NeuroRegulation

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KEYNOTE PRESENTATIONS

Functional Neuroimaging as a Window into Human Brain Function: Applications to Better Understand and Optimize Neuromodulatory Therapies

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Functional brain imaging has opened a window into brain function in humans and has significantly enhanced our understanding of neural function and connectivity supporting aversive symptom states. Our research has shown that the brain is composed of multiple primary sensory and associative networks that activate and deactivate over time as distinct assemblies. These networks can become blurred when chronic, recurring activation of network nodes is maintained. For example, recurring, spontaneous pain in a distinct body area brings saliency to specific somatosensory and nociceptive input, blurring the Salience Network (SLN) and somatotopically-distinct subregions of the Somatomotor Network (SMN). Additionally, catastrophizing about pain activates the Posterior Cingulate Cortex (PCC) and brings saliency to ruminative thought, blurring SLN and default mode network (DMN). Functional MRI brain connectivity metrics can be used to evaluate objective brain-based markers that track with clinical pain. Applications include objective markers of disease for drug and non-pharmacological/behavioral intervention trials, baseline predictor of response, and targets for neurofeedback.

References

Harris, R. E., Napadow, V., Huggins, J. P., Pauer, L., Kim, J., Hampson, J., … Clauw, D. J. (2013). Pregabalin rectifies aberrant brain chemistry, connectivity, and functional response in chronic pain patients. *Anesthesiology, 119*(6), 1453–1464. http://dx.doi.org/10.1097/ALN.0000000000000017

Kim, J., Loggia, M. L., Cahalan, C. M., Harris, R. E., Beissner, F., Garcia, R. G., … Napadow, V. (2015). The somatosensory link in fibromyalgia: Functional connectivity of the primary somatosensory cortex is altered by sustained pain and is associated with clinical/autonomic dysfunction. *Arthritis & Rheumatology, 67*(5), 1395–1405. http://dx.doi.org/10.1002/art.39043

Kim, J., Loggia, M. L., Edwards, R. R., Wasan, A. D., Gollub, R. L., & Napadow, V. (2013). Sustained deep-tissue pain alters functional brain connectivity. *Pain, 154*(8), 1343–1351. http://dx.doi.org/10.1016/j.pain.2013.04.016

Loggia, M. L., Kim, J., Gollub, R. L., Vangel, M. G., Kirsch, I., Kong, J., … Napadow, V. (2013). Default mode network connectivity encodes clinical pain: An arterial spin labeling study. *Pain, 154*(1), 24–33. http://dx.doi.org/10.1016/j.pain.2012.07.029

Napadow, V., & Harris, R. E. (2014). What has functional connectivity and chemical neuroimaging in fibromyalgia taught us about the mechanisms and management of "centralized" pain? *Arthritis Research & Therapy, 16*(4), 425. http://dx.doi.org/10.1186/s13075-014-0425-0

Napadow, V., Kim, J., Clauw, D. J., & Harris, R. E. (2012). Brief report: Decreased intrinsic brain connectivity is associated with reduced clinical pain in fibromyalgia. *Arthritis & Rheumatology, 64*(7), 2398–2403. http://dx.doi.org/10.1002/art.34412

Napadow, V., LaCount, L., Park, K., As-Sanie, S., Clauw, D. J., & Harris, R. E. (2010). Intrinsic brain connectivity in fibromyalgia is associated with chronic pain intensity. *Arthritis & Rheumatology, 62*(8), 2545–2555. http://dx.doi.org/10.1002/art.27497

Impact of Childhood Maltreatment on Brain Development and the Critical Importance of Distinguishing Between Maltreated and Non-Maltreated Diagnostic Subtypes

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Childhood adversity is the most important preventable risk factor for mood, anxiety, personality, substance abuse, and psychotic disorders. Recent studies suggest that clinical sequelae may stem, at least in part, from enduring effects on brain development. Research will be reviewed highlighting the effects of childhood abuse on EEG coherence and the development of the hippocampus, white matter tracts, and cortical regions. Evidence will be presented identifying sensitive periods when specific brain regions are most vulnerable and unique effects of difference types of abuse on sensory systems and pathways that convey the adverse experience. These findings will be placed into context illustrating how exposure to abuse affects multiple components of the brain circuit responsible for threat detection and also affects the network architecture of the brain. Finally, the case will be made that maltreated and non-maltreated individuals with the same primary DSM diagnosis differ clinically, neurobiologically, and genetically. We refer to the disorder in the maltreated cohort as an *ecophenotype* and show that it is associated with earlier age of onset, more severe course, more comorbid diagnoses, and poorer response to first-line treatments. Recognition of this distinction may be of paramount importance in effectively identifying appropriate interventions with neurofeedback emerging as a key modality for treating individuals with the ecophenotype.

References

Teicher, M. H., & Samson, J. A. (2013). Childhood maltreatment and psychopathology: A case for ecophenotypic variants as clinically and neurobiologically distinct subtypes. *The American Journal of Psychiatry, 170*(10), 1114–1133. http://dx.doi.org/10.1176/appi.ajp.2013.12070957

Teicher, M. H., & Samson, J. A. (2016). Annual Research Review: Enduring neurobiological effects of childhood abuse and neglect. *The Journal of Child Psychology and Psychiatry, 57*(3), 241–266. http://dx.doi.org/10.1111/jcpp.12507

Teicher, M. H., Samson, J. A., Anderson, C. M., & Ohashi, K. (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience, 17*, 652–666. http://dx.doi.org/10.1038/nrn.2016.111

The Evolution of Quantitative EEG: A Perfect Storm

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The historical evolution of QEEG will be explored with emphasis on significant steps its development. The following will be highlighted: 1) Early steps in quantification that pave the way, including normative equations (John et al., 1980), source localization (Pascual-Marqui, Esslen, Kochi, & Lehman, 2002), and Default Mode Network (Buckner, Andrews-Hanna, & Schacter, 2008); 2) QEEG treatment predictive biomarkers, including cognitive decline (Jelic et al., 2000; Prichep et al., 2006) and OCD (Dohrmann, Stengler, Jahn, & Olbrich, 2017); 3) QEEG as a surrogate for advanced neuroimaging, including TBI (Hanley et al., 2017) and chronic pain (Prichep et al., 2017). Impact of the “perfect storm” represented by advances over the last decade in technology, signal processing, and machine learning classification methodologies will be discussed in this context.

References

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The Brain's Default Network: Anatomy, Function, and Relevance to Disease. Annals of the New York Academy of Sciences, 1124, 1–38. http://dx.doi.org/10.1196/annals.1440.011

Dohrmann, A.-L., Stengler, K., Jahn, I., & Olbrich, S. (2017). EEG-arousal regulation as predictor of treatment response in patients suffering from obsessive compulsive disorder. Clin Neuropsychol, 128(10), 1906–1914. http://dx.doi.org/10.1016/j.clinph.2017.07.406

Hanley, D., Prichep, L. S., Bazarian, J., Huff, J. S., Naunheim, R., Garrett, J., … Hack, D. C. (2017). Emergency Department Triage of Traumatic Head Injury Using Brain Electrical Activity Biomarkers: A Multisite Prospective Observational Validation Trial. Academic Emergency Medicine, 24(5), 617–627. http://dx.doi.org/10.1111/acem.13175

Jelic, V., Johansson, S.-E., Almkvist, O., Shigeta, M., Julin, P., Nordberg, A., … Wahlund, L.-O. (2000). Quantitative electroencephalography in mild cognitive impairment: Longitudinal changes and possible prediction of Alzheimer's disease. Neurobiology of Aging, 21(4), 533–540. http://dx.doi.org/10.1016/S0197-4580(00)00153-6

John, E. R., Ahn, H., Prichep, L. S., Trepetin, M., Brown, D., & Kaye, H. (1980). Developmental equations for the electroencephalogram. Science, 210(4475), 1255–1258. http://dx.doi.org/10.1126/science.7434026

Pascual-Marqui, R. D., Esslen, M., Kochi, K., & Lehman, D. (2002). Functional imaging with low resolution brain electromagnetic tomography (LORETA): A review. Methods & Findings in Experimental & Clinical Pharmacology, 24C, 91–95. http://www.brainm.com/software/pubs/brain/loreta/LORETA-ReviewPaper03.pdf

Prichep, L. S., John, E. R., Ferris, S. H., Rausch, L., Fang, Z., Cancro, R., … Reisberg, B. (2006). Prediction of longitudinal cognitive decline in normal elderly with subjective complaints using electrophysiological imaging. *Neurobiology of Aging,*

27(3), 471–481. http://dx.doi.org/10.1016/j.neurobiolaging.2005.07.021
Prichep, L. S., Shah, J., Merkin, H., et al. (in press, 2017). Identification of Chronic Pain Matrix Using Quantitative EEG Source Localization. *Clinical EEG and Neuroscience*.

INVITED PRESENTATIONS

Early Detection and Treatment of Attention Deficits in Preterm Infants

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This study described the application of a scale for evaluation and treatment of early attention deficits in infants, the "Infant Scale of Selective Attention" (EEAS). It is well known that attention deficit begins in infancy and adversely affects individuals throughout life; thus, the challenge is to find ways to diagnose and treat it early in life, during infancy, to try to prevent children from developing attention-deficit/hyperactivity disorder. EEAS measures the infant's overall ability to detect, locate, track, and respond selectively to visual and auditory stimuli. Also, an intervention program was designed to stimulate attention in infants with delayed attention. This program was applied daily, from 3 to 8 months corrected age. Monthly behavioral measures from 3 to 8 months and event-related potentials (ERP) recordings for a two-tone oddball paradigm were collected in 10 full-term and 21 preterm infants with white matter injury and attention deficits. Eleven preterm infants participated in the attention stimulation program (experimental group) and 10 did not (control group). The behavioral study showed that the experimental group had a faster rate of improvement in attention than the control group. ERPs showed that deviant stimuli were automatically detected and could involuntarily capture attention but only in the healthy and treated groups.

References

Gomes, H., Molholm, S., Christodoulou, C., Ritter, W., & Cowan, N. (2000). The development of auditory attention in children. *Frontiers in Bioscience, 5*, 108–120. https://www.bioscience.org/2000/v5/d/gomes/fulltext.htm
Gutiérrez-Hernández, C. C., Harmony, T., Avecilla-Ramírez, G. N., Barrón-Quiroz, I., Guillén-Gasca, V., Trejo-Bautista, G., & Bautista-Olvera, M. M. (2017). Infant Scale of Selective Attention: A Proposal to Assess Cognitive Abilities. *Evaluar, 17*(1), 94–106. Retrieved from https://revistas.unc.edu.ar/index.php/revaluar/article/download/17077/16708
Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology, 118*(10), 2128–2148. http://dx.doi.org/10.1016/j.clinph.2007.04.019
Reynolds, G. D., & Romano, A. C. (2016). The development of attention systems and working memory in infancy. *Frontiers in Systems Neuroscience, 10*, 15. http://dx.doi.org/10.3389/fnsys.2016.00015

Functional Neuromarkers for Psychiatry and Neurology: Defining Brain Dysfunctions and Constructing Protocols of Neuromodulation

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The paper describes a recently emerged methodology of extracting functional neuromarkers from spontaneous multichannel EEG, event-related de/synchronization, and event-related potentials (ERP). The methodology includes methods of blind source separation for artifact correcting and extracting latent (hidden) components from resting-state EEG and from event-related potentials, methods for constructing normative and patient databases, for comparing the extracted individual parameters with the normative data, as well for pre-post comparison. The high test–retest reliability of neuromarkers, the high level of specificity and sensitivity for defining dysfunctions in ADHD, schizophrenia, OCD, autism, depression, and dementia are described. Application of the methodology for predicting clinical outcome in response to pharmacological medication, for constructing protocols of neurofeedback, tDCS (transcranial Direct Current Stimulation) and TMS (transcranial magnetic stimulation) in clinical population is presented.

References

Kropotov, J. D. (2016). *Functional neuromarkers for psychiatry: Applications for diagnosis and treatment*. Amsterdam, Netherlands: Academic Press, Elsevier.

STUDENT AWARD WINNERS – PLENARY PRESENTATIONS

Is A/T Neurofeedback Training (NFT) a Successful Treatment Method for Women with Moderate to Severe Trait Anxiety: A Clinical Trial and Methodological Considerations

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Since the late 1960s neurofeedback (NFT) has been used to treat adult individuals with anxiety disorders. Yet most related, evidence-based research studies

were conducted between the mid-1970s and late 1990s. Therefore, NFT as an efficacious treatment for anxiety problems remains unclear. The literature research discloses that of few studies, most used sample sizes 10 subjects or less per experimental or control group, results have been mixed, and the U.S. NIH's National Center for Complementary and Integrative Health (NCCIH) at this time does not endorse NFT as an efficacious treatment for anxiety problems.

In the present study 27 women between (age range = 19–67) with moderate to high trait anxiety were randomly assigned to either experimental or control condition, and received either 10 sessions of A/T NFT to up-regulate Theta (5–7 Hz) and Alpha power (8–11 Hz) or received ten 25-min sessions of alternately up- and down-regulating beta (15–19 Hz) and hibeta (20–24 Hz), respectively, at the Pz location, while getting auditory and visual feedback. Activation/Deactivation was assessed before and after each session via the Activation Deactivation Adjective Checklist (AD-ACL) list. Pre- and post-EEGs, anxiety (BAI, STAI, GAD-7), treatment expectancy, locus of control, and a variety of qualitative measures such as cognitive strategies, treatment group belief, and best times and worst times of day for learning were assessed.

Preliminary results using growth curve modeling (GCM using lmer), as well as traditional 2x2 and 2x3 ANOVAs and regression statistical analyses, indicate that both participants of experimental (EG) and control groups (CG) were able to successfully up-regulate their theta and alpha power, as well as the T/A ratio during the course of a session as well as over the course of the 10 treatment sessions. Self-perceived anxiety as measured by the two of the three anxiety measures went down significantly. No significant difference between EG and CG could be observed.

References

Aliño, M., Gadea, M., & Espert, R. (2016). A critical view of neurofeedback experimental designs: Sham and control as necessary conditions. *International Journal of Neurology and Neurotherapy, 3*(1), 041. http://dx.doi.org/10.23937/2378-3001/3/1/1041

Alkoby, O., Abu-Rmileh, A., Shriki, O., & Todder, D. (2017). Can we predict who will respond to neurofeedback? A review of the inefficacy problem and existing predictors for successful EEG neurofeedback learning. *Neuroscience*. http://dx.doi.org/10.1016/j.neuroscience.2016.12.050

Bates, D., Maechler, M., & Bolker, B. (2013). lme4: Linear mixed-effects models using S4 classes. R package version 0.999999-0. 2012. Retrieved from http://CRAN.R-project.org/package=lme4

Baxter, A. J., Scott, K. M., Vos, T., & Whiteford, H. A. (2013). Global prevalence of anxiety disorders: A systematic review and meta-regression. *Psychological Medicine, 43*(5), 897–910. http://dx.doi.org/10.1017/S003329171200147X

Éismont, E. V., Lutsyuk, N. V., & Pavlenko, V. B. (2011). Moderation of increased anxiety in children and teenagers with the use of neurotherapy: estimation of efficacy. *Neurophysiology, 43*(1), 53–61. http://dx.doi.org/10.1007/s11062-011-9185-5

Ferreira, A., Celeste, W. C., Cheein, F. A., Bastos-Filho, T. F., Sarcinelli-Filho, M., & Carelli, R. (2008). Human-machine interfaces based on EMG and EEG applied to robotic systems. *Journal of NeuroEngineering and Rehabilitation, 5*, 10. http://dx.doi.org/10.1186/1743-0003-5-10

Gidron, Y. (2013). State Anxiety. In M. D. Gellman & J. R. Turner (Eds.), Encyclopedia of Behavioral Medicine (p. 1877). New York, NY: Springer. http://dx.doi.org10.1007/978-1-4419-1005-9

Kohn, P. M., Kantor, L., DeCicco, T. L., & Beck, A. T. (2008). The Beck Anxiety Inventory-Trait (BAI): A measure of dispositional anxiety not contaminated by dispositional depression. *Journal of Personality Assessment, 90*(5), 499–506. http://dx.doi.org/10.1080/00223890802248844

Marzbani, H., Marateb, H. R., & Mansourian, M. (2016). Neurofeedback: A comprehensive review on system design, methodology and clinical applications. *Basic and Clinical Neuroscience, 7*(2), 143–158. http://dx.doi.org/10.15412/J.BCN.03070208

Mennella, R., Patron, E., & Palomba, D. (2017). Frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety. *Behaviour Research and Therapy, 92*, 32–40. http://dx.doi.org/10.1016/j.brat.2017.02.002

Mirman, D. (2014). *Growth curve analysis and visualization using R* (p. 168). Boca Raton, FL: CRC Press.

Mirman, D., Dixon, J. A., & Magnuson, J. S. (2008). Statistical and computational models of the visual world paradigm: Growth curves and individual differences. *Journal of Memory and Language, 59*(4), 475–494. http://dx.doi.org/10.1016/j.jml.2007.11.006

Monastra, V. J., Lynn, S., Linden, M., Lubar, J. F., Gruzelier, J., & LaVaque, T. J. (2005). Electroencephalographic biofeedback in the treatment of Attention-Deficit/Hyperactivity Disorder. *Applied Psychophysiology and Biofeedback, 30*(2), 95–114. http://dx.doi.org/10.1300/J184v09n04_02

Moore, N. C. (2000). A review of EEG biofeedback treatment of anxiety disorders. *Clinical Electroencephalography, 31*(1), 1–6. http://dx.doi.org/10.1177/155005940003100105

Moore, T. J., & Mattison, D. R. (2017). Adult utilization of psychiatric drugs and differences by sex, age, and race. *JAMA Internal Medicine, 177*(2), 274–275. http://dx.doi.org/10.1001/jamainternmed.2016.7507

Paluch, K., Jurewicz, K., Rogala, J., Krauz, R., Szczypińska, M., Mikicin, M., ... & Kublik, E. (2017). Beware: Recruitment of Muscle Activity by the EEG-Neurofeedback Trainings of High Frequencies. *Frontiers in Human Neuroscience, 11*, 119. http://dx.doi.org/10.3389/fnhum.2017.00119

Phneah, S. W., & Nisar, H. (2017). EEG-based alpha neurofeedback training for mood enhancement. Australasian *Physical & Engineering Sciences in Medicine, 40*(2), 325–336. http://dx.doi.org/10.1007/s13246-017-0538-2

Plotkin, W. B., & Rice, K. M. (1981). Biofeedback as a placebo: Anxiety reduction facilitated by training in either suppression or enhancement of alpha brainwaves. *Journal of Counseling and Clinical Psychology, 49*(4), 590–596. http://dx.doi.org/10.1037/0022-006X.49.4.590

Raymond, J., Varney, C., Parkinson, L. A., & Gruzelier, J. H. (2005). The effects of alpha/theta neurofeedback on personality and mood. *Cognitive Brain Research, 23*(2), 287–292. http://dx.doi.org/10.1016/j.cogbrainres.2004.10.023

Rice, K. M., Blanchard, E. B., & Purcell, M. (1993). Biofeedback treatment of generalized anxiety disorder: Preliminary results. *Biofeedback and Self-Regulation, 18*(2), 93–105. http://dx.doi.org/10.1007/BF01848110

Rogala, J., Jurewicz, K., Paluch, K., Kublik, E., Cetnarski, R., & Wróbel, A. (2016). The Do's and Don'ts of Neurofeedback Training: A Review of the Controlled Studies Using Healthy Adults. *Frontiers in Human Neuroscience, 10*, 301. http://dx.doi.org/10.3389/fnhum.2016.00301

Ros, T., Baars, B. J., Lanius, R. A., & Vuilleumier, P. (2014). Tuning pathological brain oscillations with neurofeedback: A systems neuroscience framework. *Frontiers in Human Neuroscience, 8*, 1008. http://dx.doi.org/10.3389/fnhum.2014.01008

Sargunaraj, D., Kumaraiah, V., Mishra, H. & Kumar, K. A. (1987). A comparison of the efficacy of electromyograph and alpha biofeedback therapy in anxiety neurosis. *NIMHANS Journal, 5,* 103–107.

Sarkar, P., Rathee, S. P., & Neera, N. (1999). Comparative efficacy of pharmacotherapy and bio-feed back among cases of generalised anxiety disorder. *Journal of Projective Psychology & Mental Health, 6*(1), 69–77.

Simkin, D. R., Thatcher, R.W., & Lubar, J. F. (2014). Quantitative EEG and neurofeedback in children and adolescents: Anxiety disorders, depressive disorders, comorbid addiction and attention-deficit/hyperactivity disorder, and brain injury. *Child & Adolescent Psychiatric Clinics of North America, 23*(3), 427–464. http://dx.doi.org/10.1016/j.chc.2014.03.001

Snapinn, S. M., & Jiang, Q. (2007). Responder analyses and the assessment of a clinically relevant treatment effect. Trials, 8(1), 31. http://dx.doi.org/10.1186/1745-6215-8-31

Soutar, R. G., & Longo, R. E. (2011). *Doing neurofeedback: An introduction*. ISNR Research Foundation.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

Strehl, U. (2013). Lerntheoretische Grundlagen und Überlegungen zum Neurofeedback. In U. Strehl (Ed.), *Neurofeedback* (pp. 13–30). Stuttgart, Germany: Kohlhammer.

Strehl, U. (2014). What learning theories can teach us in designing neurofeedback treatments. *Frontiers in Human Neuroscience, 8*, 894. http://dx.doi.org/10.3389/fnhum.2014.00894

Studer, P., Kratz, O., Gevensleben, H., Rothenberger, A., Moll, G. H., Hautzinger, M., & Heinrich, H. (2014). Slow cortical potential and theta/beta neurofeedback training in adults: Effects on attentional processes and motor system excitability. *Frontiers in Human Neuroscience, 8*, 555. http://dx.doi.org/10.3389/fnhum.2014.00555

Thatcher, R. W., & Lubar, J. F. (2008). History of the scientific standards of QEEG normative databases. In T. Budzinsky, H. Budzinsky, J. Evans, & A. Abarbanel (Eds.), *Introduction to QEEG and neurofeedback: Advanced theory and applications*. San Diego, CA: Academic Press.

Uryniak, T., Chan, I. S. F., Fedorov, V. V., Jiang, Q., Oppenheimer, L., Snapinn, S. M., ... & Zhang, J. (2012). Responder analyses—A PhRMA position paper. *Statistics in Biopharmaceutical Research, 3*(3), 476–487. http://dx.doi.org/10.1198/sbr.2011.10070

Vanathy, S., Sharma, P. S. V. N., & Kumar, K. B. (1998). The efficacy of alpha and theta neurofeedback training in treatment of generalized anxiety disorder. *Indian Journal of Clinical Psychology, 25*(2), 136–143.

Wang, S., Zhao, Y., Chen, S., Lin, G., Sun, P., & Wang, T. (2013). EEG biofeedback improves attentional bias in high trait anxiety individuals. *BMC Neuroscience, 14*(1), 115.

Watson, C. G., & Herder, J. (1980). Effectiveness of alpha biofeedback therapy: Negative results. *Journal of Clinical Psychology*, 36(2), 508–513. http://dx.doi.org/10.1002/jclp.6120360221

White, E. K., Groeneveld, K. M., Tittle, R. K., Bolhuis, N. A., Martin, R. E., Royer, T. G., & Fotuhi, M. (2017). Combined neurofeedback and heart rate variability training for individuals with symptoms of anxiety and depression: A retrospective study. *NeuroRegulation, 4*(1), 37–55. http://dx.doi.org/10.15540/nr.4.1.37

Wyckoff, S., & Birbaumer, N. (2014). Neurofeedback. In D. J. A. Dozois (Ed.), The Wiley Handbook of Cognitive Behavioral Therapy, Part One (13, pp. 273–310). Princeton, NJ: Wiley & Sons Press. http://dx.doi.org/10.1002/9781118528563.wbcbt13

Zuberer, A., Brandeis, D., & Drechsler, R. (2015). Are treatment effects of neurofeedback training in children with ADHD related to the successful regulation of brain activity? A review on the learning of regulation of brain activity and a contribution to the discussion on specificity. *Frontiers in Human Neuroscience, 9*, 135. http://dx.doi.org/10.3389/fnhum.2015.00135

STUDENT AWARD WINNERS – POSTER PRESENTATIONS

The Effect of Slow Breathing Training on Electroencephalogram

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Background. Previous studies have confirmed that slow breathing training not only improves heart rate variability and autonomic activation but also increases subjective relaxation feeling and reduces negative emotions. The purpose of this study was to explore the effect of slow breathing training on electroencephalogram (EEG).

Methods. Fifty-three healthy participants were randomly assigned to slow breathing group (n = 27; mean age was 25.30 ± 6.86 years; 3 male and 24 female) and control group (n = 26; mean age was 31.23 ± 14.77 years; 7 male and 19 female). Participants in the slow breathing group received 60-min weekly training for 4 weeks between pre- and posttest. The control group only received pre- and posttest. All participants received 5-min resting EEG measurement with eyes-opened at Fz, Cz, and Pz by using BrainAvatar (BrainMaster Technologies, Inc., Bedford, OH) at pre- and posttest. The EEG was analyzed in the following bandpass: delta (1–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), total beta (12–32 Hz), beta1 (12–15 Hz), beta2 (15–22 Hz), beta3 (22–28 Hz), and beta4 (28–32 Hz). The change in scores of pre- and posttest EEG were compared between two groups.

Results. There was no significant difference between slow breathing group and control group in age (t = 1.86, p = .71) and gender ($\chi 2$ = 2.16, p = .14). Significant group * time interaction effects were found at pre- and posttest between two groups at Fz on beta2, $F(1, 51) = 7.09$, $p < .05$; beta3, $F(1, 51) = 6.90$, $p < .05$; and beta4, $F(1, 51) = 4.71$, $p < .05$. The post-hoc comparison showed a trend to decrease beta activity in the slow breathing group, while increasing beta activity in the control group. Moreover, there were significant differences between two groups on change scores at Fz of beta2, $t(51) = 2.66$, $p < .05$; beta3, $t(51) = 2.63$, $p < .05$; and beta4, $t(51) = 2.17$, $p < .05$. However, there was no significant interaction effect on beta1 at Fz, as well as no significant interaction effect on EEG index at Cz and Pz.

Conclusion. This study confirmed that slow breathing training is a useful intervention protocol in decreasing cortical arousal at frontal area.

References

Lehrer, P. M., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: Rationale and manual for training. *Applied Psychophysiology and Biofeedback, 25*(3), 177–191. http://dx.doi.org/10.1023/A:1009554825745

Prinsloo, G. E., Rauch, H. G. L., Karpul, D., & Derman, W. E. (2013). The effect of a single session of short duration heart rate variability biofeedback on EEG: A pilot study. *Applied Psychophysiology and Biofeedback, 38*(1), 45–56. http://dx.doi.org/10.1007/s10484-012-9207-0

The Effects of Personalized EEG-Neurofeedback in College Students with ADHD

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Background. Several neurophysiological subtypes based on EEG biomarkers have been identified in ADHD (Johnstone, Gunkelman, & Lunt, 2005). However, most studies investigating the efficacy of neurofeedback as a treatment for ADHD use a uniform treatment protocol without taking into account individual EEG biomarkers (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). A recent pilot study suggests that personalizing neurofeedback protocols to individual EEG biomarkers of ADHD might lead to increased specificity and efficacy of treatment (Arns, Drinkenburg, & Leon Kenemans, 2012). Hence, this preliminary study aims to investigate the effects of personalized EEG-neurofeedback in a population of college students with ADHD.

Methods. Eighty college students with a diagnosis of ADHD received personalized EEG-neurofeedback training (NFT) two times a week over a period of 4 months. Half of the participants was randomly assigned to the experimental condition. The other half was placed on a waiting list to serve as a control group and received treatment later. Resting-state EEG signals were recorded to evaluate overall brain activity pre- and posttraining and to determine individual EEG biomarkers for selection of personalized treatment protocol. ADHD behavioral symptoms were assessed pre- and posttraining using the Conners' Adult ADHD Rating Scale–Self-Report: Long Version (CAARS–S:L) and the IVA-2.

Results. The expected result is that a significant change will be observed in subjects trained in EEG neurofeedback, both in brain activation patterns and at the behavioral level. More specifically, normalization of targeted resting brain waves is expected in the experimental group. Changes in neural activity in the experimental group is also predicted to be correlated with improvements in ADHD symptoms.

Conclusion. This preliminary study will demonstrate the feasibility of personalizing neurofeedback protocols to individual EEG biomarkers of ADHD and the efficacy of neurofeedback as a treatment for ADHD. On a broader level, it will allow for a better understanding of the impact of neurofeedback training on neural and behavioral correlates of ADHD.

References

Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience, 40*(3), 180–189. http://dx.doi.org/10.1177/155005940904000311

Arns, M., Drinkenburg, W., & Leon Kenemans, J. (2012). The effects of qEEG-informed neurofeedback in ADHD: An open-label pilot study. *Applied Psychophysiology and Biofeedback, 37*(3), 171–180. http://dx.doi.org/10.1007 /s10484-012-9191-4

Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: Characterization of EEG phenotypes. *Clinical EEG and Neuroscience, 36*(2), 99–107. http://dx.doi.org/10.1177/155005940503600209

The Differences Between Frontal Alpha Asymmetry Among Healthy Participants and Patients with Major Depressive Disorder

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Background. Previous studies indicated that frontal alpha asymmetry (FAA) is a potential biomarker for major depressive disorder (MDD). However, some results did not support the FAA. The purpose of this study was to examine the electroencephalogram (EEG) difference among healthy participants and patients with MDD patients whom with and without FAA.

Methods. Ninety-five healthy participants and 73 patients with MDD were recruited. Five minutes resting EEG with eyes-closed was measured at frontal (F3, F4) and midline (Fz, Cz, Pz) by BrainAvatar (BrainMaster Technologies, Inc., Bedford, OH). The EEG singles were analyzed into the following bands: delta (1–4 Hz), low theta (4–6 Hz), high theta (6–8 Hz), total theta (4–8 Hz), low alpha (8–10 Hz), high alpha (10–12 Hz), total alpha (8–12 Hz), low beta (12–20 Hz), high beta (20–32 Hz), and total beta (12–32 Hz). FAA score was calculated by log(F4 alpha) − log(F3 alpha). FAA score higher than 0 refers F4 alpha is higher than F3 alpha (FAA+); on the other hand, FAA score lower than 0 refers F4 alpha is lower than F3 alpha (FAA−). Participants were divided into one of four groups based on their FAA score: healthy control with FAA+ (H+ group), healthy control with FAA− (H− group), MDD with FAA+ (M+ group), and MDD with FAA− (M− group).

Results. No significant difference between four groups on age, $F(3, 168) = 0.43$, $p = .73$, and sex, $\chi^2 = 2.60$, $p = .46$. Significant differences were found between four groups on total theta and high beta, the post hoc comparison found that M+ and M− group had lower total theta at Fz and Cz compared with H− group ($F = 3.76$, $p = .012$; and $F = 3.85$, $p = .011$, respectively). M− group had higher high beta at Fz, F3, F4, and Cz compared with H− group ($F = 4.58$, $p = .004$; $F = 5.34$, $p = .002$; $F = 4.53$, $p = .004$; and $F = 4.32$, $p = .006$, respectively).

Conclusion. This study indicated that not all patients with MDD had FAA mechanisms in brain activity. The most significant finding was that MDD with FAA− had lower total theta and higher high beta compared to the healthy controls who with FAA−.

References

Baehr, E., Rosenfeld, J. P., & Baehr, R. (1997). The clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression: Two case studies. Journal of Neurotherapy, 2(3), 10–23. http://dx.doi.org/10.1300/J184v02n03_02

Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *Journal of Abnormal Psychology, 115*(4), 715–729. http://dx.doi.org/10.1037/0021-843X.115.4.715

Neurostructural Predictors of Cognitive Behavioral Therapy (CBT) for Obsessive-Compulsive Disorder: Implications for the Integration of Neurofeedback Training and CBT

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Background. Cognitive-behavioral therapy (CBT) is the most commonly used evidence-based practice in the treatment of mental disorders (Butler, Chapman, Forman, & Beck, 2006). CBT is an effective treatment for Obsessive-Compulsive Disorder (OCD) and is also applicable to patients with both OCD and Autism Spectrum Disorder (ASD). However, previous studies have reported that CBT for patients with both OCD and ASD might be less effective than for patients with OCD alone (Mito et al., 2014; Murray, Jassi, Mataix-Cols, Barrow, & Krebs, 2015). In addition, there is no evidence as to why autistic traits might be risk factors. Therefore, we investigated whether comorbidity between ASD and OCD might significantly affect treatment outcome, and discovered predictors of CBT outcomes using structural magnetic resonance imaging (MRI) data. Finally, we suggested implications for the integration of neurofeedback training (NFB) and CBT.

Methods. Fifteen patients were diagnosed with having OCD with ASD, and 22 patients were diagnosed with OCD without ASD. Both groups took CBT for 12 to 20 sessions. First, to examine the effectiveness of CBT for OCD patients with and without ASD, we compared CBT outcomes between both groups. Second, to investigate how the structural abnormalities profile of the brain at pretreatment influenced CBT outcomes, we performed a structural MRI comparison focusing on the total gray matter volume in both OCD patients with and without ASD, as well as those who reached remission and did not reach remission.

Results. OCD patients with ASD responded significantly less well to CBT than OCD patients without ASD. They had significantly smaller gray matter volumes than OCD without ASD in the bilateral occipital lobes and the right cuneus (BA 18 and 19), both of which play important roles in visuospatial processing. After controlling for autistic traits, the nonremission group displayed a smaller gray matter volume than the remission group in the left dorsolateral prefrontal cortex (DLPFC, BA 10, BA 46). The DLPFC has an important role in executive functions including spatial attention and working memory processes, and these cognitive processes at pretreatment might affect CBT outcomes. Our results support the revised model for OCD proposed by Menzies et al. (2008), which shows that the brain pathology of OCD is not only limited to the orbitofronto-striatal "affective" circuit associated with limbic structures but also involves abnormalities including the DLPFC that may represent the dorsolateral prefronto-striatal "executive" circuit.

Conclusion. In our study, after controlling for autistic traits, the smaller the pretreatment gray matter volume in the left DLPFC, the less likely the OCD patients would fully remit. The application of NFB in the treatment of OCD has not been systematically investigated; however, several studies showed that NFB in the limbic system would result in a reduction in experienced anxiety (Hammond, 2003). In addition to these studies, we suggest that NFB in the DLPFC prior to CBT could improve executive functions. As Mohlman and Gorman (2005) mentioned, the successful use of CBT is assumed to rely on cognitive skills known as executive functions (e.g., allocation of attention, self-monitoring) governed by the prefrontal cortex including the DLPFC.

References

Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review, 26*(1), 17–31. http://dx.doi.org/10.1016/j.cpr.2005.07.003

Hammond, D. C. (2003). QEEG-guided neurofeedback in the treatment of obsessive compulsive disorder. *Journal of Neurotherapy, 7*(2), 25–52. http://dx.doi.org/10.1300/J184v07n02_03

Menzies, L., Chamberlain, S. R., Laird, A. R., Thelen, S. M., Sahakian, B. J., & Bullmore, E. T. (2008). Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: The orbitofronto-striatal model revisited. • *Neuroscience & Biobehavioral Reviews, 32*(3), 525–549. http://dx.doi.org/10.1016/j.neubiorev.2007.09.005

Mito, H., Matsuura, N., Mukai, K., Yanagisawa, Y., Nakajima, A., Motoyama, M., ... & Matsunaga, H. (2014). The impacts of elevated autism spectrum disorder traits on clinical and psychosocial features and long-term treatment outcome in adult patients with obsessive-compulsive disorder. *Comprehensive Psychiatry, 55*(7), 1526–1533. http://dx.doi.org/10.1016/j.comppsych.2014.05.005

Mohlman, J., & Gorman, J. M. (2005). The role of executive functioning in CBT: A pilot study with anxious older adults. *Behaviour Research and Therapy, 43*(4), 447–465. http://dx.doi.org/10.1016/j.brat.2004.03.007

Murray, K., Jassi, A., Mataix-Cols, D., Barrow, F., & Krebs, G. (2015). Outcomes of cognitive behaviour therapy for obsessive–compulsive disorder in young people with and without autism spectrum disorders: A case controlled study. *Psychiatry Research, 228*(1), 8–13. http://dx.doi.org/10.1016/j.psychres.2015.03.012

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