Effects of tobacco smoking and schizotypal personality on spectral contents of spontaneous EEG

Gleb V. Tcheslavski

Bradley Department of Electrical and Computer Engineering, Virginia Tech, Blacksburg, VA 24061, United States
Department of Electrical Engineering, Lamar University, Beaumont, TX 77710, United States

A R T I C L E   I N F O

Article history:
Received 15 April 2008
Received in revised form 14 May 2008
Accepted 16 June 2008
Available online 22 June 2008

Keywords:
EEG spectral analysis
Schizotypal personality
Smoking status
Autoregressive spectral estimation

A B S T R A C T

Effects of chronic tobacco smoking on the spectral contents of spontaneous electroencephalogram (EEG) were examined in connection with schizotypal personality. EEG recorded from 19 smokers and 20 non-smokers college students were studied by means of parametric spectral estimation followed by a statistical analysis of differences. We observed that tobacco smoking affects EEG of high and low schizotypy individuals differently. Tobacco smoking may (with the probability exceeding 95%) lead to an increase in high frequency contents of EEG while evaluated for low schizotypy participants. For high schizotypy individuals, on the other hand, the most pronounced changes associated with smoking (increase of EEG power) arose in the low frequency rhythms. The most significant changes in spontaneous EEG—as attributed to smoking—were observed in the occipital region for both groups of high and low schizotypy participants. The observations are consistent with the finding reported on the schizophrenia patients and partially support the nicotine self-medication model.

Published by Elsevier B.V.

1. Introduction

Compared to other imaging techniques—such as MRI, PET, and MEG—EEG offers superior temporal resolution (except for MEG) at a significantly lower cost. Therefore, it is a convenient and yet accurate tool for the disease screening within large populations.

Quantitative analysis of the electroencephalogram (QEEG) has been used in schizophrenia research for more than 40 years resulting in a number of significant findings. In particular, patients with schizophrenia evidenced increased \( \beta \) and slow frequency powers and reduced main \( \alpha \) power compared to normal controls in majority of the studies (Morstyn et al., 1983; Sponheim et al., 2000) while others showed no differences and even opposite observations (for review, see John et al., 1994). Also, a decrease in the \( \gamma \) band synchronization to auditory stimuli in schizophrenia has been reported (Kwon et al., 1999; Light et al., 2006).

Analysis of a scalp-recorded sleep EEG showed significant decrease in coherence in both \( \beta \) and \( \gamma \) frequency bands during the wake stage in schizophrenia patients (Yeragani et al., 2006). Using both QEEG and low-resolution electromagnetic tomography (LORETA) source imaging techniques, another study (Lee et al., 2006) found that patients with auditory hallucinations (AH) had significantly increased amplitudes in both \( \beta_1 \) and \( \beta_2 \) rhythm compared with non-AH patients. Source imaging revealed significantly increased \( \beta_1 \) and \( \beta_2 \) activities in the left inferior parietal lobule and the left medial frontal gyrus in AH patients, which are the speech-related areas.

Individuals who meet the diagnostic criteria for schizotypy in young adulthood, or who show some of the symptoms, are at an increased risk for developing schizophrenia (Angst and Clayton, 1986; Wolf et al., 1991). Longitudinal studies have confirmed that schizotypy is a forerunner of schizophrenia (Chapman et al., 1994). The genetic and developmental links between schizotypy and schizophrenia suggested that they share some underlying neuropathological determinants (Walker and Gale, 1995). Thus, schizotypy can be viewed as a prodromal phase of schizophrenia. Studies on schizotypal personality among young adults will help understanding the onset mechanisms of schizophrenia. As a brain activity measurement, QEEG provides superior temporal resolution, lower cost, and easier operation compared to other techniques. QEEG studies have shown abnormalities of EEG spectrum in schizophrenia, while only few QEEG reports assessing schizotypy can be found. Kidd and Powell have reported increased left hemisphere activation among high schizotypy subjects (Kidd and Powell, 1993), Raine et al. evidence that individuals with more schizotypy showed reduced slow-wave power (\( \delta, \theta, \alpha_1 \)) over the left hemisphere (Raine et al., 2002). Therefore, one of the goals of the present study is implementation of QEEG to compare power in EEG rhythms between individuals with more schizotypal personality and individuals with less schizotypal personality.

On the other hand, cigarette smoking is strongly associated with schizotypy and schizophrenia (Adler et al., 1998; Kollaiakou and Joseph, 2000; Dinn et al., 2004). The “self-medication” model suggests that schizotypal or schizophrenic individuals smoke to reduce negative
et al., 1992; Knott and Harr, 1996; Xu and Domino, 2000) and in the administration lead to decrease in the More particularly, studies report that both acute and chronic nicotine while increasing power in the rhythm: 8–10 Hz (Domino and Matsuoka, 1994; Teter et al., 2002) while increasing power in the α rhythm: 10–12 Hz (Domino and Matsuoka, 1994; Knott and Harr, 1996; Xu and Domino, 2000; Teter et al., 2002) and in the β rhythm: 13–30 Hz (Robinson et al., 1992; Houlihan et al.; Knott and Harr, 1996; Xu and Domino, 2000). Generally, the most pronounced effects are observed in the occipital region (Domino and Matsuoka, 1994; Houlihan et al., 2001).

In this paper, we assess the chronic effect of smoking on EEG spectrum in two schizotypal groups: with low and high schizotypy. For this purpose, we utilize a parametric (Burg) spectral estimator—as a feasible alternative to traditionally used DFT-based methods—to evaluate spectra of short (and thus locally stationary) EEG fragments.

Based on the previous reports and findings, we expected that individuals with more schizotypal personality would show higher power in the low frequency EEG bands and in the α rhythm and decreased α power compared to individuals with less schizotypy. We expected that chronic smoking may lead to decrease in slow frequencies and increase in fast frequencies. Further, chronic smoking may result in more pronounced decrease in α power for individuals with high schizotypy than for individuals with low schizotypy.

2. Methods

2.1. Participants

Thirty nine strongly right-handed college students (17 men, 22 women; 17–21 years old, mean age of 18.87) participated. They were recruited from 613 online survey participants, who had been administered the Schizotypal Personality Questionnaire (SPQ; Raine, 1991), smoking history (Crawford and Wan, 2003), and medical background questionnaires (Crawford and Wan, 2003). To be selected, they must have no known history of neurological or psychiatric problems and no prescription (except birth control) or over-the-counter drugs, alcohol or illicit drugs for at least one day before the experiment.

Participants were chosen based upon scoring in the upper or lower 1/3 of the Raine’s SPQ (upper cutoff 25, lower cutoff 13). Low schizotypy group included 9 smokers and 10 non-smokers with SPQ mean score 3.33 and SD 3.08; high schizotypy group included 10 smokers and 10 non-smokers with SPQ mean score 4.05 and SD 9.47. These two groups were matched for the age and gender. Typically, the upper 10% of SPQ distributions is used as the cutoff for schizotypy (Raine, 1991). The lower cutoff (upper 1/3) used in the present study may have led to inclusion of participants high in social anxiety but low in perceptual anomalies who were arguably not true schizotypy (e.g., Claridge et al., 1996; Bergman et al., 1996). In the study, 70% participants (14 in 20 participants) in the high schizotypy group met the more stringent 10% cut-point criteria. This relatively high percentage suggests that schizotypy rather than anxiety was the dominant influence on EEG rhyme for the high schizotypy group.

Among 39 participants, 19 participants reported smoking tobacco cigarettes presently. The number of the cigarettes they smoked were from 0.5 to 20 per day (Mean=4.44, SD=5.96). Within the two groups who presently smoked, the mean smoking number per day of smokers in high schizotypy group (Mean=6.15, SD=7.49) was not significantly different from smokers in low schizotypy group (Mean=1.61, SD=2.15). 70% smokers in each group reported smoking on the average, 2 or fewer cigarettes per day. To minimize the acute effect of smoking on EEG, all smokers were asked to abstain from smoking for at least 4 h before the experiment. Exhaled Carbon Monoxide (CO ppm) was assessed before the EEG recording, with a Vitalograph Breath CO carbon monoxide monitoring device (Vitalograph Inc, Lenexa, KS). All abstaining smokers had a CO level below 10 (Mean=2.94, SD=2.46), whereas non-smokers showed considerably lower CO level, as expected (Mean=1.40, SD=0.60). The 4 hour abstinence period may result in the state of psychological smoking withdrawal, which might affect participants’ EEG. These issues are outside the scope of this paper and, therefore, are not addressed.

2.2. EEG recording

The EEG recording was conducted in the Neurocognition Laboratory at the Department of Psychology, Virginia Tech. Participants sat alone in an upright comfortable chair in the experimental room. Participants were instructed to rest with their eyes closed. Continuous EEG (0.1 to 100 Hz, 500 Hz sampling rate; gain of 150) was recorded with a cap (Electrocap Inc.), at 30 electrode sites (impedance <5 kΩ), referenced to the nose, plus vertical (above and below left eye) and horizontal EOG electrodes. Recording and digitalization were conducted with Neuroscan® SynAmps amplifier and NeuroScan® version 4.2 software. The same EEG data was previously reported by Wan (Wan et al., 2006). To minimize effects of eye movement, EEG segments of duration of approximately 60 s recorded for the participants’ eyes closed conditions were used in this study.

2.3. EEG processing

From a signal processing viewpoint, EEG is a non-stationary random process (Barlow, 1985; Bodenstein and Preatorius, 1977; McEwen and Anderson, 1975). The concept of spectrum is not defined for non-stationary processes. On the other hand, short EEG segments—of duration less than 1 s (Barlow, 1985; McEwen and Anderson, 1975)—can be considered as locally stationary and, therefore, used for spectral estimation. Assuming critically sampled EEG data with a bandwidth of 0–50 Hz, the length of locally stationary segments will not exceed 100 signal samples. The latter considerably limits applicability of DFT-based algorithms for the EEG analysis due to an increased bias of DFT spectral estimates for short sequences.

Therefore, a parametric spectral estimator was used in the present study. More specifically, discrete spectra \(P_{\text{est}}(m,\omega)\) were evaluated by autoregressive (AR) modeling for the \(i\)th EEG segment and for the \(j\)th EEG electrode. \(\omega\) denotes a frequency step in the discrete spectra, and \(m\) is the frequency index. The model’s parameters were estimated by the Burg’s algorithm (Burg, 1967). Prior the spectral estimations, EEG was re-sampled at 100 Hz and partitioned into non-overlapping 0.5 s long (50 samples) data segments. 50 consecutive segments were formed for each participant. Spectra were estimated for each segment with the AR order \(p=11\). The order was selected for the re-sampled EEG segments by performing the Akaike Information Criterion and Minimum Description Length tests (Djuric and Kay, 1992). Re-sampling procedure rejects high frequency noise (with frequencies above 50 Hz) while preserving the frequency contents of interest. DC component was also removed for each EEG segment processed.

The matrices of spectral estimates for all 30 EEG electrodes and for the \(i\)th EEG segment were formed as follows:

\[
P_i(m,\omega) = \begin{bmatrix} P_{1,i}(m,\omega) \\ P_{2,i}(m,\omega) \\ \vdots \\ P_{30,i}(m,\omega) \end{bmatrix} \tag{1}
\]

Next, vectors of average spectral powers were evaluated—for each of the following 7 EEG rhythms: \(\alpha\_0: 0–4\text{ Hz}, \beta_0: 4–8\text{ Hz}, \alpha_1: 8–10\text{ Hz}, \alpha_2: 10–12\text{ Hz}, \beta_1: 12–20\text{ Hz}, \beta_2: 20–30\text{ Hz}, \text{ and } \gamma: 30–40\text{ Hz}—as follows:

\[
x_{ij} = \frac{1}{M_j} \sum_{m=1}^{M_j} P_i(m,\omega) \tag{2}
\]

Here \(j\) indicates the EEG rhythm, \(K_j\) represents the first frequency index corresponding to the \(j\)th rhythm, and \(M_j\) is the rhythm’s...
bandwidth expressed in units of frequency indexes. Matrices of average powers we formed then as follows:

\[ X_i = \frac{1}{N} \sum_{j=1}^{N} x_{ij} \]

Finally, we form 3D matrices of average powers for each \( l \)th experimental group from the spectral matrices (3) of individuals included into the \( l \)th group:

\[ X_l = \frac{1}{N} \sum_{i=1}^{N} X_{il} \]

To investigate whether the average power in a particular rhythm is different between the two experimental groups of participants (i.e. High vs. Low schizotypy, Smokers vs. non-Smokers), statistical analysis of variances, namely the Fisher's ANOVA (Williams and Monge, 2001), was conducted. EEG processing was implemented using Matlab.

Average EEG powers followed by the ANOVA results (values of \( F \)-statistic) are represented graphically for visualization purpose.

3. Results

Differences between topographic EEG of low schizotypy (LoS) and high schizotypy (HiS) individuals were assessed first. Each experimental group, therefore, contained spectral estimates for either 19 or 20 participants, 50 estimates per individual (i.e. sample sizes were either 950 or 1000 spectral estimates per group). Fig. 1 illustrates the average EEG powers evaluated for seven rhythms and between two experimental groups: HiS vs. LoS. Values of power are represented by colors as indicated in color-bars.

We conclude from Fig. 1 that the most pronounced differences in the average power between the HiS and LoS groups are observed in

Average EEG powers evaluated for high and low schizotypy individuals.

Fig. 1. Average EEG powers evaluated for high and low schizotypy individuals.

Maps of \( F \)-statistic evaluated by one-way ANOVA between high and low schizotypy groups.

Fig. 2. Maps of \( F \)-statistic evaluated by one-way ANOVA between high and low schizotypy groups.

Average EEG powers evaluated for low schizotypy smokers and non-smokers.

Fig. 3. Average EEG powers evaluated for low schizotypy smokers and non-smokers.
the frontal lobe for the $\delta$ rhythm, in the frontal-central region in the $\gamma_1$ rhythm, and in the occipital region for all other rhythms. In all EEG rhythms—except for the $\theta$—high schizotypy experimental group is characterized by the higher—on average—powers.

ANOVA analysis, on the other hand, indicates that the most significant differences between the HiS and LoS groups occur in the $\alpha_2$, $\beta_2$, and $\gamma_1$ rhythms, which is consistent with observations reported by Raine et al. (2002).

As seen in Fig. 2, the most pronounced differences (as indicated by ANOVA) between EEG of High and Low schizotypy individuals are observed in the left hemisphere and in the $\alpha_2$, $\beta_2$, and $\gamma_1$ rhythms, which is consistent with observations reported by Raine et al. (2002).

As seen in Fig. 2, the most pronounced differences (as indicated by ANOVA) between EEG of High and Low schizotypy individuals are observed in the left hemisphere and in the $\alpha_2$, $\beta_2$, and $\gamma_1$ rhythms, which is consistent with observations reported by Raine et al. (2002).

Next, we assess spectral differences between smokers and non-smokers for the low schizotypy individuals (Fig. 3) and for the high schizotypy participants (Fig. 5).

We see in Fig. 3 that, for the low schizotypy group, smoking leads to decrease of the average power in the occipital region in the low frequency bands ($\delta$ and $\theta$ rhythms) while power of the higher bands ($\alpha_1$, $\alpha_2$, $\beta_1$, $\beta_2$, and $\gamma_1$ rhythms) increases. Also, as seen in Fig. 3, the average power at the FC3 electrode is considerably higher when evaluated for the LoS non-smokers.

ANOVA analysis indicates the most significant differences between LoS smokers and non-smokers in the $\alpha_2$ rhythm for the electrodes $O_2$ ($F = 55.8, p = 1.9 \times 10^{-11}$) and $P_2$ ($F = 50.4, p = 2.6 \times 10^{-12}$); in the $\beta_1$ rhythm for the electrodes $O_2$ ($F = 69.4, p = 3.3 \times 10^{-16}$), $F_8$ ($F = 56.1, p = 1.7 \times 10^{-15}$), $P_8$ ($F = 75.0, p = 0$), $T_P7$ ($F = 66.5, p = 1.2 \times 10^{-15}$), and $T_P8$ ($F = 76.9, p = 0$); in the $\beta_2$ rhythm for the electrode $P_6$ ($F = 61.0, p = 1.6 \times 10^{-14}$); in the $\gamma_1$ rhythm for $T_S$ ($F = 59.0, p = 4.2 \times 10^{-14}$), and $C_3$ ($F = 69.6, p = 2.2 \times 10^{-16}$). The latter is summarized in Fig. 4.

We conclude from Fig. 4 that, for the low schizotypy participants, tobacco smoking mostly affects high frequency content of EEG recorded from the occipital, temporal, and frontal–temporal regions. Next, Fig. 5 illustrates spectral differences between smokers and non-smokers among the high schizotypy participants.

As seen in Fig. 5 for the high schizotypy participants, smoking may lead to an increase of power in the $\theta$, $\alpha_2$, and $\beta_1$ rhythms while assessed in the occipital region. Average power in the $\alpha_2$, $\beta_2$, and $\gamma_1$ is higher for the non-smoking group when evaluated for the occipital region. We emphasize that this observation is nearly opposite to the one shown previously for the low schizotypy group.

ANOVA analysis indicates the most significant differences between HiS smokers and HiS non-smokers in the $\theta$ rhythm for the $O_2$ electrode ($F = 72.2, p = 1.1 \times 10^{-16}$); in the $\alpha_1$ rhythm for the electrodes $O_2$...
We conclude that subjects with more schizotypy may exhibit higher power in the α rhythm of EEG collected from the frontal region and in the rhythms α1 through γ1 of EEG acquired from the occipital region. ANOVA analysis reveals that more significant changes between high and low schizotypy individuals occur in high frequency EEG rhythms in the left hemisphere, which is consistent with previous reports (Raine et al., 2002). We also conclude that—for the low schizotypy participants—smoking may lead to a decrease of EEG power in the α and β rhythms and to an increase of EEG activity in the α1 through γ1 rhythms. Both observations are in the agreement with the previous reports (Domino and Matsuoka, 1994; Houlihan et al.; Knott and Harr, 1996; Robinson et al., 1992; Teter et al., 2002; Xu and Domino, 2000). ANOVA analysis indicates that most significant changes occur in the occipital region and in the right hemisphere. We also observed that smoking may lead among the low schizotypy individuals—to a decrease of high frequency (β2 through γ1) EEG activity evaluated at the FC1 electrode location. For the high schizotypy individuals, smoking may increase EEG power in the θ rhythm (frontal lobe) and in the θ, α, and β rhythms and to an increase of EEG activity in the α1 through γ1 rhythms. Our observations of the most pronounced changes associated with the occipital region are also consistent with the results reported previously (Domino and Matsuoka, 1994; Houlihan et al., 2001).

Lastly, ANOVA analysis reveals that, tobacco smoking may lead to significant changes of high frequency contents of EEG while evaluated for low schizotypy participants; whereas, for high schizotypy participants, the most pronounced changes arose in the low frequency EEG rhythms. Perhaps, the latter may help further revealing origins of schizophrenia and its connection with tobacco smoking.

In summary, this study indicates that tobacco smoking may have long-term effects on spontaneous EEG that are different between normal and pathological brains. The latter may be useful for functional brain modeling. Future studies may focus on long-term effects of tobacco smoking on electrical brain activity of individuals diagnosed with common neurological disorders. It would also be of interest to compare EEG spectra of the two groups from the present study (i.e., with low and high schizotypal personality) with EEG of schizophrenia patients in conjunction with their smoking status.

Acknowledgements

Author thanks Dr. Li Wan for her contributions and for sharing EEG data. Author also acknowledges the help and guidance of Dr. Helen J. Crawford of the Department of Psychology at Virginia Tech.

References


