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Journal

Physiology & Behavior, 29(4)

ISSN

00319384

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Publication Date

1982-10-01

DOI

10.1016/0031-9384(82)90246-3

Data Availability

The data associated with this publication are within the manuscript.

Peer reviewed

Physiology & Behavior **29**: 727-731, 1982

Perception of Nasal Pungency in Smokers and Nonsmokers

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Abstract

Two experiments explored the difference in common chemical sensitivity between smokers and nonsmokers. The first experiment confirmed and extended our previous finding that smokers have a higher threshold than nonsmokers for a short-latency, reflex transitory apnea in response to a nasal irritant (carbon dioxide). The experiment revealed that even short periods of smoking (6-10 min) just before the measurement will further impair the smoker's sensitivity to an irritant. Application of the novel scaling technique magnitude matching, whereby persons judged the perceived magnitude of various levels of an irritant, odorant, and tone in the same session, implied that smokers actually perceive irritation less keenly than nonsmokers. The psychophysical functions for pungency in the two groups suggested that the insensitivity may arise from conductive factors (e.g., thickening of the mucus barrier) rather than from neural factors. The results of the scaling and those for the threshold for transitory apnea showed excellent quantitative agreement and indicated that the threshold for the reflex occurred at a criterion level of perceived pungency.

Keywords: Common chemical sense, Irritation, Odor perception, Reflex apnea, Smoking, Trigeminal nerve

Introduction

In various mammalian species, inhalation of an irritant can cause a reflex depression or inhibition of breathing [2, 3, 5-8, 32]. In human beings, weak irritants may cause no discernible change, but strong irritants may cause a momentary apnea of the sort that may make the person say that the inhaled agent "took my breath away." Most of us have experienced this phenomenon upon accidental inhalation of ammonia or bleach. The agent seems to cause a "catch" in the inhalation even before the agent can reach the lungs. Various experiments have implicated the trigeminal nerve in irritant-induced disruption of breathing [4, 6, 31]. This nerve also mediates the sensations of pungency triggered by irritants [11].

Two previous investigations in this laboratory have explored the threshold for reflex transitory apnea in human beings upon inhalation of an irritant. One compared the threshold for two nostrils versus one [21]. That experiment implied bilateral integration of the effect. That is, the concentration necessary to elicit the reflex bilaterally fell below that necessary to elicit it unilaterally. The phenomenon exhibited excellent quantitative agreement with psychophysical data on perceived pungency. The second study found that smokers were 29% less sensitive than nonsmokers with respect to the concentration of irritant necessary to trigger the reflex [19]. This marked perhaps the clearest indication yet of a chemosensory difference between smokers and nonsmokers for an inhaled agent.

In the present paper, we give further attention to the difference between smokers and nonsmokers. One question of interest was whether sensitivity to the reflex in smokers could be diminished further merely by smoking for just a few minutes prior to the test. Another question was whether smokers actually perceive an irritant as less pungent than nonsmokers. The newly developed method of magnitude matching offered a way to address this question. In brief, the method requires participants to judge stimuli numerically in more than one sense modality on a common scale of perceived magnitude [29]. Careful choice of modalities permits one modality to serve as a kind of standard against which to compare numerical judgments of perceived magnitude on another. If, for instance, we could assume that smokers and nonsmokers have no differences in perception of loudness, then we could use test tones of various levels as the standard for comparison of perceived pungency when both pungency and loudness are judged on a common internal scale of perceived magnitude. Loudness had indeed served as the usual standard in magnitude matching.

We chose to incorporate loudness into the present comparison since it has already served well. Nevertheless, some studies have indicated that smokers may suffer from slight hearing loss presumably because of alterations in the middle and inner ear [15, 25, 28]. This matter is in itself an interesting question to ask by means of the method of magnitude matching. Because the bulk of data

imply virtually no olfactory loss from smoking [20, 26, 27, 30], smell might then serve as a reasonable standard modality. If it is true that smokers possess impaired sensitivity to pungency, slightly impaired loudness perception, yet virtually normal olfaction, then the phenomena should show up in the relative positions of psychophysical functions obtained simultaneously in all three modalities.

Method

Subjects

In the study of reflex apnea to an irritant, 21 smokers (7 males and 14 females; average age, 25.4 years; average daily consumption, 15.3 cigarettes ±7.9 (S.D.); average years smoking, 9.1±6.6 (S.D.) and 20 nonsmokers (6 males and 14 females; average age, 25.1 years) participated. In the psychophysical scaling of irritation, odor, and sound, 17 smokers (4 males and 13 females; average age, 24.1 years; average dally consumption, 14.3 cigarettes ±9.4 (S.D.); average years smoking, 6.3±4.5 (S.D.)) and 16 nonsmokers (5 males and 11 females; average age, 22.2 years) participated. Seven of the subjects employed in the scaling experiment also took part on the reflex study.

Equipment

Reflex. An air dilution olfactometer was employed to deliver concentrations of CO_2 (99.8% purity) to the nostril (right or left). At concentrations above about 10% by volume, CO_2 arouses an irritating pungency, virtually devoid of odor. Breathing grade compressed air was used to dilute the CO_2 to appropriate concentrations. The flowrate delivered by the olfactometer equalled 6 lpm at each concentration of CO_2. The stimulus flowed from 7 mm o.d. teflon tube brought to the nostril by the subject. The participant's contralateral nostril held a fine thermocouple (Cu-Constantan) threaded through the wall of a 2-cm segment of soft hose placed just inside the rim of the nostril. The thermocouple reference lead was placed in an ice-water bath. A single-channel output on a Grass Polygraph (Model 7) recorded temperature changes caused by inhalation and exhalation through the nasal passage. The changes followed a roughly sinusoidal pattern over time, except when the reflex occurred.

Scaling. A two-channel olfactometer was used for the scaling of the pungency of CO_2 and the odor of isoamyl butyrate (Monsanto, Flavor/Essence). The final flowrate in each channel equalled 4 lpm.

A 1,000 Hz tone served as the auditory stimulus for the scaling of loudness. The tone was produced by a Heath oscillator and fed through a Hewlett-Packard attenuator to a pair of calibrated earphones (Grason-Stadler TDH 39 with MX-

41/AR ear cushions).

Procedure

Reflex. Participants were seated and instructed to insert the thermocouple assembly into one nostril and to breathe through the nose. When given the ready signal, the subject inhaled, exhaled, rapidly inserted the end of the stimulus tube into the free nostril, inhaled, removed the stimulus tube, exhaled, inhaled, and exhaled. In this way, it was possible to obtain three breathing cycles for each nostril at each level of CO_2: the first normal, the second one with CO_2, and the third one normal again. The entire procedure was then repeated with the other nostril.

The initial trial of a session entailed presentation of 0% CO_2 first to one nostril, then to the other. The second trial entailed presentation of 20% CO_2 successively to each nostril. Thereafter, increments occurred in steps of ten percentage points (30%, 40%, etc.). Control trials (0%) occurred randomly within the otherwise increasing series. The participants were told that the concentrations of CO_2 would be presented in irregular order. Throughout testing, the duration of the inspiratory phase was standardized to an interval of two beats emitted by a metronome set at 56 beats per minute.

Each session was divided in two parts. The end of the first part occurred when the level of CO_2 was high enough to disrupt the breathing pattern record, i.e., to produce the reflex interruption of inhalation (Fig. 1). Then, the subjects smoked (smokers) or rested (nonsmokers) for 6 to 10 min. After this, the second part began, and the threshold concentration to elicit the reflex was again measured.

Each participant took part in three sessions. Smokers were asked to refrain from smoking at least one hour prior to the session.

Scaling. Subjects sat under a continuously ventilated hood and wore a pair of headphones. The subjects were instructed to estimate numerically {method of magnitude estimation) the perceived intensity of the sensations evoked by three types of stimuli: the odor, the pungent stimulus, and the tone. The instructions advised the subjects to concentrate only on the intensity of the stimuli, judging all stimuli on a common scale of perceived intensity. Participants were told that the range from the lowest to the highest stimuli might be different for the various modalities (smell, tingling, tone) [29]. The subjects could assign to the first stimulus of each session any number deemed appropriate. Thereafter, they assigned numbers proportional to perceived magnitude. For subsequent data analysis, variability that arose from differences in the choice of the initial judgment was eliminated by conventional means [13]. This normalization procedure was performed separately for smokers and nonsmokers.

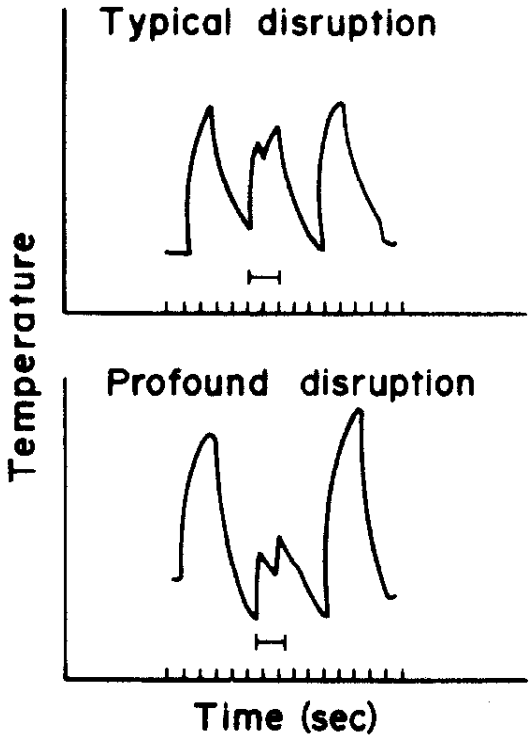

Figure 1. Breathing patterns detected by changes in temperature of a nasal thermocouple before, during, and after presentation of CO_2 at a concentration sufficient to elicit reflex, transitory apnea, The upper tracing shows a typical response whereas the lower tracing shows a particularly pronounced disruption of inhalation.

CO_2 was presented through tubing ending in a green hose, whereas isoamyl butyrate was presented through tubing ending in a white hose. When given a ready signal, the subjects had to insert the appropriate hose into either the right or left nostril (the same throughout the experiment), take a sniff, and then write down their estimation on a sheet. A ready signal also preceded the tones.

Five concentrations of each compound and five levels of sound pressure were presented in random order. Each subject made two estimates per stimulus level, rested for 5 to 10 min and then made two more estimates per stimulus level.

Stimuli

In the scaling experiment, the concentrations of CO_2 in air were (% v/v): 21, 27, 35, 46, and 60; the levels of isoamyl butyrate were (ppm): 3.4, 4.9, 9.6, 24.8, and 71.9; and the levels of the 1000 Hz tone were (dB SPL): 62, 70, 78, 86, and 94. It is important to note that the concentrations of isoamyl butyrate were carefully chosen to arouse only olfactory sensations and to cause no discernible pungency.

Results

The index of interest in the reflex study comprised the lowest concentration (threshold) of CO_2 necessary to elicit the reflex in either nostril. (The choice of

this index eliminated the need to derive an arbitrary combinatorial rule for those few subjects, 17% of smokers and 20% of nonsmokers, who exhibited an internostril imbalance that exceeded one concentration step.) The thresholds for smokers and nonsmokers appear in Table 1. The difference between the groups in the initial measurement closely parallels that obtained before [19], 25% vs 29%. However, the absolute values fall below those obtained previously, presumably because of the higher flowrate used here (6 lpm vs 3.5 lpm). After a 6-10 min respite, nonsmokers exhibited no change in threshold (41.8% vs 41.1%). After a 6-10 min interval of smoking, smokers exhibited a reliable elevation of threshold (52.3% vs 58.6%). In order to examine whether a 6-10 min interval without smoking would alter the threshold of smokers, four smokers participated in such measurements and showed no change.

TABLE 1

THRESHOLD VALUES (% CO_2, V/V IN AIR) FOR THE NASAL REFLEX BEFORE (FIRST MEASUREMENT) AND AFTER (SECOND MEASUREMENT) A PERIOD OF RESTING FOR THE NONSMOKERS AND A PERIOD OF SMOKING FOR THE SMOKERS

	First Measurement (± S.E.)	Second Measurement (± S.E.)	Difference (± S.E.)
Nonsmokers	41.8 ± 2.6	41.1 ± 2.2	−0.7 ± 1.4
Smokers	52.3 ± 2.2*	58.6±3.5†	6.2 ± 2.1‡

*Significantly different from nonsmokers at $p<0.005$, t-test.
†Significantly different from nonsmokers at $p<0.001$, t-test.
‡Significantly different from nonsmokers at $p<0.01$, t-test.

The psychophysical functions for odor, loudness, and pungency appear in Fig. 2. As expected from previous investigations, the functions for the odor of isoamyl butyrate grew more slowly whereas those for the pungency of carbon dioxide grew sharply with concentration [14]. The functions for loudness vs sound pressure grew at an intermediate rate and conformed to power functions with exponents of 0.60 for nonsmokers and 0.56 for smokers.

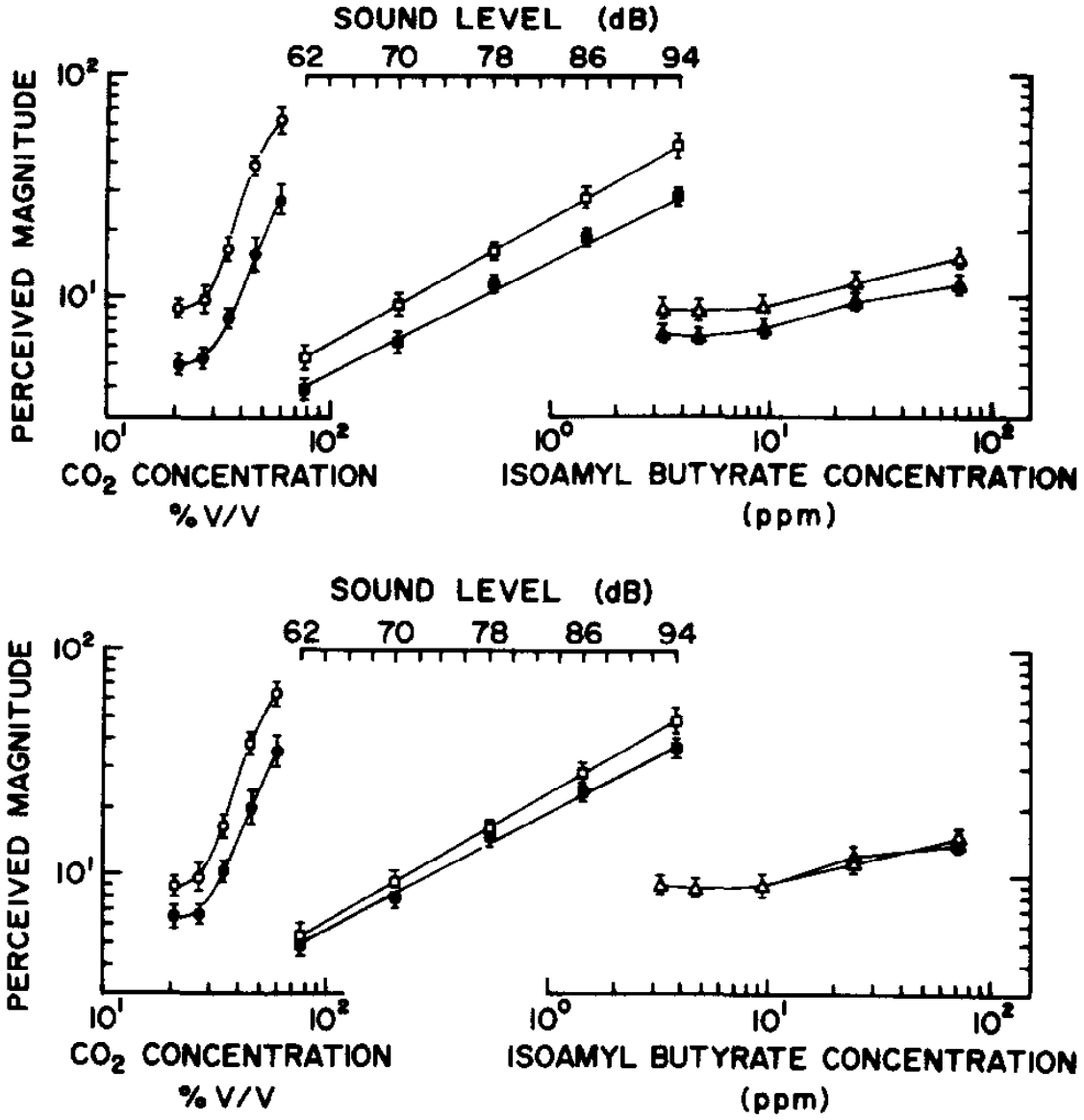

Figure 2. Upper portion shows psychophysical functions for the pungency of carbon dioxide, the loudness of the 1,000 Hz tone, and the odor of isoamyl butyrate in nonsmokers (empty symbols) and smokers (filled symbols). The coordinates are logarithmic. The points represent geometric means and the bars standard errors. The difference in the levels of the functions for the two groups reflects in part differences in the choice of modulus (i.e., in the number assigned to the first stimulus of a session). Lower portion shows the same functions as above for the nonsmokers. The functions for the smokers were transposed upward by a factor that brought the judgments of odor intensity from the smokers into coincidence with those of the nonsmokers.

The upper part of Fig. 2 reveals that smokers gave generally lower magnitude estimations than nonsmokers. This outcome has no importance since participants could choose whatever numbers they wished at the outset of the scaling. In order to allow a meaningful visual and statistical comparison of relative perceived magnitudes in smokers and nonsmokers, the three functions

for the smokers were transposed upward by a factor that brought the odor function for the smokers into coincidence with that for the nonsmokers. This transposition reveals that, relative to their perception of odor magnitude, smokers found the tones slightly less loud and carbon dioxide less pungent. A statistical comparison of the data in the lower part of Fig. 2 indicated, however, that only the difference in pungency achieved significance, $F(1,31)=4.17$, $p<0.05$, for the variable smoker vs nonsmoker.

In a previous experiment [21], it was concluded that the threshold for transitory apnea occurred at a criterion level of perceived magnitude. The present results offered another opportunity to re-examine this hypothesis. If correct, then the threshold concentrations for reflex apnea should have occurred at the same perceived magnitude in both smokers and nonsmokers. For nonsmokers, the threshold concentration of 41.8% yielded a scale value of 29 (see lower part of Fig. 2). For smokers, the threshold concentration of 52.3% yielded a scale value of 26, close enough to appear consistent with the hypothesis.

Discussion

Cigarette smoking appeals particularly to the common chemical sense, i.e., the sense of irritation, piquancy, pungency, feel, etc. in mucosal tissue. The smoker seeks a feel or impact as the primary chemosensory event in smoking [12]. Both objective (reflex) and subjective (scaling) results now indicate that smokers perceive nasally inhaled common chemical stimuli less keenly than nonsmokers. In the nose and nasopharynx, common chemical sensations arise from action of the smoke on free nerve endings of the trigeminal and glossopharyngeal nerves. These endings lie below or between the ciliated respiratory epithelial cells. In this regard, the endings differ dramatically from the specialized olfactory receptor cells with cilia that protrude directly into the nasal cavity.

Whether the sense of smell escapes deleterious effects of smoking entirely remains unclear. Some investigations have implied a rather small effect [9,22] or an occasional effect [24], whereas various carefully controlled studies have implied no effect [20, 26, 27, 30]. In the present scaling experiment, perceived pungency of CO_2 in smokers and nonsmokers was viewed relative to perceived odor magnitude. Any depression of perceived odor magnitude among our smokers would only have diminished the apparent difference in perceived pungency between the two groups. That is, if any such olfactory depression exists, then the present results would offer a slightly conservative estimate of impairment of common chemical sensitivity in smokers.

One reported adverse effect of smoking is ciliastasis [18,23]. That is, habitual smoking eventually slows down ciliary motility in respiratory epithelial cells. This in turn leads to mucostasis, the development of a deeper, viscid, relatively static layer of mucus over the respiratory epithelium. Such a layer may impede the

transfer of molecules of inhaled irritants from the air to the free nerve endings. If the layer played an obstructive role, it should alter the psychophysical function for pungency by a roughly constant factor across concentrations. This did indeed occur. In the logarithmic coordinates of Fig. 2, the functions for pungency differ by approximately a constant interval. This corresponds to a constant multiplicative factor in linear coordinates.

If ciliastasis accounts for the decreased sensitivity of smokers, why should common chemical sensitivity decrease more than olfactory sensitivity? It is unknown whether smoke may cause ciliastasis in the long, randomly motile cilia of olfactory receptors. It has been suggested that the mucus in this region differs chemically from that in respiratory epithelium [10]. Chemical differences could possibly offer differential protection to olfactory and respiratory cilia.

Other possible ways to account for a differential action of smoke on olfaction and the common chemical sense include a pharmacological explanation and an explanation based on the mere locus of the receptors in the nasal cavities. Otorhinolaryngologists routinely anesthetize the nasal mucosa with cocaine. This eliminates common chemical sensitivity in the region of application. When one of us (W. S. C.) sought, in conjunction with an otorhinolaryngologist, to eliminate olfaction through cocainization the effort failed entirely. It appeared that olfactory receptors resist the pharmacological effects of the drug far more strongly than do the free nerve endings that mediate common chemical sensations. Such differential sensitivity might also manifest itself in any direct pharmacological desensitization induced from the active constituents of tobacco smoke. Finally, the locus of olfactory receptors in the upper reaches of the nasal cavities may essentially protect them from constant stimulation by smoke. As we all know, the best way to obtain clear olfactory sensations is to sniff. This act creates the turbulence necessary to bring a stimulus into contact with these rather remote receptors. Sniffing plays no customary role in the act of smoking.

The present results on loudness perception seem to hint that smokers have slight impairment of auditory functioning. Though not verified statistically in the small sample of persons studied here, such an impairment did reveal itself in an audiological study of 250 smokers and nonsmokers [33]. The reasons for the impairment remain unspecified, but the influence of smoke on the auditory vasculature and on ciliated epithelial cells in the Eustachian tube and middle ear have seemed likely causes [15,25].

In the present application of magnitude matching, we chose to compare perception of pungency primarily to odor perception because this particular comparison seems quite natural. That is, both pungency and odor arise in the nose and the perceptual distinction between them is often ignored. It would have been possible to choose a perceptual continuum other than olfaction, e.g., cutaneous pressure. As research on the method of magnitude matching evolves, it might eventually become clear that some modalities will serve better than

others for intersensory comparisons.

The smokers in the present study refrained from smoking at least one hour prior to the measurement of the reflex. Some smokers refrained for much longer durations on some occasions. For approximately 40% of the tests, the smokers had refrained overnight and were tested before their first cigarette of the day. An analysis uncovered no systematic relation between the threshold for the reflex and duration of abstinence. This outcome suggests that the smokers have a chronic relative insensitivity to pungent stimuli. Within the sample studied here, we could find no significant correlation between the threshold and such nominal parameters as years of smoking, consumption rate, "tar" delivery of current brand, or any simple combination of these. Nevertheless, differences in style of smoking often obscure correlations between such nominal parameters and the physiological and pharmacological effects of smoking [1, 16, 17].

The effect of smoking during only a short interval caused a rather sizeable increment in the threshold. This period of smoking increased the difference in threshold between smokers and nonsmokers by 67%. Quite possibly a measurement taken mere seconds, rather than minutes, after smoking might have revealed an even larger elevation, whereas a measurement taken, say, 10 minutes later might have revealed a much smaller elevation. In short, the modulation of the smoker's sensitivity during and after smoking may prove much larger than that shown here. In order to discover the time-course of any chronic or acute effects, it would seem necessary to chart the sensitivity of individual smokers closely during periods of heavy and light smoking and during periods of abstinence. Furthermore, in order to decide whether any chronic insensitivity derives from smoking rather than from the self-selection that occurs in the decision to become a smoker, it would seem necessary to study former smokers or some youngsters who have yet to develop the habit. The threshold for reflex transitory apnea and the method of magnitude matching appear to be suitable conjoint tools in this endeavor. It would seem worthwhile to apply them also to the question of whether long-term exposure to irritants in the workplace impairs common chemical sensitivity.

Acknowledgements

Supported by NIH Grant ES00592. Performed while J. E. C. M. was visiting the John B. Pierce Foundation Laboratory from the Laboratorio de Investigaciones Sensoriales (CONICET—Fac. de Medicina, Universidad de Buenos Aires), Buenos Aires, Argentina, and was supported by a fellowship from the Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), República Argentina.

References

1. Adams, P. I. The influence of cigarette smoke yields on smoking habits. In: *Smoking behaviour. Physiological and Psychological Influences,* edited by R. E. Thornton. London and New York: Churchill Livingstone, 1978, pp. 349-360.

2. Allen, W. F. Effect on respiration, blood pressure and carotid pulse of various inhaled and insufflated vapors when stimulating one cranial nerve and various combinations of cranial nerves. III. Olfactory and trigeminals stimulated. *Am. J. Physiol.* **88**: 117-129, 1929.

3. Allen, W. F. Effect of various inhaled vapors on respiration and blood pressure in anesthetized, unanesthetized, sleeping and anosmic subjects. *Am. J. Physiol.* **88**: 620-632, 1929.

4. Andersen, P. Inhibitory reflexes elicited from the trigeminal and olfactory nerves in rabbits. *Acta physiol. scand. **30**:* 137-148, 1953.

5. Angel-James, J. E. and M. de Burgh Daly. Nasal reflexes. *Proc. R. Soc. Med.* **62**: 1287-1293, 1969.

6. Angell-James, J. E. and M. de Burgh Daly. Reflex respiratory and cardiovascular effects of stimulation of receptors in the nose of the dog. *J. Physiol.* **220:** 673-696, 1972.

7. Angell-James, J. E. and M. de Burgh Daly. The interaction of reflexes elicited by stimulation of carotid body chemoreceptors and receptors in the nasal mucosa affecting respiration and pulse interval in the dog. *J. Physiol.* **229**: 133-149, 1973.

8. Angell-James, J. E. and M. de Burgh Daly. Some aspects of upper respiratory tract reflexes. *Acta otolar.* **79**: 242-252, 1975.

9. Arfmann, B. L. and N. P. Chapanis. The relative sensitivities of taste and smell in smokers and non-smokers. *J. gen. Psychol.* **66**: 315-320, 1962.

10. Bannister, L. H. Possible functions of mucus at gustatory and olfactory surfaces. In: *Transduction Mechanisms in Chemoreception,* edited by T. M. Poynder. London: Information Retrieval, 1974, pp. 39-48.

11. Cain, W. S. Contribution of the trigeminal nerve to perceived odor magnitude. *Ann. N. Y. Acad. Sci.* **237**: 28-34, 1974.

12. Cain, W. S. Sensory attributes of cigarette smoking. *Banbury Report No. 3: A Safe Cigarette?* Cold Spring Harbor, NY: Cold Spring Harbor Laboratories, 1980, pp. 239-249.

13. Cain, W. S. and H. R. Moskowitz. Psychophysical scaling of odor. In: *Human Responses to Environmental Odors,* edited by J. Turk, J. W. Johnston, Jr. and D. G. Moulton. New York: Academic Press, 1974, pp. 1-32.

14. Cain, W. S. and C. L. Murphy. Interaction between chemoreceptive modalities of odour and irritation. *Nature* ***284****:* 255-257, 1980.

15. Cantrell, R. W. Myringoplasty failure related to smoking: A preliminary report. *Otolar. Clins N. Am.* **III, I**: 141-145, 1970.

16. Creighton, D. E. and P. H. Lewis. The effect of different cigarettes on human smoking patterns. In: *Smoking Behaviour. Physiological and Psychological Influences,* edited by R. E. Thornton. London and New York: Churchill Livingstone, 1978, pp. 289-300.

17. Creighton, D. E. and P. H. Lewis. The effect of smoking pattern on smoke deliveries. In: *Smoking Behaviour. Physiological and Psychological Influences,* edited by R. E. Thornton. London and New York: Churchill Livingstone, 1978, pp. 301-314.

18. Dalhamn, T. and A. Rosengren. Effect of different aldehydes on tracheal mucosa. *Archs otolar.* **93**: 496-500, 1971.

19. Dunn, J. D., J. E. Cometto-Muñiz and W. S. Cain. Nasal reflexes: Reduced sensitivity to CO_2 irritation in cigarette smokers. *J. appl. Toxicol.* **2**: 176-178, 1982.

20. Fordyce, I. D. Olfaction tests. *Br. J. ind. Med.* **18**: 213-215, 1961.

21. García Medina, M. R. and W. S. Cain. Bilateral integration in the common chemical sense. *Physiol. Behav.* **29**: 349--353, 1982.

22. Joyner, R. E. Effect of cigarette smoking on olfactory acuity. *Archs. otolar.* **80**: 576-579, 1964.

23. Kensler, C. J. and S. P. Battista. Components of cigarette smoke with ciliary-depressant activity. Their selective removal by filters containing activated charcoal granules. *New Engl. J. Med.* **269**: 1161-1166, 1963.

24. Koelega, H. S. and E. P. Köster. Some experiments on sex differences in odor perception. *Ann. N. Y. Acad. Sci.* **237**: 234-246, 1974.

25. Maffei, G. and P. Miani. Experimental tobacco poisoning. *Archs otolar.* **75**: 18-28, 1962.

26. Martin, S. and R. M. Pangborn. A note on responses to ethyl alcohol before and after smoking. *Percept. Psychophys.* **8**: 169-170, 1970.

27. Pangborn, R. M., I. M. Trabue and N. Barylko-Pikielna. Taste, odor, and tactile discrimination before and after smoking. *Percept. Psychophys.* **2:** 529-532, 1967.

28. Prescod, S. V. *Audiological Handbook of Hearing Disorders.* New York: Van Nostrand Reinhold, 1978, pp. 65, 110-112, 201.

29. Stevens, J. C. and L. E. Marks. Cross-modality matching functions generated by magnitude estimation. *Percept. Psychophys.* **27**: 379-389, 1980.

30. Venstrom, D. and J. E. Amoore. Olfactory threshold in relation to age, sex or smoking. *J. fd Sci.* **33**: 264-265, 1968.

31. White, S. W. and R. J. McRitchie. Nasopharyngeal reflexes: integrative analysis of evoked respiratory and cardiovascular effects. *Aust. J. exp. Biol. med. Sci.* **51**: 17-31, 1973.

32. Widdicombe, J. G. Respiratory reflexes. *Handbook of Physiology, Section 3, Respiration,* vol. 1. Washington, DC: American Physiological Society, 1964, pp. 585-630.

33. Zelman, S. Correlation of smoking history with hearing loss. J. *Am. Med. Ass.* **223**: 920, 1973.

This is a pre-copyedited, author-produced version of an article accepted for publication in Physiology and Behavior following peer review. The version of record Physiology & Behavior 29: 727-731, 1982 is available online at: http://www.sciencedirect.com/science/article/pii/0031938482902463 - DOI: 10.1016/0031-9384(82)90246-3