

Trauma Informed Neurofeedback of Adolescents in Residential Treatment

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Clinical Neuroscience Association**

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Financial Disclosure

*I am an instructor with Stress Therapy Solutions, Inc. as an independent contractor (GNI Neurofeedback Bootcamp program, including teaching and mentoring).

*I have assisted Brainmaster, Inc. at trade shows.

*The residential treatment center featured in this presentation has not compensated me for this presentation.

Seeking APA Credit?

"Materials that are included in this course may include interventions and modalities that are beyond the authorized practice of mental health professionals. As a licensed professional, you are responsible for reviewing the scope of practice, including activities that are defined in law as beyond the boundaries of practice in accordance with and in compliance with your professions standards."

Notice required by the American Psychological Association regarding APA CE credit.

Part I: Background to Study

A FEW FACTS ABOUT CHILD & TEEN MENTAL HEALTH AN

- *One in five children birth to 18 has a diagnosable mental disorder.
- *One in 10 youth has a serious mental illness sufficient to impair function in daily life.
- *Roughly half of all lifetime mental health disorders start by the mid-teens.

Stageman, Shannon & Cooper, Janice. (2010). Children's mental health: what every policymaker should know. National Center for Children in Poverty, Columbia Univ.

Facts About Residential Treatment of Adolescents

- * In 2008 an estimated 50,000 adolescents were in a residential treatment center (RTC) at any given time (www.bazelton.org).
- * Typical histories included foster placements, outpatient treatment, multiple inpatient hospitalizations, as well as previous group home and residential treatment.

Five Years Later

In 2013, according to the National Conference of State Legislatures, just shy of 56,000 kids were in group homes, residential treatment facilities, psychiatric institutions and emergency shelters.

www.ncsl.org/research/human-services/congregate-care-home-state-legislative-enactments.aspx

More Facts about this Population

- *Some youths in RTC's are Court ordered for admission or are admitted by their family.
- *Some will become among the seriously and persistently mentally ill largely served by public mental health centers.
- *Some wind-up in prison.
- *However, many of these young people will rise above adversity and become well-adjusted productive citizens.

Jackson-Feild Behavioral Health Services

Location of the study

Jackson-Feild Behavioral Health Services (JFBHS) Jarratt, VA



What kind of kids receive residential treatment?

Answer: Kids who've flunked-out of about everything else that has been tried in an effort to help them regulate their behavior and emotions!

Typical Resident Profile

- * History of multiple psychiatric admissions, multiple residential admissions, disrupted adoptions, physical and/or sexual abuse, trouble with the law and so-on.
- * Many of these young people represent those having developmental trauma as described by Fisher and van der Kolk.
- * Fisher, Sebern F. (2014). Neurofeedback in the treatment of developmental trauma, Norton.
- * van der Kolk, M.D. (2014). The body keeps score, Penguin/Random House.

Characteristics of Developmental Trauma and Common Diagnoses

- * Attachment rupture and the motherless child
- * Poverty
- * Affect dysregulation
- * Sensory dysregulation
- * Failure to bond
- * Helplessness/hopelessness
- * A lack of sense of self/others
- * Neglect
- * Failure to develop empathy

*Mood disorders (MDD, BPD/O, etc.)

*Anxiety disorders (GAD, social anxiety, phobias, etc.)

*Reactive Attachment Disorder

*Post-Traumatic Stress Disorder

*Dissociative Identity Disorder

Fisher, Sebern. (2014).

Neurofeedback in the treatment of developmental trauma. Norton.

Jackson-Field Demographics 1-1-16 to 6-30-16

Age: 6-12 = 3 (7.5%) 13-17 = 35 (87.5%) 18-24 = 1 (2.5%)
Unknown = 1 (2.5%)

Gender: Female = 28 (70%) Male = 12 (30%)

Race: African-American = 22 Bi-Racial = 1 Filipino = 1
Hispanic = 3 mulato = 1 Caucasian = 12
Total/Census = 40



Part II: The Effects of Stress

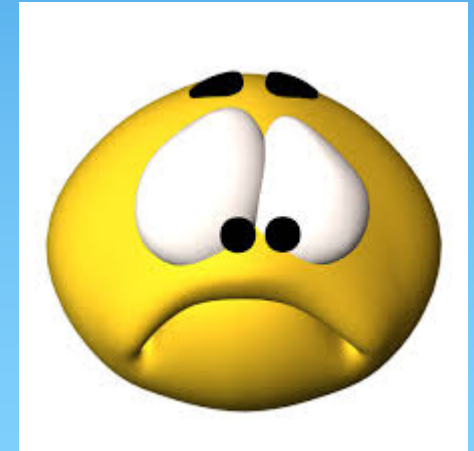
Types of Stress

Eustress (good)

Distress (bad)

How do we adapt to stress?

GAS



(sometimes literally!)

Figure 1. General Adaptation Syndrome aka **GAS** (Hans Selye, 1936)

Physiological response to stress

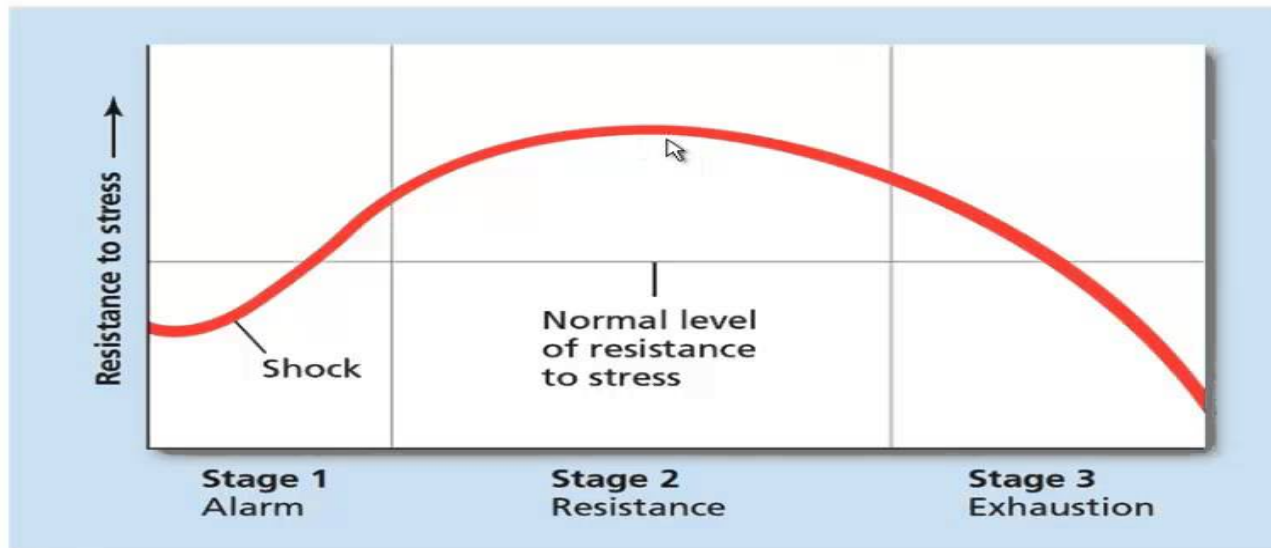


Figure 2. Stress, **GAS** and Psychopathology



SO, WHAT WHICH BRAIN SYSTEMS
ARE IMPACTED by STRESS?

***Answer: ALL OF
THEM!***

via the

lymbic system, hypothalamus, pituitary and

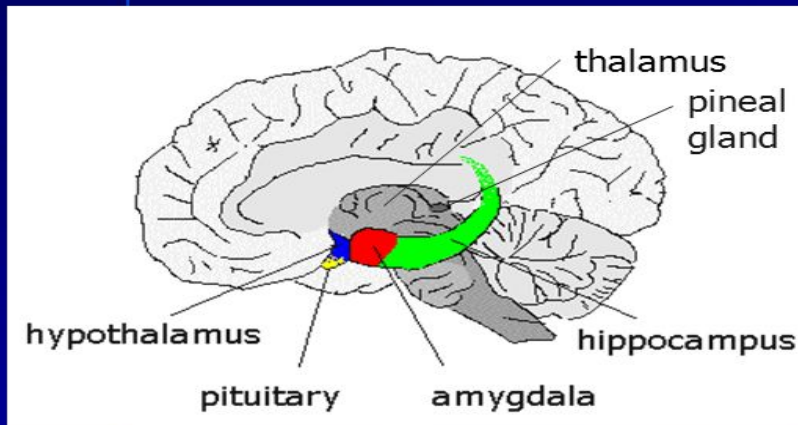
Olfactory bulbs Hippocampus Hypothalamus
Anterior thalamic nuclei Fornix Columns of fornix
Cingulate gyrus Mammillary body Septum
pellucidum Habenular commissure Cingulate
gyrus Entorhinal cortex Parahippocampal gyrus
Amygdala and Limbic mid-brain areas

The Lymbic System

(With thanks to Wikopedia)

Figure 3. The “white matter” lies under the cerebral cortex. The limbic system is part of white matter.

Limbic System & Associated Structures



- Limbic System
 - Hippocampus
 - Amygdala
 - Thalamus
 - Hypothalamus
- Pituitary Gland
- Pineal Gland

Homeostasis

The tendency of the body to achieve and maintain a state of relative internal balance; requires various feedback mechanisms (ex. The HPA axis)

Figure 4. The HPA Axis is Especially Important in the Maintenance of Homeostasis

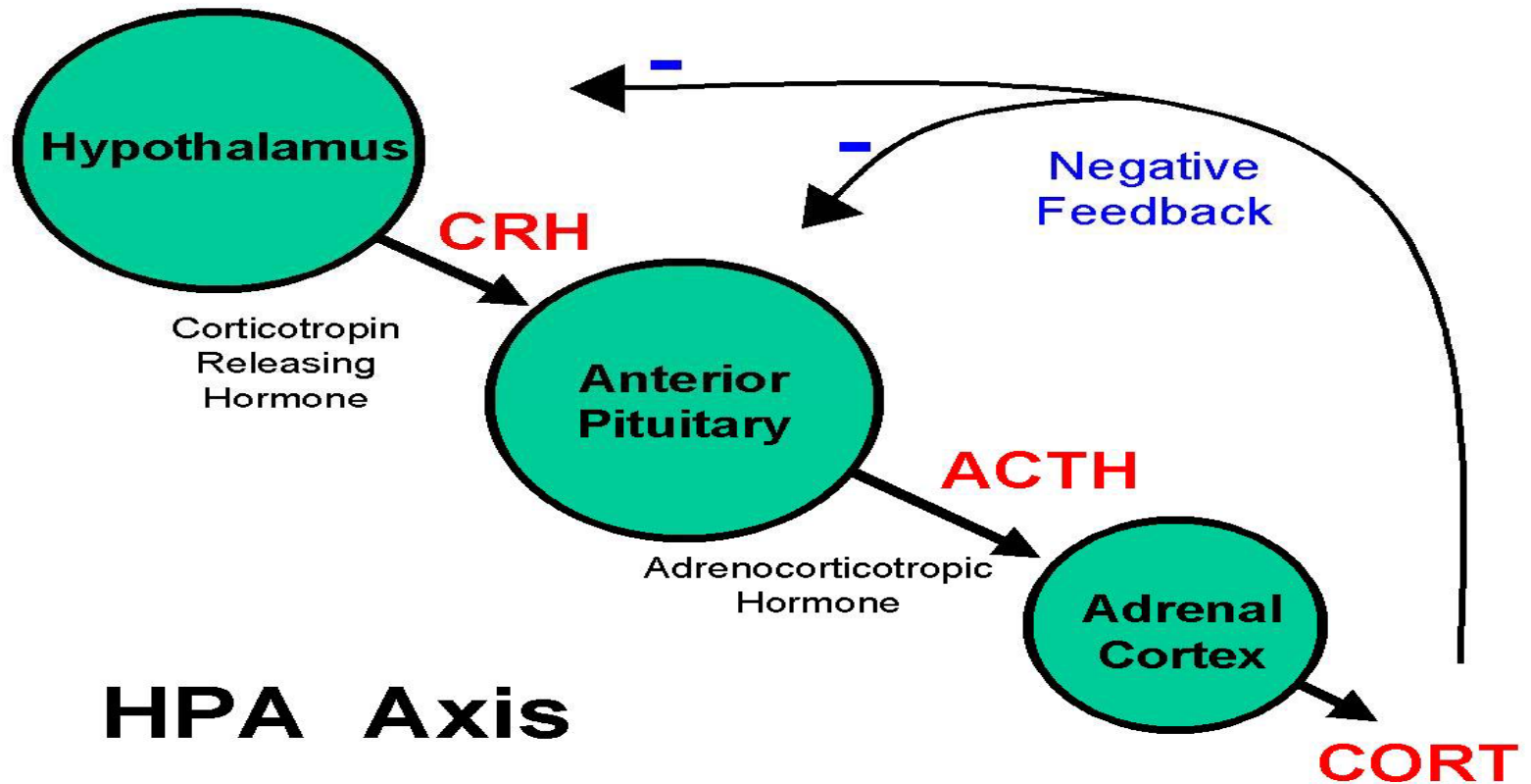
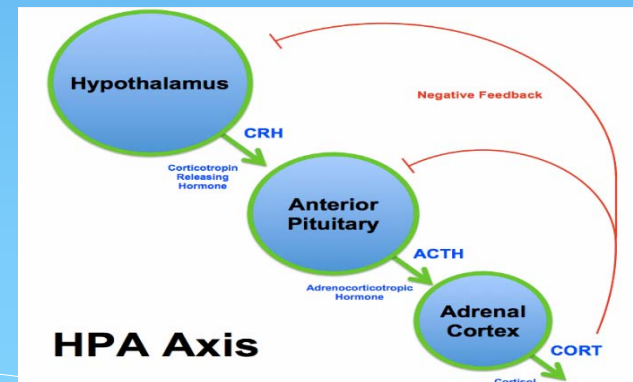
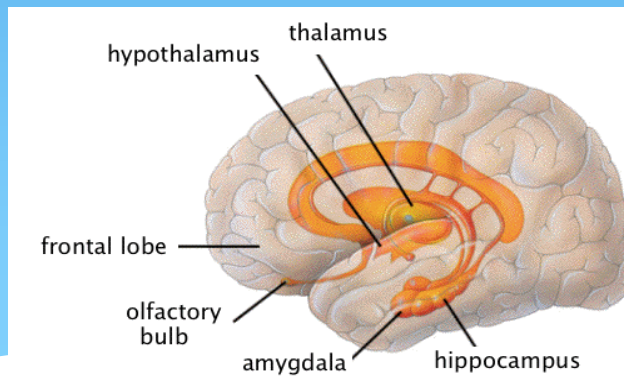


Figure 5. Cortical and HPA Interaction

Brain and body are linked by nerves *and* neurochemicals. The hypothalamic–pituitary–adrenal axis (HPA) is an important component of this mechanism. The HPA is a chain of interaction and feedback ... **Cortisol** produced in the **adrenal cortex** will “feedback” to inhibit both the hypothalamus and the pituitary gland. Cortisol can pass through the blood **brain** barrier where it can interact with the **brain** and activate **the HPA axis**.



The brain-body neuroendocrine connection is not further addressed .

(with thanks to Wikipedia; Meewisse et al.,2007; Flory, et al, 2013; Gers & Rapport, 2013)

HOW **STRESS** AFFECTS THE BODY

BRAIN

Difficulty concentrating, anxiety, depression, irritability, mood, mind fog

CARDIOVASCULAR

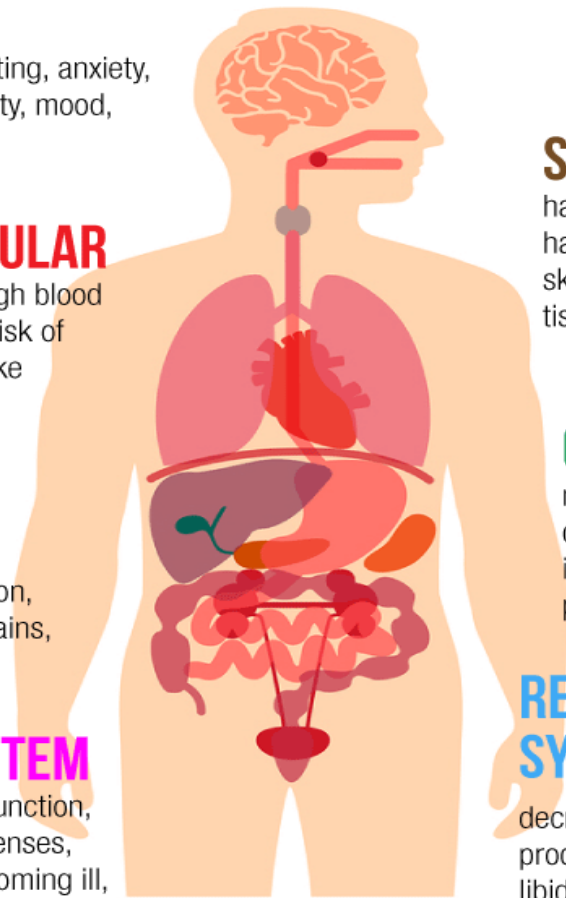
higher cholesterol, high blood pressure, increased risk of heart attack and stroke

JOINTS AND MUSCLES

increased inflammation, tension, aches and pains, muscle tightness

IMMUNE SYSTEM

decreased immune function, lowered immune defenses, increased risk of becoming ill, increase in recovery time



SKIN

hair loss, dull/brittle hair, brittle nails, dry skin, acne, delayed tissue repair

GUT

nutrient absorption, diarrhea, constipation, indigestion, bloating, pain and discomfort

REPRODUCTIVE SYSTEM

decreased hormone production, decrease in libido, increase in PMS symptoms

Figure 6.

A new diagnostic category has been proposed to describe the type of individuals described in this presentation.

Developmental Trauma Disorder

Figure 7. What are some of the known consequences of developmental trauma on the brain? Dysregulation of various systems needed for homeostasis!

Debellis, Michael D., MD, MPH. and Zisk, Abigail A., A.B. 2014. *Child & Adolescent Psychiatr. N Am*, April 23(2), 185-222.

Neurotransmitters

Dopamine, serotonin,
norepinephrine

Cortisol

Prefrontal
Cortex/DFM

Amygdala

Hypothalamus

More Brain Effects of Developmental Trauma

Plasticity/synaptogenesis disrupted
Glial cells impacted
Neuronal injury and death
Myelination patterns changed
Neuroendocrine alteration

Figure 8. Allostasis (the process by which the body responds to stressors in order to regain homeostasis)

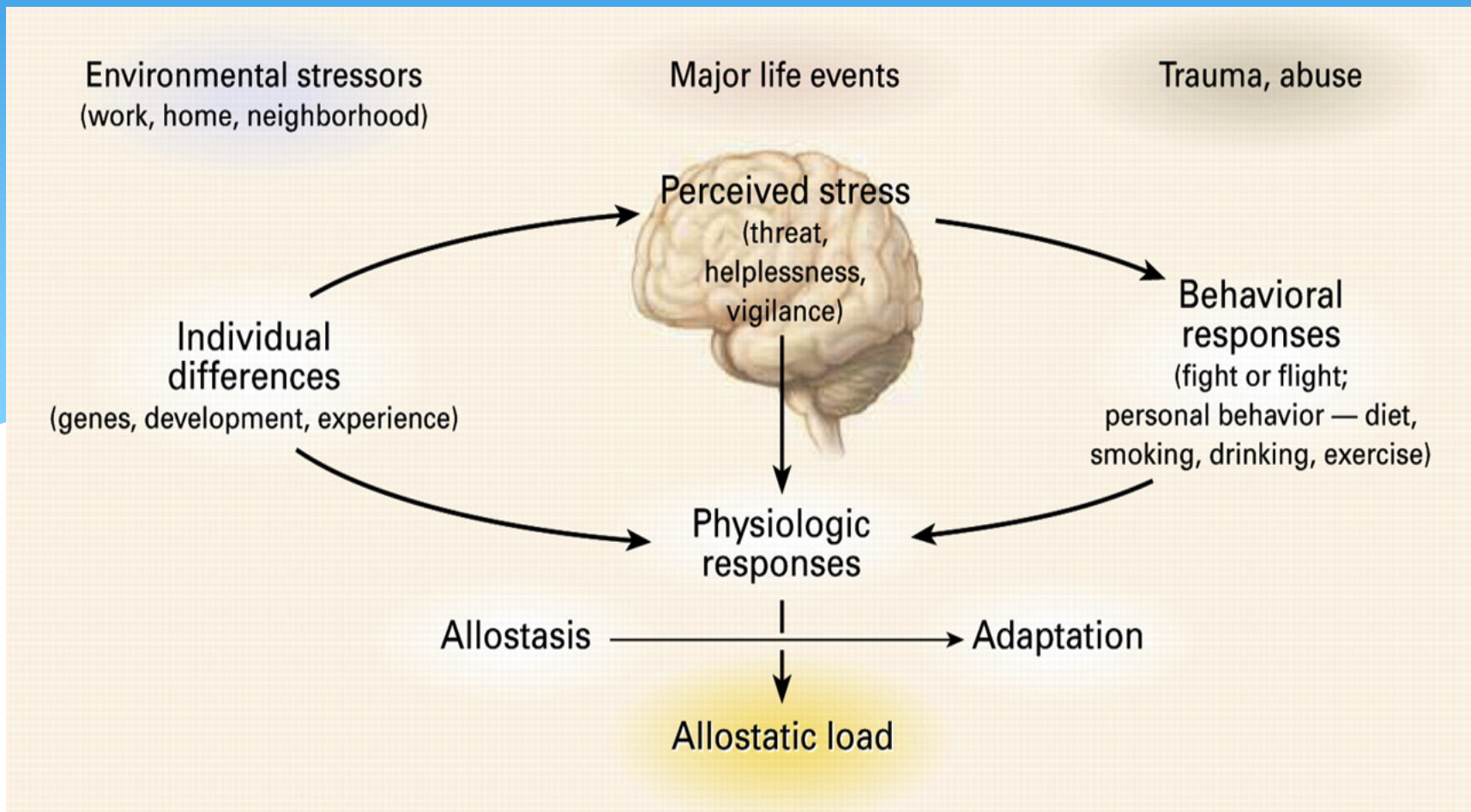


Figure 9. Traumatic Stress Delivers a Grand Slam to the Victim...A Real Knock-Out Blow!



Part III: The JFBHS Study

Significant misbehavior and emotional outbursts result when allostasis breaks down.

At Jackson-Feild and other RTCs Incident Reports reflect the dysregulation caused by trauma.

Incident Reports were the Outcome Measure used in this 2016 Study

What is an IR Anyway?

An IR is a report of unacceptable behavior, a violation of the code of conduct at JF. Violations carry consequences, usually a decrease in level of privileges.

Table 1. IR Examples (conduct issues)

- *Resident to Resident Verbal Aggression
- *Self Injury (ex. Cutting, ingestion of toxic material, etc.)
- *Resident to Staff Physical Aggression
- *Possession of Contraband
- *Boundary Issues
- *Need for Restraint
- *Resident to Resident Physical Aggression
- *Runaway Attempt



Treatment at JF Includes

- * **Psychotropic medications are used according to current medical practices**
- * **Trauma-informed psycho-social interventions (individual, group, family, art, and play therapies)**
- * **PLUS**
- * **Neurofeedback**

Psychopharmacology

TABLE 2. Partial list of psychotropic and other medications used at JF. Most patients are on two or three of these medications QD or BID, plus another QHS, and, an IM for acute acting-out that can't be managed otherwise.

Celexa	Ritalin	Trileptal	Lithium
Prozac	Concerta	Buspar	Minipress
Zoloft	Focalin	Clonazepam	Anafranil
Vistaril	Tenex	Naltrexone	Geodon
Wellbutrin	Adderall	Navane	Zyprexa
Lexapro	Abilify	Anafranil	Lamictal
Trazodone	Vyvanse	Saphris	Clonidine
Depakote	Amphetamine	Haldol	Latuda



Diagnoses

At the time of the current study, this was the mix of diagnoses at JFBHS:

TABLE 2. Primary diagnoses of JF patients (N=84).

<u>Diagnoses</u>	<u>Number</u>	<u>Percent</u>
Mood D/O (Depression, Bipolar, etc.)	11	13.5
Mood D/O + Psychotic D/O	2	2.7
Mood D/O + Anxiety D/O (other)	3	4.0
Mood D/O + Trauma Related D/O (PTSD)	6	6.8
Mood D/O + Conduct D/O	14	16.2
Mood D/O + Substance Use D/O	5	5.4
Mood D/O + Paraphilic D/O	2	2.7
Mood D/O+ Reactive Attachment D/O	1	1.4
Mood D/O + Borderline Personality D/O	23	27.0
Conduct D/O + Borderline Personality D/O	3	4.0
Mood D/O + Antisocial Personality D/O	1	1.4
Mood D/O + ADHD	13	14.9
Total	84	100.0

Table 3: Trauma-Related Diagnoses

PTSD

BPD

Reactive Attachment Disorder

These diagnoses represented over
33% of the total at JFBHS in 2016.

Neurofeedback at JFBHS

Neurofeedback has been used since 2003 with brief interruptions when providers relocated.

Selected Types of Neurofeedback

I. Hemoencephalography (HEG)

A. Near Infrared HEG or nIR (Toomin)

B. Peripheral HEG or pIR (Carmen)

II. Electroencephalographic Biofeedback

A. Neurofeedback (NFB)

1. Amplitude training (1 to 4 channels)

2. Z-Score Neurofeedback (2-19 channels)

3. sLoreta Neurofeedback (19 channels)

4. Infralow Neurofeedback (ISF)

B. Combined Methods (NFB, pEMF, auditory, photic)

Neurofeedback Principles

(Collura, 2014; Demos, 2005; Hammond, 2014)

- * Neurofeedback takes advantage of brain **plasticity** to support and reinforce clinical goals in a manner consistent with evidence-based practice;
- * EEG equipment then provides instantaneous real-time feedback about the brain's activity;
- * **Learning principles** at work in neurofeedback include (1) operant conditioning, (2) classical conditioning, (3) concurrent learning, and (4) self-efficacy (resulting from self-empowerment and self-awareness).

What is EEG?

- * EEG is produced mostly produced by neurons.
- * EEG is a graphic display of a difference in voltages [measured] from two sites of the brain....recorded over time (Tatum, 2008).
- * It consists of electrical energy assigned to bands of differing frequency, measured in cycles per second or Hertz.
- * Typical bands include delta, theta, alpha, low beta, beta, high beta, and gamma, recognized by appearance and frequency (cycles per second or Hertz).
- * Units of measurement include amplitude (uV), frequency (Hz), amperage, asymmetry, coherence and phase.

Functional Neuroanatomy I

- * Cortical neurons (largely pyramidal cells) arranged in layers averaging a total of about 3 mm in depth, produce most of what we call EEG. Neurons are mostly impacted by NFB.
- * Operant conditioning and other learning mechanisms influence neuronal electrochemical behavior. Volume conduction and other mechanism carry EEG within the brain, through tissues and fluids, and to the scalp (Collura, 2014) where they are measured.

Functional Neuroanatomy II

*The amygdala, thalamus, hippocampus and other subcortical structures may also be impacted by neurofeedback, thus influencing emotions and behavior. Thalamo-cortical reverberations carry EEG encoded messages in a top-down and bottom-up manner (Collura, 2014).

*The “Connectome” links cortical networks and subcortical regions into function-related units.

*Trauma interferes with normal brain maturation and regulation.

EEG Bands and Typical Related Brain-Behavior Relationships

(With thanks to Google Images)

FIGURE 10. EEG basics

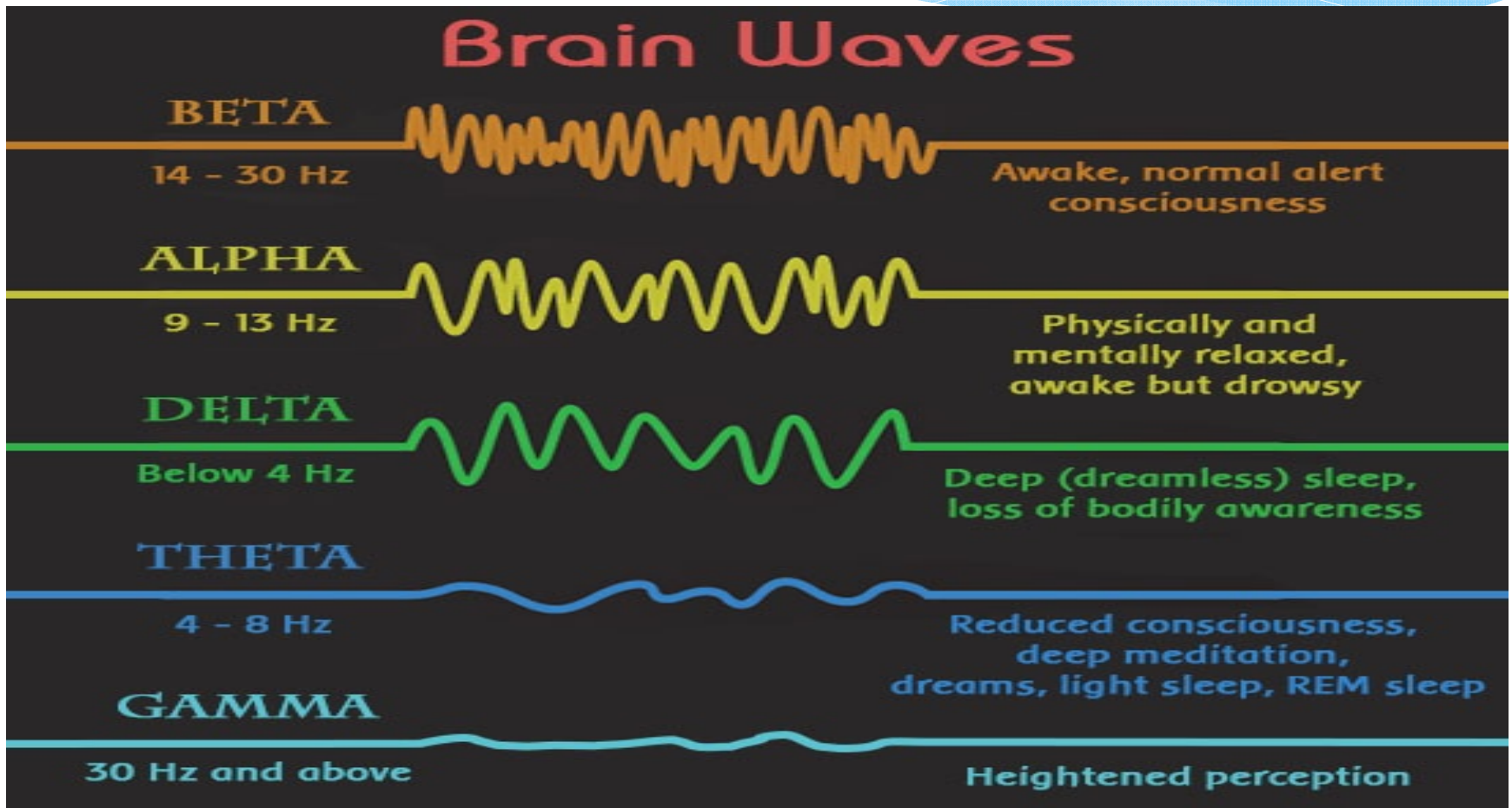
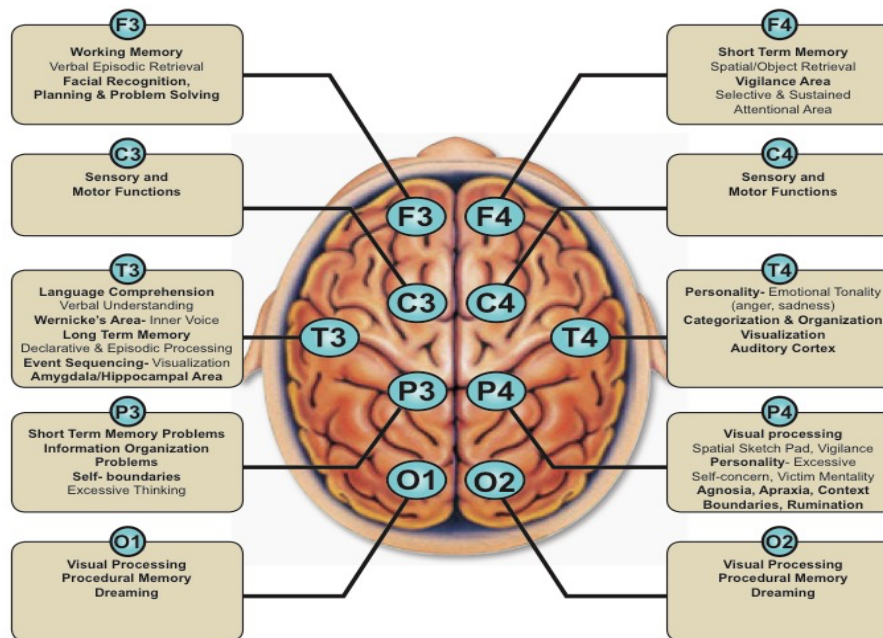


Figure 11. 10-20 Sites and Behavioral Correlates

CLEAR MIND CENTER[®]
 CREATING EEG SENSORY SOLUTIONS
 EQUIPMENT • TRAINING • CLINICAL SETUP

10-20 Correlations of Function



Three Brain Networks You Should Know About

Default Mode Network: According to Fisher (2014) the DFM is also called the “resting mode (state) network” and is self-referential and responsible for self-reflection...and sets the context for future information processing. It is also in-charge of the “sense of self.”

Salience Network: The “alertness network,” according to Fisher “responds to stimuli prequalified as worthy of attention.” It also facilitates switching between other important networks allowing working memory and attention to activate appropriately.

Central Executive Network: This is a frontoparietal network crucial to working memory and cognitive control of thought, emotion and behavior (wikipedia).

Figure 12. Stress Networks of the Brain: Salience network, Executive control network and Default mode network

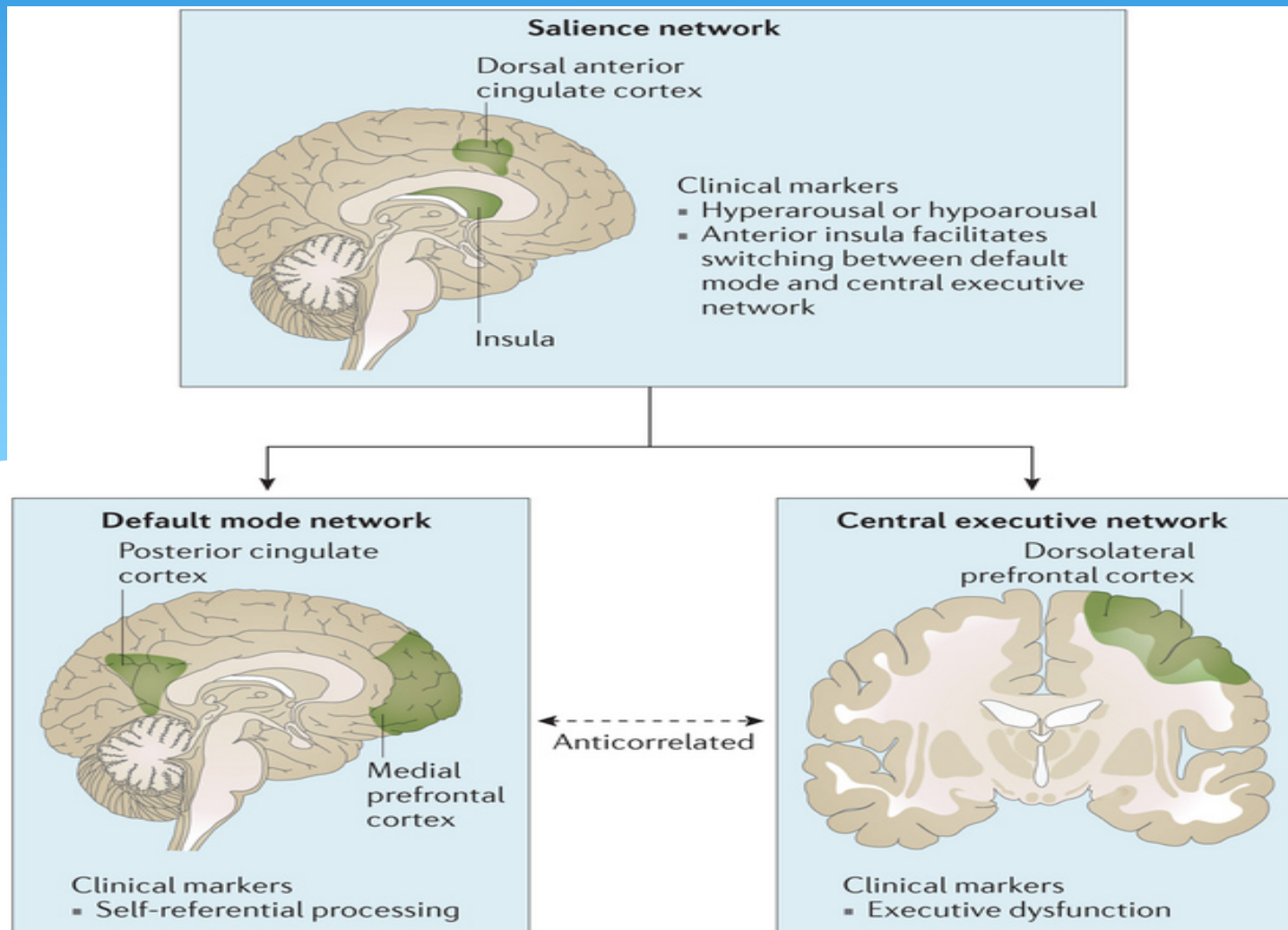
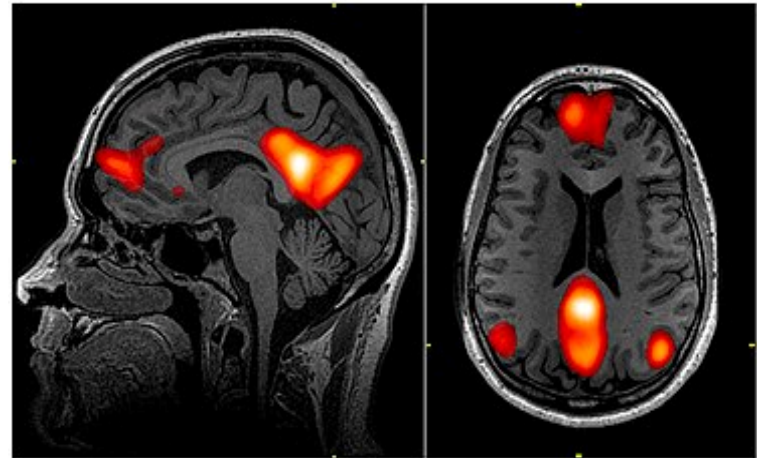
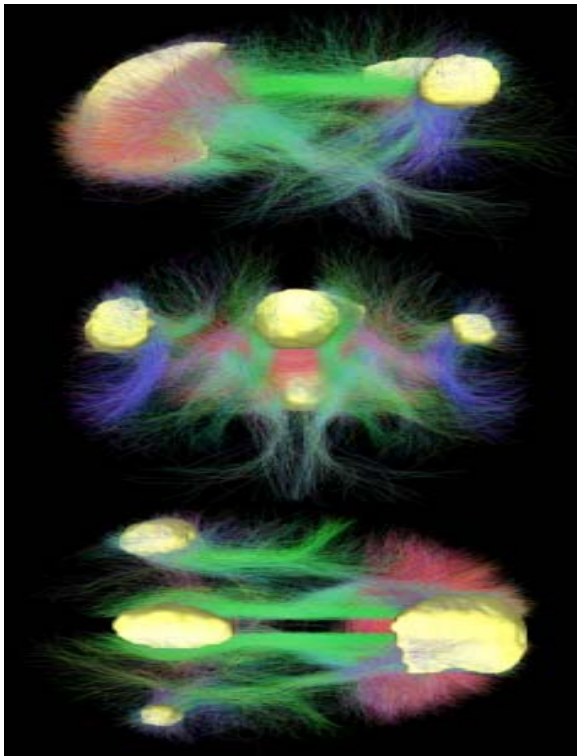


Figure 13. The Default Mode Network and Connectome (with thanks to Wikipedia)

Left: diffusion tensor image Right: fMRI image

The medial pfc, dorsal cingulate, and dorsolateral DFM is shown

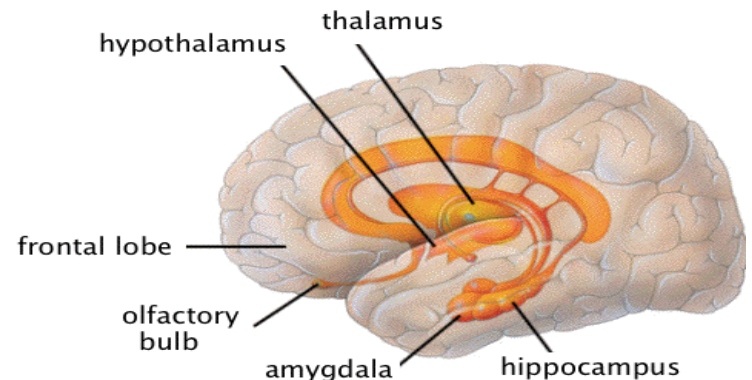
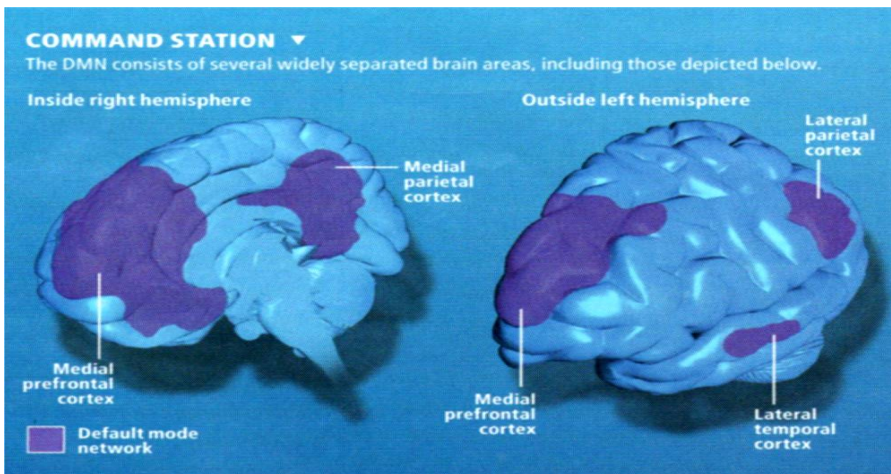
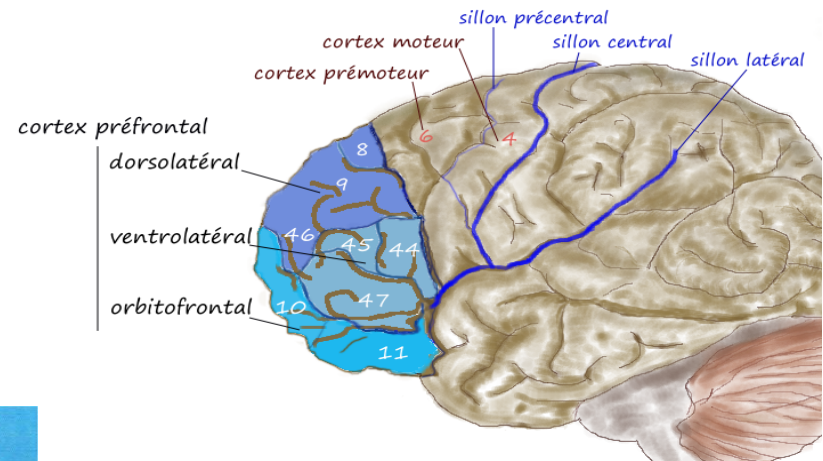
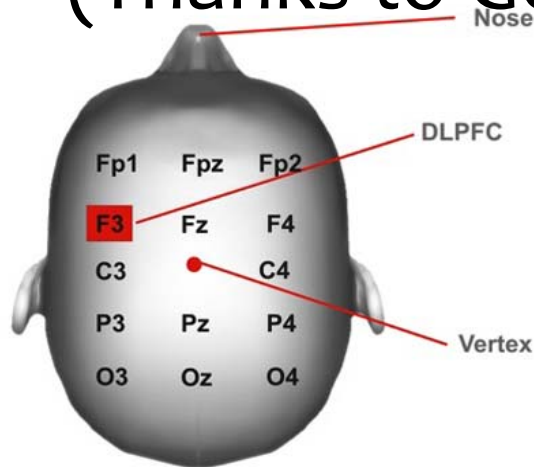


The DFM Gets Most of the Attention!

(with thanks to Wikipedia; Broyd, 2014; Buckner, et al., 2008)

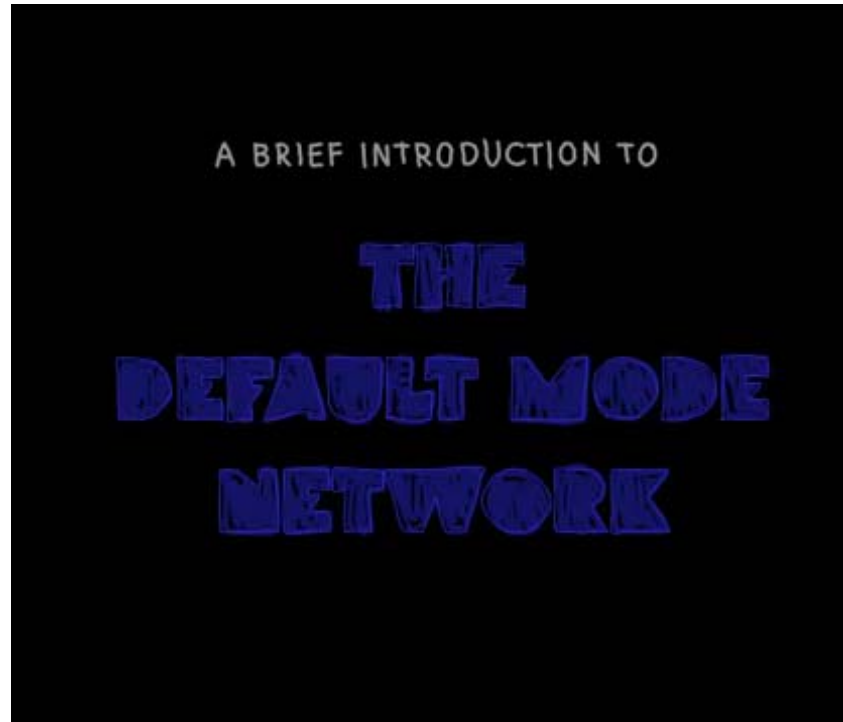
- * The ***default mode network*** activates when a person isn't focused on the outside world and the brain is at wakeful rest, such as during daydreaming and mind-wandering. It is also activated when one thinks about others, about themselves, remembers the past, and plans for the future. The network activates "by default" when an individual is not involved in a task.
- * It is the major region of interest in trauma treatment.

Figure 14. Brodmann Areas Involved in Emotional Control, 10-20 Sites, and Subcortical Structures Important to Emotional Regulation (Thanks to Google and Wikipedia Images)



The Default Mode Network

(with thanks to [youtube.com](https://www.youtube.com))



Brain Network Review I

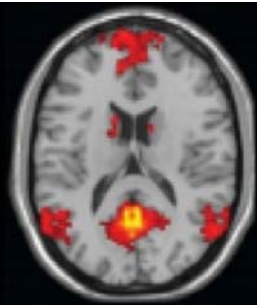
(Thanks Google Images)

The Three Primary Neural Networks

- **Default Mode Network** (medial PFC; post cingulate cortex)
- **Salience/Attention Network** (anterior insula; anterior cingulate)
- **Executive Network** (lateral PFC; lateral parietal)

Brain Networks Review II

(Thanks to Google Images)



Default Mode Network

Activates when not performing a task; daydreaming, mind-wandering, thinking about others



Sensory and limbic inputs

Salience Network

Switching between the Default Mode Network and the Central Executive Network

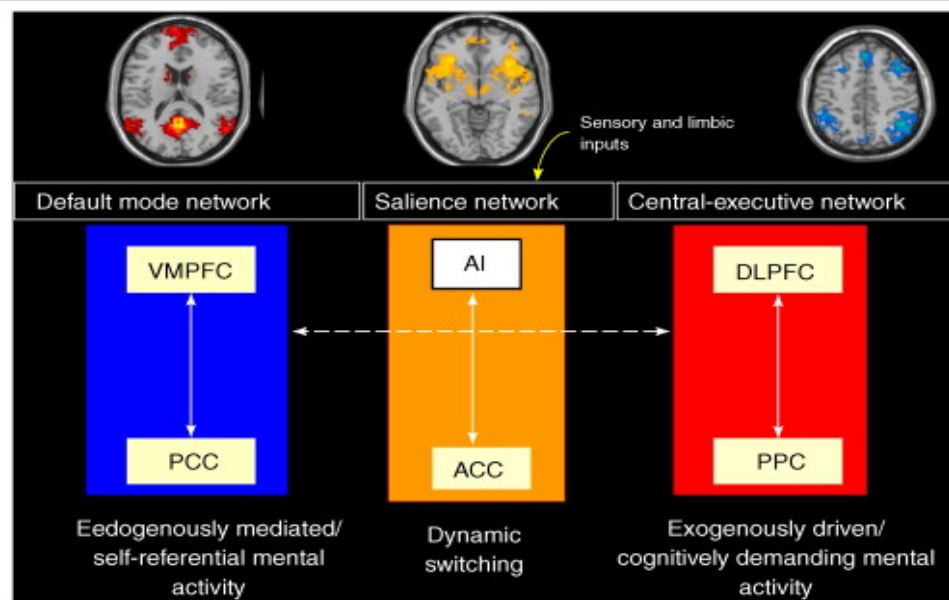


Central Executive Network

Engages your conscious brain to think and maintains attention on a prioritized task

Brain Networks Review III

(Thanks to Google Images)



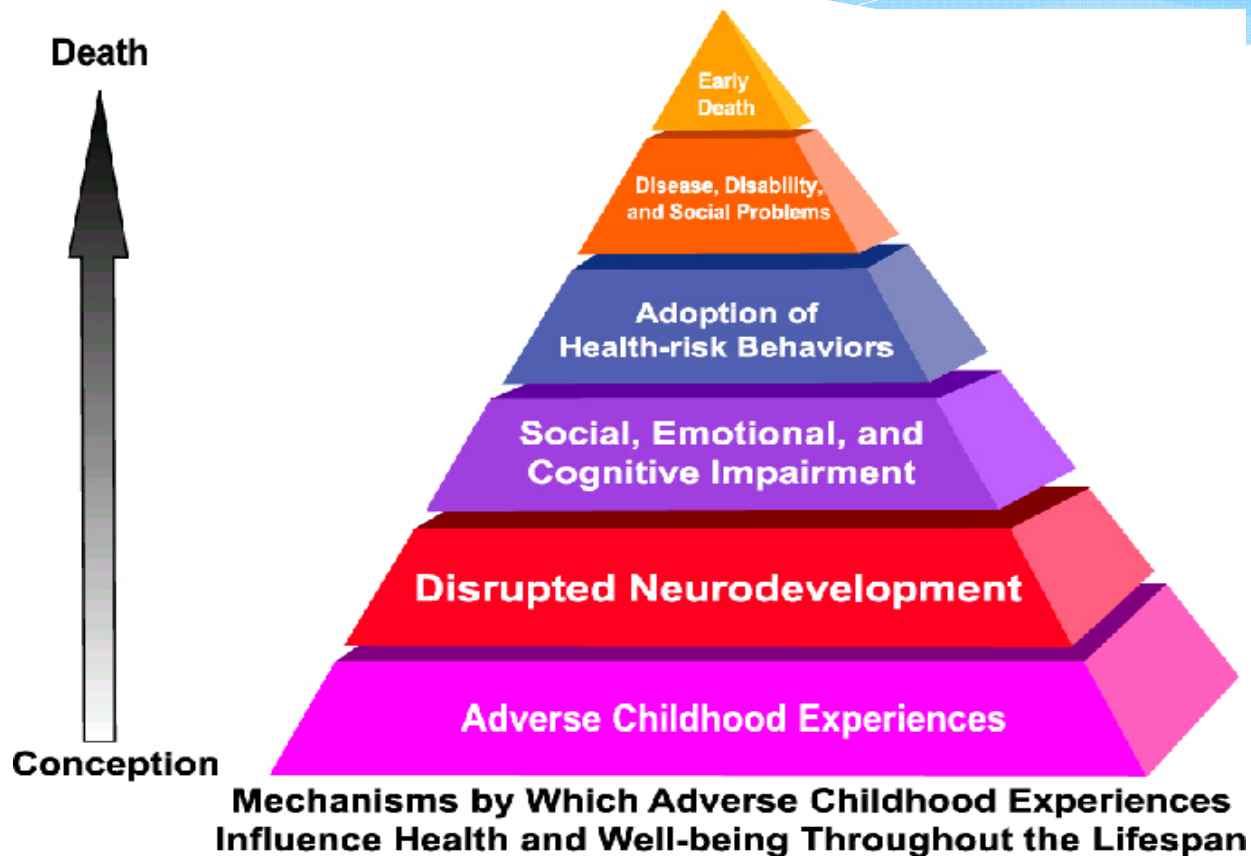


Now, back to the Jackson-Field
Study!

ACEs Scores as a Measure of Trauma

Five sources of stress or **A**dverse **C**hildhood **E**xperiences (ex. abuse, neglect, etc.) impacting personal developmental and five environmental factors (ex. Poverty, substance abuse, etc.) are considered. The scale is scored 0 to 10 with the higher scores reflecting greater severity of psychopathology and likely-hood of physical illness over the life-span (scores of 2 or higher are considered significant). There is also a scale for resilience.

Adverse Childhood Experiences (ACEs) with thanks to Google Images



Jackson-Field ACEs Scores

January 1, 2017 – June 30, 2017 (n = 33)

<u>Score</u>	<u># of Adolescents with Score</u>
1	5
2	8
3	2
4	1
5	2
6	1
7	6
8	3
9	3
10	2

mean = 5

Depression, Anxiety and PTSD

Symptom Severity

Symptom Severity - Depression

Children's Depression Inventory² (n = 43)

mean t-score = 56.9*

standard deviation = +/- 12.0

75th percentile = 67.3

*t-score of 60 or higher generally considered clinically significant

Symptom Severity – Anxiety

Manifest Anxiety Scale for Children² (n = 38)

mean t-score = 52.5*

standard deviation = +/- 25.4

75th percentile = 63.8

*a t-score of 60 or higher is generally considered clinically significant

Post-Traumatic Stress Disorder

DSM-V Criteria (n = 42)

mean raw score = 32.2

standard deviation = +/- 24.9

75th percentile = 55.5



What Do their Brains Look Like?

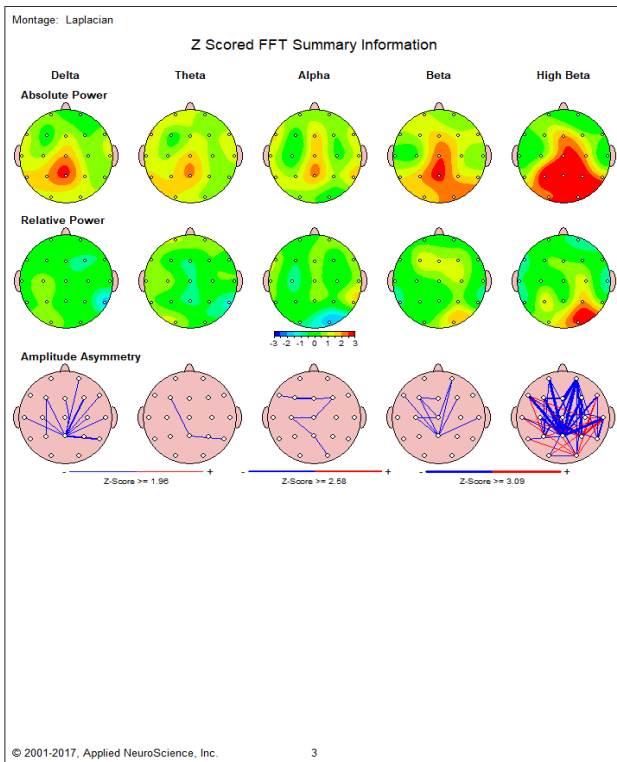
Dx RAD (age 13)

Eyes open, raw EEG (LE), cingulate is imaged, etc. collected by Freedom24

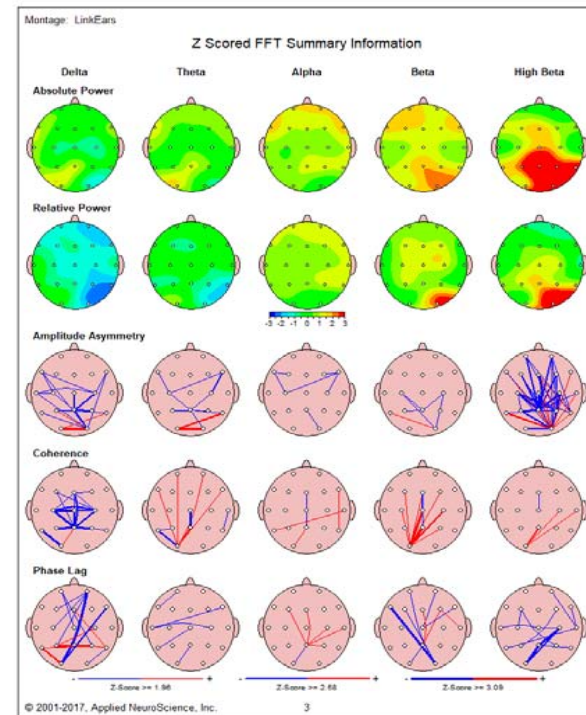


\$RUY7VHH.edf

QEEG of RAD Male (age 13)




\$RUY7VHH.edf



Dx Depression (age 13.9)



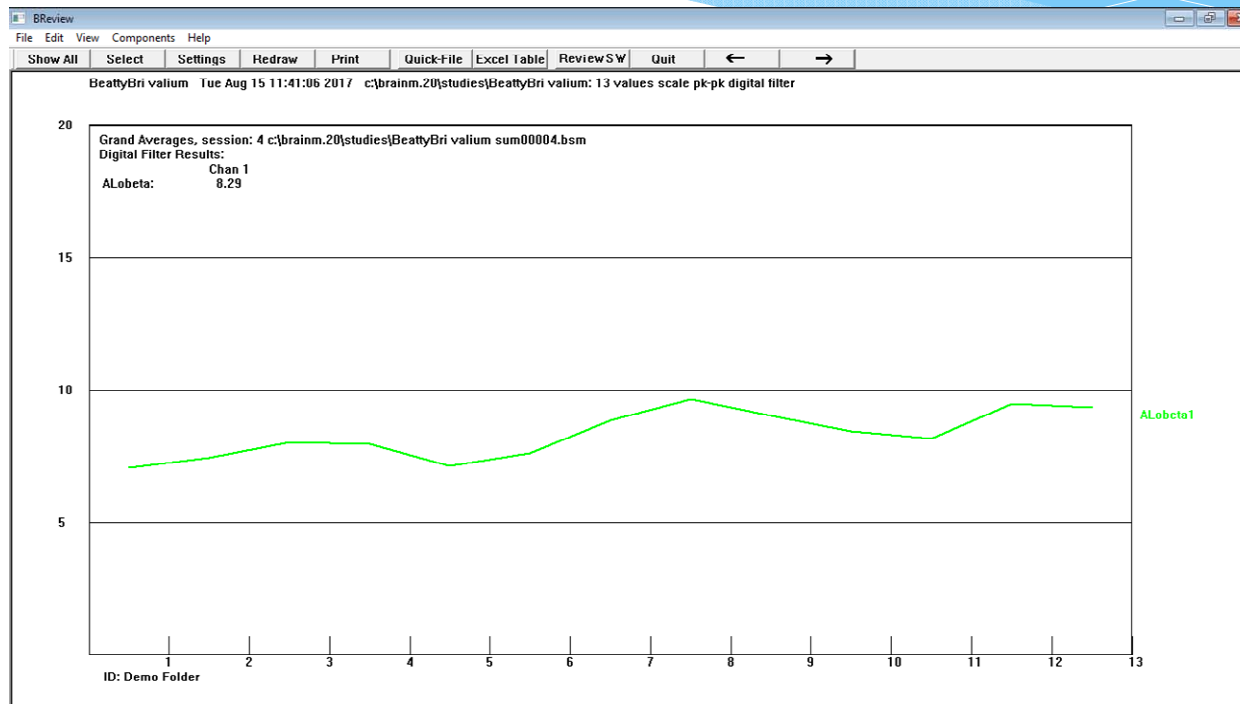
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What Does NFB Treatment Results Look Like?

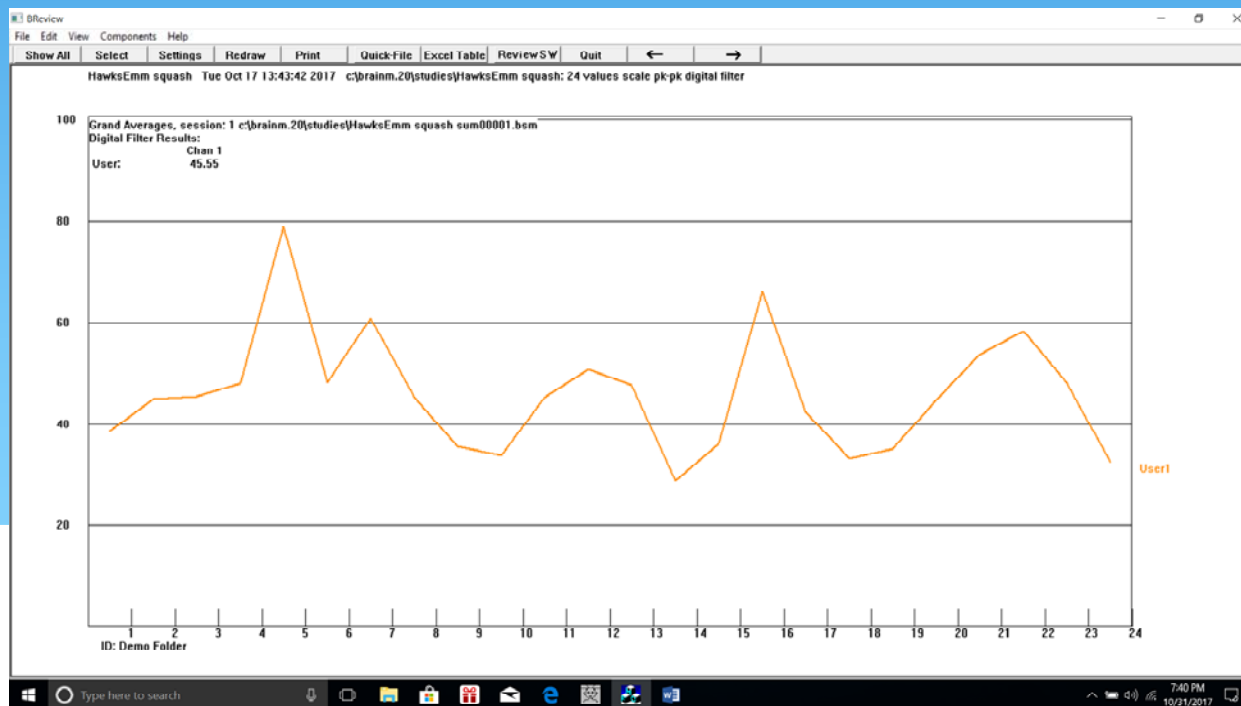
Some review screens of simple amplitude training look like what you want them too, and some don't!

DITTO for the 2-channel Z score results!

1-channel amplitude training at Fpz (8-15 reward; inhibit theta and hibeata)



1-channel Amplitude Training using 2-20 Hz Inhibit at Fpz(Squish)



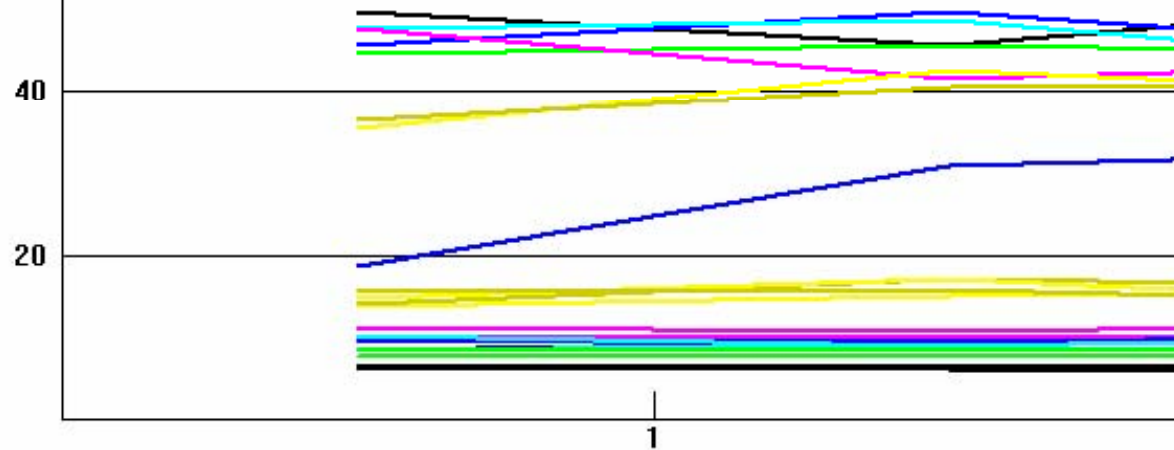
2-channel Z Score (eegPro norms) for TA () with Dx of



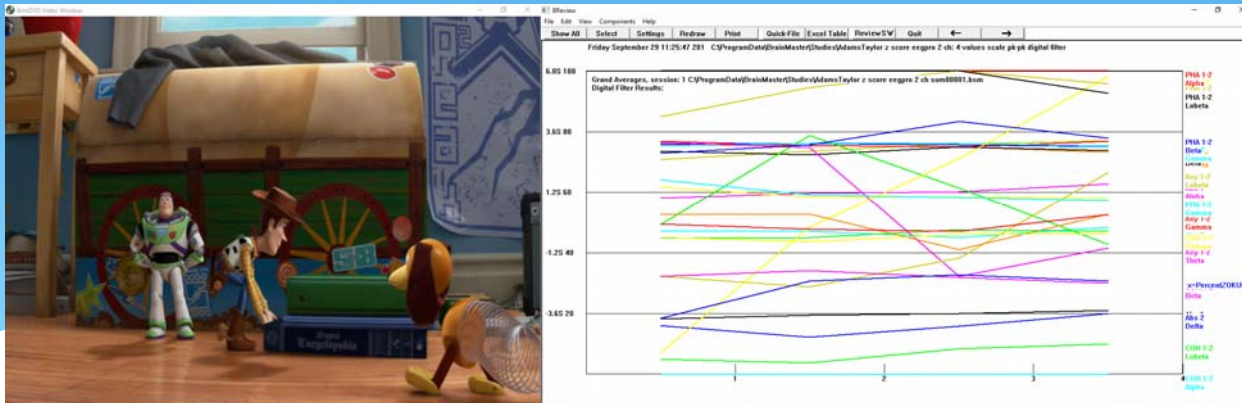
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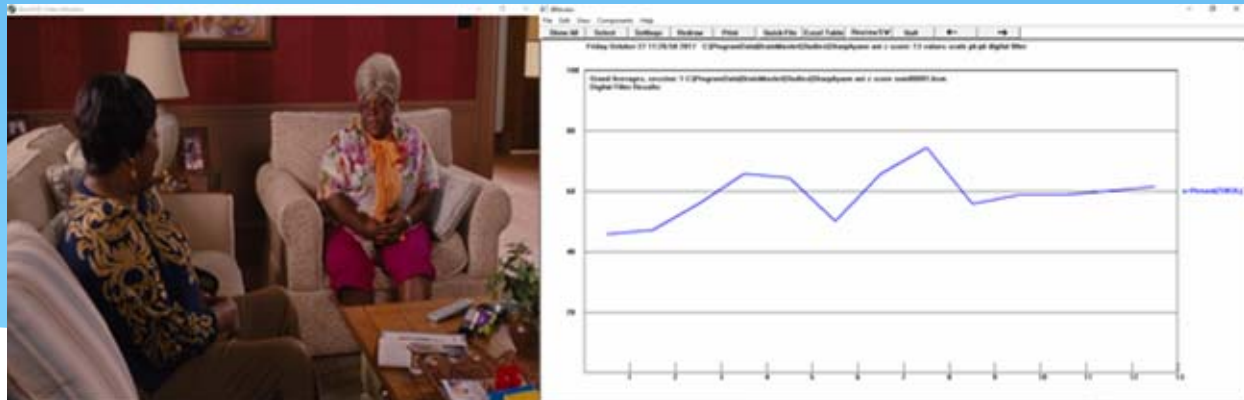
	Chan 1	Chan 2	Coher1/2
Delta:	6.07	6.67	48.36
Theta:	9.44	9.71	46.06
Alpha:	9.04	9.84	45.70
Lobeta:	7.71	8.67	44.41
Beta:	9.97	11.17	43.70
Hibeta:	14.61	15.17	38.71
Gamma:	15.64	15.10	38.71



TA



Within Session Improvement in Z-Score



QUESTION STUDIED: Can Neurofeedback Reduce Incidence Reports (IRs) in a Residential Treatment Center for Adolescents?

- * This question was suggested by recent statements/work by Bessel A. van der Kolk, M.D. and Sebern Fisher, M.A. who think neurofeedback offers an important alternative method of *treating the effects of psychological trauma.*

Dr. van der Kolk wrote in the forward of Sebern Fisher's recent book

“It amazes me that this powerful approach has been available as long as it has yet without finding widespread acceptance—it is a new frontier in helping innumerable people who up until now have been condemned to just make the best of feeling chronically fearful, unfocused and disengaged....”

Bessel A. van der Kolk, M.D.

Medical Director, The Trauma Center at JRI

*Professor of Psychiatry, Boston University School of
Medicine*

Neurofeedback in the Treatment of Developmental Trauma
by Sebern Fisher, M.A. (2014)

Review of the Literature

Neurofeedback Technology and Clinical Applications I

(selected references)

- * **Technical Foundations** (Collura, 2014; Demos, 2006; Hughes, 1994)
- * **Neuroscience** (Broyd, et al., 2009; Zotev, et al., 2013; Buckner, et al., 2008)
- * **Depression** (Hammond, 2005; Jaworska, et al., 2012; Walker & Lawson, 2013)
- * **Anxiety** (Creswell, et al., 2014; Hammond, 2005; Smith, 2013)

Neurofeedback Technology and Clinical Applications II

(selected references)

- * **ADHD, TBI** (Abbott, 2010; Arns, et al. 2009, 2015; Monastra, et al., 2012)
- * **Conduct, ODD, Anger** (Baeza, et al., 2013; Quirk, 1995; Scholte & van der Ploeg, 2006; Walker, 2013)
- * **Trauma/PTSD** (Fisher, 2014; Gerson & Rappaport, 2013; Peniston, 1993; van der Kolk (2014); Zelechowski, et al, 2013)
- * **Statistics and Research Design** (Cohen, 1992; Cook & Campbell, 1979; Green, 1991; Harris, et al., 2006; Heiman, 1992)

Methods and Materials

The bottom of the slide features a decorative graphic consisting of several overlapping, wavy lines in various shades of blue and white, creating a sense of movement and depth.

a. Experimental Design

- * A *quasi-experimental design* with the control group consisting of wait-list patients (non-randomized).
- * Patients were assigned to treatment group(s) by the clinical team as vacancies and need occurred. Diagnosis and history of acting out were considered in making assignments.
- * There were two NFB control groups (e.g., C1 and C2) and two treatment groups (e.g., T1 and T2). Groups T1 and T2 were treated by different neurotherapists using similar, but not identical techniques. T1 was made up of adolescents treated June-November, 2015 and T2 consisted of individuals treated December, 2015-May 31, 2016, some of whom were also in the T1 group.

b. Subjects

Subjects were selected for the neurofeedback program by the clinical team. Selection criteria included prior pre-admission history of acting-out, diagnosis, response to treatment, and history of acting out after admission. Assignment to the neurofeedback program occurred whenever a discharge opened a position for a new patient, usually from three days to three months after their admission.

c. Procedures

*Ten-20 paste was used to affix flat silver sensors following cleaning sites with Nu-Prep/alcohol at Fpz and ear lobes. Fpz and other frontal sites allow access to the Default Mode PFC and Salience Network.

*One-channel amplitude training (manual thresholding) was performed for 20-30 minutes two to four-times per week. Early in the study the training goal was to reduce arousal by rewarding 12-15 Hz while inhibiting 4-7 Hz and 20-30 Hz (Brainmaster “Focus” protocol) and later, the “*Valium*” protocol (Cohen, 2014) in which 8-15 Hz was rewarded while inhibiting theta and hibeta.

Treatment Rationale

Most adolescents presented with mixed symptoms of trauma-related diagnoses including depression, anxiety, PTSD, BPD, etc. A Fpz placement was used to address functions subsumed by the DFM and Salience Networks, including decision making, impulse control, sense of self, others and the world, working memory, attention, and mood stabilization.

Since we know left prefrontal hypoactivation may reflect a depressive state and right prefrontal hyperactivation may reflect anxiety (Warner, 2014, personal communication), rewarding Beta 15-18 Hz at Fp1 was often used to address depression and inhibiting Beta at Fp2 was used for anxiety (Coben, 2014).

IV. Statistical Analysis

Attempting to make sense of the data

Limitation of Poorly Executed Statistics



a. Hypothesis

- * **THE QUESTION STUDIED:**

- * Does neurofeedback reduce the number of IRs?

- * 1.a. H_0 (Null Hypothesis): NF does not reduce the number of incidence reports of adolescents in residential treatment.

- * 1.b. H_i (Alternate Hypothesis): NF reduces the number of incidence reports of adolescents in residential treatment.

b. Variables Studied

- * **Variables:**
- * **Dependent Variable** - Number of Incident Reports (IRs)/month
- * **Independent Variable** - Number of Neurofeedback Sessions/month
- * **Control Group** - the IR rate for each subject *prior* (e.g., wait-list) to the implementation of neurofeedback (3 days to 3 months).

- * **Covariables** (Possible confounds):
- * Age, Gender, Length of Treatment, and Diagnosis(es)

- * **Other Covariables** that are important but not addressed in this study include family variables, co-existing medical issues (ex. Diabetes), educational level, specific nature of trauma, race, etc.

c. Statistics Used

- * The electronic medical record system at JFBHS tallied IRs each month for each subject
- * Manual search of charts gleaned other data such as medications prescribed.
- * Descriptive statistics were performed
- * Spearman correlations (IRs/month vs NF Tx/month) were determined for age, gender, length of treatment, and diagnosis
- * t-tests of significance (non-parametric) was performed for treatment groups (T1 and T2) mean IRs/month vs mean IRs/month for control group# (IRs/month during wait-list period)
- * Cohen's d was used to determine treatment effect size.

#Rounded to nearest whole number

V. RESULTS

Does neurofeedback reduce the
number of IRs?

What does the data show?

Summary of Spearman Correlations for Covariables

Table 2

Correlation	Sample Size (n)	Correlation Coefficient	Significance Level (p = 0.05)
Age vs IRs/Month	84	-0.136	Not Significant
Gender vs IRs/Month	84	0.029	Significant
Months of Treatment vs IRs/Month	84	0.026	Significant
Diagnosis vs IRs/Month	84	-0.067	Significant

IR Means/Month of Group T1 and T2 Compared to the Control?

Treatment Group 1: Mean IRs = 3.8 ± 3.7

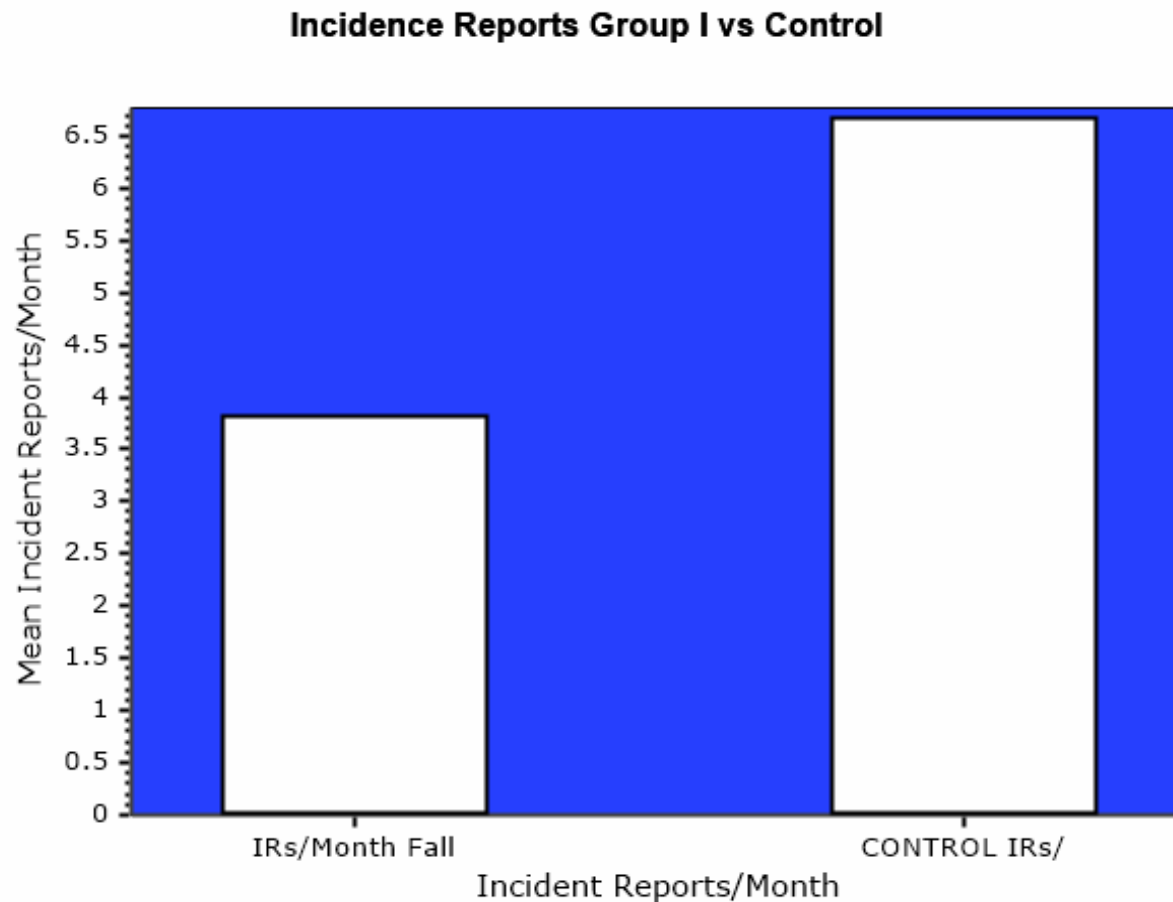
Control Group 1: Mean IRs = 6.7 ± 9.9

Treatment Group 2: Mean IRs = 3.8 ± 3.0

Control Group 2: Mean IRs = 6.7 ± 10.6

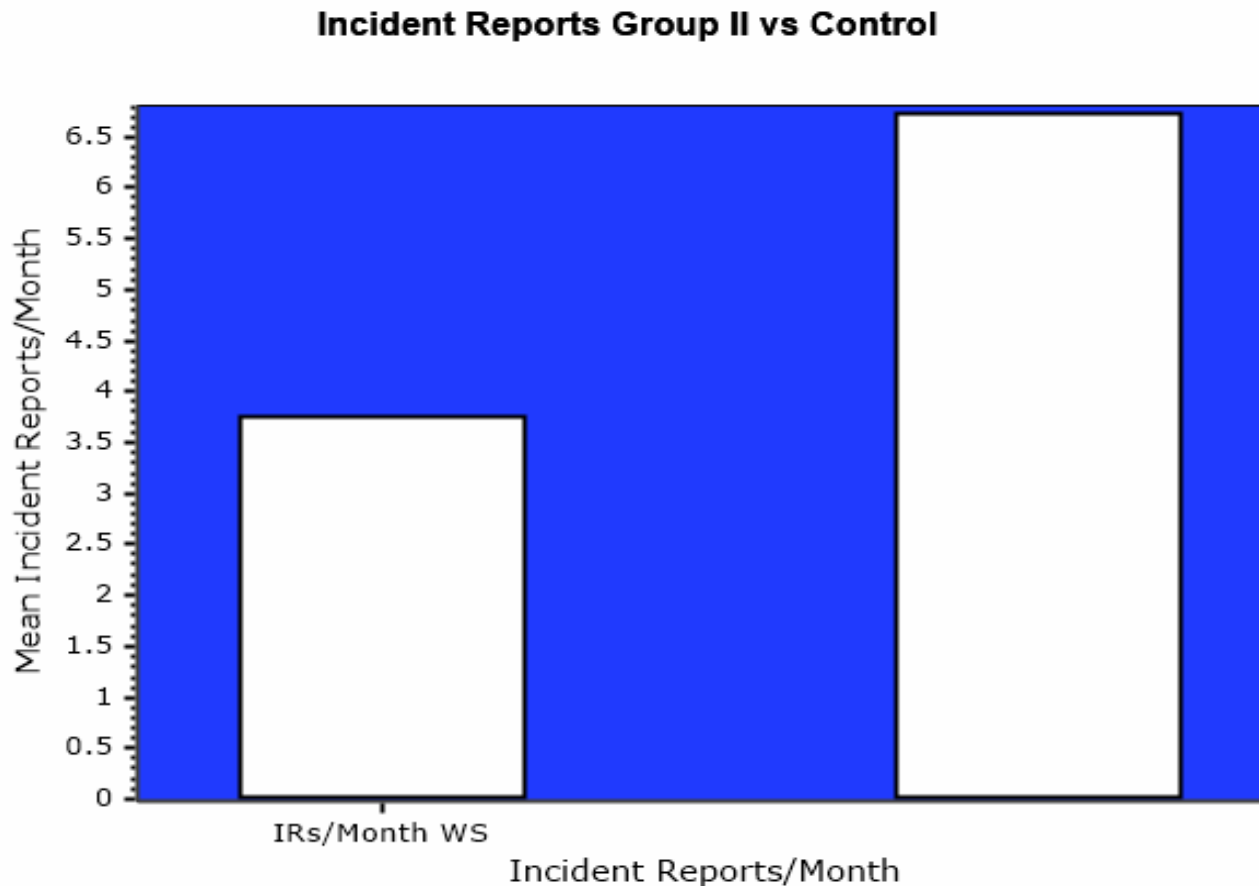
Comparison of Mean Incident Reports for Group 1 (n=61)

FIGURE 6. Group 1 (2 tx/month) vs Control 1



Comparison of Mean Incident Reports for Group 2 (n=56)

FIGURE 7. Group 2 (4 tx/month) vs (control 2 tx/month)



IRs Compared to Control

TABLE 3. Analysis of difference

Group T1 vs Control:

N = 61
T = 2.534
t, critical = 2.000
p value = 0.0139

Group T2 vs Control:

N = 56
T = 2.041
t, critical = 1.977
p value = 0.0432

(unpaired t-test of difference between means of control groups vs treatment groups)

Effect Size

(Cohen, 1992)

TABLE 4: Effect sizes compared

* Group T1:

* Cohen's $d = -0.3664$

* effect size $r = -0.1802$

* Interpretation:

* **Medium Effect Size**

* Group T2:

* Cohen's $d = -0.381$

* effect size $r = -0.1872$

* Interpretation:

* **Medium Effect Size**

VI. Discussion and Conclusion

Discussion (1)

Treatment team meeting members were asked what they had observed in the treated groups. Team members cited behavioral changes such as....

- *increased self-awareness,*
- *better impulse control,*
- *reduced hyperactivity,*
- *improved attention,*
- *reduced number of IR's.*

Reduction in IR's should make the management of the young people studied easier and less stressful for the adolescent patients, cottage staff and school personnel. No abreactions were reported.

Discussion (2)

- * In the JF study there was a statistically significant ($p < 0.05$) difference (reduction) in IRs in T1 ($p = 0.0139$) and T2 ($p = 0.0432$) compared to wait-list controls (C1 and C2).
- * The difference in IRs between T1 and T2 was fairly small. Group 2, which received neurofeedback an average of 4 times per month, had 16% fewer IRs than Group 1 which received neurofeedback 2 times per month.
- * Was the greater “dose” of neurofeedback responsible for this difference or was it just due to chance variation? Further study may answer this question.

Discussion (3)

von Hilsheimer (2006), reported D. A. Quirk found SMR neurofeedback significantly reduced recidivism in a population of criminals. He also reported that this reduction was a function of the number of neurofeedback sessions. Those treated 16 sessions were more often jailed again than those receiving 32 sessions. There also appeared to be some benefit for those who only received a few sessions. Smith and Sams (2005) reported similar benefit of neurofeedback for juvenile offenders.

Discussion (4)

In the current study adolescents who received an average of four neurofeedback sessions per month had 16% fewer IRs than those receiving an average of two sessions per month. There is general agreement among neurofeedback researchers and practitioners that properly applied neurofeedback in out-patient settings reduces the symptoms/behaviors of disorders such as ADHD, ODD, Conduct Disorder, depression, anxiety, PTSD, and TBI.

Discussion (5)

However, with the exception of the study by Breteler, et al. (2012) which addressed neurofeedback treatment of intellectually challenged youths in residential settings, similar research in the use of neurofeedback in other residential treatment populations, is scant. Use of medication is ubiquitous in adolescent units and use of isolation and restraint is not un-common (Baeza et al., 2012). These authors point-out how badly alternative methods of treating these individuals is needed. The current study offers a step toward addressing this need.

Conclusion

- * The groups treated with neurofeedback demonstrated a statistically significant reduction in IRs compared to the control groups.
- * The findings of this study demonstrated an average 57% reduction in IRs in two treatment groups. Neurofeedback thus appears to be an efficacious and safe treatment to reduce problematic behaviors in adolescent residential treatment facilities.

Section VII: Trauma Treatment

NFB Protocols that have been
found helpful by others.

Helpful Interventions in Trauma Treatment

1. Raising or lowering CNS arousal depending upon chief symptoms.
2. Optimizing coherence, phase and asymmetry.
3. Improving regulation of blood/oxygen supply.
4. HRV style breathing.
5. Psychotherapeutic methods including EMDR.

Name	Settings/Sites	References
FpO2	Reward 2-8; inhibit hibeta; FpO2	Fisher (2014)
Valium Protocol	Reward 8-12 or 8- 15 Hz; inhibit theta and hibeta; Fpz	Cohen (2014), Griffin (2016)
Alpha/Theta	Reward alpha and theta; P4, Pz, C4	Peniston (1993)
Infraslow Fluctuation	Generally 0.0020- 0.0060 Hz; various placements	Smith (2017)
pIR Hemoencephalo- graphy (pIR HEG)	Fpz	Coben (2006)

Table 5a. Neurofeedback
Protocols

Name	Settings/Placement	References
Interhemispheric Instability	Reward 9-12 Hz; Inhibit 0-6 Hz and 22-36 Hz; T3-T4 bipolar montage	Fisher (2014); Othmers (2001, 2006, 2009)
Beta Reset	Reward 16-19 Hz briefly, increase gradually to 38-42 Hz and then work down to 16-19 Hz	Teicher, et al., as cited by Fisher (2014)
Alpha Down	Inhibit 8-12 at Pz	Lanius and Ros, as cited by Fisher (2014)
Squash	Inhibit 2-20 Hz; Cz and various placements adjusted as needed	Collura (2014)

Table 5b. Neurofeedback Protocols

Future Directions

- * Replication studies are needed.
- * Impact of demographic and treatment co-variables needs to be examined.
- * Evaluation of efficacy of other neurofeedback techniques (e.g 4-channel Z-score, 19-channel surface Z-score, 19-channel sLoreta Z-score, magnetic stimulation augmented Z-score neurofeedback, Infracore Neurofeedback, etc.).
- * Exploration of which changes in EEG (e.g. power, coherence, etc.) predict specific improvement in behavior/symptom reduction.
- * Examination of the relationship between number of sessions and outcome for different Dx groups.



References and Literature Cited
available on SBCNA Website

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*Thank you for attending this
presentation*

