A Psychophysiological Re-evaluation of Eysenck’s Theory Concerning Cigarette Smoking

Part II. The Autonomic Nervous System

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SUMMARY

Eysenck states that neuroticism is a function of the autonomic nervous system (ANS). In contrast to extraversion, no study undertaken by Eysenck could reveal any relationship between neuroticism and cigarette smoking.

In view of the fact that small doses of nicotine stimulate the nervous system, whereas large doses depress it, the hypothesis was tested that light and moderate smokers have a less active ANS than heavy smokers. They therefore smoke for the stimulation afforded by small doses of nicotine. Heavy smokers, however, smoke for the inhibition afforded by large doses of nicotine.

None of the autonomic variables fully confirmed this hypothesis. The results for pulse volume and heart rate were only partly reconcilable with the hypothesis. These results are interpreted in terms of the complex action of nicotine on the cardiovascular system, as well as the probability that smokers smoke primarily for the effect of nicotine on the central nervous system (CNS), and that the concomitant changes in the ANS are coincidental.


According to Eysenck, extraversion-introversion and neuroticism are the two major dimensions of personality. The former is related to the activity of the central nervous system (CNS), whereas the latter is a function of the activity of the autonomic nervous system (ANS). The neurotic is characterized by a highly labile ANS, which is aroused too easily and for much too long.

Eysenck’s theory concerning cigarette smoking states that extraverts, characterized by inhibited activity of the cerebral cortex, and therefore by inefficient cortical functioning, will have a ‘stimulus hunger’ in order to enhance cortical efficiency. Assuming that nicotine is a stimulant drug, they will therefore smoke cigarettes more than will introverts, to whom the reverse applies. A significant positive relationship was in fact found between the degree of extraversion and the number of cigarettes smoked.

In a previous report, which re-evaluated Eysenck’s theory concerning smoking and the CNS, it was indicated that his assumption that nicotine is a stimulant drug is erroneous. Rather, small doses stimulate the nervous system, whereas large doses tend to inhibit it. The results of the study indicated that light and moderate smokers probably smoke for stimulation, whereas heavy smokers probably smoke for inhibition.

As part of the previously reported study, the same hypothesis concerning the CNS was tested regarding the ANS, i.e. (i) light and moderate smokers are characterized by a less active ANS than heavy smokers; they therefore smoke for the stimulation afforded by small doses of nicotine, whereas heavy smokers smoke for the inhibition afforded by large doses of nicotine, and (ii) in association with this, the further hypothesis was investigated that non-smokers, light smokers and moderate-to-heavy smokers differ in the reactivity of their ANS to stimulation.

EXPERIMENTAL PROCEDURE

The subjects, apparatus and experimental procedure were the same as in the study previously reported. Together with the EEG, a continuous recording of the following physiological variables was also made: (i) respiration rate; (ii) pulse volume of the right middle finger; (iii) skin resistance and the galvanic skin response of the palm of the right hand and (iv) heart rate.

TREATMENT OF THE RAW DATA

For each stimulus presented, the prestimulus and response levels of activity were obtained for each physiological variable of each subject. These scores were converted to Lacey’s Autonomic Lability Scores (ALS), using the formula

\[ ALS = 50 + 10 \left( \frac{Z_s - Z_a r_{sy}}{\sqrt{1 - r_{sy}^2}} \right) \]

when a large increase in a function was to obtain a large ALS (e.g. heart rate), and the formula

\[ ALS = 50 - 10 \left( \frac{Z_s - Z_a r_{sy}}{\sqrt{1 - r_{sy}^2}} \right) \]

when a large decrease in a function was to obtain a large ALS (e.g. pulse volume).

These ALSs were mainly subjected to profile analysis by multivariate statistical methods, employing Hotelling’s T²-statistic. The raw scores for prestimulus and response levels were also analysed in detail. The mean level of activity during the rest period was also obtained for each physiological variable.
RESULTS AND CONCLUSIONS

The relationship between the number of cigarettes smoked and the activity and reactivity of the ANS did not show the same distinct pattern as did the CNS. Only the results related to pulse volume and heart rate were partly reconcilable with the tested hypothesis.

Profile analyses of pulse volume (PV) revealed that group I (non-smokers) showed significantly higher reactivity to stimuli 1 - 8 than did group III (moderate-to-heavy smokers) ($t = 2.32; P<0.05$). Group II (light smokers) also showed significantly higher reactivity to these stimuli than did group III ($t = 2.135; P<0.05$), but this did not apply to stimuli 9 - 14. Group III had a significantly higher mean PV than group I during the rest period ($t = 2.666; P<0.05$).

In order to interpret these results in terms of a possible causal relationship between PV and the number of cigarettes smoked, the PVs between stimuli and during stimulus presentation were compared with those during the rest period. For this purpose group III was, as in the previously reported study, again divided into the same 2 subgroups as for the amount of alpha brain rhythms (group IIIA — moderate smokers, smoking 12 - 18 cigarettes/day and group IIIIB — heavy smokers, smoking 20+ cigarettes/day). The mean PVs (in mm²) for each of groups I, II, IIIA and IIIIB in the intervals between stimuli, during stimulus presentation and during the rest period, appear in Table I and are graphically presented in Fig. 1.

The median test indicated that the increase in PV from non-smokers to light smokers to moderate smokers, was significant ($\chi^2 = 6.164; P<0.05$). Group IIIIB had a decrease in PV, but not significantly so. Their PV was indeed greater than that of non-smokers. (The mean PV of the non-smokers was regarded as the optimal level of functioning.)

These results could only partly confirm the stated hypothesis. In accordance with the hypothesis, light smokers (group II) probably smoke for mild stimulation (and therefore vasoconstriction). Moderate smokers (group IIIA), whose PV is further from the optimal level, will probably have to smoke more for a higher degree of stimulation to reach the optimal level, and to overcome tolerance to the action of nicotine.

The results for heavy smokers (group IIIIB) are, however, not reconcilable with the hypothesis. Their PV should be under the optimal level and they should therefore smoke more in order to reach optimal vasodilatation, but in fact their PV lies above the optimal level (although not significantly so).

TABLE I. MEAN PULSE VOLUME

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>Location of measurement</th>
<th>Group I</th>
<th>Group II</th>
<th>Group IIIA</th>
<th>Group IIIIB</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - 8</td>
<td>Between stimuli</td>
<td>0.503</td>
<td>0.729</td>
<td>1.151</td>
<td>0.855</td>
</tr>
<tr>
<td></td>
<td>During stimulation</td>
<td>0.367</td>
<td>0.459</td>
<td>0.769</td>
<td>0.673</td>
</tr>
<tr>
<td>9 - 14</td>
<td>Between stimuli</td>
<td>0.528</td>
<td>0.792</td>
<td>1.195</td>
<td>0.976</td>
</tr>
<tr>
<td></td>
<td>During stimulation</td>
<td>0.351</td>
<td>0.442</td>
<td>0.771</td>
<td>0.639</td>
</tr>
<tr>
<td>Rest period</td>
<td></td>
<td>0.659</td>
<td>0.794</td>
<td>1.150</td>
<td>0.988</td>
</tr>
</tbody>
</table>

Fig. 1. Mean pulse volume (O—O rest period; •—• between stimuli 1 - 8; △—△ during stimulation (1 - 8); ▲—▲ between stimuli 9 - 14; +++ during stimulation (9 - 14).)

Profile analyses of heart rate (HR) indicated no significant differences in reactivity between any of the groups. For groups II, IIIA and IIIIB combined, however, the Bravais-Pearson correlation between mean HR during the rest period and number of cigarettes smoked was significantly positive ($r = 0.48; P<0.05$). The mean HRs of groups I, II, IIIA and IIIIB during the rest period appear in Table II, and are graphically presented in Fig.2.

TABLE II. MEAN HEART RATE

<table>
<thead>
<tr>
<th>Group</th>
<th>X</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>74.83</td>
</tr>
<tr>
<td>II</td>
<td>65.50</td>
</tr>
<tr>
<td>IIIA</td>
<td>70.88</td>
</tr>
<tr>
<td>IIIIB</td>
<td>83.00</td>
</tr>
</tbody>
</table>

The results for heavy smokers (group IIIIB) are, however, not reconcilable with the hypothesis. Their PV should be under the optimal level and they should therefore smoke more in order to reach optimal vasodilatation, but in fact their PV lies above the optimal level (although not significantly so).
be lower than that of group II, so that they should smoke more than group II for more stimulation in order to reach the optimal level. Their HR is, however, higher than that of group II, although not significantly so.

These results can possibly be explained tentatively in terms of the complex and often unpredictable action of nicotine on the cardiovascular system. Nicotine can increase HR by activating the sympathetic or inhibiting the parasympathetic cardiac ganglia, and decrease it by inhibiting the sympathetic or activating the parasympathetic cardiac ganglia. HR is additionally influenced by the action of nicotine on the chemoreceptors in the carotid and aortic bodies, as well as on the centres in the medulla which regulate HR. Cardiovascular compensatory reflexes resulting from changes in blood pressure as a reaction to nicotine also influence HR, as does the adrenaline secretion stimulated by nicotine.

Another possibility is that smokers primarily smoke for the stimulatory or inhibitory action of nicotine on the CNS and that the concomitant changes in the activity of the ANS are coincidental. A smoker's CNS may, for instance, be in a state of inhibition, and therefore also his ANS. He now primarily smokes to stimulate the CNS. This in fact happens, but simultaneously the ANS is also activated. The relationship between activation of the CNS and ANS is, however, not perfect. Should he therefore smoke primarily for the effect of nicotine on the CNS, it could be expected that the relationship between the activity of the CNS and smoking would be more perfect than that between the activity of the ANS and smoking. This could explain the results obtained for pulse volume and heart rate, and the difficulty in obtaining a relationship between neuroticism and smoking, should such a relationship exist at all.

REFERENCES