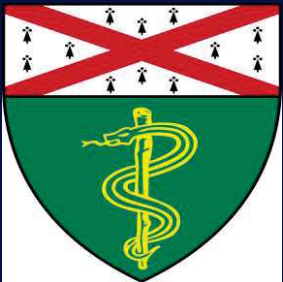


# How Our Understanding of ADHD Has Changed: Executive Functions

Thomas E. Brown, PhD

Associate Director,  
Yale Clinic for Attention and Related Disorders  
Department of Psychiatry  
Yale Medical School



"Your view of human nature will change profoundly  
as you read this brilliant book."  
—DANIEL KAHNEMAN, author of *Thinking, Fast and Slow*

# THE marshmallow TEST

Mastering Self-Control

WALTER MISCHEL



# **Preschoolers to Adults: ability to exercise self-control is often consistent**

Those able to delay gratification when in preschool, when 25-30 years old had:

- ◆ Higher scores on SAT
- ◆ More schooling completed
- ◆ Maintained close relationships better
- ◆ Less use of risky drugs
- ◆ Lower body mass index scores

## Professor Mischel claims:

- ◆ Ability to delay gratification now for more advantages later is not an inborn trait, but a skill that can be learned
- ◆ “Self-control skills...can be learned, enhanced and harnessed so they become automatically activated when you need them.”

## Yet Prof Mischel recognizes that:

- ◆ Ability to delay gratification depends upon executive functions of the brain
- ◆ “EF gives us the freedom to inhibit and cool impulsive urges, and to think and deploy attention flexibly...to reach our goals.”

## Prof Mischel also recognizes that:

- ◆ In adulthood **brain images** of those who were more able to resist temptation as preschoolers **were different** than those less able to delay
- ◆ “If EF does not develop well...those children are at **increased risk for ADHD**”

# What is essential problem in ADHD?

- ◆ **Old:** behavior problems & not listening
- ◆ **New:** developmental impairment of the brain's management system: EF
- ◆ Aspects of brain's EF don't come online in usual time frame.
- ◆ And don't work consistently



# Executive Functions

- ◆ Wide range of **central control processes** of the brain
- ◆ **Connect, prioritize, and integrate** cognitive functions—moment by moment
- ◆ Like conductor of a symphony orchestra

# “Will you do it and, if so, how and when?”

(Lezak, 2004)

Will you do it?

Motivation/Activation

How will you do it?

Planning/Organizing

When?

Timing/Remembering

# Development of Brain Structures that Support Executive Functions

- ◆ Structures and functions that support EF are not fully developed at birth
- ◆ Neural networks underlying effortful control begin development at 2-4 years old, but don't fully develop until one's 20s
- ◆ Development of EF capacities continues into early adulthood

Rothbart MK, Posner MI. Mechanism and variation in the development of attentional networks. In: Nelson CA, Luciana M, eds. *Handbook of Developmental Cognitive Neuroscience*; 2001.

# Continuing Brain Development in Late Childhood and Adolescence

- ◆ Between 6-15 years, **extreme growth** (to 80%) occurs at the colossal isthmus that supports associative relay, while considerable **synaptic pruning** occurs
- ◆ Brain **myelination increases 100%** during the teenage years
- ◆ Dopamine (DA), norepinephrine (NE), and serotonin (5-HT) **transmitter systems** in the brain **continue to develop** into one's 20s

Thompson PM, et al. *Nature*. 2000;404(6774):190-193.  
Benes FM, et al. *Arch Gen Psychiatry*. 1994;51(6):477-484.

# Characteristics of ADHD Symptoms

- ◆ **Dimensional, not “all-or-nothing”**
  - Everyone sometimes has some impairments in these functions;  
in ADHD: chronic, severe impairment
- ◆ **Situational variability: “If I’m interested”**
  - Most persons with ADHD have a few activities where ADHD impairments are absent

ADHD looks like willpower problem, but it isn't!

# The Mystery of ADHD: Situational Variability of Symptoms

- ◆ Why focus for this, but not that?  
“If it really interests me” (attraction)
- ◆ Why focus then, but not now?  
“If I feel the gun to my head” (fear)

# 2 hypotheses re: motivation in ADHD

“Willpower”

VS

Erectile Dysfunction of the Mind

# Brown's Model of Executive Functions Impaired in ADHD

## Executive Functions

Organizing,  
prioritizing,  
and  
activating  
to work

Focusing,  
sustaining  
focus, and  
shifting focus  
to tasks

Regulating  
alertness,  
sustaining  
effort, and  
processing  
speed

Managing  
frustration  
and  
modulating  
emotions

Utilizing  
working  
memory and  
accessing  
recall

Monitoring  
and self-  
regulating  
action

1.

Activation

2.

Focus

3.

Effort

4.

Emotion

5.

Memory

6.

Action

*Brown TE. Manual for Attention Deficit Disorder Scales for Children and Adolescents; 2001.*



# 1. Organize, Prioritize, and Activate

- ◆ Difficulty organizing tasks, materials
- ◆ Difficulty estimating time, prioritizing tasks
- ◆ Trouble getting started on work

**T.Brown, Attention Deficit Disorder: The Unfocused Mind in Children & Adults (2005)**

## 2. Focus, Shift, and Sustain Attention

- ◆ Loses focus when trying to listen or plan
- ◆ Easily distracted—internal/external
- ◆ Forgets what was read, needs to re-read

**T.Brown, Attention Deficit Disorder: The Unfocused Mind in Children & Adults (2005)**

### 3. Regulating Alertness, Effort, and Processing Speed

- ◆ Difficulty regulating sleep and alertness
- ◆ Quickly loses interest in task, especially longer projects; doesn't sustain effort
- ◆ Difficult to complete task on time, especially in writing—"slow modem"

T.Brown, *Attention Deficit Disorder: The Unfocused Mind in Children & Adults* (2005)

## 4. Manage Frustration, Modulate Emotion

(Not included in DSM-IV criteria)

- ◆ Emotions impact thoughts, actions too much
- ◆ Frustration, irritations, hurts, desires, worries, etc., experienced “like computer virus”
- ◆ “Can’t put it to the back of my mind”

T.Brown, Attention Deficit Disorder: The Unfocused Mind in Children & Adults (2005)

## 5. Utilize Working Memory, Access Recall

- ◆ Difficulty holding one or several things “on line” while attending to other tasks
- ◆ Difficulty “remembering to remember”
- ◆ Inadequate “search engine” for activating stored memories, integrating these with current info to guide current thoughts and actions

**T.Brown, Attention Deficit Disorder: The Unfocused Mind in Children & Adults (2005)**

## 6. Monitor and Self-Regulate Action

(Not just hyperactive/impulsive behavior)

- ◆ Difficulty controlling actions, slowing self and/or speeding up as needed for tasks
- ◆ Doesn't size up ongoing situations carefully
- ◆ Hard to monitor and modify own actions to fit situation/aims

T.Brown, Attention Deficit Disorder: The Unfocused Mind in Children & Adults (2005)

# Executive Functions are complex and operate in dynamic, integrated ways

For example, EF of “focus”

- Does not mean
  - as in holding the camera still to take a photo of an unmoving object
- Does mean
  - as in focusing on the task of driving a car

# A Working Definition of ADHD

(TEBrown, 2013)

- ♦ a **complex syndrome** of
- ♦ **developmental impairments of executive functions,**
- ♦ the self-management **system of the brain,**
- ♦ a system of **mostly unconscious operations.**
- ♦ These impairments are **situationally-specific,**
- ♦ **chronic,** and **significantly interfere with functioning** in many aspects of the person's daily life.



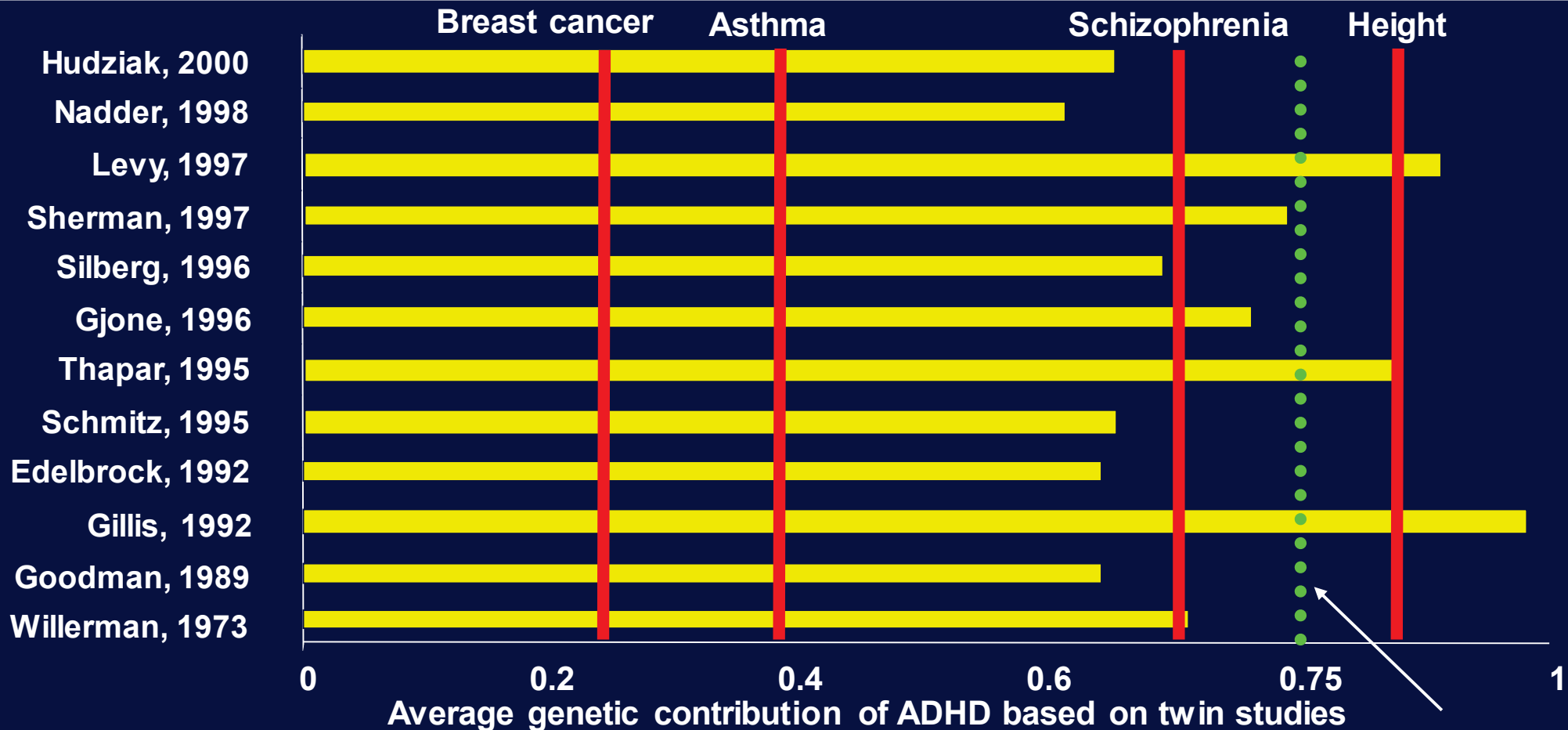
# Executive Functions: Development and Demands

- ◆ EF capacity develops through childhood, into adolescence, and beyond; **it is not fully present in early childhood**
- ◆ Environmental **demands** for EF **increase with age**, from preschool through adulthood
- ◆ EF impairments **often are not noticeable by age 12 yrs!**

# When Are ADHD Impairments Noticeable?

- ◆ Some are obvious very early and are noticeable in **preschool years**
- ◆ Some are not noticeable until middle elementary or **junior high**
- ◆ Some are not apparent until child leaves home to go to **college or later**

# ADHD Genetics: Heritability Coefficient



Faraone. *J Am Acad Child Adolesc Psychiatry*. 2000;39:1455-1457.

Hemminki. *Mutat Res*. 2001;25:11-21.

Palmer. *Eur Resp J*. 2001;17:696-702.

# How Can ADHD Be Assessed?

---

- ◆ When ADHD was seen as just a disruptive behavior disorder in childhood,  $d_x$  was based on observing overt behavior
- ◆ EF impairments of ADHD are largely cognitive, covert, not easily observed directly
- ◆ EF are complex, context dependent, & interactive, not easily captured in “laboratory tests of EF”

(Rabbitt, 1997; Burgess, 1997; Wilson, 1997)

# Measures for Assessment of Executive Functions

---

- ◆ EF modulate and control other functions
- ◆ Single function = pooled outcome of multiple sub-functions
- ◆ **Rating scale & clinical reports** of how the person manages everyday tasks are **far more sensitive & valid diagnostic indicators than neuropsych laboratory “tests of EF”**

(T.Brown, 2005, 2006, 2013; R.Barkley, 2011)

# Brain differences underlying ADHD (temporary and/or longer term)

1. Delay in unfolding of **brain development** that supports executive functions
2. Impaired white matter **connections between brain regions**
3. Impaired control of **oscillations that coordinate** brain region communications
4. Inadequate **release/reloading of transmitter chemicals** at synapses

# Cortex Maturation in ADHD vs NC

- ◆ MRI studies of 40K cortex sites in 223 youths with ADHD vs matched controls
- ◆ Brain maturation was delayed ~3yrs in specific regions in ADHD youths vs NC
- ◆ Frontal areas of cortex slower in ADHD
- ◆ Medial PFC developed lagged 5 yrs

(Shaw, et al, PNAS, Nov, 2007)

# Brain Differences & Similarities in Persistent vs Remitted ADHD

MRI comparisons of persons with remitted vs persistent ADHD at 23.8 yrs indicated increased medial PFC cortical thinning in those with persistent ADHD while cortical thinning gradually reduced and began to thicken in those whose ADHD sx remitted, making their cortical thickness more similar to TDC. (Shaw, Malek, Watson, et al. 2013)



# Brain Differences & Similarities in Persistent vs Remitted ADHD

- ◆ fMRI comparison of adults w/persistent vs remitted ADHD showed clear **differences in functional connectivity** btwn the 2 grps (Mattfeld, Gabrieli, et al. 2014)
- ◆ DTI comparison of adults dx w/ADHD 33 yrs earlier vs controls showed persistent **decrease in white matter connections for both** current and remitted ADHD vs NC  
(Cortese, Imperati, Zhou, et al. 2013)

# Is ADHD Brain Wired Differently?

- ◆ New model shifts focus from regional brain abnormalities to dysfunction in distributed network organization.

(Cortese, Kelly, Chabernaud, et al, 2012)

- ◆ DTI shows converging evidence for white matter pathology & disrupted anatomical connectivity in ADHD

(Konrad & Eichoff, Human Brain Mapping, 2010)

# Chemical Dynamics of Brain also contribute to impairments of ADHD

- ◆ **Not** due to overall “imbalance of chemicals” (not too much/too little salt in soup)
- ◆ But to **inadequate release and/or reloading** of transmitter chemicals in countless **infinitesimal network junctions**
- ◆ **Except for** “messages” re priority interests or fear of imminent unpleasantness

# A Chemical Problem

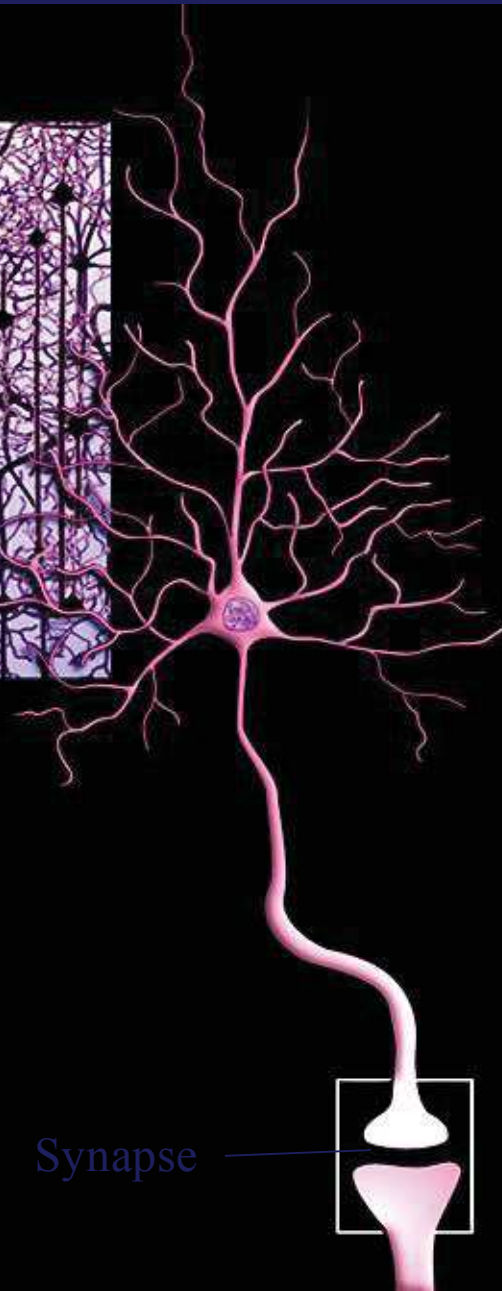
- ◆ ADHD is fundamentally a chemical problem
- ◆ Most effective treatment is to change the chemistry with medication
- ◆ Unless the problematic chemistry is changed, other interventions are not likely to be very effective

# In the Human Brain

- ◆ 100 billion neurons
- ◆ each one linked to >1000 others
- ◆ in complex sub-systems
- ◆ that have to “talk to each other”
- ◆ using low voltage electrical impulses
- ◆ that have to jump across gaps
- ◆ so fast that 12 can cross in 1/1000 sec.







Neuro

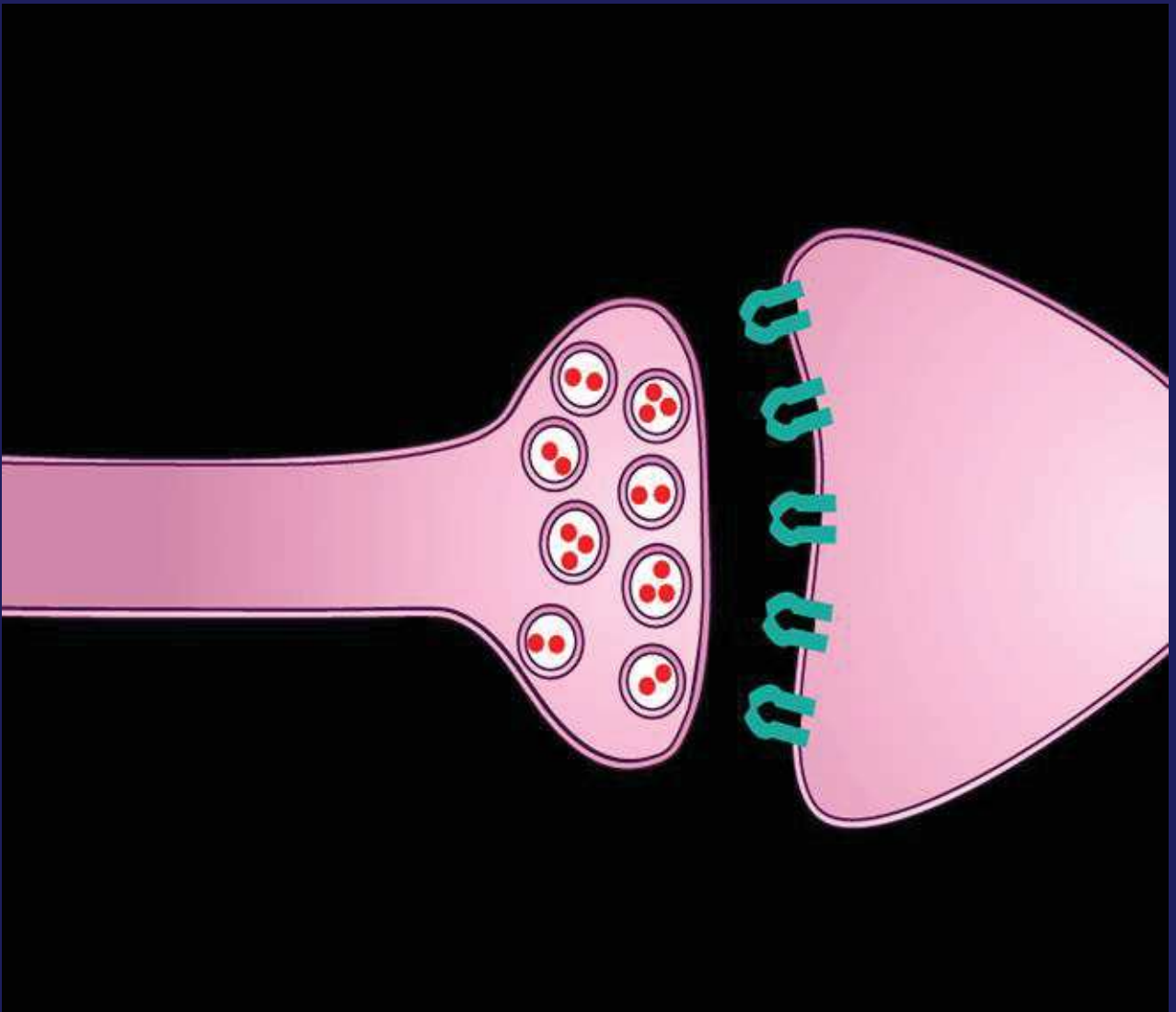
Synapse

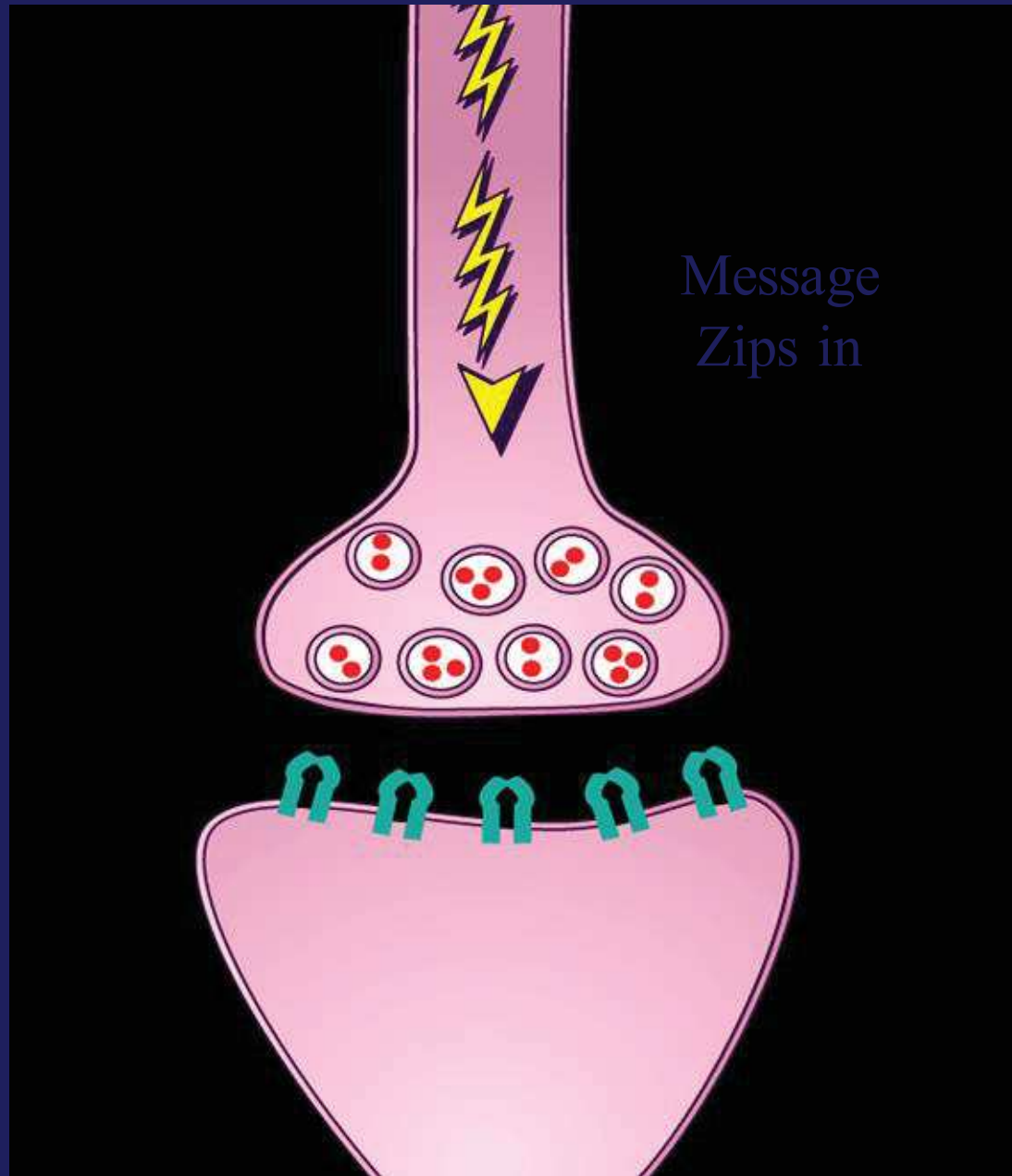
Intertw  
neuron

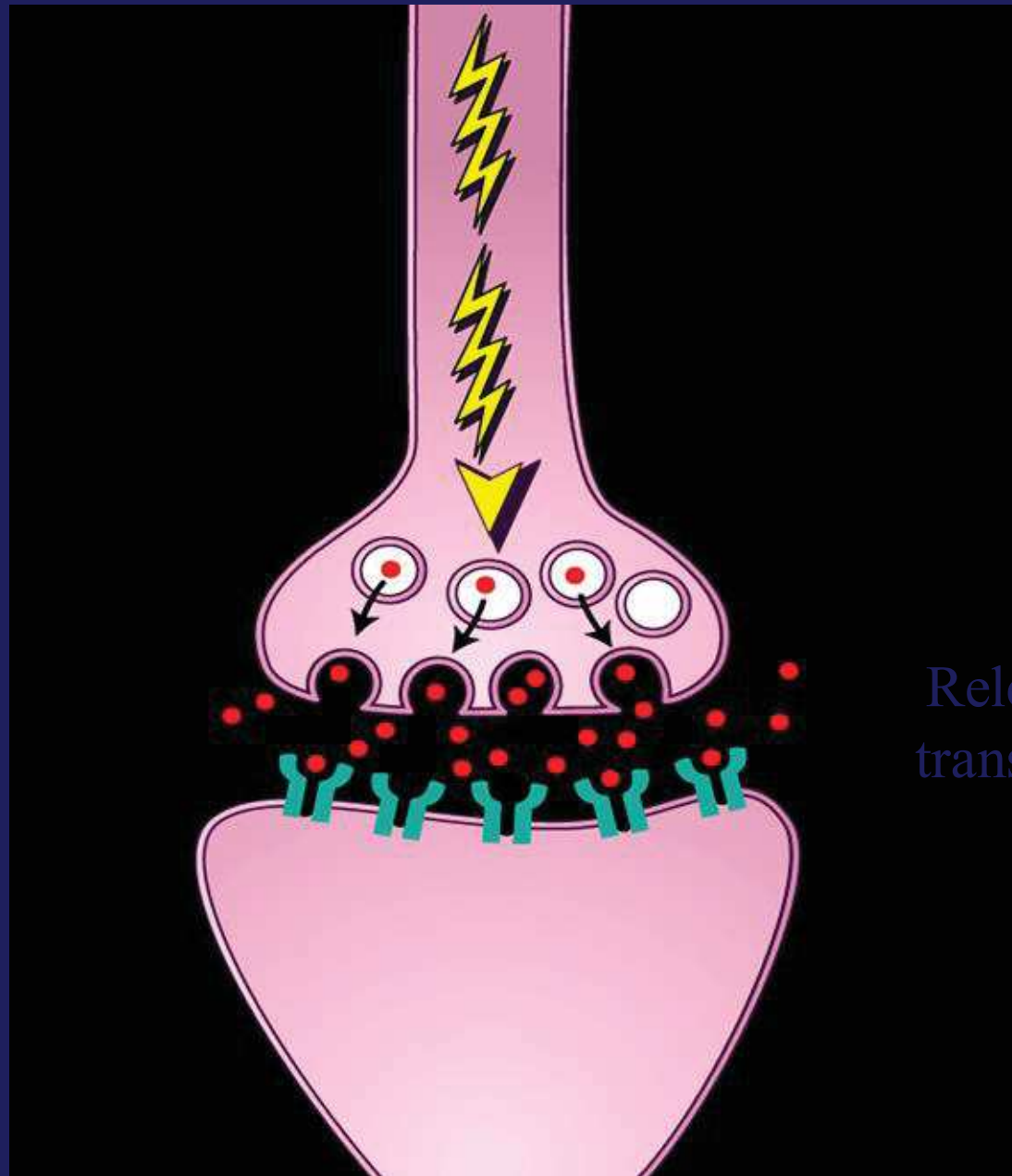
# Chemicals Jump the Gaps

- ◆ Inside brain >50 different chemicals are continuously made
- ◆ every neuron system uses 1 of them
- ◆ stored in little vesicles near tip of neuron
- ◆ when electrical impulse comes, mini-dots of that chemical are released,
- ◆ cross the gap, fire next neuron, then reload in fractions of a second

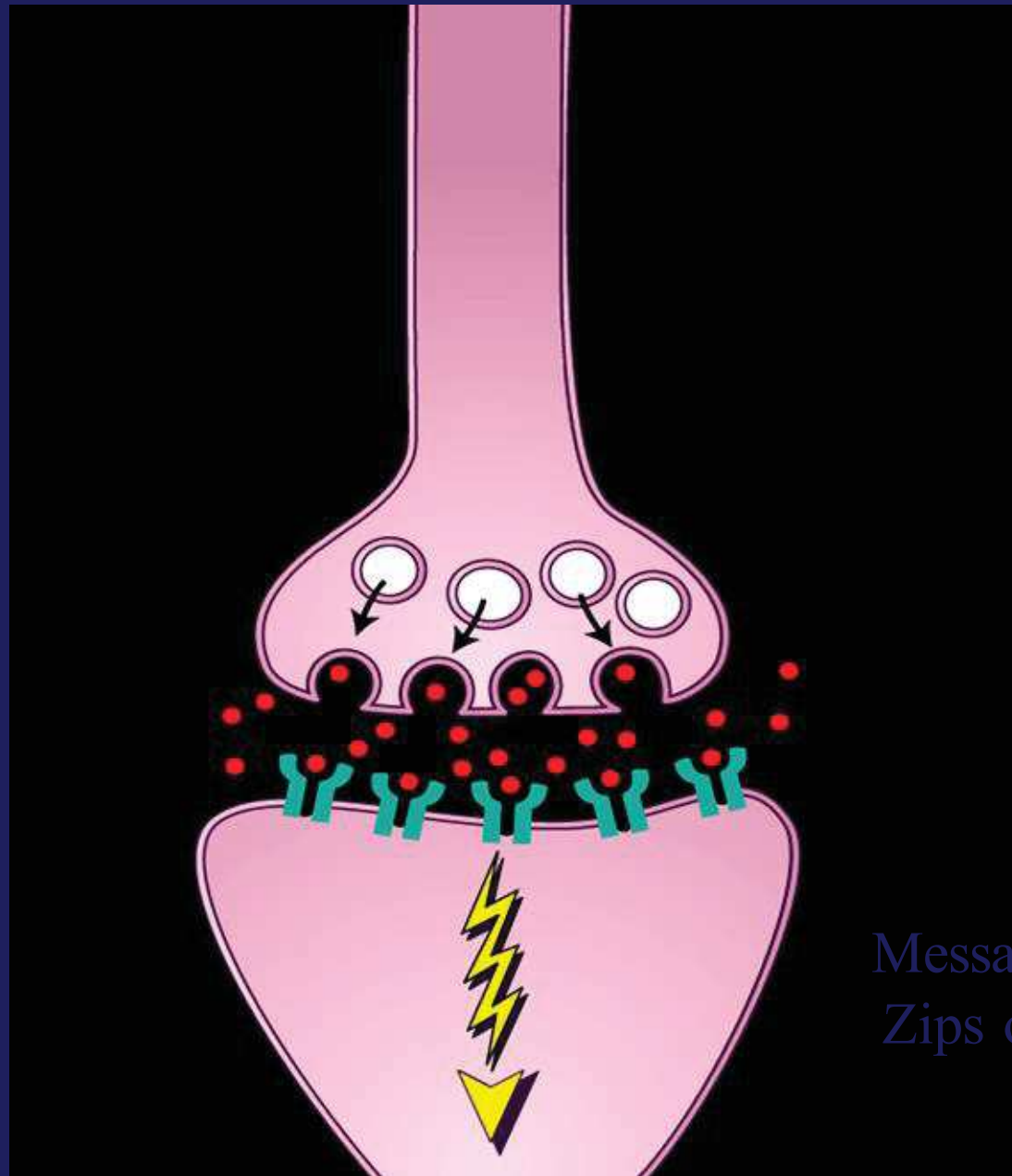




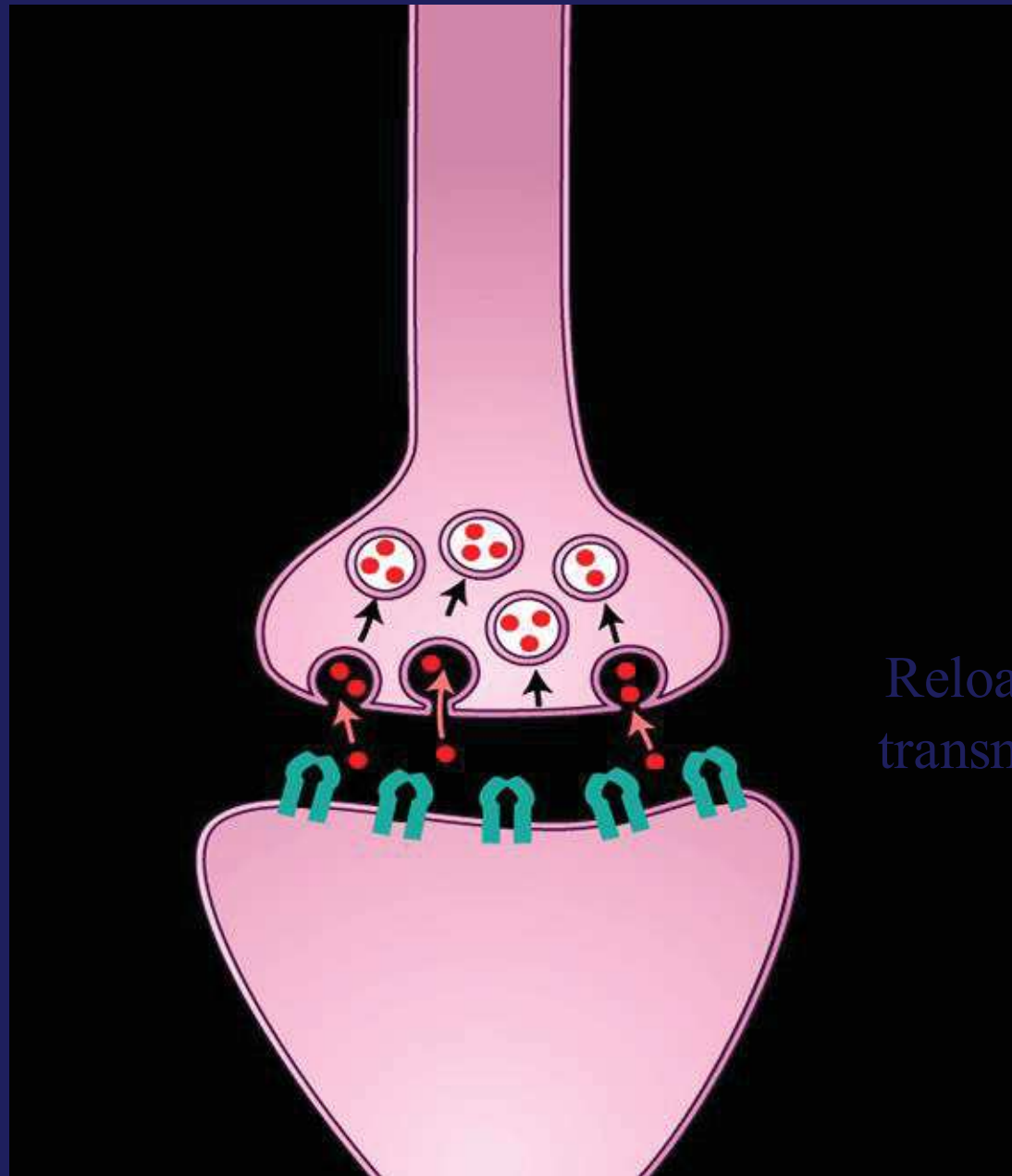




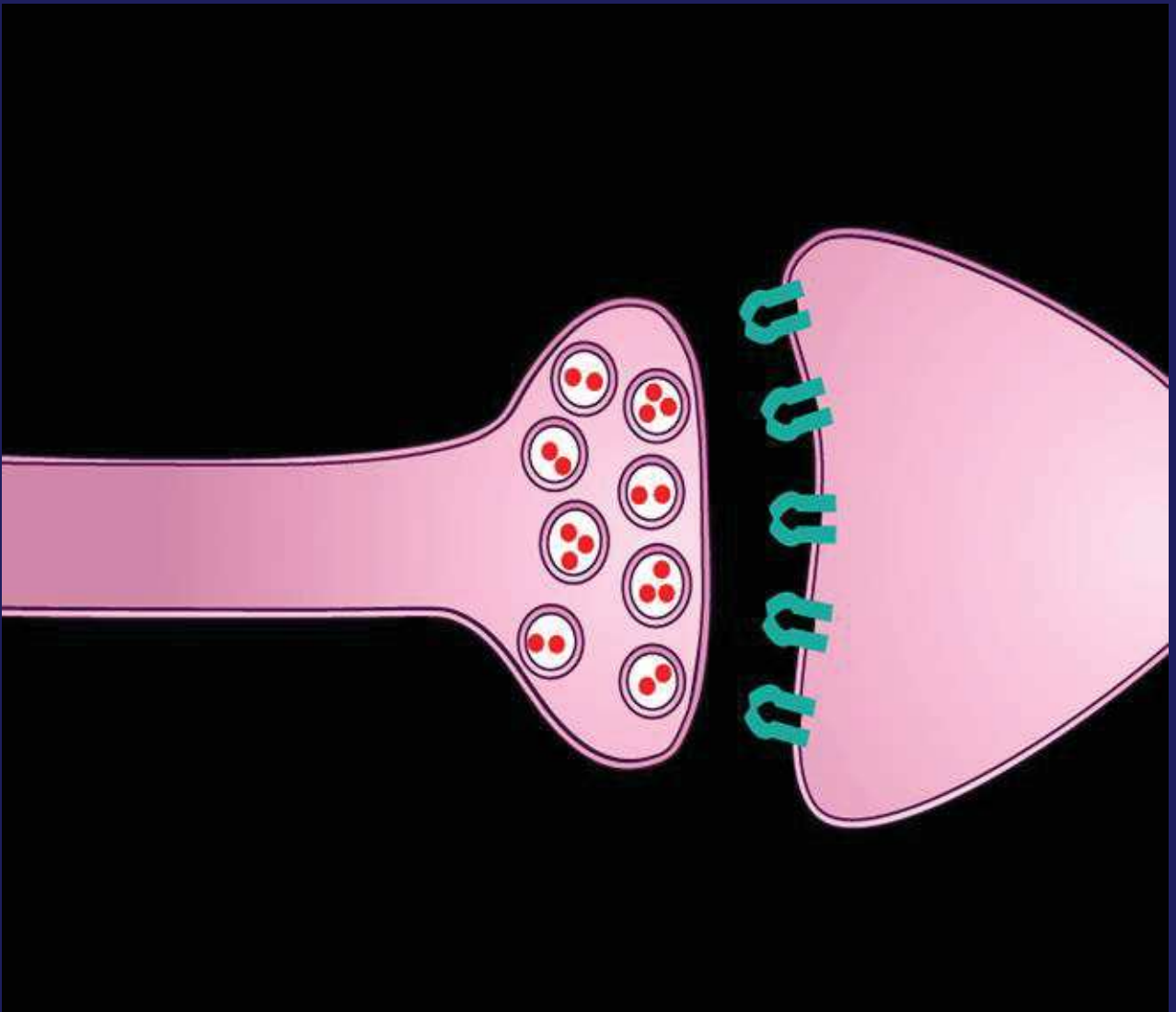
Rele  
trans



Message  
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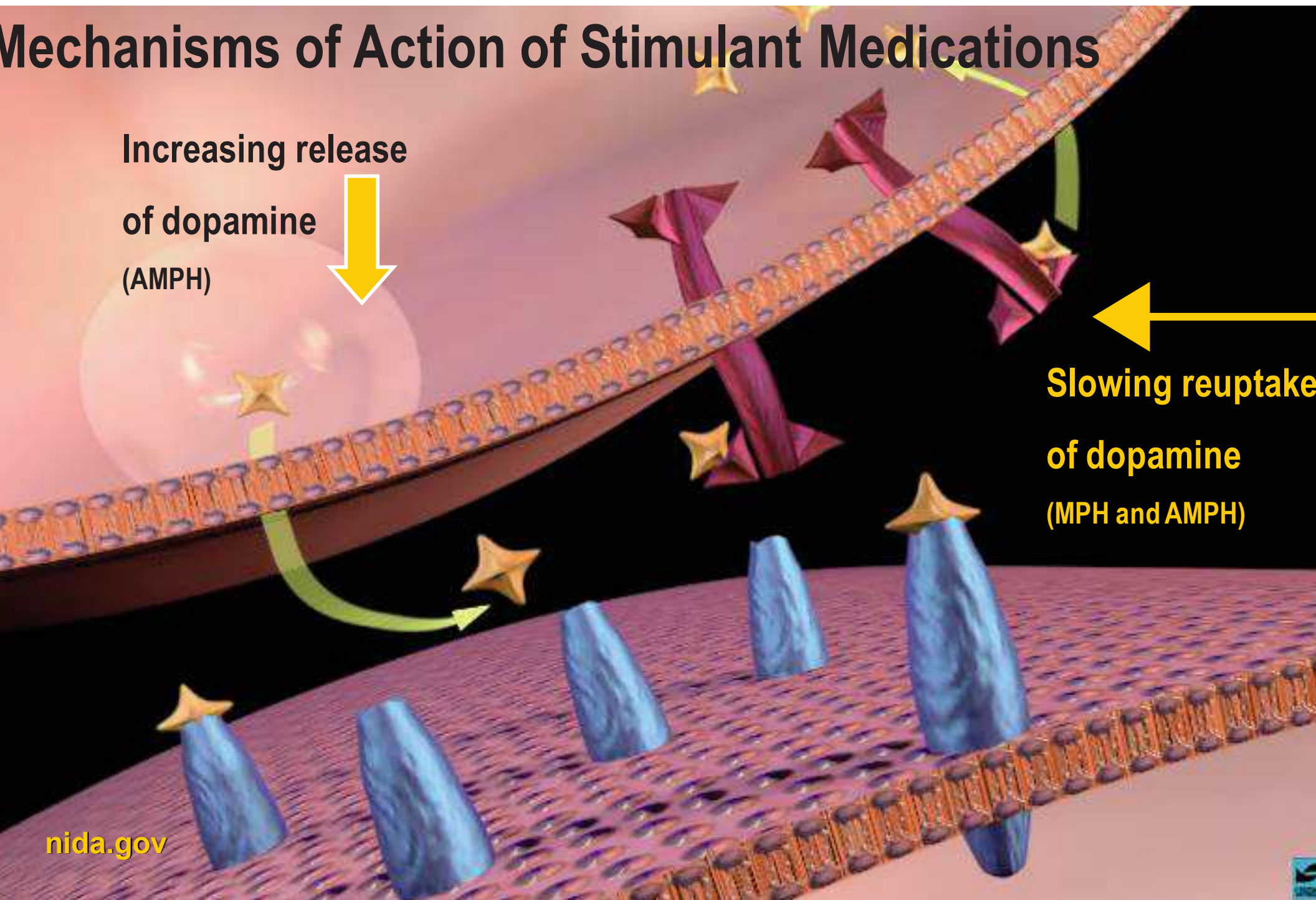


# Mechanisms of Action of Stimulant Medications

Increasing release  
of dopamine  
(AMPH)



Slowing reuptake  
of dopamine  
(MPH and AMPH)



# How do ADHD Impairments of EF Usually Respond to Medication?

- ◆ This wide range of cognitive impairments **responds to medication treatment in 70-90%** of cases in children, adolescents and adults
- ◆ Symptom **improvement varies from modest to very dramatic**
- ◆ Adverse effects are usually transient, not significant



# Set Realistic Expectations for Tx

## Medications do not cure ADHD!

- ◆ Cannot realistically promise “there will be no problematic effects” for any medication for any disorder.
- ◆ Cannot realistically promise that medication will effectively treat ADHD.  
~80% success rate w/stims
- ◆ Close prescriber-patient collaboration is essential for “fine-tuning”

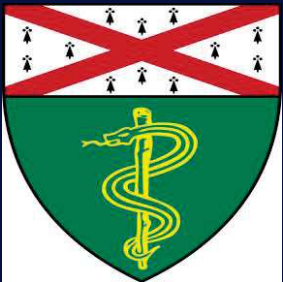
# Key Points

1. Essential problem in ADHD is developmental **impairment of EF**
2. Those with ADHD **usually can focus well for some tasks**, though not for most others.
3. **Inherited brain differences** underlie ADHD.
4. Those differences can make it **more difficult** for those with ADHD to use EF
5. **Tailored medication** treatment helps 80%

# ADHD Comorbidity With Learning Disorders and Autism Spectrum Disorders

Thomas E. Brown, PhD

Associate Director,  
Yale Clinic for Attention and Related Disorders  
Department of Psychiatry  
Yale Medical School



# ADHD Is a Complex Disorder Often Complicated by Comorbidity

- ◆ In 50-70% of cases, ADHD is further complicated by one or more additional psychiatric or learning disorders
- ◆ Not only is it possible to have another disorder with ADHD, **it is 6 times more likely** in lifetime than for those without ADHD

Pliszka SR, et al. *ADHD with Comorbid Disorders*; 1999. Brown TE. *Attention-Deficit Disorders and Comorbidities in Children, Adolescents, and Adults*; 2000.

# Types of Comorbidity

1. Cross-sectional (within past 6-12 mos)
2. Lifetime (ever within entire life)
3. Dynamic (waxing and waning)
4. Subthreshold (impairing w/o full criteria)

# Comorbidity of ADHD in Children

n=61K, ages 6-17 yrs (Larson, et al, 2011)

Disorder	ADHD%	Non-ADHD%
Learning Disorder	46.1	5.3
Conduct Disorder	27.4	1.8
Anxiety	17.8	2.1
Depression	13.9	1.4
Speech Problem	11.8	2.5
Autism Spectrum	6.0	0.6

ADHD prevalence = 8.2% No. of Comorbid Disorders: 33% = 1, 16% = 2, 18% = 3 or more

# Lifetime Psychiatric Disorders in Adolescents (13-18 yrs) (n=10,123)

◆ Any mood disorder	14.3%
◆ Any anxiety disorder	31.9
◆ Any behavior disorder	19.6
◆ Any substance use disorder	11.4
◆ Eating Disorders	2.7
◆ Any disorder	49.5%

1 class: 58% 2 classes: 24% 3+ classes: 18%

# Psychiatric Comorbidities in adults with ADHD

	12 mo.		Lifetime	
	%	OR	%	OR
Any mood	25.5	3.5	45.4	3.0
Any anxiety	47.0	3.4	59.0	3.2
Any substance	14.7	2.8	35.8	2.8
Any impulse <sup>1</sup>	35.0	5.6	69.8	5.9
Any psychiatric	66.9	4.2	88.6	6.3



(<sup>1</sup>impulse = antisocial pd, ODD, CD, Intermittent explosive disorder, bulimia, gambling)

(from Ntnl Comorbidity Survey-Replication data presented by R.Kessler at APA, 5/1/04)



# Comorbidity in NCSR

- ◆ Included any disorders at any point in entire lifetime
- ◆ Included only 18-44 year old adults
- ◆ Did not include learning disorders
- ◆ Based on self-report of sx

# PUZZLING QUESTIONS!

Why are there such high rates of comorbidity between ADHD and so many other disorders?

Why is an adult with ADHD 6 times more likely to have at least one additional DSM-IV disorder at some point in life?

# “Fruit Salad” Theory of Comorbidity

- ◆ Each of 200+ disorders in DSM is seen as a discrete entity—like a separate tree producing its own fruit
- ◆ Comorbidity is seen as chance convergence of genetics
- ◆ No recognition of overlap between disorders or hybrid variants

# Problems with Categorical Dx (Cloniger, 1999)

- ◆ “There is no empirical evidence for natural boundaries between major syndromes...No one has ever found a set of sx, signs or tests that separate mental disorders into non-overlapping categories...the categorical approach is fundamentally flawed.”

# Usefulness of Categorical Diagnosis

(Kendell & Jablensky, 2003)

“Diagnostic categories provide invaluable information about the likelihood of future recovery, relapse, deterioration, and social handicap; they guide decisions about tx; and they provide a wealth of information about similar patients in clinical populations or community surveys throughout the world.”

# Fuzzy Boundaries Between Disorders (Pennington, 2002)

“...disorders are likely **regions with fuzzy boundaries** in continuous, multivariate space. What will likely distinguish disorders is the **weighting of different risk factors**, not a distinct set of risk factors, and the different epigenetic and developmental **interactions** that result from that particular weighting.”

\*\*\*\*\*

**We do not treat categories; we treat patients.**

# Impairments of EF can have wide impact upon mind

- ◆ “without basic attention and working memory there is no prospect of coherent mental activity...” (1)
- ◆ “...attention serves all the other functions.” (2)
- ◆ Exec Functions are the “operating system” of the mind

<sup>1</sup>Damasio (1994); <sup>2</sup>Fuster (2003)

# An Alternative Theory of Comorbidity

- ◆ ADHD = developmental impairment of executive functions
- ◆ ADHD is not just one disorder among many
- ◆ ADHD is a foundational disorder that cross-cuts other disorders
- ◆ ADHD increases risks of other disorders



# Quantitative vs Qualitative Differences

“...some differences between ADHD and other disorders may be quantitative rather than qualitative.”

(Banaschewski, Hollis, Oosterlaan, et al. 2005)

# How does ADHD increase risk?

- ◆ **Adaptive impairments of ADHD expose to environmental risks**, eg. Poor school achievement → drop into peer group less motivated for school and higher exposure to substance abuse
- ◆ **Genetic impairments of ADHD may be associated with heightened risk for other genetic problems**, e.g. reading disorder and ADHD have some common genetic risk of slow processing speed

# DSM-5 Specific Learning Disorder

- ◆ Difficulties in learning & using academic skills
- ◆ Affected skills substantially and quantifiably below expected for age
- ◆ Learning difficulties started in school-age yrs, but may not be fully manifest until challenged
- ◆ Not better accounted for by other factors

# DSM-5 LD Subtypes

## 315.00 Impairment in reading (dyslexia)

(Word reading accuracy, fluency, comprehension)

## 315.2 Impairment in written expression

(spelling, grammar & punctuation accuracy, clarity or organization of written expression)

## 315.1 Impairment in mathematics (dyscalculia)

(number sense, memorization of arithmetic facts, accurate or fluent calculation, accurate math reasoning)

# LD in ADHD Children

## Clinical Sample of ADHD Children:

Reading LD	27%
Math LD	31%
Written Expression LD	65%
One or more LD	70%

(Mayes, Calhoun, Crowell, 2000)

## Epidemiological Sample:

CDC National Health Interview Study:

50% of ADHD-diagnosed children are also identified as having LD.

(CDC, 2002, 2008)

# ADHD vs LD

## Separate entities?

Some think:

**ADHD** = chemical problem in brain

**LD** = “hard-wiring” problem in brain

ADHD and LD may not be so separate

- **shared genetic etiology in Reading Disorder and ADHD**  
(Willcutt, Pennington & DeFries, 2000)
- **ADHD & LD both involve EF**, esp working memory

# Reading Disorder

- ◆ core is phonological processing deficit
- ◆ knows words when heard, but can't recognize them in written form
- ◆ normally distributed in population
- ◆ boys and girls equally affected
- ◆ linked to chromosomes 6 & 15
- ◆ early language problems predict RD

# Overlap btwn ADHD & Reading

- ◆ Fluency and comprehension in reading requires:
- ◆ not only ability to decode words
- ◆ but also ability to sustain focused attention
- ◆ Medications used to treat ADHD may be helpful in treatment of dyslexia



# Executive Skills in Reading Comprehension

(Sesma, Mahone, et al, 2009)

“Reading comprehension is more complex than single word reading, with demands that go beyond phonological decoding and word identification to include higher order cognitive processing of meaning conveyed through sentences and paragraphs.

Executive skills... become more necessary as length & complexity of text increases.”

# EF in Reading Comprehension

(Brown, Reichel, Quinlan, 2011)

“Most of the time when I’m reading assignments in my textbooks, I’m just licking the words rather than chewing them. That’s why I have to keep going back to read it all over again.”

Many students report that often such impairments are not present in self-selected reading rather than assigned texts.

# EF in Reading Comprehension

(Brown, Reichel, Quinlan, 2011)

“Executive functions often impaired in ADHD, especially processing speed and working memory, play an important role in reading, particularly in reading fluency and comprehension.”

# Study of Reading Comprehension & Time Pressures in Students with ADHD

145 students aged 13-18 yo

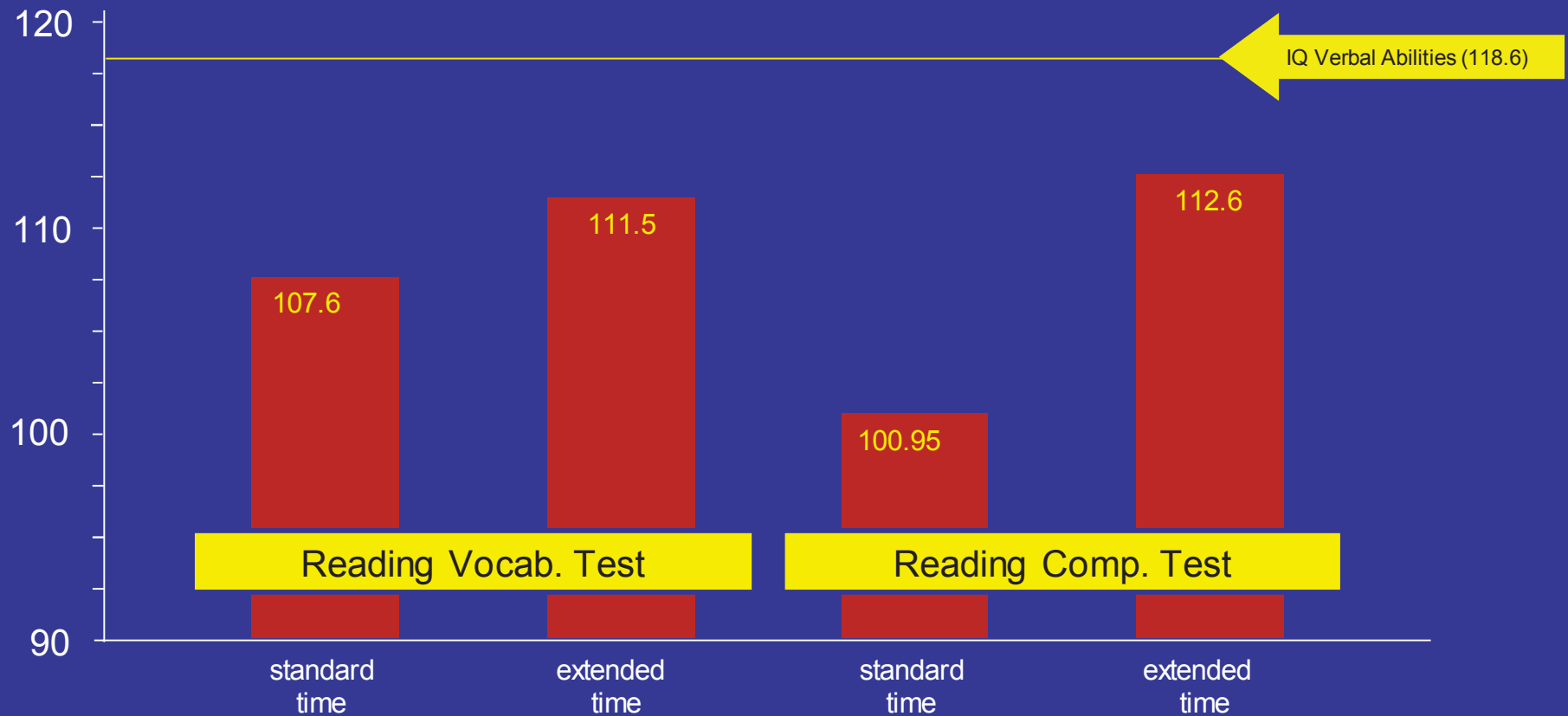
On WISC-IV or WAIS-III IQ Test:

- ◆ Verbal Comp Index = 118.6
- ◆ Working memory Index = 102.8
- ◆ Processing speed index = 99.9
- ◆ Basic reading skills = 106.5

(Brown, Reichel, Quinlan, 2011)

## Standard vs Extended Time vs IQ Verbal Abilities

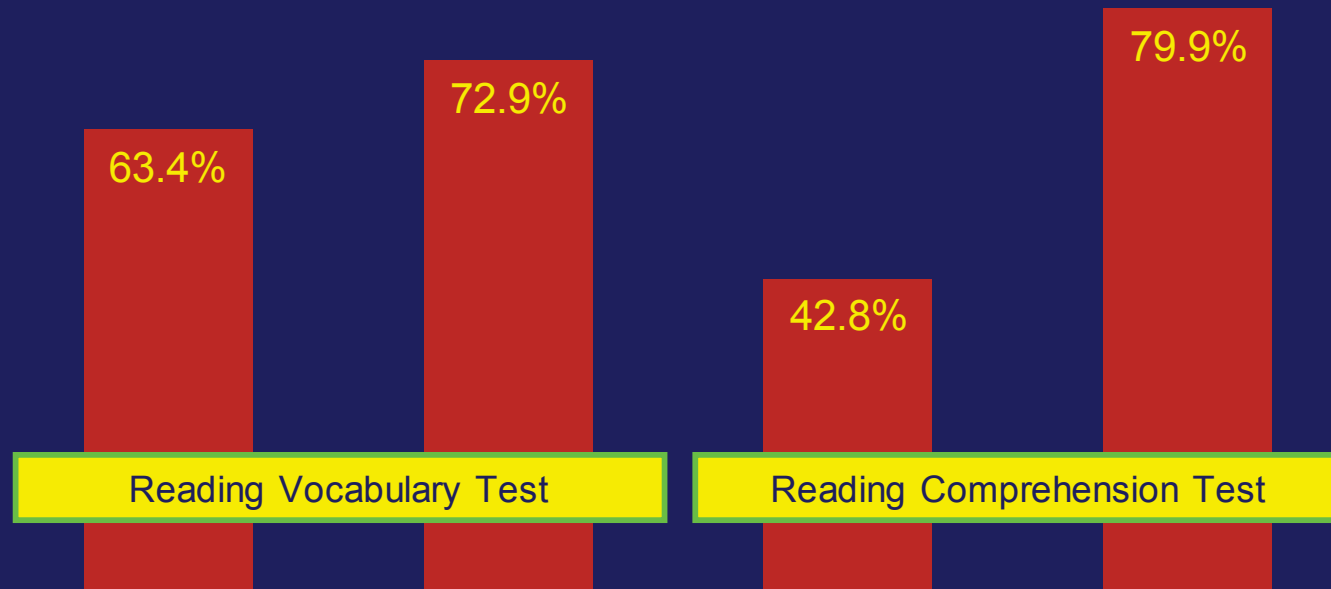
Group Means



$p = < .0001$  for paired t-tests on both comparisons of standard vs. extended time

# Percentage of Subjects Scoring within 1 SD of their IQ Verbal Ability Index

Standard Time vs. Extended Time



# Disorder of Written Expression

Significant impairment in writing grammatically correct sentences & organized paragraphs; often, but not

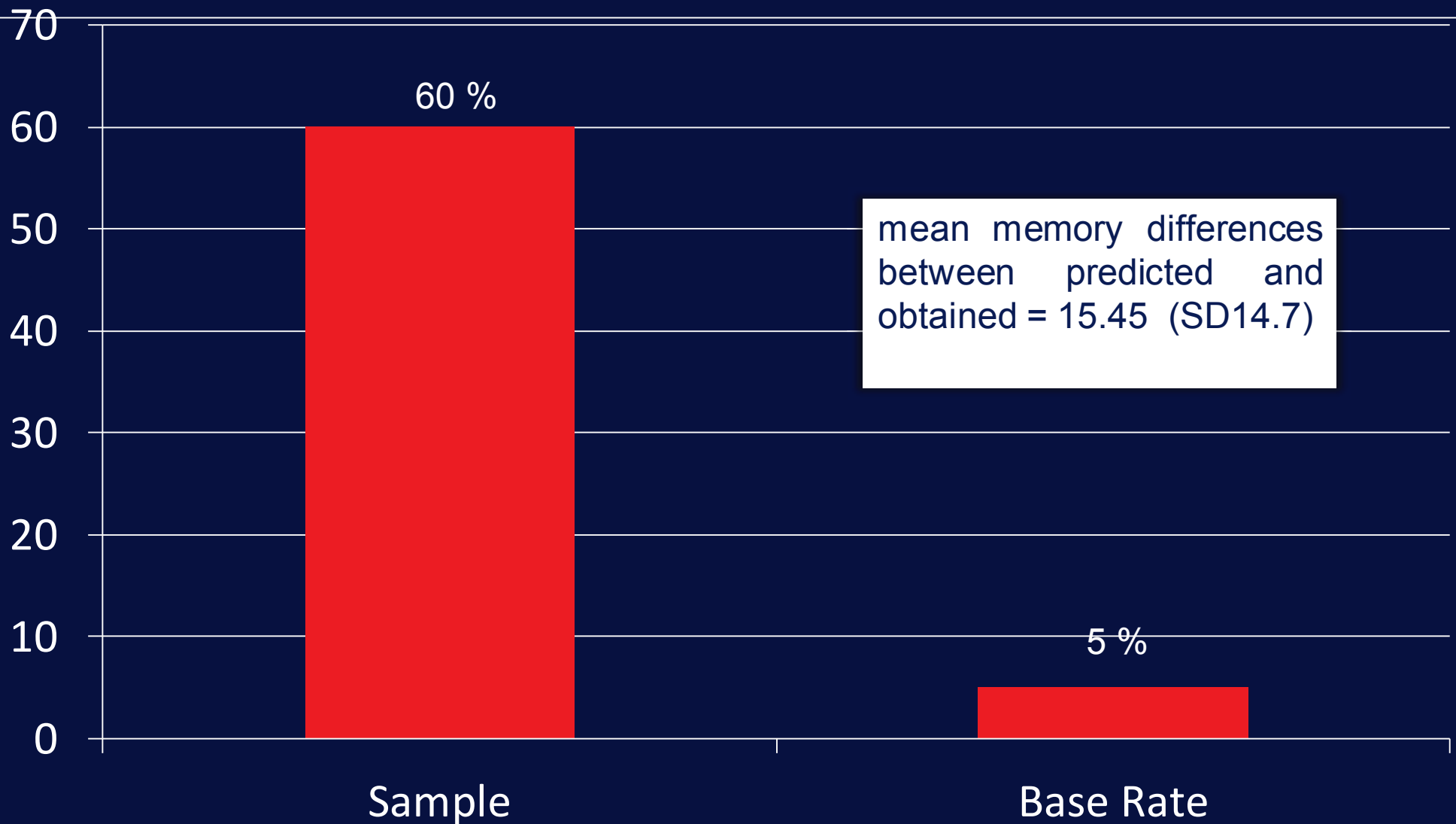
always, poor spelling

chronic difficulty in what to write, organizing ideas, and elaboration

oral expression may be adequate or strong

while weak in written expression

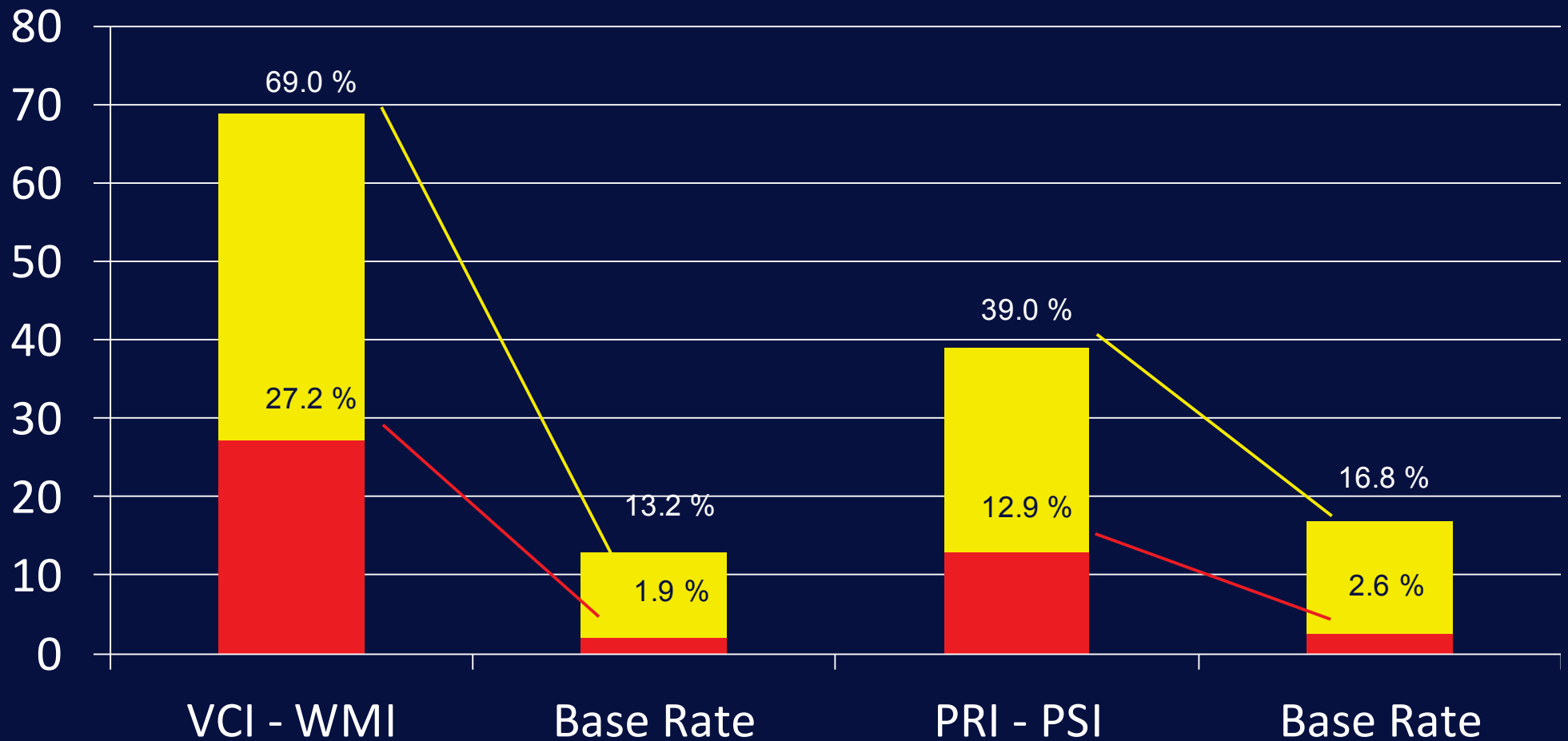
Written Expression Subtest;  
percentage of participants scoring  $\leq 1$ SD of score predicted by VCI  
n = 53





# Wechsler IQ Index Score Weaknesses in WMI/PSI vs. VCI/PRI

% of participants with 1 or 2 SD discrepancy relative to standardization sample  
n = 53



# Assessment for Learning Disorders

- ◆ Always screen students w/academic probs
- ◆ Query re: hx of chronic difficulties  
in specific courses modalities?  
adequate instruction?
- ◆ WJ-III or WIAT-III Achievement Tests  
Reading - Math - Writing
- ◆ Nelson-Denny Reading Test ( $\geq 9^{\text{th}}$  grade)

# Pills and Skills

- ◆ Careful diagnostic workup needed to identify needs for LD remediation:

**Is there lack of skills and/or deployment?**

- ◆ Basic skills deficits in reading, math or written expression may require special education services or tutoring
- ◆ Treatment of ADHD may make student more available to learn

# DSM-5 Autism Spectrum Disorders

- ◆ Persistent deficits in social communication & social interaction across multiple contexts:

(social-emotional reciprocity, non-verbal communication, developing, maintaining & sustaining relationships)

- ◆ Restricted, repetitive patterns of behavior:

(stereotypies, insistence on sameness, restricted-fixated interests, hyper or hypo reactivity to sensory input)

# Autistic Traits in Children with ADHD

(Grzadzinski, Di Martino, Brady, et al. 2011)

Parent reports re 75 ADHD children 7-17 yo rated on Social Responsiveness Scale (SRS):

32% identified as ADHD + ASD

Those with ADHD+ did not differ on sx of ADHD, anxiety or IQ

Ratings of ODD were higher in ADHD+

# Overlap Between ASD and ADHD

Latent Class Analysis n=644 (5-17 ys)

Children w/ ASD and ADHD are heterogeneous group

- ◆ All children with ASD also had some ADHD sx
- ◆ The most severe cases of ADHD also had most severe ASD sx

In other studies:

up to 80% of those with ASD also have ADHD sx

up to 50% of those with ADHD also have ASD sx

(Van der Meer, Oerlemans, van Stein, et al, 2012)

# Autism Spectrum Disorders with ADHD

- ◆ In a clinical sample:
- ◆ 76% of High Functioning ASD youths had ADHD with similar presentation and severity. Other studies report 59% to 83%.
- ◆ Yet many had received no ADHD treatment  
(Joshi, Faraone, Wozniak, et al. 2014)
- ◆ If signif. ADHD sx in ASD, consider ADHD tx

# Genetic Overlap of ADHD and ASD

(Martin, Cooper, Hamshere, et al., 2014)

- ◆ Twin studies have shown that **shared inherited factors explain much of the strong comorbidity between ADHD/ASD**
- ◆ Recent genetic studies suggest that rare chromosomal deletions and duplications (**CNVs: copy number variants**) in **ADHD kids overlap with those in ASD kids.**



# Lifespan Perspective

- ◆ In considering impact of overlap between ADHD and LD, ASD, it is important to consider **not only impact during childhood, but also during adolescence and adulthood**. Impact may be **reduced and or increased over time**, depending upon **context and supports**.

eg **LD** jobs, social situations, parenting

eg **ASD** when parents are unable to help

# Medications for ADHD + ASD

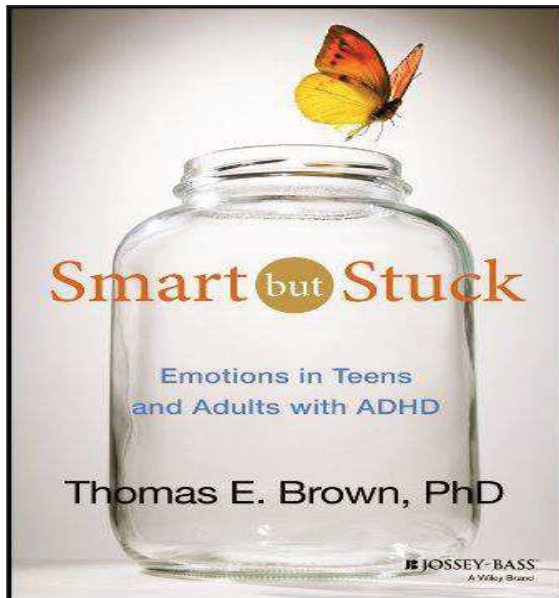
Archives of Gen Psych 2005;61,1266-1274)

Few controlled studies, most with MPH  
RUPP study of MPH in 72 PDD (5-14 yo)  
Wt based dosing!? ES: 0.20 – 0.54  
49% were responders to MPH  
18% started MPH but DC due to ae  
Often efficacious, but more adverse  
effects than in TDC.

# Complicated ADDs

- ◆ Expect complications in >50% cases
- ◆ complicating factors often interact
- ◆ family stress: contributory & reactive
- ◆ individual probs may mask other probs
- ◆ setting may make big difference +/-
- ◆ monitor meds carefully, ?change/combine
- ◆ attend to health as well as illness
- ◆ improvement is often slow and mixed

## Books by Thomas E. Brown, Ph.D. ([www.DrThomasEBrown.com](http://www.DrThomasEBrown.com))



- “Smart but Stuck: Emotions in Teens and Adults with ADHD ” – 2014
- “A New Understanding of ADHD in Children and Adults: Executive Function Impairments” – 2013
- “ADHD Comorbidities: Handbook for ADHD Complications in Children and Adults” – 2009
- “Attention Deficit Disorder: The Unfocused Mind in Children and Adults” - 2005

