



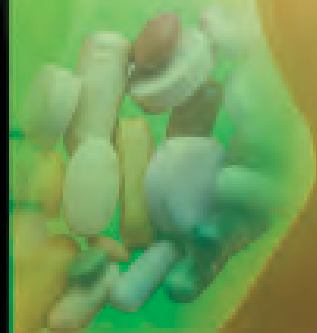
NEURO CONNECTIONS



April 2007

Newsletter

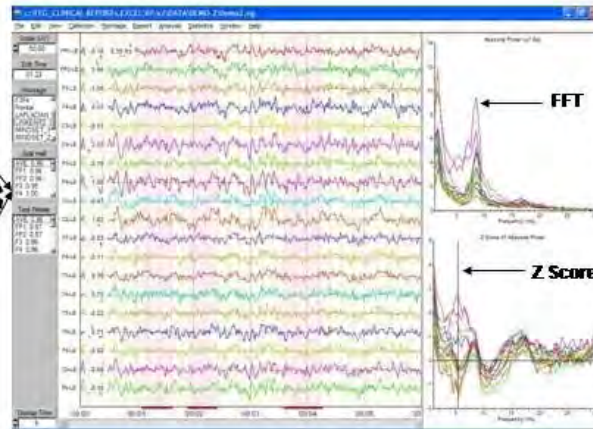
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A joint newsletter from the  ISNR
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NeuroConnections is published four times a year and will consider all materials pertaining to the practice and/or promotion of neurofeedback.

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LETTER FROM THE ISNR PRESIDENT

It is always a pleasure to write about good news, and thanks to the cooperative efforts of many of our members and board members we have some nice news about our society to share and some ideas on which we would like you to comment.

A lot of the new projects have come to fruition and will enhance the society's everyday function and will make our Journal, our membership drive, and our future conferences more efficient and productive.

You have probably visited our new website and seen the changes in format and content. It is still a work in progress but it has already been effective. As you have already discovered, it is allowing us to advertise our conference in a more attractive format. This has already translated in some new members who have asked to be registered for the conference. I understand that this is the earliest that we have had requests for registrations.

Our two program chairs, Jay Gunkelman and Hank Weeks and the program committee have done a great job in getting interesting and well-known speakers. Many of them are European researchers, some of them we have heard before but they have

different and very interesting new research to present. Others, both Europeans and Americans, are bringing us the cutting edge of neuroscience research. Information about them is being posted on our website as they confirm their presentations.

I do not know how many of you are aware of this, but we are having a second generation of neurofeedback practitioners and researchers entering and contributing to the field. James Thomson, Ph.D. candidate at Penn State will present his dissertation research on head injury in athletes. We encourage our other student members and potential members to submit presentations and posters for the 2007 conference.

This year we have received several excellent research proposals that we funded. We are receiving requests for research at prestigious universities as well as research that will answer some fundamental questions about the effectiveness of neurofeedback. As a result we have thought of some possible funding venues for putting together some projects that would make more money for the fund. We would like to hear your opinion on how effective any of these proj-

ects would be. Think about your reaction to them and that of other members you know and please share them with us at annemarie@isnr.org.

One of them is raffling a cruise for two. The second is a golf tournament that the Research Committee could sponsor for the day right after the 2007 meeting and the vendor seminars. The hotel is willing to assist the organizers in setting up the tournament but we would have to find among our members a golfer who would be willing and able to take over this task. He or she would need to have the experience to set up something that would bring in other players and to make sure that some portion of the money goes to the research fund. If one or several of you have the knowledge, capacity and willingness to carry through such a project please let us know.

We are looking forward to seeing you at the 07 meeting,

Judith Lubar
President, ISNR



LETTER FROM AAPB NEUROFEEDBACK DIVISION PRESIDENT

I am honored to be the new president of the AAPB neurofeedback section. Tom Colura did an excellent job the last two years. I hope we can follow through on his initiatives and begin some of our own.

My top priority will be to "spread the news" of the value of neurofeedback to a wider audience of users in the various fields in which we practice. Neurofeedback practitioners come from a variety of backgrounds—psychology, counseling, neurology, pediatrics, psychiatry, nursing, person training, and education, to mention several of them. Unfortunately, most individuals in

our primary fields do not understand what a powerful technique it is. For example, my colleagues in neurology tend to think of neurofeedback as just another form of biofeedback, primarily used for relaxation training. Most are not aware it is effective in normalizing brain disorders like ADHD, epilepsy, head injury, dyslexia, learning disabilities, anxiety, and depression. Why is this so? First, their training programs teach them to use drugs and surgery for those disorders. They receive little or no training in neurofeedback. They are not aware of the literature showing the remarkable efficacy

of neurofeedback in remediating those disorders or of the freedom from side effects and complications using neurofeedback. Second, the journals in which we report most of our studies (*The Journal of Neurotherapy* and the *AAPB Journal*) are not read by neurologists, and *The Journal of Neurotherapy* is not yet in the *Index Medicus*, so that literature searches often fail to



Continued on page 6

ISNR MISSION STATEMENT

To promote excellence in clinical practice, educational applications, and research in applied neuroscience in order to better understand and enhance brain function. Our objectives are:

- Improve lives through neurofeedback and other brain regulation modalities
- Encourage understanding of brain physiology and its impact on behavior
- Promote scientific research and peer-reviewed publications
- Provide information resources for the public and professionals
- Develop clinical and ethical guidelines for the practice of applied neuroscience

AAPB NEUROFEEDBACK DIVISION MISSION STATEMENT

To improve human welfare through the pursuit of its goals. The specific goals are:

- The encouragement and improvement of scientific research and clinical applications of EEG technology and neurofeedback.
- The promotion of high standards of professional practice, peer review, ethics, and education in neurofeedback.
- The promotion of neurofeedback and the dissemination of information to the public about neurofeedback.
- The division is organized for the purpose of carrying on educational and scientific objectives and is not to be operated for profit.

LETTER FROM ISNR CO-EDITOR



This April edition of the *NeuroConnections* continues the collaboration with the AAPB Neurofeedback Division. The aim of the editors and the respective organizations is to provide information around a theme for each issue. Nutrition and Neurofeedback is the focus of this edition.

Geir Flabab MD takes us through his own personal journey into Neurofeedback and the importance of nutrition in assessing and then providing comprehensive services to the clients. His ideas and practices are well worth the read.

Don Barrs PhD has provided us with a re-look at ways all of us can make the techniques and benefits of neurofeedback more accessible to the scientists of other disciplines and his generous offer to help formulate presentations is welcome.

Kimberly Weeks MS writes a follow-up to the first student survey on the accessibility and knowledge of neurofeedback and QEEG in the colleges. The article certainly gives support to Don Barrs letter.

Mike Gismondi's Best Practice's interview with Rob Cohen, PhD offers more information regarding assessing client's needs and addressing them in a multi modal approach.

The next edition in July will be around two themes: electrical issues and pediatrics. Of course should the number of articles prove to be too many for one edition they will be split between the July and October issues.

Do have a wonderful time reading these very informative articles and hope you will find them informative and useful in your clinical and research efforts.

Also, have a joyous Spring. Here in NYC, we cannot make up our minds whether to have winter or spring. By June we should have it sorted out!!

Best regards to all!

Merlyn Hurd PhD, BCIA/EEG Fellow

LETTER FROM AAPB CO-EDITOR



Welcome to the spring edition of *NeuroConnections*. This special issue is devoted to an exploration of the role of nutrition in neurofeedback and brain health. Contributors have broadened the focus of this discussion to address allergic, toxic and drug-related patterns which may be represented in the EEG and may modulate our patients' response to neurofeedback intervention.

The issue includes a discussion of emerging scientific insights into the role of nutrition in brain health and psychopathology, as well as the contributions of a group of visionary clinicians who are leading the way in integrating this emerging science into their clinical practice.

Dr. Jacques Duff contributes a scholarly review of the emerging field of nutrigenomics, representing the interface between nutrition, genetics and health. His provocative paper includes a discussion of innovative approaches to many of the most challenging chronic health conditions and developmental disorders which we may see in our clinical practices.

Dr. Curtis Cripe brings our topic into clinical focus with a discussion of qEEG patterns which may serve as "red flags" for underlying nutritional, allergic, and toxic metabolic issues. Dr. Cripe includes a discussion of the integrative multidisciplinary assessment and treatment model used in his clinic to provide neurotherapy in the context of a full spectrum of health promotion interventions to re-regulate brain function.

Doctors Margaret Ayers and Penny Montgomery describe specific EEG signatures correlating with nutritional status, infectious and toxic conditions which have emerged out of their work with the NeuroPathways system over the past several decades, while Dr. Mark Gustafson contributes a dramatic case history, which illustrates the diagnostic power of these signature patterns in the hands of a skilled clinician.

I hope that you will find, as I have, within the contents of this issue, one or more clinical gems which will inform and enhance your practice of neurotherapy.

Roger Riss, PsyD

Editor, AAPB Neurofeedback Division

LETTER FROM ISNR EXECUTIVE DIRECTOR



Another season has passed and I'm very much looking forward to spring. We, at ISNR have completed a few of the many projects we've set out to accomplish this year.

Since the last issue, we completed the new Web site. If you haven't done so already, please check out the Member Section- a great marketing and networking tool. Now that we've ironed out the glitches, the site is quite functional and useful.

Our membership is up from last year this time, with many new members. However, there are still a few of you who haven't renewed. Check to see if you're listed on the site as it's updated whenever one joins (within a few hours) rather than once a month as it was in the past.

We are working hard preparing for the conference in September. The site (isnr.org) is a great way to find out who's agreed to present, find updates on the accommodations and how to submit papers for presentations. We hope that whatever your capacity - presenter, attendee, vendor, that the conference proves to be a worthwhile event for you. This year, we are coordinating the event with the Biofeedback Society of California. Many of you may enjoy having exposure to peripheral biofeedback presentations. Please check the BSC's website for updates. The site address is biofeedbackcalifornia.org. And, on Saturday night, the BSC will host the San Diego Zoo Dinner. Eating amongst the elephants - could make you feel right at home. We hope to gain enough sponsorship to make this a free event to the attendees (prospective sponsors, if you're reading this, please contact me for details).

Looking forward to serving our members throughout the year,

Cynthia Kerson
office@isnr.org

LETTER FROM THE
AAP NEUROFEEDBACK DIVISION
PRESIDENT
CONTINUED FROM PAGE 4

yield articles on the use of neurofeedback in these various articles. Third, we tend to present our work at the ISNR and AAPB rather than at neurology meetings.

How can we remedy this problem? My personal goals are:

1. We can strive to get articles on neurofeedback published in more typical neurology journals, such as, *Epilepsia*, *Clinical Neurophysiology*, *Neurology*, and *Clinical Electroencephalography*.
2. We can present reports on the efficacy of neurofeedback at the annual meetings of the American Epilepsy Society, and the EEG and Clinical Neuroscience Society.
3. Tom Collura and I have been invited by Frank Andrasik to co-edit an issue of the *AAPB Journal* on neurofeedback. We are lining up authors for review of the use of neurofeedback in several neurological and psychiatric disorders.
4. I will apply to give a workshop on neurofeedback at the American Academy of Neurology next year, featuring some of our best, brightest, and most persuasive practitioners.
5. We can encourage our teachers of courses to have booths at the neurology meetings.
6. We can encourage our manufacturers to have booths at the neurology meetings where they can give out papers showing the efficacy of neurofeedback in various disorders.
7. We can recruit practitioners from other fields to use similar approaches with their specialty journals and at their annual meetings.

I am looking forward to working with our Board. They are all capable practitioners and can help me to make our colleagues aware of this remarkable approach, and begin to use it. Our cooperation with ISNR on the newsletter is working out very well. Good job, Cindy, Merlyn and Roger!

Jonathan Walker, MD
President, AAP Neurofeedback Division

ISNR RESEARCH COMMITTEE ANNOUNCES 2007 AWARDS

David L. Trudeau, MD, Chair Research Committee


Due to cancellations of past awards the ISNR research committee has been able to offer funding to four research projects for the coming year. This is the most awards and largest dollar amount for any year to date. A total of ten proposals were received for this funding cycle. Each applicant for the ISNR research fund had an extensive review by the ISNR research committee and opportunities to revise their applications. Fiscal restraints and remarks from the committee regarding methods, design, appropriateness and pertinence of research were considered in the rounds of revisions. The applications were then ranked by committee members and after a consensus was reached, recommendations for awards were ratified by the elected ISNR board of directors. Here are the research committee's suggestions for awards:

1. Mario Beauregard. Effects of Neurofeedback Training on Spatiotemporal Patterns of Response Inhibition in AD/HD Children: A Magnetoencephalography Study. \$20,000.
2. Bojana Knezevic. Pilot Project to Ascertain Utility of Tower of London to Assess Outcomes of Neurofeedback in Clients with Asperger's Syndrome. \$1000
3. Robert Coben. A Randomized Double Blind Placebo Controlled Clinical Trial of Neurofeedback for Autistic Spectrum Disorder. \$14,000 for first year, with subsequent funding to be considered in second year.
4. Tato Sokhadze. Neurofeedback and Motivation Enhancement Therapy Based Bio-Behavioral Treatment in Psychoactive Substance Use Disorder (PSUD) \$12,000

Other proposals considered included:

- Rex Cannon. Alcoholism: The Assessment of negative schemata, negative self-image and the involvement of particular brain areas in a dysfunctional context.
- Julie Weiner A naturalistic exploration of the value of neurofeedback in childhood primary nocturnal enuresis.
- Leslie Sherlin A Pilot Efficacy Study Evaluating Peak Performance in High School Golf Athletes using Quantitative Electroencephalographic (QEEG)-Based Neurofeedback:

Three other proposals were considered and eventually withdrawn.

The ability of ISNR to fund research is the direct result of the generous support of the membership and supporting vendors. 

ISNR WOULD LIKE TO THANK THE FOLLOWING CONTRIBUTORS TO THE 2007 RESEARCH FUND:

Delta Contributors: Efthymios Angelakis, Don Bars, Sarah Bramblett, Curtis Cripe, Marilyn DeBoer, Richard Dombrowski, Ann Frick, Carol Kershaw, Lynda Kirk, Pavel Krivulka, Ladell Lybarger, Lilian Marcus, Manuel Morales, Robert Phillips, Deborah Rabeck, Ann Richman, Aharon Shulimson, Corey Snook, Douglas Starr, David Thomson, Charles Valentine, Roxana Vasiliu, and Nancy White

Theta Contributors: Michael Anderson, David Borrmann, Tom and Helen Budzynski, Jeffrey Crotty, Emily Elliot, Maritza Rivera Gaxiola, Fritz Helmut Hemmerich, Cynthia Kerson, Marilyn Mason, Lisa Merrifield, Cynthia Perlin, Susanne Schmid-Grether, Susan and James Sendelbach, Andrea Sime, Marta Tyler and Hans Volke.

SMR Contributors: Eugenia Bodenhamer-Davis, Richard E. Davis, Carol Lee Hilewick, Ed Hamlin, Peter Kelsey, Burton Kittay, Randall Lyle, Tanju Surmeli and Alberto Texidor.

Want to make a contribution to the fund that can help our field grow? <http://www.isnr.org> and click on ISNR Research Fund.

NUTRIGENOMIC FACTORS AND MENTAL HEALTH

Jacques Duff MAPS, MANSA, MECNS, AMACNEM



NUTRIGENOMICS, MODERN DISEASES AND PSYCHIATRIC DISORDERS

Over the last decades, epidemiological studies have repeatedly demonstrated a relationship between diet and disease.¹ Nutrigenomics is the study of the interaction between nutrition and genetics. This branch of science seeks to understand the effects of nutrition on health by studying the interaction between genes and bioactive nutrients in food. Polymorphisms, small sequence differences that vary between individuals, encode protein functions and influence cell structures. Some have been identified as screening tools for predicting disease risk. Consider for example the E4 allele of the APOE gene, a marker for cholesterol homeostasis and Alzheimer's disease.² Although these single nucleotide polymorphisms correlate with certain diseases, it is often the combination of genes and nutrient deficiencies that determine whether the phenotype is expressed.³

We start life as a single cell whose DNA interacts with nutrients to make cells which divide, multiply, differentiate and eventually form a whole human being. Throughout the lifespan, Nutrigenomic interactions maintain the integrity of our cells and promote their optimum functioning. These Nutrigenomic interactions can fail due to antigens damaging DNA and caus-

ing pathogenic mutations (such as cancer); or more commonly when there are nutrient deficiencies leading to suboptimal cellular structures and therefore cellular dysfunction. It is also conceivable that some DNA weaknesses may require additional levels of nutrients to maintain cellular integrity and normal function.³ Virtually all modern diseases are due to impaired cellular integrity and the resulting cellular dysfunction.¹⁻⁴ In turn, cellular dysfunction leads to systemic dysfunctions, neurodevelopmental disorders, psychiatric disorders and diseases.³ In this paper, some common known mechanisms are introduced to highlight the importance of considering nutrient interactions in the treatment of mental disorders. However, due to space restrictions, several other mechanisms and a discussion of nutrients in modern diseases such as cardiovascular disease, cancer and diabetes have been left out.

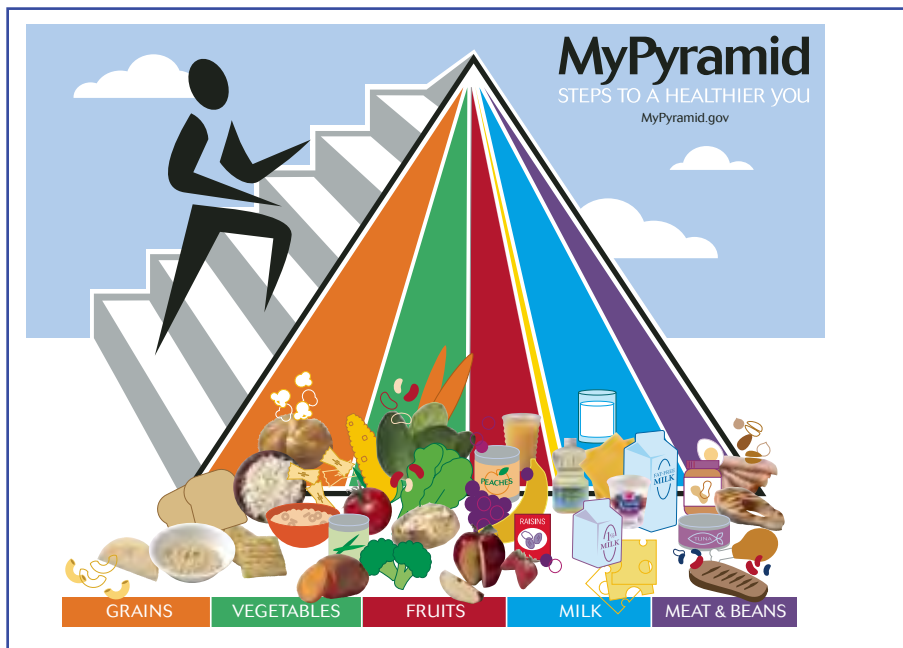
INTESTINAL DYSBIOSIS AND GUT BACTERIA

The digestive process starts to breakdown (denature) food into nutrients (amino acids, sugars, fatty acids, vitamins and micronutrients) and foods with simple structures are readily absorbed through the stomach and proximal small intestine, which are free of bacteria. However most of the breakdown of complex foods into nutrients takes place in the distal small intestine and the large bowel by gut bacteria. Several studies have

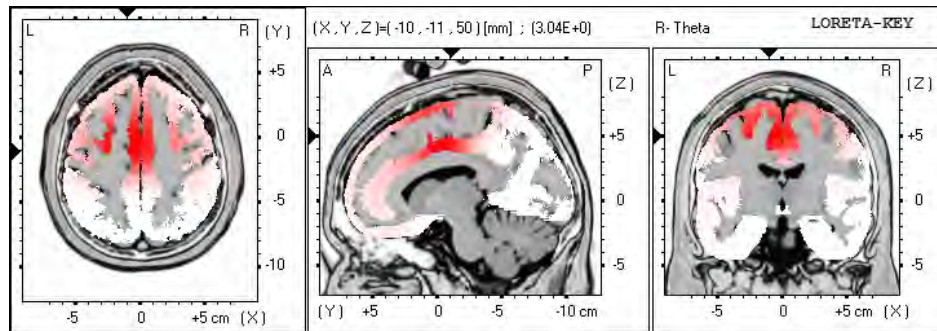
established the normal bacteria profile in healthy human populations. Intestinal dysbiosis is a condition whereby the various bacteria usually found in the large bowel are abnormally distributed. Often there is an overgrowth of streptococcus and enterococcus for example and a reduction in the beneficial flora escherichia -coli, lactobacillus and bifidobacteria. This imbalance can interfere in the proper breakdown of food into nutrients and may lead to nutrient deficiencies, leaky gut and malabsorption.

The additives, artificial colourings, flavourings, antibiotics found in the food chain, prescribed medications (including antibiotics), and other chemicals that we ingest daily may interfere with the delicate balance of beneficial bacteria in our gut. For example, preservatives protect foods on the supermarket shelves by killing bacteria and fungi; unfortunately they also kill beneficial bacteria and yeasts in our gut. These "foreign" substances, were not in the diet of our evolutionary ancestors in the Paleolithic period, and many of us are genetically ill-equipped to cope with these toxins effectively. Modern agricultural methods using chemical fertilisers and food processing have also significantly reduced the availability of nutrients in the foods that we consume in a western diet.

Intestinal dysbiosis is responsible for irritable bowel syndrome and inflammatory bowel disease. Irritable Bowel Syndrome in turn is associated with psychiatric disorders. A meta-analysis of the literature at Mount Sinai Psychiatric Hospital in New York revealed that 70 to 90% of IBS sufferers have a psychiatric comorbidity, mostly depression and anxiety. After reviewing a number of recent studies, an editorial expert opinion published in Gastroenterology, July 2000, stated that the studies established unequivocally that in some genetically susceptible persons there is an autoimmune response to the normally occurring (commensal) bacteria in the gut causing inflammatory bowel disease. A double-blind placebo-controlled study which eliminated foods causing immunoglobulin G (IgG) reactions in 150 patients suffering from IBS found that only those who eliminated IgG sensitive foods improved their IBS and mood symptoms.



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qEEG Reporting and Consultation

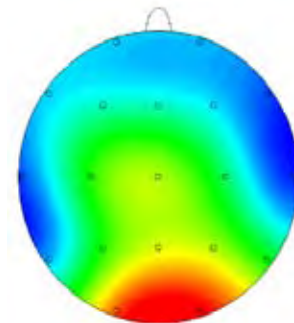
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**NUTRIGENOMIC FACTORS AND
MENTAL HEALTH**
CONTINUED FROM PAGE 7

**RECURRENT ABDOMINAL
PAIN IN CHILDREN AND MOOD
DISORDER**

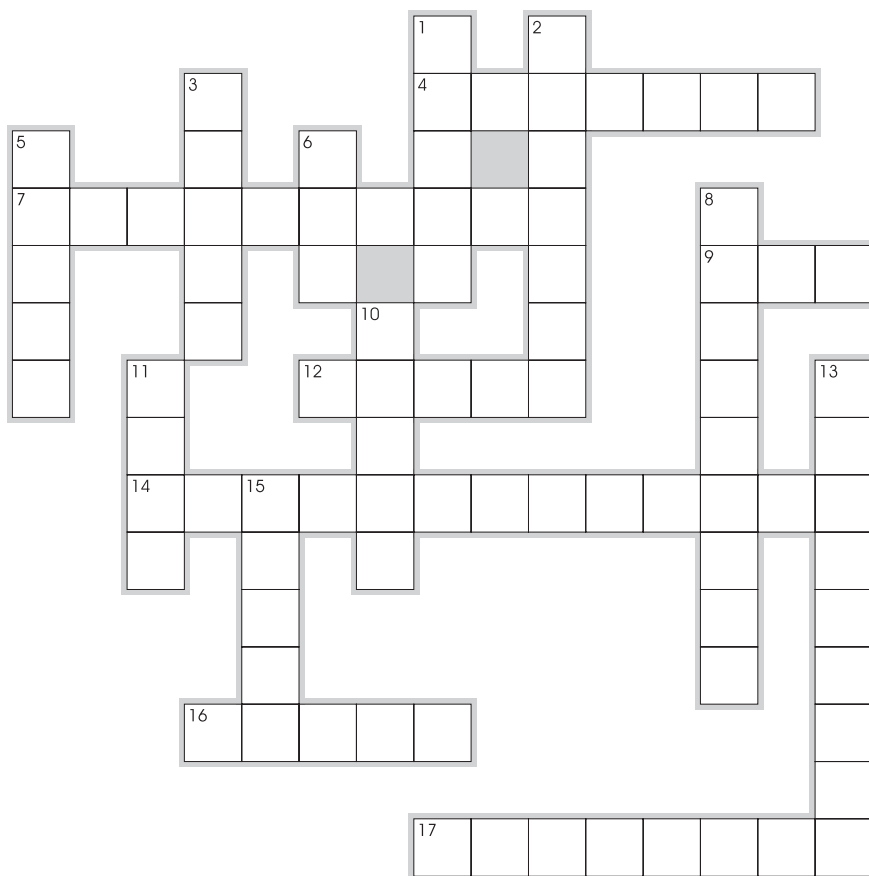
There is emerging evidence suggesting that children with recurrent abdominal pains may suffer from intestinal dysbiosis. Dr Campo and colleagues from the University of Pittsburgh School of medicine conducted a study which found that children with unexplained recurrent abdominal pains did not seem to simply outgrow them. In a controlled study, they found trends suggesting associations between childhood recurrent abdominal pains and lifetime psychiatric disorder, depression, migraine, and family history of depression at 11-year follow-up.

In our own extensive clinical experience testing the faecal microbiology of children with neurodevelopmental disorders, many children with anxiety, irritability and temper outbursts, irregular stools, frequent tummy aches and sometimes recurrent headaches have abnormal bowel bacteria profile, and more often than not, a leaky gut. Leaky gut is a medical expression meaning that large molecules of food that have not been adequately denatured by gut bacteria are able to cross the mucosal gut barrier and irritate the gut wall causing IgG mediated autoimmune reactions and inflammation. Such autoimmune reactions can be measured by IgG food sensitivity tests. Treating the bacteria imbalance and leaky gut and improving nutrient malabsorption significantly improves or normalise symptoms. Bioscreen Medical Laboratory at Melbourne University is the only Pathology Laboratory in the world at time of writing that collects faecal samples in refrigerated anaerobic conditions and grows the faecal bacteria over a period of weeks, counting the colonies to provide a detailed bacterial profile of both aerobic and anaerobic bacteria. Other laboratories do not provide such a detailed and reliable faecal microbiology profile.

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BRAIN FOOD CROSSWORD PUZZLE



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Across

4. for Ayers, high amplitude 22 hz pattern may signify this
7. this medicine may kill off healthy bacteria in the gut
9. (abbrev) 1% increase in this nutrient caused 59% decrease in postpartum depression
12. _____ amines ; too much in diet triggers depression and mental fog
14. (pl); study of interaction between genes and nutrients
16. this kind of gut may make you sick
17. Lacto _____; beneficial bacteria found in yogurt

Down

1. These acids make up 25% dry volume of the brain
2. only fuel normally used by brain cells
3. these acids build proteins for brain networks
5. this makes up 80% of brain volume
6. Omega _____; overabundant in Western diet
8. For Duff, these found in food may trigger ADHD
10. Patients with injury to this are often deficient in fatty acid DHA
11. 70% in US don't get daily allowance of this mineral
13. may cause irritable bowel disease
15. Omega _____; "good" fatty acids found in fish

Answer on page 27

Continued on page 11

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San Francisco, CA	September 15-19, 2007
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Professional EEG 4-day Certificate Program

Anaheim, CA	April 12-15, 2007
Long Island, NY	May 17-20, 2007
San Francisco, CA	June 16-19, 2007
Chicago, IL	July 26-29, 2007
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NUTRIGENOMIC FACTORS AND MENTAL HEALTH CONTINUED FROM PAGE 9

or fish-oil consumption. All modern diseases have been linked to deficits in fish-derived Omega 3 fatty acids.³ Several studies and reports by the World Health Organisation and the National Institute of Health recommend that we should consume a ratio of Omega 6 to Omega 3 of around 1.5:1. Countries and communities who consume this ratio have a very low incidence of all the modern diseases. In our modern western diet we consume a ratio of Omega 6 to omega 3 which is between 20:1 to 40:1.^{1, 2, 5, 6}

Every cell in the body has a lipid membrane which delineates the cell from its surrounding and acts to protect the cell from extracellular antigens and toxins. Omega 3 fatty acids play a crucial role in this protective system. A lack of Omega 3 EFAs renders every cell in the body vulnerable.⁷

OMEGA3 EFAS AND BRAIN FUNCTION

The dry volume of the brain consists of 25% Omega 3 EFAs, DHA which we get mostly from fish. There are in excess of 8000 studies on Essential Fatty Acids in the scientific literature. Many are controlled animal studies proving that deficiencies of the long chain Omega 3 EFAs (derived from fish) are associated with serious brain and systemic dysfunctions. Many prove that Omega 3 EFAs are essential for brain function. It is known that deficits are associated with all kinds of Psychiatric disorders including ADHD⁸⁻¹⁷, Neurodevelopmental disorders¹⁸⁻²³, Learning Difficulties^{12-17, 21, 24}, depression^{21, 25-30}, Alzheimer's dementia^{21, 26, 27, 31, 32} and Post-partum depression^{3, 30} amongst others.

The relationship between Omega-3 status, in particular docosahexaenoic acid (DHA), and post-partum depression has been investigated of late. In a cohort of 380 Australian women, plasma DHA was investigated at 6 months post-partum. Logistic regression analysis indicated that a 1% increase in plasma DHA was associated with a 59% reduction in the reporting of depressive symptoms³⁰. It is well known that during pregnancy there is a significant transfer (up to 2.2 g/day) of Essential Fatty Acids (EFAs) from the mother to the developing fetus, particularly DHA³³. Increased risk of post-partum depressive symptoms has recently been associated with a slower normalization of DHA levels after pregnancy.³⁴ During lactation, there is an ongoing transfer of DHA from the mother to the child through breast milk. Low maternal

DHA is likely to result in low DHA in the offspring and be associated with neurodevelopmental disorders. Supplementation with fish oils effectively raises Red Cell and Plasma DHA.^{8, 21, 23} The average level of Red Blood Cell DHA, in normal controls with no Neurodevelopmental disorders, was found to be 4.2%, those with ADHD 1.8% and in Autism 0.8%.³⁵

OMEGA3 EFAS AND GUT FUNCTION

The gut cell wall, the epithelium, is constantly exposed to billions of organisms and toxins daily. The importance of the protective effect of the lipid membrane, zinc and of each cell in the epithelium cannot be understated. If the lipid membrane is faulty, such as if there are deficits in Omega 3 fatty acids, the lipid membrane integrity fails and unfriendly organisms and toxins irritate the gut wall, possibly giving rise to Irritable Bowel Syndrome and Inflammatory Bowel Diseases. Hundreds of other factors, nutrients, enzymes and their interactions are also involved. However, Omega 3 fatty acid deficiencies and deficiencies in associated nutrients, such as zinc, magnesium and B group vitamins may be the common denominator amongst the biological predispositions that underpin most modern diseases.

OMEGA 3 EFAS AND TBI

Several animal studies have identified that increased concentrations of free fatty acids in cerebrospinal fluid after a minor TBI are recognized as markers of brain damage. One recent study examined FFA concentrations in cerebrospinal fluid from 15 patients with TBIs compared to those from 73 contemporary controls. Concentrations of Arachidonic (AA), docosahexaenoic (DHA) and myristic acid, from TBI patients, obtained within 48 hours of the insult were significantly ($P < 0.001$) greater than those in the control group. Higher concentrations of total polyunsaturated fatty acids obtained 1 week after the insult were associated with a significantly ($P < 0.001$) worse outcome at the time of hospital discharge using the Glasgow Outcome Scale as a measure [36]. The study highlights the fact that TBIs result in a substantial loss of DHA, which unless replaced through a high fish diet or with fish oil supplementation, may result in impaired mental function.

ZINC AND PSYCHIATRIC DISORDERS

After potassium and magnesium, zinc is the most abundant intracellular micro nutrient in the human body. It is an essential trace element used in hundreds of biochemical pathways. Thus zinc deficiency may be involved in a wide range of metabolic disorders, affecting protein synthesis, cell membrane integrity, immune and neurological functioning. Zinc is an essential component of desaturase enzymes responsible for EFA metabolism. Over 70% of the US and Australian populations do not receive the recommended daily intake of zinc and therefore may be zinc deficient. The determination of zinc deficiency is a complex issue. Blood tests for zinc do not provide adequate indication of zinc deficiency, as blood maintains zinc within tight limits in circulation, denying it to tissues where the mineral is needed. A zinc taste test is a better way of screening for possible deficiency. Currently the only way of proving zinc deficiency is by supplementation, followed by observation of improvements in biochemical or behavioural functioning.

Apart from dietary shortfalls, chronic zinc deficiency may result from Pyroluria, a condition whereby excessive pyrrole production from haemoglobin binds zinc and excrete it through urine output. Pyrolurics can exhibit varying degrees of psychiatric problems, most commonly Mood Disorders and Schizophrenia, which are exacerbated by stress.

FOOD ADDITIVES AND ADHD

In a landmark double-blind placebo-controlled crossover study, twenty-seven children, whose behaviours had improved significantly on the Royal Prince Alfred Hospital (Sydney) elimination diet, which excludes food additives, natural salicylates, amines and glutamates, were either challenged with calcium propionate (preservative 282) or given a placebo in their daily bread. There was a statistically significant difference in the proportion of children whose behaviours 'worsened' with challenge compared to placebo. Dengate and Ruben who conducted the study concluded that irritability, restlessness, inattention and sleep disturbance in some children who are sensitive to amines, glutamates and food additives may be caused by this common mould inhibiting preservative. In our own experience, anecdotal as it may be, these

Continued on page 13

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NUTRIGENOMIC FACTORS AND MENTAL HEALTH CONTINUED FROM PAGE 11

children respond best to fish oils and associated essential nutrient supplementation and for most, their sensitivities and behaviours resolve in a matter of a few months.

In addition to showing that additive 282 may exacerbate childhood behaviours, this study also highlighted that some children benefit significantly from a diet that exclude a number of substances including trace amines. In this study removing the irritants from the GUT helped reduce inappropriate behaviours. When reintroduced the behaviours returned.

In addition to removing irritants from the gut, the gut wall needs to be repaired using specific nutrients, including Omega 3 EFAs and the balance of the gut bacteria restored. Although this can take several months, in our experience most children thrive physically and emotionally on this approach and parents and teachers report that their behaviours improve dramatically. Many of the children that we treat also need Neurotherapy to redress abnormal brain-wave patterns and CBT to help them adapt to their environment and promote more beneficial behaviours.

Increased amine production can result from food allergies and abnormally high levels of some naturally occurring bowel bacteria. For example gram +ve bacteria produce more Amines and less aminoacids from proteins under acidic gut conditions.

TRACE AMINES, DEPRESSION AND IMPAIRED MENTAL FUNCTION

Trace amines are naturally-occurring substances in food, which the body can cope with in small amounts. However in larger amounts they can cause serious disturbances in the brain, triggering depression and reduced mental alertness. Recent studies have found G-Protein coupled receptors for amines in the brain of mammals, suggesting that increased dietary amines, and increased amine production from overgrowth of some gut bacteria may cause treatment-resistant depression, fatigue mental fog and tiredness.

Jacques Duff is a registered Psychologist who since 1997 has been conducting clinical research in the causes and treatment of ADHD at the Brain Sciences Institute, where he is a PhD candidate in the Clinical Neurosciences stream. Since then he has also been the Director of the Behavioural Neurotherapy Clinic, which specialises in ADHD, Behavioural and Neurodevelop-

mental disorders. Jacques has assessed and treated over 1200 children in that period and treated many children with difficult behavioural conditions. Jacques has presented at dozens of seminars and symposiums, in Australia and Overseas on the subject of ADHD causes, QEEG, Neurotherapy, Nutrition and Neuroscience.

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LETTERS FROM THE MEMBERS

HELLO FELLOW MEMBERS,

For many years we have been trying to increase the acceptance of neurotherapy by other mental health groups. The field has come a long way since I started working with ADHD and the old Autogen 120. ISNR has grown tremendously from its early days as SSNR and we have put on many outstanding conferences with experts from the world over. Over the years Jay Gunkleman as the conference chair, tried and was extremely successful at getting individuals at the top of their respective fields to attend and present their research, hoping that they would also stay and listen to what we as a group were doing.

While some of our members have been successful at getting papers printed in journals other than The Journal of Neurotherapy helping the field get better known, there still remain many professionals with doubts that it is possible to do what is claimed can be done to improve behaviors with neurotherapy. Also, as Christopher deCarms, Ph.D. pointed out in his talk at the last conference, there are some who doubt

the quality and findings of the research already conducted or indicate that it is more than likely the improvement in behaviors is just a placebo effect. Recently in Scotland, I talked with Professor Marc Nuwer from UCLA Medical Center and past president of the International Federation of Clinical Neurophysiology about neurotherapy to gauge his reaction. I found that while he did not dismiss it out of hand he was nevertheless not impressed, a little like his early thoughts on the clinical utilization of evoked potentials. His primary concern is that the science behind the claims does not seem to be there; exactly the concern of many health care providers. The reason I bring this up is because I have been for years trying to get members interested in presenting their research not only at our own conferences, which is like preaching to the choir, but to also present at the International Congress of Clinical Neurophysiology that occurs every 4 years. I have attended and presented at five international congresses and other than when I presented in Buenos Aires in 2001, about

using qEEG and EPs to separate affective disorders from disorders of attention, there have been few studies about the behaviors we work with and none concerning neurotherapy as a treatment. The studies usually given deal with the use of Transcranial Magnetic Stimulation (TMS) and its effect on behavior or biofeedback in the training for prevention against urinary continence.

I would like to propose that to be accepted, our group needs to present at these congresses. The world experts in neurophysiology attend them and we need to get our findings accepted by this group to gain the world wide acceptance of our techniques we all talk about. The next Congress is in early September 2010 in Kolbe, Japan. I plan on presenting some of my research and would like to be joined by as many other members of ISNR as possible. Also, I would like to offer my assistance to any clinicians who have good data that they might like to present but need help or have questions about how to go about it.

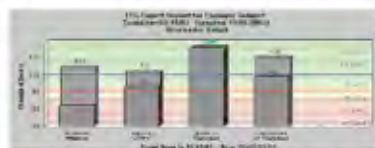
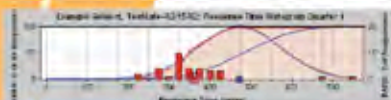
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THE NEED FOR VITAMIN D

Dave Siever



INTRODUCTION

The winter of 2005-2006 was a real challenge for me. For no apparent reason, I had developed full-blown restlessness and insomnia by December, and come January, I began feeling mildly depressed, had severe bouts of fuzzy-headedness and lethargy. I was waking up at 2, 3 and 4 AM, and because of that, I would sleep in until 9:30 to 10 AM, as I tried to make up for the lack of it. I was craving chocolate like mad—and simple carbs (candy, cake, etc.) as well—but mostly chocolate. By February, I was feeling like a tired old man. I had no physical energy and felt very weak. I quit working out on the weights because it took just too much effort. By April, my lower right molar and gums began getting quite painful when I ate, making it difficult to chew. Later in April my upper left molar also began aching as well. Also, my elbows, knees and ankles began to ache. I was really feeling old, if not ancient. This was of great concern for me, as I normally feel physically strong.

In early April, just before attending the AAPB conference, I went for an 8-minute tan and noticed that later on in the day, I felt relatively very good, but by the next day I was back in the slumps. A few days later, I went back and tanned to the point of getting mildly pink. The next day there was no change but the following three days I felt very good, and in particular I had an excellent head-space, even though my joints and mouth continued to hurt.

Suspecting a connection, I took time off from work every day at noon for the first two weeks in May, stripped down to my shorts and took in at least 30 minutes of tanning in the warm May sun. Never have I got so much sun so early in the year and fortunately, our Spring was the warmest and sunniest ever, with leaves sprouting and flowers blooming a full three weeks ahead of schedule.

By May 15, my mind was as sharp as a well-honed knife and all of my body pain vanished. I was suddenly bursting with energy and strength and biked 25 miles on Mothers' Day! Best of all, my wit and humor returned and I enjoyed being the life of the party again. On the May 20, Canada long weekend, I went to Jasper National Park and participated in the annual SCUBA diving competition – feeling GREAT!

WINTER 2006

This winter was an unusual one. We normally have a good foot of snow by Christmas, but with global warming looming, the past 10-years have seen three winter droughts. This year, we didn't get snow until March 15! What this has meant to me and some of my clients whom I have since spoken to since, was that this was a particularly severe year for certain people suffering in symptoms of a kind-of seasonal affective disorder (SAD),

A possible reason for this is due to a lack of reflection off of the snow. Edmonton is situated about 300 miles north of the USA/Canada border at 53 north latitude. Edmonton is a fairly sunny place and in Edmonton the sun shines at roughly a 20-degree angle in around the winter solstice. This means that plenty of sunlight bounces off of the snow and given that most clothes pass 10-20% of the sunlight that strikes them, everyone would get some sunlight exposure. This year, without the snow, I missed my weekend cross-country skiing and the concurrent nourishment from the sun.

THE VITAMIN D STORY

Vitamin D is a potent steroid, much like testosterone. It is essential for the metabolizing of calcium (which is also essential for generating the voltage potentials in neurons). This explains the emotional collapse and the subsequent depression, anxiety, restlessness, insomnia and so on in those who are deficient. Vitamin D also plays a key role in muscle strength and the integrity of connective tissue and the maintenance of bones as well as playing a part in controlling swelling and in tissue repair. Vitamin D supplementation has been shown to reduce the risk of breast, colon, pancreatic and prostate cancer. Current research indicates vitamin D deficiency is a causal factor in 17 varieties of cancer, heart disease, stroke, hypertension, autoimmune diseases, diabetes, depression, chronic pain, osteoarthritis, osteoporosis, muscle weakness, muscle wasting, birth defects, periodontal disease, rickets, and the common flu.

Vitamin D is involved in brain function. Nuclear receptors for vitamin D are localized in neurons and glial cells. Genes encoding the enzymes involved in the metabolism of vitamin D are also expressed in brain cells. Vitamin D is important in neurotransmitter synthesis. Vitamin D has been shown to have neuroprotective and immunomodulatory effects and may help us resist bacterial and viral infections. A study of arterial and venous blood samples from 101 healthy Australian men over a one-year period found strong

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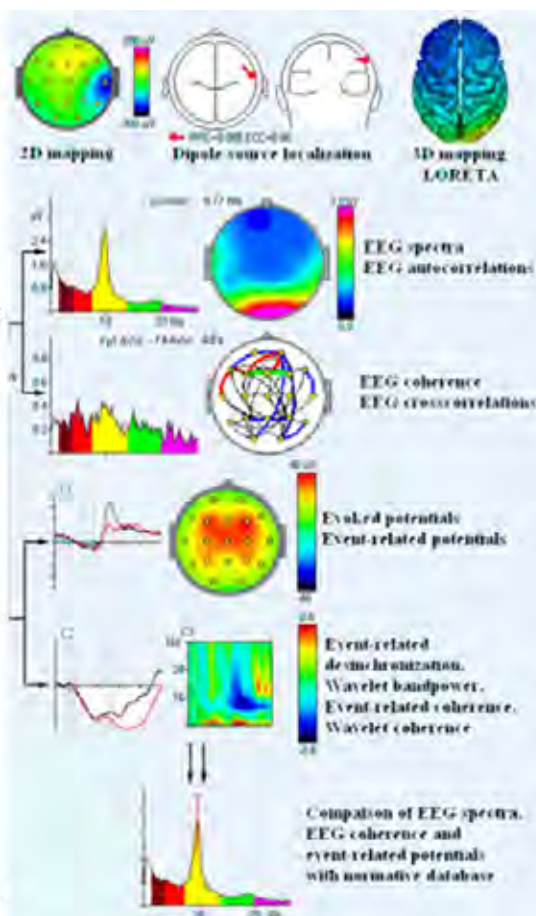
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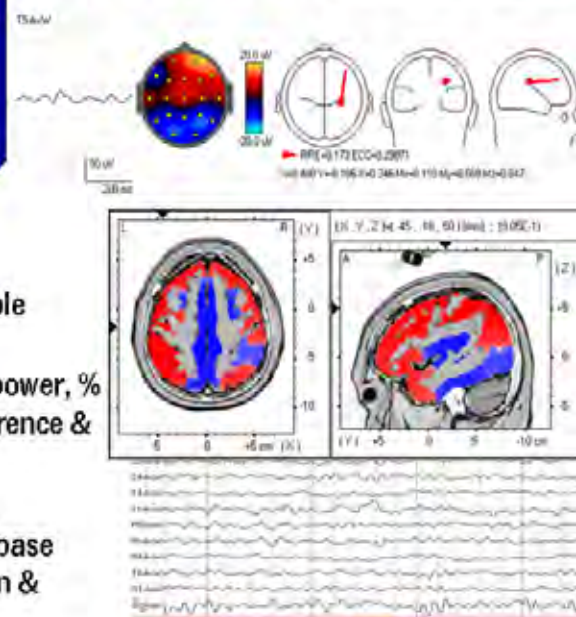
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THE NEED FOR VITAMIN D CONTINUED FROM PAGE 15

correlations between ambient sunlight and production of serotonin in the brain. In fact, depression and schizophrenia are highly correlated with vitamin D deficiency. Autism is also effected by seasonality, which may be related to vitamin D deficiency during pregnancy.

It has been thought that the average human utilizes roughly 1500-2000 IU of vitamin D per day. However, recent research suggests that healthy men require upwards from 3000-5000 IU per day. Those who live above or below 30 degrees latitude are likely to become vitamin-D deficient throughout the winter. Over one billion people worldwide have become deficient in vitamin D including many Americans who work daylong in office buildings. Estimates in the USA suggest that somewhere from 21 – 58% of adults and adolescents are vitamin D deficient (and this is using the old low-level standard).

Vitamin D has been shown to significantly enhance the genetic expression of antimicrobial peptides in macrophages, thus improving up-front ability to attack and

destroy a broad spectrum of invasive microbes, spanning both viruses and bacteria. This may be why there is a seasonal effect with flu. Flu season occurs in the winter of both the northern and southern hemispheres, and it is not attributed to more people staying indoors as previously thought.

natural sources of vitamin D are sunshine and fatty fish such as salmon and mackerel. Milk is fortified with vitamin D, but at very low levels - enough to prevent Rickets.

ABOUT SAD

Each year, 6% of northern populations are

OVER ONE BILLION PEOPLE WORLDWIDE HAVE BECOME DEFICIENT IN VITAMIN D INCLUDING MANY AMERICANS WHO WORK DAYLONG IN OFFICE BUILDINGS.

The best kind of vitamin D, vitamin D3, or cholecalciferol (its chemical name), comes from the sun. Most vitamin supplements also contain vitamin D3. Calcidiol (25-hydroxy vitamin D) is a prehormone in your blood that is directly made from cholecalciferol. When being tested for vitamin D deficiency, calcidiol is the only blood test that should be drawn. When doctors refer to vitamin D blood levels, they are referring to calcidiol levels, but the lab will know calcidiol as 25-hydroxy vitamin D. The only

affected with Seasonal Affective Disorder (SAD) and another 14% have a milder form of SAD, called the winter blues. Surprisingly, SAD may occur at any time of year and even in equatorial regions although the ratio of northerners with SAD as compared to those living in the tropics is about 10-1. People in the southern USA experience SAD in the summer from staying indoors where air conditioning allows them to es-

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THE NEED FOR VITAMIN D CONTINUED FROM PAGE 17

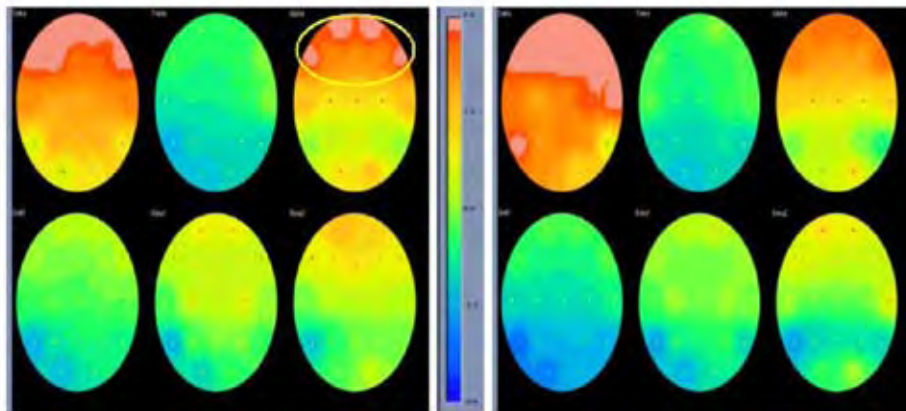


Figure 1. *Vitamin D Deficiency vs Sunlight Exposure*

cape the unbearable summer heat. People have also experienced SAD moving into a basement suite or an office on the north side of a building or after painting the interior of their home a darker shade of color. People have experienced SAD following the development of cataracts or after wearing sunglasses for an extended period of time and during overcast, rainy periods (Rosenthal, 1993).

The common symptoms are depression, anxiety, extreme fatigue, hypersomnia, carbohydrate cravings, and weight gain. Women between the ages of 20 to 40, (their sexually reproductive years), are most susceptible (Rosenthal, 1993). The first controlled study using light therapy to treat SAD was published in 1984. SAD was officially accepted as a clinical malady in 1987 by the American Psychiatric Association and described in its then current diagnostic manual, the DSM-III-R. Since that time, a great number of studies on the topic have been completed.

THE “CAPTAIN AND PINEAL”

All species studied to date have been observed to have a biological clock. This

clock is essential for survival, regulating various types and levels of arousal to provide cues for alertness, eating, sleep and the release of hormones. Light waves striking the retina activate electrical output that is sent down the optic nerve to the brain for visual processing. A secondary, smaller nerve tract from the retina, originating from specialized cells that utilize a light detecting pigment called melanopsin, also carries signals to the suprachiasmatic nucleus (SCN) of the hypothalamus. The SCN, in turn, sends nervous outputs to various parts of the brain including the pineal gland. Four genes that govern circadian cycles in flies, mice and humans have been discovered that not only reside within the SCN, but in all cells of the body. When cultured in a petri dish under constant lighting, these cells continue with gene activity, hormone secretion and energy production in a 24-hour cycle that varies less than 1% (Wright, 2002).

In the mid 70s, Dr. Alfred Lewy of the National Institute of Mental Health (NIMH) discovered the neurotransmitter melatonin. The wake/sleep cycle in animals and humans is controlled by melato-

nin, which is produced by the pineal gland, a structure the size of a pea and located in the mid-brain. Every night, the pineal gland excretes melatonin into the bloodstream and continues to do so until dawn. However, under normal exposure to sunlight, secretions of melatonin follow the earth’s light/dark time frame and therefore more melatonin is typically released during the long dark hours of the winter months. Henceforth, the pineal gland is in charge or “captains” our wake/sleep arousal states.

A COMPARISON BETWEEN SAD AND VITAMIN D DEFICIENCY

Although most anxiety and depression inventories could be used to detect SAD, one popular SAD test is the Seasonal Pattern Assessment Questionnaire or SPAQ, developed by Rosenthal and his colleagues at the NIMH. The SPAQ is a self-assessment questionnaire that evaluates one’s level of SAD. However, one big problem with the SAD test is that there is much overlap between the symptoms of SAD and Vitamin D deficiency, and more than just the basic SAD type questions must be asked.

The literature on the brain wave effects of SAD is inconsistent, ranging from increased broad alpha/theta to increased left frontal alpha activity as is known in depression. In Figure 1 below are eyes-closed brain maps from the Skil database at a scale of ± 2.6 SD before and following 2-weeks of sun tanning. Notice the exceptionally high alpha (encircled in yellow) while deficient in vitamin D. The high delta is primarily artifact. If this was a case of true SAD, it would have resolved itself by the time the “sunlight” QEEG was taken on May 15, as eye/pineal exposure is significant by Ma

TREATMENT OF SAD

A number of coping techniques are used to reduce the symptoms of SAD. These include long walks outside, aerobic exercise, a diet rich in complex carbohydrates and protein, relocating to sunnier locations, winter vacations to tropical areas, and frequenting sun-tanning centres. Light-based clinical interventions include light box therapy and audio-visual entrainment.

“Light box” therapy has been used to reduce the symptoms of SAD in 60% to 80% of SAD patients. White light therapy, using intensities of 2,500 lux, requires exposure times from 2 to 6 hours, a considerable behavioral investment for the user.

SYMPTOMS OF SAD

1. anxiety
2. depression
3. fatigue
4. carbohydrate cravings
5. hypersomnia
- 6.
- 7.

SYMPTOMS OF VITAMIN-D DEFICIENCY

- anxiety & restlessness
- depression
- fatigue
- carbohydrate cravings
- insomnia
- physical weakness
- connective tissue swelling

Light exposures in the intensity of 10,000 lux for 30-minute exposures has been found to be more effective than 2,500 lux intensity with exposure times of several hours. Some people have reported that over-use of light therapy can leave them feeling "wired" and restless.

Audio-Visual Entrainment (AVE) using flashing lights and pulsing tones has been shown to enhance EEG activity at the stimulation frequency. However, a lesser-known attribute of AVE lies in its inhibition effect at roughly the half-frequency of stimulation. In QEEGs (brain maps) collected at our office of those with SAD, we have observed long spindles of 10 Hz alpha brain wave activity, globally, with particularly increased activity in the left frontal regions. In light of these findings we ran a study of 74 people struggling with SAD. AVE at 20 Hz produced profound reductions in anxiety, depression, carbohydrate cravings and body weight. Energy and quality of life increased.

TREATMENT OF VITAMIN D DEFICIENCY

The only treatment for vitamin D deficiency is vitamin D3, whether it be from food sources or via the sun. Many supplements are available. Studies have shown that it is difficult to become toxic on vitamin D, and a wide variance of a suggested toxic limit exists in the literature. An adult may have to consume 4000-8000 IU for more than four months in order to become toxic.

RESOURCES

FOR MORE INFORMATION ON VITAMIN D:

www.vitamindecouncil.com

Dale Kiefer. Why is Flu Risk So Much Higher in the Winter? Life Extension, February, 2007, 23-28.

FOR MORE INFORMATION ON SAD AND ITS TREATMENT WITH AVE:

www.mindalive.com/1_0/articles%204.pdf

Dave Siever graduated in 1978 as an engineering technologist. He later worked in

the Faculty of Dentistry at the University of Alberta designing TMJ Dysfunction related diagnostic equipment and research facilities where he organized research projects, taught basic physiology and the advanced TMJ diagnostics course. Dave had noted anxiety issues in many patients suffering with TMJ dysfunction, prompting him to study biofeedback, which he applied to their patients and later design biofeedback devices.

In 1984, Dave designed his first audio-visual entrainment (AVE) device—the "DigitalAudio-visualIntegrationDevice," or DAVID1. Since this time, through his company, Mind Alive Inc., Dave has been researching and refining AVE technology since, specifically for use in relaxation, and treating anxiety, depression, PMS, ADD, FMS, SAD, pain, cognitive decline and insomnia, which he presents at conferences and for special interest groups. Dave also designs Cranio-Electro Stimulation (CES) and biofeedback devices and continues to conduct research and design new products relating to personal growth and development.

INTERMEDIATE AND ADVANCED QEEG, NEUROFEEDBACK & LORETA WORKSHOP

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JULY 20, 21, 22, 2007

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- *Learn methods for obtaining quality recordings
- *Learn detailed methods for artifact removal
- *Learn how to distinguish muscle artifact from EEG in the recordings.
- *Learn how to coherence neurofeedback

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MINDFULL

Preattention

David Kaiser, PhD



What is the most important part of the brain? What most keeps us informed and the information flowing? What brain area is most responsible for making us us?

Our mental abilities are often attributed to an enlargement of the frontal lobes during human evolution, but this isn't entirely true. It's true that our frontal lobes are larger in absolute volume than any other primate, nearly four times the size of a chimpanzee's, but relatively speaking the mass behind our eyes occupies the same space in our head as it would any ape our size: 38% of our cortex is frontal lobe, compared to 35-38% for chimps, gorillas, and orang-utans. Our frontal lobes are proportional to our size, nothing more; they did not balloon out in any respect during the last few million years.

Perhaps if we took a microscope to our lobes we could determine how most we

differ from other primates. For the sake of philosophy and religion we might find out that our frontal lobes are more richly interconnected than our furry brethren's, or contain a greater amount of "human" cells, whatever they may be, which explains the various behaviors unique to our species such as language and warfare, but we're getting off-track. If we presume that the most important part of the brain is specific to humans, we've entered a circular argument.

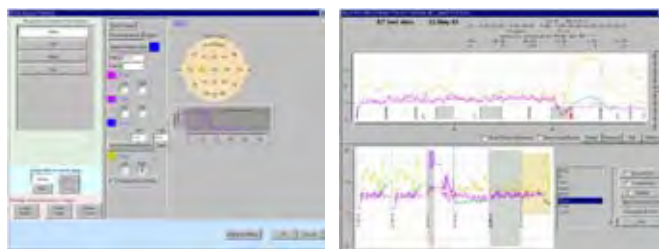
Let's look at the brain as a general information processing device. Can neuroscientists and people in the know agree upon its most important structure? Well, why ask onlookers when we have access to the source itself? Brain--- which part do you most cherish? Which do you service ahead of all others? Which part do you get online first and keep online most? And given your reliance on it, which part is most



overextended, most likely to be run into the ground?

Using any of these criteria, the most important brain structure in humans, or any animal, is the inferior colliculus. Overlooked, ignored, a brain site that gets sand kicked in its face, called inferior -- this geeky site controls auditory information at its earliest flow. Neither cortical nor limbic, the colliculi are midbrain, although I've seen it mistakenly relegated to lowly brainstem. Talk about not getting any respect; and yet it is the most metabolically active structure

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in the brain (Sokoloff, 1981), the first myelinated and most vascularized (Moore et al., 1995); hands down, the most important structure in the brain, human or non.

Landau et al. (1955) was one of the first neuroscientific teams to investigate this humble structure and when they detected the highest blood flow in the inferior colliculus (IC) compared to all others, they immediately assumed they had got it wrong. They didn't believe the results. Surely their measuring equipment was to blame. It was loud and noisy so the high readings in this unassuming structure were likely due to artifact, so they thought, especially given the IC's function (sound processing). So they repeated the experiments with deafened cats and got the same results. This time they believed them. Others determined that the IC possessed the greatest capillary supply (Craigie, 1938) and consumed the most glucose per volume (Fisch, 1970). Most of the cortex takes years, even decades to myelinate; but the IC requires a mere 25 gestational weeks (Yakovlev & Lecours, 1967; Moore et al., 1995) and is only one of two brain structures fully functional (i.e., myelinated) at birth. In fact it may be respon-

sible for triggering maturation of "more important" cortical structures above it (Von Hungen et al., 1975). (The other structure fully myelinated at birth is the inner division of the inferior cerebellar peduncle. A baby needs to integrate proprioceptive sensory input with motor vestibular functions in order to balance, maintain posture, and drool effectively.)

The inferior colliculus is also the first structure to fail, as it alone is selectively damaged during the earliest moments of asphyxia. Injury of the inferior colliculus causes "word deafness," the inability to comprehend spoken language, and even slight damage is suspected in hypometabolism of language receptive areas. Caspary et al. (1995) detected a decline of neurotransmitter function in the IC with advancing age, which may reduce our capacity to detect and extract meaningful signals from background noise, making it difficult to follow a conversation in a noisy environment. (At least that's what I thought they said.)

So what task does this important structure perform?

In fish the IC acts as their ears – vibration detectors, that is. In mammals the

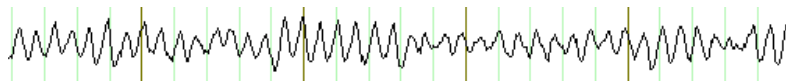
IC acts as our sentinel as it monitors the environment preattentively. It continually evaluates the environment, seeking changes in the periphery, relegating background noise to just that, background noise to ignore and habituate to, and directing attention to that in the environment which ought to be attended to. Hearing-impaired individuals often resemble hyperactive (ADHD) individuals on tests of vigilance because they lack this mechanism, a passive sentinel, and are subsequently forced to use vision for both environmental awareness and inspection.

So the lesson that might be taken from the IC is the importance of preattention. Forget consciousness and the cortex; mental dysfunction probably lies here, in subcortical preattention, to paraphrase Freud. Disconnecting the IC from structures above it produces a dramatic loss of attention, affect, and motivation (Sprague et al, 1961), the basis of all mental illness.

In regard to EEG, the preconscious layer happens to be where neurotherapy does it work. Operant conditioning of the

Continued on page 20

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MINDFULL

CONTINUED FROM PAGE 19

EEG is volitional but preconscious (i.e., not fully conscious). We initiate changes in brain states but are unable to characterize and control these changes as we would a motor movement. They remain available but without discrete starts and stops to grasp at. The preconscious is not foreign to us, simply unremarkable, in both senses of the word. The preconscious is ubiquitous as well as difficult to describe, despite the fact that we spend so much time here (especially as we grow older). When we think of something to say but lose the handle on it and we flounder for words or meaning, we're there, preconscious, a sea of multiplicity without proper priorities. Rest assured, the preconscious is volitional, just one in need of order. So the lesson of IC is priority, importance through sequential hierarchy, and it reveals that often the point of entry for information is the most important point. *M*

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NEUROFEEDBACK AND QEEG RECOGNIZED AT CHADD CONFERENCE

Joseph Barr PhD

This last October neurofeedback providers from the Chicago Area broke new ground as they manned an exhibitor's booth at the Annual CHAD Conference. CHADD, Child and Adults with ADHD, has historically been reticent to acknowledge the merits of EEG Biofeedback. Nonetheless, this fall they welcomed the local neurofeedback providers interested in presenting the case for our work to this international audience. Further, the conference program on complementary treatments noted the strong research on neurofeedback assessment and treatment of attention disorder.

The CHADD Program on Complementary Treatments was chaired by Drs David Rabiner and Barbara Ingersoll. Developmental Pediatrician Patty Quinn, a member of the Professional Advisory Board of CHADD, addressed the value of an objective measurement of ADHD. She spoke of the strong effects size in the research and clinical utility of quantitative EEG.

She noted that in 2004 in an American Academy of Pediatrics Guideline for treating ADHD that the "QEEG will help experts more clearly document the neurology and behavioral nature of ADHD paving the way for a better understanding and treatment". "We know that EEGs have been available for a long time, that the brain shows continuous electrical activity, and that what we are looking for are changes in the ratios of the electrical activity".

Dr Dominic Greco presented the research on neurofeedback referencing the work of Monastra and the many others. David Rabiner highlighted the increased quality in the field over the last five years.

The Neurofeedback Centers of Greater Chicago (www.eegchicago.com) is a provider group that collaborates in informing the community on the applications and efficacy of neurofeedback. The group, now two years old, has providers across the metropolitan area. Members Ann Stout, Kathy Abbott, Ann Richman, Marilyn DeBoer, Charles Warren, Jim Kowal, and Joe O'Donnell and Joseph Barr staffed the informational site. They answered the questions of an interested audience and directed national and international conference attendees to provider referral resources.

Joseph Barr is a psychologist in practice in Deerfield, IL; he is BCIA Board Certified, past president of the Biofeedback Society of Illinois, and former chief of psychology at Lutheran General Hospital. He has taught at Northwestern and University of Illinois Medical School. You can contact him at <http://www.bfnorth.com>. *M*



l. to. r.: Charles Warren, Ann Richman and Kathy Abbott



Joseph Barr and Kathy Abbott

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AUTUMN'S STORY

Roger H Riss, Psy.D.

When she first began to experience light headedness, Autumn didn't pay much attention, assuming that her symptoms would pass on their own. After all, she was only in her thirties, and in excellent health. Aside from the births of her two young children she had never been in the hospital. Eventually, she could no longer deny that her puzzling symptoms were growing worse. Her dizziness was becoming more persistent, accompanied now by problems with balance, coordination and constant fatigue. A self-employed cosmetologist, she was finding it increasingly difficult to spend a day on her feet, working with her clients. She wondered if they noticed her catching her balance against the edge of the chair as she trimmed their hair. Soon she began to notice that her thinking was slower, and that she was becoming forgetful. When she could no longer ignore her symptoms, she shared her concerns with her family physician.

At first the answer seemed simple. She breathed a sigh of relief when she learned that she was pregnant with her third child. Her doctor agreed that her problems could simply be related to the pregnancy. "When I entered my second trimester, my symptoms seemed to go away, and I thought my problems were behind me." However, once her beautiful baby boy was born, her all too familiar problems returned with new severity. She had to be careful when coming to standing from a chair, lest she lose her balance with baby in arms. Her days at work seemed interminable. The fatigue and mental fog made it increasingly difficult for her to juggle her responsibilities as a working mother. It seemed increasingly difficult for her to stay focused when driving...

Thus began a frustrating two year journey through the offices of countless specialists and countless rounds medical tests as she searched for answers to her deteriorating health. One by one, the specialists tested for, and ruled out the frightening possibilities...multiple sclerosis, Lou Gehrig's

disease, Lyme disease... CT scans, MRI's, EEG's, spinal taps failed to yield clues to her puzzling decline. She was one week away from brain surgery to repair a Chiari malformation, before last minute retesting failed to confirm the diagnosis.

An extrovert and natural optimist, Autumn tried to stay upbeat for her children, but the unrelenting progression of her symptoms and discouraging rounds of medical tests left her feeling increasingly frightened and helpless....

Her long search for a diagnosis seemingly was rewarded with a trip to the offices of a prominent Salt Lake City neurologist. After another round of extensive tests, he explained that she suffered from "channelopathy," a term describing a family of rare genetic disorders affecting ion transport through cell membranes of the body. Her initial relief at finally receiving a diagnosis was short-lived, when it became obvious that her body was unable to tolerate the medications which the neurologist offered to control her condition.

Her chiropractor suggested that she consider an alternative therapy. He told her that he had recently met Dr. Mark Gustafson, a chiropractor who incorporated neurotherapy, a form of "brain training" into his practice, and was reporting very encouraging results with symptoms of mental inefficiency. In Autumn's own words, "Within a week of my first session, I felt better than I had in two years."

Dr. Mark explained: "Using Margaret Ayers Neuropathways system, I immediately recognized a pattern of excessive frontal slowing in Autumn's raw EEG signal, which I interpreted as a possible sign of toxicity. This led me to do further testing for sensitivities to foods, medications or additives in her diet. By the end of her initial evaluation, we had identified a sensitivity to aspartame products, and I instructed Autumn to substitute a natural chewing gum for the brand that she had been chewing on a daily basis."

A few weeks later, when this writer had the opportunity to meet with Autumn, it was only her third session with Dr. Mark. Her mother and nine year old daughter accompanied her, confirming the story of her near miraculous recovery following a simple change in chewing gum! Dr. Mark performed follow-up balance and muscle testing, confirming that there were no signs of the balance and coordination problems that had been present for the past two years. Moreover, repeat evaluation of Autumn's raw EEG failed to reveal any evidence of the frontal slowing which had been so prominent earlier.

However, Autumn sheepishly admitted, "I had a relapse this week." She told Dr. Mark that her symptoms had briefly returned a couple days earlier, following a yogurt snack. When she looked more closely at the carton, she discovered that it contained another artificial sweetener, **sucralose (Splenda)**, to which she was apparently also sensitive!



Dr. Mark Gustafson, a chiropractic physician in Lincoln, Nebraska, addresses the emotional nutritional and detoxification aspects relating to recovery and wellness in his practice. A

Palmer College of chiropractic graduate, Dr. Gustafson researches integrative approaches for chemical sensitivities, heavy metal toxicity and allergies. Dr. Gustafson is certified in acupuncture and LIFE system biofeedback. A consultant for wellness education and kinesiology for over a decade, Dr. Gustafson is an avid runner and has completed nine marathons including the New York City and Honolulu marathons.

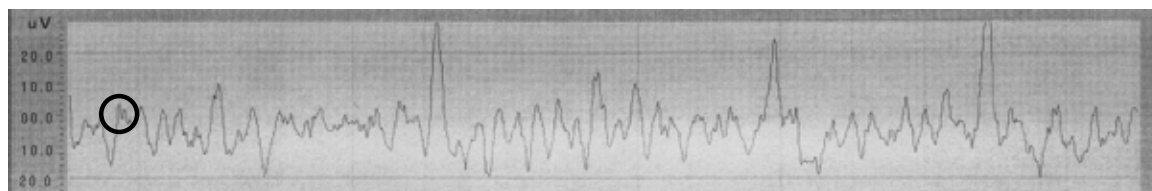


Figure 1: Bipolar EEG, F4-T4 montage. "Toxic" pattern characterized by prominent slow wave activity. Ayers and Montgomery report that they have seen similar EEG patterns in cases of poisoning. One distinctive feature is a pattern of deeply collapsed wave peaks, such as that circled above.

PATTERNS IN THE RAW EEG RESULTING FROM NUTRITIONAL CONCERNS OR SUBSTANCE ABUSE

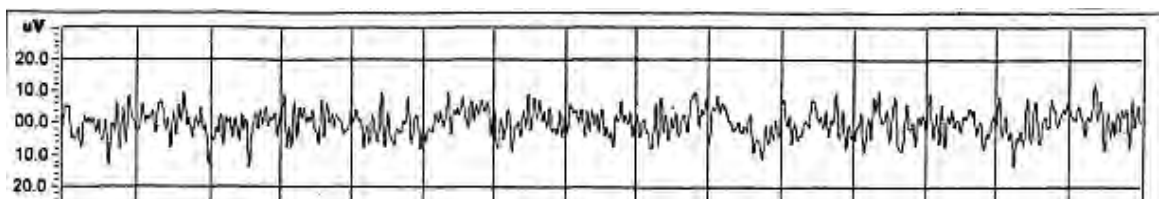
Margaret E. Ayers and Penny S. Montgomery

What we eat or don't affects classroom and on-the-job performance. Since behavior, memory and cognition are determined by brain function, it is reasonable to consider the effects of nutrition and substance abuse on brainwaves. Using Neuropathways all digital real time EEG technology, brainwaves of individuals have consistently correlated with specific states. Even though brainwaves are as individual as fingerprints, patterns or signatures of pathology appear the same in all EEGs.¹

For comparison, a raw EEG from an individual with no evidence of damage or disease is shown.

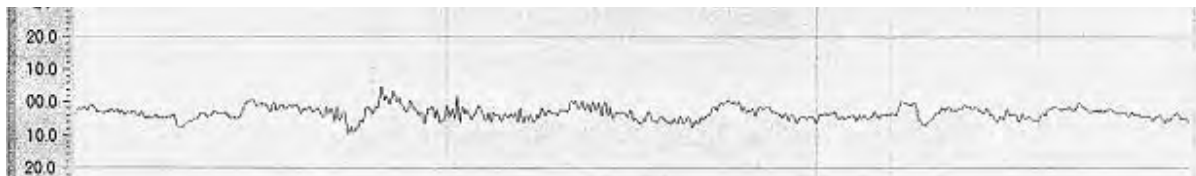


Margaret E. Ayers and Penny Montgomery



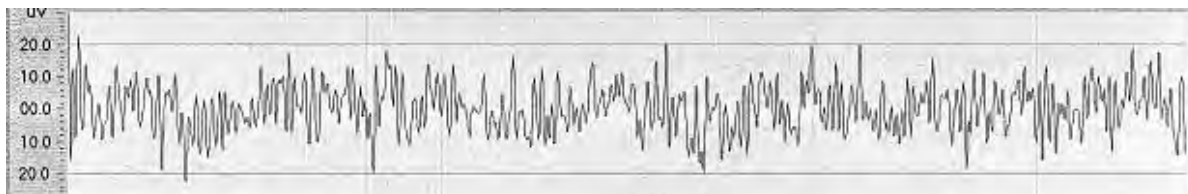
Normal EEG with no evidence of damage or disease Bipolar montage, C4-T4

Starting the day well fed is thought to improve function throughout the day. If there was ever a doubt, the following two EEGs are convincing. The first is from the frontal lobe of a middle-aged woman at about ten in the morning, before eating. Note the extremely low voltage and lack of organized activity.



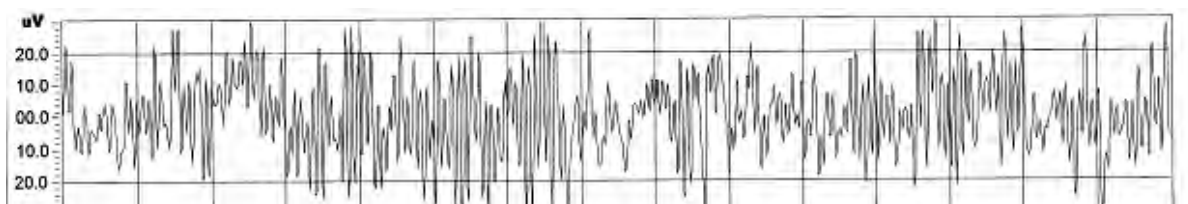
Signature of middle age woman at 10:00am without food since night before Bipolar montage, C4-T4

She was instructed to eat from a breakfast buffet and to include animal protein. The next EEG is about an hour later, after eating. Note dramatically improved voltage and organization of the activity. This demonstrates how important a good breakfast is for school children as it would be very hard to concentrate with such low voltage.



Normal EEG with no evidence of damage or disease Bipolar montage, C4-T4

The signature of allergy appears fast and hyperactive, as does the resulting behavior. This frontal lobe EEG recording demonstrates the signature. It is easy to understand how someone with this high voltage fast activity has difficulty being calm and focused.



Signature of allergy Note bursts of fast (22Hz) high voltage activity Bipolar montage, F3-F4

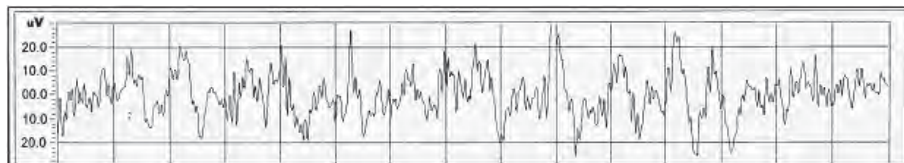
Continued on page 26

PATTERNS IN THE RAW EEG RESULTING FROM NUTRITIONAL CONCERNS OR SUBSTANCE ABUSE

CONTINUED FROM PAGE 25

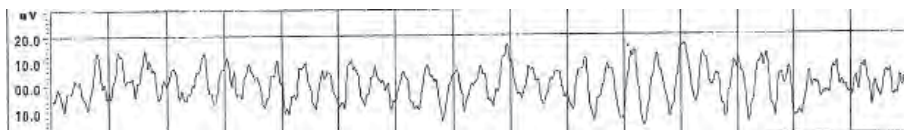
A more comprehensive explanation of this signature may be found in our recent Journal of Neurotherapy paper.²

Alcohol abuse is consistent with a signature characterized by “hooks” which look like “M” at the top of the waveforms. The EEG below from an individual suffering from alcohol abuse for several years demonstrates this characteristic.



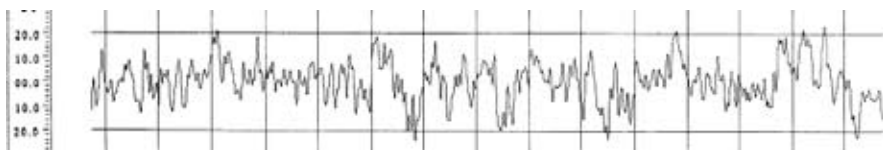
*Signature of damage due to alcohol abuse. Note sharp waves and “hooks” on top of waveforms
Bipolar montage, F4-T4*

Years of severe drug abuse have damaged the frontal lobe of this 40 year-old woman who lives in a nursing home. She has frequent seizures and lacks judgment and social skills. Delta (.5-4Hz) such as seen here indicates severe damage.



*Signature of frontal lobe damage in a 40 year-old woman due to severe drug abuse
Bipolar montage, F7-F8*

Brainwave patterns found in “drug babies” persist for many years and the impaired functioning of these individuals can be better understood from the raw brainwave recording below.



Eleven year-old boy showing effects of maternal substance abuse during pregnancy. Note spikes and sharp waves resulting in slowing and loss of function. This pattern is seen at all electrode sites in “drug babies.” Bipolar montage, C4-T4

From the distinct signatures which accompany specific states, it is obvious that the raw EEG is rich in information vital to clinicians. Such information provides better un-

derstand of the pathology and may guide neurofeedback training to bring about faster and more effective outcomes. In our experience, these signatures can only be seen when the raw brainwaves are recorded on an analog or all digital real time EEG machine. EEG machines using Fast Fourier Transform (FFT) lose this information in the process of averaging samples. Thus, qEEGs cannot display this information, nor can these patterns be readily seen when raw signal is viewed on EEG feedback machines using FFT or quadrature signal processing. However, signatures are very easy to see in the raw EEGs recorded from analog or all digital real time machines.

REFERENCES

1. Ayers, M.E. and Montgomery, P.S. (2004, August), All digital real time EEG patterns. Presentation at the Neuropathways Annual Conference, Manhattan Beach, CA.
2. Montgomery, Penny S. “Allergy Pattern in the EEG.” J. Neurotherapy, Vol. 10(1) 2006.

Margaret Ayers is the inventor of Neuropathways EEG Imaging all digital real time EEG and holds patents in the United States, England, Germany and Japan. She started the first clinic devoted to neurofeedback in 1975. She has an international reputation as an authority in neurofeedback and has written and lectured extensively for the past 30 years.

Penny Montgomery began her work in biofeedback in 1968 and co-authored the first clinical biofeedback text in 1979 with a second edition in 1981. Her focus has been on brainwave biofeedback for the past 30 years. She has taught graduate and medical students and has served as a consultant to the Cleveland Clinic and the Department of the Army.

Margaret Ayers and Penny Montgomery are in private practice. They may be contacted at 427 No. Canon Drive, Suite 105, Beverly Hills, California, 90210. (310) 276 9181.

CALL FOR PAPERS FOR THE 2007 ISNR CONFERENCE

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PART TWO

STUDENT SURVEY OF RECOGNITION OF NEUROFEEDBACK

Kimberly Weeks

This is Part Two of a two-part pilot survey. Part One appeared in the ISNR Newsletter last fall. The purpose of this survey is to explore ways to start to increase visibility of these organizations to attract student members, and to explore how current student member needs are being met.

To recap, Part One of this survey, it focused on getting input from current and recent graduate students in psychology, counseling, and social work where specific instruction in biofeedback (BFB), neurofeedback (NFB) or psychophysiology (PsyP) is not already a curriculum focus or where AAPB/ISNR member faculty do not teach.

Results on Part One demonstrated that none of the thirteen students in Part One of the survey had been aware of either AAPB or ISNR, most had no academic exposure to BFB or PsyP, and none had any academic information about, or even mention of, NFB. Nearly all felt that learning about these modalities in their program would have been helpful to them so that they could be aware of possible interventions. Most also stated that workshops and conferences should be offered. Several respondents even mentioned that a professional organization should be started. Many gave suggestions regarding how professional organizations could help them learn more about the field, including:

- Encourage the development of academic courses devoted to this area of study;
- Encourage more coverage of these topics in textbooks used in related courses;
- Work with academia to help educate students about what BFB, NFB and PsyP are;
- Present information in academic programs;
- Offer training workshops through universities' cooperative education or professional workshop programs;
- Advertise workshops and confer-



ences more widely, particularly those leading to certification;

- Start working with professional CE presentation bodies to increase the visibility of workshops;
- Sponsor workshops for university professors in related fields;
- E-mail students information about it [psychophysiological interventions], along with information about practitioners who use the technique working in the area who might be prepared to come lecture.

For Part Two, the goal was to survey current and recent student members of ISNR and AAPB's Neurofeedback Division or students who attend universities where there is specific instruction in BFB, NFB or PsyP. The faculty members belonging to AAPB and ISNR were also asked to complete the survey from the perspective of their students. Student and faculty recipients of the survey were also asked to assist in distributing the survey among academic colleagues.

Part Two consisted of a 45 yes/no and qualitative/narrative questions and it was distributed via e-mail to 148 current or recent (within the past 2 years) student members and 73 faculty members. Of the 221 surveys distributed, fifteen were returned as undeliverable. Recipients were provided a protocol for ensuring anonymity of their responses and were given two weeks to respond.

Twelve current graduate students and three faculty members responded to the survey. The significant lack of responses in this survey is remarkable, and the responses received cannot be interpreted as being rep-

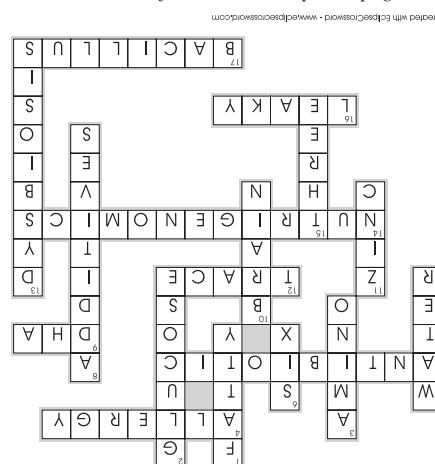
resentative. One respondent indicated feeling the questionnaire was too long, and others reported that two questions were stated in a confusing way, so the survey itself may have contributed to the low response rate. Further exploration on the survey topics is clearly warranted. However, the effort and quality of the 15 responses received warrants their exploration.

Of the 45 survey questions:

- Five questions related to membership in and familiarity with AAPB and ISNR;
- Three questions related AAPB/ISNR membership benefits;
- Two questions related to professional goals and opportunities;
- Six questions related to familiarity with and academic exposure to BFB, NFB and PsyP;
- Two questions related academic opportunity for and support of study of BFB, NFB, and PsyP;
- Three questions related to experience with workshops/conferences/trainings in BFB, NFB, and PsyP;
- Seven questions related to conducting/presenting research in BFB, NFB, and PsyP;
- Six questions related to participation in and support from professional organizations;

Continued on page 29

Answers to Brain food crossword puzzle page 9



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**Robert L. Gurnee, MSW,
BCIA:EEG, QEEG Diplomate,
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PART TWO STUDENT SURVEY CONTINUED FROM PAGE 27

- Six questions related to BCIA certification;
- Three questions related to promoting the field and organizations to students and professionals; and
- Two questions related to how NeuroConnections could meet student needs.
- Additional optional questions related to academic affiliation were also included which included university name and location, student or faculty status, and estimated program completion date for students.

The universities and programs represented by respondents who gave academic affiliation information are:

- Alliant International University, San Diego, CA
- Barry University, Miami Shores, FL
- Capella University, Minneapolis, MN
- Northcentral University, Prescott,

AZ

- St. Mary's University, San Antonio, TX
- Saybrook Graduate School and Research Center, San Francisco, CA
- University of California, Los Angeles, Los Angeles, CA
- University of North Texas, Denton, TX
- University of Tennessee, Knoxville, Knoxville, TN (undergraduate education)
- University of West Florida, Pensacola, FL

Students who replied with this information were pursuing doctoral degrees in: transpersonal psychology, clinical psychology, health psychology, marriage and family therapy, counselor education and supervision, and cognitive neuroscience, with one student completing a master's in psychology.

Two faculty and six student respon-

dents are current members of ISNR and one student is a past member; four of the student non-members had no familiarity with ISNR. Two of the faculty are current members of AAPB and one is a past member. Nine of the students are AAPB members. One student respondent is a non-member of both organizations at this time, but did report future intention to be a member of both. While six students reported learning of these organizations through academic instructors, two listed BCIA as their source of information, three listed non-academic training programs in BFB or NFB, and one listed internet research. Six students indicated having attended AAPB's conference and three of those six have attended ISNR conferences. Eight students plan to participate in AAPB and/or ISNR, while one indicated plans to participate in attendance only.

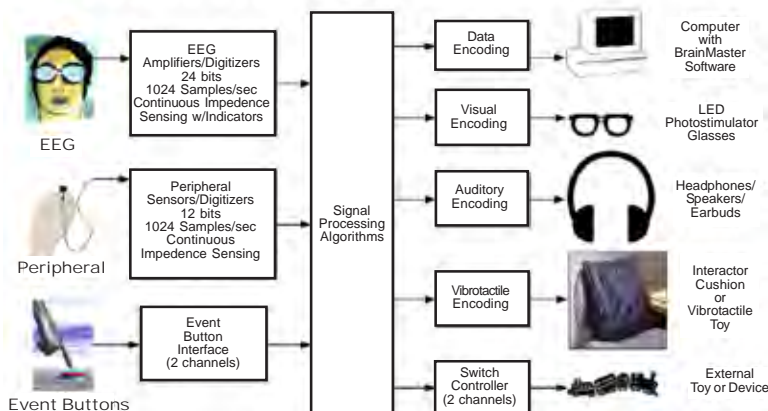
Three students reported that limited resources and organizational benefits play

Continued on page 30

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PART TWO STUDENT SURVEY CONTINUED FROM PAGE 29

a role in determining which organizations they join and/or participate in. Other organizations students listed include:

- American Association of Marriage and Family Therapists,
- American Counseling Association
- American Psychological Association,
- American Society of Clinical Hypnosis,
- Association for Psychological Science,
- Society for Clinical and Experimental Hypnosis,
- Society of Neuroscience
- State and Regional Biofeedback Associations,
- State Counseling & Marriage and Family Therapy Associations,
- State and Regional Psychological Associations.

Thirteen respondents indicated that journals and access to research is one of the most valued benefits of membership. Other valued benefits, listed in descending order of mention, are:

- Networking/community access,
- Workshops and conferences/education,
- Student discounts for conference attendance,
- Opportunities to present research,
- Web resources,
- Travel scholarships, and
- Bookstores.

Benefits and supports respondents proposed for adoption by AAPB and ISNR include:

- Find a way to have more affordable supervision;
- Mentoring offered by qualified members;
- Offer/sponsor equipment training;
- Offer dedicated vendor seminars for students at conferences;
- Increase the availability of hands on training;
- Offer equipment loans for research;
- Establish a professional advisor or liaison;
- Provide ambassadors for students at conferences;

- Improve student networking opportunities;
- Improve opportunities to network with professionals within the field;
- Have student meetings at the conferences;
- Better scheduling of student conference presentations;
- Coordinate more with regional organizations or offer regional student meetings;
- Offer professional guidance or editorial board to instruct on research design;
- Offer assistance in grant writing and learning about accessing research funding;
- Offer reduced-cost new-professional membership category for students transitioning into careers;
- Provide information on and resources for practice building and marketing;
- Offer “free workshops”/ “deeply discounted conferences” for students.

One respondent suggested that offering “high levels of support for student members is nice, since we often travel at our own expense and are sometimes intimidated by the prospect of presenting. So having a social mixer meal/cocktails for students is really great and reduced cost overall is a wonderful thing.”

Faculty members suggested increased scholarship assistance for students, and reaching out more to graduate students across the country.

Thirteen respondents felt that it would be beneficial to establish a student liaison², though one faculty member indicated that it has been tried in the past with lackluster response.

Additionally, students indicated the following additional web resources would be beneficial and appreciated:

- Have student web sections;²
- Have a student listserv, mailing list, or forum;
- Have a database of willing/potential supervisors;
- Have a scientific research library, or at a minimum an online collection of key research articles;
- Provide a password-protected web listing of other student members with location and interests;

- Offer regular web courses for students by experts/pioneers in field;
- Have a database of opportunities to assist in research being conducted in the field;
- Offer outlets for resource sharing on joint projects;
- Have a centralized events calendar;³
- Provide archival access to past conference materials through internet and/or lending library;
- List a timeline of historical developments in the field on the website;
- Create a list of graduate programs friendly to psychophysiology along with faculty research topics;
- Feature student/other research on a rotating basis on the organizations’ homepage;
- Provide clarifications of common terminology;⁴

Student reported that improved benefits would increase the likelihood of their joining or maintaining membership and increasing participation in these organizations. A majority of them indicated that the journal and access to research was too valuable to not continue membership after graduation, with several respondents citing the difficulty of accessing these journals without membership. Other reasons cited for continuing memberships: continued networking and community building opportunities, access to continuing education, promotion of standards within field, and one respondent stated “Because of the investment of the organizations [in] me as a student, I feel obligated to them.”

Related to professional goals, all of the students indicated an interest in direct clinical work utilizing psychophysiological approaches (including BFB and NFB) that they report finding effective and powerful interventions, well supported by research. Half the students indicated that this work would be in private practice. Eight indicated plans to conduct research in their careers, and eight indicated a desire to teach. When asked about perceived limitations to pursuing their professional goals, five students indicated they perceived no limitations. One student noted that in terms of research, no limitations were perceived, but that professional clinical pursuits would be limited by need for additional training. Other students noted the following perceived limitations:

- Difficulty and expense of obtaining supervision;
- Diversity in the field among more research oriented practices and "fringe practitioners";
- Poor insurance reimbursements;
- Poor acceptance by health care providers;
- Lack of awareness outside the field; and
- Need for more research in the field to establish efficacy of psychophysiological interventions.

While two faculty perceived sufficient opportunity for their students to work in this field, one stated, "I am concerned with the obstacles to newcomers in the field. I would like to see a national trend toward licensure in biofeedback/clinical psychophysiology."

One student noted, "Time constraints for full time students, as well as the supervision issues [the cost of and difficulty in finding supervision], and the financial burden dissuade many potential practitioners."

Only three of the twelve students indicated that they feel they have sufficient opportunity for psychophysiological study, clinical practice or research in their academic programs, with one student reporting a need for more clinical internship opportunities. One faculty member indicated that her/his program does not provide enough of these opportunities for students there, and another stated a need to expand training opportunities for students.

Six students are currently involved in academic research in psychophysiology. Another two students are involved in non-academic research. Two students indicated that they might have difficulty incorporating EEG into their academic research. Three students have presented at AAPB, with one of them also presenting at ISNR. AAPB and ISNR have each provided a research grant to one of these students.

Half the student respondents indicated their programs support BCIA certification. Ten have completed didactic requirements and seven have completed other requirements, with three having completed certification. Two students noted difficulty in finding and affording mentoring to complete certification, and two others stated that the exam was very challenging in its' breadth. Two students indicated that they felt certification provided no benefit to them; one of these students has already completed certification, the other does not intend to. One student hoped the arrangement BCIA has in New York with regard to licensure would become more widespread. A faculty member pointed out a need for BCIA to reach out more to student programs on other campuses, and stated "I would like to see this standard more recognized nationally, as there is very uneven quality in the biofeedback services now delivered. The consumer has no other way than certification to know if the provider is well prepared."

In terms of helping students and other professionals learn more about BFB and NFB, many suggestions from Part One of the survey were duplicated. Additional ideas include:

- Increasing visibility of research;
- Work to have psychophysiology added to core curriculum requirements for graduate degree programs;
- Increase and advertise student scholarships;
- Increase visibility by having booths and speakers at psychological, neuroscience and other related conferences;
- Offer didactic training courses in university towns and advertise them at graduate programs with student discounts; and
- Regular distribution of research to academic programs and

libraries, as well as to newsletter producers for related organizations.

It is clear from the responses that were received that there is more both organizations can do to support and meet the needs of student members. Additionally, further examination of ways to increase visibility of both the field and the professional organizations would seem warranted. Perhaps the responses from the individuals who took time to participate in this survey will act as a catalyst to increase conversation in these areas.

For any new recipients of NeuroConnections who are interested in the receiving the article about Part One or the survey or in receiving the Survey questions for Part Two, please send an email request to breetheasy@earthlink.net.

1. I want to express my thanks to the ISNR and AAPB staffs for helping me to reach student and faculty members of these organizations.
2. One student respondent volunteered in web development for students and to serve as student liaison. Because the surveys were blinded, we are unable to reach that student. Please send an email to breetheasy@earthlink.net.
3. An event calendar has been established on the ISNR website. Anyone with an event to post can submit the event on the calendar page of the website.
4. A Lexicon of psychophysiological terminology is in development for use by AAPB and ISNR.

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WELL . . . WHAT IS IT ADD—FOOD ALLERGIES, HEAVY METAL TOXICITY OR ????

Curtis Cripe, PhD

Most of us have been faced with what seemed to be a fairly straightforward presenting set of client complaints that would easily fit an Attention Deficit Disorder profile. When using the DSM-IV criteria and standardized parent and teacher questionnaires such as the Connors Profiles everything appears to line up and a classic set of data points concur. But when we take a look at the client's qEEG the seemingly straightforward pattern often becomes a bit more complex, and may even be more confounding when we apply classic neurotherapy or qEEG guided ADD protocols and find that at best we have either minimal success and/or progress doesn't hold for very long.

Across the country at the Crossroads Institute Centers, we too have been perplexed by cases such as these. Over the years, we have been collecting data in order to determine if there is anything in common among these cases, that may explain the variability of response to similar treatment in seemingly similar patients. As we analyzed our clinical data, we have begun

to learn that environmental and nutritional factors may play a crucial role in explaining these differences. Moreover, we have begun to identify several recurring qEEG patterns that we have come to recognize as indicators of underlying nutritional, toxic or allergic factors which are likely to modulate our client's response to treatment. We have been asked to share some of these emerging findings with readers of NeuroConnections for this special issue.

Have you ever seen Qs like those in fig 1? Unfortunately across the country in the last few years we have begun to find these types of cases to be more common than not, especially cases such as those illustrated by patterns 2 and 4. Of the four cases illustrated in the figure, one is truly and simply an ADD child. The other three are not, but their presenting symptoms could easily be construed as such. Sadly, in all four cases, their teachers and schools insisted that they be put on medications before they were allowed to return to class. One parent refused, the other three com-

plied, with resulting negative medication reactions.

In order to more effectively assess and treat to these types of presenting challenges, at Crossroads we have organized ourselves into multi-disciplinary teams. Each local team consists of a psychologist, a physician with expertise in functional or integrative medicine, a neurodevelopmental specialist and a talented team of electrophysiologists who interpret and integrate qEEG data with the findings from the different disciplines.

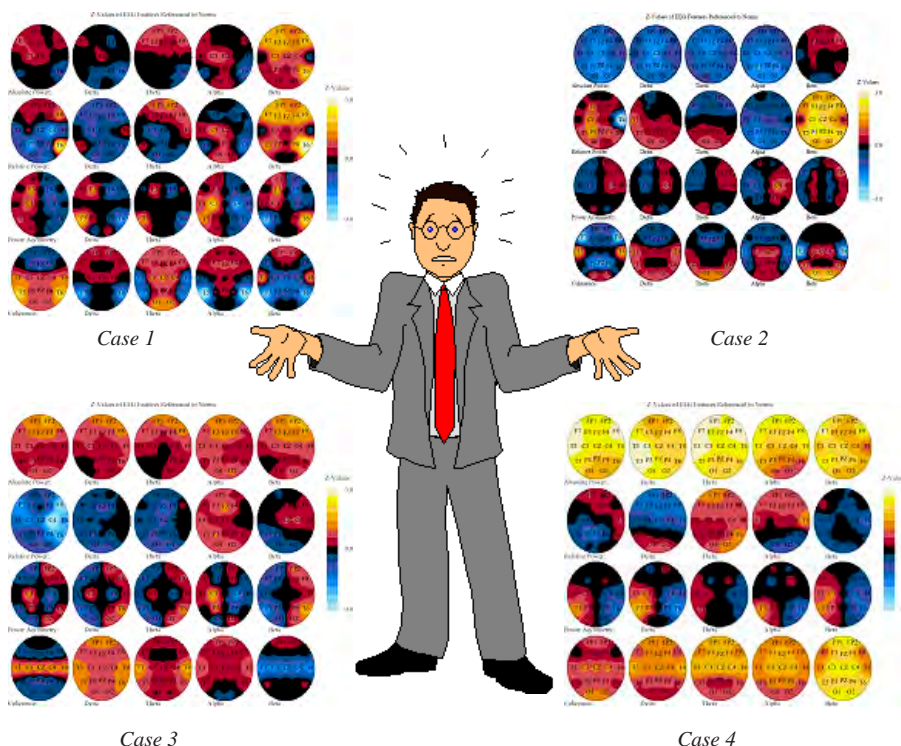
The cases presented in Figure 1 highlight this integrated approach: Case 1 is a classic ADD child who responded to qEEG driven protocols. However, neurotherapy gains didn't hold until the medical team found a significant imbalance in the fatty acids. After additions of the proper balance of Omega 3 supplementation the child responded very well.

Case 2 was a child with heavy metal toxicity which it appeared to have been acquired in utero. This was determined after heavy metal testing with mom as well as child. Additionally, significant developmental delays also were also measured. These which included memory function, auditory processing system, and the ATNR primitive reflex which affected the child's eye tracking ability. The child's attentional and learning issues resolved well in response to an integrated program including neurodevelopmental exercises, heavy metal detoxification (for lead and mercury), and neurotherapy.

In Case 3, the team found an allergy to wheat and milk, which may have accounted for the failure of initial treatment gains to hold over time. After eliminating the allergens from the diet, further testing continued to indicate ADD symptoms, but the child now responded well to a program of neurofunctional training activities and returned to class symptom-free, as long as the diet is maintained.

In Case 4, the team isolated several factors. These which included an active Candida infection in the gut combined with several allergy symptoms related to a "leaky gut" syndrome and significant delay in maturational speed of processing. Combining neuro-functional development activ-

Fig 1. Four different ADD clients presenting with similar complaints and severity of symptoms



ities with neurotherapy and the medical healing protocols allowed the child to gain the proper neurofunctional abilities required be accepted by his teacher with out medications.

As a team, we've collected clinically derived empirical data sets in an attempt to look at many of these issues from a multi-disciplinary perspective. These measures include results from neurofunctional/developmental profiles, allergy profiles, neurotransmitter profiles, standard biomarker medical profiles of liver, heart, and sugar conversion functions, TOVA and any other bio/performance markers we can find. In addition to traditional eyes open and eyes closed qEEG, we utilize functional qEEG during cognitive task, using Evoked Potential and Event Related Potential techniques, with reference to the database developed at the Institute of The Human Brain of Russian Academy of Science by Dr. Juri Kropotov.

To summarize some of the empirical patterns that our team has begun to identify, the following chart will hopefully be useful:

Findings	Absorption	Heavy Toxic	Allergies	Delayed Cortical/SubCortical Maturation	NeuroTransmitter
High Delta or theta or Alpha ADD			X Foods	X	X
High Beta ADD		X	X	X	X
C3/C4 ADD			X		X
High Beta ADD		X	X Chemicals/molds	X	X
Resistive Depression P3/P4 asymmetry	X	X	X Foods Chemicals/molds	X	X
High Beta Anxiety complaints	X		X Chemicals/molds		X
PDD	X	X	X	X	X
Autistic	X	X	X	X	X
Speech Delay	X	X	X	X	X

Curtis Cripe, PhD is the director and founder of Crossroads Institute and its centers and affiliates, which include eight locations in Arizona, Texas, Florida, Maryland, and California. He has published three scientific articles and a recent book chapter on neurotherapy. Curtis Cripe holds a Ph.D. in Research Psychology with emphasis in Neurodevelopment and Psychophysiology as well as a Masters Degree in Aerospace Engineering. He is board certified in Neurodevelopment and Neurotherapy. Due to his early professional years as a top aerospace engineer for NASA's Jet Propulsion Laboratory, Dr. Cripe understands the need for precision and has gained an appreciation for concrete, scientifically-based (objective) measures.

The CrossRoads Clinic, PLC

<http://www.crossroadsinstitute.org>
ctcripe@att.net

BEST PRACTICES: AN INTERVIEW WITH ROBERT COBEN, PHD

Michael Gismondi, MA

INTRODUCTION

Dr. Robert "Rob" Coben is one of the most successful and highly regarded neurofeedback (NFB) practitioners in the greater New York City area. He is a UCLA-trained neuropsychologist whose innovative clinical work and outcome research in the growing area of the autistic spectrum has garnered him national attention. Like most neuropsychologists who offer NFB services, Dr. Coben also specializes in the diagnosis and treatment of ADHD, closed head injuries, stroke, specific learning disabilities and other neurobehavioral and neurological disorders. He is known for his innovative work with HEG both in terms of documenting it's clinical effectiveness as an ancillary to NFB, as well as it's neuroimaging via thermal cameras/infrared imaging. Over the years Dr. Coben has developed a close working relationship with Dr. William Hudspeth, and Rob's clinic serves as a key test site for Hudspeth's software innovations in the mapping of cortical connectivities, i.e, multivariate measures of coherence relations. Dr. Coben impressed me as being one of the most articulate individuals I have ever interviewed for this "BEST Practices" series.



MG: What got you interested in working with coherence training?

RC: In my experience, more traditional NFB, focusing on amplitude training and normalization, does a very good job at addressing dysregulations of arousal, impulse and affect. It can also enhance learning abilities and academic performance via it's impact on attention control systems in the brain. What I noticed

over time, however, is that if the presenting problem included problems with memory, language processing, visuospatial analysis and/or problem solving, or complex presentations as we see in autism, the success rate is more tenuous. Success rates of about 75% may be expected, and the number of sessions to get reasonable results increases to an unacceptable level. As a result, I was inclined to seek out ways to increase my success rates with these specific cognitive or learning difficulties, while at the same time reducing treatment times to a more desirable level. Addressing more specific locations of dysfunction, and the precise disconnects and lack of coordination between parts of the brain needed for a specific cognitive function or modality, became an important part of my work. Using the techniques that I do, I notice an increase in treatment effectiveness with both the more specific cognitive problems as well as the more "general" complaints such as simple ADHD or certain mood disorders. Let me be more specific here. It is important to not just look at percentages of "success" vs. "non-success" within a given population.

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BEST PRACTICES: AN INTERVIEW WITH ROBERT COBEN, PH.D CONTINUED FROM PAGE 33

We are concerned with not only the percentage of people that improve, but also degrees of success achieved, across populations. Take the case of informing parents how much you can improve an ADHD child's level of functioning, based on our outcome criteria that may include continuous performance tests, QEEG profiles, neuropsych testing, subjective rating scales and symptom checklists, and so on. I don't think we as a field have a good understanding, much less a shared vocabulary or set of metrics, to talk about how much improvement we can offer. Said another way, our field hasn't really grasped this distinction between general NFB vs. more specialized treatment refractory population NFB, and NFB approaches that succeed where more general approaches fall short or fail altogether. It is one thing for us to say "NFB will greatly reduce ADHD symptoms 75% or 80% of the time vs. "we will take your child to a specified level of cognitive function 90% of the time".

MG: Who will be in a position to really address these degrees of cognitive performance improvement?

RC: Once you move beyond remediation of impulse and attention problems, you move beyond what most traditional NFB is really prepared to assess effectively. Reliance on TOVAs and IVAs fails us past that point. The clinicians who are able or interested in moving beyond these limits have a variety of ways of assessing client functioning and documenting progress, everything from sophisticated neuropsych batteries to certain computerized tests that don't really require a neuropsychologist to administer. Objective measurement of progress can also be done through parent ratings scales, personal inventories, neuropsychological testing, and pre- and post- QEEG or other neurophysiological measurements. The key is to monitor progress in some objective fashion.

MG: So what do you use, and why?

RC: In my clinic, we have the advantage of two full time neuropsychologists on site and we have come to rely upon multiple and varied paper and pencil tests that best fits the client's needs. We like the flexibility. Also, there has been a research track over the last 15 years that has asked the question do computerized tests measure the same thing that established paper and pencil tests do, and the results of that research so far has been less than conclusive. There is clearly a movement, though,

towards computerizing neuropsychological measures. That said, we have to get more specific and move beyond thinking in terms of "good tests" vs. "bad tests". For example, the Micro-Cog, a computerized cognitive battery is not a bad test per se, but it is designed and normed for adults, so when it is used to assess children, that's a problem. Also, some tests, computerized or not, are fine for measuring some kind of change, but you can't make strong statements about how far the treatment was able to take a client in some normed way, across some developmental dimensions. That is one of the reasons for a more sophisticated neuropsych before-and-after evaluation. IntegNeuro, a computerized cognitive test battery by the Brain Resource Company, is very useful, but does not cover all neuropsychological or other related functions. Now, for NFB offices where there is, for one reason or the other, no ready access to a good neuropsychologist, some of these computerized tests like Integ-Neuro or even Micro-Cog could be a real improvement compared to depending upon just TOVA or IVA.

MG: Kirt Thornton's cognitive activation QEEG system has emphasized listening and reading "free recall" memory tasks, both immediate and delayed. These kinds of tasks seem to possess, for lack of a better term, face validity. What would you say about the concern that many established neuropsych tests don't capture "real world" cognitive or academic performance variables?

RC: The ecological validity of neuropsychological tests has been estimated at about .40 - .60. That is the correlation between test scores and everyday functioning. Neuropsychological testing has been shown to be a good, if not the best, predictor of school, work or social functioning. When you add neurophysiology (QEEG data) to this your predictions become much better. Testing takes place in a relatively "contrived" setting, in a quiet room with no distractions with a supportive person doing the administration and observation. And don't forget much of the art of this type of testing involves getting the best performance possible from the client, giving extra time, or coaching or giving feedback...the "testing of limits" so to speak, so all that must be factored in if your goal is true ecological validity. But I will answer your question this way; it is the combining of neuropsych test data with Hudspeth's advanced QEEG

information about amplitudes but especially connectivities associated with parts of the brain thought to be relevant to a presenting problem, that really makes things work for us. The combination of Neuropsychological and QEEG data is very powerful in assessing clients and measuring progress.

MG: What do you think about cognitive task activated QEEG?

RC: I have tried cognitive activation, but when you use it, noise and artifact is a problem. During reading or math or even recalling digits backwards, we observe all sorts of subtle and not so subtle eye movements, vocalizations, and so on. Now, when you then try to compare the activation record with the resting EEG, teasing out the true cognitive activation from artifact can be a challenge. Making inferences as to what types of cognitive processes were in fact taking place in a certain part of the record is another issue that has to be examined. What I have preferred to do, in keeping more with Jon Walker's model, is to get as specific as possible about the parts of the brain that are most involved or specialized in a particular cognitive deficit, and you can assess the amplitudes (or power) and the connectivities between the different parts of the neural circuit, at rest, and detect the problem that needs to be addressed by NFB.

If you can see the EEG components of the cognitive weakness without task activation, why take on the overhead of additional artifact? I think with the capabilities of Bill Hudspeth's software we can get good at assessing the different views and types of connectivity so that seeing the problem that needs to be addressed via NFB becomes quite manageable. While a complex skill like reading may involve various brain functions and systems, I find that usually one or two parts or phases of the information processing loop are the main culprits, and when you find and normalize them, the brain improves and so do the related abilities. And through the use of Bill's newest software, we can usually see where that key obstacle is, even at rest.

MG: I understand that some basic arousal, perceptual, associative and attention functions have been localized fairly successfully. The fact still remains that almost any given part of the brain plays host to multiple simultaneous local and more globalized activities of a higher order nature like memory and executive processes and lower order activity. With an eyes-closed recording with

no cognitive activation to anchor it, how can you be sure you are looking at the impaired cognitive information processing activity you think you are looking at?

RC: First, it is clear that certain brain systems are devoted to certain cognitive or other functions. It is important to understand these multiple systems, how they interrelate and the functions subserved by different brain systems.

Next, is the question of how we know if a QEEG finding is related to a specific cognitive function. QEEG does not measure cognitive processing, it measures brain processing. Neuropsychological or other testing is the only way to objectively measure cognitive functioning. In our work, we assess and conceptualize the cognitive defect with neuropsychological testing, use QEEG to assess for neurophysiological defects, and use our knowledge of neuroscience to relate to two. When these match up, we then have our target for NFB intervention.

MG: OK, let's turn for a moment to the idea that Hudspeth has found a way to not depend upon a normative database to get treatment planning information; how is that possible?

RC: I use norms, but I also use non-normed data. EEG/QEEG analysis should include quantitative analysis, but also an analysis of the raw EEG and views that are conceptual in addition to numerical. In terms of the communication between and across neural systems, Bill's software allows us to go beyond pair-wise coherence comparisons and look at multivariate relationship patterns in the brain so we can determine the direction of the information processing problem "flow" as well as the system of sites involved with a given problem. Autism is an interesting problem in that unlike most learning disabilities or head injuries where most of the problems involve hypo-coherence or under-connectivities, autism seems to involve a simultaneous combination of over and under connectivities. You want some kind of visual tool to see how the under and over-connectivities interact simultaneously, and Hudspeth has done just that with what he calls his "neuro-electric image", where you can visually inspect individual site and more "systemic" deviations. It's the distance that given electrodes deviate from the 10-20 system that alerts you to the individual and systemic anomalies.

MG: In that way, analogous to the distinction between relative vs. absolute magnitudes or power, Bill's approach allows

you to look at relative connectivity distortions within the client, whereas most other QEEG reports only look at coherence and phase as "absolute" numbers or values.

RC: Yes. And with Bill's system, we are not limited to pairs of homologous sites or pairings of any kind as other QEEG reports or databases are. There was a study done by Robert Barry and Adam Clarke that showed how the physical distance separating different sites will effect the coherence values. Any system that measures coherences must take that into account, as Hudspeth does, where every electrode is compared with every other electrode, both intra-hemispherically and inter-hemispherically. The strength of connectivity relationships under study is averaged and gives each site its overall connectivity "score" if you will. This way we eliminate physical distance between electrode sites as a confounding variable. It is important to interpret all QEEG connectivity data in the context of our knowledge of neuronal pathways. Also, pairwise notions of coherence imply a linear quality while the connections in the brain are in fact curved. Spatial and anatomical issues are important.

MG: That sounds similar to what Kirt Thornton calls his "flashlight values" for a site.

RC: Yes, and I want to be clear, I don't think Kirt's cognitive activation approach is "wrong" or that it isn't effective, it's just that I find I don't need to do that because I expect a good neuropsych testing work up to be done before I do the Q, and it helps me isolate the probable sources of EEG anomalies without the overhead of additional artifact risk that is a concern with any cognitive activation approach. Kirt's and Bill's approaches are both multivariate in nature. A head-to-head comparison would be interesting. What is not clear, is how much of Kirt's results are a function of his use of cognitive activation vs. his use of a more multivariate vs. pair wise approach to coherence mapping. Another variable is the importance of the high hertz work he does above say 32 hertz, which I don't currently do. Now, Jon Walker and his team don't use a multivariate approach or high hertz work at all, and they get very good results too. I think the common factor with clinicians who get superior results with higher-order cognition problems is the use of some type of connectivity training, but we need to do the comparative research to nail down what is contributing what to


which treatment outcomes.

MG: Since you have spoken about your working closely with Bill Hudspeth and Bill's on-going software innovations, is there any new feature of Bill's NeuroRep software that you particularly value or helps you deal more effectively with difficult populations like, say, autism?

RC: Actually, yes. There is a new feature called the "spectral sort" that is very intriguing. This was the main program we used for our Mu oscillation study in autism. What it does is it allows you to take parts of the record with oscillations of magnitude or power over a given location - say the left temporal region in a case of dyslexia - and analyze the difference between high and low oscillations in terms of connectivity. This gives us a clearer view of the connectivity problems underlying magnitude excess and abnormalities. I'll elaborate. You highlight whatever power or magnitude irregularity is of greatest interest. For example, let's say you have a LD case and you run your Q study and you see in the posterior left area excessive theta. In spectral sort, you then highlight the electrodes where this problem is, and highlight the frequency range where the problem is located, and the program automatically isolates only those variables, and it performs a median split of the activity, and then divides the waveforms into high and low segments. Now you have higher and lower oscillations within that Theta band, and separate that out into two files, i.e., the times when the oscillations are surging too much as compared to the times when they are within acceptable ranges. We now have two files, then we do a difference comparison between the two files and look at the coherence abnormalities that are magnified when there is a high vs. low oscillation. This allows us to pinpoint with great specificity what mechanisms are going wrong in the brain. It shows us what are the coherence abnormalities we need to be most concerned with and gives us "cognitive task activation-like" information.

MG: In capturing the brain in the act, so to speak, in it's coherence abnormalities in this manner, you are adding the variable of power fluctuations or "variability" to the analysis of power and connectivity data.

RC: Yes, the brain works in an oscillatory fashion. The underlying mechanisms for these changes should be our target or targets.

MG: Thanks for your time, Rob and best of luck with your work! 

NUTRITION AND NEUROFEEDBACK

Geir Flatabø, MD

My Road to Neurofeedback and EEG was through nutrition.

As an MD, and even before that, I have always been interested in other ways of managing ill health than mere medication. Modalities where you could get healthier through some effort of your own seemed particularly attractive to me.

One technique is acupuncture, another is healthy eating, yet others are nutritional supplements, herbs etc. Some of these are technical like pills, but dietary supplements differ from pharmacological drugs by in general being physiological and natural, while drugs most often are synthetic and impose possible unknown threats and more often unwanted side effects...

Healthy eating i.e., getting enough of every nutritional item necessary for good health through food seems the obvious right choice for everyone. Still, unhealthy eating is apparently preferred by most people, who prefer tastes of sweet and some stimulating effects of food and drink – addiction eating, from all that you can choose in a modern supermarket. For some people it is, of course, also a question of lack of money to spend on the right kinds of healthy food.

Fifteen years ago I read about professors Bjørn Ellertsen and Hallgrim Kløve at the University of Bergen, Norway. They were treating ADHD, epilepsy and headaches by relaxation techniques, called Biofeedback, with remarkable success. These were problem diagnoses I would have attacked with nutritional means, diet and/or supplements (and acupuncture and pills).

The intriguing thought came to me, that if both nutrition and biofeedback had documentation showing about 80% effectiveness, what would happen if you combined them. I had to learn biofeedback, what it was, and what it could do.

There was not too much reading to do before I was convinced—applied neurophysiology like practiced in Neurofeedback was convincing, and had a sound scientific basis, but most of all intriguing, fascinating and exciting - to a complete newbie like myself.

I read among Barry Sterman's early basic findings in his cat studies that rocket fuel, dimethylhydrazine, also fueled epileptic seizures. These seizures were partly pre-

ventable by operant conditioning training, and were the foundation on which SMR rhythm training and neurofeedback later grew and developed. But he also found that these seizures were preventable by giving them Vitamin B6, Pyridoxine.

There was a link.

German studies on pathologic seizure activity in EEG in celiac children showed that it took a year using a gluten free diet to abolish pathologic EEG and normalize EEG. (Possibly, some neurofeedback would have made this transition faster).

It is well known that other drugs may have a significant effect on EEG. Diazepam is most often mentioned, and produces an increase in beta wave readings...

Recently a video made by Stan Kurtz showed rapid normalization of the EEG after administering vitamin MethylB12 nasally to a young man diagnosed with ADHD. Jack Johnstone of Q-Metrx used him as an example of procedure and real EEG changes. (www.childrenscornerschool.com/recoveries.htm)

When questioned about how Neurofeedback training can help, I always refer to studies done, and then state: theoretically NF might have an impact on any disease where brain waves are involved. This is because brain waves more or less mirror everything; chemical activity and physiologic activity.

Nutrition is chemistry. The main problem is the diversity, and complexity. All the different chemicals in all the strange foods, and gut microbial activity add to the complexity. The point is that too little or too much of nutrients, and too much toxic food or non food substances like gluten and/or pyridoxine in epilepsy, or Vitamin B12 in ADD might also show up in brainwaves.

New scientific literature findings in ADHD (not correlated to the subgroups) is lack of various minerals like Mg, Zn, Fe, Sulphate, Vitamins like B6, and/or increased lead. This is due partly to picky eating, lots of sugar, and low protein food. Supplementing these deficiencies leads to obvious symptom relief. To me it would, in a way, be wrong to use neurofeedback to suppress symptoms of nutritional imbal-

ances and/or toxic overload.

Still I will do neurofeedback if that is what the client wants, but also use the opportunity to provide nutritional information. I often find patients reluctant to try some kind of restrictive diet, or costly supplement, and they may also change focus during neurofeedback training.

To combine nutritional counselling and NF has great positive impact. I would still prioritise improving chemical imbalance. But not all patients want to quit dairy and wheat as they are not convinced that it is the cause of their problem and it is difficult to alter their diets to accommodate the allergy.

If you have little knowledge about nutritional impacts on the diseases you manage in your practice, referring to a nutritionist would perhaps be the way the most stubborn cases could be helped. This may be the case when 20 or 60 NF training sessions have not made a big difference. Your success with others might also improve—they get results with NF faster.

Nutritional counselling and supplements are very seldom dangerous. There are wider limits in trial and error than in ordinary pharmacological treatments, and most supplements are virtually non toxic.

My basics are: perhaps everyone should use CLO - Cod liver oil, and ordinary multi vitamin mineral supplementation—further supplementation would then depend on lab findings, and/or the symptoms being treated. Empowerplus (www.truehope.com) is an all-round high dosed supplement with good reputation and some science behind it.

And regarding diet: perhaps no one should drink milk or eat food with milk in it. When there is any chronic illness a diet without dairy ought to be tried for some time.

And if your client already has done a nutritional workout without significant success, then it is time for NF—or other biofeedback approaches. *M*

