Some Physiological Responses to Acute Passive Smoking in Healthy Young Adults

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INTRODUCTION

Exposure to an inhalation of environmental pollutants from tobacco smoke is referred to as passive smoking. At present, it is pointed out that pertinent findings have emerged concerning (1) levels of specific substances such as carbon monoxide, nicotine, and particulate matter, (2) concentrations of specific substances in passive smokers, (3) effects of passive smoking on healthy children and adults, and (4) effects of passive smoking on persons with preexisting diseases.

The present investigation has been aimed to study acute effects of indoor pollution due to cigarette smoking on physiological functions, especially on cardiovascular functions of healthy adults under realistic conditions of laboratory experiments, with special regard to the smoking habit.

METHODS AND MATERIALS

Subjects

Twenty male and female healthy adult volunteers were engaged in the present study. Five male subjects, 20–40 years old, were habitual smokers. The remaining 15 subjects were life-long nonsmokers consisting of 5 subjects of male twenty-agers and female twenty- and forty-agers, respectively.

Procedures: Fig. 1.

Every subject took parts in 2 successive experiments, that is, a control experiment by sham smoking with non-lit cigarettes and an actual passive smoking experiment. The habitual smokers took parts in both the experiments as passive smokers and active smokers. All of the subjects participated in the experiments in groups of 2 nonsmokers and 2 smokers.

The subjects spent 3 and half hours in an exposure chamber of 16 m³ at about 20°C of ambient temperature and about 60% of relative humidity. The air in the chamber was ventilated at ventilation rate of 20 changes/hr for the resting period during which the door was opened, and at 1 change/hr for the smoking period during which the door was closed, respectively.

Throughout the experiments, air pollutants were measured continuously, and physiological indexes such as blinking rates, expired carbon monoxide concentrations, blood pressures, heart rates, products of systolic blood pressure and heart rate (Katz index), finger blood flows (wave heights of photoelectric plethysmograms), and skin temperatures were measured at appropriate time intervals.

For the air of the chamber, CO concentrations were monitored by the Ecolyzer (2500), CO₂ concentrations by the infrared analyzer (model ZFP5 of Fuji Denki), NO, NO₂ and NOₓ by the chemiluminescence apparatus (Monitor Lab 8440), and total particulate matter by the particulate monitor (model P-5H of Shibata-Kagaku-Kikai). Hemodynamic changes were conventionally monitored with a polygraph and blinking rates were assessed by the same inspectors according to the visual counting.

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The passive smoking conditions were prepared by successive smoking of respective 3 cigarettes by 2 smokers during a period of 1 hour under the poor conditions of ventilation, and thereafter the ventilation was switched to the high rate in the recovery period. In addition to this 2 nonsmokers-2 smokers experiment, 1 nonsmoker-1 smoker experiment and 1 nonsmoker-3 smokers experiment were appropriately performed.

Content of nicotine and tar in the mainstream smoke of a cigarette adopted was 0.9 mg and 14 mg, respectively.

RESULTS AND DISCUSSIONS

Measurements of Air Pollutants
Chronological changes in averaged maximum and minimum values for each pollutant are presented according to sham and actual passive smoking sessions.

Fig. 2 shows changes in CO concentrations throughout the experiments. The shaded area indicates the sham or actual smoking period of 1 hour. At the end of actual passive smoking, CO concentrations ranging 1–2 ppm at the initial level reaches the peak level ranging 14–19 ppm, while no appreciable change is noticed throughout the sham smoking period. The peak concentration of CO exceeds the concentration of 10 ppm and approaches 20 ppm, which is the Air Quality Standard for Japan for 8 hours' exposure and 24 hours' exposure, respectively.

Fig. 3 illustrates changes in CO₂ concentrations of which overall trends for both the sham and actual passive smoking experiments are quite similar to each other. The initial levels around 1000 ppm reach the peaks of 3000–4000 ppm for both the experiments. This similarity of the changes indicates that the quantity of CO₂ physiologically expired by 4 subjects is much larger than that in the secondhand smoke generated by 2 smokers.

Fig. 4 demonstrates changes in NO concentrations. The initial level ranging 0.01–0.05 ppm reaches the plateau of peak concentrations around 0.2 ppm, 50 minutes after the start of smoking; maximum 0.23 and minimum 0.18 ppm.

Fig. 5 presents changes in NO₂ concentrations. The overall trends for both the sham and actual passive smoking experiments are very similar to each other. Slight but definite decrease in NO₂ concentrations is noticed even during the sham smoking experiment. This similarity may indicate that the quantity of NO₂ in the secondhand smoke is quite small in comparison with the background levels. Moreover, the declining change during the experiments suggests the adsorption of NO₂ onto clothes of the subjects and surfaces of walls and others of the exposure chamber. The initial background level of 0.03–0.04 ppm appears rather high level, because the Air Quality Standard of Japan refers to the concentrations within or less than the zone of 0.04–0.06 ppm.

Fig. 6 demonstrates changes in NOₓ concentrations of which initial level ranging 0.03–0.08 ppm reaches the plateau around 0.2 ppm, 45 minutes after the start of smoking; maximum 0.26 ppm and minimum 0.18 ppm. The trends of changes in both NO and NOₓ concentrations are quite similar to each other and the concentration changes of NOₓ run parallelly with those of NO at merely high levels. Accordingly, the changes in NOₓ concentrations might reflect mainly those in NO concentrations.

Fig. 7 illustrates changes in total particulate matter (TPM) concentrations. The initial level of 18 counts/min, that means 0.18 mg/m³, reaches the peak ranging 200–250 counts/min, i.e., 2.0–2.3 mg/m³. The background concentration of TPM seems rather high comparing with the Air Quality Standard, 0.10 mg/m³ or less for 8 hours' exposure and 0.20 mg/m³ or less for 24 hours' exposure. However, the obtained peak level of 2 mg/m³ is about 20 times higher than the standard value for the short time exposure.

Physiological Responses to Passive Smoking
All measurements were averaged for 15 minute period and changes during the two experiments were expressed in per cent changes of the initial levels. The illustrated column and bar indicates mean and standard error, respectively. Annotations of T₁ to T₄ indicate four sequential periods of the passive smoking experiments and T₅ and T₆ indicate two sequential periods of recovery.

Fig. 8 demonstrates changes in blinking rates (B.R.) and expired CO concentration (CO) for the nonsmokers (NSm) on the left side and for
the smokers (Sm) on the right side. Blinking rates of both the nonsmokers and smokers show remarkable increases along with the progress of the actual passive smoking. Although the changes are less pronounced for the smokers, the eyes of smokers who had been accustomed to inhale tobacco smoke, could not bear the irritation by the secondhand smoke.

Concentrations of expired CO also increase remarkably along with the progress of passive smoking for both the nonsmokers and smokers. While the changes are much pronounced for the smokers who smoke actively as well as passively, the change even for the nonsmokers is approaching almost half the change in the smokers.

Fig. 9 presents appreciable increases in heart rates and systolic blood pressures for the nonsmokers, and marked increases in them for the smokers throughout the actual passive smoking experiment. Although the changes in both the indexes for the nonsmokers are much less pronounced than those for the smokers, these findings indicate definite acute cardiovascular responses of the nonsmokers to the passive smoking in the same direction as the responses of the smokers to the active smoking.

Fig. 10 presents changes in product of systolic blood pressure and heart rate, that means oxygen cost of the heart, and in skin temperature. Although the extents of changes in both the cardiotoxnic and vasoconstrictive measurements are much less for the nonsmokers than the smokers, these responses also indicate definite acute cardiovascular changes of the nonsmokers during passive smoking in the same direction as those of the active smokers.

Table 1 shows overall comparisons between selected physiological responses to the sham and actual smoking experiments and the same between the physiological responses of the smokers and nonsmokers to the actual passive smoking. The extents of all the physiological responses of both the nonsmokers and smokers to the actual passive smoking except for diastolic pressures the nonsmokers, are significantly higher than those to the sham smoking experiment. The acute physiological effects of the passive and active smoking under the present conditions are quite evident in the nonsmokers and in the habitual smokers, respectively. Therefore, it is clear that the passive smoking under certain realistic conditions can inevitably cause acute changes in some physiological, especially cardiovascular functions of nonsmokers, in addition to the annoyance and irritation.

Moreover, the extent of the change in blinking rates of the smokers does not differ from that of the nonsmokers while the remaining responses of the smokers to the actual passive smoking significantly differ from those of the nonsmokers. This means that the eyes of the smokers as well as the nonsmokers suffer similarly from the irritation of the secondhand smoke generated by themselves.

CONCLUSIONS

Under realistic conditions of the indoor pollution due to the secondhand tobacco smoke, even healthy young adults should experience some physiological responses to passive smoking regardless of smoking habit, although the habitual smokers respond in much less pronounced magnitudes. Even if the acute cardiovascular effects of passive smoking could be underestimated from the clinical point of view at present, the disturbance of normal physiological function which was typically evidenced by the increasing rates of eye blinking suggests an infringement by passive smoking on the amenity of nonsmokers from the point of view of public health. Moreover, the cardiovascular responses imply definitely unnecessary physical and mental burdens on the homeostasis of normal living body and the increased CO-Hb levels, indicated by elevated concentrations of expired CO during and after the passive smoking, may give warning on the chronic influences of inhalation of concomitant carcinogens in the sidestream smoke on development of lung cancer at least.

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REFERENCES

Table 1  Statistical Significances for Comparisons of Some Selected Physiological Responses between Sham and Actual Passive Smoking, and between Nonsmokers and Smokers. P.P.G. refers to maximum amplitudes of photoelectric plethysmographic tracing of a finger tip. Concerning other abbreviations, see in the text.

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COMPARISON BETWEEN EFFECTS ON SMOKERS(Sm) AND NONSMOKER(NSm) OF ACTUAL EXPERIMENT

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Fig. 1  Experimental Procedures.

Fig. 2  Time Course of Changes in CO Concentrations.
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Fig. 3  Time Course of Changes in CO₂ Concentrations.

Fig. 4  Time Course of Changes in NO Concentrations.

Fig. 5  Time Course of Changes in NO₂ Concentrations.
Fig. 6  Time Course of Changes in NO$_X$ Concentrations.

Fig. 7  Time Course of Changes in Total Particulate Matter Concentrations.
Fig. 8 Changes in Blinking Rates (B.R.) and Concentrations of Expired CO (CO) Comparing between Nonsmokers (NSm) and Smokers (Sm).

Fig. 9 Changes in Heart Rates (H.R.) and Systolic Blood Pressures (Syst. B.P.) Comparing between Nonsmokers (NSm) and Smokers (Sm).
Fig. 10 Changes in Katz Indexes (Syst. B.P. × H.R.) and Skin Temperatures (S.T.) Comparing between Nonsmokers (NSm) and Smokers (Sm).