ADD and ADHD

Neurological Basis and Neurofeedback Treatment of ADHD
Joel F. Lubar, Ph.D., and Judith O. Lubar, LCSW, BCD, April, 1995

Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.

Recent evidence based on regional cerebral blood flow, PET scan studies and quantitative multichannel EEG have shown that attention deficit disorder, particularly of the inattentive subtype has a neurological basis. Our data supports and extends these findings and provides a rationale for employing neurofeedback as a significant part of a multicomponent treatment program.

We will demonstrate the relationship between EEG changes and success in learning neurofeedback, objective measures of change and the effect of stimulant medication on the QEEG. Cerebral substrates of different types of selective attention will be covered in relation to ADD/HD and learning disabilities. The direct influence of family dynamics on the EEG and ability to change certain EEG parameters is critical for achieving long term success.

Issues relating to referential vs. bipolar recording and training and the importance of measuring phase and coherence will be discussed in relation to neurofeedback application in general.

Review of Journal Article by Joel F. Lubar, et. al., on EEG Neurofeedback Training for ADHD in Biofeedback and Self-Regulation, March 1995

Review of a New Study by Joel F. Lubar, et.al, on EEG Neurofeedback Training for ADHD (*)

by Brian Thiel, April, 1995

A new report by Joel F. Lubar, et. al, has just been published in the March, 1995 issue of the Biofeedback and Self-Regulation journal. This reports what were the effects of EEG neurofeedback as measured by two 'objective' instruments and one 'subjective' rating scale. During the summer of 1992, 23 children and adolescents received daily 1-hour sessions of EEG neurofeedback training for up to 40 total hours. The outcomes were measured using three different standard instruments, and compared with the scores using the same instruments that had also been administered pre-training. The main purpose of the EEG neurofeedback training regimen was to reduce the amplitude of theta brainwaves.
T.O.V.A. Scores
Of the 23 total subjects, 19 were measurable with pre/post T.O.V.A. scores. From this group, 12 were responsive and produced EEG changes, and 7 did not produce EEG improvements.
T.O.V.A. scores for the 12 who were EEG responsive improved on an average of 3+ of 4 possible scales. This finding is similar to the oft-reported T.O.V.A. score differences between non-med and subsequent challenge with-med. But T.O.V.A. research also shows that scores retreat to baseline as soon as the med wears off. That did not happen with these subjects. Their scores remained at the 'improved' level. The 7 of 19 who did not show improved EEG similarly did not have improved T.O.V.A. scores.

ADDES Results
The McCarney Attention Deficit Disorders Evaluation Scale (ADDES) pre/post scoring was available for 13 subjects. For this part of the study, there was no difference in the post-training scores between those who had improved EEG and those who did not. All were rated as better on this measure.

WISC-R Comparisons
Ten subjects had this analysis since WISC-R scores were desired to be from tests approximately 2 years before the training. All of the subjects in this group had improved EEG and all of them registered significant IQ increases. Neither the pre-training nor post-training testing was done by any of the EEG neurofeedback training staff.

Conclusions
The scores of the two objective measures, T.O.V.A. and WISC-R, corresponded with changes in EEG. The more subjective rating, ADDES, did not correspond with the EEG changes. The article ends, significantly I believe, with a cautious statement that EEG neurofeedback training "is a powerful adjunctive technique which is part of a multicomponent process."


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This study was done at the University of Alberta (Canada).

Establishing an EEG Norm-Base for ADD v. non-ADD


An important lack in the research about EEG effects is the dearth of data on the measurements of 'normal' brain activity while performing typical cognitive tasks. This study is intended to be a beginning point for building a baseline for both those with ADD and for those without. Sixteen 9-12 year old boys were studied, 8 with ADD but not hyperactive and 8 controls without ADD. ADD diagnoses were based on SSM-III-R.

Three well known tests (WISC-R, WRMT-R, WRAT-R) were administered to all subjects prior to the main part of the study, a series of cognitive tests performed while connected to a 19 lead EEG cap.

Findings:

- The most consistent finding was that ADD subjects have significantly higher theta amplitudes ($p < .05$) for all sites at both baseline and while performing cognitive tasks.
- There were also differences in the ratios of theta to beta and theta to SMR for baseline and all tasks at all sites, but the differences were significant only for some tasks at the parietal sites.
The raw beta and SMR amplitudes themselves were not significantly different between the two groups.

The authors conclude that although the number of subjects was small, there were significant differences that could be observed. These findings form a starter set of data for additional efforts.

Those who do neurofeedback training with ADD subjects are probably not surprised

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**Addictions-Alcoholism, Alpha-Theta Training**

Abstract of "Alpha-theta brainwave training and beta-endorphin levels in alcoholics"
Peniston EG, Kulkosky PJ, Veterans Administration Medical Center, Fort Lyon, Colorado 81038

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Abstract of "Alcoholic personality and alpha-theta brainwave training"
Eugene G. Peniston and Paul J. Kulkosky, Veterans Administration Medical Center, Fort Lyon, CO 81038 and University of Southern Colorado, Pueblo, CO 81001

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**Biogenetic Studies Relating to Neurofeedback**

*Reward Deficiency Syndrome: Electro-Physiological and Biogenetic Evidence*

Kenneth Blum, Ph.D., April 1995

Reward Deficiency Syndrome:
Electro-Physiological and Biogenetic Evidence

**Kenneth Blum, Ph.D.**

*Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.*

Drug and alcohol seeking behavior has become a great global problem affecting millions of inhabitants with a cost to society in the billions. While psychosocial modalities are essential for prevention as well as treatment of this disorder(s), demand reduction could be significantly impacted if the biological root cause was unraveled. It is in this spirit that we submit a number of important findings which might serve as examples of dissecting this most complex disease.

A most important revelation comes with the understanding that drug and alcohol seeking behavior is simply a symptom of a family of disorders which constitute the disease first termed by Blum to be "The Reward Deficiency Syndrome" (RDS).

It is further proposed that certain childhood behaviors such as conduct disorder and attention deficit hyperactivity are premorbid states which significantly increase the risk for adult drug and alcohol seeking behavior by a factor of 5.5 times the general population.

Moreover, a pattern of behaviors including aggressiveness, anger, anxiety, inability to cope with stress, low ego strength and craving behavior seem to drive the overall behavior of compulsive manifestation of self-healing. Depending on availability of a particular substance and/or biogenetic compulsive anomalous seeking of drugs, alcohol, food or other behavioral acts such as sex and gambling, the type and extent of compulsivity will manifest.
Many years of research have established that pleasure-seeking has as its physiological basis the interaction of neurotransmitters in a cascade fashion centered in the mesolimbic structures of the brain. The final pathway involves the activation of dopaminergic receptors in the N. Accumbens. It has been conjectured that at least four neurotransmitter systems are intimately involved in the so-called "Feel Good Response" (FGR).

Therefore it is conjectured that serotonergic, opioidergic, GABAergic and catacholaminergic systems breakdown of either synthesis, storage mechanisms, metabolic mechanisms, release at receptor(s) via genetic and/or environmental causes, could result in a lack of FGR. Certainly, animal research especially in the area of genetics reveal important biological substrates that seem to regulate intake of a variety of psychoactive substances through common biochemical pathways including dopaminergic activation of D2 receptors in the N. Accumbens. In this regard a number of animal models including C57 vs. DBA mice; Sardian SP vs. SNP rats; P rats vs. NP rats, and AA vs. ANA rats all show reduced D2 densities in the mesolimbic areas in the preferring vs. the non-prefering animals.

While additional cases could be made for other neurotransmitter deficits, to date, the only molecular genetic defect, in terms of association with alcohol and drug seeking behavior (as well as other compulsive or neuropsychiatric disorders such as attention deficit hyperactivity and Tourettes) which has been confirmed, are a number of variants of the dopamine D2 receptor gene (DRD2). A recent meta-analysis reveals an odds ratio for this association to be 2.18 with a p<10-7. Supportive findings also include an effective sib-pair linkage study which also confirms this association in families with a p<.002 for heavy drinking, and p<.0002 for alcoholism. Other work suggests that the variants of the DRD2 gene increase with severity of the disease (i.e., alcoholism and medical complications, cocaine addiction and family history of alcoholism, potency of the form of cocaine utilized and early deviant behavior).

It is the contention of the author that understanding the molecular genetic basis of the disease (addictive brain/compulsivity or RDS) ultimately would lead to a more targeted treatment. Moreover, through the use of certain biological predictors such as anomalous P300 waves (event-related potential) which have been shown to be lowered in children of alcoholics as well as DNA marker tests such as the DRD2 gene and other dopamine variants together could assist parents in early diagnosis of potential vulnerability to substance seeking in the future as well as other behavioral anomalies, i.e. attention deficit hyperactivity and conduct disorder.

Early diagnosis could dictate the utilization of early interventions such as neurotherapy (brain-wave training), subluxation based chiropractic, auricular therapy, cranial-electrical stimulation, nutritional supplementation (i.e., gamma-hydroxybutyrate), as well as psycho-pharmacological adjunctive therapies. By understanding the true biogenetic substrate(s) involved in craving behavior, it would significantly remove the current stigma, while eventual gene therapy (i.e., albeit a premature suggestion, replacing Taq 1 A1 allele of DRD2 gene with A2 DRD2 gene) among other novel modalities, would lead us toward a solution and would ultimately support the disease "precept" of the "biogenic paradigm."

A paper of similar title by Kenneth Blum, John G. Cull, Eric R. Braverman and David E. Comings, 'Reward Deficiency Syndrome' was published by the American Scientist, March-April 1996. The publishers have made it accessible to the internet as either an abstract or as a full text version including its several graphic figures. (The full text version is a very large file, over 100KB, and it may take quite a while to load.)
Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.

EEG feedback for quieting the cortex is helping people nowadays discover things about themselves that in previous decades were either hidden in the 'unconscious' or not manifested without yogic training. Theta feedback, for instance, can be used for becoming aware of subconscious and superconscious aspects of mind in the same way that vipassana, a form of Buddhist meditation, is used.

Vipassana, described as 'mindfulness', is a meditation method in which the 'witness', or 'observer' aspect of mind develops. When mindfulness is achieved, the 'I' of a person is capable of watching what is happening in body, emotions, and mind without reacting. Instead, the 'I' acts in whatever way seems useful from its transpersonal point of view, which in Zen is called the point of view of the True Self. Experimentally, theta feedback and vipassana lead to states of 'mindfulness' that are so much alike that theta training might, without exaggeration, be called 'instrumental vipassana.'

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**Epilepsy**

Remediation of Nocturnal Seizures by EEG Biofeedback
Jonathan Walker, M.D., Neurologist, Dallas, TX, May, 1995

**Remediation of Nocturnal Seizures by EEG Biofeedback**
Jonathan Walker, M.D., Neurologist, Dallas, TX

Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.

Two cases of epilepsy are presented with seizures occurring only at night.

The first case was a 34 year old woman with post-traumatic seizures dating from early childhood. Her seizures always occurred after going to bed at night, either while falling asleep (20%) 3-5 hours, or after falling asleep (80%). She would feel fatigued and confused for three days afterwards. One awake electroencephalogram (EEG) revealed spiking in the left fronto-temporal region. Quantitative EEG (QEEG) revealed an increase in absolute power of theta over the entire scalp except for the right mid-temporal region (consistent with residual effects of a closed head injury). Following sessions of EEG biofeedback (reward 12-15 Hz, reward inhibit of 7-12 Hz, and above 18 Hz), at Fz the patient experienced a marked reduction in seizure frequency and severity, as well as a disappearance of the postictal confusion and tiredness.

The second case was a 22 year old woman with post-traumatic seizures beginning at age 13, beginning with jerking in both hands and proceeding to loss of consciousness. Her original EEG revealed bilateral posterior spike and wave activity in response to 10 Hz photic stimulation. Her seizures were originally controlled on Depakote and it was discontinued at age 17. She had a psychotic break at age 19 and was placed on Haldol with recurrence of her grand mal seizures, which typically occurred early in the morning (on awakening). Her mental state and generalized seizures stabilized on Lithium and Depakote, but she continued to hear voices on going to bed at night several times per week. Training was carried out at the same parameters as first case for 46 sessions, then switched to reward 15-18 Hz for last 11 sessions. By completion of training she was off all medication, was seizure-free, hallucination-free, was no longer seeing her psychotherapist, and was no longer depressed or anxious.

Both patients have maintained their improvement for over six months.
Resolution of nocturnal seizures suggests that the effect of EEG biofeedback in reducing seizures does not completely rely on conscious brain mechanisms. The second case suggests that EEG biofeedback may play an important role in remediating psychoses associated with epilepsy.

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Exceptional Results with 'Exceptional Children'
Lynda Thompson, Ph.D. and Michael Thompson, M.D., Mississauga, Ontario, Canada

This paper will describe the effects of combining neurofeedback training with instruction in metacognitive strategies on a group of 15 boys between the ages of 8 and 14 who presented with a combination of: severely inappropriate, immature, social interactions, Attention Deficit Disorder and Learning Disabilities. The emphasis in neurofeedback training was to encourage changes in their brain wave profiles toward a more mature, age appropriate pattern by decreasing the initial predominance of slow wave activity (usually 4-8 Hz), decreasing the variability of these slow waves, and increasing fast wave (13-15 and/or 16-20 Hz) activity. Metacognitive strategies taught depended on the individual child's initial academic levels and usual emphasized skills for improved reading.

A number of authors have noted improvement in the symptoms of ADD and academic functioning using neurofeedback; this pilot project has the unique feature of gathering data on severely socially impaired children who also exhibited the symptoms of ADD and LD.

The interesting observation was that the most important variable to the families, the social behaviour of their children, was the first major positive change to be noted. Only children with very severe social impairments were chosen for inclusion in this study. Nine of these children met all the criteria for Asperger's Syndrome and seven of this group had moderate to severe symptomatology. Asperger's Syndrome is a neuropsychiatric disorder who's symptoms picture rarely changes with traditional approaches so this report of improvement following training with neurofeedback combined with metacognitive strategies is of particular interest.

Parental, school and trainer reports of social behaviour, academic and intellectual testing, school report cards and feedback from parent-teacher interviews, ADD check-lists, and the T.O.V.A. were the measures used. All fifteen boys demonstrated improvements in all areas of functioning. 13 are now off stimulant medications (2 remain on low dose for specific situations only) and all improved in academic functioning by at least a year's growth on standard tests in their first six months of training (some showed as much as 5 year's growth in grade equivalent scores.) However, the most dramatic and somewhat unexpected change was the shift toward normalization of their social interactions.

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Autism/Asperger's/Obnoxious Child, 3 Case Histories
How We Get Positive Results with Complex ADD Clients
Lynda Thompson, Ph.D. and Michael Thompson, M.D., Mississauga, Ontario, Canada

Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.
Three brief case histories on the scale of primarily organic to primarily psychologically based social-behavioural disorders are described. All three children and their families, previous to coming for neurofeedback, had had very considerable intervention both from the school system and from other clinical resources. All previous efforts from other clinical resources had minimal benefit.

This paper presents a simple, theoretical understanding of the mechanism by which coping styles characteristic of specific stages of early development can shift and normalize if the clinician understands the basic principles which underlie these behaviours and combines a positive learning environment to counter the negative assumptions of "self" with neurofeedback which allows the child to settle, focus, and perhaps to neurologically mature.

The term, coping style, rather than, disorder, is used to reflect the opinion that the child's behaviour constitutes an attempt to find a solution to difficulties encountered during the normal course of psychosocial development and that these styles of relating are not static. The coping styles, for the most part, arise from partial blocks to the stages of normal development in the first three years of life. In some children, the Autistic and the Asperger's syndrome, the block may be due to neurological immaturity, particularly in the social development arena and in the ability cognitively for inferential thinking and abstraction.

We are asking ourselves if neurofeedback could be accelerating neurological maturation in these children? This represents an avenue for further research. In addition to remarkable academic gains, these three children now are developing friendships and are socially not being handled differently in school than other children and do not require special behaviour management at school.

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**Immune Diseases**

**Neurofeedback and Immune Response (HIV)**
Gary J. Schummer, Ph.D., M. Crane, L. Wong, C. Aguirre, May, 1995

**Neurofeedback and Immune Response (HIV+)**
Gary J. Schummer, Ph.D., M. Crane, L. Wong, C. Aquirre

Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.

A pilot study titled, "The effect of alpha/theta neurofeedback and alpha-stim treatment of immune function, physical symptoms and subjective stress within a group of HIV+ subjects, a controlled study" is in the process of being completed.

We investigated the effects of neurofeedback training (8-12 Hz, 6-8 Hz) and Alpha-Stim therapy on 40 subjects with HIV. The goal of the study is to document previously observed clinical changes and to justify the utility of further investigation. Effects were measured over four months by changes in blood panels (T-4 helper cells), k physical symptomatology (SCL-90) and subjective measures of stress.

To date, in 10 subjects given neurofeedback therapy only, the group averaged 31% increase in T-4 level. In 10 subjects given neurofeedback and Alpha-Stim, the group averaged 34% increase in T-4 level. All the subjects reported a decrease in physical symptoms and subjective stress within the first month of the study.

The research is currently being completed and will have a control group and Alpha-Stim only group (10 subjects each.) Since the results thus far have been extremely compelling in favor of positive immune modulation with neurofeedback, the authors would like to offer the preliminary data to interested researchers and clinicians so they may replicate the study and/or utilize the protocol in clinical practice. At
present, the authors are analyzing the data and will be prepared to discuss both the preliminary results of this research as well as to offer a theoretical model for why the immune system would be amenable to neurofeedback.

**Neurofeedback and Lyme Disease: A Clinical Application of the Five Phase Model of CNS Functional Transformation**
Valdeane W. Brown, Ph.D., Hermitage, PA, May, 1995

**Neurofeedback and Lyme Disease: A Clinical Application of the Five Phase Model of CNS Functional Transformation**

Valdeane W. Brown, Ph.D., Hermitage, PA, May, 1995

*Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation.*

Neurofeedback has generated enormous interest recently due to its demonstrated efficacy with ADD, PTSD, and Substance Abuse, and especially now in regards to pioneering efforts of using it with immune system involved disorders like Lyme Disease and CFIDS. However, a major problem in the rapidly emerging field of neurofeedback is the wealth of unintegrated clinical and research findings due to the lack of a systematic approach to implementing this approach across a range of disorders.

While intriguing bits of clinical wisdom float about chaotically, practitioners have little sense of how to relate these various nuggets into a comprehensive approach to restoring and optimizing CNS function. SMR augmentation appeals to many practitioners, whereas others prefer variants of the original Alpha/Theta or Beta/Theta protocols.

In this presentation a Five Phase Model of CNS Functional Transformation is described through its application to working with individuals suffering from Lyme Disease. A step-by-step framework for restoring function to these individuals is described in detail, with indications of how the comprehensive framework of the Five Phase Model can be use effectively to organize the triage and treatment of the entire range of disorders and foci of treatment, including ADD, anxiety, depression, substance abuse, PTSD, and peak performance.

**Multiple Sclerosis**

Remediation of Neurologic Deficits in Patients with Multiple Sclerosis by EEG Biofeedback
Jonathan Walker, M.D., Neurologist, Dallas, TX, May, 1995

**Remediation of Neurologic Deficits in Patients with Multiple Sclerosis by EEG Biofeedback**
Jonathan Walker, M.D., Neurologist, Dallas, TX

*Abstract of a presentation at the 1995 Annual Conference of the Society for the Study of Neuronal Regulation*

Multiple sclerosis is a demyelinating disease of the central nervous system. Lesions may occur in the white matter of the cortex, cerebellum, brain stem, or spinal cord. Lesions which have been stable for many years
are associated with glial scarring ("sclerosis") and have generally been considered irreversible, at least with regard to drug therapy.

We report here cases in which EEG biofeedback training was associated with remediation of deficits which had been present and stable (no improvement or worsening over several years' training including diplegia, spasticity, gait ataxia, and extremity ataxia.)

EEG biofeedback appears to offer a potential method of reversing stable deficits in multiple sclerosis patients who are not likely to benefit from therapy with drugs.