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# Face recognition in Asperger syndrome: A study on EEG spectral power changes

Han Hsuan Yang[a], Alexander N. Savostyanov[b,c,*], Arthur C. Tsai[b], Michelle Liou[b]

[a] Fo Guang University, Taiwan, ROC
[b] Institute of Statistical Science Academia Sinica, Taipei, Taiwan
[c] Research Institute of Physiology, Siberian Branch of Russian Academy of Medical Sciences, Novosibirsk, Russia

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ABSTRACT

EEG reactions in emotional face recognition were studied in five participants with Asperger syndrome (AS) and seven control subjects. Control subjects showed a spectral power increase following the stimulus onset in two time-frequency intervals—(1) 150–300 ms in the 1–16 Hz frequency range and (2) 300–650 ms in the 1–8 Hz range. Also, alpha/beta desynchronization occurred 400–1000 ms after the stimulus onset with maximal amplitude in the posterior region. Theta synchronization (4–8 Hz) was weaker in the AS group than in the control group, but beta2 desynchronization was stronger in the AS group. The results were interpreted in terms of automatic and voluntary control of perception.



Asperger syndrome (AS) has been characterized as social and communication deficits of the type seen in autism disorders, but with relatively high verbal intelligence and cognitive abilities [32]. Individuals with AS also demonstrate cortical impairments in discrimination between stimuli, which can be found in early ERP peaks [27]. Such impairments could result from deficits in the early stage of signals perception [5,14,30]. For this reason individuals with AS have difficulty in the estimation of emotional state of other people [34]. However, they could compensate for this problem by using other perceptual strategies [15]. They could be trained to recognize the emotion of others by means of more focused attention on some special features in faces or intonations in voice.

According to [29], there are two classes of mental functions: (1) elementary (or automatic) functions executed by the evolutionary ancient brain structures and irrelevant to conscious regulation of behavior and (2) higher level functions executed by the phylogenetically new neocortical structures and connected with consciousness and speech. Later studies confirmed that the elementary and higher mental functions are connected with different brain structures [17]. Significant differences in relationships between the two kinds of functions were obtained in children and adult participants [24,26]. In pathology, the different symptoms are related with damage in the different classes of mental functions [17,26]. Automatic recognition of emotional stimuli could be damaged in individuals with AS, but this deficit could be compensated for by intensively engaging the higher mental functions.

According to evolutionary interpretation of EEG rhythms, slow-wave (delta and theta) oscillations are activities sourced from the phylogenetically ancient structures responsible for execution of automatic brain functions, whereas higher frequency rhythms (alpha and beta) are generated in the later-forming structures during execution of higher mental functions [7,20]. Delta band reflects the brain motivational activity [16], and theta band relates to working memory [18] and automatic recognition of emotions [1]. The alpha and beta activities are related with voluntary-controlled visual perception [3,6], and attention [18]. Also, alpha activity reflects the motor regulation and inhibitory control of behavior [19,24].

We compared EEG-reactions between healthy subjects and participants with AS during interpretation of photographs of emotional faces. Event-related spectral perturbation (ERSP) was applied to characterize the oscillatory activity in the control and AS groups [9]. We would expect that AS and healthy participants should demonstrate distinct ERSP patterns while performing the face recognition task [34]. In emotional face recognition, it is typical to find short-term power increases in low-frequency rhythms [21,23]. Desynchronization of alpha and beta would also occur in face recognition [8,21]. In this study, we hypothesized that low-frequency reactions in the face recognition tasks would serve as a pattern of automatic brain functions, and their amplitude be essentially lower in the participants with AS. However, the participants with AS would show stronger alpha and beta reactions as compared with healthy subjects.

EEG data were obtained for five subjects (4 males), aged 16–22 years (average 19.2) with Asperger syndrome. Diagnosis of AS was carried out by psychiatrists on the basis of Gillberg [11] and DSM-IV criteria [2] and was also confirmed by the ICD-10 criteria. All partic-

* Corresponding author. Tel.: +886 2 27835611; fax: +886 2 27831523.
*E-mail addresses:* Alexander.Savostyanov@gmail.com, a-sav@ngs.ru (A.N. Savostyanov).

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ipants with AS were patients of either National Taiwan University Hospital or Taipei City Hospital. Only one of the participants with AS was left-handed. Three of the participants with AS were hypersensitive to either sound or touch. Two participants with AS never used any pharmacological medicine for therapy of their syndrome. The other three were under medication for at least two years before the experiment. None of the participants with AS had a history of verbal language delay or the co-morbid psychiatric disorder.

For stimulation, we selected 30 photographs of five different females and five different males, with three facial expressions (angry, happy, and neutral) each, from an ensemble of photographs presented by Ekman and Friesen [10]. During the experiment, each subject was seated comfortably in a chair with eyes open in a sound insulated dimly lit chamber. The photographs were presented in black and white ($15 \times 15$ cm) via a $24.4 \times 18.3$ cm monitor located 60 cm in front of the subject. After about 12 min of spontaneous EEG registration, they were instructed to evaluate the emotional expression of each face stimulus on a continuous scale ranging from −100 (very angry) to 0 (neutral) to 100 (very happy). A fixation cross appeared at the center of the screen for 1 s before the task onset, followed by a face photograph presented for 4 s. Angry, happy, and neutral faces were delivered randomly, and the inter-stimulus-interval randomly varied between 4 and 7 s. The total number of presented faces was 30 for each subject, with 10 faces for each emotional category. EEGs corresponding to the three types of emotional faces were processed together in one data analysis.

In the pre-test of the experimental stimuli, the participants with AS showed extreme fatigue and failed to complete the test when the test lasted longer than 30 min (note: none of the pre-test takers participated in the EEG experiment). In the design of experiment, we limited the experimental session to approximately 20 min. For this reason, we could not present enough images to achieve a tolerable accuracy level for comparison of reaction to the faces from different emotionality categories. The ensemble of photographs presented by Ekman and Friesen [10], which has been validated in different countries, contains only Caucasian faces. It is known that ERP reactions to other-race faces differ from those to the own-race faces [37]. Thus far, there is no validated database of photographs with Chinese faces available for EEG experiments. Also, several experiments with healthy subjects have shown that emotionality evaluation of other-race faces is usually more difficult than that of own-race faces [28]. In our opinion, the more difficult other-race recognition task reveals the cause of the perceptual deficit in the participants with AS better than recognition of own-race faces.

EEGs were recorded using 132-channels (122 EEG, VEOG, HEOG, EKG, EMG, and 6 face muscles channels) via Ag/AgCl electrodes. The EEG electrodes were placed on 122 head sites according to the extended International 10-10 system and referred to Cz with ground at FzA. The monopolar reference scheme was applied for the montage of electrodes. The Quik-Cap128 NSL was used for electrode fixation. The electrode resistance was maintained below 5 kΩ. The signals were amplified using "Neuroscan (USA)" amplifiers, with 0.1–100 Hz analog bandpass and digitized at 1000 Hz.

Event-related spectral perturbations (ERSPs) were calculated using the *timef* function in the EEGLAB toolbox (http://sccn.ucsd.edu/eeglab/) [9]. The ERSP shows mean log event-locked deviations from baseline-mean power at each frequency. For time-frequency representation of EEG data, the wavelet transformation using the *Morlet* waveform as a mother wavelet was chosen. In addition, the ERSPs on relative power were calculated. In this case the power in narrow frequency band was related to the power in wide (1–45 Hz) band. However, the application of relative power did not improve statistical significance in comparison with absolute power. In this reason, just ERSPs on absolute power were used here.

Ongoing EEGs from 2.0 s before to 1.5 s after the stimulus onset were selected for data analyses. EEGs from 2.0 to 1.25 s before the stimulus onset were used for baseline-correction. This interval was chosen as "baseline" because it directly preceded the representation of the readiness sign (the fixation cross) before the photo onset. In other words, the "baseline" interval did not contain any task-related activity. EEGs were preliminarily band-pass filtered in 1–50 Hz using elliptic filters. Re-reference to average reference and baseline adjustment procedures were performed during data preprocessing using *pop_reref* and *pop_rmbase* EEGlab functions [9]. Baseline for each channel and each epoch was removed by subtracting the mean EEG value in the baseline time range from epoch EEG. Some trials (1–7 per person) with non-stereotyped artifacts were removed after visual inspection of EEG records. The presence of eye-blink or eye-movement artifacts was not a criterion for rejection of the episode. The 23–26 (mean 24.6; SD 1.6) trials were selected in the control group and 23–30 (mean 27.0; SD 2.7) trials in the AS group. A particular EEG-channel was reconstructed by means of the EEG-*interp* function, if there were irremovable artifacts.

Independent component analysis (ICA) was used for correcting eye blink and movement artifacts [9]. The component's weights were computed individually for each subject. The components corresponding to eye artifacts were identified by visual inspection of component sets together with VEOG and HEOG records. For each person, 5–15 components were identified as "artifactual". These components had the highest weights in the VEOG and HEOG channels and contained spectral power changes in time intervals corresponding to eye activity in occulogram. Components of artifacts were removed in the preprocessing of EEGs. After removing artifacts, we computed ERSP-indices separately for each EEG-channel and each subject.

The averaged ERSP patterns were firstly investigated separately for different groups without direct statistical comparison between groups and regions. Given a subject group, ERSPs were averaged across subjects and all channels. The random permutation method (bootstrap) with $p < 0.05$ significance level was applied in the statistical analysis of ERSPs for the healthy and AS groups. Here we used the bootstrap statistical method realized in EEGLAB toolbox (*statcond* function) and based on random data re-sampling. In this method, a surrogate data distribution is constructed by selecting spectral estimates from randomly selected samples and then averaging these. Applying this process 500 times produces a surrogate data distribution where the specified percentiles are then taken as significance thresholds [9]. This method was used to visualize significant deviations from baseline random fluctuations that were observed after stimulus presentation, such that non-significant features of the output plots were zeroed out (i.e., plotted in green).

We partitioned scalp channels into 9 regions: the left (10 channels), midline (11 channels), and right frontal (10 channels); left (17 channels), and right temporal (17 channels); and central (27 channels), left (9 channels), midline (12 channels), and right occipital–parietal (9 channels). ERSPs were averaged across channels within each region for each individual subject. The Kolmogorov–Smirnov test did not show significant deviations from normal distribution in our data. It is reasonable to apply ANOVA for testing between-group differences. The Greenhouse–Geisser correction for violation of the sphericity assumption was used whenever necessary. Repeated measures ANOVA was applied to testing the main effects of regions (9 levels) and groups (AS vs. control), as well as the interaction effect between regions and groups.

According to behavioral responses, the emotional evaluation of different categories of faces had the following averaged scores: angry faces −60.3 (SD 29.6) in the control group and −49.2 (SD 19.7) in the AS group, neutral faces −16.7 (SD 19.0) in the control group and −9.6 (SD 10.1) in the AS group, and happy faces 66.8 (SD 18.8) in the control group and 47.8 (SD 23.5) in the AS group. Emo-

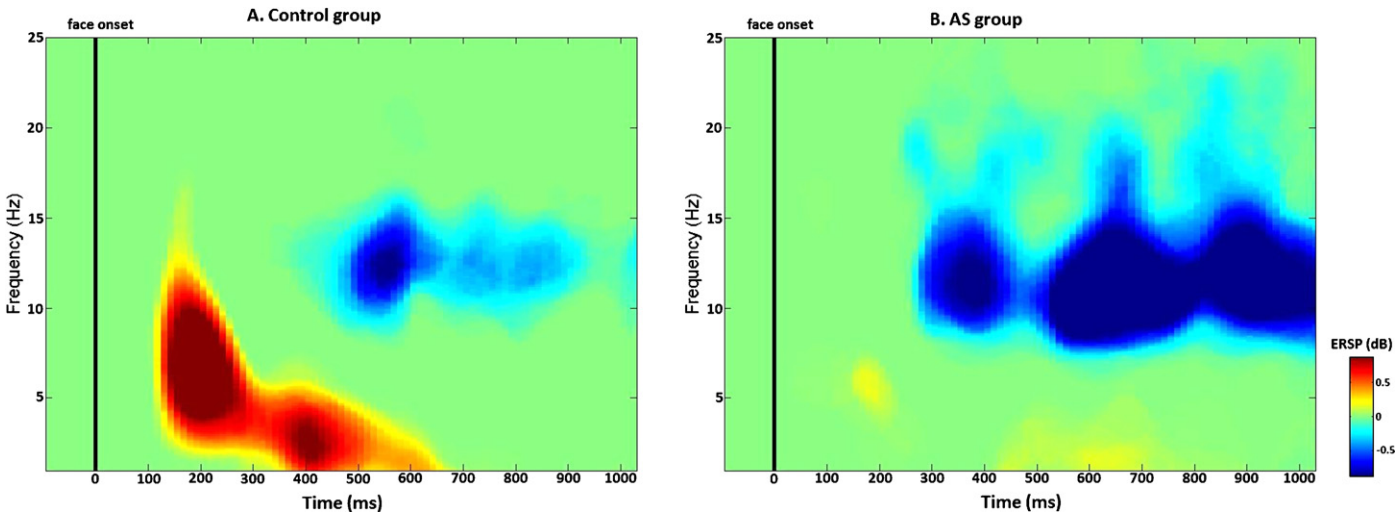


**Fig. 1.** ERSPs for the control (A, left panel) and AS (B, right panel) group. The plot shows the averaged ERSP values across channels and subjects. Only significant deviations from the mean baseline level are highlighted ($p < 0.05$). Increase of power is highlighted in warm colors, and decrease of power is highlighted in cold colors. The vertical line on the left indicates the onset time of the face stimulus.

tional evaluation for all categories was not significantly different between the two groups ($p > 0.8$). In subjective reports all AS individuals mentioned that the task was difficult for them, whereas the control subjects did not note any difficulty.

According to the ERSP pattern for the control group, there were two different time-frequency intervals with increased spectral power, and another interval with decreased spectral power. A significant decrease from the baseline in EEG spectral power (desynchronization) was found in the control group between approximately 400–1000 ms after the stimulus onset (see Fig. 1A) in the 9–16 Hz frequency range, that corresponds to boundaries of upper alpha and beta1 rhythms. The early power increase (synchronization) was found between 150 and 300 ms in the 1–16 Hz range, and the late synchronization was found between 300 and 650 ms in the 1–8 Hz range. In the AS group, significant synchronization was found in 150–250 ms in 4–6 Hz and also in 400–700 ms in 1–4 Hz, but the amplitude of synchronization in both intervals was weaker than that of the control group (see Fig. 1B). In contrast, alpha/beta desynchronization in the 8–20 Hz range was stronger in the AS group.

Three time periods (150–300 ms, 300–500 ms and 200–1000 ms) were further analyzed using repeated measures ANOVA. For the first and second periods, the frequency bands 1–4 (delta) and 4–8 (theta) Hz were chosen, and for the third time-period, the frequency bands at 8–12 (alpha), 12–16 (beta1), 16–20 (beta2) and 20–25 (beta3) Hz were chosen. ERSP values within each interval were averaged across trials and time-frequency points separately for each subject.

In the 150–300 ms time interval and the theta range (4–8 Hz), the region effect was significant ($F(8, 80) = 10.64$; $p < 0.001$). The maximum amplitude of synchronization appeared in the occipital–parietal regions. Post hoc comparisons (Fisher LSD) showed that the theta synchronization in all occipital–parietal regions significantly differed ($p < 0.01$) from all other cortical regions. The between group (control vs. AS) effect was marginal ($F(1, 10) = 3.99$; $p = 0.074$). However, in the theta band the Group*Region interaction effect was significant ($F(8, 80) = 3.46$; $p = 0.048$). The post hoc comparison suggests that the two groups only significantly differed in the left ($p = 0.052$), midline ($p = 0.052$) and right ($p = 0.020$) occipital–parietal regions. In these cortical regions, theta synchronization was stronger for the control subjects than for the participants with AS. In the delta frequency range, only the region effect was significant. Similar to the theta band, the maximum amplitude appeared in the posterior regions.

In the 300–500 ms time interval and the theta range (4–8 Hz), the region effect was significant ($F(8, 80) = 3.41$; $p = 0.033$). The maximum amplitude was located in the midline frontal region. Post hoc comparisons (Fisher LSD) showed that the theta synchronization in frontal regions significantly ($p < 0.01$) differed from all other regions. The group effect was also significant ($F(1, 10) = 5.51$; $p = 0.041$). The overall theta synchronization was stronger for control subjects than for the participants with AS. The group by region interaction was not significant ($p > 0.05$). At the delta (1–4 Hz) frequency band, only the region effect was significant, and maximum amplitude appeared in the frontal regions.

In the 200–1000 ms time interval and the beta2 range (16–20 Hz), the region effect was significant ($F(8, 80) = 6.04$; $p < 0.002$). Post hoc comparisons showed that beta2 desynchronization was strongest in all occipital–parietal regions. The group effect was also significant ($F(1, 10) = 6.36$; $p = 0.030$). The group by region interaction was not significant ($p > 0.05$). The ANOVA results for all other bands (alpha, beta1 and beta3) showed that the region effect was significant (desynchronization was stronger in the posterior regions) and group and interaction effects were insignificant, except for the interaction effect for the alpha band ($p < 0.01$). The post hoc comparison for alpha band suggests that the group difference was maximal in the posterior regions with alpha desynchronization stronger for the AS group.

A review of behavioral and neuroimaging results of facial emotion recognition in persons with autism spectrum disorders yielded mixed results [15]. According to this review some patients with ASD can successfully perform the task in spite of atypical processing of the stimuli. However, the brain compensation mechanism that allows the patients to recognize emotions is still not clear [34]. In our hypothesis, the EEG findings can reflect simultaneously the reason for abnormality in facial emotion recognition and the mechanism of compensation.

The participants with AS gave similar emotionality evaluations as their control counterparts, but their patterns of oscillatory activities differed from each other. In the subjective reports the execution of the face recognition task was difficult for participants with AS and they needed strong concentration, whereas it did not induce any fatigue in the control group. Therefore, the participants with AS were able to recognize the emotions of other people, but such

recognition was connected with additional mental effort. We have interpreted our ERSP results in terms of automatic and voluntary mental functions [17,26,29]. According to our hypothesis, AS is a result of impairments in elementary functions, which can be compensated for by more intensive use of voluntary functions.

ERSP patterns in the control group were generally in agreement with those in the literature on the face recognition task with healthy subjects [12,21–23,35]. We have observed two components of spectral power increase following the face onset. The early synchronization had the maximum amplitude in the occipital–parietal region. In face recognition, this early component was shown to be independent of face emotionality [23]. This component was tentatively interpreted as an indicator for activation of the visual system, which is connected with recognition of physical parameters in a visual image [6]. The late component was found in control subjects predominately in the anterior regions. This oscillatory activity depends strongly on the type of experimental tasks and emotionality of faces [21]. Theta synchronization depends on individual personality factors, such as gender, anxiety and aggressiveness [8,21–23]. This reaction is stronger for more emotionally sensitive persons in comparison with less sensitive ones [25]. Anterior synchronization in the low-frequency range could be interpreted as an indicator for recognition of emotionality in faces. We have also observed the desynchronization of alpha/beta rhythms with maximal amplitude in the posterior region in healthy subjects. Traditionally, this oscillatory activity has been interpreted as an indicator of participation of the visual system, which is correlated with directed attention to stimuli [3,6,19,20]. In a face recognition task, the amplitude of alpha desynchronization depends on the degree of attention to stimuli, but is independent of reaction to emotionality [8,21–23]. In our data, the frequency range of alpha desynchronization differed greatly among control subjects as observed by inspection of their individual ERSP-plots. This corresponds to the experimental findings on individual differences in alpha rhythm oscillations in the literature [19]. Based on a collection of findings on face recognition in healthy subjects [8,21–23], and functional interpretation of EEG rhythms across experimental tasks [6,19,20], the early and late slow-wave synchronizations can be interpreted as brain activities engaging automatic recognition of stimuli, and alpha/beta desynchronizations as those engaging voluntary attention.

Theta synchronization (4–8 Hz) was weaker in the participants with AS than in the control group for both the early and late reactions. In the literature, theta synchronization relating with recognition of stimuli reflects the activity in thalamus-cortical and hippocampal-cortical circuits [33]. Our findings support the hypothesis that both circuits might contain some functional disorders in participants with AS [14]. Besides, emotion-related theta synchronization is connected with activity of the amygdale [31]. In facial emotion recognition the activation of the amygdale differs between healthy subjects and persons with AS [13]. Thus, the inter-group differences in theta activity could reflect functional deficits in connections between cortex and subcortical structures that are one of main reasons for deficiencies in automatic emotion recognition [17].

The participants with AS showed desynchronization in the beta2 range that was significantly stronger as compared with the control group. A between-group difference was found in the alpha range in the occipital–parietal region, though it was less significant. As was mentioned, we observed high variability in the alpha desynchronization level in control subjects, which could also enlarge the residual error in the ANOVA test. In several empirical studies [3,19,20,36], alpha and beta desynchronization reflects the degree of attention in performing tasks. The beta desynchronization has been stronger in tasks that require verbal thinking as compared with those without verbal knowledge [36]. In facial emotion recognition, beta desynchronization was also stronger for unknown than for known faces [35]. We interpret the strengthened beta2 (and to some degree alpha) activity as an indicator of more intensive participation of voluntary attention and verbal thinking in the face recognition task in participants with AS.

A serious limitation of this study was that the number of subjects was small. The between group difference could have been significant in other frequency ranges if the number of participants with AS was large enough. It is possible that our findings on the five participants with AS simply contribute one example of many subtypes of AS, and are yet to be validated in further studies with more participants with AS and with other experimental modalities. In spite of the small number of subjects, our results of inter-group comparisons are statistically significant. Also, our results for the control group correspond to the findings that were obtained in several experiments with a large number (40–60 participants) of healthy adults [4,8,12,21–23,25]. It is possible to assume that for a relatively smaller (7 person) group of control subjects the ERSP pattern of reaction after emotional faces onset was steady enough and repeatable for different experimental groups. Besides, we have obtained the inter-group differences in the same time-frequency intervals that were most sensitive to such personality traits as a gender, age, trait anxiety, aggressiveness, and such in healthy individuals during similar experimental tasks [8,12,21–23]. Summing up, we can suggest that our findings are not casual and reflect real differences in brain activity between healthy subjects and participants with AS.

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