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EEG Characteristics in Males at Risk for Alcoholism

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Baseline EEG activity was recorded with eyes closed in 15 males, aged 19 to 24 at risk (HR = high risk) for the development of alcoholism and 15 matched controls (LR = low risk). Four EEG frequency bands were examined: slow alpha (7.5–10 Hz), fast alpha (10.25–12.75 Hz), slow beta (13–19.5 Hz) and fast beta (19.75–26 Hz). The HR and LR groups were compared on four measures of EEG activity for each frequency band: band power, absolute and relative area and laterality differences. Statistical analysis of the data via ANOVA revealed no significant difference between the HR and LR groups for any comparison. The results indicate that EEG measures prior to alcohol administration do not effectively discriminate between individuals at high and low risk for alcoholism.

Key Words: EEG, Alpha, Beta, Risk, Alcoholism.

ELECTROPHYSIOLOGICAL investigations of EEG activity in individuals at risk for alcoholism have been few, and their results have often been either equivocal or contradictory. Early studies of EEG activity in abstinent alcoholics revealed abnormalities such as decreased alpha activity and increased delta, theta, and beta activity.[1,2] Thus, one might hypothesize that these or other aberrations would characterize the EEG of an individual at risk. However, these studies evaluated groups of alcoholics rather than non-alcoholic individuals at risk for alcoholism. Therefore, it could not be determined whether the EEG findings preceded the development of alcoholism or were in fact a consequence of the disease.

More recently, one study[3] tested the hypothesis that children (ages 11–13) of alcoholic fathers have EEG activity characterized by a greater percentage of fast activity than the offspring of non-alcoholic fathers. While multiple frequency bands were examined, only the 18- to 26-Hz and greater than 26-Hz frequency bands showed absolute increases in activity. This pattern characterized the high risk (HR) males, as did lower EEG amplitudes in each frequency band; these amplitude differences were opposite those observed in the HR females. Based upon these results, the authors concluded that the excess fast activity preceded exposure to alcohol and argued for the heritability of this pattern. However, they also noted that they did not control for the psychiatric classification of either or both parents. While they posit that psychopathy is reflected in increases in slower frequencies in the EEG, they did not address the possible consequences of the pharmacologic treatment of the parent on the EEG in the offspring.

In another study, Pollock et al.[4] examined theta (3.51–7.03 Hz), slow alpha (7.42–9.46 Hz), and fast alpha (9.75–12.10 Hz) energy and mean alpha frequency in a population of 19- to 21-year-old high and low risk males. The subject's risk classification was based on records from psychiatric or alcoholism clinics attended by the father. Subjects at high risk for alcoholism were defined as sons of male alcoholics. Since the age of the subjects indicated they had likely been exposed to alcohol, measures of drinking history were also obtained. Neither group was found to differ significantly in the quantity of alcohol consumed during the week prior to testing. The results obtained following an ethanol challenge clearly showed that HR individuals manifested more slow alpha energy, less fast alpha energy, and a lower mean alpha frequency than did the LR individuals. In contrast, the results obtained prior to ethanol were more difficult to interpret. While the graphic display of the data indicated that HR individuals had less slow alpha energy and less fast alpha energy than did the LR individuals, the statistical significance of this difference was not indicated. The authors failed to address this point, as well as the psychiatric classification of the subjects' parents.

In two studies, Ehlers and Schuckit[5,6] examined frequencies in the fast alpha range (9–12 Hz) and the beta range (12–20 Hz) in a population of 21- to 25-year-old family history positive (FHP) and family history negative (FHN) males. A FHP male was one whose biological father was classified as an alcoholic. The former study[5] found that in the absence of an alcohol challenge, FHP subjects had more energy in the fast alpha range (9–12 Hz) than did FHN subjects. The latter study[6] found no differences between the two groups at 12 to 20 Hz. Interestingly, this study was designed to replicate one[3] in which significant differences were found only in the 18 to 26 Hz and greater than 26-Hz frequency bands; yet, the authors neither examined these frequencies nor explained their failure to do so. In this same study they also reported that FHN subjects classified as "moderate" drinkers had significantly more power in the 12 to 20 Hz range than did those classified as "low" drinkers. This relationship was not evidenced in the FHP subjects. Thus, based on the results of both studies, the authors concluded that both genetic factors and drinking history may contribute to an individual's EEG pattern.

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Lastly, Kaplan et al.[7] recorded bandwidths between 2 and 20 Hz: slow wave (2–4 Hz), theta (not defined), alpha (9–12 Hz), and fast wave activity (13–20 Hz) and found no differences in baseline EEG between FHP and FHN subjects. The subjects ranged from 20 to 28 years of age and all were social drinkers. Those in the FHP group each had a father diagnosed as alcohol-dependent (DSM III) and may have had first or second degree relatives similarly classified. In contrast, those in the FHN group had neither an alcohol-dependent father nor alcohol-dependent first or second degree relatives. An individual was rejected from either group if his mother had a history of alcohol dependence or drug abuse.

Thus, in view of the fact that few studies have examined the EEG in individuals at high risk for alcoholism, that these studies have typically focused on a limited portion of the frequency spectrum, and that the results were at times contradictory, it is the intent of the present investigation to examine both the alpha and beta frequency ranges in a carefully defined population of HR and LR individuals by employing multiple measures of EEG activity in order to determine if EEG features can be used to identify individuals at risk for alcoholism (Table 1).

METHODS

Subjects

Thirty males ranging from 19 to 24 years of age were used as subjects. High risk (HR, $N = 15$) subjects were individuals whose fathers were undergoing treatment for alcohol dependency (DSM III-R criteria). Low risk (LR, $N = 15$) subjects were either recruited via newspaper ads or were volunteers from the Health Science Center. The first stage of the screening process required each prospective subject to fill out a questionnaire detailing alcohol and drug use and the medical and psychiatric histories for both themselves and their relatives. The responses to this questionnaire determined whether an individual would be invited to participate in the study. Inclusion in the HR group required that at least the prospective subject's father be classified as alcohol-dependent (DSM III-R). A high incidence of alcoholism in the first and second degree relatives of these individuals was sought for HR subjects. However, alcoholism in one's mother was cause for exclusion from the study. Candidates for the LR group were rejected if any of their first or second degree relatives was diagnosed as alcoholic. Candidates for each group were rejected if they had major medical problems, were taking medication that affected the central nervous system, or had a history of psychiatric problems and/or drug abuse. Upon arriving in the laboratory, each subject underwent a detailed psychiatric interview focusing on questions of drug and alcohol use and the medical and psychiatric history for both themselves and their first and second degree relatives. Table 2 summarizes the characteristics of the subjects comprising each group. They were matched on the basis of age, weight, education, smoking history, marijuana use, frequency of alcohol intake, and number of drinks per occasion.

Table 1. Studies in Which Measures of EEG Activity Have Been Used to Distinguish between Individuals at Low Risk and High Risk for the Development of Alcoholism without an Alcohol Challenge

	Frequency range examined 0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30
Cohen et al. 1990	|----------|
Ehlers and Schuckit 1990[5]	|***|
Ehlers and Schuckit 1990[6]	|----------|
Gabrielli et al. 1982[3]	|----------**********|
Kaplan et al. 1988[7]	|----------|
Pollock et al. 1983[4]	|----------|

*** Indicates the frequency band in which a significant baseline difference was found between the high risk and low risk group before the ingestion of alcohol.

RECORDING METHODS AND PARAMETERS

Each subject was seated comfortably in a soundproof, temperature regulated booth (Industrial Acoustics Company). They were asked to keep their eyes closed and not fall sleep. Each subject wore a fitted electrode cap (Electro-Cap International, Inc.) using the entire 10/20 International system with the nasion serving as reference and the forehead as ground. Both vertical and horizontal eye movements were monitored.

Cortical activity was amplified 20 k (bandpass 0.1–100 Hz) via a Grass Model 12 Neurodata Acquisition System. The data were sampled continuously for 128 sec at a sampling rate of 128 Hz. According to the Nyquist Theorem, this sampling rate would not influence the frequency interval (7.5–26 Hz) examined. Artifact rejection (EOG, EMG, and saturation artifacts) was performed off-line by a highly trained technician.

DATA ANALYSIS

A 128-sec EEG record was obtained for each subject at each of 21 leads. A Fast Fourier Transform (FFT) was then performed on the first 12, artifact-free, 4-sec intervals. The resulting power spectral densities were then summed and averaged at 0.25-Hz intervals over a range from 0.25 to 63.75 Hz. The power (integrals of power density over frequency) were calculated for the following frequency bands: slow alpha (SA, 7.5–10 Hz), fast alpha (FA, 10.25–12.75 Hz), slow beta (SB, 13–19.5 Hz) and fast beta (FB, 19.75–26 Hz) at electrodes P4, P3, O2, O1. These frequency bands and recording sites were common to most of the studies evaluated.

RESULTS

Analyses of variance were used to determine whether differences in band power existed between the HR and LR groups. Comparisons were made between the two groups for the following electrode (P4, P3, O2, O1)-frequency band (SA, FA, SB, FB at 0.25-Hz intervals) combinations. No statistically significant differences were obtained between HR and LR subjects (Fig. 1).

Repeated measures multivariate analyses of variance

Table 2. Characteristics of the Individuals in the High Risk and Low Risk Groups

	High risk	Low risk
Age (years)	Mean 22, SD 2.9 Range 18–27	Mean 21.8, SD 2.6 Range 19–28
Education (years)	Mean 13.8, SD 1.6 Range 12–17	Mean 15.7, SD 2.08 Range 13–20
Drinks per occasion	Mean 4, SD 3.1 Range 0–10	Mean 2.3, SD 1.1 Range 1–5
Days per month	Mean 7.1, SD 7.8 Range 0–25	Mean 4.3, SD 3.6 Range 1–14
Number of alcoholic relatives	Mean 3.5, SD 2.0 Range 1–8	Individuals in this group could not have any alcoholic relatives

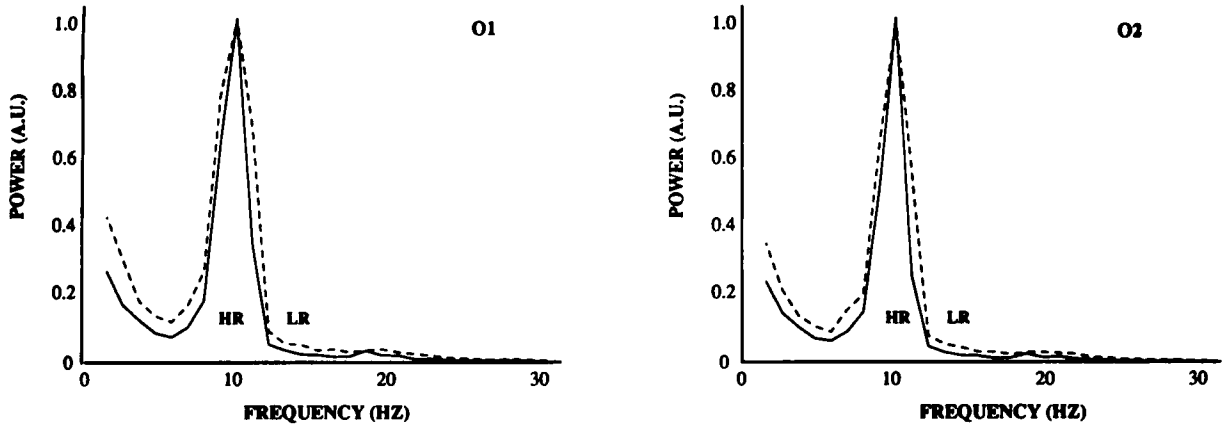

Fig. 1. The mean EEG power spectra over the 2- to 30-Hz frequency interval for both high risk individuals (HR, $N = 15$, solid line) and low risk individuals (LR, $N = 15$, broken line) obtained from electrodes O1 and O2. The sampling rate was 128 Hz and the buffer size was 128 data points.

were used to determine whether laterality differences in band power spectral densities were present in either the HR or LR groups. Therefore, comparisons within a risk group were made between electrodes P4 and P3, and O2 and O1, for each frequency band (SA, FA, SB, FB). The results indicated that there were no significant laterality differences in either the HR or LR groups.

Analyses of variance were used to compare both the absolute and relative percentages of slow and fast alpha and slow and fast beta between the HR and LR groups. Comparisons were made for each electrode (P4, P3, O2, O1)-frequency band (SA, FA, SB, FB) combination. The results indicated that there was no difference, between groups, for any of the comparisons.

DISCUSSION

This study demonstrates that over the frequency bands investigated, the EEG in individuals at high risk for the development of alcoholism does not differ significantly from the EEG in matched, low risk controls. This investigation, using measures of EEG activity that included band power, and both absolute and relative percentages of power within each frequency band, found that none of the HR-LR comparisons for each combination of parietal and occipital electrode and frequency band was statistically significant. A similar lack of significance was obtained from the within group analyses of laterality differences in band power.

There have been few investigations of baseline EEG activity without ethanol ingestion in individuals at risk for the development of alcoholism. In those studies examining baseline EEG, numerous methodological differences have often made it difficult to compare results (e.g., psychiatric classification of the subject's parents, subject's age when tested, number and placement of recording electrodes). For the frequency bands examined and measures of EEG activity used, only two studies[3,5] found significant differences between HR and LR individuals without the administration of alcohol (more power at 9–12 Hz in FHP individuals, more fast frequency beta at 18–30 Hz in HR individuals, respectively). The majority of studies[4,6,7] including the present one found no such differences.

Thus, the general failure to demonstrate a significant baseline difference between HR and LR individuals means that measures of spontaneous EEG activity may not reliably differentiate HR from LR individuals. This finding contrasts with the results of several studies in which the P300 component of the event-related potential (ERP) has been found to effectively discriminate between high risk and low risk individuals without an alcohol challenge. The significantly reduced P300 voltage has often characterized the ERP from both alcohol-dependent men[8–10] and their not as yet alcoholic male offspring.[11–15] Interestingly, all 15 of the HR individuals and 13 of the 15 LR individuals who participated in our EEG study also participated in a visual P300 paradigm.[14] The HR individuals manifested significantly lower P300 magnitudes than did their LR counterparts. However, it should also be noted that several studies have failed to replicate this finding.[16–18] Thus, while a reduced P300 component may be antecedent to the development of alcoholism, no similar result has been detected in the majority of studies for the EEG.

While the reason for this difference is not readily apparent, it may reflect the fact that the EEG is generally recorded under conditions in which the subject's task is ill-defined and there is poor experimental control over the subject's focus of attention as well as mentation. Thus, variations in the EEG appear to be more subject to random influences. In contrast, the ERP is a time-locked response to a discrete stimulus. The P300 component of the ERP has been shown to be related to stimulus variables such as task relevance,[19] unpredictability, and infrequency[20] and to subjective variables such as motivation.[21] Therefore, it is possible that the electrophysiological correlate of degree of risk may be manifested only under conditions in which specific stimulus features and cognitive processes interact to dynamically stimulate the central nervous system. Studies are presently underway to determine whether differ-

ences in EEG between subjects at risk for alcoholism and controls become apparent with alcohol administration.

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REFERENCES

1. Begleiter H, Platz A: The effects of alcohol on the central nervous system in humans, in Kissin B, Begleiter H (eds): The Biology of Alcoholism, vol 2: Physiology and Behavior. New York, Plenum Press, 1972, p 293

2. Volavka J, Pollock V, Gabrielli WF Jr, Mednick SA: The EEG in persons at risk for alcoholism, in Galanter M (ed): Recent Developments in Alcoholism. New York, Plenum Press, 1985, p 21

3. Gabrielli WF Jr, Mednick SA, Volavka J, Pollock VE, Schulsinger F, Itil TM: Electroencephalograms in children of alcoholic fathers. Psychophysiology 19:404–407, 1982

4. Pollock VE, Volavka J, Goodwin DW, Mednick SS, Gabrielli WF Jr, Knop J, Schulsinger F: The EEG after alcohol in men at risk for alcoholism. Arch Gen Psychiatry 40:857–861, 1983

5. Ehlers CL, Schuckit, MA: Evaluation of EEG alpha activity in sons of alcoholics. Neuropsychopharmacology (in press)

6. Ehlers CL, Schuckit MA: EEG fast frequency activity in the sons of alcoholics. Biol Psychiatry 27:631–641, 1990

7. Kaplan RF, Hesselbrock VM, O'Connor S, De Palma N: Behavioral and EEG responses to alcohol in nonalcoholic men with a family history of alcoholism. Neuropsychopharmacology Biol Psychiatry 12:873–885, 1988

8. Pfefferbaum A, Rosenbloom M, Ford JM: Late event-related potential changes in alcoholics. Alcohol 4:275–281, 1987

9. Porjesz B, Begleiter H, Bihari B, Kissin B: The N2 component of the event-related brain potential in abstinent alcoholics. Electroencephalogr Clin Neurophysiol 66:121–131, 1987

10. Porjesz B, Begleiter H, Garozzo R: Visual evoked potential correlates of information processing deficits in chronic alcoholics, in Begleiter H (ed): Biological Effects of Alcohol. New York, Plenum Press, 1980, p 603

11. Begleiter H, Porjesz B, Bihari B, Kissin B: Event-related potentials in boys at risk for alcoholism. Science 225:1493–1496, 1984

12. O'Connor S, Hesselbrock V, Tasman A: Correlates for increased risk for alcoholism in young men. Neuropsychopharmacol Biol Psychiatry 10:211–218, 1986

13. O'Connor S, Hesselbrock V, Tasman A, DePalma N: P3 amplitudes in two distinct tasks are decreased in young men with a history of paternal alcoholism. Alcohol 4:323–330, 1987

14. Porjesz B, Begleiter H: Event-related potentials in individuals at risk for alcoholism. Alcohol 7(5):465–469, 1990

15. Whipple SC, Parker ES, Nobel EP: An atypical neurocognitive profile in alcoholic fathers and their sons. J Stud Alcohol 49:240–24, 1988

16. Polich J, Bloom FE: P300 from normals and children of alcoholics. Alcohol 4:301–305, 1987

17. Polich J, Bloom FE: Event-related potentials in individuals at high and low risk for developing alcoholism: Failure to replicate. Alcohol Clin Exp Res 12:368–373, 1988

18. Baribeau JC, Ethier M, Braun CMJ: Neurophysiological assessment of selective attention in males at risk for alcoholism, in Johnson R Jr, et al (ed): Current Trends in Event-Related Potential Research, Suppl 40. The Netherlands, Elsevier Science Publishers, 1987, p 651

19. Sutton S, Tueting P, Zubin J, John ER: Information delivery and the sensory evoked potential. Science 155:1436–1439, 1967

20. Tueting P, Sutton S, Zubin J: Quantitative evoked potential correlates of the probability of events. Psychophysiology 7:385–394, 1971

21. Begleiter H, Porjesz B, Chou CL, Aunon JI: P3 and stimulus incentive value. Psychophysiology 20:95–101, 1983