Delta sleep inducing peptide-like immunoreactivity and sleep EEG results in schizophrenia

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Delta-sleep-inducing-peptide (DSIP) is a non-peptide that has received some attention in animal and human studies, including depressed and schizophrenic patients, and controls. Sleep EEG studies in schizophrenic patients have shown a variety of abnormalities. The purpose of this study was to explore the relationship between CSF DSIP-like immunoreactivity (DSIP-li) and sleep EEG measures in schizophrenic patients.

This report includes 15 consenting, physically healthy males (mean age 35 ± 7.0 years, range 24–45 years) studied on an inpatient research unit. Patients were maintained on a low monoamine, caffeine restricted, alcohol free diet. Three nights of polysomnography were performed when drug-free at least 2 weeks (46 ± 6 days, N = 13). Two patients were neuroleptic-naive. Lumbar punctures were performed in the morning under standard conditions after sleep studies. CSF DSIP-li was analyzed by radioimmunoassay. Sleep studies were scored visually by standard criteria. Fourteen patients remained clinically stable and four patients met criteria for psychotic relapse at the time of the studies.

CSF DSIP-li did not correlate with psychosis levels, height, weight, age, age of illness onset, or duration of illness. CSF DSIP-li correlated significantly with stage 3 sleep ($p = 0.05$), with stage 3 and delta sleep during the first Non-REM period ($p = 0.02$ and $p = 0.05$, respectively) and negatively with stage 2 ($p < 0.05$).

This is thought to be the first report of such findings. Whether the findings are relevant to the pathogenesis of schizophrenia awaits further study.

Characteristics of some auditory evoked potential (AEP) and field (AEF) responses (100 ms) in schizophrenia

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Evoked potential techniques have been used to study the characteristics and patterns of responses of the brain in schizophrenia, showing that there are both attentional and processing deficits associated with this illness. The specificity of findings often remains in question because of uncontrolled variables affecting the results, such as chronicity of mental illness, diagnostic homogeneity, specific controls by diagnostic type, medication status, fluctuation in level of attention and performance. We have attempted to examine these factors in a study of auditory evoked responses recorded by electroencephalography (EEG) and magnetoencephalography (MEG) in patients with schizophrenia and manic-depressive illness, in patients taking neuroleptics for other reasons, and in age and sex-matched normal controls. Auditory stimuli were 50 msec tones of 1000 Hz and 1500 Hz delivered in an odd-ball paradigm, wherein the subject was required to attend to and count the number of rare tones heard in a run of 100–300 stimuli. MEG and EEG recordings were made simultaneously. Data were analyzed to compare location of equivalent dipole sources for the N100 (AEP) and N100m (AEF) to the unattended frequent tones, to compare degree of background brain activity prior to and after the stimulus, to identify the response amplitude and latency variation for each individual and between groups.

There was considerable overlap in location of the equivalent current dipole of the N100 response (AEP or AEF) between all groups. The increase in variation of latency and amplitude of response in the patients with schizophrenia could not be attributed to increased variability in brain activity prior to the stimulus. Other work in our laboratory supports lack of specificity of neuroleptics to contribute to alterations in the AEPs or AEFs. These findings suggest that it is important to pursue real time analysis of brain processing in this disorder so that we may understand what areas of the brain are involved in processing sensory (auditory) information and how that processing can be affected by other inputs. (Supported by NARSAD Junior Investigator Award.)