ADHD & Neurofeedback

The Original Research

- Lubar, J. F. (1977). Electroencephalographic biofeedback, methodology and the management of epilepsy. Pavlovian Journal of Biological Science, 12, 147-189.
- • Shouse, M. N. & Lubar, J. F. (1978). Physiological bases of hyperkinesis treated with methylphenidate. Pediatrics, 62, 343-351.
- Shouse, M. N., & Lubar, J. F. (1979). Sensorimotor rhythm (SMR) operant conditioning and methylphenidate in the treatment of hyperkinesis. Biofeedback and Self-Regulation, 4, 299-311.
- Lubar, J. F., Shabsin, H. S., Natelson, S. E., Holder, G. S., Woodson, S. F., Pamplin, W. E., & Krulikowski, D. I. (1981). EEG operant conditioning in intractable epileptics. Archives of Neurology, 38, 700-704.
- Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. Biofeedback and Self-Regulation, 9, 1-23.

Later Peer Reviewed Studies

- Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, B. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention deficit/hyperactivity disorder (ADHD). A controlled study with clinical implications. Pediatric Neurology, 8, 30-36.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. Biofeedback and Self-Regulation, 20, 83-99.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & Timmermann, D. L. (1995). Quantitative EEG and auditory event-related potentials in the evaluation of Attention-Deficit/Hyperactivity disorder: Effects of methylphenid ate and implications for neurofeedback training. Journal of Psychoeducational Assessment (Monograph Series Advances in Psychoeducational Assessment) Assessment of Attention-Deficit/Hyperactivity Disorders, 143-204.

Many Studies Followed

- In collaboration with researchers from Tubingen University (Germany), Radboud University (Nijmegen, the Netherlands), Brainclinics and EEG Resource Institute a so-called meta-analysis was conducted on all published research about Neurofeedback treatment in ADHD. This meta-analysis included 15 studies and 1194 ADHD patients. Based on this study - which was published in the July issue of EEG and Clinical Neuroscience - it could be concluded that Neurofeedback can indeed be considered an Evidence-Based treatment for ADHD. The results show that neurofeedback treatment has large and clinically significant effects on Impulsivity and Inattention and a modest improvement of Hyperactivity.
- Arns, M., de Ridder, S., Strehl, U., Breteler, M. & Coenen, A. Efficacy of Neurofeedback Treatment in ADHD: The effects on Inattention, Impulsivity and Hyperactivity: a Meta-Analysis. EEG and Clinical Neuroscience; 40(3), 180-189.

MRI Research & Neurofeedback

Changing Function of the Brain

MRI Research shows that EEG Biofeedback changes the functioning of the brain.

- Functional magnetic resonance imaging investigation of the effects of neurofeedback training on the neural bases of selective attention and response inhibition in children with attention-deficit/hyperactivity disorder.
- <u>Beauregard M</u>, <u>Lévesque J</u>.
- Centre de Recherche en Neuropsychologie et Cognition, Département de Psychologie, Université de Montréal, Montréal, Canada. mario.beauregard@umontreal.ca
- Two functional magnetic resonance imaging (fMRI) experiments were undertaken to measure the effect of neurofeedback training (NFT), in AD/HD children, on the neural substrates of selective attention and response inhibition. Twenty unmedicated AD/HD children participated to these experiments. Fifteen children were randomly assigned to the Experimental (EXP) group whereas the other five children were randomly assigned to the Control (CON) group. Only subjects in the EXP group underwent NFT. EXP subjects were trained to enhance the amplitude of the SMR (12-15 Hz) and beta 1 activity (15-18 Hz), and decrease the amplitude of theta activity (4-7 Hz). Subjects from both groups were scanned one week before the beginning of NFT (Time 1) and 1 week after the end of NFT (Time 2), while they performed a "Counting Stroop" task (Experiment 1) and a Go/No-Go task (Experiment 2). At Time 1, in both groups, the Counting Stroop task was associated with significant activation in the left superior parietal lobule. For the Go/No-Go task, no significant activity was detected in the EXP and CON groups. At Time 2, in both groups, the Counting Stroop task was associated with significant activation of the left superior parietal lobule. This time, however, there were significant loci of activation, in the EXP group, in the right ACC, left caudate nucleus, and left substantia nigra. No such activation loci were seen in CON subjects. For the Go/No-Go task, significant loci of activation were noted, in the EXP group, in the right ventrolateral prefrontal cortex, right ACC, left thalamus, left caudate nucleus, and left substantia nigra. No such activation and response inhibition in AD/HD children vere seen in CON subjects. These results suggest that NFT has the capacity to functionally normalize the brain systems mediating selective attention and response inhibition in AD/HD children.
- PMID: 16552626 [PubMed indexed for MEDLINE]

Neurofeedback Changes Brain Structure

Neurofeedback Training Induces Changes in White and Gray Matter

Clinical EEG and Neuroscience 00(0) 1-8 © EEG and Clinical Neuroscience Society (ECNS) 2013 Reprints and permission: sagepub.com/journalsPermissions.nav DOI: 10.1177/1550059413476031 eeg.sagepub.com



J. Ghaziri¹, A. Tucholka², V. Larue¹, M. Blanchette-Sylvestre¹, G. Reyburn¹, G. Gilbert², J. Lévesque¹, and M. Beauregard^{1,2,3}

Abstract

The main objective of this structural magnetic resonance imaging (MRI) study was to investigate, using diffusion tensor imaging, whether a neurofeedback training (NFT) protocol designed to improve sustained attention might induce structural changes in white matter (WM) pathways, purportedly implicated in this cognitive ability. Another goal was to examine whether gray matter (GM) volume (GMV) might be altered following NFT in frontal and parietal cortical areas connected by these WM fiber pathways. Healthy university students were randomly assigned to an experimental group (EXP), a sham group, or a control group. Participants in the EXP group were trained to enhance the amplitude of their β I waves at F4 and P4. Measures of attentional performance and MRI data were acquired one week before (Time I) and one week after (Time 2) NFT. Higher scores on visual and auditory sustained attention were noted in the EXP group at Time 2 (relative to Time I). As for structural MRI data, increased fractional anisotropy was measured in WM pathways implicated in sustained attention, and GMV increases were detected in cerebral structures involved in this type of attention. After 50 years of research in the field of neurofeedback, our study constitutes the first empirical demonstration that NFT can lead to microstructural changes in white and gray matter.

Keywords

neurofeedback, structural magnetic resonance imaging, white matter, gray matter, sustained attention

Pre Post ADHD Child: 40 Sessions









Magnitude

Alpha

H

Beta

OK LO VLO

HI

LO LO VLC



Inhibit Theta Using Two Channel Protocol #2

Typical Changes In ADHD

- Improved Behavior
- Improved School Performance 1-2 grade levels.
- Increase IQ by 5-15 points.
- Reduced Medications. (Hammond, 2006)



American Academy of Pediatrics

ADDRESSING Mental Health CONCERNS IN PRIMARY CARE A CLINICIAN'S TOOLKIT	EVI This repo using the health We	DENCE-BASE rt is intended to guide practitioners, PracticeWise Evidence-Based Serv eb site (www.aap.org/mentalhealth)	D CHILD AND ADOLE educators, youth, and families in developing appropriate ices (PWEBS) Database, available at www.practicewise.co for updates.	SCENT PSYC plans using psychosocial intervent m. If this is not the most current v	HOSOCIAL IN ions. It was created for the period Nu ersion, please check the American A	VTERVENTIONS ovember 2012–April 2013 ccademy of Pediatrics mental
Problem Area	a	Level 1- BEST SUPPORT	Level 2- GOOD SUPPORT	Level 3- Moderate Support	Level 4- MINIMAL SUPPORT	Level 5- No support
Anxious or Avoidant Behaviors		Cognitive Behavior Therapy (CBT), CBT and Medication, CBT with Parents, Education, Exposure, Modeling	Assertiveness Training, Attention, CBT for Child and Parent, Cultural Storytelling, Family Psychoeducation, Hypnosis, Relaxation, Stress Inoculation	Contingency Management, Group Therapy	Biofeedback, CBT with Parents Only, Play Therapy, Psychodynamic Therapy, Rational Emotive Therapy	Assessment/Monitoring, Attachment Therapy, Client Centered Therapy, Eye Movement Desensitization and Reprocessing (EMDR), Peer Pairing, Psychoeducation, Relationship Counseling, Teacher Psychoeducation
Attention and Hyperactivity Behavio	Drs	Behavior Therapy and Medication, Biofeedback, Parent Management Training, Self-Verbalization	Contingency Management, Education, Parent Management Training (with Problem Solving, or with Teacher Psychoeducation), Physical Exercise (with or without Relaxation), Social Skills and Medication, Working Memory Training	Biofeedback and Medication	Parent Management Training and Social Skills, Relaxation, Self-Verbalization and Contingency Management, Social Skills	Attention Training, Client Centered Therapy, CBT, CBT and Anger Control, CBT and Medication, Family Therapy, Parent Coping/Stress Management, Parent Management Training and Self-Verbalization, Problem Solving, Psychoeducation, Self-Control Training, Self-Verbalization and Medication, Skill Development
Autism Spectrum Disorders		Intensive Behavior Therapy, Intensive Communication Training	Parent Management Training, Peer Pairing, Physical/ Social/Occupational Therapy	None	Cognitive Behavior Therapy, Massage, Social Skills	Auditory Integration Training, Biofeedback, Eclectic Therapy, Hyperbaric Treatment, Modeling, Structured Listening

PracticeWise, the proprietary company that creates the American Academy of Pediatrics "Evidence-based Child and Adolescent Psycho-social Interventions" has just *elevated biofeedback to "Level 1 - Best Support" as an intervention for Attention Problems.*

The brain is capable of changing and remodeling itself

The Research Community Reports The Real Findings

- In a recent paper *Update on attention-deficit/hyperactivity disorder* published in <u>Current Opinion in Pediatrics</u> Katie Campbell Daley reviewed the research and practice standards on treatment of ADHD. Dr. Campbell serves on the staff of the Department of Medicine, Children's Hospital Boston and in the Department of Pediatrics of the Harvard Medical School. She concluded:
- "Overall, these findings support the use of multi-modal treatment, including medication, parent/school counseling, and EEG biofeedback, in the long term management of ADHD, with EEG biofeedback in particular providing a sustained effect even without stimulant treatment...parents interested in non-psychopharmacologic treatment can pursue the use of complementary and alternative therapy. The therapy most promising by recent clinical trials appears to be EEG biofeedback."

More Recently

Eur Child Adolesc Psychiatry DOI 10.1007/s00787-010-0109-5

ORIGINAL CONTRIBUTION

Neurofeedback training in children with ADHD: 6-month follow-up of a randomised controlled trial

Holger Gevensleben · Birgit Holl · Björn Albrecht · Dieter Schlamp · Oliver Kratz · Petra Studer · Aribert Rothenberger · Gunther H. Moll · Hartmut Heinrich

Received: 8 September 2009/ Accepted: 31 March 2010 © Springer-Verlag 2010

Abstract Neurofeedback (NF) could help to improve attentional and self-management capabilities in children with attention-deficit/hyperactivity disorder (ADHD). In a randomised controlled trial, NF training was found to be superior to a computerised attention skills training (AST) (Gevensleben et al. in J Child Psychol Psychiatry 50(7): 780-789, 2009). In the present paper, treatment effects at 6-month follow-up were studied. 94 children with ADHD, aged 8-12 years, completed either 36 sessions of NF training (n = 59) or a computerised AST (n = 35). Pretraining, post-training and follow-up assessment encompassed several behaviour rating scales (e.g., the German ADHD rating scale, FBB-HKS) completed by parents. Follow-up information was analysed in 61 children (ca. 65%) on a per-protocol basis. 17 children (of 33 dropouts) had started a medication after the end of the training or early in the follow-up period. Improvements in the NF group (n = 38) at follow-up were superior to those of the control group (n = 23) and comparable to the effects at the end of the training. For the FBB-HKS total score (primary outcome measure), a medium effect size of 0.71 was obtained at follow-up. A reduction of at least 25% in the primary outcome measure (responder criterion) was observed in 50% of the children in the NF group. In conclusion, behavioural improvements induced by NF training in children with ADHD were maintained at a 6-month follow-up. Though treatment effects appear to be limited, the results confirm the notion that NF is a clinically efficacious module in the treatment of children with ADHD.

Keywords ADHD · Neurofeedback · Randomised controlled trial (RCT) · Follow-up · Children

Introduction

And It Continues . . .

ARTICLE IN PRESS

Clinical Neurophysiology xxx (2010) xxx-xxx



Resting-state EEG gamma activity in children with Attention-Deficit/Hyperactivity Disorder

Robert J. Barry^{a,*}, Adam R. Clarke^a, Mihaly Hajos^b, Rory McCarthy^c, Mark Selikowitz^c, Franca E. Dupuy^a

⁸ Brain & Behaviour Research Institute and School of Psychology, University of Wollongong, Wollongong 2522, Australia ^b Department of Neuroscience, Pfizer Global Research and Development, Groton, CT 06340, USA ^cSydney Developmental Clinic, 6/30 Carrington A., Sydney 2000, Australia

ARTICLE INFO

ABSTRACT

Article history:	Objective: Children with Attention-Deficit/Hyperactivity Disorder (AD/HD) have well-described abnor-
Accepted 21 April 2010	malities in the four traditional EEG bands. However, to date the gamma band has not been widely inves-
Available online xxxx	tigated. This study investigated resting-state EEG in children with AD/HD and matched controls, with a
Keywards: Attention-Deficit/Hyperactivity Disorder Children EBG Gamma	Method: Forty children with AD/HD, and 40 age- and sex-matched controls, participated. EEG was recorded from 19 sites during an eyes-closed resting condition and Fourier transformed to provide estimates for absolute and relative power in the delta, theta, alpha, beta and gamma bands. Results: Children with AD/HD had elevated levels of absolute delta and theta power, and decreased levels of absolute beta and gamma power, compared to controls. With relative power measures, children with AD/HD showed enhanced delta and theta activity, with reduced alpha, beta and gamma activity. Inattention scores on the Conners' Parent Rating Scale were negatively correlated with absolute gamma. Conclusions: These patients demonstrate the typical EEG profile in the eyes-closed resting state, over the delta, theta, alpha and beta bands, associated with AD/HD. In addition, compared with controls, they demonstrate reduced absolute and relative gamma activity. These differences appear to contribute importantly to their dysfunctional stimulus processing and impact their behavioural outcomes. Significance: This resting-state study extends the well-established fast-wave EEG deficits in children with AD/HD to the gamma band, and links that to increased inattention, which is of special importance in understanding their cognitive-processing problems.

qEEG Findings Fail To Support Unidimensional Disorder Theory



Available online at www.sciencedirect.com

SCIENCE dDIRECT.

International Journal of Psychophysiology 58 (2005) 81-93

INTERNATIONAL JOURNAL OF PSYCHOPHYSIOLOGY

www.elsevier.com/locate/ijpsycho

The neurophysiology of attention-deficit/hyperactivity disorder

Flavia di Michele^{a,b,c}, Leslie Prichep^a, E. Roy John^a, Robert J. Chabot^{a,*}

^aBrain Research Labs, New York University School of Medicine, 27th and 1st Ave., 8th Floor Old Bellevue Admin. Bldg., New York, NY 10016, United States ^bDepartment of Neuroscience, Tor Vergata University of Rome, Italy ^cIRCCS S. Lucia, Rome, Italy

> Received 23 September 2004; received in revised form 8 January 2005; accepted 27 January 2005 Available online 24 June 2005

Abstract

Recent reviews of the neurobiology of Attention-Deficit/Hyperactivity Disorder (AD/HD) have concluded that there is no single pathophysiological profile underlying this disorder. Certainly, dysfunctions in the frontal/subcortical pathways that control attention and motor behavior are implicated. However, no diagnostic criteria or behavioral/neuroimaging techniques allow a clear discrimination among subtypes within this disorder, especially when problems with learning are also considered. Two major Quantitative EEG (QEEG) subtypes have been found to characterize AD/HD. Here we review the major findings in the neurophysiology of AD/HD, focusing on QEEG, and briefly present our previous findings using a source localization technique called Variable Resolution Electromagnetic Tomography (VARETA). These two techniques represent a possible objective method to identify specific patterns corresponding to EEG-defined subtypes of AD/HD. We then propose a model representing the distribution of the neural generators in these two major AD/HD subtypes, localized within basal ganglia and right anterior cortical regions, and hippocampal, para-hippocampal and temporal cortical regions, respectively. A comprehensive review of neurochemical, genetic, neuroimaging, pharmacological and neuropsychological evidence in support of this model is then presented. These results indicate the value of the neurophysiological model of AD/HD and support the involvement of different neuroanatomical systems, particularly the dopaminergic pathways.

Conclusions

6. Conclusions

The diagnostic category AD/HD, as defined by APA, seems not to fit with the real heterogeneity of the symptoms exhibited in AD/HD and must be explained by multiple specific causes. Even though the intent of DSM-IV is to provide hierarchically organized categories of mental disorders that aid clinicians in differential diagnosis, certain criteria are too restrictive. In regard to AD/HD and LD, these are considered two mutually exclusive and non-overlapping entities. By this definition, a child cannot simultaneously meet criteria for AD/HD and LD. It is clear that AD/HD is a heterogeneous disorder. Therefore, it cannot be conceptualized as only one disease entity with a very narrow phenotype and a distinct etiology. Rather, it is believed to constitute a spectrum of disorders that subsume different subtypes of a larger population of children with attention and learning problems. To date, AD/HD is diagnosed solely on the basis of patterns of observable behavior. It has been difficult to identify specific biochemical or neurophysiological tests that may contribute to more accurate diagnosis. Identifying a biological measure that could aid in this distinction would help to refine diagnostic criteria and may provide more specific diagnostic tests for AD/HD and LD.

Clinical Evidence Is Supportive



Gurnee, Robert: Compliments of The ADD Clinic Scottsdale Arizona

Amen's 6 Spect Subtypes



Do Not Match 5 qEEG Subtypes Identified

Classic ADHD Pattern



NOTE: Low Beta Phenotypes Are Often More Commonly Found

ADD Type Theta vs Over 75





Inhibition of Default

- Reduces (attenuates) in activity during focused attentional network activation.
- Reduces activity during memorization tasks.
- Reduces in activity during novel motor tasks.
- Reduces in response to intentional network activity (Buckner et al)

FIGURE 15. Intrinsic activity suggests that the default network is negatively correlated (anticorrelated) with brain systems that are used for focused external visual attention. Anticorrelated networks are displayed by plotting those regions that negatively correlate with the default network (shown in blue) in addition to those that positively correlate (shown in red). These two anticorrrelated networks may participate in distinct functions that compete with one another for control of information processing within the brain. Data are the same as analyzed for FIGURE 7.



Excitation of Default Network Navigating Social Interactions

- Self-reflective thoughts and judgments that depend on inferred social and emotional content. dMPFC
- Episodic memory HF
- Fantasy, daydreams, envisioning the future, past ruminations, moral judgments, inferring thoughts of others PCC.



FIGURE 12. The default network is activated by diverse forms of tasks that require mental simulation of alternative perspectives or imagined scenes. Four such examples from the literature illustrate the generality. (A) Autobiographical memory: subjects recount a specific, past event from memory. (B) Envisioning the future: cued with an item (e.g., dress), subjects imagine a specific future event involving that item. (C) Theory of mind: subjects answer questions that require them to conceive of the perspective (belief) of another person. (D) Moral decision making: subjects decide upon a personal moral dilemma. Data come from prior studies and are here displayed using procedures similar to FigURE 2. Data in A and B are from Addis et al. (2007). Data in C uses the paradigm of Saxe and Kanwisher (2003). Data in D is from Greene et al. (2001). Note that all the studies activate strongly PCC/Rap and dMPCC. Active regions also include those close to PR and IIC, although further research will be required to determine the axoct degree of anatomic overlap. It seems likely that these maps represent multiple, interacting subsystems.

Two Thetas Midline vs Temporal Lobe



Fig. 1 – Schematics of the pathways involved in A. ascending theta modulation and B. recurrent theta networks described in text. Abreviations: ctx – cortex, MD – mediodorsal thalamus, SuM – supramammillary nucleus, post. cing. – posterior cingulate, med. mam. bodies – medial mamillary bodies.

998

Ian J. Kirk and James C. Mackay

Not All Theta Is Bad Theta and Insight



TRENDS in Cognitive Sciences

Idling Zone of Default System



 Activation levels in the optimal idling zone reflect functional connectivity

ADD Pathways

Or Depression vs Attention Problems ?



Michele et al, 2005

Diagnosed With ADHD



Testing revealed depression as key issue.

Diagnosed With ADD



Testing revealed key issue was anxiety.



Diagnosed With ADHD





CPT Testing For Attention

Cognitive Performance Testing (CPT)



TOVA Confirmation



Beta Spindles



Diagnosed With ADD 2





CEC Analysis



CEC Response Assessment

Category	Response Count	Average Response	Category Score
Attention	6	2.33	14.00
Memory	11	2.36	26.00
Impulsive	7	1.86	13.00
Depression	7	2.71	19.00
Anxiety	9	2.11	19.00

* Red indicates an abnormally high score.

CEC Responses

Answer	Question
3	Lack Of Motivation/ Poor Follow through
3	Procrastination/Puts Things Off
3	Difficulty With Decisions
3	Daydreams Or Spaces Out
3	Careless Mistakes
3	Poor Short Term Memory

Metabolics ?

Normal Dominant Frequency



Metabolic Assessment

Probability	Score	Metabolic Category	Symptoms Reported
Θ	9	Pituitary	Insomnia
Θ	4	Thyroid (hyper)	Insomnia
Θ	6	Adrenals	Insomnia Fatigue Non-restorative sleep
Θ	3	Blood Sugar	Irritable with missed meals Eating relieves fatigue
Θ	1	Thyroid (hypo)	Fatigue
Θ	1	Kidney	Fatigue
Θ	1	Cardio-Vascular	Fatigue
Θ	1	Liver	Fatigue
Θ	1	Somatic	Restless Legs



Probability Legend

Dashboard An Emotional Profile

	Underarousal	Inhibited	Overarous	al
	Global Measures	Global Measures	G	lobal Measures
			6	
			Inhib	ited
				Global Measures
Note tha green lig mostly re	t the EEG is largely s hts while the CEC s ed and yellow lights	showing howing	CEC * * * * * * * *	Local Measures EEC Symptom Victim Mentality Excessive Self-concern Rumination Anger Self-Deprecation Self-Deprecation Irritability Passive Aggressive

Probability Legend

Executive Functions ?



TOVA

Name:	кауlа на	tfield			Test Date:	10/04/14	Version: 7.	2-4062
Subject:	01 0158	Gender :	Female	71 d	Birth Date:	10/13/96	Serial: 01	L6015
session:	01	Age:	1/9 110	210	Test Time:	03:11 PM	Test Type: V	15UA 1
S								
t 115					XX	XX	~~ ~~	84
n					ŶŶ	- XX XX	XX XX XX	P
d 100					XX	XX XX	XX XX XX	50 e
a		xx			XX X	x xx xx	XX XX XX	r
r er		XX	XX		XX X	x xx xx	XX XX XX	C C
d 85		XX XX	XX	XX	XX X	X XX XX	XX XX XX	16 e
5	X	-xxxx	XX	-XX	XXX XX X	xxxxx- x xx xx	-XXXXXX XX XX XX	1 T
c 70	XX XX	XX XX	XX XX	xx	XX X	x xx xx	XX XX XX	2 i
0	XX XX	XX XX	XX XX	XX	XX X	x xx xx	XX XX XX	1
r	XX XX	XX XX	XX XX	XX	XX X	x xx xx	XX XX XX	e
e 55	XX XX	XX XX	XX XX	XX	XX X	X XX XX	XX XX XX	.1
1.				<u>~~</u> _		2 03 04	_XXXXXX	
		variabili	itv	1 - C	Q± Q	Response	Time	
			-					
Std_Sc	79 84	87 97	73 93	89	119 9	8 115 113	108 114 113	
%ile	8 14	19 42	4 32	23	90 4	5 84 81	/0 82 81	
1								
S 115								
t								
a								
n 100					XX X	x	XX	
a	xx					x		
r	xx				XX X	x xx	XX	
d	XX-		-xx		XXX	xxx-	-XXxxXX	
	XX XX	XX XX	XX XX	xx	XX X	x xx xx	XX XX XX	
S 70	XX XX	XX XX	XX XX	XX	XX X	X XX XX	XX XX XX	
	$\sim \sim \sim$	\$\$ \$\$	\$\$ \$\$	XX XX				
r 55	$\hat{\mathbf{x}}$ $\hat{\mathbf{x}}$	$\hat{\mathbf{x}}$ $\hat{\mathbf{x}}$	$\hat{\mathbf{x}}$ $\hat{\mathbf{x}}$	xx	$\hat{\mathbf{x}}$		$\hat{\mathbf{x}}$ $\hat{\mathbf{x}}$ $\hat{\mathbf{x}}$ $\hat{\mathbf{x}}$	
e	XX XX	XX XX	XX XX	XX	XX X	x xx xx	XX XX XX	
.	XXXX	_xxxx_	_XXXX_	_XX_	XXX	xXXXX	_XXXXXX	
	Q1 Q2	Q3 Q4	H1 H2	т	Q1_Q	2 Q3 Q4	Н1 Н2 Т	
std sc	70 03	75 70	81 77	76	101 10	SSTOR (10a 1 75 88	102 81 82	
Stu St	15 95	13 19	01 //	10	101 10.	1 / 3 00	102 01 03	
Graph Ke	y: XX =	valid		ii =	above scale	b	lank = no data	a
	=	invalid		!! =	below scale			

The T.O.V.A. test results (below) are a quarter by quarter analysis of the test. These results, in combination with the ADHD Score (below and on Form 4) determine the T.O.V.A. Interpretation (see Form 1).



CPT For Attention

Cognitive Performa	nce Testing (CPT)		
		— Close	Report 🖓 Download Report
Baseline - 9/22/2014	-		
Paying Attention	Not Paying Attention	Impulsive	Response Time (ms)
100 What is this?	What is this?	2 3 What is this?	460.98 What is this?

CPT Short Term Memory

Cognitive Performance Testing (CPT)



Asymmetry Is Key Measure Mood Issue, Not ADD



Significant Changes In Five Sessions Asymmetry Training



External Factors Interfering With Biofeedback Training

- Poor Sleep
- Poor Diet
- Lack of Exercise
- Physical Trauma
- Mental Trauma
- Trauma Inducing Social Environments
- Trauma Inducing Physical Environments

Stress, Arousal & Physiology



Arousal & Social Accuracy Factors That Modify Behavior

- Individuals who cannot adjust their level of CNS arousal to the proper level required to perform a task are likely to make excessive errors in social exchanges.
- Excessive errors reduce their ability to successfully achieve their goals and get their needs met, resulting in distress.
- The ability to continuously monitor ongoing social cues and respond appropriately is a function of CNS processing as well as proper socialization.



Arousal

High

Low

Low

Figure 1: The Yerkes-Dodson Human Performance and Stress Curve

Sterman's Arousal Theory Connecting Arousal To Brainwave Correlates

- Sterman's research indicates that arousal is highly correlated with function in the human brain.
- Without the proper level of vigilance individuals cannot pay attention to ongoing social interaction.
- Without the proper level of sensorimotor integration individuals cannot control the behavior they emit in social environments.
- Without the proper cognitive integration individuals cannot make the correct social decisions.

Three Systems of brain activity influence thalamic generation of field potentials (Sterman, 1996).

• 1. Vigilance

If vigilance is withdrawn then theta brainwaves appear.

• 2. Sensorimotor Integration

If sensorimotor inputs are withdrawn then SMR brainwaves appear.

• 3. Cognitive Integration

If cognitive processing is withdrawn then alpha brainwaves appear.

Too Much Arousal Worry Impairs Cognitive Performance



Figure 2: Cells in the caudate known as tonically active neurons (TANs) tend to be found between striosomes and matrisomes. Striosomes are areas where information from an emotionprocessing part of the brain, the amygdala, reaches the caudate; matrisomes are clumps of axon terminals where information from the thinking, reasoning cerebral cortex reaches the caudate. By virtue of their position, TANs can integrate emotion and thought. They fire in a characteristic pattern when the brain senses something with positive or negative emotional meaning. Cognitive-behavioral therapy may change how TANs respond to OCD triggers.

Schwartz & Begley, 2002



TECHNICAL REPORT

The Lifelong Effects of Early Childhood Adversity and Toxic Stress

abstract

Advances in fields of inquiry as diverse as neuroscience, molecular biology, genomics, developmental psychology, epidemiology, sociology, and economics are catalyzing an important paradigm shift in our understanding of health and disease across the lifespan. This converging, multidisciplinary science of human development has profound implications for our ability to enhance the life prospects of children and to strengthen the social and economic fabric of society. Drawing on these multiple streams of investigation, this report presents an ecobiodevelopmental framework that illustrates how early experiences and environmental influences can leave a lasting signature on the genetic predispositions that affect emerging brain architecture and long-term health. The report also examines extensive evidence of the disruptive impacts of toxic stress, offering intriguing insights into causal mechanisms that link early adversity to later impairments in learning, behavior, and both physical and mental well-being. The implications of this framework for the practice of medicine, in general, and pediatrics, specifically, are potentially transformational. They suggest that many adult diseases should be viewed as developmental disorders that begin early in life and that persistent health disparities associated with poverty, discrimination, or maltreatment could be reduced by the alleviation of toxic stress in childhood. An ecobiodevelopmental framework also underscores the need for new thinking about the focus and boundaries of pediatric practice. It calls for pediatricians to serve as both front-line guardians of healthy child development and strategically positioned, community leaders to inform new science-based strategies that build strong foundations for educational achievement, economic productivity, responsible citizenship, and lifelong health. Pediatrics Jack P. Shonkoff, MD, Andrew S. Gamer, MD, PhD, and THE COMMITTEE ON PSYCHOSOCIAL ASPECTS OF CHILD AND FAMILY HEALTH, COMMITTEE ON EARLY CHILDHOOD, ADOPTION, AND DEPENDENT CARE, AND SECTION ON DEVELOPMENTAL AND BEHAVIORAL PEDIATRICS

KEY WORDS

ecobiodevelopmental framework, new morbidity, toxic stress, social inequalities, health disparities, health promotion, disease prevention, advocacy, brain development, human capital development, pediatric basic science

ABBRE VIATIONS

ACE—adverse childhood experiences CRH—conticotropin-releasing hormone EBD—ecobiodevelopmental PFC—prefrontal contex

This document is copyrighted and is property of the American Academy of Pediatrics and its Board of Directors. All authors have filed conflict of interest statements with the American Academy of Pediatrics. Any conflicts have been resolved through a process approved by the Board of Directors. The American Academy of Pediatrics has neither solicited nor accepted any commercial involvement in the development of the content of this publication.

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

All technical reports from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

Toxic Stress

Adverse Childhood Experiences (ACE)

- "From strong, frequent of prolonged activation of the body's stress response system in the absence of the buffering protection of a supportive adult relationship."
- Child Abuse
- Neglect
- Parental Substance Abuse
- Maternal Depression

Shonkoff & Garner, 2011, American Academy of Pediatrics

Health Effects of Toxic Stress

- Diminished Immune Function
- Inflammatory Process
- Neural Excitotoxicity
- Telomere Shortening- Early Aging
- Abnormal Fat Deposits
- Arterial Degradation

Toxic Stress Degrades Cognitive Abilities Childhood Trauma & Adult Emotional Trauma

- General Cognitive
- Linguistic Abilities
- Socio-emotional Skills
- Developmental Landmarks
- Decision-making
- Working & Short Term Memory
- Behavioral Self Regulation
- Mood and Impulse Control

Shonkoff & Garner, 2011, American Academy of Pediatrics

How Stress Effects Structure and Function of the Brain

- Hypertrophy of Amygdala
- Hypertrophy of Orbital Frontal Cortex
- Reduced Hippocampal Function
- Reduced Medial PFC Function
- Neuronal Death
- Degradation of Neuronal Connections

Shonkoff & Garner, 2011, American Academy of Pediatrics

Hypersensitivity to Stress **Reduced Socio**emotional Contextual **Evaluation** Degradation of **Memory Function** Loss of Top Down **Control and Increased** Impulsivity Loss of Cognitive Resources Loss of Cognitive **Function**

Fear and Arousal Exposure To Toxic Stress Enhances Fear Response





The amygdala modulates activity in the hypothalamus through the ventral amygdalofugal pathway

The Amygdala & The CNS

- The amygdala has direct control over hypothalamic, somatic, endocrine and brain stem functions.
- It indirectly precipitates immune system suppression through gluccocortical levels.

Extensive trauma can generate periodic recurrent paroxysmal activity in the amygdala. It is an important survival mechanism



Emotion- Memory- Attention The Three Frontal Circuits

- Chronic Anxiety generates excess cortisol which degrades hippocampal function.
- Loss of hippocampal function degrades ST Memory
- Loss of ST memory undermines sustained attention.



Anterior Cingulate

Trans-generational Behavior The Silent Process

- Parenting style alters RNA (Posner & Peterson, 2012).
- Behavior can be transmitted through

genes (Shonkoff & Garner, 2011).

• EEG is 80-90% heritable (Zietsch et al, 2007).



Gluten & Slow Wave Pre Post Gluten Map



8 Year Old: ASD/ ADHD

sex:	M	AGE:	8	DATI	12/28	/12 LAR	3 NO. 51756	6	CIJENT ACC	CT NO.	95321
	1.4	-		-	NUTR	IENT MIN	ERALS			_	
120.4	18		75	30-	10.5	7.5-	0.12	1 17 -	0.36	0.54 1	45.04
	17		777	749	4.0	70			0.34	0.51	44.0
118	10			20	0.0	1.0	0.11		0.32 -	0.48	49.0
104	15		60	201	8.1	0.0	0.10	56 -	0.30	0.45	41.0
96 -	14		60	24	8.4 -	6.0		4E -	0.28 -	0.42	38.4
88	13		56 -	22-	7.7	5.5 -	0.09	44 -	0.26	0.30	35.2 -
80	12	a company	50 -	20-	7.0	5.0 -	0.06	4C -	0.24	0.36 -	32.0 -
72-	11		45 -	18-	6.3 -	4.5 -	0.07	36 -	0.22 -	0.33	28.8
64-	10		40 -	36-	5.6-	4.0 -		35-	0.20	0.30	25.6 -
55-	9		35 -	14-	4.9 -	3.5 -	0.06	26 -	0.18-	0.27 -	22.4 -
48.4	8		30 -	12-	42 -	3.0	0.05	24 -	0.10-	0.24	19.2
40			15	-	9.6	25	0.04	4	0.14	0.19	16.0
40		10			0.5		0.01		0.10-	0.15	10.0
32			20.	2	2.01	201	0.03 -		0.08	0.12	12.0
24-	3		15.1	° .	2.14	1.5	- 50.0	12 -	0.05 - 2	0.09 -	9,6
16-		=	10 -	1	1.4 1	1.0		자물	0.04	0.06	6,4 -
	1	1	5 E	Z-	0.7	0.5	0.01	- 신물	0.02	0.m -	3.2
1.1	200						0.017	150	0.055	0.053	10.0
	2	2.0	5.0	- 3.0	1.4	11.9	0.012	,	0.000	0.032	10.0
all.	1 18		Sugar	all us	Se.	de.	diller.	the second	Alas	aller.	de m
90 (C	10 90 0	minin;	PRESENT TIT	PRIVER THT	10-0)	ADART JUL	Market Jan	HIGHLI WIT	THETHER SET.	(130) (30)	Con (P)
1 10		1	OXIC ME	TALS	1 10	12	1000	ADDIT	IONAL MIN	ERALS	
2,5	.45		0.10	.45-	4,0-		0.40	0.10	0.44	0.8	0.8
							0.35 -	B.ta.		0.7 -	0.7
2.0	.35		.08-	.35-	3,0-		0.30	0.12	0.33	0.6	0.0
					1000		0.25 -	0.10		0.5	0.5
1.5-	.25		0.06	25-	2,0		0.20-	0.05	0.22	0.4 -	0.4
					=		0.15	0.06		0.3	0.3
1.0	,15		2,04	.15-	1.0		0.10	0.04	0.11	0.2	0.2
~h			· · · · ·				0.05-	0.02-		0.1 -	0.1
0.0	0.00		0.00	0.00	0.0		0.00 =	0.00	0.00	0.0 =	1.0
0.01	4 0	009	0.003	0.007	1.80		0.013	C.001	0.003	0.001	N/A
	7 -	e -	a	a	N		10/	a	N	a	" M
Pipbi	1	Had	(Cd)	Man ales	E (AD		PIND	1Col	(P Mol	1 0.0	9 (B)
Mayouts 1	07 THO		PROPERTY AND	PRACE LES	PROVIDE THU		THE WEAK THESE	MONTON.	ANSAGA TED	PROMOTION	PREVENUE THE
_										-	
DEIRA I	IDEAL	CLIMPORT	16.05	PREMOUS	SIGNIFIC	ANT MIN	ERAL RAT	IOS		Lange of	
DITAR	HATID	10.00	0041	MATRO	anne	VVVVVV	~~~~~	854		THUR	
end terral (4.00	2.30	44	-	000000	00000)				
CAR	1.000	2.20	60	-	000000	00000	0				
GAK	4.17	1.000.000			and the second second second second	and an other designs of the local distance o					
GAK ANG NAK	4.17	1.00	40		000000	00		and the second s		And the second second	
GA/K AVMG NA/K NA/K	4,17 1.00 8,00	1.00	40 208		000000	000000	000000	000000	000000	00000	00000
GA/K AANG NA/K N/CU DA/P	4,17 1.90 8,00 2.50	1.00 16.67 1.10	40 208 44		0000000		000000		0000000	00000	00000



Note: Elevated Theta, Alpha, Delta Slowed Alpha Dominant Frequency Frontal Connectivity Abnormality

8 Year Old Metabolics

is		
Scor	Metabolic Category	Symptoms Reported
7	Blood Sugar	Crave Sweets Irritable with missed meals Agitation
6	Gall Bladder	Reddened Skin Dry or flaky skin Food intolerances Itchy Skin
4	Thyroid (hypo)	Fatigue Cold all the time
3	Gastrointestinal	Food intolerances Diarrhea Constipation
2	Liver	Fatigue Loss of Appetite Diarrhea
2	Kidney	Fatigue
2	Cardio- Vascular	Fatigue
1	Adrenals	Fatigue
1	Somatic	Restless Legs

MetaMetrix Lab Test Findings: IgG4 Tested Positive for Egg Intestinal Hyperpermeability Oxidative Stress

RX

Given Glutithione and B6 with Dietary Restrictions

8 Year Old: Hair Analysis Details

EX:	м	AGE:	8	DATI	12/28/	12 LA	B NO. 51756	6	CLIENT AG	CT. NO.	95321
-		-	-	-	NUTRI	ENT MIN	FRAIS		All second second		
120.4	18		70-	304	10.5	7.5-	0.12	60 -	0.36	0.54	46.04
	17		m	200	9.9	7.0-		10	0.34	0.61	44 B
112	16		~	204	0.0		0.11		0.32	0.48	44.0
104	15	11	60 -	201	6.1	6.5	0.10	50 -	0.30	0.45	41.0
96 -	14		60	24-	8.4 -	6.0		4E -	0.28	0.42	38.4
88	13		55 -	22-	7.7	5.6 -	0.09 -	44 -	0.26	0.39	35.2
80	12	the second	50 -	20-	7.0	5.0 -	0.06	40 -	0.24	0.36 -	32.0
72-	11		45 -	18-	6.3 -	4.5 -	0.07	36 -	0.22 -	0.33 -	28.8
64-	10		40 -	16-	5.6-	4.0 -		35 -	0.20	0.30	25.6
55-	9		35 -	14-	4.9 -	3.5 -	0.06	26 -	0.18-	0.27 -	22.4
10	8	1 3	30 -	12-	42.	30.	0.05	24.	0,16-	11,24 -	19.2
40				- 4.5	95	9.6	0.04	45	0.14-	0.21	10.0
40					0.5		0.01		0.10	0.15	10.0
35	4		201	2	2.91	2.01	0.03 -	10.	0.08 -	0.12	12.0
24-	3.		15-	•	2.1	1.5	- 50.0	12 -	0.05 - 2	0.09	9,6 -
16-		=	10	418	1.4 1 🗃	1.0	100	자물	0.04	0.06	6.4
8-	1.	=	5 m	~ 물	0.7	0.5	0.01	41 E	0.02 -	0.03 -	3.2
L											
11.	<u>0</u>	2.0	5.0	5.0	1.4	0.9	0.012	15.0	0.066	0.052	10.0
with.	. A	e .	M	155	D.	all	aletter.	N	de.	- State	Alle
9° (C	a) 98 (h	Agi 2	P-{Na}	50° 10	(Fe)	27 (Cu)	49"(Mn)	_1Zn)	(Cr)	9 (Se)	(P)
				10000		100000000				1	1
1		T	OXIC MI	ETALS	The second second	1000		ADDIT	IONAL MIN	ERALS	
2.5	.45	0	10	.45-	4.0		0.40	0.16	0.44	0.8	0.8
							0.35	n.ta.		0.7	0.7
2.0	.35		.08-	35-	3.0 -		0.30	0.12	0.33	0.6	0.0
		1 1 1					6.25	0.10		0.5	0.5
1.5	.25	0	06	25-	2.0		0.20	0.05	0.22	0.4	0.4
100	1000	1 2	233	2012	=		0.45	0.00			
1.0	.15										0.01
28			V/# 1	.15-	1.0		0.10	0.04	0.11	0.3	0.2
				.15-	1.0		0.10	0.04	0.11	0.2	0.2
	0.00		00	.15-	1.0		0.10	0.04 0.02 0.00	0.11	0.2	0.2
0.0	0.00		0001	0.007	0.0		0.05	0.04	0.00	0.3 0.2 0.1 0.0	0.2 0.1 0.0
0.0	4 0.0	009	0.003	0.00	1.0 0.0		0.10	0.04 0.02 0.00 0.00	0.00	0.2 0.1 0.0 0.001	0.2 0.1 11.0 N/A
0.0	4 0.0	009	0.003	.15- 0.00- 0.007	1.0 0.0 1.80		0.10	0.04 0.02 0.00 C.001	0.11	0.2 0.1 0.001	0.2 0.1 0.0 N/A
0.01	4 0.00	009 009	0.003	.15- 0.00- 0.007 (A6)			0.10 0.05 0.00 0.013	0.04 0.02 0.00 C.001	0.11 0.00 0.003	0.2 0.1 0.0 0.001	0.2 0.1 11.0 N/A
0.0 0.01	4 0.0 4 0.1 9 00 10 00		0.003	.15- 0.00 0.007 (0.007 (0.6) (0.007) (0.007) (0.007) (0.007) (0.007) (0.007) (0.007) (0.007) (0.007) (0.00)	1.0 0.0 1.80		0.10 0.05 0.00 0.013	0.04 0.02 0.00 0.001 C.001	0.11 0.00 0.003 (Moj monario	0.3 0.2 0.0 0.001	0.2 0.1 1.0 N/A
0.0 0.01	4 0.0 4 0.1		00 0.003	.15- 0.007 0.007 	1.0 0.0 1.80	ANT MIN	0.10 0.05 0.00 0.013 0.013	0.04 0.02 0.00 C.001 (Co) (Co)	0.11 0.00 0.003 0.003	0.3 0.2 0.1 0.001 0.001	0.2 0.1 10 N/A
0.0 0.01	4 0.00 4 0.1 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9		0.003	15- 0.001 0 007 9463 78630 TH	1.0 0.0 1.80 MIAIN SIGNIFIC	ANT MIN	0.10 0.05 0.00 0.013 040 10000	0.04 0.02 0.00 0.00 0.00 0.00 00 00 00 00 00	0.11 0.00 0.003 (Ma) Measurin	0.3 0.2 0.1 0.00 0.001 PREMI TO PREMI	0.2 0.1 0.0 N/A
0.0 0.01 996	4 0.00 4 0.1 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	009 009 009 009 009 009 009 009	0.003 0.003 4Cd) 10004 10004 8004 82	.15- 0.007 94.6) 94.60 94.60 TH 94.60 TH 94.60 TH	1.4 0.0 1.80 Main Signific		0.10 0.05- 0.00 0.013 0.013 040 10000 100000	0.04 0.02 0.00 C.001 F(Co) F(Co) F(Co) F(Co) F(Co) F(Co) F(Co) F(Co) F(Co) F(Co)	0.11 0.00 0.003 Mag Mag	0.3 0.2 0.1 0.001 0.001 PREMIER TEX 1900H	0.2 0.1 0.0 N/A
0.0 0.01 PPb MINUE MINUE ArMS CAVK	4 0.00 4 0.1 70 70 70 70 70 70 70 70 70 70 70 70 70	009 009 009 009 009 009 009 009 009 009	0,003 0,003 4Cd) 1004 1004 1004 1004 1004 1004 1004 100	.15- 0.007 946) 9460 TH 94000 TH	1.4 0.0 1.50 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5	ANT MIN	0.10 0.05 0.013 0.013 0.013 0.013 0.013 0.013	0.04 0.02 0.00 C.001 /(Co) //(Co) //(Co)	0.11 0.00 0.003	0.3 0.2 0.1 0.001 0.001 PERMIT	0.2 0.1 1.0 N/A
0.0 0.01 996	0.00 4 0.1 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9	0009 0000 0009 0000 0000 0000 0000 0000 0000	000 0.003 4C41 0.003 800 800 800 800 800 800 800 800 800	15- 0.007 0.007 94.6) 94600 TH			0.10 0.05 0.013 0.013 0.013 0.013 0.013 0.013 0.013 0.013 0.000 0.000 0.0000000000	0.04 0.05 0.00 0.001 0.00000000	0.11 0.00 0.003	0.3 0.2 0.1 0.0 0.001 PREMIT	0.2 0.1 10 N/A
0.0 0.01 VPb MINUET	0.00 4 0.1 9 9 10 10 10 10 10 10 10 10 10 10	6,009 6,000 6,	000 0.003 4Cdt 0.003 8004 8004 8004 8004 8004 8004 8004 8	15- 0.007 (0.007 (0.007 (0.007) (0.007	1.6 0.0 1.80 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7 7	ANT MIN 1000 00000 000000 000000	0.10 0.05 0.00 0.013 0.013 0.013 0.013 0.013 0.013 0.000 0.013	0.00- 0.00- 0.00- C.001 P(Ga)P	0.11 0.00 = 0.003 Mol	0.3 0.2 0.1 0.0 0.001 70001 70001	0.2 0.1 10 N/A
0.0 0.01 2001 2000 2000 2000 2000 2000 2	0.00 4 0.1 10 10 10 10 10 10 10 10 10 1	CLEVENT 6000 5.50 2.20 2.50 1.00 16.67	0,00 0,003 4Cd) 8000 BF 8000 B	15- 0.09- 0.007 	1.6 0.0 1.50 1.50 SIGNIFIC 0.00000 0.00000 0.00000 0.000000	ANT MIN 1000 30000 30000 30000 30000	0.10 0.05 0.00 0.013 0040 0040 0040 0040 0040 0040 000000	0.04 0.05 0.00 C.001 MICel MURRINIT	0.11 0.00 = 0.003	0.3 0.2 0.1 0.0 0.001 0.00000000	0.2 0.1 1.0 N/A (B) Weeks TUT

Very low readings overall:

Electrolyte Imbalances Poor Eliminator, Digestive Issues

"Four Lows:" First four categories low Indicates exhausted system Difficulty breaking down protein, Adrenals at 60% function

Sodium low

Zinc/copper Imbalance

Indicates Thyroid Regulation Problem Selenium Low- needed for T3-T4 Conversion

Chromium low

Blood Sugar Regulation Problem

8 Year Old: Pre Post Map

30 Sessions Improved Sleep Better Concentration Emotional Stabilization

Temporary Suspension of NFB to focus on metabolics.



NOTE: Pre-Post Maps Do Not Have To Turn All Green To Have Significant Changes In Symptoms!