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Effects of Single Doses of Alcohol and Caffeine on Cigarette Smoke Puffing Behavior

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NIL, R., R. BUZZI AND K. BÄTTIG. *Effects of single doses of alcohol and caffeine on cigarette smoke puffing behavior.* PHARMACOL BIOCHEM BEHAV **20**(4) 583–590, 1984.—Puffing behavior (number of puffs, puff duration, puff volume, peak pressure, peak flow, peak latency, and puff interval) and pre- to postsmoking Δ tidal CO difference were measured in female subjects in order to assess separate and combined effects of ethanol and caffeine. The subjects smoked two cigarettes of their habitual brand in a preliminary familiarizing session and in each of the subsequent four test sessions. The treatments administered after smoking the first cigarette in the test sessions were: alcohol placebo and caffeine placebo; alcohol placebo and caffeine; alcohol and caffeine placebo; alcohol and caffeine. Test-retest reliability across the first cigarette of each session (which was not smoked under the influence of the treatments) was remarkably high for all the puffing parameters. Ethanol in the dose of 0.7 g/kg intensified cigarette smoking of the second cigarette by increasing Δ tidal CO, average puff volume, and total puff volume per cigarette, whereas 0.5 g/kg ethanol and 5 mg/kg caffeine given alone or combined with ethanol failed to influence puffing behavior consistently.

Alcohol Caffeine Cigarette smoking

POSITIVE correlations between average daily cigarette consumption and consumption of alcoholic and caffeine-containing beverages have been found in several investigations [3, 4, 28, 29]. Such correlations raise the question whether this might be a consequence of life style or of pharmacological interactions between alcohol, caffeine, and nicotine or other smoke constituents. Under laboratory conditions Griffiths *et al.* [11] found that in alcoholics ethanol consumption was followed by an enhanced rate of cigarette smoking. Similar effects of ethanol consumption on cigarette smoking were also shown for social drinking [21]. In a recent review article Adesso [1] proposed that these results, together with the numerous reports on correlations between consumption of alcohol and nicotine, suggest a dose related pharmacological interaction between the two substances.

Experiments on the effect of caffeine on smoking have yielded more controversial results. Kozlowski [17] found that nicotine mouth intake during smoking was higher in the caffeine placebo condition than in any of three different caffeine dosage conditions, especially in light coffee consumers. Marshall *et al.* [19,20] observed that a greater number of cigarettes was smoked by subjects receiving coffee (with or without caffeine) than by subjects in no-drink or water control groups.

Several studies suggest that smokers adjust their smoking behavior so as to regulate nicotine intake [18,27]. Since individual puffing behavior and inhalation during the smoking of single cigarettes seem to be the main contributors in this regulation [2,5], the present experiment was designed to investigate the pharmacological effects of alcohol and caffeine on parameters of smoke puffing behavior and respiratory smoke inhalation. Each subject was monitored for puffing and inhalation behavior while smoking two personal-brand cigarettes. Such cigarettes were preferred to any standardized cigarettes, because it has been shown that nicotine and CO uptake are nearly independent of the respective smoke yields of the cigarettes [5,27] and also because personal-brand cigarettes were expected to interfere less with "natural" smoking. Data obtained with the first cigarette, smoked before the experimental treatments with caffeine, alcohol, their placebos and combinations, were used to assess test-retest reliability of puffing and inhalation behavior, whereas the data obtained with the second cigarette were used to assess for the treatment effects.

METHOD

Subjects

Twenty female paid volunteers (between the ages of 18 and 50) served as subjects. They were all regular smokers and reported themselves to be in good health. On the test

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days they were required to abstain from caffeine and alcohol, but not from cigarette consumption. They were instructed about the experiment, but were not informed about any placebo treatments. They all signed a consent form before starting the experiment.

Apparatus and Questionnaires

The following technical equipment was used: (a) A cigarette-holder puffing-flowmeter [9] was used to obtain analogue signals for puffing flow and puffing pressure (Projects CGC Ltd., Tewkesbury, England). (b) A puffing analyzer, developed in this laboratory, was connected with the puffing flowmeter. It delivered puff-by-puff digital printouts for the six parameters: puff volume, puff duration, peak pressure, latency to peak pressure, peak flow, and the interval from the preceding puff. (c) A CO analyzer (model 866, Beckman Instruments Inc.) was used to measure the concentration of mixed expiratory tidal air, which was collected in a Teflon bag for 5 min during normal breathing and simultaneously analyzed until a stable CO concentration reading was obtained [26]. (d) The module Elag Be-207-S (Elag, Cologne, Germany) was used to measure pulse rate and blood pressure. (e) The Alcohol Test Computer ATC1 (Joma Trading AG, Winterthur) was used for breath ethanol level assessments. (f) For graphical recording of tidal breathing an elastic belt fitted with a strain gauge was fixed around the chest. Temporal positioning of puffing within an ongoing respiratory cycle was scored with 1 for expiratory or end inspiratory puffing, with 2 for end inspiratory puffing, with 3 for middle inspiratory puffing, with 4 for early inspiratory puffing, and with 5 for puffing at the onset of the inspiratory phase. (g) Saliva samples were stored in a freezer (−23°C) until being delivered to the Institute for Toxicology of the University of Zürich and the Swiss Federal Institute of Technology for analysis of caffeine concentration (ppm) by the HPLC method [17].

The set of questionnaires consisted of: (a) A 24-item smoking-habit questionnaire including questions about alcohol and coffee use. (b) The FPI questionnaire, a German language personality test yielding scores for extraversion (E), neuroticism (N), and openness (O), according to the Eysenck Personality Inventory, and a series of additional personality subscales [10]. (c) The d2 vigilance test [6]. This task involved crossing out each d marked with two apostrophes in a row of 47 d's marked with one to four apostrophes. A subject was permitted 20 sec to complete each row. To assess the subjective need for smoking a 10-cm analogue scale marked at the end points with "no need to smoke at all" and "very strong need to smoke" was presented immediately before smoking.

Experimental Design and Procedure

Each subject came to the institute for five sessions at weekly intervals. All experimental sessions were started between 4:30 and 5:30 p.m. and were carried out according to the following experimental protocol:

Arrival at laboratory 4:30	saliva sample and breath alcohol assessment (to verify alcohol and caffeine abstinence on the test days) CO analysis of expired air (CO baseline) measurement of pulse rate and blood pressure assessment of the subjective need for smoking
4:45	smoking the first cigarette (own brand) measurement of pulse rate and blood pressure (immediately after the last puff) "alcohol" cup CO analysis of expired air (5 min after the last puff)
5:15	"coffee" cup (within 1 min) d2 vigilance task for occupational standardization (18 min) saliva sample
5:30	breath alcohol assessment CO analysis of expired air measurement of pulse rate and blood pressure assessment of the subjective need for smoking
5:45	smoking the second cigarette (own brand) measurement of pulse rate and blood pressure CO analysis of expired air

For the first familiarizing session no beverages were administered, and the period between smoking the two cigarettes was used to fill out the FPI personality questionnaire instead of performing the d2 letter cancellation task. Each subject consumed the "coffee" and "alcohol" beverages in a double blind latin square design in the following combinations: alcohol placebo and caffeine placebo; alcohol placebo and caffeine; alcohol and caffeine placebo; alcohol and caffeine.

Alcohol, Caffeine and Placebo Preparations

Ethanol was administered to ten subjects (Group I) at a dose level of 0.5 g/kg body weight and to ten other subjects (Group II) at a dose level of 0.7 g/kg body weight. Gin flavoring and 96 Vol% ethanol were mixed and diluted with orange juice to yield a 15 Vol% ethanol beverage. The alcohol placebo beverage consisted of an equal volume of orange juice with gin flavoring and 3–5 ml of ethanol floated on the surface. Caffeine (anhydrous powder) (Fluka AG, Buchs, Switzerland), administered to both groups at a dose level of 5 mg/kg body weight, was dissolved in 100 ml decaffeinated coffee, which was also used as caffeine placebo.

Data Analysis

All experimental data were punched on cards and statistically analyzed on a large CDC computer using SPSS and BMDP software systems. The analyses involved a repeated measure design ANOVA (BMDP program P2V) in order to test for treatment effects on the posttreatment puffing variables and physiological pre- to postsmoking differences, separately for Group I and Group II and for the two groups combined. The puffing variables of the first cigarette of each session were also submitted to the Kendall coefficient of concordance procedure (BMDP program P3S) in order to assess the test-retest reliability of the puffing measurements. The final crosscorrelational analysis used the Kendall procedure and included, in addition to all experimental data, the entire set of questionnaire scores and answers.

TABLE 1

AVERAGE PUFFING BEHAVIOR FOR THE FIRST CIGARETTE SMOKED IN THE FIRST SESSION, SEPARATED FOR THE TWO GROUPS

Variable	Mean ± SEM Group I	Group II	*t* Group I vs. Group II
Total puff vol (ml)	619 ± 329.3	579 ± 244.8	0.30
Puff volume (ml)	41.1 ± 13.6	39.5 ± 8.9	0.31
Peak pressure (cm H_2O)	25.4 ± 10.1	27.9 ± 8.5	0.6
Peak latency (sec)	0.60 ± 0.13	0.61 ± 0.15	0.02
Puff duration (sec)	1.67 ± 0.50	1.57 ± 0.41	0.47
Peak flow (ml/sec)	41.1 ± 13.7	39.5 ± 8.9	0.31
Puff interval (sec)	25.9 ± 14.8	25.7 ± 11.9	0.04
Number of puffs	14.8 ± 3.8	14.7 ± 4.7	0.05
Δ tidal CO (ppm)	4.8 ± 2.5	3.0 ± 2.6	1.56
Inhalation score	2.7 ± 0.83	2.1 ± 0.62	1.71

RESULTS

Test-Retest Reliability and Averages of Puffing Behavior

Table 1 shows the average puffing behavior of each of the two groups for the first cigarette which was smoked in the first, "familiarizing," test session. According to the *t* values, no differences could be found between the groups. This was also the case for the other first cigarettes smoked (without treatment) in the following four test sessions. Kendall coefficients of concordance across the puffing variables obtained with the first cigarettes smoked in all five sessions are presented in Table 2 and reached exceptionally high levels of significance in both groups, indicating that the individual ranking order of puffing parameters turned out to be remarkably constant. Within the two inhalation variables, the low value of the Kendall coefficient of concordance for Δ tidal CO in Group I suggests wide intraindividual differences in the depth of inhalation for this group. This is in contrast to the high value of this coefficient for the inhalation score in the same group, giving evidence of a more stable time patterning of puffing and respiration. On the other hand, the same analysis in Group II showed both stable Δ tidal CO values and inhalation scores.

Effects of Alcohol and Caffeine

According to the breath analyzer data, the blood alcohol levels rose to 0.025%±0.08 in Group I and to 0.052%±0.06 in Group II. The caffeine saliva concentration for both groups combined rose to 4.5±2.2 ppm. In a first step, separate 2×2 ANOVA analyses were performed within each group for each of the posttreatment puffing parameters and for the pre- to postsmoking differences in tidal CO, pulse rate, and blood pressure. The averaged cell means and F values are summarized in Table 3 for Group I and in Table 4 for Group II. Alcohol significantly increased Δ tidal CO, average and total puff volume in Group II, which was given 0.7 g/kg ethanol, and had no significant effects in Group I, given 0.5 g/kg ethanol. Caffeine was significantly effective in Group I only with prolonged puff intervals and a decreased subjective need for smoking. Significant ethanol-caffeine interactions were obtained in Group I with relatively high values for peak pressure with the combined treatment and in Group II for relatively long latencies to peak pressure with the combined treatment. None of the other variables (puff duration, peak flow, number of puffs, inhalation score, and the pre- to posttreatment differences in pulse rate and systolic and diastolic blood pressure) were significantly affected by the treatments.

A second-step ANOVA procedure with the two groups combined attempted to test for the effects of caffeine on a larger sample by neglecting the alcohol dose effect. Although this failed to produce significance for any of the parameters with caffeine as the single factor, it confirmed single factor effects of alcohol (as obtained previously only in Group II) with $F(1,18)=8.48$ ($p<0.01$) for Δ tidal CO and, marginally, with $F(1,18)=3.88$ ($p=0.064$) for the average puff volume. In addition, marginal significance was also obtained with $F(1,18)=4.20$ ($p=0.054$) for the puff intervals and with $F(1,18)=3.60$ ($p=0.074$) for peak pressure. Interactions between alcohol and caffeine reached significance with $F(1,18)=8.06$ ($p<0.01$) for peak pressure and with $F(1,18)=6.84$ ($p<0.02$) for peak latency.

Sample Characteristics and Crosscorrelational Data

No significant differences could be found between the

TABLE 2

KENDALL COEFFICIENTS OF CONCORDANCE ACROSS THE FIRST CIGARETTES SMOKED (BEFORE TREATMENT) FOR ALL FIVE SESSIONS

	Group I		Group II	
Variable	Kendall coefficients of concordance	p	Kendall coefficients of concordance	p
Total puff vol (ml)	0.84	0.00001	0.65	0.0006
Puff volume (ml)	0.70	0.0002	0.77	0.0011
Peak pressure (cm H_2O)	0.64	0.0007	0.88	0.0002
Peak latency (sec)	0.71	0.0002	0.89	0.0002
Puff duration (sec)	0.87	0.00001	0.75	0.0015
Peak flow (ml/sec)	0.74	0.0001	0.81	0.0006
Puff interval (sec)	0.73	0.0001	0.77	0.0011
Number of puffs	0.83	0.00001	0.79	0.00001
Δ tidal CO (ppm)	0.09	—	0.60	0.0014
Inhalation score	0.70	0.026	0.59	0.0016

TABLE 3

REPEATED MEASURE ANOVA RESULTS FOR GROUP I (F VALUES AND CELL MEANS)

	Cell means				F values		
Alcohol Caffeine	Placebo Placebo	Placebo 5 mg/kg	0.5 g/kg Placebo	0.5 g/kg 5 mg/kg	Alcohol F(1,9)	Caffeine F(1,9)	Alcohol × Caffeine F(1,9)
Variables							
Total puff vol (ml)	641.0	621.0	570.0	625.0	0.5	0.2	2.2
Puff volume (ml)	41.4	42.3	38.4	46.4	0.9	3.94	2.68
Peak pressure (cm H_2O)	25.8	26.1	25.2	33.6	3.7	4.3	7.0*
Peak latency (sec)	0.61	0.55	0.62	0.60	0.7	0.8	0.64
Puff interval (sec)	23.0	34.5	30.2	34.9	2.3	8.0*	2.2
Number of puffs	14.4	13.7	14.0	13.2	0.28	2.58	0.01
Δ tidal CO (ppm)	1.7	2.7	3.0	2.9	2.8	1.4	0.9
Subjective need for smoking (mm)	52.0	33.0	49.0	44.0	0.5	10.0*	2.0

*$p<0.05$.

two groups for personality, smoking habit, anamnestic data, or standard smoke yield data of the cigarettes, as presented in Table 5. Correlation analysis between variables of sample characteristics were therefore performed for both groups combined: Openness correlated positively with daily cigarette consumption ($r=0.34$, $p<0.05$) and negatively with latency to the first cigarette/day ($r=-0.44$, $p<0.01$). A positive correlation was obtained between extraversion and machine standard nicotine yield of the cigarettes ($r=0.37$, $p<0.05$). The positive correlation between the number of years smok-

TABLE 4

REPEATED MEASURE ANOVA RESULTS FOR GROUP II (F VALUES AND CELL MEANS)

	Cell means				F values		
Alcohol Caffeine	Placebo Placebo	Placebo 5 mg/kg	0.7 g/kg Placebo	0.7 g/kg 5 mg/kg	Alcohol F(1,9)	Caffeine F(1,9)	Alcohol × Caffeine F(1,9)
Variables							
Total puff vol (ml)	479.0	455.0	521.0	513.0	5.2*	0.2	0.0
Puff volume (ml)	36.2	34.1	38.0	39.0	15.4†	0.2	0.6
Peak pressure (cm H_2O)	30.8	29.4	29.7	31.5	0.3	0.0	1.8
Peak latency (sec)	0.57	0.51	0.54	0.59	0.8	0.1	9.3*
Puff interval (sec)	29.9	28.3	34.2	32.1	2.9	0.4	0.0
Number of puffs	13.5	13.7	13.7	13.3	0.02	0.03	0.10
Δ tidal CO (ppm)	1.7	1.2	3.3	2.4	5.5*	4.6	0.6
Subjective need for smoking (mm)	48.0	40.0	48.0	55.0	2.6	0.0	1.3

*$p<0.05$.
†$p<0.01$.

ing and daily cigarette consumption (r=0.40, $p<0.05$) and the negative correlation between years smoking and latency to the first cigarette/day (r=−0.45, $p<0.01$) suggest stronger smoking habits in subjects with long smoking histories. Alcohol consumption correlated positively with condensate yield (r=0.35, $p<0.05$), but coffee consumption correlated negatively with condensate yield (r=−0.38, $p<0.05$) and CO yield (r=−0.40, $p<0.05$). High intercorrelations between machine standard smoke yields of the cigarettes were to be expected (nicotine yield with condensate yield, r=0.75, $p<0.01$; nicotine yield with CO yield, r=0.58, $p<0.01$; condensate yield with CO yield, r=0.78, $p<0.01$).

Among correlations between sample characteristics and puffing, average daily cigarette consumption was significantly correlated with total puff volume per cigarette in three out of five test sessions only for Group I (Session 3, r=0.55, $p<0.037$; Session 4, r=0.60, $p<0.023$; Session 5, r=0.69, $p<0.009$). All the other correlations showed similar trends in the two groups and are therefore presented in Table 6 for both groups combined. In order to restrict to consistent correlations, only those correlations are presented which reached the level of significance ($p<0.05$) for at least three of the five sessions. The first four correlations underline the consistency of the questionnaire answers, as the number of cigarettes smoked on the test day prior to the experiment increased with the number of years having smoked and with habitual daily consumption, but decreased with prolonged latency to the first morning cigarette. Furthermore, habitual daily consumption also correlated with the pretest baseline measures of expiratory CO. The following five correlations suggest compensation phenomena for light cigarettes. Furthermore, high pH cigarettes seemed to be preferred by heavy smokers (CO baseline and N of cigarettes prior to test). The CO baseline level increased with the number of cigarettes smoked prior to the experiment. The pre- to postsmoking Δ tidal CO levels increased with increasing CO baseline levels. Finally, Δ tidal CO increased with increasing CO inhalation. CO inhalation was estimated by multiplying CO mouth intake (total puff volume ÷ 350×CO machine yield) with the respiratory inhalation score.

DISCUSSION

Alcohol intensified inhalation by increasing Δ tidal CO and puffing behavior by enhancing average puff volume and total puff volume per cigarette in Group II, receiving 0.7 g/kg, but not in Group I, with a 0.5 g/kg ethanol dose. The failure to find alcohol effects in Group I must be attributed to the smaller alcohol dose which was used. However, the larger intraindividual variability in Δ tidal CO shown for this group across the first cigarettes smoked in the sessions could also have biased possible alcohol effects on Δ tidal CO. The affected smoking variables in Group II are the same variables which have been seen to be important factors in "compensation puffing" for different cigarette smoke deliveries [5,12]. Since neither the intervals between puffs nor the number of puffs per cigarette was significantly affected by ethanol, it appears that the single puff volumes and the depth of inhalation, which in this experiment were scored qualitatively, are the main contributors to the enhanced smoke exposure. This effect of ethanol was not accompanied by any enhanced subjective need for smoking a cigarette. The results may, therefore, favor the hypothesis of a pharmacological facilitating effect of ethanol on smoking, as suggested by Griffiths *et al.* [11] and Mello *et al.* [21]. However, the nature of such an interaction might be complex. Myrsten and

TABLE 5

ANAMNESTIC DATA, PERSONALITY RATINGS, SMOKING HABIT, AND CIGARETTE CHARACTERISTICS FOR BOTH GROUPS

	Mean ± SEM		t
Variable	Group I	Group II	Group I vs. Group II
Age (years)	32.8 ± 9.7	28.2 ± 5.9	1.28
Weight (kg)	53.6 ± 6.6	54.3 ± 3.9	0.29
Openness	6.0 ± 1.7	5.1 ± 2.6	0.92
Extraversion	5.0 ± 2.3	6.2 ± 2.0	1.25
Neuroticism	5.3 ± 2.0	5.3 ± 1.2	0.0
Age began smoking (years)	19.3 ± 4.2	17.8 ± 2.0	1.0
Years smoking	14.3 ± 9.8	10.4 ± 5.9	1.05
Duration of longest smoking abstinence (weeks)	11.2 ± 30.1	4.0 ± 7.4	0.72
Alcohol consumption (times/week)	5.0 ± 2.4	4.0 ± 2.5	0.92
Coffee consumption (cups/day)	4.9 ± 4.7	4.4 ± 1.8	0.31
Number of cigarettes/day	19.5 ± 6.8	20.5 ± 11.3	0.24
Latency to 1st cig/day (hours)	2.4 ± 1.1	2.6 ± 1.4	0.37
Nicotine yield (mg/cig)	0.75 ± 0.32	0.74 ± 0.17	0.09
Condensate yield (mg/cig)	11.4 ± 4.0	10.1 ± 3.1	0.80
CO yield (mg/cig)	11.2 ± 2.7	11.0 ± 4.9	0.09
pH	6.9 ± 0.24	7.0 ± 0.69	0.14

TABLE 6

CORRELATIONS OF ANAMNESTIC DATA AND SMOKE YIELD MEASURES WITH PUFFING BEHAVIOR FOR THE FIRST CIGARETTE SMOKED IN EACH OF THE FIVE SESSIONS

Variables		Session 1	Session 2	Session 3	Session 4	Session 5
Years smoking	—N cig before test	0.43*	0.37*	0.35*	0.29*	0.28*
Latency to 1st cig/day	—N cig before test	−0.85†	−0.77†	−0.71†	−0.75†	−0.71†
Number of cig/day	—N cig before test	0.78†	0.84†	0.72†	0.72†	0.68†
Number of cig/day	—CO baseline	0.68†	0.48†	0.57†	0.54†	0.62†
Nicotine yield/cig	—Total puff vol/cig	−0.24	−0.30	−0.36*	−0.40*	−0.31
Condensate yield/cig	—Total puff vol/cig	−0.36*	−0.39*	−0.46†	−0.51†	−0.47†
Condensate yield/cig	—Puff volume	−0.45†	−0.32	−0.44†	−0.50†	−0.38*
Condensate yield/cig	—Number of puffs	−0.17	−0.45†	−0.37*	−0.40*	−0.34*
CO yield/cig	—Puff volume	−0.51†	−0.33	−0.57†	−0.38*	−0.28
pH	—CO baseline	0.48†	0.37*	0.41*	0.18	0.20
pH	—N cig before test	0.38*	0.37*	0.47†	0.37*	0.22
CO baseline	—N cig before test	0.73†	0.47†	0.46†	0.43†	0.69†
CO baseline	—Δ tidal CO	0.17	0.57†	0.38†	0.38†	0.59†
Estimated CO inhalation	—Δ tidal CO	0.33	0.39*	0.39*	0.56†	0.10

$^*p<0.05$, $^{\dagger}p<0.01$.

Andersson [22] found synergistic interactions between alcohol and cigarettes for the variables heart rate (increase) and hand steadiness (impairment), but antagonistic interactions for skin temperature and reaction time. Deleterious effects of alcohol on performance in simple and in choice reaction time tasks tended to be counteracted by cigarette smoking. No clear-cut effects on EEG variables (dominant alpha frequency and CNV) were found by Knott and Venables [14,15], who discussed their results in relation to antagonistic and synergistic interactions between alcohol and tobacco smoking.

Only inconsistent effects of caffeine on cigarette smoking behavior were found in the present study, showing that caffeine tended to prolong puff intervals with an accompanying decrease in subjective smoking need in Group I but not in Group II. These findings, together with the relations between average daily coffee consumption and total puff volume per cigarette found in the same group for the first cigarettes smoked in a session (without treatments), suggest different relations between caffeine and cigarette smoke intake in the two groups. Other sample characteristics which were not used in the present experiment, including the evaluation of caffeine side effects, might therefore be important for caffeine effects on cigarette smoking and might be a possible reason for the rather conflicting findings in the earlier literature. Using anamnestic data and questionnaire ratings Kozlowski [17] and Bättig *et al.* [5], but not Marshall *et al.* [19,20], found positive correlations between average daily cigarette and coffee consumption. Under laboratory conditions coffee drinking failed to affect the number of cigarettes smoked during a one-hour period in experiments carried out by Ossip *et al.* [24] and by Ossip and Epstein [23], but increased the number of cigarettes in experiments by Marshall *et al.* [19,20] carried out in the same laboratory. Whereas Marshall *et al.* [19,20] suggested that the increased rate of cigarette smoking with coffee was independent of caffeine content, Kozlowski [17] found in other laboratory experiments that caffeine depressed nicotine mouth intake as evaluated by butt analysis. In an experiment by Chait and Griffiths [7] acute doses of caffeine produced dose dependent increases in subjective drug-effect scores and hand tremor and depressed cigarette smoking behavior in some, but not all subjects, while d-amphetamine increased cigarette smoking in all the subjects.

Findings on the effect of coffee drinking on smoking remain, therefore, highly controversial, suggesting that any possible effect of coffee drinking on cigarette smoking might be a rather subtle one and different for the individual subjects, leaving open the possibility of a merely habitual connection between the two habits [20]. An additional factor may be seen in the increased clearance of caffeine by smokers as compared with nonsmokers [13,25], which, however, is dependent on the validity of the assumption that coffee is consumed in order to obtain caffeine.

In addition to the effects of ethanol and caffeine, the present results might also merit some additional interest in view of the reliability of both the puffing and inhalation measurements. Under the conditions of this experiment, puffing behavior remained not only remarkably constant within one session, as already seen earlier [5], but also over five sessions at weekly intervals. Consistent correlations between anamnestic data and puffing measurements further supported the validity of this technique. The great interindividual variability of the puffing behavior could, in part, be interpreted in terms of compensation phenomena for different machine standard smoke yields of the cigarettes, as these were negatively correlated with puff volume but not correlated with Δ tidal CO. These results are in line with other investigations showing that nicotine and COHb levels were similar for smokers of different types of cigarettes [27] or that Δ tidal CO was not correlated with the smoke deliveries of the cigarettes [5].

Carbon monoxide uptake during smoking depends upon the amount of CO which reaches the respiratory part of the pulmonary system [9]. The depth of inhalation was estimated by Rawbone *et al.* [26] by recording the chest pneumogram trace during cigarette smoking. He found a good relationship between his smoke exposure index (estimated from the pneumogram, reflecting the depth of inhalation of smoke and the time during which this smoke remains in the lungs) and the increment of alveolar CO concentration. The positive correlation obtained in the present experiment between Δ tidal CO and estimated respiratory CO inhalation suggests that further approaches of this kind might be a promising way for the noninvasive estimation of smoke intake.

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