EFFECTS OF SMOKING WHEN BEING EXPOSED TO DIFFERENT ENVIRONMENTAL FACTORS IN FIELD AND LABORATORY CONDITIONS; A CRITICAL REVIEW

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Abstract

This report discusses recent studies on smoking and the results of an experimental study conducted in an exposure chamber. The purpose of this two-stage project was to chart in brief recent studies on smoking that are of interest in terms of labour protection and to investigate the effects of smoking in complex exposure situations involving simultaneous exposures to numerous different environmental factors. Despite the large number of published works on smoking and the detailed documentation available of the health effects of smoking, the review revealed that at the moment there is rather limited knowledge on the additional and combined effects of smoking in different work and exposure situations. The experimental study, in turn, proved that during the tests the differences between smokers and non-smokers in the values and changes of the temporary hearing threshold \((TTS_2)\) and blood count \((HbO_2, Hcr, ESR)\) were particularly high when the subjects were besides noise simultaneously exposed to a whole body vibration at an elevated ambient temperature. The rise in the \(TTS_2\) values was higher with non-smokers than with smokers and more pronounced at 4 kHz than at 6 kHz. To obtain more useful information with a view to labour protection future studies should have a specific focus on the viscosity of blood, the relations between blood fat and sugar contents and hearing both in multi-exposure laboratory experiments and in real life working situations involving both hot and cold temperatures.

Smoking and carbon monoxide exposure

Cigarette smoke is one important and widespread source of carbon monoxide among people. Therefore there seems to be reason to consider the carbon monoxide contents reported by different studies as an indicator of cigarette smoke exposure, provided that the other sources can be identified and subtracted. In general the population should be protected against carbon monoxide exposures that produce carboxyhaemoglobin contents higher than 5 per cent. This recommendation is based on tests made on animals and on other observations concerning carbon monoxide exposure. The measured blood carboxyhaemoglobin levels however vary on a wide range, irrespective of the amount of smoking, which suggests that the number of daily smoked cigarettes is only one of the many factors affecting the total amount of carbon monoxide inhaled by a smoker and the potential health hazards (see Manninen 1988a).
The amount of carbon monoxide transferred to blood circulation depends on the carbon monoxide content of the alveoli, which in turn is affected by the dilution of smoke into other gasses. Apart from the amount of smoke inhaled the ventilation of lungs is also an important factor affecting the dilution. Thus it has been proposed that physical exercise, which increases the ventilation of lungs, also increases the dilution of smoke. In fact this does not necessarily cut down the carbon monoxide dose inhaled, because a more effective ventilation and perfusion of the pulmonary alveoli probably means increased carbon monoxide uptake. In this light the most important factors seem to be the rate at which carbon monoxide exits the body during non-smoking periods and the carbon monoxide quantity inhaled during smoking.

**Smoking and functional capacity**

Many smokers claim that smoking helps them to tackle stressful situations better. Yet there are very few studies on the effects of smoking in real-life situations or in situations simulating reality. At this junction I refer to two studies that are most relevant for the topics discussed here:

Heimstra et al. (1967) noted that smoking did not compensate or reduce the effect of tiredness during a long drive. Aston et al. (1972), by contrast, noted clear differences in the performances of smokers and non-smokers. All changes in performance were limited to the first half of the test, when the smokers smoked, and disappeared during the second half, when they no longer smoked. In the light of these results it seems evident that the changes were not caused by intrinsic differences in the subjects, such as differences in personality between smokers and non-smokers; in one way or another the differences were related to smoking. It could further be noted that the lack of differences in easy or relatively stress-free situation suggests that the effects of smoking are rather subtle and mostly appear in complex situation involving a high degree of stress.

As far as physiological functions are concerned, perhaps the most surprising finding was that no differences could be observed in heart rate between smokers and non-smokers. It is generally known that smoking increases heart rate; but here the finding worth noting is that the smokers' increased heart rate continued to be high after the test and during pauses, about 20-30 minutes after smoking. It seems that smoking has a long-lasting effect on heart rate. The noted difference between people smoking high- and low-nicotine cigarettes, on the other hand, may be attributable to the fact that those smoking light cigarettes take more puffs of one cigarettes than those smoking high-nicotine cigarettes, which in turn increases the blood
carboxyhaemoglobin level. As to other physiological changes, worth mentioning is that smokers generally had weaker peroneal blood flow than non-smokers, but the differences were not statistically significant. During driving blood pressure rose equally in smokers and non-smokers, and it seems that the rise is due to the stress involved in the task itself, which may have covered the single effect of smoking on blood pressure.

Smoking and hearing

The results of Barone et al. (1987) show that smoking coincides with an increased risk of hearing loss in people exposed to noise. The risk seems to have a statistically significant correlation with the total exposure (annual number of packs) and current smoking frequency (number of packs per day). Thus it seems that current smoking anticipates a risk of hearing loss, while earlier smoking does not have a significant correlation with hearing loss.

The role of a noisy hobby has also been prominently displayed in all studies on noise-induced hearing loss. Although the effect of a noisy hobby is relatively small compared to the effect of a life-time working in a noisy factory, out-of-work noise exposure may still be an important contributor to hearing loss in young people with a short history of work-induced exposure. In fact Kryter (1985) has noted that in particular factory workers under 30 years of age are more frequently exposed to out-of-work or leisure-induced noise than other people.

Thomas et al. (1981) compared a group of 42-45 years old navy pilots with normal hearing to a group with a reduced hearing. The main factors that distinguished the bad-hearing group from those with normal hearing were higher smoking frequency and a light eye pigment. Chung et al. (1982) have also shown that there is a positive correlation between hearing loss and smoking.

It has been proposed that predisposition to noise-induced hearing loss is caused by a sort of ischaemic condition in the inner ear. A hazardously strong noise causes increased metabolism and capillary vasoconstriction in the cochlea, which leads to reduced oxygen tension and lactate accumulation (Hawkins 1971; Schneider 1974). The relative carboxyhaemoglobinemia caused by smoking may aggravate the situation and cause increased sensory cell damages. Browning et al. (1986) noted that hearing loss at a high frequency range was directly correlated with high blood viscosity and reduced cell tension. Smoking can cause rheological changes with an adverse effect on micro-circulation. This, in turn, may exacerbate the existing metabolism disturbance of the ear previously exposed to noise.
Smoking and changes in health

There is plenty of evidence of the contribution of smoking to the emergence of fatal or mild myocardial infarcts. Still epidemiological research has not been able to show a direct causal relation between the number of smoked cigarettes and mortality in the coronary disease.

What has been proved is a certain proportion between the number of smoked cigarettes and the content of high-density lipoprotein-cholesterol; this content is considered one indicator of the risk of coronary disease (Hill et al. 1983).

The major components of cigarette smoke, nicotine and carbon monoxide, are key factors in exacerbating cardiovascular disturbances. Smoking itself, or nicotine in general increases pulse heart rate and blood pressure by increasing myocardial contraction and after-load. Smoking also stimulates catecholamine and cortisol secretion. These in turn are connected to the development of a ventricular arrhythmia and myocardial infarct. Carbon monoxide as such is a cause of the coronary disease. As carbon monoxide is more easily bonded with haemoglobin than oxygen, it increases the myocardial oxygen demand and causes hypoxia in patients with blocked arteries. Cigarette smoke also contains cytotoxic ingredients such as hydrocyanide, which is converted to thiocyanate and accumulates in body fluids preventing vesicular breathing.

Epidemiological research has further shown that the risk of coronary disease increases with cigarette consumption, but the correlation between the risk and inhaled smoke is not completely clear, because the studies have used data reported by the subjects and estimates of the nicotine and carbon monoxide doses of different cigarette brands based on machine smoking measurements (Kozlowski et al. 1980). As the risk of coronary disease caused by smoking depends equally on the way of smoking and the components of the smoke, it is obvious that the real amount of inhaled smoke is related to the subject's behaviour and the intensity of smoking.

Although epidemiological studies prove smoking to increase the risk of coronary disease as one factor, the single and combined effects of carbon monoxide, nicotine and thiocyanate on the risk of coronary disease are still unclear.
Combined effects of smoking and environmental factors

When driving or working in automotive machines people are often smoking. This means above all that due to smoking the drivers are exposed to certain combinations of environmental factors consisting of cigarette smoke, noise, vibration, temperature, illumination level and various chemical environmental factors. Although smoking and such combinations are nowadays very common, we still have no knowledge on the significance of smoking in complex exposure and working situations.

The results of a recent laboratory study (for details see Manninen 1988a) showed that the averages of TTS₂ values at 4 kHz in darkness (0 lux) in non-smoking subjects were significantly higher when the subjects were exposed simultaneously to stochastic vibration and a 90 dBA noise than in an exposure to a 90 dBA noise alone. When the duration of the exposure was extended from one hour to two hours this difference was enhanced. When the illumination level at the exposure chamber was 300 lux, no difference of such magnitude between a noise exposure and a combination of simultaneous noise and vibration could be observed. Likewise, among smokers an exposure to simultaneous noise and vibration did not accelerate the rise in the TTS₂ values from the levels caused by noise exposure alone. In non-smoking subjects, by contrast, a simultaneous noise and vibration exposure seemed to accelerate the rise of the TTS₂ values at 6 kHz when the subjects were exposed to this combination at an illumination level of 300 lux. Only when the exposure combination included darkness (0 lux) and a 90 dBA noise, the average TTS₂ values at 4 kHz increased in smoking subjects more than in non-smoking subjects. The differences in the averages were 5-6 decibels.

Haemoglobin levels increased virtually in all exposure combination classes with the exception of a combination consisting of darkness (0 lux), stochastic vibration and a 90 dBA noise in the group of non-smokers. The rise in the average haemoglobin levels was also rather small when smoking and non-smoking subjects were exposed to a 90 dBA noise in darkness (0 lux). Similarly, the average haemotocrit values also increased in all exposure classes from the pre-exposure levels.

The results of variance analysis showed that the variation of the TTS₂ values at the frequencies of 4 and 6 kHz were correlated not only with a simultaneous noise and vibration exposure but also with the illumination level of the exposure situation. It was also worth noting that smoking together with vibration had a significant effect on the variation of the
TTS$_2$ values at 4 kHz during an exposure of 2 hours. However, the most significant combined effect on the variation of the TTS$_2$ values at 6 kHz was found for simultaneous noise, vibration and illumination. As a whole the variance analysis model explained 54-63 percent of the variation of the TTS$_2$ values at 4 kHz and 53-54 percent at 6 kHz. The results of the variance analyses further showed that noise had a significant effect on the variation of haemoglobin levels during a two-hour exposure, which could also be inferred from the averages calculated by noise classes. Smoking alone and vibration alone had a marked effect on the variation of the haematocrit levels. Likewise, noise and vibration had a significant combined effect on haematocrit values. Further it could be noted that smoking and vibration together and noise and vibration together had very significant effects on the blood erythrocytes sedimentation rate (ESR).

**Summary**

In recent times there has been a lively discussion on the effects of smoking on people's health and working. A number of aspects relevant to labour protection have also been discussed in connection with smoking, such as the need to limit smoking at work-places and to protect non-smokers from the smoke produced by their smoking fellow workers.

Cigarette smoke contains many hundreds of chemical elements and compounds (USDHHS 1988). Carbon monoxide is generally known to be the most active component of cigarette smoke. It is also one of the main impurities produced by traffic. The carbon monoxide in cigarette smoke and exhaust gases have been suggested to have several different effects on the body functions. Changes in the blood count may be very rapid, because compared to oxygen carbon monoxide is much more active in forming bonds with the haemoglobin of erythrocytes in pulmonary alveoli.

Eisen and Hammond (1956) have rather early reported increased haemoglobin and haematocrit levels in smokers. The high levels are probably due to a chronic carbon monoxide poisoning. An increased blood carboxyhaemoglobin content in smokers reduces the blood's capacity to transport oxygen and increases the affinity of blood. The low oxygen content in tissues increases the haemoglobin and erythrocyte synthesis in smokers, which in turn increases the mass of erythrocytes and haematocrit levels. Finally the blood viscosity changes. A low blood viscosity would affect hearing thresholds at 2-4 kHz, while a high blood viscosity seems to affect hearing threshold values at 6-8 kHz (Browning 1986). Besides smoking age also seems to influence blood viscosity and the factors controlling it. In general smokers at all ages tend to have higher blood viscosity levels than non-smokers.
Many researchers agree that a high blood viscosity is one factor contributing to the risk of blocked arteries, and in particular it is believed that smoking increases the predisposition of young people to atheroma or thrombosis; carbon monoxide or hypoxia increases the deposition of lipoid plaques and fat and other changes in arteries. The assumption is supported by the fact that after smoking even light physical effort or a mild exposure to air impurities in traffic jams may launch an attack of illness.

As a whole both the literary review and the laboratory tests produced some exceptionally interesting results and shed some new light on smoking and its effects on body functions in complex exposure situations. A short but regularly repeated simultaneous noise and vibration exposure accelerates the rise of the TTS$_2$ values at a high temperature much more than a mere noise exposure. However, the rise in the TTS$_2$ values is higher in non-smokers than in smokers and at 4 kHz than at 6 kHz. In this respects the results agree with the results of Dengerink and Dengerink (1986): smokers who smoked before the noise exposure had only half of the TTS$_2$ level measured for non-smoking subjects. The differences in the blood count and in changes of the blood count between smokers and non-smokers were particularly high when the subjects were besides noise exposed simultaneously to a whole body vibration.

All in all, at present there is little knowledge of the effects of smoking in complex and real-life exposure situations. Particularly in situations where smoking coincides with noise, vibration and different temperatures, smoking may alter the single effects of noise, vibration or temperature. Existing studies of smoking and the discussion on the effects of smoking have largely overlooked these additional or combined effects, ostensibly for the simple reason that our knowledge on these effect is extremely scarce. There are only a few studies on traffic safety which have established a potential relationship between traffic accidents and smoking in particular if the driver has a hangover and is driving crowded and polluted roads (see Manninen 1988b). In terms of labour protection it would evidently be useful to locate such combinations of smoking and environmental factors that cause more than normal disturbances in body functions and/or are the most hazardous to the operational and working performance of employees. It was therefore one of the main objectives of the present study under carefully controlled conditions to give answers to these urgent questions in labour protection.

Acknowledgements

The study was financed by the National Board of Labour Protection in Finland.
References


