulation hypothesis of nicotine's stress-reducing and affect-modulating effects. We consider the possible explanations for this incoherence after reviewing our findings in terms of the relevant literature.

Nicotine increased heart rate, EEG activation, and vigilance performance during both noise and quiet conditions. In contrast to the effects of nicotine, noise elevated negative affect and had no effect on vigilance or heart rate. Hasenfratz et al. (1989) also found that nicotine increased vigilance while noise failed to have a significant effect. Nicotine has repeatedly been shown to have attention-enhancing effects (reviewed by Warburton 1990). The contrasting pattern of effects of nicotine vis a vis noise is consistent with models proposing that nicotine's affect-modulating effects are influenced by a system that is at least partially independent of its influences on arousal and vigilance (see O'Neill and Parrot 1992; Gilbert 1995).

While our findings replicate work showing nicotine to reduce slow-wave and increase fast-wave activity (Golding 1988; Hasenfratz et al. 1989; Knott 1990; Pritchard 1991; Domino 1995), these stimulant effects varied as a function of noise, hemisphere and time. Changes in relative delta magnitude and in activation ratio indicated greater left- than right-hemisphere activation after smoking in the 1.0-mg relative to the 0.05-mg QSDS during the quiet condition. In contrast, during noise there were no hemispheric differences between the two QSDS doses though greater left than right activation was found in the own ad-lib condition.

Our noise-stress-dependent EEG effects of nicotine are consistent with findings of Mangan and Golding (1978) and of Hasenfratz et al. (1989), but by using bilateral electrode sites we were able to detect lateralized noise-dependent effects of nicotine. Others have observed relatively right lateralized effects of nicotine on EEG during relatively relaxing and during highly arousing conditions (Gilbert 1987; Gilbert et al. 1989; Pritchard 1991; Norton et al. 1992; Domino 1995; Pritchard et al. 1995). The tendency of the present subjects to exhibit relatively greater left-hemispheric activation in response to nicotine during the quiet condition may reflect somewhat elevated arousal during this condition due to associations (during the orientation session and subsequent sessions) of noise with the experimental setting. Consistent with this possibility, Gilbert et al. (1995) found that caffeine changed the EEG-activating effects of nicotine from relatively

stronger in the right hemisphere to relatively greater in the left hemisphere.

Evidence suggests that nicotine can have a greater stimulant effect on the less activated hemisphere, while potentially decreasing activation in the more activated hemisphere (Gilbert et al. 1989, 1994). Such findings are consistent with the more general and commonly held view that nicotine stimulates cortical activation when initial arousal is low, while decreasing it when pre-nicotine arousal is high. This two-hemispheres-two-inverted- "U"s hypothesis was suggested to explain earlier differences in the effects of nicotine on the two hemispheres as a function of arousing versus relaxing conditions (Gilbert and Welser 1989).

The failure of nicotine to modulate negative affect and subjective arousal suggests that further articulation of specific stressors and affective states is important when assessing the effects of nicotine. Evidence (reviewed by Gilbert 1995) suggests that nicotine's stress-reducing properties occur primarily when stress-inducing stimuli are anticipatory and/or ambiguous and when distracting stimuli are present. Changes in the processing of such stimuli can be brought about by nicotine's priming relatively lateralized and localized bioinformational networks and by enhancing attention (Gilbert 1995; Gilbert et al. 1989). Since the noise stimuli used in the present study were largely predictable, repetitive, and unambiguous, and there were no significant distractors, processing of the stimuli was unlikely to be significantly altered by nicotine. Consistent with this information-processing-priming model, findings by Arci and Grunberg (1992) showed that only certain kinds of loud, irritating stimuli (three of 11 assessed) differentiated emotional responses of smoking-deprived smokers from smoking smokers and nonsmokers. Perlick (1977) observed that smoking reduced jet aircraft noise-induced irritability in the context of smokers watching a highly engaging (distracting) television drama, possibly by attenuating noise-induced loss of concentration on the drama.

In conclusion, our findings are consistent with the view that nicotine's effects on EEG are pre-nicotine arousal-state-dependent, but are inconsistent with the hypothesis that nicotine-induced EEG changes consistently result in parallel changes in affective state. Thus, the findings provide mixed support for both the arousal modulation and lateralized arousal modulation models.

Acknowledgements This research was supported in part by a grant from the R.J. Reynolds Tobacco Company.

References

Arci JB, Grunberg NE (1992) A psychophysical task to quantify smoking cessation-induced irritability: The Reactive Irritability Scale (RIS). Addict Behav 17:587–601

Cooley J, Tukey JW (1967) An algorithm for machine calculations of complex Fourier series. Math Comput 19:197–301

Davidson, RJ (1995) Cerebral asymmetry, emotion, and affective style. In: Davidson RJ, Hugdahl K (eds) Brain asymmetry. MIT Press, Cambridge, Mass, pp 361–387

Domino EF (1995, March) Variable brain localization effects of tobacco smoking depending upon the increment in plasma nicotine levels. Poster presented at the First Annual Meeting of the Society for Research on Nicotine and Tobacco. San Diego, Calif.

Eysenck HJ (1973) Personality and the maintenance of the smoking habit. In: Dunn WL (ed) Smoking behavior: motives and incentives. VH Winston, Washington, D.C., pp 113–146

Eysenck HJ, O'Connor K (1979) Smoking, arousal and personality. In: Remond A, Izard C (eds) Electrophysiological effects of nicotine. Elsevier, Amsterdam, pp 147–157

Gilbert DG (1979) Paradoxical tranquilizing and emotion-reducing effects of nicotine. Psychol Bull 86:643–661

Gilbert DG (1987) Effects of smoking and EEG laterality as a function of personality. Person Individ Diff 8:933–941

Gilbert DG (1995) Smoking: individual differences, psychopathology, and emotion. Taylor & Francis, Bristol, Pa.

Gilbert DG, Meliska CJ (1992) Individual differences in and reliability of EEG, cortisol, beta-endorphin, heart rate, and subjective responses to smoking multiple cigarettes via a quantified smoke delivery system. In: Lippiello PM, Collins AC, Gray JA, Robinson JH (eds) The biology of nicotine: current research issues. Raven Press, New York, pp 141–155

Gilbert DG, Welser R (1989) Emotion, anxiety and smoking. In: Ney T, Gale A (eds) Smoking and human behavior. Wiley, Chichester, pp 171–196

Gilbert DG, Jensen RA, Meliska CJ (1988) A system for administering quantified doses of tobacco smoke to human subjects: plasma nicotine and filter pad validation. Pharmacol Biochem Behav 31:905–908

Gilbert DG, Robinson JH, Chamberlin CL, Spielberger CD (1989) Effects of smoking/nicotine on anxiety, heart rate, and lateralization of EEG during a stressful movie. Psychophysiology 26:311–320

Gilbert DG, Meliska CJ, Williams C, Jensen RA (1992) Subjective correlates of smoking-induced elevations of plasma beta-endorphin and cortisol. Psychopharmacology 106:275–281

Gilbert DG, Meliska CJ, Welser R, Estes SL (1994) Depression, personality and gender influence EEG, cortisol, beta-endorphin, heart rate and subjective responses to smoking multiple cigarettes. Person Indiv Differences 16:247–264

Gilbert DG, Plath LC, Hiyane SG, Masson CL, Dibb WD, McClernon FJ (1995) Effects of nicotine and caffeine, separately and in combination on cortisol, mood, vigilance, and EEG. Poster presented at the 35th annual meeting of the Society for Psychophysiological Research, Toronto, Ontario

Gilbert DG, Meliska CJ, Plath LC (1997) Noise stress does not modulate effects of smoking/nicotine on β-endorphin, cortisol, ACTH, glucose, and mood. Psychopharmacology (in press)

Golding JF (1988) Effects of cigarette smoking on resting EEG, visual evoked potentials and photic driving. Pharmacol Biochem Behav 29:23–32

Greenhouse SW, Geisser S (1959) On methods in the analysis of profile data. Psychometrika 24:95–112

Hasenfratz M, Michel C, Nil R, Battig K (1989) Can smoking increase attention in rapid information processing during noise? Electrocortical, physiological and behavioral effects. Psychopharmacology 98:75–80

Knott VJ (1990) A neuroelectric approach to the assessment of psychoactivity in comparative substance use. In: Warburton DM (ed) Addiction controversies. Harwood, Chur, Switzerland, pp 66–89

Lykken DT, Tellegen A, Thorkelson K (1974) Genetic determination of EEG frequency spectra. Biol Psychol 1:245–259

Mangan GL, Golding J (1978) An enhancement model of smoking maintenance. In: Thorton RE (ed) Smoking behaviour: physiological and psychological influences. Churchill Livingstone, Edinburgh, Scotland, pp 87–114

Maxwell Consumers Report (1990) Year-end industry tobacco sales. Wheat First Securities, Philadelphia

Norton R, Brown K, Howard R (1992) Smoking, nicotine dose and the lateralisation of electrocortical activity. Psychopharmacology 108:473–479

Oldfield RC (1971) The assessment and analysis of handedness: the Edinburgh Inventory. Neuropsychologia 9:97–114

O'Neill ST, Parrot AC (1992) Stress and arousal in sedative and stimulant cigarette smokers. Psychopharmacology 107: 442–446

Perlick DA (1977) The withdrawal syndrome: nicotine addiction and the effects of stopping smoking in heavy and light smokers. Dissertation Abstracts International, 38(01), 409B, University Microfilms No. 77/4833

Pritchard WS (1991) Electroencephalographic effects of cigarette smoking. Psychopharmacology 104:485–490

Pritchard WS, Robinson JH, DeBithizy JD, Davis RA, Stiles MF (1995) Caffeine and smoking: subjective, performance and psychophysiological effects. Psychophysiology 32:19–27

Warburton DM (1990) Psychopharmacological aspects of nicotine. In: Wonnacott S, Russell MAH, Stolerman IP (eds) Nicotine psychopharmacology: molecular, cellular and behavioural aspects. Oxford Press, Oxford, pp 77–111

Warburton DM, Wesnes K (1984) Effects of cigarettes of varying yield on rapid information processing performance. Psychopharmacology 82:338–342