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ADHD From A to Z: Advances in the Understanding and Management of Attention Deficit Hyperactivity Disorder

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Symptoms and Demographics



Dr. Barkley's Disclosure

Retirement Pension: State of Massachusetts (UMASS Medical School)
Speaking Fees Received From the Following (2012):
University of Alabama, Student Disabilities Service, Annual ADHD Conference, Tuscaloosa, AL
Alberta Learning Disabilities Association (Edmonton, Canada)
LDA Life and Learning Services, Rochester, NY
CMI Education Institute/Premier Educational Solutions (CMI/PESI)
Cincinnati Children's Hospital & Springer School
Yulius Academy, Rotterdam, The Netherlands
ADHD Network, Utrecht, The Netherlands
Maasstad Hospital, Rotterdam, The Netherlands
Association for Personal Training & Development, Bucharest, Romania
Milwaukee Children's Hospital/Medical College of Wisconsin
Nebraska Psychological Association, Omaha
ADHD Support Network, Moose Jaw, Saskatchewan, Canada
Horizon Health Network, Moncton, Nova Scotia, Canada
ADHD School Specialties & Groves Academy, Minneapolis, MN
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Speaker: Eli Lilly, Shire.
Consultant: Theravance

Obvious Symptoms

Age-Inappropriate Hyperactivity-Impulsivity

- Impaired verbal and motor inhibition
- Impulsive decision making; cannot wait or defer gratification
- Greater disregard of future (delayed) consequences
- Excessive task-irrelevant movement and verbal behavior
 - Fidgeting, squirming, running, climbing, touching
- Restlessness decreases with age, becoming more internal, subjective by adulthood
- Emotionally impulsive; poor emotional self-regulation
 - Impatient, low frustration tolerance, quick to anger, easily excitable, and generally greater levels of expressed emotions

More Obvious Symptoms

Age-Inappropriate Inattention

- But there are at least 6 types of attention:
 - Arousal, alertness, selective, divided, span of apprehension, & persistence
 - Not all are impaired in ADHD
- Poor persistence toward goals or tasks
- Greater reactivity to distractions
- Less able to re-engage the incompleting task following disruptions
- Impaired working memory (remembering so as to do)

ADHD Varies by Setting

Better Here:

Worse Here:

- Fun _____ Boring
- Immediate _____ Delayed Consequences
- Frequent _____ Infrequent Feedback
- High _____ Low Salience
- Early _____ Late in the Day
- Supervised _____ Unsupervised
- One-to-one _____ Group Situations
- Novelty _____ Familiarity
- Fathers _____ Mothers
- Strangers _____ Parents
- Clinic Exam Room _____ Waiting Room



Prevalence (United States)

- 2-5% of children (using DSM-III or III-R)
- 7-8% of children (using DSM-IV) (~3-4 million)
 - Adding Inattentive Type doubles prevalence over III-R
- 4-5% of adults (~12 million in US)
- Varies by sex, age, social class, & urban-rural
 - 3:1 Males to females in children (5:1 in clinical samples)
 - <2:1 males to females in adults
 - More common in children; less so in adults
 - Somewhat more common in middle to lower-middle classes
 - More common in population dense areas
 - For instance, 12-15% of U.S. military dependents (DSM-III-R)
 - No evidence for ethnic differences to date that are independent of social class and urban-rural



Persistence to Adulthood

- 70-80% persistence into adolescence
- Young Adulthood (age 20-26) (Barkley et al. 2002; Milwaukee Study)
 - 3-8% Full disorder (self-report using DSM3R)
 - 46% Full disorder (parent reports using DSM3R)
 - 66% - Using 98th percentile of severity (parent report)
 - 85-90% remain functionally impaired
- Adulthood (mean age 27)
 - 14-35% recovered from disorder
 - 44-55% still fully disordered (diagnosable)
 - 15-30% highly symptomatic but not diagnosable



Diagnostic Criteria

DSM-IV Criteria for ADHD

- Manifests 6+ symptoms of either inattention or hyperactive-impulsive behavior
- Symptoms are developmentally inappropriate
- Have existed for at least 6 months
- Occur across settings (2 or more)
- Result in impairment in major life activities
- Developed by age 7 years
- Are not best explained by another disorder, e.g. Severe MR, PDD, Psychosis
- 3 Types: Inattentive, Hyperactive, or Combined

Changes Expected in DSM5

- Symptom list remains the same (18) but with parenthetical clarifications for teens and adults
- Symptom threshold for children and teens remains the same (#6) but is reduced for adults (#5)
- Age of onset adjusted to age 12 years
- Requires corroboration of self-reports
- Replaces subtypes with "presentations"
- Will impairment be defined?? (should be the "average person" standard)

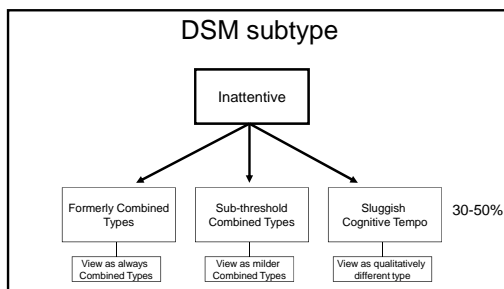
Other Issues DSM5 Failed to Address

- Inattention list is mislabeled
 - Include executive functioning (working memory)
- Symptoms of impulsiveness or poor inhibition are chiefly verbal
 - needed to add poor impulse control generally and motor, cognitive, and affective/motivation specifically
- Symptoms of poor executive emotion self-regulation are important central features but receive no mention
- Symptoms and wording are not appropriate past childhood – parenthetical clarifications may help but not enough
 - Need more items for adult stage of disorder

More Issues

- Symptom threshold (6) not appropriate past childhood – adjustment to 5 for adults helpful but not enough
 - May have to adjust thresholds down to 4 of 9 if > age 17 and higher than 6 if < 4 yrs
- Threshold for children based mainly on boys (3:1)
 - May need to be lower for girls; use rating scales
- Duration may be too short for preschoolers:
 - try 1 year or more
- Requires cross-setting occurrence of symptoms that implies need for parent-teacher agreement
 - Instead, blend reports of both and use history of cross setting impairment

What About the Inattentive Type (ADD)?



A Second Disorder of Attention: Sluggish Cognitive Tempo

SCT Symptoms on Rating Scales

- Daydreaming excessively
- Trouble staying alert or awake in boring situations
- Easily confused
- Spacey or "in a fog"; Mind seems to be elsewhere
- Stares a lot
- Lethargic, more tired than others
- Underactive or have less energy than others
- Slow moving or sluggish
- Doesn't seem to understand or process information as quickly or accurately as others
- Apathetic or withdrawn; less engaged in activities Gets lost in thought
- Slow to complete tasks; needs more time than others
- Lacks initiative to complete work or effort fades quickly

What do we know about SCT?

- Most symptoms of Sluggish Cognitive Tempo (SCT) are not characteristic of the C-Type^{1,2}
- SCT Symptoms form 2 dimensions of daydreamy-confused and sluggish/lethargic in factor analysis. The former are the more diagnostic from ADHD²
- Slow, Error Prone Response Style & Processing
 - Less able to use relevant environmental cues in task responding^{2,3}
- Poor Focused or Selective Attention
 - Slower reaction times, more omission errors^{1,4}
 - Unlike ADHD-C type, sluggish style is cross-situational⁴

1. Milich, R. et al. (2001). *Clinical Psychology: Science and Practice*, 8, 463-488.
2. Penny, A. M. et al. (2009). *Psychological Assessment*, 21, 380-389.
3. Solanto, M. V. et al. (2007). *Journal of Abnormal Child Psychology*, 35, 729-744.
4. Derefinko, K. J. et al. (2008). *Journal of Abnormal Child Psychology*, 36, 745-758.

ADHD Inattentive Type - SCT

- Socially shy, reticent or withdrawn – less impaired socially than ADHD children
- No motor disinhibition problems or impulsiveness on ratings or on cognitive testing in most studies^{1, 2}
 - If anything, they can be overly inhibited⁴
- Little evidence for executive function deficits on tests; if present are in working memory and problem-solving.³
- But some EF deficits are evident on EF ratings in daily life. In children, these are far milder than in ADHD. In adults, they are more prominent. In both, they are mostly in Self-Organization and Problem-Solving

1. Milich, R. et al. (2001). *Clinical Psychology: Science and Practice*, 8, 463-488.
2. Penney, A. M. et al. (2009). *Psychological Assessment*, 21, 380-389.
3. Solanto, M. V. et al. (2007). *Journal of Abnormal Child Psychology*, 35, 729-744.
4. Derfinko, K. J. et al. (2008). *Journal of Abnormal Child Psychology*, 36, 745-758.

More Distinguishing Features of SCT

- Comorbidity: Rarely show Aggression or ODD/CD
- Greater risk may be for anxiety symptoms
- Possibly greater risk for depression (?)
- Lower levels of parenting stress
- Greater parental concerns regarding school failure
- Equally impaired in educational performance
 - But ADHD is a productivity disorder while SCT is an accuracy disorder
 - Greater frequency of math disorders in SCT (?)
- Greater family history of anxiety and LD (?)

Treatment Implications for SCT

- All research has been with children, not with adults
- All drug research was with methylphenidate and used ADD without H cases (or Inattentive Only) – not selected specifically for SCT
- Less Likely to Have a Clinically Impressive Response to Stimulants (based on a few studies; need more research)
 - (Barkley Study finds 65% improve modestly in symptom ratings but only 20% showed a good clinical response warranting continued medication)
- Better response to social skills training in children than ADHD cases
 - Up to 25% of ADHD cases become more aggressive in social skills groups due to peer deviancy training
 - Training works best for shy, withdrawn, anxious children
- Good (better?) response to joint home-school treatments
 - MTA study: anxious cases did the best in psychosocial treatment
 - Pfiffner (2007) study shows good response to home-school behavioral training and child training in social and organizational skills that is targeted at ADHD-I specific problems^{*}

* Pfiffner, L. et al. (2007). *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 1041-1050.
** Geller, D. et al. (2007). *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 1119-1127.

More SCT Treatment Considerations

- More responsive to cognitive therapy (??)
 - It doesn't work for children with ADHD but if this is not ADHD then try it again?
 - It does work for anxiety disorders and depression
- Do adults respond to CBT focusing on EF deficits as well as do ADHD adults?
 - And do they need to be on medication like ADHD adults?
 - Doubtful, as ADHD medications don't seem as useful for SCT
- Consider atomoxetine (??) Why? It may treat anxiety in ADHD cases – SCT cases are more likely to have anxiety
- Consider modafinil (anti-narcoleptic) (??) Why? Is SCT a disorder of arousal?
- If SCT is ruminative or related to OCD, consider clomipramine or fluvoxamine used to treat OCD (??)



Life Course Impairments



Childhood Physical/Medical Risks

- Seizures – 2.5x increase in risk
- 1.5 x more likely to be overweight
(Waring et al. (2008). *Pediatrics*, 122, e1-e6 (online 8/28/08)
- Sleep problems (39-56%)
 - bedtime behavior issues, delayed onset and greater night waking leading to shorter sleep time
 - parent reports of sleep problems are not related to academic achievement scores*
 - but sleep problems are related to daytime inattentiveness**
- Poorer oral health practices including lower likelihood of dental brushing each evening, more dental visit behavior problems, and more dental decay at age 11 (but not at 13)

*Moyles, S. et al. (2006). *Journal of Developmental and Behavioral Pediatrics*, 29, 206-212.
**Willoughby et al. (2008). *Journal of the American Academy of Child and Adolescent Psychiatry*, 47, 186-194

More Health Risks

- **Developmental Coordination Disorder (30-50+%)**
 - Deficient movement skills relative to peers* and lasts into adolescence**
 - More left-sided un-coordination in both ADHD cases and siblings
 - Reduced physical fitness, strength, & stamina (physical fitness tests)
 - Girls as affected as boys, unaffected sibs have same motor problems
 - Motor problems and ADHD share same risk genes**
 - Motor incoordination related more to inattention dimension of ADHD**
- **Accident Proneness 57%+**
 - 1.5 to 4x risk of injuries (greater in ODD)
 - 3x risk for accidental poisonings
 - Why? Impulsive, risk-taking, less coordinated, more oppositional, and less parental monitoring

2-3 times the medical costs of normal children or those with non-ADHD behavior problems (also greater maternal medical and job-related costs – sick days, absenteeism, etc.)***

*Harvey, W. J. et al. (2007). *Journal of Abnormal Child Psychology*, 35, 871-882.

**Fliers, A. et al. (2010). Papers from dissertation are in press – contact clarifline.vanzelji@gmail.com

***Haackart-van Roijen, L. et al. (2007). *European Child & Adolescent Psychiatry*, 16, 316-326

***Leibson, C. et al. (2001). *Journal of the American Medical Association*, 285, 60-66.

Childhood Developmental Risks

- **Greater Risk for Language Disorders**
 - Expressive: 10-54% Pragmatic deficits in 60%
 - Excessive speech, reduced fluency, less logical, coherent, & organized
 - Delayed internalization of language
 - Reduce capacity for rule-governed behavior
- **Low Average Intelligence (7-10 point deficit)**
 - An apparent failure to keep pace with peers but could also result from poor executive functioning that partly affects IQ
- **More Adaptive Disability – 1 to 2 SD difference between IQ and adaptive functioning (self-sufficiency), particularly in communication and socialization domains**
- **Positive illusory bias in self-evaluations of competence**
 - Not evident in their evaluations of other children's abilities

Childhood Academic Impairments

- **Poor School Performance (90%+)**
 - greater inattention to classwork
 - reduced productivity is greatest problem
 - accuracy is only mildly below normal (85%)
 - more hyperactive & disruptive behavior
- **Low Academic Achievement (10-15 pt. deficit)**
 - May be deficient even in preschool readiness skills
- **Learning Disabilities (24-70%)**
 - Reading (8-39%); (effect size (ES) = 0.64)
 - Spelling (12-30%) (ES = 0.87)
 - Math (12-27%) (ES = 0.89)
 - Handwriting (60%+)
 - Reading, viewing, & listening comprehension deficits
 - Likely due to impact of ADHD on working memory

Family Relations Impairments

- Increased parent-child conflict & stress
 - Greater parental commands, hostility, reduced responsiveness, more lax yet harsh discipline
 - More child noncompliance, hostility, disruption
 - Poorer sense of competence in parental role
 - Greater parenting stress and maternal depression
 - Especially problematic for ODD/CD subgroup
 - Higher rates of parental divorce and earlier divorce (related to parental education & IQ and child age, race, and ODD/CD)*

*Wymbs, B. et al. (2008). *Journal of Consulting and Clinical Psychology*, 76(5), 735-744.

Parental ADHD

- Screening of parents for ADHD and related disorders and management as needed to optimize child ADHD treatment. Why?
 - Parental inattention is related to inconsistent discipline and low child monitoring while parent impulsivity is related to low child social reinforcement. Chen & Johnston (2007). *Journal of Clinical Child and Adolescent Psychology*, 27(3).
 - Maternal ADHD is related to lower rates of involvement with their children and lower positive reinforcement, higher rates of negative behavior and greater rates of commands to their ADHD children. Chronis-Toscano et al. (2008). *Journal of Abnormal Child Psychology*, 36(8), 1237-1250.
 - Parental ADHD is related to reduced success in behavioral parent-training programs.

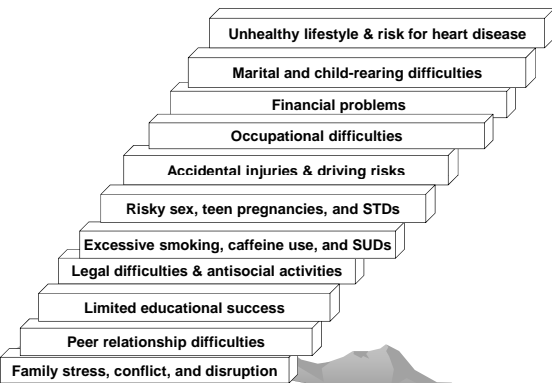
Peer Relationship Impairments

- Peer Relationship Problems (50-70%+)
 - Less sharing, cooperation, turn-taking
 - More directive of others, more off-task during interactive play activities
 - More intrusive, verbally impulsive, emotional
 - Greater reactive anger and aggression
 - Consequently, higher rates of peer rejection
 - Some show reduced empathy and guilt
 - Subset having early onset CD at higher risk for psychopathy
 - Relations are more impaired in ODD/CD subgroup
 - More likely to be bullied and to be bullies in childhood
 - More likely to be beaten up, mugged, or assaulted with a weapon by young adulthood

Comorbid DSM-IV Disorders

- Oppositional Defiant Disorder (40-84%; M = 55%)
- Conduct Disorder (15-56%; Mean = 45%)
 - Predicts personality disorders by age 27 - 10-21%; antisocial & borderline most ly
- Anxiety Disorders (20-35%; odds = 3.0): increases with age
- Major Depression (25-35%; odds = 5.5)
 - Suicidal ideation & attempts increase during high school; decrease by age 27
- Risk for substance use disorders (20-30%) by adolescence
- No elevated rates of PTSD except in comorbid ODD and especially Bipolar cases (22-24%)
- Autistic Spectrum Disorders (22%)
 - 28-52% of ASD cases have ADHD
- Bipolar Disorder (0-27%; likely 6-10% max.)
 - A one-way comorbidity? (80-97% BPDs have ADHD but only 2-3% of ADHD cases have BPD)
- Females have risk for bulimia – binge eating (16%)

Adaptive Impairments by Adulthood



The Causes of ADHD

Etiologies

- Disorder arises from multiple causes
- All currently recognized causes fall in the realm of biology (neurology, genetics)
- Causes may compound each other
- Final common pathway for disorder appears to be the fronto-striatal-cerebellar circuits in the brain
- Social causes lack credibility



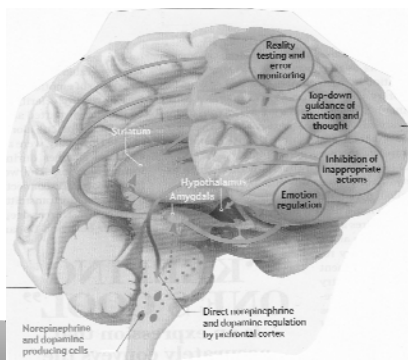
Neuro-Imaging Findings

Smaller, Less Active, Less Developed Brain Regions (found on MRI, fMRI, PET, QEEG)

- Orbital-Prefrontal Cortex (primarily right side)
- Anterior cingulate cortex
- Basal Ganglia (mainly striatum & globus pallidus)
- Cerebellum (central vermis area, more on right side)
- Anterior of corpus callosum (splenium)
- Possibly smaller thalamus
- Size of this network is correlated with degree of ADHD symptoms, particularly inhibition
- No gender differences
- Results are not due to taking stimulant medication



Executive Networks



Acquired Cases: Prenatal

- Maternal smoking in pregnancy (odds 2.5)
- Maternal alcohol drinking in pregnancy (same)
- Prematurity of birth, brain bleeding (45% ADHD)
- Total increased pregnancy complications
- Maternal high phenylalanine levels in blood (?)
- Maternal pre-pregnancy obesity
- Maternal Pitocin exposure during delivery
- High maternal anxiety in second trimester (?)
- Maternal methylmercury ingestion (fish in diet)
- Cocaine/crack exposure not a risk factor after controlling for the above factors



Acquired Cases: Post-Natal (3-7%)

- Head trauma, brain hypoxia, tumors, or infection
- Febrile seizures
- Lead poisoning in preschool years (0-3 yrs.)
- Survival from acute lymphoblastic leukemia (ALL)
 - Treatments for ALL cause brain damage resulting in most survivors having ADHD (SCT) symptoms*
- Post-natal Streptococcal Bacterial Infection
 - triggers auto-immune antibody attack of basal ganglia
- Post-natal elevated phenylalanine (dietary amino acid related to PKU)
 - Prenatal – hyperactivity
 - Post-natal – inattention

*Reeves, C. et al. (2007). *Journal of Pediatric Psychology*, 32, 1040-1049.



Heredity – Family Studies

- Family Aggregation of Disorder:
 - 25-35% of siblings
 - 78-92% of identical twins
 - 15-20% of mothers
 - 25-30% of fathers
 - If parent is ADHD, 20-54% of offspring (odds 8+)



Heredity – Twin Studies

- Heritability (Genetic contribution)
 - 57-97% of individual differences (Mean 80%+)
 - (91-95%+ using DSM criteria)
- Shared Environment (common to all siblings)
 - 0-6% (Not significant in any study to date)
- Unique Environment (events that happen only to one person in a family)
 - 15-20% of individual differences
 - (but includes unreliability of measure used to assess ADHD)

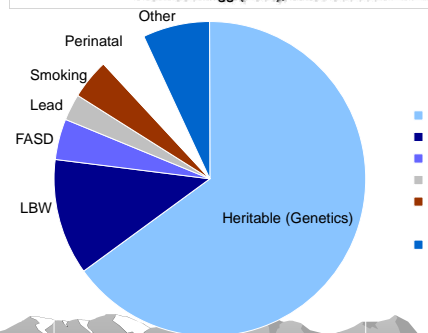
Molecular Genetics^{1,2,3}

- **DRD4 – 7+ repeat and 4 repeat absent (?)**:
 - Related to novelty seeking, exploratory behavior, possibly human migration patterns; Longer genes blunt dopamine sensitivity. Those lacking 4 repeat do better on methylphenidate.⁶
- **DAT1 – 480 bp (9/10 heterozygous differs from 9/9, 10/10)**
 - Function not well known; likely serving as a tag for other nearby functional gene regions; May build the dopamine transporter (reuptake pumps); Those with single copy 10 variants or with homozygous pairings (10/10) may respond less well to methylphenidate.⁶ 10 repeat interacts with maternal alcohol use to increase risk for ADHD; 9/10 pairing has marked effect on severity of ADHD across childhood to adulthood.
- **DBH – TaqI (A2 allele)**
 - May create chemical (DBH) that converts dopamine to norepinephrine
- **MAO-A**: produces an mitochondrial enzyme that regulates presynaptic dopamine signals and other neurotransmitter systems
- **LPHN3 (latrophilin) gene** – linked to G-protein-coupled receptors in amygdala, caudate, cerebellum, and cortex. Controls GABA release presynaptically; GABA is an inhibitory transmitter^{4,5}
- **Genome wide scans** – suggest that 20 or more additional chromosome sites may contain minor genes that are possible candidates²

1. Mick, E. & Faraone, S. V. (2008). *Child and Adolescent Psychiatric Clinics of North America*, 17, 261-284.
2. Franke, B. et al. (2009). *Human Genetics*, published online April 2009.
3. Bakschewski, T. et al. (2010). *European Child and Adolescent Psychiatry*, 19, 237-257.
4. Ribases et al. (2010). *Genes, brain, and behavior*, online first, doi:10.1111/j.1601-183X.2010.00649.x
5. Arcos-Burgos, M. (2010). *Attention deficit hyperactivity disorder*, 2, 139-147.
6. Froehlich, T. (2011). *Journal of the American Academy of Child and Adolescent Psychiatry*, 50, 1129-1139.

Etiologies of ADHD

From Joel Nigg (2006). *What Causes ADHD?*



ADHD As A Disorder of Executive Functioning (Self-Regulation)



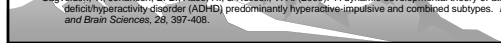
The Prefrontal Cortical Networks Involved in EF Are Also the Networks Implicated in Self-Regulation and in ADHD

- **The frontal-striatal circuit:** Associated with deficits in response suppression, freedom from distraction, working memory, organization, and planning, known as the “cool” or “**what**” EF network
- **The frontal-cerebellar circuit:** Associated with motor coordination deficits, and problems with the timing and timeliness of behavior, known as the “**when**” EF network
- **The frontal-limbic circuit:** Associated with symptoms of emotional dyscontrol, motivation deficits, hyperactivity-impulsivity, and proneness to aggression, known as the “**hot**” or “**why**” EF network

Nigg, J. T., & Casey, B. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development and Psychology*, 17, 785-806.

Castellanos, X., Sonuga-Barke, E., Milham, M., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Science*, 10, 117-123.

Sagvolden, T., Johnsen, E. B., Kase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive-impulsive and combined subtypes. *Behavioral and Brain Sciences*, 28, 397-408.



Problems with the EF Construct

- Lacks any consensus definition
 - More than 20 definitions exist
 - Most emphasize self-regulation, goal directed behavior, and problem-solving
- Lacks any theory of EF, especially as it extends into daily life
- Considered to be a meta-construct serving as an “umbrella” term for a set of more specific components
 - But up to 33 components have been attributed by experts to EF
- Assessment of EF often employs psychometric tests. But:
 - They are unreliable and often poorly normed
 - They lack ecological validity
 - do not correlate with EF rating scales or observations
 - Do not predict impairment in major domains of life in which EF is important for effective functioning; EF ratings do predict impairment



Most Common EF Components

- Inhibition and interference control
- Self-Awareness and self-monitoring
- Nonverbal working memory
- Verbal working memory
- Planning and problem-solving
- Anticipation and preparation to act
- Self-Regulation across time
- Emotional Self-Control

Defining EF as Self Regulation

- Self-regulation is:
 - Any action directed at one's self
 - Intended to change one's behavior from what it otherwise would have been
 - So as to change the likelihood of a later consequence (so as to attain a goal)
- The EFs are types of self-directed actions
- *"EF is the use of self-directed actions (self-regulation) to choose, enact, and sustain actions across time toward a goal usually in the context of others and often relying on social and cultural means for the maximization of one's longer-term welfare as the person defines that to be."*
(Barkley, R. A. *Executive Functioning and Self-Regulation*, 2012, New York: Guilford Publications)

EFs as Self-Directed Actions

- Inhibition = Self-Restraint
- Self-Awareness = Self-Directed Attention
- Nonverbal working memory = Self-Directed Sensory Motor Actions (Imagery)
- Verbal working memory = Self-Speech
- Planning and problem-solving = Self-Play
- Emotional Self-Control = Self-Directed Emotion and Motivation

The EFs Create Four Developmental Transitions in What is Controlling Behavior

- External → Mental (private or internal)
- Others → Self
- Temporal now → Anticipated future
- Immediate → Delayed gratification
(Decreased Temporal Discounting of Delayed Consequences)

Think of Executive Functioning
As A Hierarchy That Forms an
Extended Phenotype Essential for
Social Existence

Michon's Model of Driving

Level IV: Strategic Abilities

i.e., Purpose or goals for using the car, best routes through traffic to attain the goals, time likely needed to attain each goal, knowledge needed to enact the plan effectively (weather, traffic, construction, known accidents, etc.)



Level III: Tactical Abilities

i.e., abilities required to operate the vehicle on roadways in the presence of and interactions/conflicts with other drivers and their vehicles, such as driving laws, knowledge of safe driving tactics, etc.



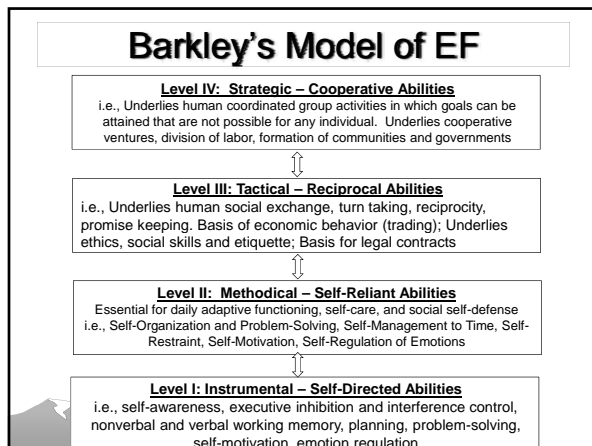
Level II: Operational Abilities

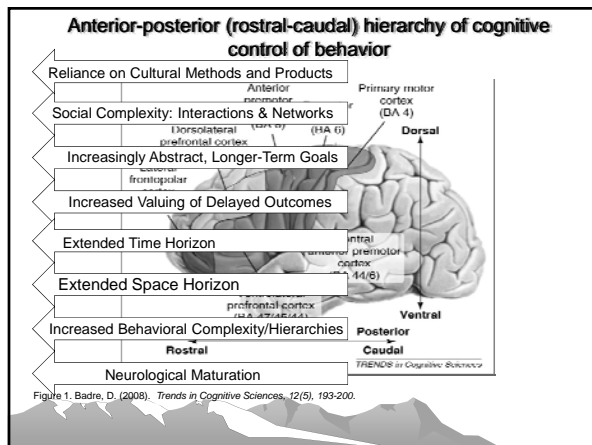
i.e., familiarity with and sound management of the vehicle and its components such as steering, braking, acceleration, signaling, mirrors, seat belts, other safety equipment [ex. Driving a car in an empty parking lot]

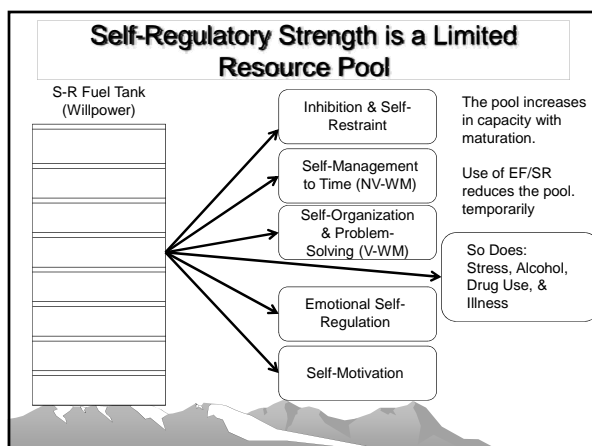


Level I: Basic Cognitive Abilities Required to Drive

i.e., normal reaction time; visual field perception; motor speed, agility, coordination, and range of motion; visuo-spatial reasoning; hearing; language and reading abilities, etc.

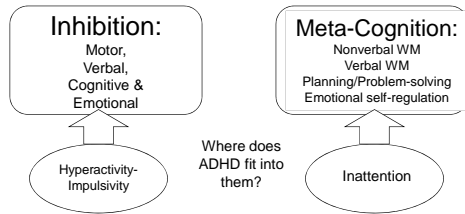






How Does ADHD Fit Into EF?

EF Comprises a Single Domain that Can Be Usefully Subdivided into two Broad Dimensions



Understanding ADHD

- ADHD disrupts the 5 levels of EF/SR but especially the tactical and higher levels thereby creating a disorder of self-regulation across time
- ADHD can be considered as "Time Blindness" or a "Temporal Neglect Syndrome" (Myopia to the Future)
- It adversely affects the capacity to hierarchically organize behavior across time to anticipate the future and to pursue one's long-term goals and self-interests (welfare and happiness)
- It's not an Attention Deficit but an Intention Deficit (Inattention to mental events & the future)

Understanding ADHD

It's a Disorder of:

- Performance, not skill
- Doing what you know, not knowing what to do
- The when and where, not the how or what
- Using your past at the "point of performance"

The point of performance is the place and time in your natural settings where you should use what you know (but may not)

Implications for Treatment

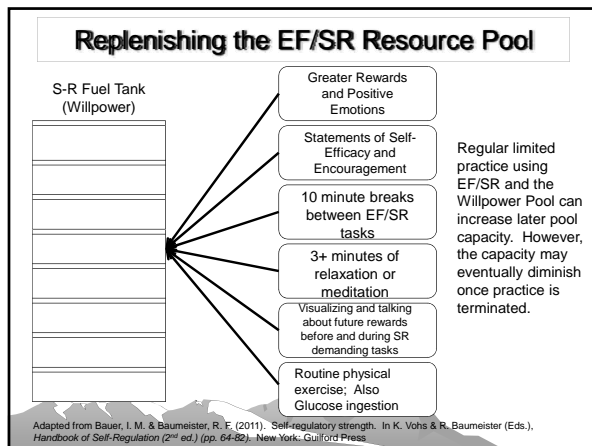
- Teaching skills is inadequate
- The key is to design prosthetic environments around the individual to compensate for their EF deficits
- Therefore, effective treatments are always those at the "point-of-performance"
- The EF deficits are neuro-genetic in origin
- Therefore, medications may be essential for most (but not all) cases – meds are neuro-genetic therapies
- But some evidence suggests some EFs may also be partly responsive to direct training
- While ADHD creates a diminished capacity: Does this excuse accountability?
 - (No! The problem is with time and timing, not with consequences)

More Treatment Implications

- Behavioral treatment is essential for restructuring natural settings to assist the EFs
 - They provide artificial prosthetic cues to substitute for the working memory deficits (signs, lists, cards, charts, posters)
 - They provide artificial prosthetic consequences in the large time gaps between consequences (accountability) (i.e., tokens, points, etc.)
 - But their effects do not generalize or endure after removal because they primarily address the motivational deficits in ADHD
- The compassion and willingness of others to make accommodations are vital to success
- A chronic disability perspective is most useful

How can we compensate for EF deficits? By reverse engineering the EF system

- Externalize important information at key points of performance
- Externalize time and time periods related to tasks and important deadlines
- Break up lengthy tasks or ones spanning long periods of time into many small steps
- Externalize sources of motivation
- Externalize mental problem-solving
- Replenish the SR Resource Pool (Willpower)
- Practice incorporating the 5 strategies for emotional regulation in daily life activities



Conclusions

- ADHD is more than just a disorder of attention and inhibition
- It is a developmental disorder of executive functioning
- It is persistent into adulthood in 65-84% of childhood diagnosed cases
- It is associated with numerous impairments in major life activities
- It arises largely from neurological and genetic causes
