COMBINED EFFECTS OF NOISES AND CIGARETTE-SMOKING

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Abstract

A total of 40 healthy male smokers and 40 healthy male non-smokers were exposed seven times to stable broadband noise of 70 dBA and 90 dBA at 35 °C. Total exposure duration was 112 minutes. During the 4 min pauses between consecutive exposures smokers smoked altogether five filtered cigarettes, one per pause. Functional changes were characterized through determining blood haematocrite, haemoglobin, erythrocyte sedimentation rate, glucose, haemodynamic activity and temporary threshold shifts of hearing. The results showed a significant rise in the mean haematocrite, blood glucose and haemodynamic index values after smoking. Smoking and noise combined to increase glucose response. Significant increases in blood glucose were observed more often after smoking under coexisting intense noise exposure of 90 dBA than under moderate coexisting noise exposure of 70 dBA. Provable increase in blood glucose seemed to inhibit the increase of temporary threshold shift TTS2 of hearing at 4 kHz among smokers. Similarly among smokers haematocrite values were significantly higher after smoking at 90 dBA noise than after smoking at 70 dBA noise. These findings conclude that unidimensional models are inadequate to explain the effects of smoking.

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Introduction

Carbon monoxide (CO) present in the human body has two origins: an endogenous production, as a consequence of the catabolism of hemoproteins, and an absorption of inhaled carbon monoxide generated from natural and anthropogenic sources (Castellino and Sannolo 1987). Under normal conditions the haemoglobin catabolism is responsible for 80 % of CO endogenous production, while the remaining 20 % results from the catabolism of the other intracellular hemoproteins (myoglobin, cytochromes, catalase etc).

With the exception of some professions (like waiter’s work) the major non-occupational sources of CO exposure in human are cigarette smoke, automobile exhaust, and other emissions from fossil fuels (Manninen 1988b, Aviado 1996, Morgan et al 1997). However, CO is only one of many constituents of tobacco smoke. Earlier reviewers have reported inorganic oxides and sulfides nitrogenous compounds (including nicotine), non-halogenated solvents and other miscellaneous compounds as other constituents of tobacco smokes (Aviado 1996). These other components in cigarette smoke are to be cytotoxic.

It has been noted that in smokers there is a positive correlation between the number of cigarettes smoked and the levels of carboxyhaemoglobin (COHb); moderate smokers (up to 10 cigarettes/day) have COHb less than 4 %; smokers (up to 20 cigarettes/day) have between 5 to 7 %; heavy smokers (more than 20 cigarettes/day) have greater than 10 %. Even COHb concentrations exceeding 15 % can be observed in some heavy smokers (Castellino and Sannolo 1987). On the average, the concentration of CO in cigarette smoke varies from about 400 ppm (Goldsmith and Landaw 1968 cited in Birnistingl et al 1971) to about 500 ppm (Castellino and Sannolo 1987) in the alveolar space if the appropriate dilution of the smoke with clean air is taken in consideration.
The alleged association between cigarette smoking and health consequences has led to numerous speculations as to the mechanisms (e.g. Turner 1979, US Department of health and human services 1988). However, there are many aspects of this process that are not yet well understood.

What is more puzzling is that some individuals smoke 20 cigarettes per day throughout their lives with little apparent ill effect. Some of the difference in the risk of developing these ill-effects and diseases among such smokers may be genetically determined, but the degree to which tobacco smoke is inhaled and absorbed is also likely to be important. The COHb boost per cigarette will also depend on person's total haemoglobin and myoglobin content. Filtered cigarette smokers must inhale more deeply to obtain the same amount of nicotine as nonfiltered cigarette smokers, and therefore they are exposed to a greater amount of CO, which is not trapped by filters. On the other hand, environmental sources of carbon monoxide can influence obtained levels, and the half-life of CO is very short, making it dependent upon very recent cigarette use (Perkins 1985).

Moreover, the discrepancy depends partly on methodological differences in various studies, partly on the situational environment and work related factors linked to smoking which unfortunately have frequently been omitted. For example, we know that noise exposure, moderate heat or work or exercise may accelerate excretion rates of catecholamines in the same way as the cigarette smoking itself. The results have also showed that the heart rate tended to rise in particular among smokers during the first and second hour of exposure. Unexpectedly, the rise of hearing threshold values was slightly higher among the non-smokers than among the smokers and at 4 kHz than at 6 kHz (Manninen 1989). Thus the short-term effects of tobacco smoking on haemodynamic activity seem to be hypertensive by nature. It is conceivable that environmental tobacco smoke and nicotine exposure raises circulating catecholamines, which, in turn, may increase platelets and the potential for thrombus formation (Dintenfass 1975).

Besides the well-known circulatory effects of cigarette smoking some investigations have shown an association with elevated serum-lipids while others found no such relationship. Some earlier authors reported that cigarette smoking was followed by a significant rise in blood-glucose and a transient increase in the total platelet count (Murchison and Fyfe 1966). The factors responsible for platelet adhesiveness are not completely established, but some which have been suggested are blood-glucose and various plasma-lipid components including triglyceride and fatty acids. So high cholesterol might promote the development of vulnerable plaques, and smoking promotes the formation of blood clots overlying vulnerable plaques. The increased carboxyhaemoglobin level in smokers impairs blood oxygen-carrying capacity and increases blood-oxygen affinity (Lowe et al 1980).

Since these are factors affected by cigarette smoking, the present investigation was carried out to determine the short-term effects of cigarette smoking on all these factors and the relationship between them. This kind of investigation is needed, because we still have a lack of tightly controlled studies on the effects of smoking under simultaneous noise exposure mimicking daily exposure conditions. To shed more light on this actual issue we studied blood glucose, haemodynamic activity, blood viscosity determinants and hearing thresholds in healthy men with respect to smoking and noise at elevated ambient temperatures. Due to practical reasons it has not been possible to realize these kind of comparisons and further analyses ever before.

Material and methods

Exposure arrangements

The study was carried out in a special exposure chamber and as a factorial experimental design. One personal test comprised a pre-exposure period and seven consecutive 16 minutes taking exposures each followed by a pause of 4 min for measurements and
rest. The dry bulb temperature inside the chamber was kept constant at 35 °C and relative humidity at 50 percent during the whole experimental session. The air velocity was less than 0.1 m/s. The noise used in the tests was stable broadband noise. Its intensity was either 70 dBA or 90 dBA. During the experiments the subjects were working in sitting position with a choice reaction apparatus. The work was very light and resembled control room work. The measurement arrangements and equipment used for the exposures involved have been reported previously elsewhere (e.g. Manninen 1983, 1985, 1986, 1989).

Subjects

A total of 80 healthy male volunteers were selected for the experiment. 40 of the subjects were smokers and 40 were non-smokers. The smoking subjects had smoked on average for 3 years. Among smokers (n=40) there were 14 persons who had smoked less than 5 years, 15 persons who had smoked for 5-10 years and 11 persons who had smoked longer than 10 years. All smokers smoked cigarettes. At the moment of the experiment 18 smokers reported to smoke less than one pack (under 20 cigarettes) and 22 smokers reported to smoke more than one pack (over 20 cigarettes) daily.

The subjects were thoroughly familiarized with all the techniques in pilot experiments. Each subject wore similar experimental clothes during the experiments.

Smoking

During the 4 min break the smokers smoked their cigarettes and non-smokers rested sitting in the vibration chair. During the experiment the smokers smoked a total of five filtered red Marlboro cigarettes, each with a tar content of 15 mg, carbon monoxide content of 12 mg and nicotine content of 0.9 mg. A selection of five cigarettes per one trial was arrived at by approximating the minimum daily exposure and the shortest treatment period that supposedly could produce noticeable changes in cardiovascular function as well as blood variables. The smoking style was controlled by researchers: they ensured that each smoker smoked as agreed, inhaled the smoke and finished the cigarette during the break. Sham-smoking was not allowed. Each smoker was given a free pack of cigarettes about a week before the test so they could get accustomed to the blend.

Determination of variables

During the exposure period changes in the body functions were characterized by determining the $TTS_2$ values of both ears (dB), systolic and diastolic blood pressure (SBP, DBP; mmHg), haemodynamic index (Ind) and heart rate (HR; beats/min). The heart rate function of the subjects was monitored continuously with a two-channel memory cardioscope (Olli 431D). The monitoring electrodes were in positions $V_1$, $V_3$ and $V_5$ on the subjects chest (see Manninen 1997). The Ind values were calculated on the basis of the product of systolic blood pressure and heart rate (see Robinson 1967, Manninen 1988a).

Changes in the blood variables were characterized by measuring haematocrit (Hct; %), haemoglobin (Hb; g/L) and erythrocyte sedimentation rate (ESR; mm/h). Blood glucose concentrations (mmol/L) were determined with the orthotoluidine method (Yee et al 1971). The subjects fasted overnight.

The pure-tone hearing threshold ($TTS_2$) was determined with a pure tone audiometer (type Interacoustics Diagnostic Audiometer AD17) using the ascending technique. Hearing thresholds at the frequencies involved were measured for both ears. In this article only $TTS_2$ values 4 kHz have been counted.

All determinations on both smokers and non-smokers were performed in duplicate.

Analysis of data

The pre-exposure values were determined in this way before the exposure period and the post-exposure values were determined on the basis of blood samples taken after the exposure period.

The subjects served as their own controls. Results obtained from exposure measurements were corrected by subtracting the results of the second measurement made during the pre-exposure control period. The results are described using means calculated over the whole material in classes of noise
and smoking. The significance of the differences of means were characterized using the t-test of differences of independent means. The statistical significance of the main effects of and interactions between the independent exposures was also tested by an analysis of variance (F-test).

Results

The results of the experiment are shown in Figures 1-6. The figures depict mean differences of the variable values determined immediately before and after single exposures and exposure combinations.

There was a significant rise in the mean haematocrite \( (p<0.05) \), glucose \( (p<0.01) \) and haemodynamic index \( (p<0.001) \) values after smoking (Fig. 1). Mere noise exposure to 90 dBA increased glucose \( (p<0.05) \) and TTS values at 4 kHz \( (p<0.001) \) more significantly than noise exposure to 70 dBA (Fig. 2).

Significant increases in blood-glucose \( (p<0.001) \) were more frequent after smoking under coexisting intense noise exposure of 90 dBA than under moderate coexisting noise exposure of 70 dBA. Similarly among smokers haematocrite values were significantly \( (p<0.001) \) higher after 90 dBA noise than after 70 dBA noise (Fig. 3).

Mean sedimentation rates decreased significantly \( (p<0.1) \) both among non-smokers after intense noise exposure of 90 dBA than after moderate noise exposure of 70 dBA (Fig. 4, 5) and after moderate noise exposure of 70 dBA among smokers compared to the corresponding mean values \( (p<0.01) \) among non-smokers (Fig. 6).

Regardless of different changes in haemoglobin concentrations there were no statistically significant differences in the means of the haemoglobin values calculated by any groups of smokers, non-smokers, noises or their mutual combinations.

The results of the variance analyses showed that both noise exposure \( (F=4.99, p<0.05) \) and smoking/non-smoking \( (F=8.52, p=0.005) \) had significant single effects on the variation of the differences of blood glucose values. Noise and smoking formed a significant two factor combination \( (F=8.08, p=0.005) \) that affected the variation of differences of blood glucose values, too.

Discussion

The COHb boost per cigarette will depend on the method of smoking (the size and number of puffs and the depth of inhalation), the pulmonary transfer factors, and the CO yield from the cigarette (Wald et al 1975). According to the same authors the between-person variation in COHb boost per cigarette might be at least tenfold. So, a further possible predictor of adverse health effects is the peak COHb value during the day (Astrup 1972).

This indicates that the method of smoking is the main factor affecting COHb boost per cigarette, although from the data collected in this study it was not possible to isolate the independent effects of size of puff, number of puffs, or depth of inhalation. To standardize cigarette smoking in this study, the researchers controlled the smoking styles of subjects as far as possible, and no sham-smoking was allowed. Other arrangements involving the pretraining sessions, state of health, eating and drinking, leisure activities of the subjects as well as the operation of the exposure chamber during the actual experiments were also under control.

Our results show that cigarette smoking as such as performed in our tests is followed by an increase in haematocrit, blood glucose and haemodynamic activity. It has been shown that acute exposure of human subjects to tobacco smoke produces a perturbation of the cardiovascular system. The response appears to result from a combination of peripheral arterial constriction and cardiac stimulation and is probably mediated by a nicotine-induced release of catecholamines (e.g. Richardson et al 1975, Cryer et al 1976). Results of Dembroski et al (1985) revealed that the combination of smoking cigarettes and a mildly stressful activity (induced by video game) produced heart rate and diastolic and systolic blood pressure responses that were larger than the effects of factors causing separately. Woodson et al (1986) also reported that during noise, smoking induced cardiovascular stimulation (i.e. heart rate acceleration). So our findings that smokers have increased levels of haematocrit agree very well with those of Eisen and Hammond (1956), Dintenfas (1975) and Lowe et al (1980). Furthermore, results from our experiments show that a short but regularly
Fig. 1. Mean (X±SEM) differences of haematocrite, haemoglobin, sedimentation rate, glucose, haemodynamic index and TTS₂ values in non-smoker group (n=40) and smoker group (n=40)
Fig. 2. Mean (X±SEM) differences of haematocrite, haemoglobin, sedimentation rate, glucose, haemodynamic index and TTS$_2$ values in noise group of 70 dBA (n=40) and in noise group of 90 dBA (n=40)
O Manninen. Combined Effects of Noises and Cigarette Smoking

Fig. 3. Mean (X±SEM) differences of haematocrite, haemoglobin, sedimentation rate, glucose, haemodynamic index and TTS₂ values in noise group of 70 dBA (n=20) and in noise group of 90 dBA (n=20) among smokers
Fig. 4. Mean (X±SEM) differences of haematocrite, haemoglobin, sedimentation rate, glucose, haemodynamic index and TTS₂ values in noise group of 70 dBA (n=20) and in noise group of 90 dBA (n=20) among non-smokers.
Fig. 5. Mean (X±SEM) differences of hemocrit, hemoglobin, sedimentation rate, glucose, haemodynamic index and TTS$_2$ values in non-smoker group (n=20) and smoker group (n=20) under 90 dBA noise.
Fig. 6. Mean (X±SEM) differences of haematocrite, haemoglobin, sedimentation rate, glucose, haemodynamic index and TTS₂ values in non-smoker group (n=20) and smoker group (n=20) under 70 dBA noise.
repeated noise and smoking combined accelerated blood glucose values considerably more than a noise exposure and smoking alone. These findings are supported in the literature, too. Simpson et al (1974) noted changes in blood glucose levels under chronic exposure to high intensity noise. This is also partly consistent with the findings done by Roth and Shick (1960) and Asbel (1965).

Likewise, there is some evidence that work or exercise during heat stress conditions may result to greater increases in catecholamines and blood glucose than exercise in a thermoneutral environment (e.g. Hartung et al 1987). During sedentary light work in the moderate heat like in this investigation, the relative stability of cardiac filling pressure is likely the consequence of an adequate compensation for a reduction in central blood volume accompanying cutaneous vasodilation and the associated increase in volume of blood residing in the periphery (Tripathi et al 1990). On the basis of previous findings of ours (Manninen 1988a) we can further note that a temperature of 35 °C and 90 dBA noise together increase the catecholamine ratio more than a temperature of 35 °C alone.

It is a reasonable assumption that the glucose removed from the blood is metabolized for the production of energy, rather than storage as glycogen. The accelerated secretion rate of catecholamines increases the production of glucose in the liver and lipolysis in the fat tissue. Besides their effect on metabolism catecholamines also have a stimulating effect on the blood circulation. By increasing the heart rate they cause the circulation to bring more glucose to the brain. As the results show, smokers have an elevated glucose level compared to non-smokers and, correspondingly, higher glucose level in the brain. The acute hyperglykemia seems to compensate the evident adverse effects of the blood hypoxia (rheological changes with increases in haematocrit) and viscosity in the sensory cells of the hearing organ and thus prevent hearing impairment in smokers during short-term but repeated noise exposures (see also Browning et al 1986).

On the other hand, we have to consider that glucose is somehow in connection with the transportation of the other substances like red cells. Evidence has shown that glucose can enter or leave the erythrocytes and does not accumulate with the cells above the concentration existing externally (e.g. Vosteen 1970). So acute increment in haematocrit values means corresponding increasement of glucose in red cells. However, the rate of influx of sugar is proportional to the external sugar concentration only to a certain extent. Therefore, in a long run, when bodily stores of glucose become exhausted, deterioration of hearing might happen due to recurrent noise and smoking.

As a whole, these findings conclude that unidimensional models are inadequate to explain the effects of smoking.

In a large Japanese cross-sectional study (Momose et al 1997) the number of cigarettes smoked per day was found to relate negatively to HDL-cholesterol, positively to diastolic blood pressure and not at all to total cholesterol level or systolic blood pressure. Formerly Kannel et al (1986) reported the strong relationships between heart disease mortality and the smoking and elevated cholesterol, as well as hypertension. The data presented by the latter authors showed that the combination of smoking and elevated cholesterol confers greater than additive effects on coronary heart disease risk. What is also important, the same data illustrated a common issue that arises in epidemiological data analysis: the presence or absence of an interaction between risk factors may depend on the model chosen (i.e. Perkins 1987).

A further clarification of these relationships may therefore help to explain the underlying mechanism. To minimize this kind of uncertainties linked to data treatments and controversial effects induced by smoking, it is acceptable to include both cholesterol and fatty acids into the set of variables in the continuous analyses of ours in near future. To describe the obvious degree of acute dehydration (see Dill and Costill 1974) among subjects and its association to the variables involved in that context it would be wise to estimate percentage changes in volumes of plasma and of red cells, too.

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