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The dependence of P300 amplitude on gamma synchrony breaks down in schizophrenia

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Abstract

Introduction—Auditory P300 amplitude reduction in schizophrenia is canonical and may be explained by poor synchronization or reduced power of the underlying neural activity. We asked if patients have reduced synchrony and power, and whether together with P300 amplitude, they make unique or overlapping contributions to the discrimination between patients and controls. We also asked whether people who have large P300s have higher power and greater synchrony of neural activity, and if the relationships between P300 and power and synchrony are different in patients and healthy controls.

Methods—We recorded EEG data from 22 controls and 21 patients with schizophrenia (DSM-IV) while they performed an auditory target (p=.10) detection task. We used wavelet analyses to estimate total power and synchrony of delta, theta, alpha, beta, and gamma activity in a 50ms window around the peak of the P300 to the target from the single trial data. We measured P300 amplitude from the average of the single trials, in a 50ms window around its peak.

Results and Conclusions—P300 amplitude and delta and theta synchrony were reduced in patients; delta power and synchrony better distinguished between groups than P300 amplitude. In healthy controls, but not patients, gamma synchrony predicted P300 amplitude, but delta did not. In patients, P300 and gamma synchrony are affected by independent factors; the relationship between them is attenuated by an additional pathophysiological process.

Keywords

Schizophrenia; EEG; phase synchrony; power

1. Introduction

Communication and coordination failures between different brain regions may account for a wide range of problems in schizophrenia, from psychosis to cognitive dysfunction (Phillips and Silverstein, 2003). The phenomenology of schizophrenia suggests a disturbance in integration of brain activity through loss of “inner unity” (Kraepelin, 1919) or “cognitive coordination”(Phillips and Silverstein, 2003). It has been hypothesized that many of the deficits in schizophrenia can be attributed to core abnormalities in the timing,

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synchronization, and efficiency of neural processes that bind and integrate information from different brain regions, as measured by EEG (Green and Nuechterlein 1999; Spencer et al 2004b).

EEG-based averaged event-related potentials (ERPs) recorded at the scalp are evidence of underlying synchronous activity among large assemblies of neurons firing at the same frequency(Makeig et al., 2002) The finding that the P300 component in the average ERP is reduced in amplitude in schizophrenia (Jeon and Polich, 2003) suggests that there is a deficit in the power and/or trial-to-trial synchrony of the neural activity generating the average P300. In a single trial analysis of the P300, we used a 2Hz half-sine wave as a "P300 template" and fitted it to the EEG following a target tone. We found that patients with schizophrenia have greater latency variability from trial to trial and have smaller amplitudes on each trial (Ford et al., 1994). In that analysis, we reported that even when the single trials were aligned on the peak latency, the average of these latency-adjusted trials still produced a smaller amplitude in the patients. This suggested that both the consistency of the latency of the peak across trials and power on each trial were diminished in this group.

With the advent of sophisticated EEG time-frequency analysis algorithms, we are in a position to address whether (1) P300 amplitude depends on single trial estimates of power across a wide range of frequencies (2) whether P300 amplitude depends on trial to trial phase synchrony of that activity with respect to stimulus onset across trials, and (3) whether the reduction of P300 in schizophrenia can be entirely accounted for by deficits in power or synchrony at specific frequencies. Trial-to-trial phase coherence, termed phase-locking factor (Tallon-Baudry et al., 1997) or inter-trial coherence (Makeig et al., 2004) reflects the consistency across trials of the phase of the EEG at a particular frequency, or in a particular frequency band, with respect to the onset of a stimulus. When power is extracted from the average ERP, it is called "evoked power", whereas power extracted from the single trials that go into the average ERP is called "total power". Evoked power reflects the amplitude of oscillations that are phase-locked to a stimulus event, since averaging across trials tends to cancel out non-phase locked oscillatory activity. When evoked power is removed from total power, it is referred to as "induced power" (Tallon-Baudry et al., 1997), but this practice has been questioned(Truccolo et al., 2002). Here, we report phase-locking factor and total power extracted by wavelet-based spectral decomposition of single trial EEG epochs time-locked to target stimuli presented during an "oddball" target detection task.

In this paper, we conducted two sets of analyses. In the group analyses, we compared patients to controls in the traditional time-voltage domain (P300 amplitude) and in the time-frequency domain (phase-locking factor and total power). We tested these primary hypotheses: patients will have smaller auditory target P300s than controls, the canonical finding in the literature (Jeon and Polich, 2003); patients will have reduced phase-locking factor in the frequency range of the P300 component (~3Hz), consistent with single trial analyses of P300 indicating increased cross-trial latency variability in patients (Ford et al., 1994; Roschke et al., 1996; Roth et al., 2007); patients will have reduced power at the lower frequencies, consistent with our earlier report of smaller amplitude P300s regardless of the latency variability(Ford et al., 1994). In addition, we asked whether these different measures make unique or overlapping contributions to the discrimination between patients and controls.

In the correlational analyses, we asked whether people who have large P300s have higher power and greater cross-trial phase synchrony of neural activity, and if the relationships are different in patients and healthy controls. In addition, we asked whether P300 amplitude is reflective of synchrony and power at slow frequencies (3Hz) characteristic of slow P300 itself, or whether it is reflective of synchrony and power of faster neural activity, such as

gamma band activity, invoked during complex cognitive and perceptual tasks(Miltner et al., 1999; Tallon-Baudry and Bertrand, 1999) or in the binding of sensory with executive cortical areas(Basar et al., 2001), which might be required in an auditory target detection task.

2. Results

Group Analyses

The results of the Group × Site analyses of variance can be seen in Table 1.

P300 Amplitude—As can be seen in Figure 1, P300 amplitude was significantly reduced in the patients with schizophrenia. As can be seen in Table 1, P300 was different at the different sites, having the expected Fz<Cz<Pz distribution. P300 amplitude was not affected by a Group × Site interaction.

Phase-Locking Factor and Total Power—The phase-locking factor plots are shown in Figure 2, and total power plots in Figure 3. The means extracted from these data are plotted for each frequency band in Figure 4, and the results of the results of the statistical analyses are presented in Table 1. Patients with schizophrenia have significant reductions in both phase-locking factor and power at the lower frequencies (delta and theta) that likely contribute directly to P300. That is, not only was there a reduction in the power or amplitude of these slower oscillations, their phases were less consistently synchronized to stimulus onset across trials. In the beta band at Pz, patients had reductions in phase-locking factor, but not power, suggesting they had normal power in this band, but could not synchronize it across trials. There were no group differences in phase-locking factor or power in the alpha or gamma bands.

Phase-locking factor for delta and gamma activity was affected by Site, with a Fz<Cz<Pz distribution. Phase-locking factor for beta activity was affected by a Group × Site interaction, with controls having a central maximum (Fz<Cz<Pz), but patients having a fairly uniform distribution of beta phase-locking factor across Fz, Cz, and Pz. Total power was not affected by Site or a Group × Site interaction for any of the frequencies.

We asked whether P300 amplitude, delta phase-locking factor, delta power, theta phase-locking factor and theta power made significant, independent contributions to the group discrimination. The results of the multiple logistic regression analysis appear in Table 2. Delta phase-locking factor and power made significant, independent contributions to the group discrimination, and there was a tendency for theta phase-locking factor to also contribute. With these low frequency power and phase-locking measures in the model, P300 itself did not independently contribute to the group discrimination. The overall model for accurately identifying patients had a sensitivity of 85.7% of and a specificity 90.9%.

Correlational Analyses

The correlation coefficients are listed in Table 3.

P300 vs. Phase-Locking and Power

Healthy controls: Contrary to our expectation, although P300 is predominantly a slow component, P300 amplitude at Pz is not related to 3 Hz phase-locking factor or power. However, P300 amplitude does significantly depend on phase-locking factor in the theta, beta, and gamma bands. That is, healthy controls with greater inter-trial phase-locking across a broad range of frequencies have larger P300s. Scatter plots depicting these relationships are presented in Figure 5.

Also, people with larger P300s had less alpha power, consistent with the view that alpha reflects a relatively idle state and that P300 elicitation reflects attentional engagement.

To determine the independent contributions of the different frequencies to P300, we conducted separate multiple regression analyses for synchrony and power. As can be seen in Table 3, in the phase synchrony analysis, larger P300 amplitudes were significantly predicted by greater phase-locking in the gamma band, with a trend (p=.08) toward an independent contribution from phase-locking in the theta band. In the power analysis, larger P300 amplitudes were significantly predicted by less alpha power and more theta power. We further asked whether the three phase-locking and power predictors of P300 that emerged as significant from these separate analyses would remain significant when combined in a single regression model. Results showed that gamma phase-locking continued to significantly predict P300 amplitude (p=.0001), with theta power making a marginal trend-level contribution (p=.078) and alpha power making no incremental contribution to P300 prediction.

Schizophrenia patients: None of these relationships is significant in the patients, suggesting that the normal dependence of P300 amplitude on concurrent gamma and theta activity is attenuated or absent in schizophrenia.

3. Discussion

Group Analyses

As predicted, patients with schizophrenia have smaller auditory target P300s than healthy comparison subjects. This confirms a large literature showing P300 amplitude reductions to auditory targets in patients with schizophrenia (for reviews, see Bramon et al., 2004; Ford, 1999; Jeon and Polich, 2003). We have also extended this literature to include power and synchrony of neural activity associated with auditory targets, at the latency of P300. We found reductions in both single trial power and cross-trial phase synchrony at delta and in the theta band in schizophrenia patients. Although we focused on even slower activity (2 Hz) in our previous report and used more rudimentary methods (Ford et al., 1994), reductions in cross-trial phase synchrony are consistent with the earlier findings of more single trial P300 latency variability in patients with schizophrenia, and reductions in single trial power are consistent with earlier findings of smaller single trial P300 amplitude in these patients. When P300 was considered along with 3 Hz activity as discriminators of patients and controls, both delta power and delta synchrony, but not P300, made significant independent contributions to the group discrimination. Perhaps because the average P300 is a composite of single trials that vary in both amplitude and latency, it cannot distinguish between groups as well as the separate measures of power and phase. Moreover, when deprived of the variance it shares with these 3 Hz power and phase measures, P300 amplitude is no longer sensitive to the schizophrenia effect. Similarly, Brockhaus-Dumke et al (Brockhaus-Dumke et al., 2007) concluded that phase and amplitude of single trials provides more information on auditory information processing and reflects differences between schizophrenia patients and controls better than the averaged ERP. It is important to distinguish between the delta activity evoked by a target tone in this analysis and slow wave activity in resting EEG, which is emblematic of sleep and neurological disorders.

We did not find reductions in gamma synchrony or power in patients in response to the auditory target tone, at least in the 50 ms time window surrounding the P300 peak. It is difficult to fit this into the context of the existing schizophrenia-target tone literature because of the diversity of methods used. For example, regarding synchrony, Gallinat et al (Gallinat et al., 2004) did not calculate phase locking across trials, and others have reported a hybrid measure of spatial and temporal phase locking (Haig et al., 2000). Although Spencer et al

(Spencer et al., 2007) examined early gamma (50–100 ms) phase locking across trials for standard tones, they did not examine later phase synchrony following *target tones*, limiting our ability to compare our results with theirs. Nonetheless, they found no evidence of reductions in the well-characterized early sensory "evoked" gamma response in patients. Regarding power, Gallinat et al (Gallinat et al., 2004) calculated power from both the single trials (total power) and from the average of those trials (evoked power), and removed evoked power from total power to produce a measure of "induced" power. However, because there was no increase of induced gamma activity after stimulus onset in either group, it was not reported. Importantly, they did see *evoked* power reductions in gamma in the schizophrenia patients between 220 and 350 ms following stimulus onset. Although we did not include evoked power data in this report, we calculated it and found no evoked gamma power reductions in patients (p=.52) in a similar latency window to that examined by Galinat and colleagues.

Correlational Analyses

P300 is a large, slow component, seen widely across the scalp. We suggested that its generation might depend on power and synchrony at delta, a frequency that characterizes the P300 and the lowest frequency we could resolve with our wavelet method. However, in the correlational analysis, we did not find a significant relationship between P300 amplitude and power or phase synchrony at delta. Instead, we found that controls with larger P300s had greater stimulus-locked cross-trial phase synchrony across a broad range of frequencies from 4–50Hz. A progressive series of regression analyses revealed that only gamma synchrony, but not gamma power, made a significant and independent contribution to the prediction of P300 amplitude, although there was also a trend for theta power to independently contribute to this prediction. This suggests that subjects who generate larger P300s are capable of tightly synchronizing the phase of their neural responses from one trial to the next, in the gamma band. Whether this relationship is an enduring trait or a fluctuating state is not known, although we do know that the test-retest reliability of P300 is high (.93)(Mathalon et al., 2000). Whether this ability to synchronize gamma phase across trials is associated more generally with other neurobiological variables and cognitive performance is also not known.

Although not calculating the correlation between P300 amplitude and power or phase synchrony, others have observed theta power increases following a target tone (Basar et al., 1999; Basar et al., 2001; Demiralp and Basar, 1992) and gamma power increases (Lee et al., 2007) or decreases (Fell et al., 1997; Marshall et al., 1996). Ours may be the first correlational analysis showing a correlation between auditory target P300s and gamma cross-trial phase synchrony.

We did the same correlational analysis described above in schizophrenia patients, and surprisingly, we found no relationships between P300 amplitude and synchrony or power in any of the frequencies studied. In spite of the strong correlation between P300 amplitude and gamma synchrony in controls (r=.79), and in spite of P300 amplitude reduction in the patients, gamma synchrony was not reduced in the patients. Thus, some other factor must be contributing to P300 amplitude reduction, including gray matter volume deficits, poor clinical state, or fluctuations in attention (Ford, 1999). Perhaps, one or all of these affects P300 amplitude but not gamma synchrony in patients. The breakdown of a lawful relationship among neurobiological (Ford et al., 2007) or neuropsychological (Sullivan et al., 1992) variables in schizophrenia patients is not uncommon in the field of biological psychiatry. It suggests that the variables are affected by independent factors and the relationships between them are attenuated by additional pathophysiological processes.

4. Experimental Procedure

Participants

EEG data were acquired from patients with schizophrenia (n=21) and age-matched healthy comparison subjects (n=22). All gave written informed consent after procedures had been fully described. Demographic and clinical data are summarized in Table 4. Informed consent was obtained from all subjects. This study was approved by the Human Subjects Committees at the Connecticut VA Healthcare System and Yale University.

Patients were recruited from community mental health centers, outpatient services of the Veterans Affairs Healthcare System in West Haven, and outpatient services of Connecticut Mental Health Center in New Haven. Also, some patients were recruited by Dr. Hoffman to participate in an rTMS clinical trial for auditory hallucinations. All patients in the treatment study were studied before rTMS treatment was begun. All but one patient were on stable, therapeutic doses of antipsychotic medications and met DSM-IV(American Psychiatric Association, 1994) criteria for schizophrenia based on a Structured Clinical Interview for DSM-IV (SCID; (First et al., 1995)) conducted by a clinical psychologist, or a SCID conducted by a clinically trained research assistant. No patients with a diagnosis of schizoaffective disorder are included. Patients were excluded if they met DSM-IV criteria for alcohol or drug abuse within 30 days prior to study. In addition, patient and control participants were excluded for significant head injury, neurological disorders, or other medical illnesses compromising the central nervous system. Symptoms were rated using the Positive and Negative Symptom Scale(Kay et al., 1987).

Comparison subjects were recruited by newspaper advertisements and word-of-mouth, screened by telephone using questions from the SCID(First et al., 1995) non-patient screening module, and excluded for any history of Axis I psychiatric illness.

Tasks

Subjects listened to a random series of infrequent (15%) high tones (1000Hz), frequent (70%) low tones (500Hz), and infrequent novel sounds (15%). Subjects were asked to press a response key to each occurrence of the infrequent, higher (target) tone. Target tones were 80 dB SPL and 50 ms in duration. Because the novel sounds had variable rise times and durations(Friedman et al., 1993), responses were imprecisely phase locked and were not analyzed.

EEG Acquisition

EEG data were acquired at 1000Hz from 26 sites (F7, F3, Fz, F4, F8, FT7, FC3, FC4, FT8, T3, C3, Cz, C4, T4, TP7, CP3, CP4, TP8, T5, P3, Pz, P4, T6, O1, Oz, O2), low pass filtered at 100Hz, high pass filtered at 0.05Hz, and referenced to linked ears. Additional electrodes were place on the outer canthi of both eyes and above and below the left eye to record eye movements and blinks (vertical and horizontal electro-oculogram [EOG]; VEOG, HEOG). All impedances were maintained at or below 10kOhm throughout the recording session with most EEG sites around 5kOhm.

Epochs were stimulus-locked to target onset. Individual trials were baseline corrected using the 100ms period preceding stimulus onset after correcting for eye movements and blinks using EOG data(Gratton et al., 1983). Finally, trials containing artifacts (voltages exceeding ± 100μV) at Fz, Cz, Pz were rejected. Because our hypotheses were restricted to phenomena surrounding P300, and because P300 is traditionally seen best at Fz, Cz, and Pz, we simplified our analysis by only analyzing data from those sites. These data are presented in Figure 1.

Time-Voltage Analysis

P300 was identified as the positive peak between 230 and 400ms at Pz for each subject. This peak latency defined the 50ms search window (±25 ms) for the P300 peak at Fz and Cz. The average area around the peak (±25 ms) was measured relative to the average voltage in the 100ms pre-stimulus baseline at Fz, Cz, and Pz.

Time-Frequency Analyses

The time-frequency analysis was done with a Morlet wavelet decomposition(Tallon-Baudry et al., 1997) using FieldTrip software (http://www2.ru.nl/fcdonders/fieldtrip/). Its Gaussian shape was defined by a constant ratio ($\sigma_f = f/7$) and wavelet duration ($4\sigma_t$), where f is the center frequency and $\sigma_t = 1/(2\pi\sigma_f)$. At 40Hz, the wavelet duration is over four cycles ($4\sigma_t$ = 111.4ms) with a spectral bandwidth of $4\sigma_f$ =22.8179Hz. Typical wavelet decompositions convolve the EEG signal with complex wavelets for all frequencies of interest, moving sample-by-sample in the time domain. FieldTrip achieves the same result by multiplying the Fast Fourier Transform (FFT) of the wavelet by the FFT of the EEG signal. The inverse FFT of the resultant is then adjusted so that the time course of the data corresponds to the time course of the original signal. This series of calculations progresses in 1Hz steps in the frequency domain and is equivalent (Lyons, 2004) and computationally more efficient than convolution in the time domain.

Phase-locking factor—After applying this method to every trial for the frequencies between 3–80Hz at Fz, Cz, and Pz, the phase-locking factor was calculated as 1 minus the phase variance. This calculation is identical to that used by others(Tallon-Baudry et al., 1997). High phase locking at a specific frequency and within a specific time window indicates that oscillations have become phase-synchronized across trials with respect to event onset. Plots of the phase-locking factor are presented in Figure 2.

Total Power—In addition, total power was calculated for each individual trial and for each frequency and time point; complex results of the wavelet decomposition were squared. Each time and frequency point was baseline (−100ms to −25ms) corrected by dividing that point by the average of the data points in the baseline interval for that frequency. That power quotient was then 10*log 10 transformed, returning values on a decibel (dB) scale because of the logarithmic correction for the baseline. Plots of total power are presented in Figure 3.

The phase-locking factor and total power values that were entered into the statistical analysis were extracted for each frequency of interest (Delta 3Hz; Theta: 4–7Hz; Alpha: 8–12Hz; Beta: 13–30Hz; Gamma: 30–50Hz) over a 50ms window centered on each individual subject's P300 peak latency.

Statistical Analysis

Group differences in P300 amplitude, phase-locking factor and total power at each of the 5 frequencies were assessed in 2-way ANOVAs for Group (NC vs. SZ) and Site (Fz, Cz, Pz). Interactions were parsed by running separate ANOVAs. Group was regressed on P300 amplitude, phase-locking factor, and power at Pz in a multiple logistic regression analysis to determine if each made significant and independent contributions to the group difference.

Correlational analyses were done separately for controls and patients by correlating P300 amplitude with phase-locking factor and total power, only at Pz to reduce the number of tests. To determine which frequency made the largest independent contribution to P300, all 5 frequency bands were entered into a multiple regression analysis, for phase-locking factor and total power. Those making a significant contribution were entered into a final multiple regression analysis using both power and phase-locking factor variables as predictors.

Abbreviations

EEG	electroencephalogram
ERP	event related potential
P300	positive component of the ERP at 300ms post-stimulus
PLF	Phase Locking Factor

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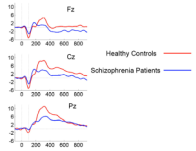

Figure 1.
ERPs to auditory targets from healthy control subjects (red) and schizophrenia patients (blue) recorded from Fz, Cz, and Pz. Negative voltage is plotted down. Time in milliseconds is on the x-axis, and voltage in microvolts is on the y-axis.

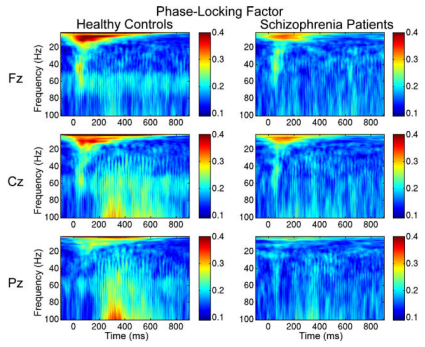

Figure 2.

Phase-locking factor plots from healthy control subjects (left) and schizophrenia patients (right) from Fz, Cz and Pz. EEG frequency is indicated on the y-axis and spans 0–100Hz. Time is indicated on the x-axis and spans −100 to 1000ms. The auditory target occurred at 0ms. Greater trial-to-trial PLF, reflecting phase synchronization with respect to target onset, is shown in hot colors, as indicated on the color scale located to the far right of each plot.

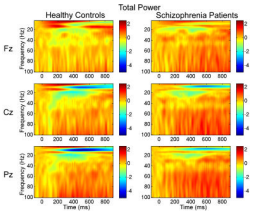

Figure 3.

Total power plots from healthy control subjects (left) and schizophrenia patients (right) from Fz, Cz and Pz. EEG frequency is indicated on the y-axis and spans 0–100Hz. Time is indicated on the x-axis and spans −100 to 1000ms. The auditory target occurred at 0ms. Greater trial-to-trial PLF, reflecting phase synchronization with respect to target onset, is shown in hot colors, as indicated on the color scale located to the far right of each plot.

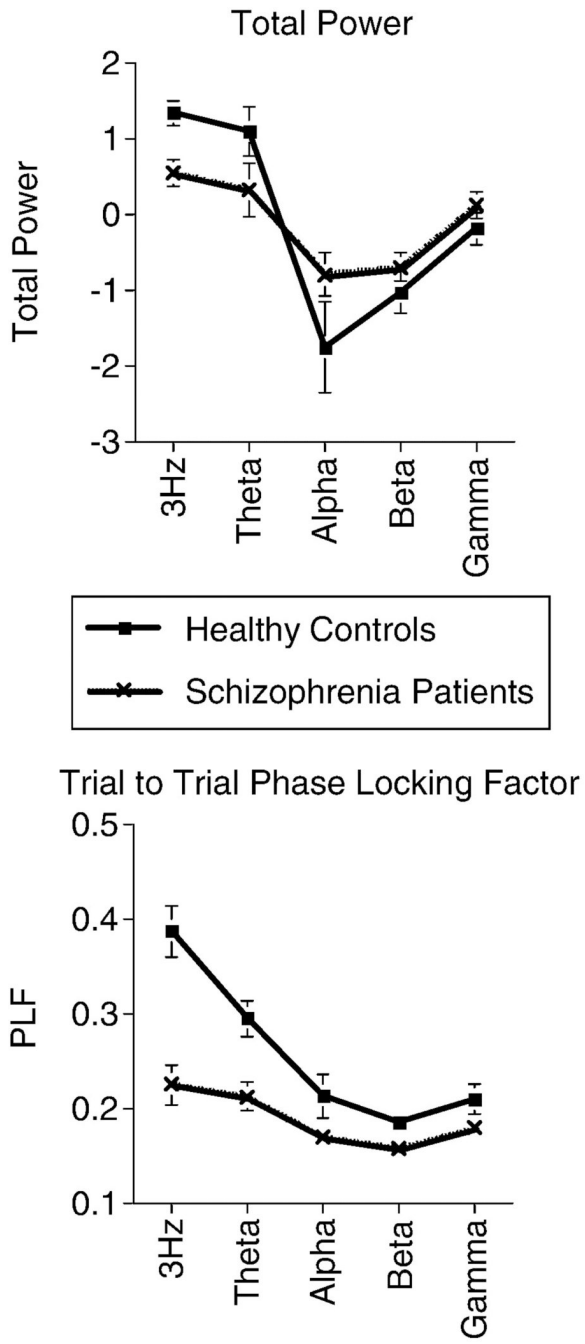

Figure 4.
Means and standard errors of the mean are plotted for Total Power (top) and Phase-Locking Factor (bottom) for each of the 5 frequency bands for both groups.

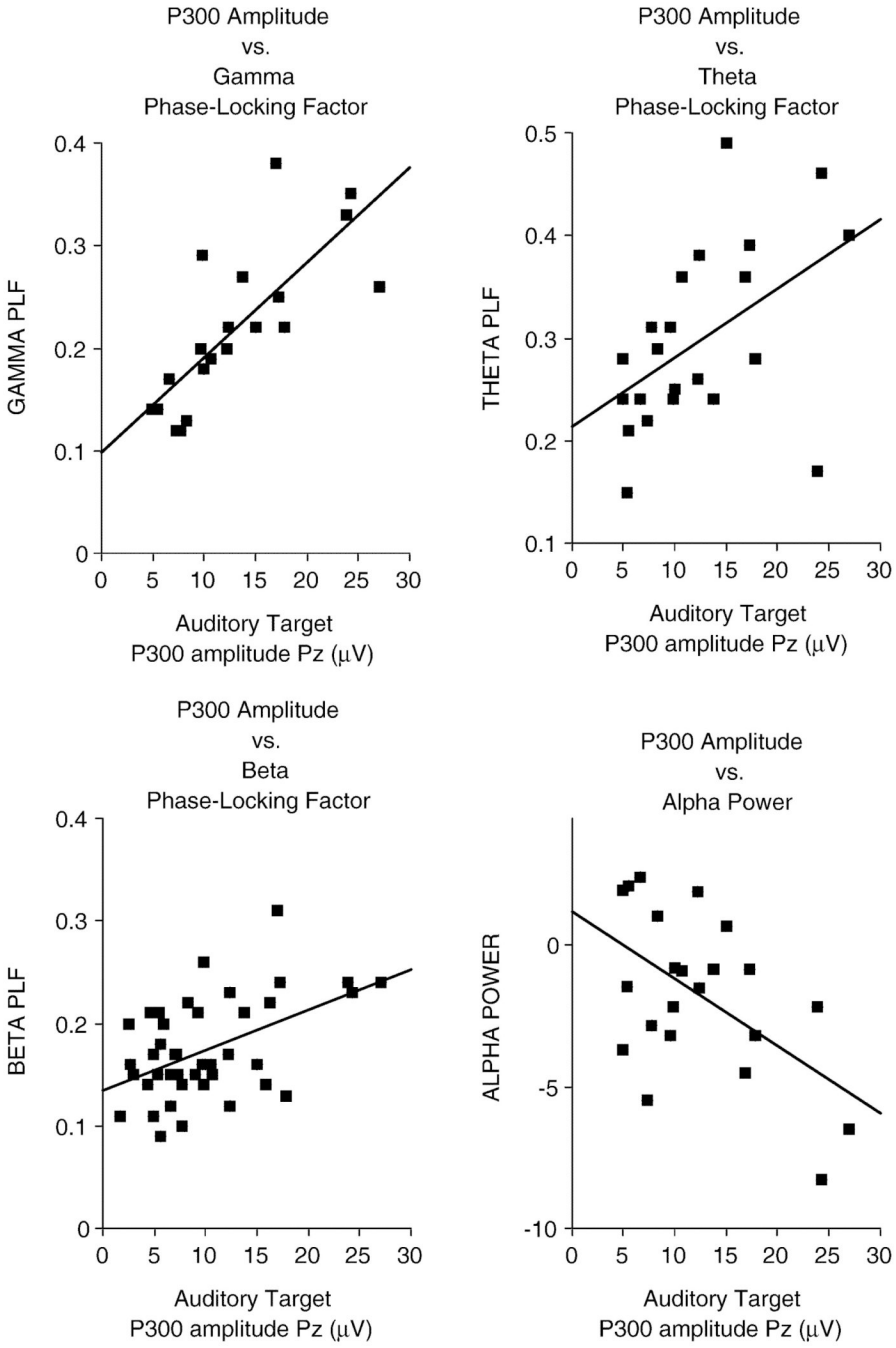

Figure 5.
Scatter plots showing the relationship between P300 amplitude (μV) at Pz and Phase-Locking Factor (PLF) for Gamma, Theta, and Beta at Pz, and Total Power for Alpha at Pz. Data are from healthy control subjects.

Table 1

Group Analyses

P300 Amplitude ANOVA											
	df	**F**	**Sig.**								
Group	1,41	10.03	0.003								
Site	2,82	77.84	0								
Group × Site	2,82	0.961	0.371								
Total Power ANOVA											
		Delta		**Theta**		**Alpha**		**Beta**		**Gamma**	
	df	F	Sig.	F	Sig.	F	Sig.	F	Sig.	F	Sig.
Group	1,41	8.941	0.005	5.06	0.03	1.89	0.18	0.76	0.39	1.23	0.27
Site	2,82	0.574	0.516	1.46	0.24	2.93	0.07	2.72	0.09	1.60	0.22
Group × Site	2,82	0.524	0.54	1.83	0.18	0.46	0.59	0.80	0.43	0.17	0.76
Phase-Locking Factor ANOVA											
		Delta		**Theta**		**Alpha**		**Beta**		**Gamma**	
	df	F	Sig.	F	Sig.	F	Sig.	F	Sig.	F	Sig.
Group	1,41	14.53	0.001	11.26	0.002	3.56	0.07	0.85	0.36	0.98	0.33
Site	2,82	10.45	0.001	2.37	0.10	1.29	0.28	4.44	0.02	8.73	0.002
Group × Site	2,82	0.024	0.951	0.17	0.83	1.39	0.26	5.13	0.01	1.33	0.26
							Fz	1.69	0.20		
							Cz	0.96	0.33		
							Pz	4.61	0.04 (nc>sz)		

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Table 2

Distinguishing Groups: Logistic Regression

Variables in the Equation	B	S.E.	Wald	df	Sig.
P300 Amplitude	−0.12	0.162	0.548	1	0.459
Delta PLF	−27.6	11.62	5.661	1	0.017
Theta PLF	−24.3	12.649	3.678	1	0.055
Delta Power	−2.73	1.346	4.098	1	0.043
Theta Power	0.213	0.747	0.082	1	0.775

Table 3

Correlational Analyses: Predicting P300 amplitude from Time-Frequency Data

Bivariate Correlations

	Controls		Patients	
	PLF	**Power**	**PLF**	**Power**
Delta	0.167	−0.3	0.087	−0.007
Theta	.498(*)	0.058	−0.119	0.033
Alpha	−0.119	−.543(**)	0.091	0.023
Beta	.514(*)	−0.153	0.082	−0.194
Gamma	.791(**)	0.125	0.285	0.034

Multivariate Correlations

Controls

PLF Model Summary

R	R Square	Adj Rsquare Std.	Error of the Estimate
.852)	0.726	0.64	3.92459

	Unstandardized Coefficients		Standardized Coefficients		sig.
	B	Std. Error	Beta	t	
(Constant)	−2.267	4.627		−0.49	0.63
Delta PLF	−3.299	7.01	−0.065	−0.471	0.64
Theta PLF	19.527	10.581	0.266	1.845	0.08
Alpha PLF	−14.328	9.871	−0.227	−1.452	0.17
Beta PLF	−0.347	29.153	−0.003	−0.012	0.99
Gamma PLF	62.69	20.005	0.731	3.134	0.006

Power Model Summary

R	R Square	Adj Rsquare Std.	Error of the Estimate
0.73	0.533	0.388	5.11673

	Unstandardized Coefficients		Standardized Coefficients		Sig.
	B	Std. Error	Beta	t	
(Constant)	11.214	3.203		3.501	0.003

Bivariate Correlations

	Controls		Patients		
	PLF	**Power**	**PLF**	**Power**	
Delta Power	−1.97	1.892	−0.212	−1.041	0.31
Theta Power	1.858	0.869	0.434	2.137	0.05
Alpha Power	−1.875	0.584	−0.819	−3.208	0.005
Beta Power	1.226	1.347	0.24	0.91	0.38
Gamma Power	1.89	1.334	0.296	1.417	0.18

PLF and Power Model Summary

	R	R Square	Error of the Estimate	Error of the Estimate
	0.832	0.693	0.642	3.91355
	Unstandardized Coefficients		Standardized Coefficients	Sig.
	B	Std. Error	Beta	t
(Constant)	−6.462	3.39		−1.906	0.073
Gamma PLF	57.863	12.262	0.675	4.719	0
ThetaPLF	24.834	12.529	0.338	1.982	0.063
Theta Power	−0.693	0.681	−0.162	−1.017	0.323

Patients

PLF Model Summary

	R	R Square	Adj Rsquare Std.	Error of the Estimate
	0.32	0.102	−0.197	4.37697
	Unstandardized Coefficients		Standardized Coefficients	Sig.
	B	Std. Error	Beta	T
(Constant)	3.527	6.513		0.541	0.60
Delta PLF	3.984	11.467	0.095	0.347	0.73
Theta PLF	−5.367	15.184	−0.092	−0.353	0.73
Alpha PLF	12.066	23.796	0.146	0.507	0.62
Beta PLF	−9.669	32.334	−0.085	−0.299	0.77
Gamma PLF	19.773	20.353	0.269	0.971	0.35

Power Model Summary

Bivariate Correlations

	Controls		Patients	
	PLF	**Power**	**PLF**	**Power**
	R	R Square	Adj Rsquare	Std. Error of the Estimate
	0.309	0.095	−0.206	4.3939
	Unstandardized Coefficients		Standardized Coefficients	Sig.
	B	Std. Error	Beta	t
(Constant)	6.16	1.685		3.656 0.002
Delta Power	−0.083	1.271	−0.017	−0.065 0.95
Theta Power	0.394	0.714	0.157	0.552 0.59
Alpha Power	0.444	0.833	0.147	0.533 0.60
Beta Power	−1.901	1.541	−0.399	−1.234 0.24
Gamma Power	1.393	1.585	0.278	0.879 0.39

Table 4

Demographics of populations studied

	Healthy Controls		Schizophrenia Patients	
Variable	**Mean**	**SD**	**Mean**	**SD**
Age (years)	37.29	12.62	39.19	10.38
p=.59				
Education (years)	16.23	2.28	13.29	2.12
p<.0001				
Parental Socioeconomic Stat	34.61	15.16	38.30	19.66
p=.50				
Mean Symptom Scores*				
Positive PANSS			16.82	4.26
Negative PANSS			16.22	4.49
General PANSS			33.39	8.15
Handedness	19 right		20 right	
	2 left		1 left	
	1 ambidextrous			
Gender	9 women, 13 men		4 women, 17	
Diagnosis			3 Undifferentiated	
			18 Paranoid	
Medication type			17 atypical, 1 typ,	