Dark Adaptation and Recovery from Light Adaptation: Smokers versus Nonsmokers

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Dark Adaptation and Recovery from Light Adaptation: Smokers Versus Non-Smokers

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Since the published data concerning the effects of smoking on visual sensitivity at night are inconsistent, a new study was initiated to investigate this question. Thirty Army aviators between the ages of 19 and 39 volunteered to participate in this study. Of these subjects, 15 smoked and 15 were non-smokers. Each subject was seated in a light-controlled room and exposed to a standardized bright light for 5 minutes. Immediately after the bright light was extinguished, the subject's visual sensitivity was tested by gradually increasing the intensity of a test light until the subject could see it. This was continued over a period of 35 minutes by which time the subjects had reached their maximum light sensitivity. Each subject then wore a pair of AN/PVS-5 Night Vision Goggles for 5 minutes after which his visual sensitivity again was tested for 20 minutes. Our data do not show any differences in visual sensitivity between aviators who smoke and those who do not smoke. Blood samples were analyzed to compare serum levels of nicotine, cotinine and carboxyhemoglobin with the visual data. Again, no correlation exists between.
sensitivity and blood measures related to smoking. Aviators who smoke reach the same level of sensitivity to light as non-smokers and they do so in the same amount of time. Visual recovery after wearing the Night Vision Goggles also followed the same time course regardless of smoking history. The conclusion from these data is that light sensitivity, the ability to see the dimmest lights at night, is independent of smoking history.
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The possible biological effects of smoking tobacco products have been studied intensively from a variety of different aspects. However, despite many efforts, the reported changes in visual sensory functions are inconsistent. The earlier reports indicated subjects who smoke cigarettes demonstrated reduced visual thresholds. For example, McFarland and coworkers (1944, 1953, 1970) reported a loss in visual sensitivity associated with smoking cigarettes. They used, as a measure of visual sensitivity, discrimination thresholds, i.e., the ability to detect light stimuli presented against backgrounds of various brightnesses. These authors stated that they could detect a change in the discrimination threshold if the subject smoked just one cigarette. Since no change in threshold was reported if the subjects did not inhale the smoke, the authors concluded that carboxyhemoglobin saturation was the cause of reduced visual sensitivity. They reached this conclusion after considering that nicotine still reaches the blood even without inhaling the smoke while carboxyhemoglobin saturation is not present without inhalation. In partial support of this conclusion, Sheard (1946) reported that the immediate effect of inhaling smoke was a reduction of from 0.25 to 0.75 log units in absolute light sensitivity. However, he ascribed his results to nicotine since his data indicated no effect if the nicotine was filtered from the smoke. These early reports have not remained unchallenged.

In contrast to the previous reports, Troemel, Davis and Hendley (1951) found that nicotins actually facilitated the course of dark adaptation in their subjects. Johansson and Jansson (1964) used a visual discrimination threshold and a repeated measures design to assess smoking effects and failed to show any change in thresholds after their subjects smoked cigarettes. Calissendorff (1977) also used a repeated measures design and reported a slight reduction in mesopic, but not scotopic, light sensitivity when measured after his subjects smoked cigarettes. Durazzini, Zazo, and Bertoni (1975) attempted to correlate the presence of thiocyanates in the urine secondary to smoking with several measures of visual function. These authors reported about half of their subjects demonstrated a reduction in absolute visual sensitivity thresholds after smoking cigarettes. In comparison to these investigations which have addressed primarily scotopic or mesopic function, Fine and Kobrick (1987) studied the effects of smoking on visual contrast sensitivity which is primarily subserved by the photopic system. No differences in contrast sensitivity were found in their test subjects pre- and postcigarette smoking. However, habitual smokers had slightly lower contrast sensitivities to certain spatial frequencies.
While many of the above investigations have used only a limited number of subjects or examined either the scotopic or photopic system using psychophysical procedures, Luria and McKay (1979a, b) used both psychophysical and electrophysiological techniques to assess the effects of carbon monoxide exposure on smokers and nonsmokers. Using age-matched subjects (40 smokers and 40 nonsmokers), they tested scotopic sensitivity, reaction time, color vision, visually evoked cortical potentials, and EEGs. As a group, the smokers had a poorer scotopic sensitivity score and a slower reaction time. The remaining tests in their battery did not show any differences between the two groups. Further, their results did not demonstrate any trends to indicate that a history of smoking caused a cumulative decrement in visual sensory function.

There is a growing body of evidence to indicate that smoking cigarettes can cause many different physical infirmities (US Surgeon General Report, 1979). However, the effects of smoking on visual sensory function are equivocal. A review of the published evidence presents a confusing picture. Cigarette smoking does, or possibly does not, cause a change in visual perceptual processes; if visual processes are changed, they might be enhanced or reduced. Finally, if a change occurs, the photopic, mesopic, and/or scotopic systems might be affected.

The objective of the present investigation is to determine if there are changes in scotopic sensitivity and its recovery which possibly could be attributable to chronic tobacco use. To assess this, we measured absolute scotopic sensitivity using standardized clinical testing procedures in a group of Army aviators who smoke cigarettes and compared those results with an age-matched group of aviators who do not smoke. Additional data were obtained to determine if differences exist between the two groups in recovery of absolute scotopic sensitivity after viewing a military electro-optical device (AN/PVS-5 night vision goggles).

**Materials and methods**

**Subjects**

Thirty Army aviator volunteers served as subjects for this study. Of these, 15 subjects did not smoke or use any tobacco products and 15 smoked cigarettes. All among the smoking group had smoked for more than 1 year with 11 of them having smoked for more than 10 years. Daily usage ranged from about 10 cigarettes to more than 40. The ages among the smokers ranged between 28 and 38 years (mean = 32.87 years) and among the nonsmokers between 19 and 39 years (mean = 30.20 years).
Since the purpose of this study was to investigate the cumulative rather than immediate effects of cigarettes, no attempt was made to control the subjects' smoking prior to data collection. However, the testing procedures required approximately 2 hours during which the subjects were not allowed to smoke. The testing schedule required complete data collection on two subjects daily, and subjects from the smoking and nonsmoking groups were interspersed.

An identical test procedure was followed on every subject. When the individual arrived at the laboratory, he was thoroughly briefed on the purpose of the experiment and trained on the observations required of him. He then sat in a dimly illuminated room (5.12 footcandles) for 5 minutes. Following this period, all lights were extinguished in the specially prepared dark room and the subject remained in the dark for 3 minutes. During this time, his left eye was occluded, and he positioned himself comfortably in front of the hemispherical ganzfeld of a clinical Goldmann/Weekers Adaptometer. The instrument then was turned on and the subject was light adapted by staring at the uniformly illuminated hemisphere having a brightness of 312 footlamberts. In accordance with standard clinical testing procedure, this period of light adaptation lasted for 5 minutes, after which the hemisphere lighting was extinguished and the fixation light became visible. Testing light sensitivity thresholds started immediately.

An ascending method of limits was used to measure the threshold with the subject indicating when the test stimulus became visible by tapping on the instrument table. The angular subtense of the test stimulus was 10 degrees and it stimulated a portion of the retina approximately 10 degrees below the fovea. During the first 15 minutes of dark adaptation, the threshold was measured every 15 seconds. Measurements were made every 30 seconds during the remainder of the 35 minutes.

After completing the 35 minutes of threshold testing, the subject donned a pair of AN/PVS-5 night vision goggles (NVGs). He was instructed simply to observe objects in the darkened test room using the infra-red source incorporated into the NVGs to illuminate them. The goggle output tubes provide a brightness of 0.098 footlambert and the subject was exposed to this brightness for 5 minutes. Following the 5 minute NVG exposure, the subject immediately positioned himself in the Dark Adaptometer again and threshold testing was resumed for an additional 20 minutes to assess the speed with which he recovered his absolute sensitivity.

When the psychophysical testing had been completed, the
subject was allowed to light adapt and a medical technician, using standard medical laboratory technique, took two venous blood samples. A 15 ml sample was forwarded to the Alabama Reference Laboratory which had been contracted to analyze each sample for nicotine and cotinine levels. A 7 ml sample was analyzed immediately to determine the percentage of carboxyhemoglobin (COHb).

Results

The primary results from this study are shown in Figure 1. In this figure, the changing threshold light sensitivity is graphed as a function of time in the dark. The averaged data obtained from the smoking and nonsmoking groups are practically identical. Both groups started at the same level of desensitization following the pretest bleaching exposure and achieved an approximate 4 log unit increase in visual sensitivity, demonstrating an average time to absolute sensitivity of about 28 minutes. There was an intermediate window of time (9 minutes to 24 minutes) during which the averaged thresholds from the nonsmoking group showed a very slight, and statistically insignificant, greater sensitivity.

![Figure 1. Average threshold luminance for smokers (circles) and nonsmokers (triangles) following white light bleach or night vision goggle exposure. Brackets indicate ±1 standard deviation.](image-url)
After 35 minutes of testing, the subjects used the AN/PVS-5 night vision goggles (NVGs) to view randomly around the darkened test room. The infrared light emitting diode provided in the NVGs was used as the illumination source. By doing this, the output phosphor (S20) screen had a luminance of 0.098 foot-lambert to which the subjects were exposed for five minutes. Data showing the visual sensitivity recovery from this exposure also are shown in Figure 1. Again, no differences between the smokers and nonsmokers were revealed, the two averaged curves being practically identical. After viewing with the NVGs, the subjects were reduced to about the same level of sensitivity which they previously had demonstrated at the 6-minute point during the initial testing following a more intense bleaching exposure. However, recovery back to baseline sensitivity following the NVGs exposure was much more rapid. This is shown in Figure 2 and has been reported previously (Glick, et al., 1975). Since the two groups' data were almost identical, only

![Figure 2](image)
the smokers' data are shown. In Figure 2, the initial threshold data are plotted from 6 minutes until 32 minutes. As stated previously, maximum sensitivity was reached by the 28th minute of testing. For comparison, the threshold recovery data following exposure with the NVGs also are shown in the figure. In this latter condition, threshold recovery is much more rapid, approaching the maximum sensitivity within 5 minutes after removing the NVGs.

Since the curves shown in Figure 1 represent grouped data which conceivably could mask subtle individual effects, the absolute sensitivity thresholds for each of the subjects who smoked were plotted with the results from their respective blood analyses. Figures 3A, B, and C show these thresholds plotted against the blood nicotine, cotinine, and carboxyhemoglobin results from each of the subjects. These truly are scattergrams, showing no correlation or even gross trends between visual threshold and the several physiological byproducts which presumably are related to smoking history.
Figure 3. Absolute threshold sensitivities as a function of serum nicotine (A), serum cotinine (B), and serum carboxyhemoglobin (C).
Discussion

The impetus for this investigation has been provided by considerations at the Department of the Army staff level to broaden the restrictions on smoking among Army aviators. At present, smoking is not allowed in Army aircraft during flight. A further restriction under consideration would be to not allow any smoking by Army aviators at any time, both official and personal. By this restriction, smoking tobacco could be the basis for nonselection for aviation training or removal from flight status if already rated.

A restriction on tobacco products would significantly impact the personal lives and professional careers of the affected aviators. Such a restriction should not be taken precipitously without clear indications that the use of tobacco products negatively affect military performance or endanger mission accomplishment. There are many precedents for prohibition based upon potentially compromising performance. Almost simultaneous with the dawn of aviation, the use of alcohol along with or prior to operating an aircraft has been forbidden. However, the adverse sensory and motor effects of alcohol are well-documented (Collins, et al., 1987). That is not the case with tobacco. As discussed previously, the visual sensory effects of tobacco are contradictory. Several investigations have reported a reduction in absolute light sensitivity with smoking while others have failed to show any change in threshold or even showed a facilitation. Among the investigations which have reported a visual change, the visual change has been variously photopic, mesopic, or scotopic and the effect has been attributed to nicotine or carboxyhemoglobin.

The results from the present investigation which are shown in Figure 1 support previous reports of no change in visual threshold secondary to tobacco use. The average sensitivity profiles are practically identical over the course of dark adaptation for the two test groups. In addition, exposure by the AN/PVS-5 NVGs subsequent to reaching absolute light sensitivity caused the same average visual desensitization in the smoking and non-smoking groups and the measured recovery of sensitivity occurred at the same rate. The data shown in Figure 2 are similar to comparison curves reported previously by Glick, et al. (1974).

As shown in Figure 2, recovery of visual threshold following exposure to the light output from the NVGs is much faster than recovery following the white light initial bleaching. As mentioned in the earlier report, the more rapid recovery from NVG exposure possibly can be attributed to the narrower wavelength
band of the NVG output (S20 phosphor). While this would not affect rod function, separate cone populations might be differentially influenced. Although the NVG output is quite dim (0.098 footlambert), it is definitely photopic as evidenced by the green color perception resulting. However, an equally acceptable explanation is provided by a consideration of neural versus photochemical adaptation. It is possible that the visual desensitization after exposure to the dim NVG tube is caused by a change in the neural gain of the visual system rather than a change in the bleached versus unbleached retinal photopigments. By this reasoning, the recovery would be faster because of the more rapid neural recovery rather than a change in photopigment state.

Realizing that grouped data analyses might fail to reveal subtle threshold changes among the smokers and that self-reports of smoking history would not be sufficiently reliable or quantitative, venous blood samples were taken from each subject. These samples were used to analyze sera concentrations of several contaminants resulting from tobacco use. Both carboxyhemoglobin and nicotine previously have been considered to be implicated in changes in visual function. Unfortunately, both of these have relatively short plasma half-lives and our subjects were prevented from smoking for at least two hours during the study. Therefore, we also measured cotinine, a major metabolite of nicotine, which has a much longer life (Pojer, et al., 1984). However, the results shown in Figure 3 indicate that there was no correlation between any of these products and absolute threshold in our subjects.
Conclusions

Our data indicate that there is no difference in visual function between smokers and nonsmokers when the measures of visual function are absolute light sensitivity and rate of recovery of sensitivity after light exposure. There is a growing body of evidence that use of tobacco products has a variety of negative health effects. Also, the immediate physiological consequences of smoking may or may not degrade visual perception. The present data show that there are no cumulative effects of smoking which degrades light sensitivity. Therefore, changes in visual function related to chronic cigarette smoking do not appear to provide a useful basis for prohibiting cigarette use.
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