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Action Profiles of Smoking and Caffeine: Stroop Effect, EEG, and Peripheral Physiology

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HASENFRATZ, M. AND K. BÄTTIG. *Action profiles of smoking and caffeine: Stroop effect, EEG, and peripheral physiology.* PHARMACOL BIOCHEM BEHAV 42(1) 155–161, 1992.—Twenty female regular cigarette smokers and coffee drinkers performed a numerical Stroop task in a 2 × 2 (caffeine × smoking) prepost crossover design. In the easier of the two different versions, caffeine and smoking reduced the reaction times (RT's) when given alone, but there was no additive effect. The Stroop effect itself (difference between RT's to numbers and RT's to symbols) was reduced by the two treatments only in the more difficult version, but the combination did not differ from the placebo condition. The physiological reactions to both treatments were additive, although the two reaction profiles were different. Smoking increased heart rate, blood pressure, finger vasoconstriction, respiratory frequency, EEG dominant α-frequency, and β power and reduced respiratory amplitude, EEG δ and θ power. Caffeine increased blood pressure, finger vasoconstriction, motor activity, frontal EMG, and EEG θ power and decreased heart rate and EEG β power.

Stroop task Mental performance Caffeine Smoking Cardiovascular

SMOKERS are known to report higher coffee consumption than nonsmokers [for a review see (2)], and in daily life they can also quite commonly be observed smoking a cigarette while drinking coffee. However, as both nicotine and caffeine have stimulant properties, one might expect the contrary, namely that coffee should diminish the desire for a cigarette and vice versa. Since both nicotine and caffeine are widely believed to help in mental concentration, to enhance vigilance, and to facilitate even complex intellectual behavior (2,14,18), the two substances may have interactive effects which might explain their combined use.

Using different tasks, Wesnes and Warburton (19) investigated the effects of smoking and nicotine tablets on mental performance. Smoking and nicotine prevented attentional decreases in prolonged vigilance tasks and improved target detection and decreased reaction times (RT's) in a rapid information processing (RIP) task. This task requires both continuous attention to the odd versus even modality of each of the single-digit stimuli presented and short-term memory for the same modality of the two previous digits, as a response is required whenever three consecutive digits are either even or odd. Similar improvements after smoking have also been found with this task using a subject-paced rather than fixed-presentation rate of the stimuli (10).

The effects of caffeine on mental efficiency, however, are, according to several reviews (1,2,7), more equivocal. Nevertheless, evidence has been presented that caffeine also improves performance in the subject-paced RIP task (3). But whereas caffeine alone and smoking alone were found to improve RIP performance, this was not the case for the combination of the two treatments (9). This negative finding was attributed to possible ceiling limitations of this task.

Another dimension of information processing is assessed with the Stroop task (17). The "Stroop effect" is defined as the performance difference between processing conflicting information (such as the word RED printed in green letters) and processing nonconflicting information (such as the word RED printed in red letters). Wesnes and Warburton (19) found that performance improved (i.e., a decrease of the Stroop effect) after the ingestion of nicotine tablets (1 and 2 mg nicotine), whereas Foreman et al. (8) reported an increase of the Stroop effect with caffeine using a numerical version of the Stroop task rather than the more classical color-word version.

In addition to general questions regarding which cognitive functions are affected by the two substances and in what manner, the above reports that both caffeine and smoking improve RIP performance, whereas nicotine improves and caffeine impairs Stroop performance, also raise the more specific ques-

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tion of whether the two versions of the Stroop task assess the same cognitive functions. Toward a possible elucidation of this problem, the present study was carried out using the numerical Stroop task to investigate the effects of smoking and caffeine alone and combined.

In order to assess the action profiles of smoking and caffeine on a broader scale, a series of physiological parameters were also measured.

METHODS

Subjects

Twenty female regular smokers and coffee drinkers with a mean age of 28.3 years (range 21–39) participated in the study. Their mean self-reported coffee consumption was 5.4 cups/day (range 3–13) of caffeine-containing coffee, and they smoked 23.4 cigarettes/day (range 15–38). They were all in good health, weighed 58.6 kg (range 47–75), and had a mean height of 169 cm (range 158–176). After 8:00 p.m. on the evening preceding the experimental days, they were required to abstain from smoking and not to eat or drink caffeine-containing substances. Further, they were required to eat a standardized breakfast provided by the laboratory (60 g Darvida crispbread and a cup of decaffeinated coffee) at home before coming to the laboratory. The subjects were selected responders to an advertisement in a local newspaper and their fee consisted of a fixed sum plus an efficiency bonus.

Numerical "Stroop" Task

The numerical Stroop task used in the present study was adopted from Foreman et al. (8). It consisted of counting up to four geometrical symbols or numerical figures appearing on the screen. The symbols constituted nonconflicting information requiring answers such as "2" for the stimulus "▲▲" or "3" for the stimulus "▲▲▲". The numerical figures constituted conflicting information, which required suppressing the numerical value of each figure and simply counting their number to give answers such as "2" for the stimulus "4 4" or "3" for the stimulus "1 1 1." The numerical figures in a single stimulus all had the same value, but this value was never congruent with the number of figures. The four response buttons (marked 1–4) had to be pressed as follows: the number 1 with the left middle finger, the number 2 with the left forefinger, the number 3 with the right forefinger, and the number 4 with the right middle finger. The subject's response started the next stimulus presentation after a delay of either 0 or 1 s.

Each task period lasted about 15 min and was divided into eight blocks, each consisting of 55 randomly selected stimuli: two blocks with symbols and two blocks with numbers, both with no delay; two blocks with symbols and two blocks with numbers, both with a 1-s delay. The sequence of these blocks was balanced.

The mean RT was computed for each condition separately. The Stroop effect was calculated as the difference between the mean RT's to the interference (numbers) and control (symbols) stimuli for the corresponding interval conditions.

Physiological Recordings

Blood pressure. Blood pressure was measured from the control room using an automatic measuring set (arm cuff; Tonomed electronic, Speidel & Keller AG, Germany) immediately before and after each rest phase.

The following signals were continuously recorded with an AT-compatible microcomputer and stored on streamer tape for later off-line analysis.

ECG. The electrocardiogram was recorded with Beckman Ag/AgCl electrodes fixed below the middle of the right clavicle, below the last rib on the left and, for the reference electrode, below the last rib on the right. The R-wave peaks of the ECG were detected using an ECG cardiometer (Cardiotronics AG, Stockholm) and were digitally recorded.

Finger pulse amplitude, finger and ear pulse arrival time. Miniature photosensors were placed at the palmar surface of the distal phalanx on the left ring finger and at the left earlobe. The finger and ear pulse arrival times were computed as the time between the R-peak of the ECG and the point at which the finger or the ear pulse amplitude began to increase.

EMG. The electromyogram of the musculus frontalis was recorded with three Beckman Ag/AgCl electrodes arranged in a horizontal line on the middle of the forehead.

Respiration. Respiratory amplitude and frequency were registered with the strain-gauge method (a conducting tape sewed on an elastic belt).

Body movement. Body movements were measured with four piezoelectrical crystals, centrally installed under the seat. The impulses of the three dimensions were recorded as sum vector (Kistler, Piezo Instrumentation, Type 9251A).

EEG and EOG. EEG activity was recorded with goldcup electrodes from C_z, P_3, and P_4 (international 10–20 system). Combined ear references with resistances between them were used, and a midforehead electrode served as ground. All electrode impedances were kept below 5 kΩ. The signals were amplified with bandpasses from 0.2 to 25 Hz. EOG activity was monitored with Beckman Ag/AgCl electrodes placed below the left infraorbital ridge and above the left supraorbital ridge. The signal was monitored with a bandpass setting of 0.5 to 25 Hz.

Carbon monoxide. The carbon monoxide (CO) concentration of the expiratory air was measured at the beginning and end of each session as well as immediately before and after smoking using the EC50 Micro Smokerlyzer (Bedford Instruments, England).

Subjective Parameters

Subjective performance after each task period and the subjective coffee strength after the treatment period were assessed using 100-mm scales. [Subjective performance: "How would you judge your task performance?" (left end labeled with: poor; right end labeled with: good); coffee strength: "How strong did you find the coffee?" (weak–strong)].

State anxiety was assessed at the beginning and end of a session using the German version of the Spielberger State-Trait Anxiety Inventory questionnaire (13).

Procedure

In a training session the subjects were familiarized with the laboratory situation and allowed to practice the Stroop task. No physiological parameters were recorded. After that, each subject took part in four test sessions according to a 2 × 2 design with the two manipulated factors caffeine and smoking. The order of the manipulations was balanced.

After the subject's arrival at the laboratory, the electrodes were attached and CO in the expiratory air was measured. Continuous recordings of the physiological parameters started with a first 5-min rest period before and after which blood pressure was measured. Then the first Stroop task period,

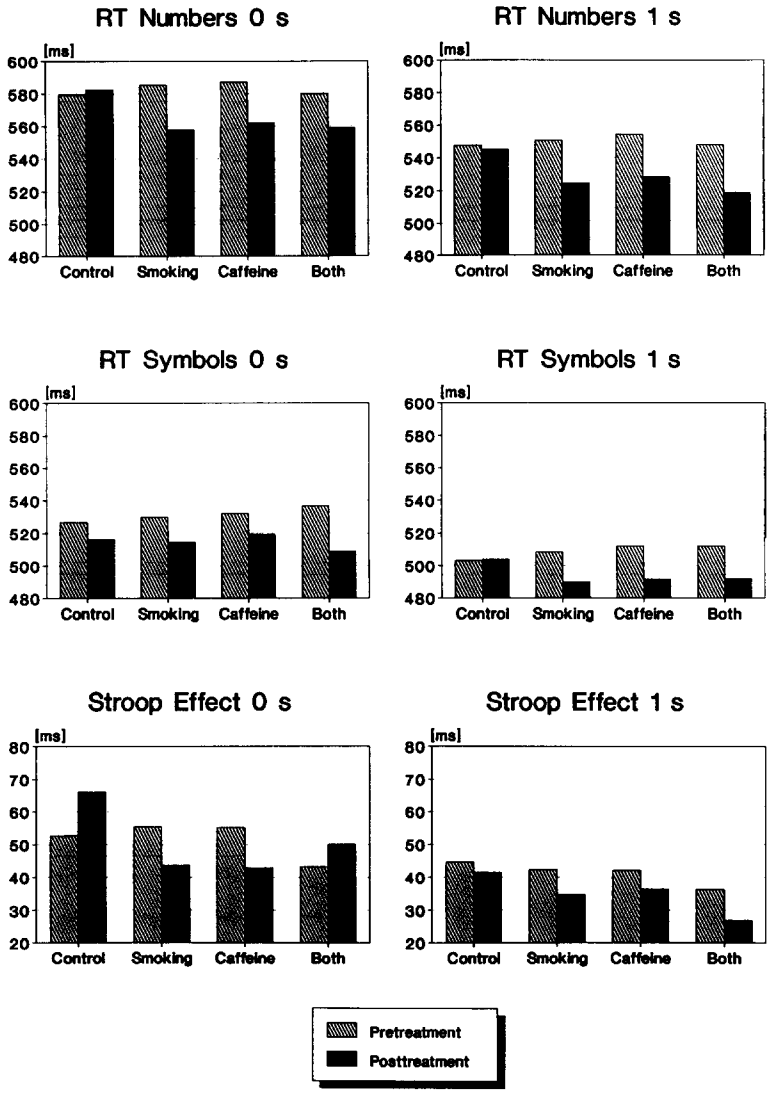

FIG. 1. Mean reaction times (RT) to the numbers and symbols of the pre- and posttreatment runs of the Stroop task (upper four panels), and mean Stroop effects of the pre- and posttreatment runs (lower two panels).

lasting about 15 min, was started. This was followed by a second 5-min rest period with blood pressure measurements immediately before and after. Then subjective performance was rated and 150 ml decaffeinated coffee (with or without an additional 250 mg caffeine) were served. Twenty min later, another item of the scalometric questionnaire was checked (subjective coffee strength), CO was measured a second time, and then the first of two cigarettes was lighted (in the smoking condition). This cigarette was smoked totally and the second one as far as the subject chose to. After the subjects had finished with the second cigarette, or after a corresponding time lag in the case of the nonsmoking condition, CO was measured again and then the same sequence as before the treatment period (5-min rest, 15-min task, 5-min rest) followed. After the last blood pressure measurement a final CO measurement was made and the electrodes were detached.

Data Processing and Statistics

The blood pressure measurements before and after the rest periods were aggregated to one mean value for each rest period.

All continuously recorded physiological data were analyzed off-line so as to obtain the mean values and standard deviations for each successive 10-s period. After a visual artifact control carried out under blind conditions, the 10-s averages were aggregated to means for each experimental period. From the EEG data, the relative power of the δ (0–4 Hz), θ (4–8 Hz), α (8–12 Hz), and β bands (12–25 Hz), as well as the peak frequencies of the α and β bands were determined for each 5-min rest period.

These reduced data sets were then statistically analyzed using the appropriate software programs of the SPSSX and

BMDP packages available on a mainframe computer. For all significance levels of the analyses of variances (ANOVA's) Greenhouse-Geisser probabilities were considered where appropriate.

RESULTS

Reaction Time and Stroop Effect

The upper four panels of Fig. 1 show the mean RT's to the different stimulus categories of the task. RT's were shorter when the stimuli were presented at 1-sec rather than 0-sec postresponse delays and shorter with the symbol than the number stimuli. In a first step overall ANOVA, this produced main factor significance for the postresponse delays, D: $F(1,19) = 73.97, p < 0.001$, and for the Stroop effect (interference), that is, the difference between the RT's to symbols and to numbers, I: $F(1,19) = 168.93, p < 0.001$. Based on this result, the treatment-induced changes in RT's were analyzed separately for each of the four response categories by ANOVA's with the factors P (pre/postadministration), C (caffeine), and S (smoking). With the 1-s postresponse delays, the RT reductions, apparent in Fig. 1, reached significance with caffeine (interaction C × P) for the symbols, $F(1,19) = 4.64$, $p < 0.05$, as well as for the numbers, $F(1,19) = 4.53$, $p < 0.05$, and the same result was obtained with smoking (interaction S × P) both for the symbols, $F(1,19) = 4.63, p < 0.05$, and for the numbers, $F(1,19) = 6.51, p < 0.05$. With the 0-s delay, however, significance for this interaction was missed, although there was a tendency in the same direction for smoking but not for caffeine, symbols: $F(1,19) = 3.13, p < 0.1$; numbers: $F(1,19) = 3.38, p < 0.1$.

Figure 1 further suggests that the combined smoking-caffeine treatment mostly failed to produce additive effects. This resulted in significant C × S × P interactions for the 1-s

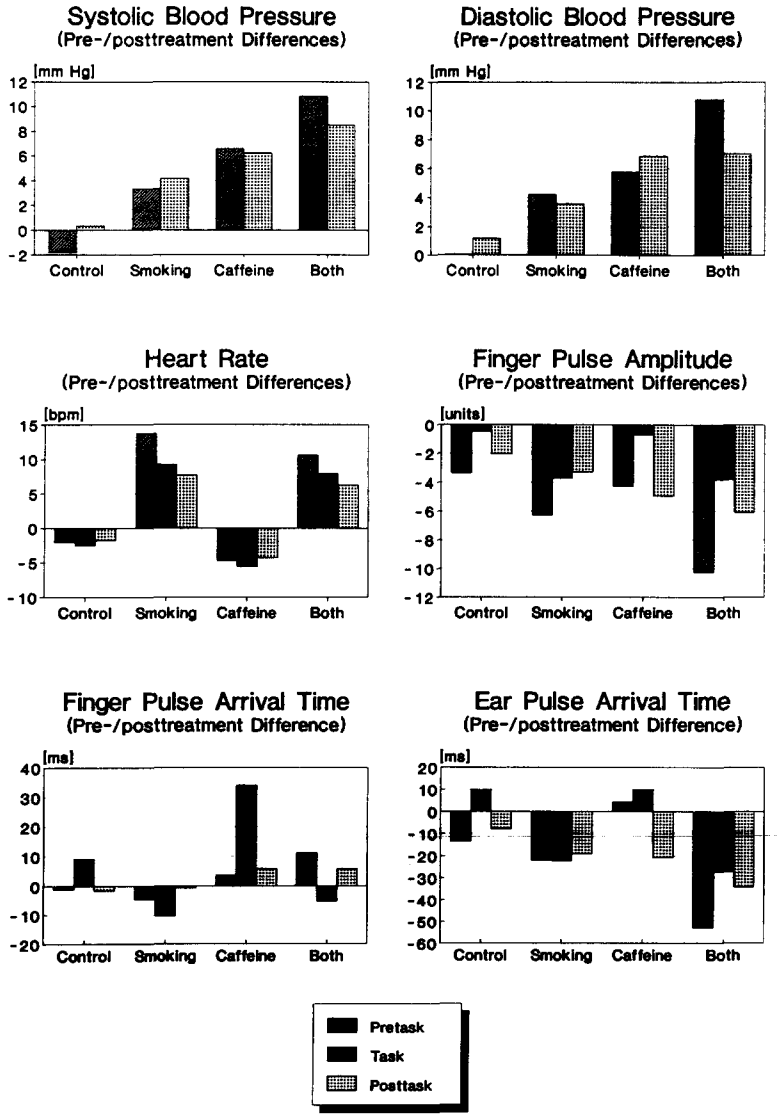

FIG. 2. Mean pre- to posttreatment differences of the cardiovascular parameters. Bars represent means of phases (5-min pretask rest, 15-min task, 5-min posttask rest). For blood pressure no measurements were available during the task phases.

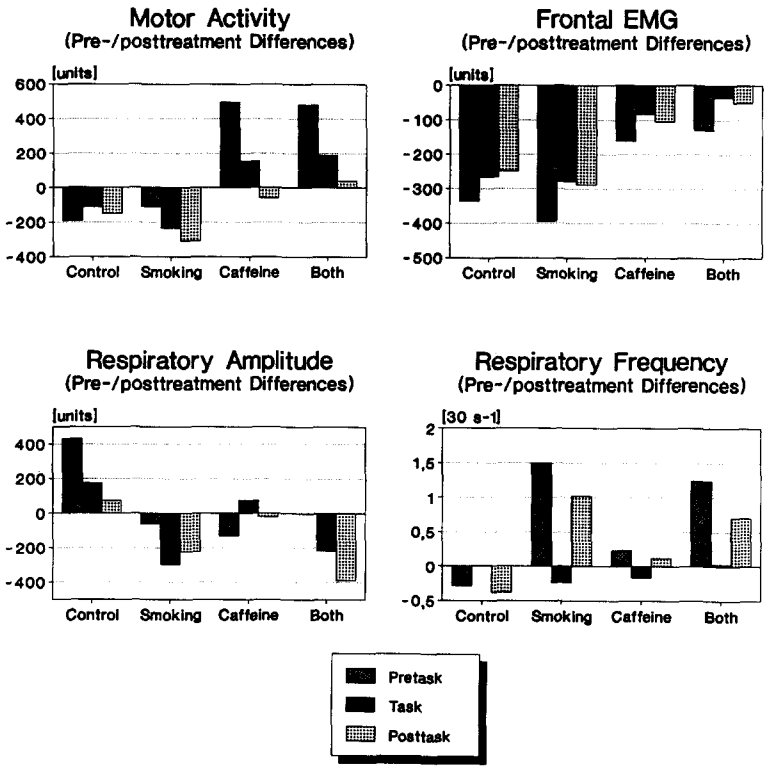

FIG. 3. Mean pre- to posttreatment differences of the peripheral physiological parameters. Bars represent means of phases (5-min pretask rest, 15-min task, 5-min posttask rest).

delay condition for both the symbols, $F(1,19) = 6.96$, $p < 0.05$, and the numbers, $F(1,19) = 9.97$, $p < 0.01$, but for the 0-s delay condition for the numbers only, $F(1,19) = 9.41$, $p < 0.01$.

Separate analogous ANOVA's carried out with the number-symbol differences, representing the Stroop effect, support the picture shown in the lower two panels of Fig. 1. With the 1-s delay, only a general and treatment-independent prepost improvement was obtained, P: $F(1,19) = 4.74$, $p < 0.05$. With the 0-s delay, the caffeine-smoking interaction suggested by Fig. 1 was supported by significance being reached by the corresponding C × S × P interaction, $F(1,19) = 12.23$, $p < 0.01$. Paired t-tests further confirmed significance for the caffeine-control difference, $t = 2.89$, $df = 19$, $p < 0.01$, as well as for the smoking-control difference, $t = 2.18$, $df = 19$, $p < 0.05$, but not for the smoking plus caffeine-control difference.

Cardiovascular Effects

Pretreatment levels of the cardiovascular parameters did not show any significant differences among the four test sessions, as confirmed by separate three-way ANOVA's. Thus, pre- to posttreatment differences were used for the further analyses. Figure 2 suggests that all cardiovascular parameters were affected by smoking as well as by caffeine and that the effects were additive for both treatments combined. Three-way ANOVA's with the factors C (caffeine), S (smoking), and W (within: time protocol) carried out on the pre- to posttreatment differences revealed significant smoking-induced increases of systolic blood pressure (SBP), $F(1,19) = 13.00$, $p < 0.001$; diastolic blood pressure (DBP), $F(1,19) = 15.55$, $p < 0.001$; and heart rate (HR), $F(1,19) = 209.17$, $p < 0.001$; as well as decreases of the finger pulse amplitude, $F(1,17) = 9.97$, $p < 0.01$; finger pulse arrival time, $F(1,17) = 5.97$, $p < 0.05$; and ear pulse arrival time, $F(1,18) = 6.99$, $p < 0.05$. Significant caffeine-induced increases of SBP, $F(1,19) = 24.89$, $p < 0.001$; DBP, $F(1,19) = 20.51$, $p < 0.001$; and finger pulse arrival time, $F(1,17) = 9.91$, $p < 0.01$; as well as decreases of HR, $F(1,19) = 9.53$, $p < 0.01$; and finger pulse amplitude, $F(1,17) = 9.91$, $p < 0.01$, were also obtained.

In addition, smoking interacted significantly with the time protocol (interaction S × W) for all cardiovascular parameters except SBP and ear pulse arrival time. Generally, the effect was the greatest immediately after smoking (pretask resting phase) and decreased thereafter, DBP: $F(1,19) = 5.89$, $p < 0.05$; HR: $F(2,38) = 14.47$, $p < 0.001$; finger pulse amplitude: $F(2,34) = 5.38$, $p < 0.01$. For the finger pulse arrival time, the greatest effect of smoking was obtained during the Stroop task, $F(2,34) = 15.58$, $p < 0.001$. In the case of caffeine, a similar significant C × W interaction was obtained for finger pulse amplitude only, $F(2,34) = 4.38$, $p < 0.05$, indicating that the caffeine-induced decrease was greatest during the task phases (Fig. 2).

No significant C × S interactions were obtained, suggesting therefore that the cardiovascular effects of the two treatments were additive.

Motor Activity, EMG, and Respiration

Analogous C × S × W ANOVA's carried out on the pre- to posttreatment differences supported the drug effects suggested by Fig. 3. Motor activity increased with caffeine, C:

$F(1,19) = 9.54$, $p < 0.01$, particularly for the pretask rest period in contrast to the decreases with the control and smoking alone conditions. The marked EMG decreases seen in the control and smoking alone conditions were considerably attenuated by caffeine, $F(1,19) = 5.09, p < 0.05$. Smoking did not affect these two parameters. On the other hand, respiratory amplitude and frequency were not affected by caffeine, but respiratory amplitude decreased, $F(1,19) = 10.00$, $p < 0.01$, and respiratory frequency increased, $F(1,19) = 9.07$, $p < 0.01$, due to smoking. The smoking effect on respiratory frequency was predominant in the rest phases but disappeared during the task, as confirmed by the significant S × W interaction, $F(2,38) = 7.64, p < 0.01$.

No interactive effects of the two treatments were obtained.

EEG Parameters

In a first step, overall ANOVA's led to significant main factor effects for the three leads in the power of all four EEG bands. Thus, in a second step, similar ANOVA's were performed for each of the three leads separately, which revealed only a few significant within factor effects (pre- vs. posttask rest phase). Based on this result, a final set of ANOVAs was limited to the differences between the pretreatment–posttask and the posttreatment–pretask measures, and those that were significant are summarized in Table 1. These differences were due to increases in α-peak frequencies and β power and decreases in δ and θ power with all three leads with smoking, and to increases in β power and decreases in θ power, but only in P_3 and P_4, with caffeine. Further, there were no significant interactions between the two treatments, indicating that their combined effect was additive.

Respiratory CO and Subjective Ratings

Compliance with the abstinence instruction was confirmed by low and stable initial respiratory CO values (on the average 10.73 ppm at the beginning of the sessions and 10.55 ppm presmoking). Smoking increased respiratory CO on the average to 21.2 ppm postsmoking and to 18.63 ppm at the end of the session, whereas the corresponding values in the nonsmoking conditions remained at 10.53 ppm and 10.2 ppm, respectively. There was no significant C × S interaction.

Subjective ratings of performance on the Stroop task were higher after caffeine than after placebo as supported by the C × P interaction, $F(1,19) = 10.50, p < 0.01$. However, for smoking no analogous effect was found.

The analysis of the subjective ratings of coffee strength revealed no significant differences between the caffeine and the placebo condition.

State anxiety, assessed at the beginning and end of a session, was not significantly affected by the treatments.

DISCUSSION

The improvements in Stroop performance obtained in the present study with both smoking alone and caffeine alone are in agreement with the Stroop effect reduction reported by Wesnes and Warburton (19) after the administration of sublingual nicotine tablets, but in contrast to the Stroop effect increase reported by Foreman et al. (8) after the ingestion of caffeine. The present study differs from the Foreman et al. study not only by including pretreatment measurements but also by manipulating task difficulty with varied stimulus delays and by contrasting caffeine with nicotine through smoking as another stimulant.

The differential results obtained with these manipulations may help to better understand the difficulties inherent in the Stroop task and the limitations of the action of mild stimulants such as caffeine and nicotine.

With 1-s response-stimulus delays as the easier version of the task, not only were the RT's to both types of stimuli shorter, but the Stroop effect as the difference between the two types of stimuli was also smaller than with the more difficult 0-s delay version. Nevertheless, both caffeine alone and smoking alone still reduced the RT's to both types of stimuli. The Stroop effect, on the other hand, seemed to become too small to leave room for a pharmacological improvement.

With the no-delay condition as the more difficult version of the task (because there was no recovery period after a single stimulus), both treatments failed to affect the RT's to both types of stimuli but, instead, smoking and caffeine both reduced the Stroop effect. A similar result was recently reported by Provost and Woodward (15), where the time taken to perform the incongruent color-word naming task was decreased after a 2-mg nicotine gum, whereas there was no effect on the time taken to name symbols or to read words written in black.

However, the most important result is that the two treatments antagonized each other so that the combination of caffeine and smoking did not differ from the control condition. This result is in line with the findings of Kerr et al. (11), who found a similar antagonistic effect of nicotine chewing gum and caffeine capsules on "memory reaction time" (Sternberg task), and with those of Hasenfratz et al. (9), who found a

TABLE 1

F-VALUES OF THE CAFFEINE × SMOKING ANOVA OF THE EEG PARAMETERS (DIFFERENCES BETWEEN POSTDRUG MINUS PREDRUG BASELINE) FOR THE THREE LEADS P_3, C_Z, AND P_4

		Caffeine			Smoking		
Parameter	(*df*)	P_3	C_Z	P_4	P_3	C_Z	P_4
Dom. α	(1,17)			4.05*	27.68§	3.31*	17.29§
Dom.β	(1,17)					4.09*	
δ power	(1,18)				19.30§	12.57‡	14.05‡
θ power	(1,18)	9.70‡		6.43†	10.13‡	13.57‡	6.60†
α power	(1,18)						
β power	(1,18)	31.70§		9.36‡	36.56§	43.31§	33.29§

*$p < 0.1$, †$p < 0.05$, ‡$p < 0.01$, §$p < 0.001$.

significant interaction between caffeine and smoking for the processing rates of the RIP task.

Another possible explanation for this interaction might be that the Stroop effect is less stable than the RT's, as suggested by the pretreatment levels of Fig. 1 as well as by findings of Bittner et al. (4), who reviewed the test-retest reliability of 114 measures of different mental performance tasks.

Taken together, it might be concluded that in the easier task version caffeine and smoking primarily affected the RTs, whereas in the more difficult task version they primarily affected the solving of the problem posed by the interference stimuli.

The electrocortical effects, as analyzed only during rest phases, are largely in line with those reviewed by Knott (12) for a series of psychoactive substances. Smoking decreased the power of the lower frequency bands (δ and θ bands) but increased the dominant α-frequency and the high-frequency (β) band. Caffeine, on the other hand, also decreased the θ band, but, in contrast to the suggestions made by Knott (12), we found an increase in β power rather than a decrease in α power. In contrast to the cognitive parameters, the effects of the two treatments on electrocortical parameters did not interact.

The effects of smoking alone and caffeine alone on cardiovascular parameters were mostly as expected on the basis of earlier findings (9,16). Caffeine increased blood pressure more than smoking, but heart rate decreased after caffeine whereas it increased after smoking. As an extension of the earlier results, it was shown that the cardiovascular effects of both treatments were additive and did not interact.

Whereas motor activity and EMG were not affected by smoking, caffeine increased both measures. This result can be compared with increased tremor in humans [as reviewed by Calhoun (5)] and increased spontaneous motor activity in animals [as reviewed by Dews (6)] after caffeine treatment.

A shift in respiration from a lower frequency with greater amplitude to a higher frequency with smaller amplitude was found after smoking but not after caffeine. With the given design, it is not clear whether smoking and caffeine affect respiration in a differential manner or whether this stimulation was a consequence of the smoke from the cigarette.

Finally, a somewhat surprising result was obtained for the subjective parameters. On the one hand, the subjects did not realize when caffeine had been added to the coffee, as suggested by the fact that the subjective coffee strength did not significantly differ between the two caffeine conditions. On the other hand, they rated their task performance higher after caffeine, thus suggesting a psychoactive effect of caffeine. Surprisingly, a similar effect was not found for smoking, which would suggest that smoking or nicotine may have different psychoactive effects.

Taken together, on the peripheral physiological level smoking and caffeine showed different profiles of action, which appeared to be additive when the two treatments were combined. On the level of cognitive functions and electrocortical parameters, the results are less clear.

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