Genetic Findings in PTSD: Along the Road to Discovery

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Disclosures

- None
Epidemiology of PTSD

• Estimated that upwards of 56% of Americans experience a lifetime trauma
• Lifetime prevalence of 8% in the general population
• Female prevalence (10.4%) twice as likely as men (5.0%) to have PTSD
• Soldiers serving in support of OEF/OIF report PTSD rates as high as 18% post-deployment (90% of active duty are male)
• Twin studies estimate 32-35% of the variance in PTSD due to genetic influences
Risk Factors for PTSD

- Pre-existing emotional disorder (e.g. depression)
- Drug or alcohol use
- History of sexual/physical abuse and/or childhood neglect
- Insomnia and/or excessive daytime sleepiness within the month following traumatic event
- Family history of anxiety disorders
Common Pathway Model for Five Anxiety Disorders

Tambs K et al. BJP 2009;195:301-307
Common Pathway Model for Five Anxiety Disorders

Tambs K et al. BJP 2009;195:301-307
Common Pathway Model for Five Anxiety Disorders

Tambs K et al. BJP 2009;195:301-307
The Search for Genes Associated with PTSD
Fear Neurocircuitry

Skelton et al., Neuropharmacology 2012
Fear Neurocircuitry

Skelton et al., Neuropharmacology 2012
# Candidate Genes for PTSD – HPA Axis

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Trauma</th>
<th>Gene (symbol)</th>
<th>GxE</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bachman</td>
<td>2005</td>
<td>Combat</td>
<td>GCCR</td>
<td>N/A</td>
<td>Negative</td>
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<tr>
<td>Amstadter</td>
<td>2011</td>
<td>Injury</td>
<td>CRHR1</td>
<td>Trauma</td>
<td>GxE for PTSD</td>
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<tr>
<td>Binder</td>
<td>2008</td>
<td>Various</td>
<td>FKBP5</td>
<td>Childhood abuse</td>
<td>GxE</td>
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<tr>
<td>Xie</td>
<td>2010</td>
<td>Childhood adversity</td>
<td>FKBP5</td>
<td>Childhood adversity</td>
<td>GxE</td>
</tr>
</tbody>
</table>
CRHR1 rs12944712 Associated with PTSD in Pediatric Sample

Amstadter et al., 2011
Interaction of FKBP5 and Childhood Abuse for PTSD

Binder et al., 2008
Replication of GxE FKBP5 Finding - rs9470080

Xie et al., 2010
Fear Neurocircuitry

Skelton et al., Neuropharmacology 2012
## Candidate Genes for PTSD – Locus Coeruleus-Noradrenergic System

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Trauma</th>
<th>Gene (symbol)</th>
<th>GxE</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lappalainen</td>
<td>2002</td>
<td>Combat</td>
<td>NPY</td>
<td>N/A</td>
<td>Negative</td>
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<tr>
<td>Mustapic</td>
<td>2007</td>
<td>Combat</td>
<td>DBH</td>
<td>N/A</td>
<td>Negative</td>
</tr>
<tr>
<td>Kolassa</td>
<td>2010</td>
<td>Rwandan genocide/various</td>
<td>COMT</td>
<td>GxE?</td>
<td></td>
</tr>
</tbody>
</table>
Interaction between COMT and Trauma Load

Kolassa et al., 2010
Fear Neurocircuitry

Skelton et al., Neuropharmacology 2012
**Candidate Genes for PTSD – Limbic-Frontal System**

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Trauma</th>
<th>Gene (symbol)</th>
<th>GxE</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voisey</td>
<td>2008</td>
<td>Combat</td>
<td>DRD2</td>
<td>N/A</td>
<td>Excess C allele in PTSD</td>
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<tr>
<td>Segman</td>
<td>2002</td>
<td>Various</td>
<td>DAT1</td>
<td>N/A</td>
<td>Excess 9-repeat in PTSD</td>
</tr>
<tr>
<td>Kilpatrick</td>
<td>2007</td>
<td>Hurricane/ various</td>
<td>SLC6A4</td>
<td>Stress in county</td>
<td>GxE</td>
</tr>
<tr>
<td>Nelson</td>
<td>2009</td>
<td>Various</td>
<td>GABRA2</td>
<td>Childhood trauma</td>
<td>GxE</td>
</tr>
<tr>
<td>Zhang</td>
<td>2006</td>
<td>Not specified</td>
<td>BDNF</td>
<td>N/A</td>
<td>Negative</td>
</tr>
</tbody>
</table>
Interaction between SLC6A4 and Trauma

Kilpatrick et al., 2007
Pituitary adenylate cyclase-activating polypeptide (PACAP)

- The neuropeptide PACAP is an informational molecule released from stress-transducing neurons.
- It exerts post-synaptic effects required to complete hypothalamo-pituitary-adrenocortical (HPA) and hypothalamo-splanchnico-adrenomedullary (HSA) circuits activated by psychogenic and metabolic stressors.
- Has been called the “master regulator” of the stress response.
- And has been linked with PTSD!
PACAP Blood Levels Predict PTSD Symptoms in Females

Ressler et al., 2011
Genetic association of PAC1 receptor with PTSD

Ressler et al., 2011
Association of PAC1 rs2267735 with PTSD Symptoms

Ressler et al., 2011
Future Genomics
Recent Gene Discovery Studies - GWAS

• Only two published genome-wide association studies published to date
• White non-Hispanic sample 295 PTSD cases and 196 controls (Logue et al., 2012)
• Genome-wide association based on Illumina microarray (2.5 million SNPs)
Retinoid-Related Orphan Receptor Alpha (RORA) Gene

Logue et al., 2012
Possible RORA Link with PTSD Risk

- RORA SNPs previously linked with depression
- Protein encoded by RORA involved in brain development, neuroprotection and the regulation of circadian rhythms and steroid hormones
- It protects cortical neurons against oxidative stress-induced apoptosis by increasing the expression of the antioxidant proteins
- In glial cells, its expression is upregulated by the presence of destructive pro-inflammatory cytokines, and during hypoxia, its expression is upregulated in neurons and astrocytes

Terracciano et al., 2010
Recent Gene Discovery Studies - GWAS

- Only two published genome-wide association studies published to date
- White non-Hispanic sample \(295\) PTSD cases and \(196\) controls (Logue et al., 2012)
- Genome-wide association based on Illumina microarray (2.5 million SNPs)
- 1,578 European Americans (\(300\) PTSD) and 2,766 African-Americans (\(444\) PTSD)
- Genome-wide association based on Illumina microarray (870,000 SNPs)
Tolloid-Like 1 Gene (TLL1)

Xie et al., 2013
Possible TLL1 Link with PTSD Risk

- TLL1 protein is expressed at high levels in the granular layer of the cerebellum of adult mice and in the hippocampus of both juvenile and adult mice.
- Functional studies suggest that glucocorticoids, which regulate stress response in mammals, could decrease mTLL1 expression in vitro.
- Elevated number of hippocampal neurons expressing mTLL1 found in mice with increased neurogenesis.
- Reptilian orthology of TLL1 isolated from turtle showed that the gene cleaves the precursor pro-brain-derived neurotrophic factor (proBDNF) into mature BDNF.

Xie et al., 2013
Emerging Gene Discovery Studies - GWAS

- Psychiatric Genetics Consortium (PGC) PTSD working group (Koenen et al.)
- CSP 575B, Genomics of Posttraumatic Stress Disorder among Veterans including 10,000 combat-exposed Veterans with PTSD as cases and 10,000 combat-exposed Veterans without PTSD as controls (Stein & Gelernter)
- Army STARRS examining factors that help protect a Soldier’s mental health and factors that put a Soldier’s mental health at risk (Stein & Ursano)
- STRONG STAR Multi-Disciplinary PTSD Research Consortium including epidemiologic, treatment, and pre-clinical studies (Peterson)
STRONG STAR

Clinical Trials

- Cognitive Processing Therapy for Combat-Related PTSD (PI: Patricia Resick, PhD)
- Prolonged Exposure for PTSD among OIF/OEF/OND Personnel: Massed vs. Spaced Trials (PI: Edna Foa, PhD)
- Individual PE vs. Couples' Cognitive-Behavioral Therapy for Combat-Related PTSD (PI: Candice Monson, PhD)
- SSRI Treatment of Dual Diagnosis PTSD and Alcohol Dependence: A Test of the Serotonergic Hypothesis (PI: John Roache, PhD)
- Brief Cognitive Behavioral Treatment of Deployment-Related PTSD in Primary Care Settings: A Randomized Controlled Trial (PI: Jeffrey Cigrang, PhD)

http://strongstar.org
STRONG STAR
Pre-Clinical Studies

• Mechanisms of Vulnerability to PTSD: The Role of Early Life Stressors (PI: Randy Strong, PhD)

http://strongstar.org
Fear Response to PNS/CAPS Exposure

Green et al., 2011 - Neuroscience
Combined PNS/CAPS Treatment Impaired Extinction

Green et al., 2011 - Neuroscience
Tyrosine Hydroxylase mRNA Expression in the Locus Coeruleus

Green et al., 2011 - Neuroscience
Effects of PNS/CAPS on Glucocorticoid Receptor Protein

Green et al., 2011 - Neuroscience
STRONG STAR

Biological Studies

- Neuroimaging Studies of PTSD and PTSD Treatment among Combat Veterans (PI: Peter Fox, MD)
- Genetic and Environmental Predictors of Combat-Related PTSD (PI: Douglas Williamson, PhD)

http://strongstar.org
Genetic and Environmental Predictors of Combat-Related PTSD

- Identify changes in gene expression (mRNA) associated with onset of PTSD symptoms/diagnosis and identify genetic DNA variants among the genes identified
- Screen 4,000+ soldiers pre-deployment including self-report assessments and blood collection
- Estimate recapture of 3,600 available post-deployment
- Reassess 1,440 soldiers post-deployment half (n=720) with significant PTSD symptoms (~20%) and/or diagnosis
Biologic Sample Collection

- PAXgene™ Blood RNA 8.5mL Tube – rapid in-tube lysing and storable at room temperature up to 5 days
- PAXgene™ Blood DNA System 8.5mL Tube – provides buffering conditions optimized for subsequent cell lysis and DNA purification. Whole blood DNA stored in PAXgene DNA Tubes is stable for 14 days at room temperature
- ACD Yellow Blood Collection 8.5mL Tube – contains acid citrate dextrose (ACD) for isolating plasma. Laboratory processing within one hour of collection

Genetic and Environmental Predictors of Combat-Related PTSD
Whole Genome Microarrays

- Illumina HumanHT-12 v3 mRNA Expression BeadChip - (genome-wide transcriptional coverage of well-characterized genes, gene candidates, and splice variants > 48,000 probes) (pre-/post-deployment)
- Illumina PsychArray DNA Analysis BeadChip - (~700,000 SNPs evenly spaced whole-genome markers and imputable to several million) (pre-deployment)

Genetic and Environmental Predictors of Combat-Related PTSD
Study Implemented at Ft. Hood, TX
• 4,112 Soldiers recruited pre-deployment
• As of January 26, 2015 – ~2,000 Soldiers followed post-deployment
• Post-deployment interviews expected to be complete Summer 2015
• Whole-genome mRNA and DNA micro-arrays planned Summer/Fall 2015
• Currently finalizing the symptom trajectory analyses examining PTSD symptoms around redeployment to US
Genetic and Environmental Predictors of Combat-Related PTSD
Genetics of PTSD in Human Postmortem Samples
Pilot Human Postmortem Study of PTSD

- Methyl-CpG Binding Domain-Based Capture Sequencing (MBDCap-Seq) of CpG sites in human postmortem tissue
- Included n=5 PTSD cases and n=5 controls
- Examined the posterior cingulate cortex (pCC – BA23) based on our fMRI meta-analytic results showing increased BOLD during script imagery task (Ramage et al., 2012)
- Included medial orbital frontal cortex (mOFC – BA11) due to its involvement in fear extinction deficits (e.g. Milad et al., 2005)
Results of the MBDCap-Seq

Post-mortem tissue PTSD (n=5) and controls (n=5)

Data from +/- 1000bp around transcription start site

1) Posterior Cingulate Cortex (pCC) – p<0.01
   5 sites hyper-methylated,
   16 sites hypo-methylated

2) Medial Orbitofrontal Cortex (mOFC) – p<0.01
   344 sites hyper-methylated
   11 sites hypo-methylated
Expanded Human Postmortem Study of PTSD

- Medial orbital frontal cortex (BA11)
- PTSD (n=18) and Controls (n=40) (postmortem tissue from the SWBB and LIBD)
- PTSD and control samples matched for age and gender
- Postmortem interval slightly lower in PTSD (P < .023) and covaried in all analyses
- Examined DNA methylation and mRNA levels in mOFC
DNA Methylation Microarray

- Infinium HumanMethylation450 BeadChip
- Data analyzed in R - Illumina Methylation Analyzer (IMA)
- 372,389 CpG sites remained after QC processing (detection < .01 and known SNPs at CpG sites removed ~65k)
- Quantile normalization and peak corrected
- FDR adjusted p-values
Gene Expression Microarray

- Illumina HumanHT-12 v4 Expression BeadChip (~47k transcripts and known splice variants)
- Data analyzed in R - Linear Models for Microarray Data (LIMMA)
- 22,622 genes remained after QC processing (detection < .01)
- Background correction using negative controls
- Quantile normalized and log transformed
- FDR adjusted p-values
Genomic and Neighborhood Locations of CpG Sites

Total = 372,389

TSS1500: 24.3%
TSS200: 14.6%
5'UTR: 10.7%
1STEXON: 9.0%
Body: 4.7%

Total = 372,389

Islands: 35.7%
Shores: 31.0%
Shelves: 23.7%
Others/Open Sea: 9.6%
Manhattan Plot of CpG Sites Stratified by Genomic Region
## Genome-wide Significant CpG Sites

<table>
<thead>
<tr>
<th>CpG Site</th>
<th>Gene</th>
<th>Location</th>
<th>Neighborhood</th>
<th>P-Value</th>
<th>LOD Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>cg16866257</td>
<td>FTSJ3</td>
<td>TSS200</td>
<td>Island</td>
<td>2.36E-07</td>
<td>6.63</td>
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<tr>
<td>cg09898154</td>
<td>CYB5RL</td>
<td>TSS200</td>
<td>Shore</td>
<td>3.00E-07</td>
<td>6.52</td>
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<tr>
<td>cg15874062</td>
<td>INPP4B</td>
<td>5’UTR</td>
<td>Island</td>
<td>4.58E-07</td>
<td>6.34</td>
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<tr>
<td>cg20485669</td>
<td>SLCO3A1</td>
<td>Body</td>
<td>Island</td>
<td>9.23E-07</td>
<td>6.03</td>
</tr>
<tr>
<td>cg01366085</td>
<td>KLF10</td>
<td>TSS1500</td>
<td>Island</td>
<td>1.01E-06</td>
<td>6.00</td>
</tr>
</tbody>
</table>
FTSJ3
FtsJ Homolog 3 (E. Coli)
Further Characterization of CpG Sites in FTSJ3

• 14 CpG sites assayed on the Infinium HumanMethylation450 BeadChip
• 4 of the 14 significantly hypermethylated in PTSD
• Current function of this gene unknown
• Probable methyltransferase involved in methylating substrates including DNA methylation
Heatmap of Genes Expressed in mOFC in PTSD and Controls
300 genes (out of 22,622) differentially expressed

All genes P < 0.05 after FDR correction
# IPA Network Analysis of Genes

<table>
<thead>
<tr>
<th>ID</th>
<th>Molecules in Network</th>
<th>Score</th>
<th>Focus Molecules</th>
<th>Top Diseases and Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26s, Proteasome, APOM, ARCN1, CCDC59, CCL5, CCL21, DDAH2, DPM1, EDA2R, EHHADH, HISTONE, Histone h3, Histone h4, IKBKAP, Insulin, KCNA1, KLF15, KLHDC10, LPCAT3, MITD1, MRPS9, <strong>NFkB complex</strong>, PIN1, POLR2B, Proinsulin, RNA polymerase II, SIGIRR, SNIP1, SRSF11, TAF1L, TMEM14C, TNFRSF10C, TSC22D1, USPL1, WHSC1L1</td>
<td>51</td>
<td>27</td>
<td>Cellular Assembly and Organization, Infectious Disease, Developmental Disorder</td>
</tr>
<tr>
<td>2</td>
<td>ACTA1, ACVR1C, <strong>Ampa Receptor</strong>, ATP1A1, caspase, CHGA, Collagen Alpha1, ERK1/2, FLCN, FXN, GRIA3, GRIA4, MEK, MTMR6, NXN, P38 MAPK, phosphatase, PLA2G7, PP2A, PPP2CA, PSMD6, PTPase, PTPN2, PTPN7, PTPRK, RANBP2, RAPGEF1, Rock, SRC (family), TAOK2, TRPC3, T ceilial protein R1, UBA2, VPS39, ZHX2</td>
<td>45</td>
<td>24</td>
<td>Post-Translational Modification, Cellular Assembly and Organization, Cellular Function and Maintenance</td>
</tr>
<tr>
<td>3</td>
<td>AIRE, Akt, Ap1 gamma, AP1G1, APOL2, BAG5, BCR (complex), CD79A, CIDEA, CIDE-C, CLTC, Creb , Cyclin A, EIF4EBP2, ETS2, F Actin, HMGA2, IFN Beta, IgG, IgM, IL10RA, IL12 (complex), IL12 (family), Immunoglobulin, Interferon alpha, LDL, LRRK2, mir-8, PCSK1, POLQ, PPP1R13L, RABEP1, SEC14L3, Tgf beta, VAMP1</td>
<td>35</td>
<td>20</td>
<td>Cell Morphology, Hematological System Development and Function, Inflammatory Response</td>
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Network 1
# Genes in NFkB Complex

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
<th>Direction</th>
</tr>
</thead>
<tbody>
<tr>
<td>TMEM14C</td>
<td>Transmembrane Protein 14C</td>
<td>↓</td>
</tr>
<tr>
<td>TNFRSF10C</td>
<td>Tumor Necrosis Factor Receptor Superfamily, Member 10c, Decoy Without An Intracellular Domain</td>
<td>↑</td>
</tr>
<tr>
<td>USPL1</td>
<td>Ubiquitin Specific Peptidase Like 1</td>
<td>↓</td>
</tr>
<tr>
<td>PIN1</td>
<td>Peptidylprolyl Cis/Trans Isomerase, NIMA-Interacting 1</td>
<td>↓</td>
</tr>
<tr>
<td>SIGIRR</td>
<td>Single Immunoglobulin And Toll-Interleukin 1 Receptor (TIR) Domain</td>
<td>↑</td>
</tr>
<tr>
<td>SNIP1</td>
<td>Smad Nuclear Interacting Protein 1</td>
<td>↓</td>
</tr>
<tr>
<td>CCL5</td>
<td>Chemokine (C-C Motif) Ligand 5</td>
<td>↑</td>
</tr>
<tr>
<td>EDA2R</td>
<td>Ectodysplasin A2 Receptor</td>
<td>↑</td>
</tr>
<tr>
<td>IKBKAP</td>
<td>Inhibitor Of Kappa Light Polypeptide Gene Enhancer In B-Cells, Kinase Complex-Associated Protein</td>
<td>↓</td>
</tr>
</tbody>
</table>
Genes in NFkB Complex

---

**TMEM14C**

- Control
- PTSD

Log(AVG Signal mRNA) vs. Control and PTSD

- P < 0.0003

**TNFRSF10C**

- Control
- PTSD

Log(AVG Signal mRNA) vs. Control and PTSD

- P < 0.0003

**USPL1**

- Control
- PTSD

Log(AVG Signal mRNA) vs. Control and PTSD

- P < 0.0004

---

**cg10011792**

Body

- Methylation (%) vs. log(AVG Signal mRNA)

- r = -0.03
- P < 0.00021

**cg03749393**

TSS200

- Methylation (%) vs. log(AVG Signal mRNA)

- r = -0.03
- P < 0.8411

---

**cg10011792**

Body

- Methylation (%) vs. log(AVG Signal mRNA)

- r = 0.05
- P < 0.6926

---
Genes in NFkB Complex

PIN1

P < 0.0006

SiGIRR

P < 0.0007

SNIP1

P < 0.0018

cg17163138
Body

r = 0.07
P < 0.5926

cg08869273
5'UTR

r = 0.34
P < 0.0093

cg20768399
TSS1500

r = -0.03
P < 0.8251
# Genes in AMPA Receptor

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
<th>Direction</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP1A1</td>
<td>ATPase, Na+/K+ Transporting, Alpha 1 Polypeptide</td>
<td>↓</td>
</tr>
<tr>
<td>GRIA3</td>
<td>Glutamate Receptor, Ionotopic, AMPA 3</td>
<td>↓</td>
</tr>
<tr>
<td>GRIA4</td>
<td>Glutamate Receptor, Ionotopic, AMPA 4</td>
<td>↓</td>
</tr>
</tbody>
</table>
Genes in AMPA Receptor

- **GRIA3**
  - Log(AVG Signal mRNA)
  - Control: 4, 5, 6, 7, 8
  - PTSD: 4, 5, 6, 7, 8
  - $P < 0.0012$

- **GRIA4**
  - Log(AVG Signal mRNA)
  - Control: 4, 5, 6, 7, 8
  - PTSD: 4, 5, 6, 7, 8
  - $P < 0.0049$

- **ATP1A1**
  - Log(AVG Signal mRNA)
  - Control: 9, 10, 11, 12, 13
  - PTSD: 9, 10, 11, 12, 13
  - $P < 0.0009$

- **cg01854931**
  - Methylation (%)
  - $r = -0.29$
  - $P < 0.0281$

- **cg21719418**
  - Methylation (%)
  - $r = -0.01$
  - $P < 0.9144$

- **cg25346117**
  - Methylation (%)
  - $r = -0.32$
  - $P < 0.0159$
Combined PNS/CAPS Treatment Impaired Extinction

Green et al., 2011 - Neuroscience
mRNA Levels of Network 1 Genes in OFC of Traumatized Rats

Not Fear Extinguished

Chemokine ligand 5: p = .01

Smith et al., SFN 2014
mRNA Levels of Network 1 Genes in OFC of Traumatized Rats

Smith et al., SFN 2014
Summary of Human Postmortem Study

- mOFC appears to be uniquely methylated in PTSD
- Methylated CpG sites in several genes unique to PTSD
Spine Density and Spine Morphology in mOFC of PTSD Brains

Decreased spine density on dendrites in BA11 in PTSD

<table>
<thead>
<tr>
<th></th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD</td>
<td>&lt; .07</td>
</tr>
<tr>
<td>Age</td>
<td>&lt; .05</td>
</tr>
<tr>
<td>Gender</td>
<td>&lt; .11</td>
</tr>
</tbody>
</table>
High FKBP5 mRNA levels correlate with reduced spine density

FKBP5 Gene Expression levels in BA11 were inversely correlated to mushroom spine density (Spearman’s rho = 0.83, p<0.001) in PTSD (crosses) and control cases (boxes).
FUTURE OF PERSONALIZED MEDICINE

Better evidence for diagnostics and therapies

Translate research... empower patients!

Give me my data!

Test before you treat

Giant leaps in medicine are just around the corner!

Get to the right drug the first time!

All of the data from the internet can be stored in DNA in a small test tube!

www.DiscoveryDoodles.com
ROC Curve for Selected CpG Sites of the FTSJ3 Gene

CpG Sites Entered
- cg05642923
- cg06024079
- cg16866257
- cg10840864
- cg24581160

Performance on 58 samples (18 PTSD and 40 Controls)
- AUC: 0.960
- Sensitivity: 0.944
- Specificity: 0.850
ROC Curve for Selected CpG Sites from the NFkB Complex and AMPA Receptor

Gene - CpG Sites Entered
ATP1A1 - cg04403423
IKBKAP - cg146147935
PIN1 - cg03560053
SIGIRR - cg02996181
TMEM14C - cg06359086
TNFRSF10C - cg23221723

Performance on 58 samples (18 PTSD and 40 Controls)
AUC: 0.985
Sensitivity: 0.944
Specificity: 0.950
Activity Details

Thursday 8 Jan 2015

BIOMETRICS

- HEART RATE
  91 avg bpm

- STEPS
  19545
  14 avg steps/min

- CALORIES
  5629.4 cal
  3.9 avg cal/min

- SKIN TEMP
  88.4 avg ºF

- PERSPIRATION
  30.9 µS/cm
  0.022 avg µS/cm

ACTIVITY

- WALKING
  16527
  avg steps/min
  3 hr 17 min total

- RUNNING
  0
  0 avg steps/min
  0 min total

- BIKING
  0 cal
  0 avg cal/min
  0 min total
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Tom Hyde, Ph.D.

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LTC Douglas Maurer, D.O
Melanie Carless, Ph.D.
Katherine Compton, R.N.
Joe Cuellar, B.S.
Katheryn Gonzales, B.S.
Tamara DeFranc, M.D.
Nellie Springston, M.S.
Cathy Orihuela, M.A.
Rene Olvera, M.D

Pre-Clinical
David Morilak, Ph.D.
Randy Strong, Ph.D.
Alan Frazer, Ph.D.

Treatment
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Thank you!