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Postlunch Smoking for Pleasure Seeking or Arousal Maintenance?

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HASENFRATZ, M., D. PFIFFNER, K. PELLAUD AND K. BÄTTIG. *Postlunch smoking for pleasure seeking or arousal maintenance?* PHARMACOL BIOCHEM BEHAV **34**(3) 631–639, 1989.—Interactions between smoking and eating might be expected, since the craving to smoke increases after a meal, since smokers tend to have a lower body weight and since they have also been suggested to differ from nonsmokers with respect to metabolism. Further, both eating and smoking have been reported to affect mental performance. In the first experiment the influences of a heavy meal, a light meal and no meal on smoking behavior and subjective ratings were compared in 15 subjects. Whereas puffing behavior was not affected by the meal conditions, craving and smoking enjoyment increased after the meals. In the second experiment the effects of smoking on postlunch performance and concomitant central and peripheral physiology were investigated. Postlunch smoking (compared to postlunch no smoking) distinctly showed the usual increases in heart rate, peripheral vasoconstriction and electrocortical arousal, but it failed to affect rapid information processing performance and its concomitant event-related EEG potentials as well as several indices of metabolic activity. It appears, thus, that under the conditions of the present experiments, pleasure seeking may be a more important factor in postmeal smoking than the effects on performance, EEG or metabolism.

Eating Smoking Rapid information processing EEG Event-related potentials Cardiovascular Puffing behavior Insulin Glucose Triglycerides

THE number of cigarettes smoked per hour appears to increase considerably after meals (8), and cluster analysis of questionnaire data on smoking behavior revealed an enhanced craving to smoke during relaxation periods, particularly after a meal (15,24). Thus, a relation between smoking and eating might be suggested, due either to positively perceived effects on metabolism after a meal or to a nonspecific increase in smoking pleasure. Jarvik (15) proposed several hypotheses concerning the question: "Does smoking decrease eating and eating increase smoking?" Eating might increase the craving to smoke because of a reduction of nicotine levels in the blood, because of the counteracting effect of smoking on the sedation produced by a meal or because of the potentiation of the rewarding effect of the meal by the release of endogenous opioids, catecholamines or other substances involved in the mediation of reward.

Within the profile of the actions of smoking, the stimulation induced by nicotine on central arousal as assessed by self-ratings [e.g., (25)] and EEG frequency analysis (9, 13, 18), together with heart rate acceleration and acrodermal vasoconstriction, are perhaps the best documented ones. Positive effects on standardized performance measures, however, were less considerable, mostly modest in magnitude (13, 18, 31, 32), and in some instances, even missing (19).

The widely presumed postprandial depressive effects of meals on arousal, attention and performance as a second aspect have been investigated thus far less systematically and the results are in part less than unequivocal. Smith and Miles (26) observed that subjects who ate lunch prior to testing detected fewer targets in a successive comparison task,whereas no impairment was seen with a proportion perception task. These authors suggested that performance changes observed in the early afternoon may be attributed to at least two factors, one of them meal dependent and the other one circadian. The latter appears to be endogenous, as it is operative even when no lunch is consumed. On the other hand, Christie and co-workers (3) found an increase in heart rate after eating, but no postlunch performance deficit in a letter cancellation task. In a review, Craig (4) concluded that—beside the time of day, size and composition of the meal—age, sex, personality and individual eating habits may all be influential factors.

The third aspect of possible effects of smoking on metabolism is based mainly on the nearly unequivocal observation that smokers tend to be lighter than nonsmokers and to gain weight upon smoking abstinence (6, 14, 28, 29). As an underlying factor for these differences, specific effects of smoking on metabolism can be taken into consideration on the basis of different reports. Stamford and co-workers (28) were unable to explain the lower body weights of smokers in terms of a lower caloric food intake, and Grunberg (11) more specifically was unable to relate the decrease of body weight of smokers to their decreased intake of sweet tasting high caloric foods. On the other hand, with the method of direct calorimetry, Hofstetter and co-workers (14) reported increased energy expenditures in smokers, and Green and Tapp (10) reported shorter feeding cycles in smokers than in nonsmokers.

In order to possibly throw some more light on this complex of questions, two experiments were carried out. The first one was done to compare postlunch self-ratings and cigarette puffing behavior between different meal sizes and the second one to

compare postlunch performance, EEG, physiological and metabolic indices for postlunch smoking and postlunch no smoking.

EXPERIMENT I

The first experiment was carried out to determine the influence of different caloric contents of meals on different psychological variables (self-ratings) and puffing behavior as well as on heart rate and CO uptake. Therefore, the meal factor was manipulated and the smoking data were compared for differences between a premeal and a postmeal cigarette.

METHOD

Subjects

The subjects were fifteen smokers (five women and ten men), all students in good health with a mean age of 24.6 years (range 19–33) and an average daily cigarette consumption of 21 cigarettes per day (range 8–33). All reported to be regular smokers since they were 17.2 years old (range 14–20). The average interval reported between waking up and the first cigarette was 72 minutes (range 0–180). On the testing day they ate only a light breakfast, and after 10:00 a.m. no food was allowed until the session began, but no restriction of smoking was imposed. The subjects were paid sFr. 60.- at the end of the last session.

Procedure

All subjects underwent three testing sessions with an identical procedure, but with three different meal conditions presented in a balanced sequence: no meal (a glass of mineral water), light meal (a small green salad with a piece of bread, an apple and a glass of water) and heavy meal (green salad, a portion of French fries with half a fried chicken, an apple and a glass of water). After the arrival at the laboratory at 11:30 a.m. the session began with the fixation of the ECG electrodes, the answering of the different questionnaires and the collection of respiratory air for CO concentration analyses. Then a first cigarette was smoked through a puffing flowmeter mouthpiece, followed by further questionnaires and air sampling. At noon, a heavy, a light or no meal was served according to the three conditions. Afterwards a second cigarette was smoked as before the meal with questionnaires and respiratory air sampling before and after smoking. During the smoking periods, emotionally neutral video films (wildlife) were shown to the subjects in order to create a habitual situation and the subjects were instructed to pace their puffs and to dose the puff durations and depth of inhalation as naturally as possible.

Heart Rate

For the heart rate measurement a miniature infrared transducer [STRT-850, (27)] was used to obtain continuous analogue signals of the vascular pulse wave at the earlobe. The signals were analyzed offline using a MINC 11/23 computer to the following two parameters for each cigarette: mean heart rate during the minute preceding smoking and the heart rate acceleration after smoking (difference between the mean heart rate during the minute immediately before lighting the cigarette and the minute immediately after the last puff).

CO

The CO concentration was measured in the mixed expiratory air rather than in the endexpiratory air, as in many other studies. Toward this goal a Beckman Instruments CO analyzer (model 866) was used. [For more details see (21,22).] The CO uptake was computed as the difference between the two measurements, before starting to smoke and 3 min after the last puff of each cigarette.

Puffing Behavior

Number of puffs per cigarette, means of puff volume, puff duration, puff interval, latency to peak pressure, peak pressure and peak flow as well as the total puff volume of each cigarette were computed for each smoked cigarette using a MINC 11/23 computer from raw data recorded by a puff-flowmeter [cigarette holder connected to a precision pressure and flow transducer system (5, 21, 22)].

Questionnaires and Subjective Ratings

For the assessment of anamnestic data, the subjects had to fill out a life-style questionnaire in the no-meal session (instead of eating). Analogue scale ratings (100-mm scales) were used to assess craving before smoking and drowsiness, subjective arousal, smoking enjoyment, perception of strength of the cigarette, tobacco taste quality and dizziness after smoking. [Craving: "How much would you like to smoke now?" (not at all–very much); drowsiness: "Do you feel drowsy now?" (not at all–very much); subjective arousal: "Do you feel aroused now?" (not at all–very much); enjoyment: "How much did you enjoy this cigarette?" (not at all–very much); perception of strength of the cigarette: "How strong was this cigarette?" (weak–strong); tobacco taste quality: "How was the tobacco taste?" (bad–good); dizziness: "Do you feel dizzy now?" (not at all–very much).]

Statistics

All data were transferred to a large CDC computer and statistically analyzed using the software packages SPSS (Statistical Package for the Social Sciences) and BMDP (Biomedical Computer Programs). All subjective rating and puffing behavior variables as well as heart rate acceleration and CO uptake were analyzed using repeated-measures analyses of variance (3×2 ANOVA, BMDP2V) with the factors meal (M: three conditions) and cigarette (C: pre- vs. postlunch cigarette). Since heart rate acceleration and CO uptake represented differences between pre- and postsmoking levels, they were also analyzed using a 3×2 ANCOVA (BMDP2V) with the same factors as described above, but with the corresponding levels before smoking as covariables.

RESULTS

Heart Rate and CO

The heart rate acceleration (Fig. 1a) after smoking the first cigarette (before the meal) reached about 10 beats per minute and was nearly identical for all three meal conditions, whereas it differed after the postmeal cigarette among the conditions [Interaction $M \times C$: $F(2,27) = 7.78$, $p<0.01$]. As indicated in the figure, the pulse acceleration following smoking after the heavy meal was nearly as high as after premeal smoking. In comparison, smoking after the light meal and after no meal produced smaller heart rate increases than premeal smoking. Significance for the covariance of these heart rate accelerations with the presmoking heart rate levels was reached for the factor meal, $F(2,27) = 8.47$, $p<0.01$, the factor cigarette, $F(1,13) = 13.97$, $p<0.01$, and for their interaction [$M \times C$: $F(2,27) = 37.67$, $p<0.0001$]. The corresponding regression coefficients of these postsmoking accelerations with the presmoking levels were negative in each case.

CO uptake (Fig. 1a) differed among the three meal conditions

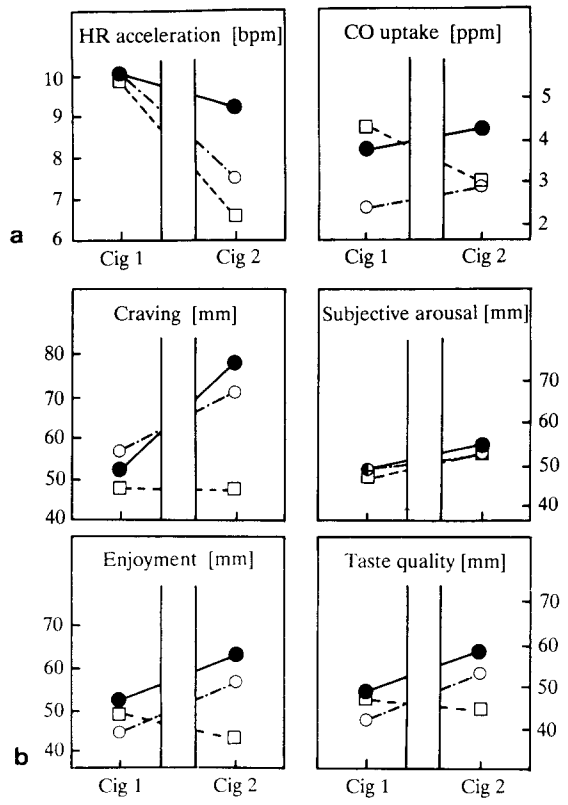

FIG. 1. (a) Heart rate acceleration (bpm), respiratory CO uptake and (b) subjective ratings (100-mm analogue scales) for both cigarettes (Cig. 1 = premeal, Cig. 2 = postmeal cigarette). ●: Heavy-meal condition; ○: light-meal condition; □: no-meal condition.

[M: $F(2,27)=6.29$, $p<0.01$], but there were no other significant effects, particularly no interaction effects. This effect seems, therefore, not to be meal dependent.

Self-Ratings

The craving to smoke (assessed before smoking; Fig. 1b) increased from the premeal to the postmeal cigarette [C: $F(1,14)=11.56$, $p<0.01$] with the two meal conditions but not with the no-meal condition [Interaction M × C: $F(2,28)=4.54$, $p<0.05$]. Subjective arousal after smoking showed a significant increase from the first to the second cigarette [C: $F(1,14)=7.4$, $p<0.05$], but no effects of meal. Smoking enjoyment and tobacco taste quality tended to develop similarly to craving, but this trend failed to reach significance [Interaction M × C: $F(2,28)=3.01$, $p<0.1$, for smoking enjoyment and, $F(2,28)=2.78$, $p<0.1$, for taste]. None of the other subjective ratings showed any effects of the meal conditions.

Puffing Behavior

There was no effect of the meal conditions or of the cigarette factor on any of the puffing behavior variables.

DISCUSSION

The different meal conditions in this experiment affected subjective ratings in that craving increased significantly from the premeal to the postmeal cigarette in the heavy- and light-meal conditions, but not in the no-meal condition. Further, there was a tendency in the same direction for smoking satisfaction and tobacco taste quality. Also, heart rate acceleration after postmeal smoking was higher in the heavy-meal condition than in the light-meal or in the no-meal condition, suggesting deeper smoke inhalation. However, CO uptake and puffing behavior were not affected by the different kinds of meals.

EXPERIMENT II

The craving to smoke, smoking enjoyment and subjective tobacco taste quality all increased with postlunch smoking. It also appeared that a meal might intensify the effects of smoking, possibly due to a transient loss of nicotine tolerance as suggested by the dependence of the magnitude of the smoking-induced heart rate acceleration on the different meal conditions. Among other possibilities, such a transient loss of nicotine tolerance might be the consequence of an increased metabolism after a (heavy) meal. On the other hand, puffing behavior remained unaffected by all the meal conditions, consistent with findings of Nil and co-workers (21), who found puffing behavior to remain rather stable under different situational conditions. In order to obtain more relevant information with respect to the factors underlying the postlunch increases in craving and enjoyment, a second experiment was carried out. In this second experiment postlunch assessments of mental performance, concomitant EEG correlates, cardiovascular and metabolic functions were compared between test lunches with and without the subsequent smoking of a cigarette.

METHOD

Subjects

The subjects were 22 male volunteers with a mean age of 32.0 years (range 27–39) recruited through newspaper advertisements. The only selection criteria were: a cigarette consumption of at least 20 cigarettes per day, a healthy condition and the absence of obesity (mean weight 73.0 kg, range 58–85, with a mean height of 179.0 cm, range 170–190). All were steady smokers according to self-reported consumption, but no assessments were made for symptoms of different types of dependence. No restriction was imposed on smoking, eating, or drinking for the experimental day. Each subject was paid sFr. 180.- at the end of the last session. None of these subjects had participated in the first experiment.

Procedure

All subjects underwent an individual training session starting at 8:45 a.m. and two subsequent lunch sessions starting at 11:45 a.m. on different days at an interval of about one week (see Fig. 2). All sessions started with the fixation of the different sensors for continuous recording of physiological functions and the implantation of an indwelling plastic tube (Venflon 2 IV cannula, Viggo AB, Helsingborg, Sweden) into the cubital vein of the nondominant forearm for the intermittent collection of blood samples. The training session consisted of two 20-minute rapid information processing trials (RIP) separated by a smoking period, during which the subjects had to smoke one of their habitual cigarettes. Four 5-minute rest phases were included in the testing design, one before and one after each of the two trials. Both lunch sessions consisted of a meal period, a subsequent smoking or no smoking period and a following 20-minute RIP period. Again, four 5-minute rest phases, one before and one after the meal and RIP periods, were included in the design. All subjects smoked in only one of the two lunch sessions,and they were instructed to do so as naturally as possible. In the other lunch session, they had a

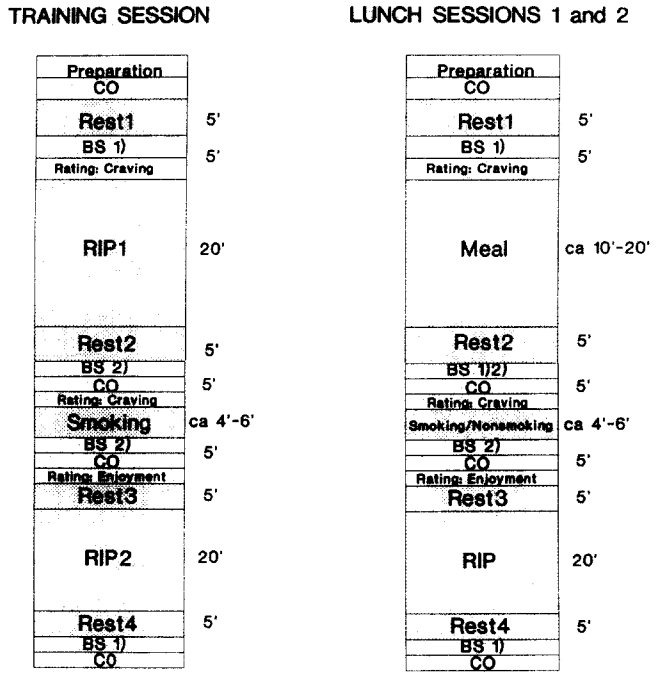

FIG. 2. Schematic diagram of the experimental protocol. CO = Carbon monoxide measurement; BS 1) = blood sample for the analysis of the biochemical parameters; BS 2) = blood sample for nicotine analysis; RIP = rapid information processing task.

relaxation period without smoking. The order of these two treatments was balanced.

Mental Performance

The rapid information processing (RIP) task used in this laboratory (1, 13, 20) required the subjects to watch single digits (1–8) presented in a pseudorandom order for 80 msec on a TV monitor, and to press a button whenever the last three digits were either odd or even. The presentation rate increased after each correct response and decreased after each error by a change in the interstimulus interval in steps of 33 msec. The initial presentation rate was 90 digits per minute, and the program allowed a minimum of 30 digits per minute and a maximum of 300 digits per minute. Digitally coded signals were recorded on a tape using the pulse code modulation (PCM) method (Johne & Reilhofer Ltd.) for the identification of the stimuli and the button presses to calculate the reaction times.

Meal

The meals presented in the lunch sessions consisted of a standardized portion of Italian-type tortellini with cheese sauce garnished with red sweet pepper and parsley, and a piece of chocolate ice cream cake as dessert. A glass of moderately carbonated water was served as beverage. The whole meal contained 3428 kJ (820 kcal).

Physiological Recording

All physiological data (except ECG) were digitized at a rate of 125 cps and recorded on PCM tape continuously throughout the experimental sessions including the baseline periods.

EEG and EOG. EEG activity was recorded with goldcup electrodes from Cz (international 10-20 system). Combined ear references with resistances between them were used and a mid-forehead electrode served as ground. All electrode impedances were kept below 5 Kohms. The signal was amplified with bandpasses from 0.2 to 25 Hz (−3 dB). 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.

ECG. The electrocardiogram was recorded with Beckman Ag/AgCl electrodes fixed at the positions aVR, V5-V6. The R-wave peaks of the ECG were detected using an ECG cardiometer (Cardiotronics AG, Stockholm) and recorded with a separate real-time unit (1-msec resolution) of the PCM system.

Finger and ear pulse amplitude, ear pulse arrival time. Miniature photosensors [STRT-850; (27)] were placed at the palmar surface of the distal phalanx of the middle finger of the nondominant hand and at the left earlobe. The ear pulse arrival time was computed as the time between the R-wave of the ECG and the point at which the ear pulse amplitude began to increase.

EMG. The electromyogram of the musculus frontalis was recorded (hardware RMS transferred) with three Beckman Ag/AgCl electrodes.

Respiration. Respiration frequency was registered with the strain gauge method (a conducting tape, No. 13, 3M Scotch, sewed on an elastic band). Two elastic belts were installed to assess thoracic and abdominal breathing.

Body movement. Strength (size of amplitudes) and level (integrated measure) of 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).

CO. For measuring the CO concentration in the expiratory air, a CO analyzer (Beckman Instruments, model 866) was used. [For more details see (21,22).] The CO uptake was calculated as the difference between the two measurements, before smoking and 3 min after the last puff.

Plasma nicotine and biochemical parameters. Plasma concentrations of nicotine were analyzed at the Institut für Klinische Chemie, Universitätsspital Zürich, using the capillary gas chromatographic mass spectroscopy method and plasma concentrations of insulin, glucose, triglycerides, cholesterol, high and low density lipoproteins and glutamate-pyruvate transaminase (GPT) were determined by routine clinical methods at the Isler Medizinische Laboratorien AG, Zürich. For analyzing nicotine, 10-ml blood samples were taken before smoking and with the last puff of the cigarette or after the corresponding relaxation period in the lunch-no smoking session. For analyzing metabolic parameters, 10-ml blood samples were taken before the meal, before the smoking period and after the final rest period. In the training session only two blood samples were taken, namely at the beginning and at the end of the session. The blood was collected in glass tubes and was stored at room temperature for one hour following the session. Then the samples were centrifuged and the serum for nicotine analyses was frozen at −70°C and for insulin analyses at −20°C. The serum for the other analyses was sent immediately to the external laboratory (fluoridated tubes for glucose analysis).

Self-Ratings

In the training session, the craving to smoke was rated before the first RIP trial and before the smoking period (see Fig. 2). Smoking enjoyment was rated immediately after smoking. In the lunch sessions, the first rating of the craving to smoke was

done before the lunch period, the second one before the smoking/no smoking period. Smoking enjoyment was rated again after smoking.

Data Processing

The PCM-stored data were read into a PDP 11/34 laboratory computer. A set of software programs reduced the data to the following main parameters: The mean R-R intervals, EMG levels, the mean finger pulse amplitudes, the mean respiration frequencies as well as body movement levels were computed for each consecutive experimental period.

The stimulus processing rate (number of digits per minute) was averaged for each consecutive 10-minute block. The EEG recordings of the baseline periods were used for power spectral analysis using a Fast Fourier Transformation [International Mathematical & Statistical Libraries (IMSL) subroutines]. The dominant frequencies (frequency of maximal power) and the mean power were computed for the commonly defined frequency bands: alpha, 7–14 Hz, and beta, 14–25 Hz.

The EEG recordings during the RIP trials were used for computing event-related potentials (ERP) to the second and third digits of the correctly detected triads. A detailed description of the ERPs to the different stimuli and conditions was reported earlier (20). The averaging period lasted from 200 msec before to 500 msec after stimulus onset. The EEG epochs with corresponding ISIs shorter than 500 msec or which saturated the a/d converter were rejected. The ERPs included between 30 and 40 averaged EEG epochs within both the first and the second 10-minute phase of each RIP trial.

The ERPs to the second digits are characterized in this task by a continuously increasing initial negativity, which is comparable to the CNV in the S1-S2 paradigms [reflecting the expectancy to the third digit; (20,23)]. This negativity is followed by a late positive wave after the correct answer of the third digit (LP) [indicating the brain processes associated with the response selection; (7,20)]. According to previous studies (13, 18, 20), the CNV-related negativity was assessed using the microvolt × millisecond products between 100 msec before and 100 msec after the third digit and the late positivity using the corresponding product between 300 and 500 msec after the onset of the third stimulus.

Statistics

The reduced data set was transferred to a large CDC computer and statistically analyzed using the software packages SPSS and BMDP.

For the lunch sessions all performance and ERP data collected during the postsmoking RIP period were analyzed using repeated-measures analyses of variance (2 × 2 ANOVA, BMDP2V). The factors were smoking/nonsmoking (Treatment, T) and the comparison of the two 10-minute periods within the RIP trial (Within, W). The 2 × 2 × 2 ANOVA of EEG power, dominant frequencies and all physiological measures of the four rest phases of the lunch sessions included the factors Treatment (T), a comparison of the two pre- versus the two posttreatment rest phases (PrePost, P) and a comparison between each of the two rest phases within the pre- and within the postsmoking period (Within, W). All biochemical data of the two lunch sessions were analyzed using 2 × 3 ANOVAs with the factors Treatment (T) and the number of repeated measurements (W).

For the training session, separate 2 × 2 ANOVAs for performance, EEG, ERP and physiological data were carried out with the factors PrePost (P: comparison of RIP performance and ERP before and after smoking as well as comparison of the two pre- versus the two postsmoking rest phases for EEG and physiological measures) and Within (W: comparison between the two 10-min blocks of RIP performance and ERP within the pre- and within the postsmoking periods as well as comparison between each of the two rest phases within the pre- and within the postsmoking period for the EEG and physiological measures).

Relations between self-ratings, performance, CO and plasma nicotine uptake were analyzed with Pearson correlation analyses. The significance of differences between the cell means of these parameters was tested with Student's *t*-test.

RESULTS

For the following statistical analyses the training session was separately analyzed for all parameters.

Mental Performance and Reaction Time

RIP performance (Fig. 3) increased from the first to the second trial, $F(1,21) = 34.09$, $p<0.001$, of the training session, but there were no differences between the postlunch trials of the smoking and no-smoking lunch sessions. Within-trial decreases in RIP performance were seen in the training session only, $F(1,21) = 7.77$, $p<0.05$, and this to a greater extent within the second than the first trial [Interaction P × W: $F(1,21) = 4.99$, $p<0.05$].

The reaction time (Fig. 3) was also not affected by smoking. This parameter showed a significant increase across the two blocks of the trials in the two lunch sessions, $F(1,21) = 4.49$, $p<0.05$, but not in the training session.

ERP and EEG Frequency and Power Analysis

The analyses of ERP components (Fig. 3) revealed a decrease of the CNV-related negativity (a correlate of attention) to the second digits of the correctly answered digit triads in the training session, $F(1,21) = 30.07$, $p<0.001$, after smoking, but this effect was smaller and failed significance in the lunch session with smoking as compared to the no-smoking lunch session. The LPs to the correctly answered third member of the digit triads (measures of cognitive recognition) showed a strong within-trial increase in the training session, $F(1,21) = 22.17$, $p<0.001$, but not in the two-lunch sessions. No smoking-related LP differences were obtained between the two postlunch trials.

In the lunch sessions, EEG frequency and power analyses (measures of cortical arousal) during the four baseline phases (Fig. 4) revealed a weak, but not significant increase of the dominant alpha frequency after smoking [Interaction T × P: $F(1,21) = 3.79$, $p = 0.06$]. Beta power as well as dominant beta frequency, which increased from pre- to postsmoking after lunch, decreased within the subsequent RIP period after smoking, whereas both increased in the RIP period following no smoking [Interaction T × P × W: $F(1,21) = 19.24$ for dominant beta frequency and $F(1,21) = 20.82$ for beta power, both $p<0.001$]. In the training session, alpha, $F(1,21) = 4.63$, $p<0.05$, and beta power, $F(1,21) = 10.89$, $p<0.01$, as well as dominant alpha frequency, $F(1,21) = 4.49$, $p<0.05$, were increased after smoking. The increase of dominant beta frequency shown in the figure failed, however, to reach significance.

Physiology

Heart rate showed a smoking-induced increase in the lunch sessions [Interaction T × P: $F(1,21) = 21.68$, $p<0.001$] as well as

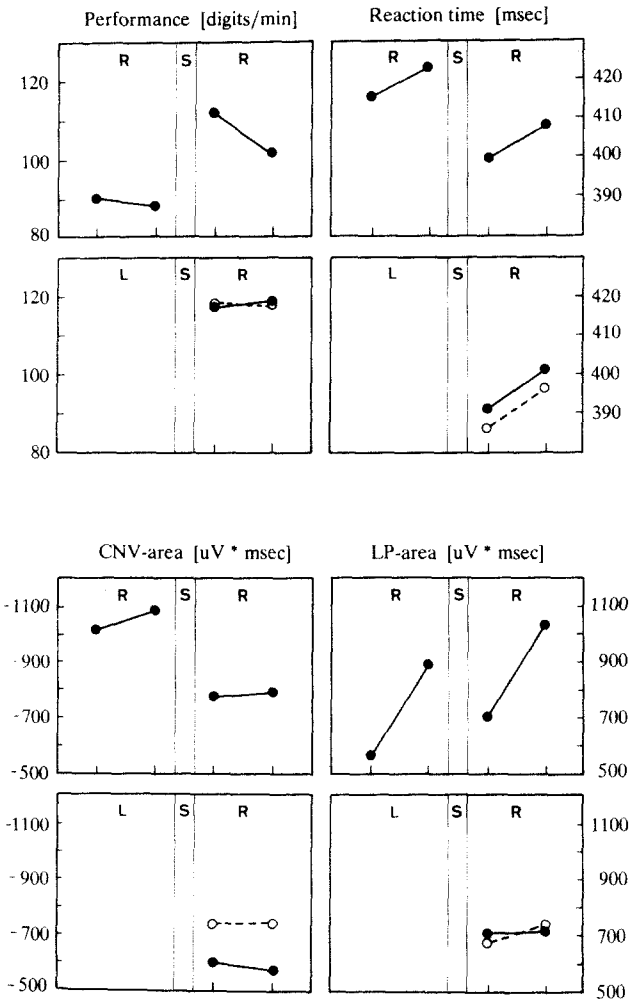

FIG. 3. RIP performance, reaction time and concomitant ERP measures for the training session (upper panels) and lunch sessions (lower panels). Each point represents the mean value for a 10-minute block of a RIP trial. R = RIP phase; S = smoking phase; L = lunch phase. ●: Smoking in the smoking phase; ○: relaxation in the smoking phase.

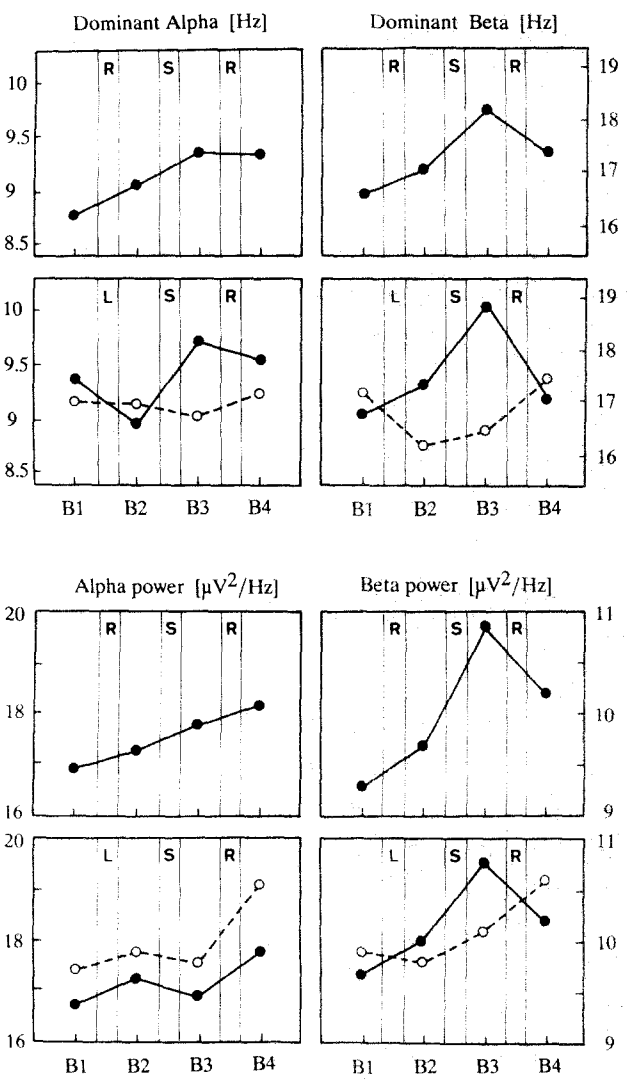

FIG. 4. EEG measures for the four rest periods (B1–B4) of the training session (upper panels) and lunch sessions (lower panels). Each point represents the mean value for the 3-minute EEG recording time in the middle of each 5-minute rest period. R = RIP phase; S = smoking phase; L = lunch phase. ●: Smoking in the smoking phase; ○: relaxation in the smoking phase.

in the training session [$F(1,21) = 5.24$, $p<0.05$; Fig. 5]. As shown in the figure, eating also increased heart rate in both lunch sessions, whereas no increase was observed in the training session (no lunch) from the first to the second rest phase.

The finger pulse amplitudes decreased more after smoking than after no smoking in the lunch sessions, $F(1,21) = 6.12$, $p<0.05$, and they also decreased after smoking in the training session, $F(1,21) = 26.61$, $p<0.001$. Ear pulse arrival time increased across the training session, $F(1,21) = 16.46$, $p<0.001$, whereas it decreased across the lunch sessions, $F(1,21) = 24.50$, $p<0.001$. No effect of postlunch smoking was found.

Respiration frequency showed a postlunch increase from the first to the second rest phase (effect of lunch) and decreased from the third to the fourth rest phase [Interaction P × W: $F(1,21) = 42.54$, $p<0.001$], but did not change across the training session.

Body movement increased across all sessions, independent of the treatment [lunch sessions: P: $F(1,21) = 36.09$, $p<0.001$; training session: $F(1,21) = 26.35$, $p<0.001$]. Frontal EMG remained unaffected by either smoking or eating.

Plasma Nicotine, Breath CO and Self-Ratings

Plasma nicotine increased after smoking (training session: $t = 5.68$, $p<0.001$; lunch smoking session: $t = 6.55$, $p<0.001$; Fig. 6). Breath CO also increased after smoking, but this reached significance in the training session only ($t = 2.05$, $p<0.05$). No correlations were found between nicotine uptake and CO uptake.

Need to smoke increased more from the first rating before eating to the second rating after eating (i.e., before the smoking period) in both lunch sessions (lunch-smoking: $t = 6.94$, $p<0.001$; lunch-no smoking: $t = 5.87$, $p<0.001$; Fig. 7) than in the training

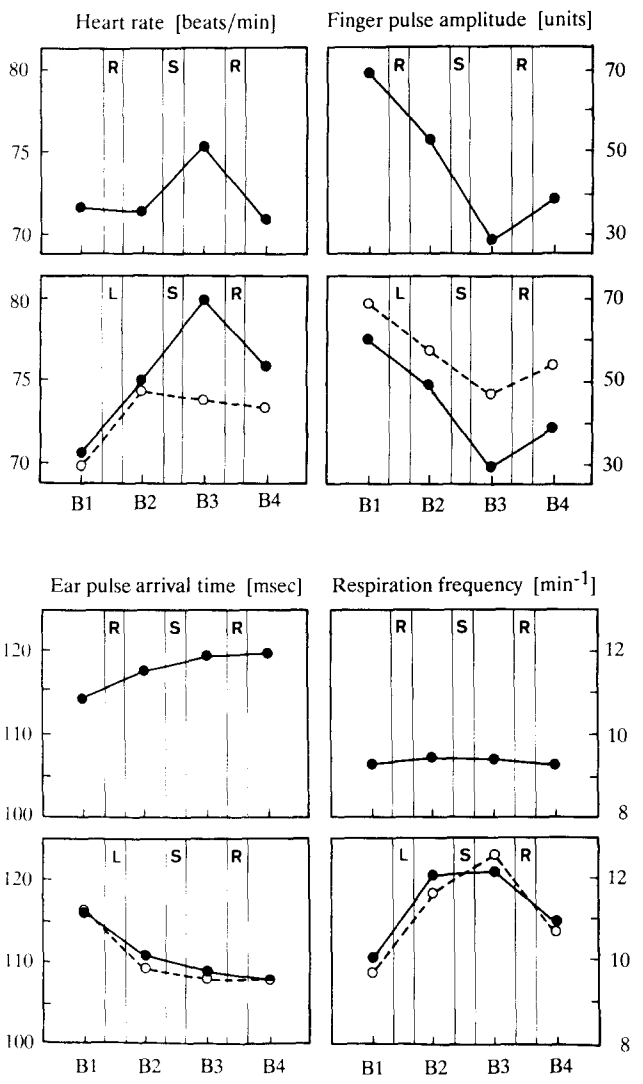

FIG. 5. Physiological measures for the four rest periods (B1–B4) of the training session (upper panels) and lunch sessions (lower panels). Each point represents the mean value for the 5-minute rest periods. R = RIP phase; S = smoking phase; L = lunch phase. ●: Smoking in the smoking phase; ○: relaxation in the smoking phase.

session, where need to smoke was rated before and after the first RIP trial ($t = 2.22$, $p < 0.05$).

Metabolic Effects

Insulin, glucose and triglycerides increased significantly as a consequence of the meal [$F(2,38) = 29.98$ for insulin, $F(2,42) = 20.50$ and 82.90 for glucose and triglycerides respectively, all p's < 0.001], but only glucose and insulin were significantly dependent on smoking [Interaction T × W: $F(2,42) = 4.37$ for glucose and $F(2,38) = 4.08$ for insulin, both $p < 0.05$], with a slightly greater postmeal decrease (after meal versus after RIP) in the smoking than in the no smoking session (see Table 1). High- and low-density lipoproteins as well as cholesterol and GPT failed to be significantly affected by either manipulation.

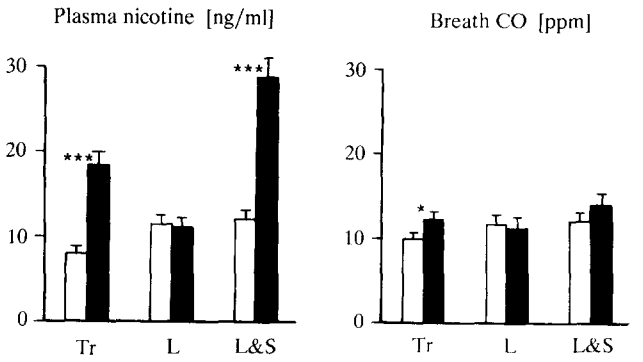

FIG. 6. Nicotine and CO uptake, measured before and after the smoking phase, in means ± S.E. Tr = Training session; L = lunch—no smoking session; L&S = lunch and smoking session. Open bars = presmoking values; filled bars = postsmoking values. (Student's t-test: $*p < 0.05$; $***p < 0.001$.)

GENERAL DISCUSSION

As the first experiment revealed postlunch increases in the craving to smoke and enjoyment of smoking without affecting CO uptake and puffing behavior, the second experiment concentrated on the comparison of a series of performance, physiological and metabolic postlunch measures between a session with and a session without postlunch smoking. Both were carried out after a preceding training session without lunch. The data of this training session, which involved two RIP periods separated by a smoking interval, were analyzed independently of the lunch session data in order to avoid biases due to order effects (e.g., learning, habituation) or to the different time of day.

A first aspect to be considered is the interacting effects of eating and smoking on measures of attention, arousal and performance. The frequency analyses of the resting EEGs revealed effects of smoking similar to those of earlier studies (9, 16, 18). Dominant alpha and beta frequency as well as beta power increased after smoking in the training session, as well as after postlunch smoking, whereas eating failed to show an effect on these measures. On the other hand, smoking induced improve-

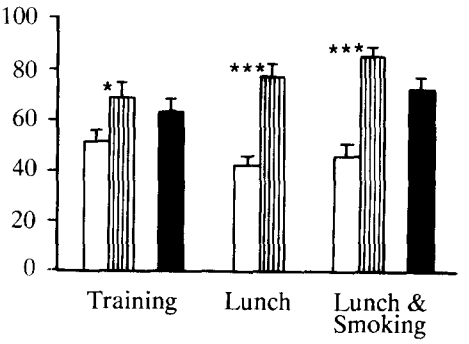

FIG. 7. Craving and enjoyment rated on a 100-mm analogue self-rating scale. Open bars = first rating of craving to smoke (before eating in the two lunch sessions and before the presmoking RIP in the training session). Hatched bars = second rating of craving to smoke (immediately after eating in the two lunch sessions and after the presmoking RIP in the training session). Filled bars = smoking enjoyment immediately after smoking (Student's t-test: $*p < 0.05$; $***p < 0.001$.)

TABLE 1

MEANS ± STANDARD DEVIATIONS OF THE PLASMA LEVELS OF GLUCOSE AND INSULIN

		Glucose (mmol/l)	Insulin (IU/ml)
Training	before RIP 1	5.35 ± 1.02	18.93 ± 21.32
	after RIP 2	5.08 ± 0.72	12.00 ± 11.04
Lunch/	before meal	4.83 ± 1.30	13.31 ± 19.10
Smoking	before smoking	6.02 ± 0.93	41.92 ± 25.22
	after RIP	5.55 ± 1.31	29.53 ± 20.89
Lunch/	before meal	4.68 ± 0.54	7.26 ± 5.47
Nosmoking	before nosmoking	6.38 ± 0.94	37.85 ± 18.72
	after RIP	6.11 ± 1.40	32.83 ± 18.63

ments of RIP performance were, in contrast to other experiments (13, 18, 20), not seen for postlunch smoking in this experiment, whereas the increase in performance from the first to the second trial of the training session could be attributed to the learning process and/or to the effects of smoking between the two trials. Several reasons could account for the fact that postlunch performance with and without smoking did not differ: one is the possibility of a nonspecific masking effect of eating, another is the fact that the subjects were not smoke deprived before the lunch sessions. The same problem arises for the reaction time and the ERP measures of the EEG, which closely paralleled the concomitant performance data across the different sessions. Only the CNV-related negativity of the ERPs tended to be smaller after postlunch smoking, a tendency which is in agreement with significant decreases seen in earlier studies both after smoking (13,18) and after a 4-mg nicotine chewing gum (19), and which has been proposed to represent an attribute of subjective tranquilisation when an optimal arousal level has been reached (2, 13, 30).

Among the vegetative measures, heart rate was increased by eating and by smoking, in an additive fashion, while respiration frequency was increased by eating only. Acrodermal vasoconstriction, as seen with finger plethysmography, appeared as a consequence of smoking only. Whereas the parallel postlunch increases of heart rate and respiration can be viewed as a consequence of the higher energy turnover and oxygen consumption after a meal, the missing parallelism of the smoking effects on these two measures fails to support the notion that smoking might significantly enhance postlunch metabolic processes. Of particular interest are the results of the biochemical analyses. Insulin, glucose and the triglycerides all increased as expected in a marked fashion from the premeal to the postmeal determination. However, for the third determination, carried out after the subsequent RIP task, both the insulin and glucose levels were slightly, although significantly lower in the smoking than in the nonsmoking session. However, as this difference was very small, its possible significance for the postmeal increases of craving and smoking enjoyment remains to date questionable. Furthermore, the existing reports of nicotine effects on insulin and plasma glucose are less than unequivocal. In the rat, Grunberg and co-workers (12) reported a decrease of insulin, but not of glucose after chronic and continuous application of high doses of nicotine. Earlier studies on smokers, laboratory animals and isolated pancreas slices, as reviewed by Larson and Silvette (17), were equivocal with respect to effects both on insulin and on plasma glucose.

Taken together, it appears, thus, that among all interactive effects of eating and smoking, a pronounced increase in the postlunch craving to smoke and in the enjoyment of smoking stands out against moderate or no effects for all other measures. In the light of these results it appears that the need to smoke after lunch is rooted more in the memory pools of the positively experienced stimulating effects of the habit than in any help nicotine might be in overcoming postprandial dips in arousal or in improving the speed of metabolism.

However, it remains open to what extent this conclusion could be generalized for detailed manipulations of the size and composition of the meals, of the time of day and of the intervals after the meals for taking biochemical probes and measuring physiological and behavioral functions.

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