Alcohol and the adolescent brain:

Why delaying the onset of drinking is so important

Aaron White, PhD

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National Institute on Alcohol Abuse and Alcoholism
National Institutes of Health

EUDL Symposium – August 8th, 2012

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Excessive drinking during adolescence is a global concern

Telegraph.co.uk

Britain's 'chronic' teenage binge-drinking problem highlighted by European poll

Britain's "chronic" teenage binge-drinking problem has been highlight
European poll.

Last Updated: 1:40PM GMT 26 Mar 2009

The survey of 35 countries found the UK had the third-highest number of 15 and 16-year-olds with an alcohol problem.

Professor Martin Plant, the University of the West of England academic who led the research, said: "The UK retains its unenviable position in relation to both binge-drinking, intoxication and alcohol-related problems among teenagers.

Binge-drinking: The survey of 35 countries found the UK had the third
16-year-olds with an alcohol problem Photo: PA

A woman lies on a bench after leaving a bar in Bristol City Centre on October 15, 2005 in Bristol, England. (Matt Cardy/Getty Images)

Binge drinking comes to France

By David Chazan
BBC News, Paris

Australia battles rise in alcohol abuse

By Phil Mercer
BBC News, Sydney

Alcohol increasingly has Australia in its poisonous grip.

One in eight Australians drink at dangerous levels. The effects on long-term health are likely to be catastrophic.

Experts say alcohol is damaging the health of Australians

Australia has always had a boozy reputation but excessive drinking is on the rise.

Doctors are warning of a surge in chronic diseases such as cirrhosis of the liver and cancers as well as brain disorders in the next 20 years.

"Unfortunately Australia has a massive alcohol drinking problem," said Associate Professor Gordian Fulde from Sydney's St Vincent's Hospital.

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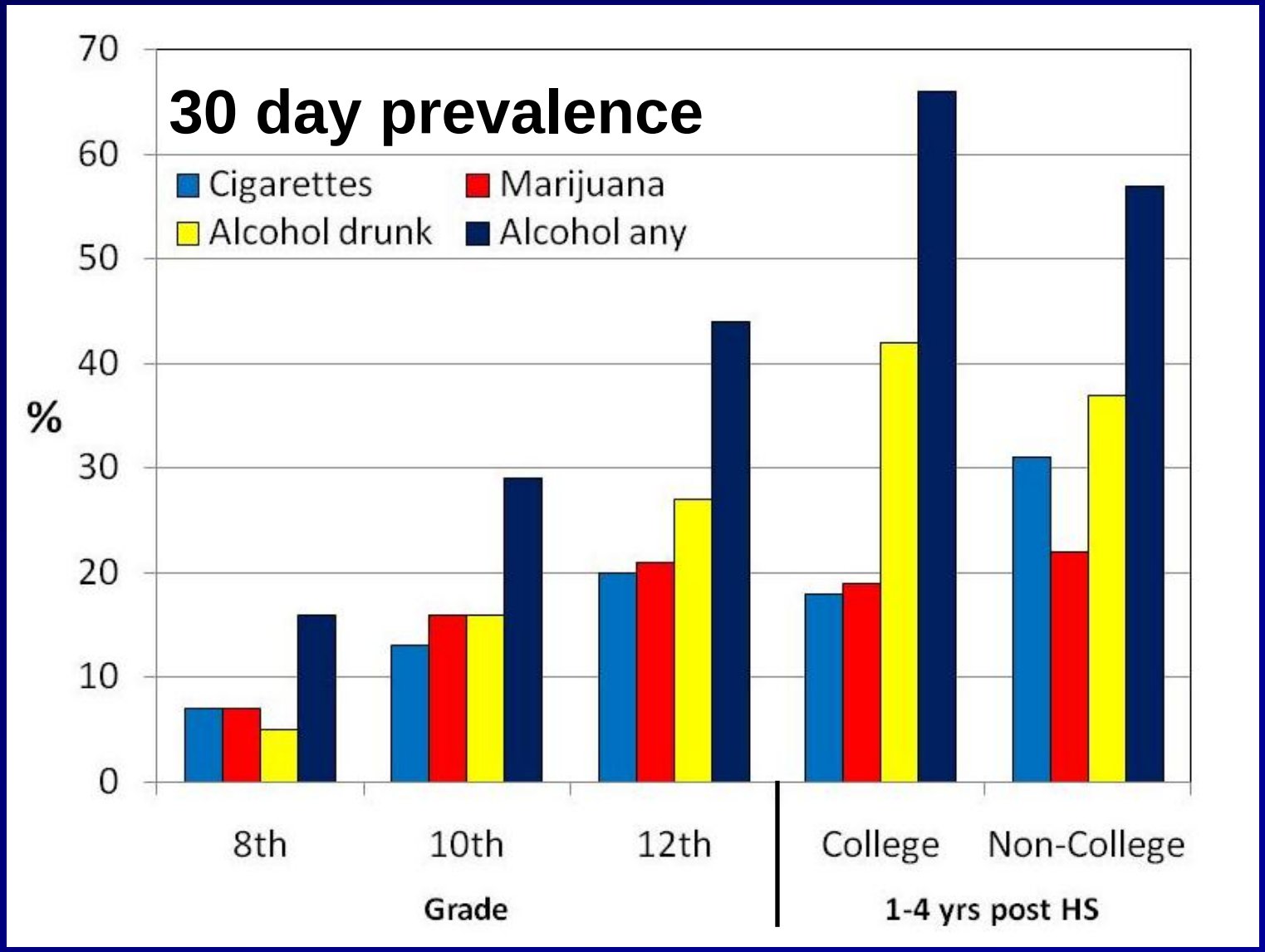
Alcohol is the drug of choice among youth in U.S.



Source: Monitoring the Future, 2010

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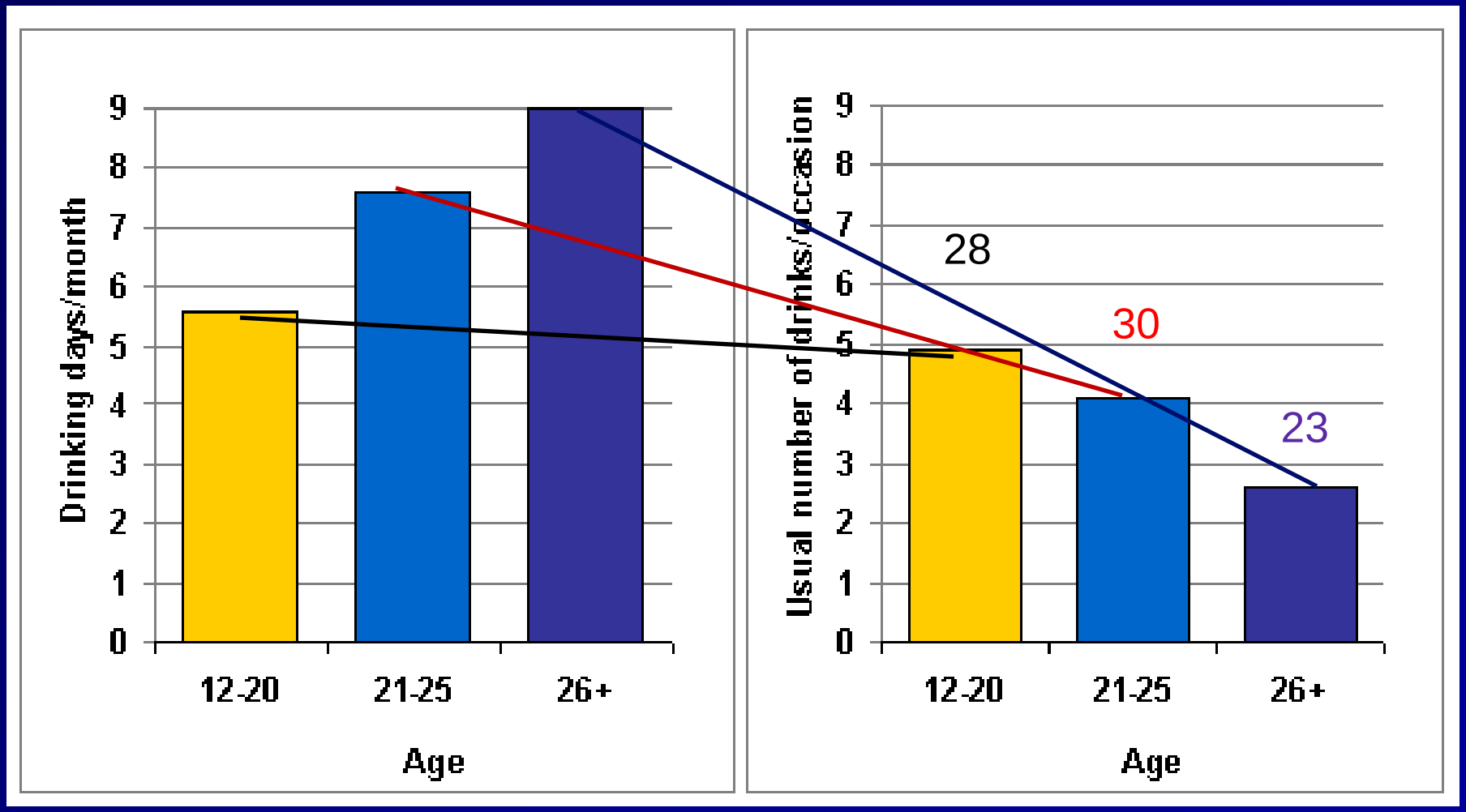
Adolescents drink less often than adults but more per occasion



Source: SAMHSA Report to Congress on Prevention and Reduction of Underage Drinking, May 2011

Risk of alcohol abuse and dependence peaks in late adolescence/young adulthood

Percentages of Persons Aged 12 or Older Who Met the Criteria for Alcohol Dependence or Abuse in the Past Year, by Age Group: 2002, 2003, and 2004

Age Group	Percentage
12 to 17	5.9
18 to 25	**17.4**
26 to 34	11.1
35 to 49	7.5
50 or Older	3.0

Source: The NSDUH Report, Issue 16, 2006, SAMHSA

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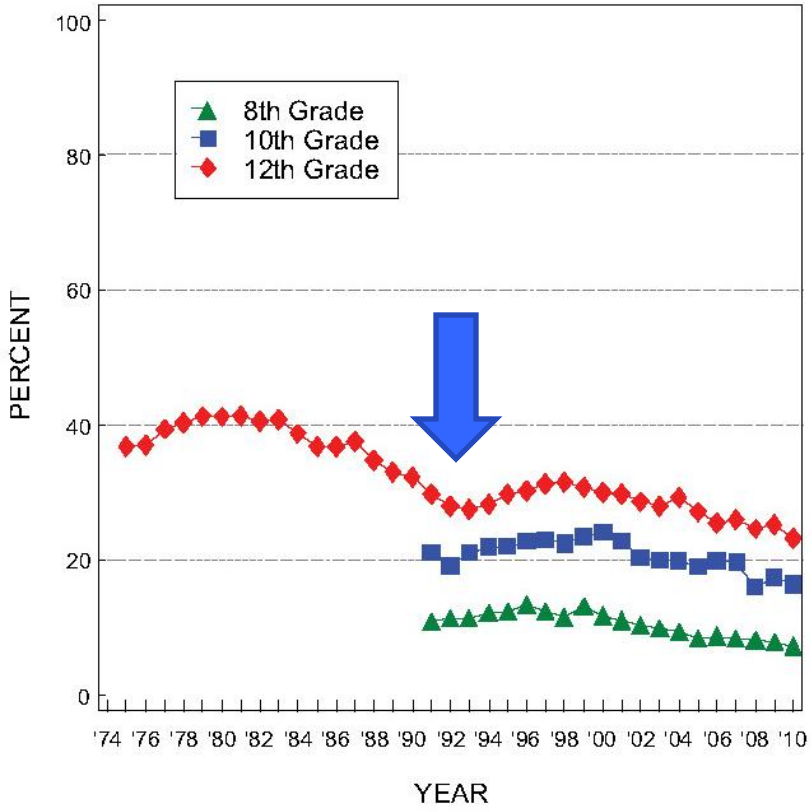
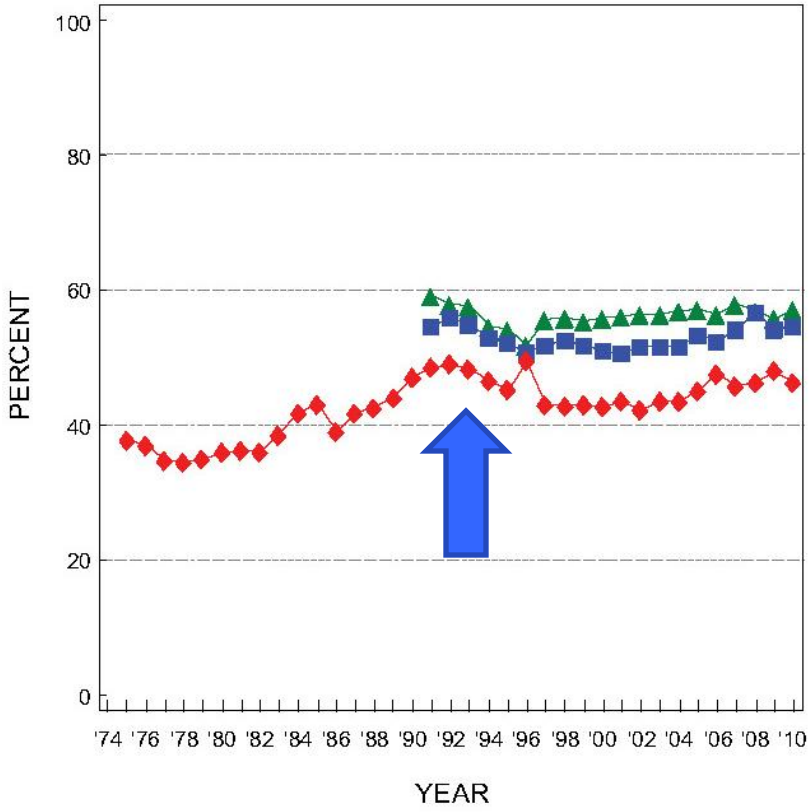
U.S. – Decrease in binge drinking

Binge (5+ drinks) past 30 days

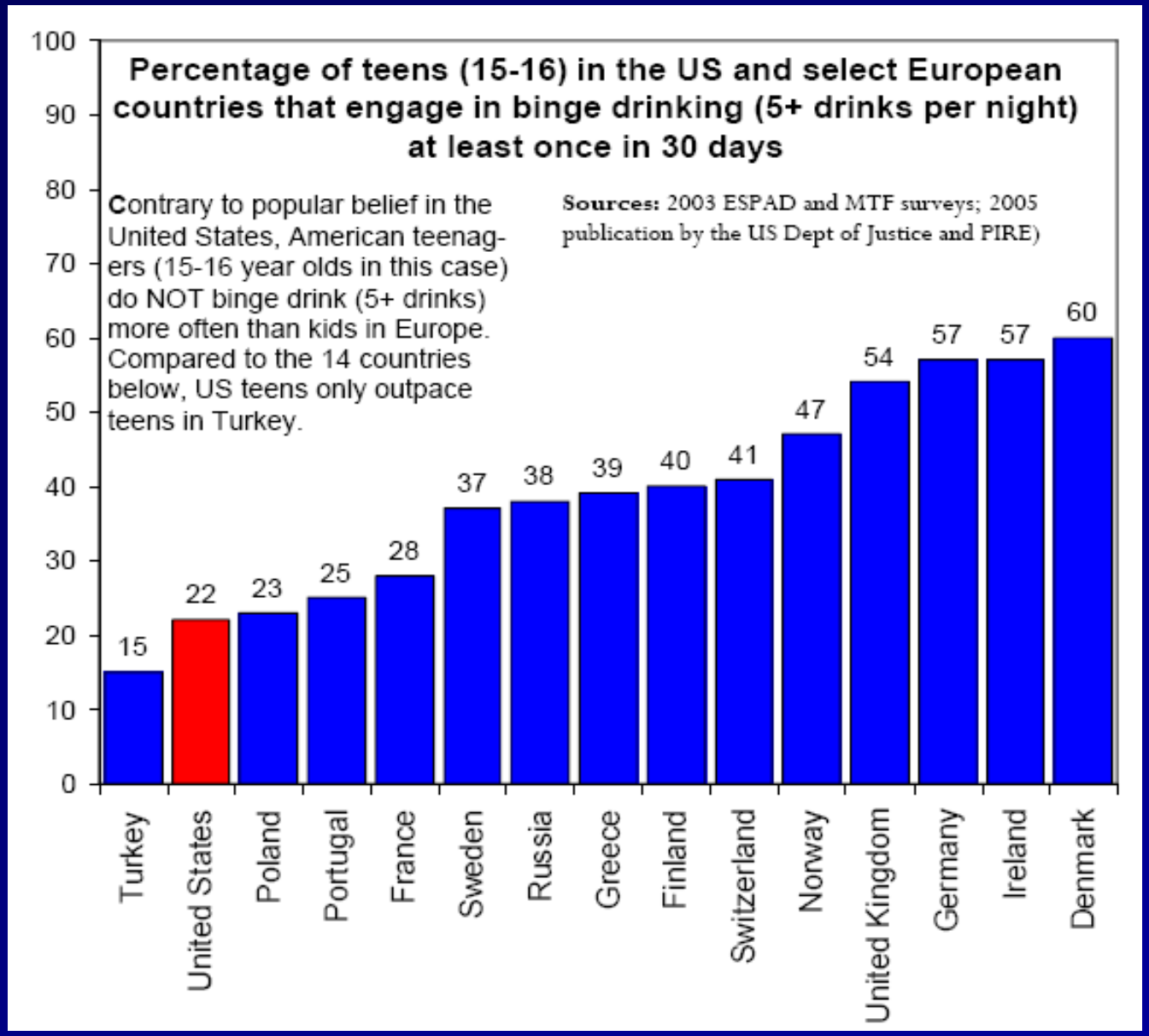


See great risk in binge 1-2 per weekend



Source: Monitoring the Future (2010)

U.S. teens compared to other countries



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Questions:

- What is it about adolescence that increases the likelihood of excessive drinking during this stage of life?
- How do the effects of alcohol on the adolescent brain help explain the **short-term** and long-term consequences of alcohol use?
- What do the findings suggest about the logic of a legal drinking age of 21?

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Adolescence – What? Why?

- Transition from dependence to independence
- Period of biological, social and psychological changes that prepare us to survive in the adult world
- Tumult (e.g., risk taking, conflict, moodiness) is normal and can be adaptive
- Changes that occur in the brain during adolescence simultaneously get us out into the world to explore and then record experiences to prepare us for independence

Adolescent storm and stress is normal and often adaptive

"Teenage brains. Beautiful brains. Moody. Impulsive. Maddening. Why do teenagers act the way they do? Viewed through the eyes of evolution, their most exasperating traits may be the key to success as adults."

David Dobbs

National Geographic Magazine

October, 2011

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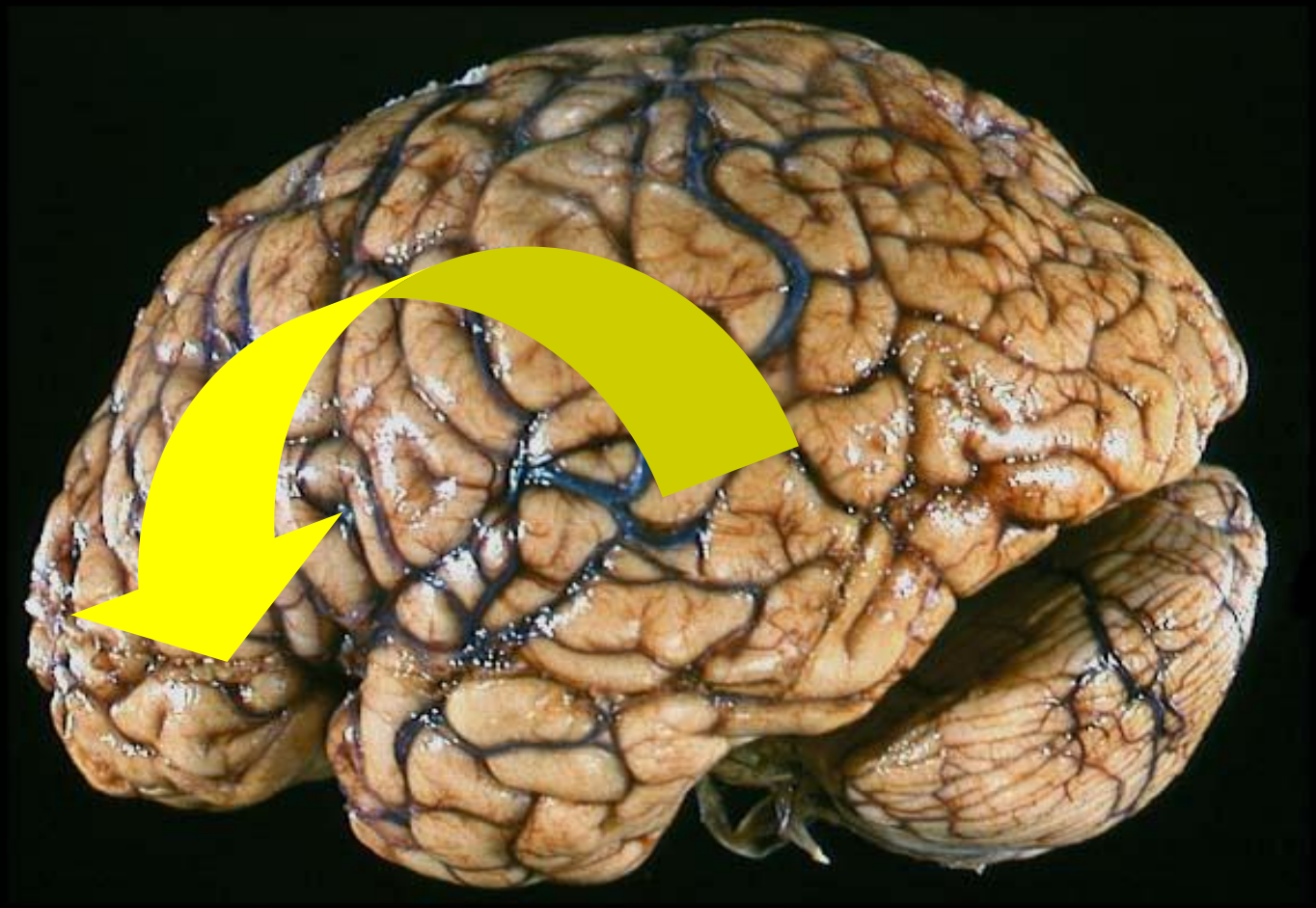
Adolescent brain development



<u>Frontalization of function</u> – Frontal lobes take on more and more organizational control over behavior as adolescence unfolds

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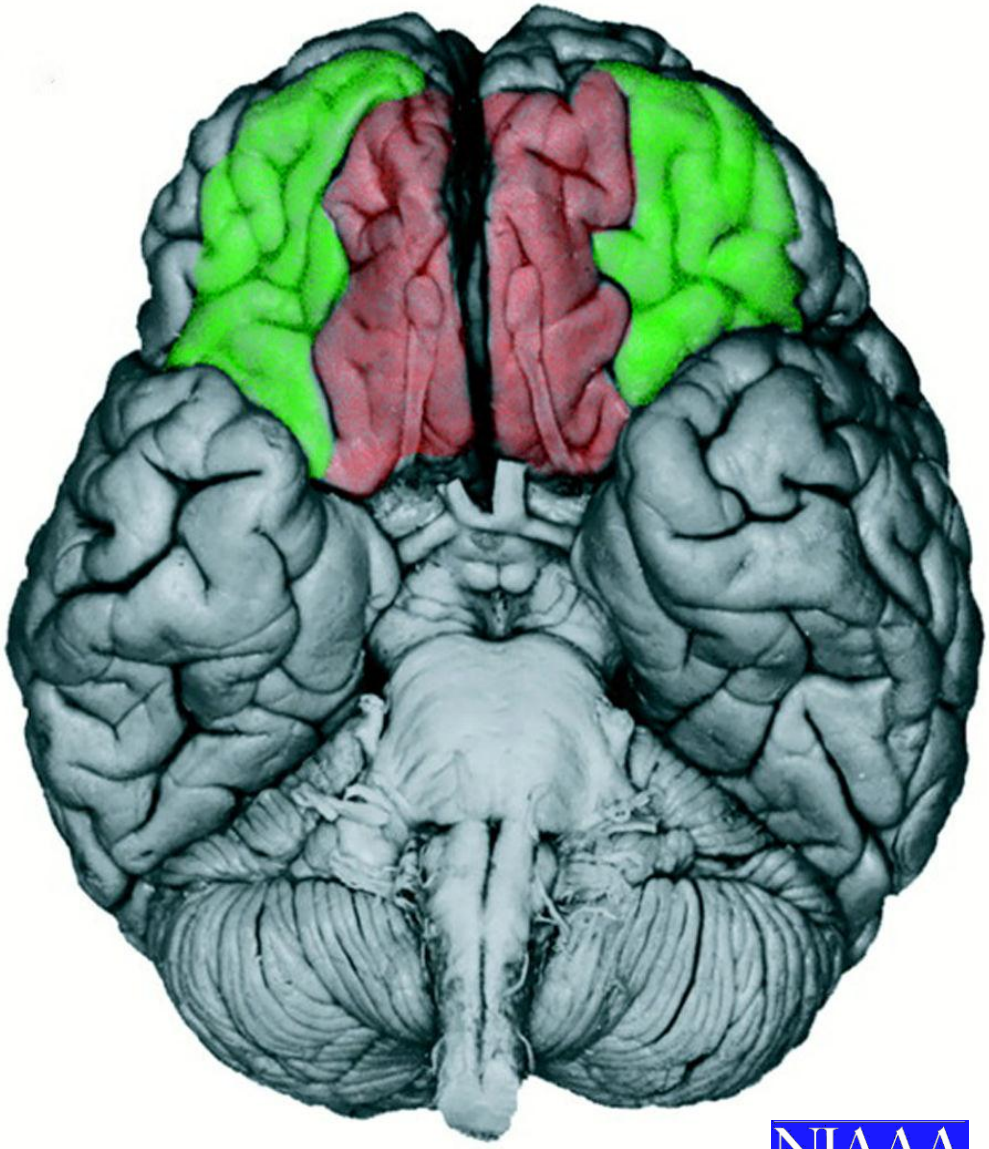
The amazing frontal lobes

- Planning, decision-making, impulse control, memory, language and more
- Remodeled during adolescence

Frontal lobe development during adolescence

Orbitofrontal and Ventrolateral Prefrontal Cortex Development

Schematic depiction of the ventrolateral prefrontal cortex (**vlPFC in green**) and orbitofrontal cortex (**OFC in red**) regions of the VPFC that contribute to the development of social flexibility. Inhibitory control and rule use are controlled by the vlPFC, whereas computation of expected value of social stimuli is performed by the OFC. All three of these functions mature slowly across development. (Adapted from Davidson, Putnam, and Larson (2000), and reprinted with permission.)



Nelson EE and Guyer AE (2011) The Development of the Ventral Prefrontal Cortex and Social Flexibility. Dev Cogn Neuroscience, 1(3): 233–245.

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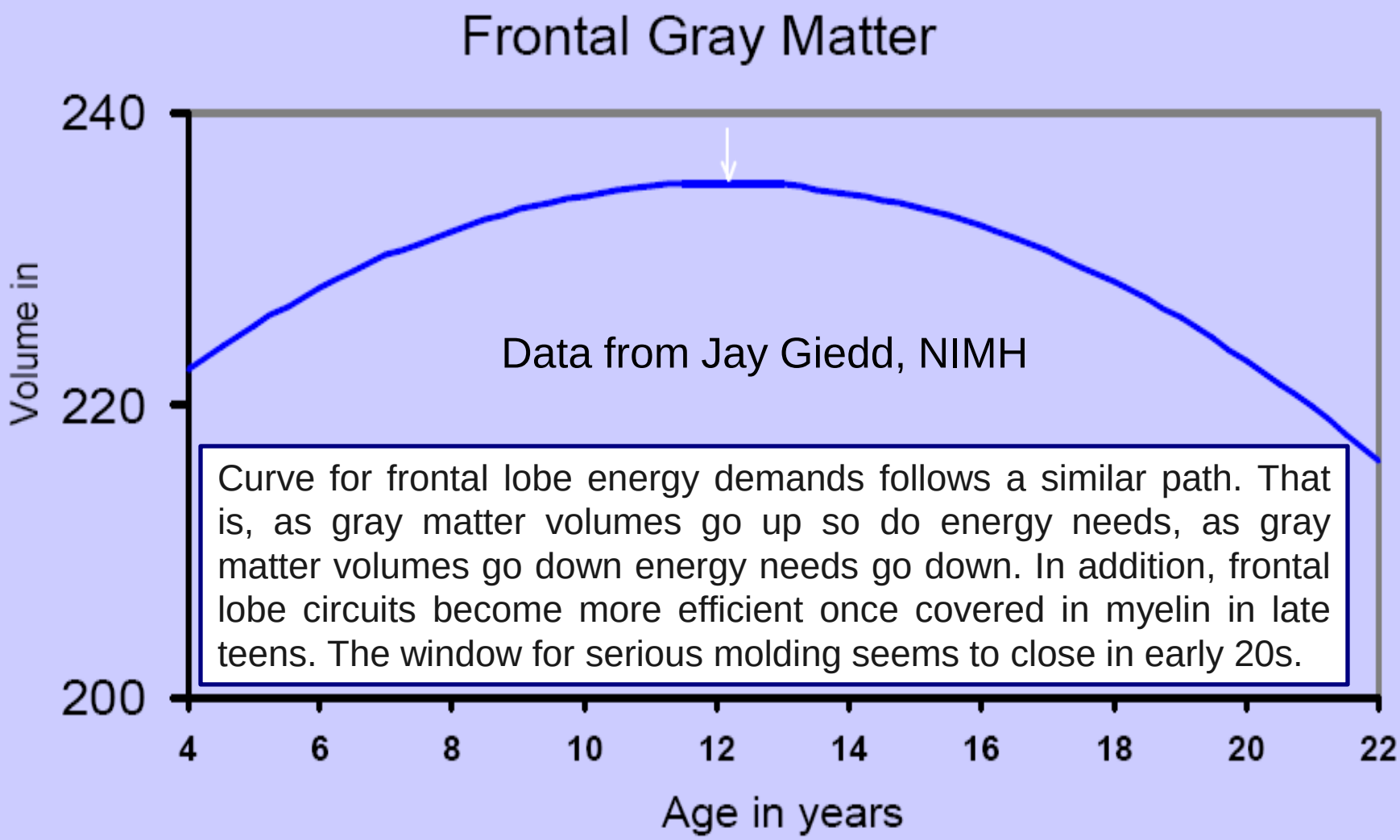
Frontal lobe development



Curve for frontal lobe energy demands follows a similar path. That is, as gray matter volumes go up so do energy needs, as gray matter volumes go down energy needs go down. In addition, frontal lobe circuits become more efficient once covered in myelin in late teens. The window for serious molding seems to close in early 20s.

Reward (monetary) activates ventral striatum more strongly in adolescents than children or adults

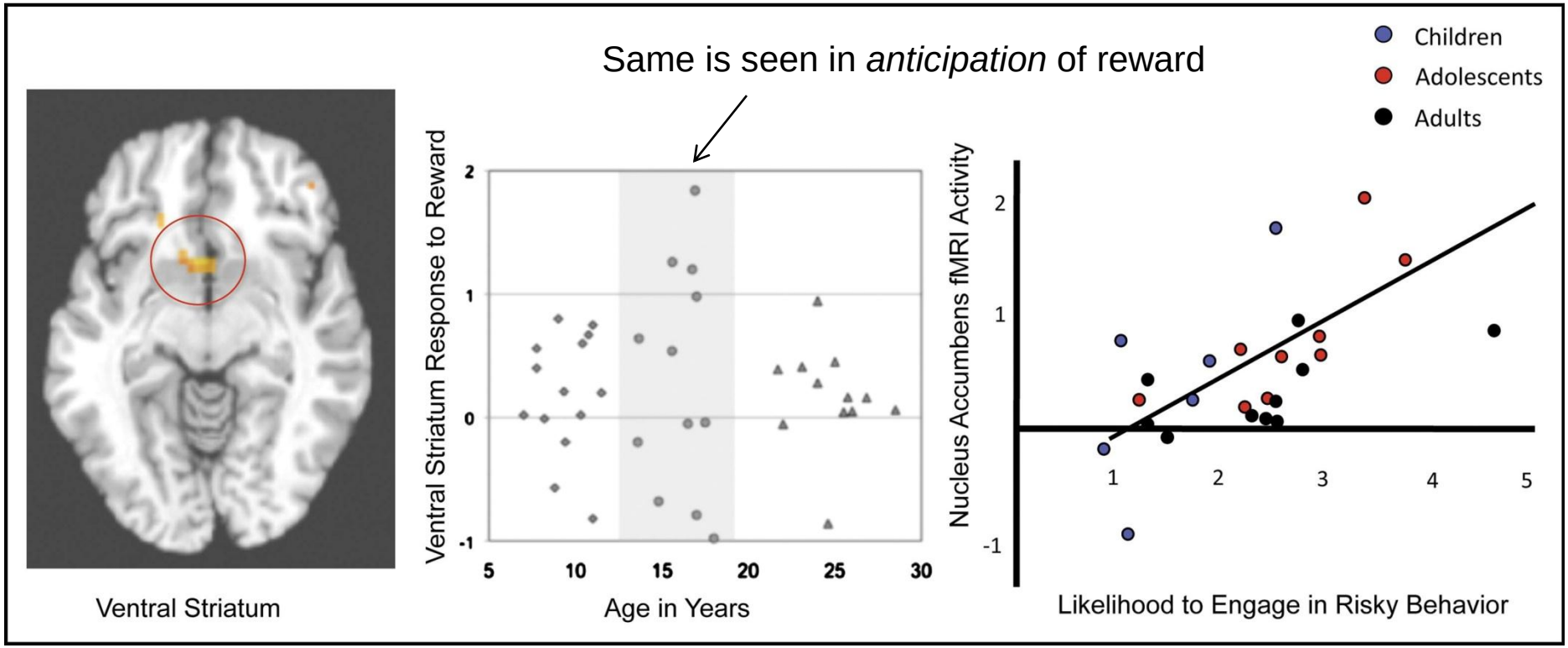


FIGURE 3. Ventral striatal activity to reward and association with risk-taking. Note: Ventral striatum (left) is activated with reward (middle) and correlated with risk-taking (right) (adapted from Galvan et al.[6] and Galvan et al.[16]). Such findings could mean that adolescents are more motivated for reward and more reinforced by it.

SOURCE: Casey BJ, Jones RM. Neurobiology of the adolescent brain and behavior: implications for substance use disorders. J Am Acad Child Adolesc Psychiatry. 2010 Dec;49(12):1189-201;

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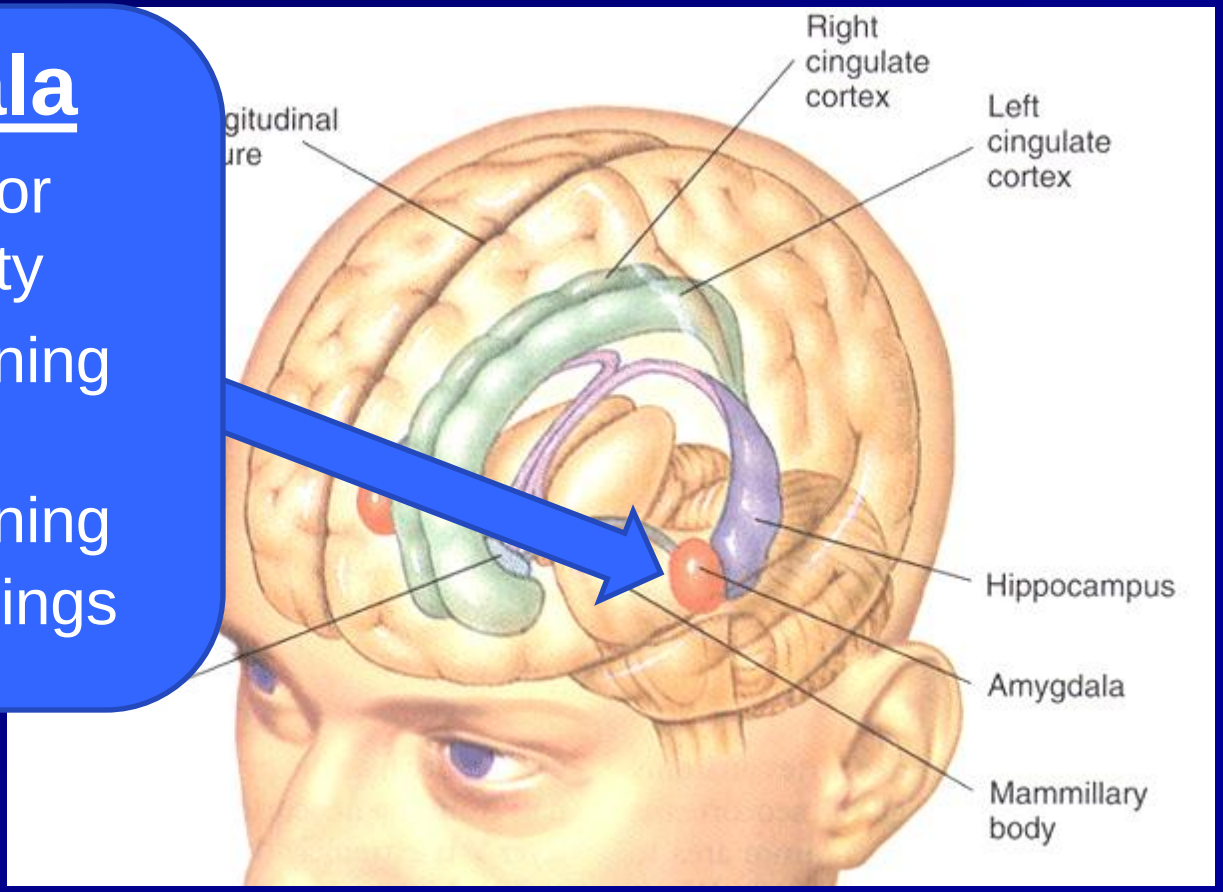
"Stressed out" teenagers

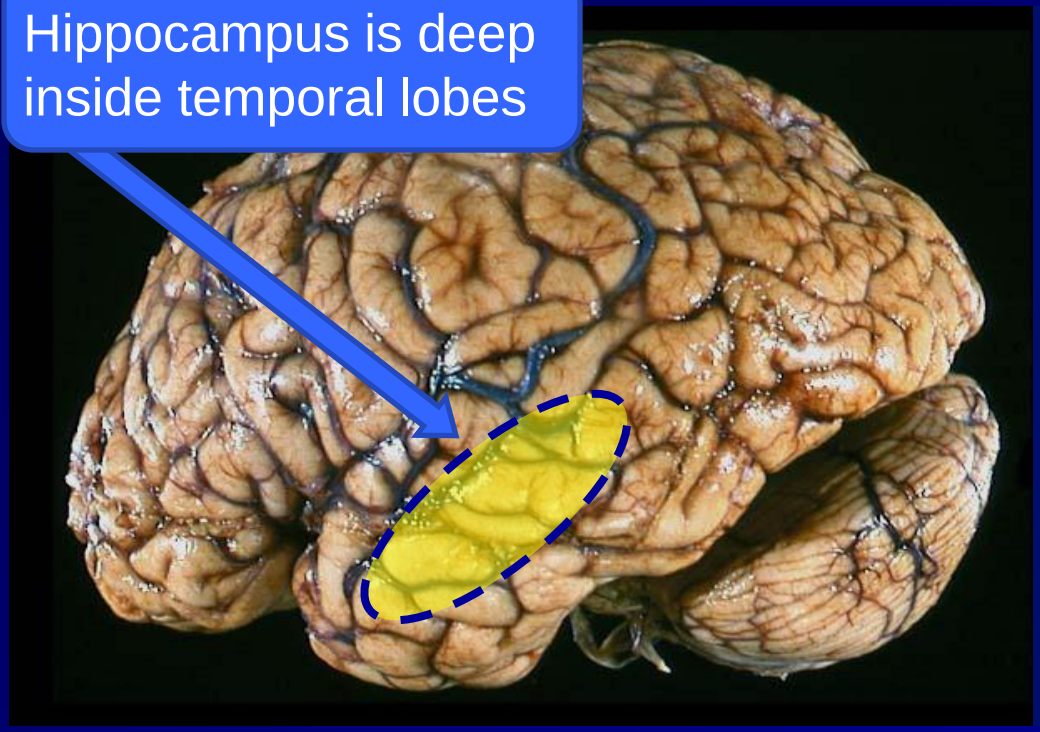
- Adolescent amygdala highly reactive to stressful stimuli
- Stress hormone (cortisol) levels higher during adolescence
- Adolescence is a time of high stress and uncertainty

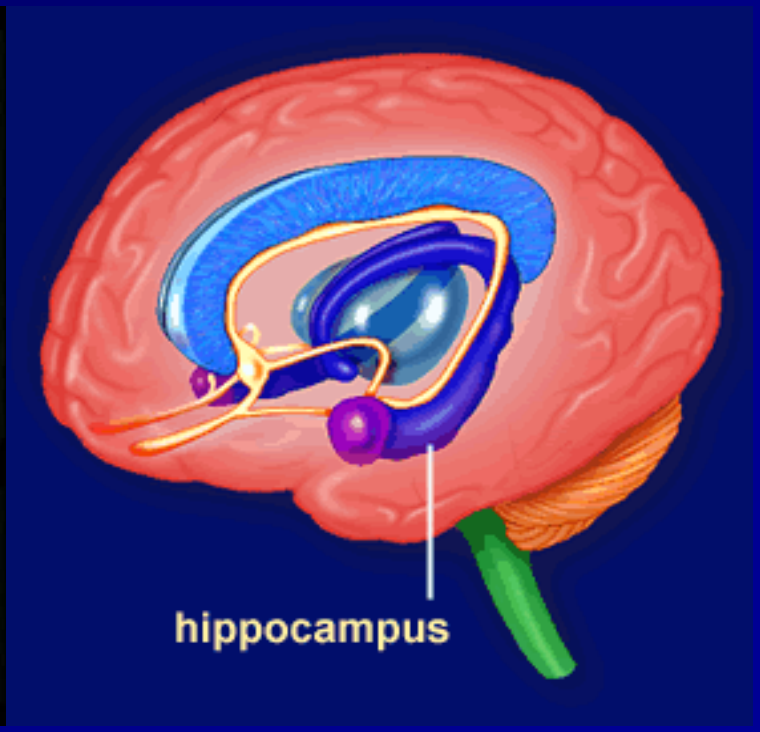
Amygdala

- Volume knob for fear and anxiety
- Allows for learning **to fear** things
- Allows for learning **not to fear** things



How the brain records memories





- Hippocampus records facts and life events
- New brain cells born here throughout life
- Neurogenesis peaks in adolescence

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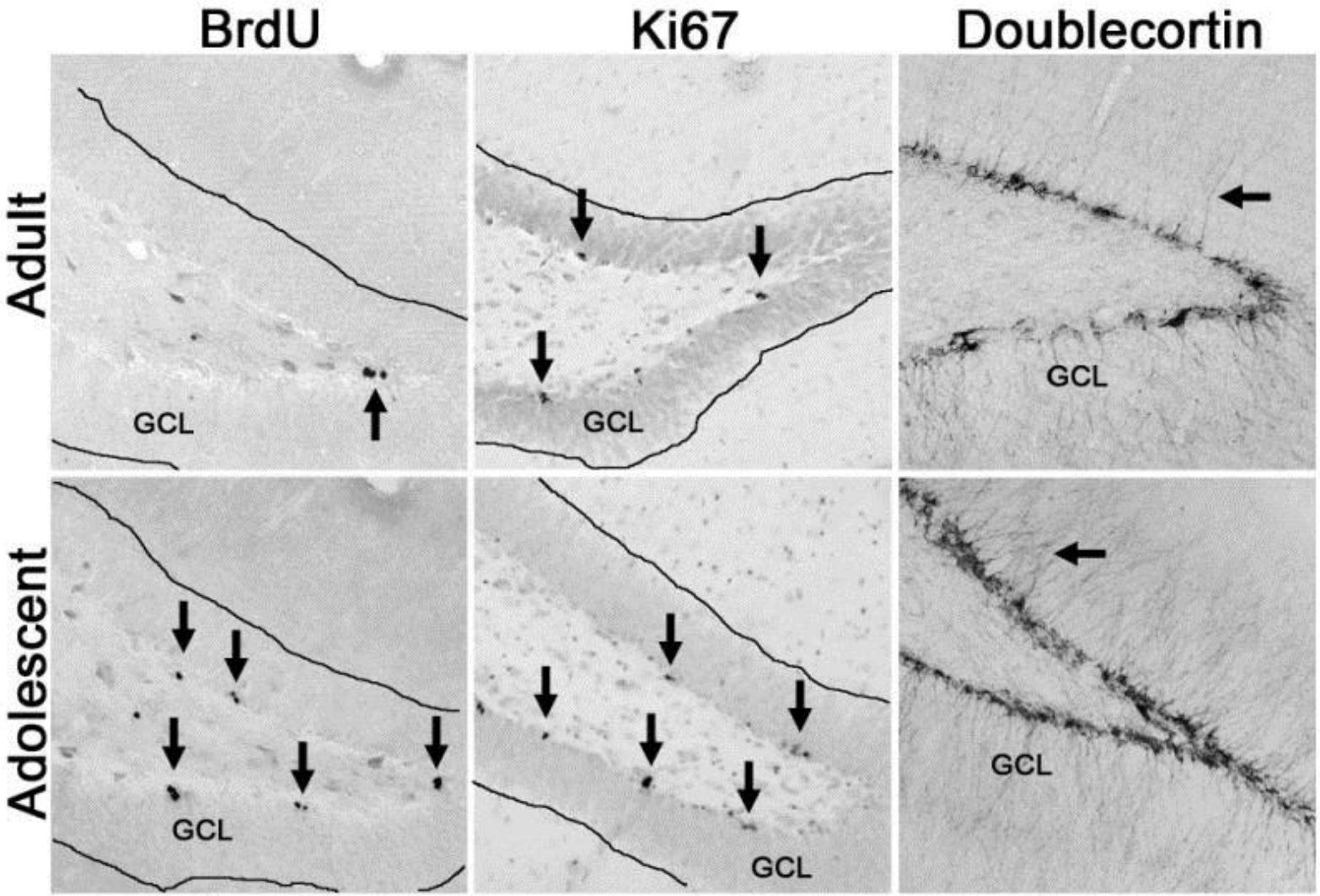
Neurogenesis peaks during adolescence



He J, Crews FT. Neurogenesis decreases during brain maturation from adolescence to adulthood. Pharm Biochem Behav. 2007;86:327–333.

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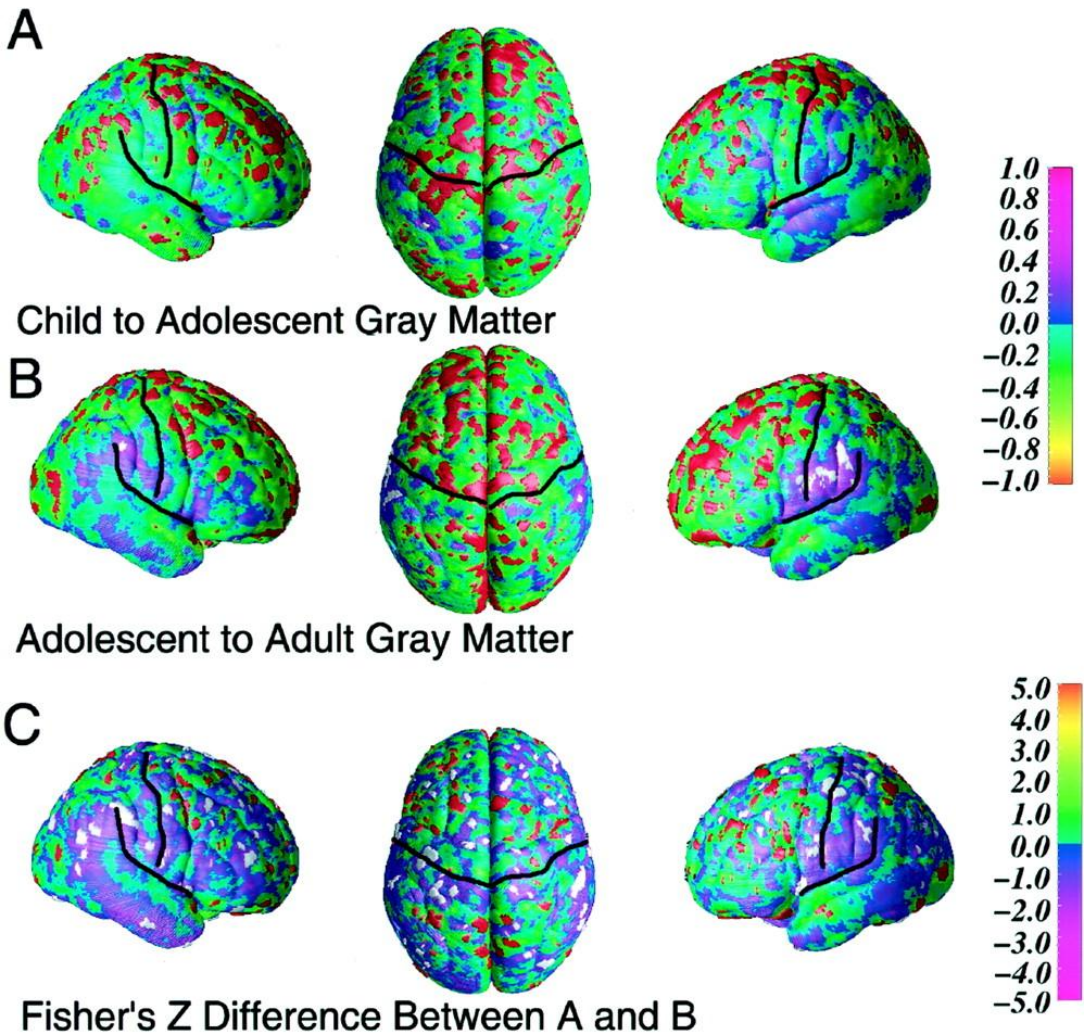
Myelination increases as gray matter density decreases



Sowell E R et al. J. Neurosci. 2001;21:8819-8829

Decreased DFC gray matter density but increased total DFC volume

"Close temporal linkage between dendritic arborization–synaptic density changes and increased myelination could be consistent with our in vivo findings of cortical gray matter density reduction spatially concomitant with late brain growth."

Gray matter density age effect statistical maps showing gray matter density changes between childhood and adolescence (A) and between adolescence and adulthood (B).

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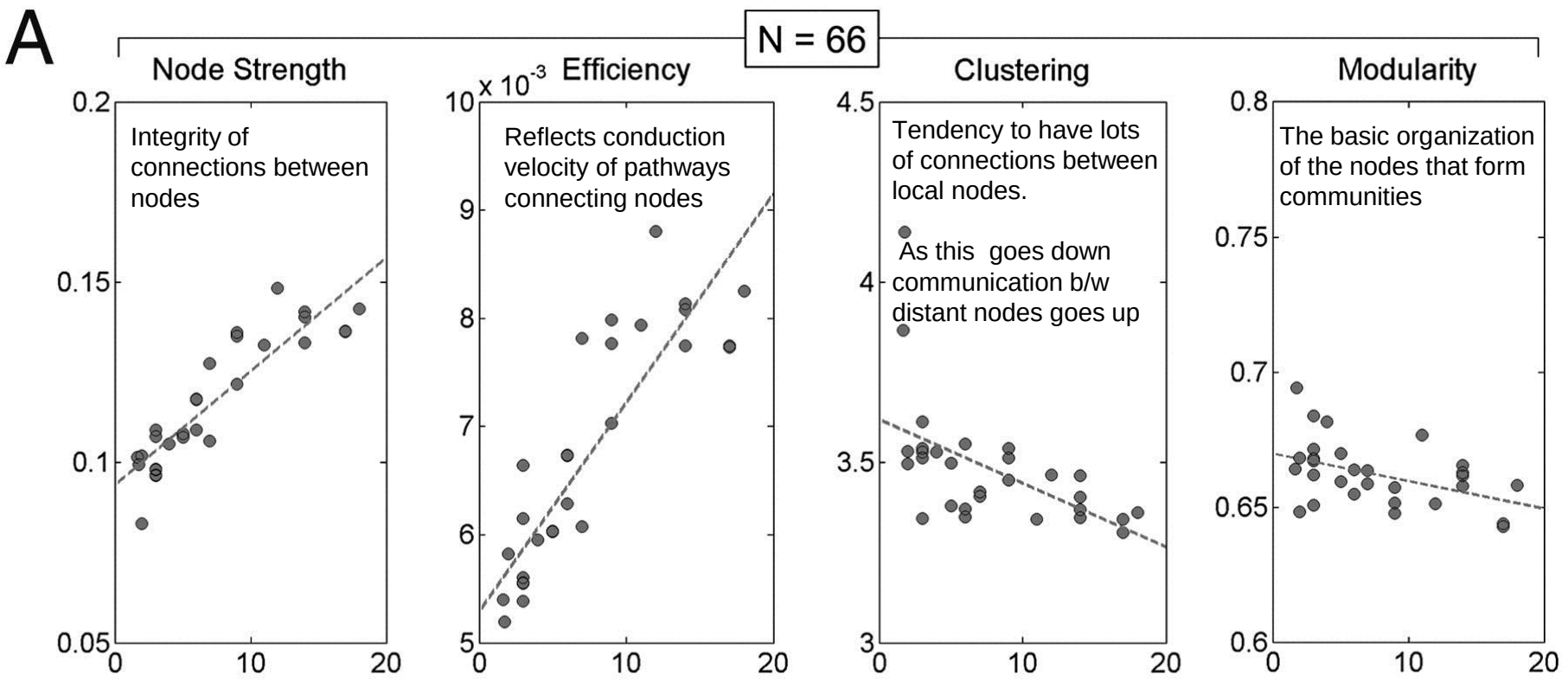
White matter maturation during adolescent development

Relationship of network metrics and developmental age based on measurements of white matter



Hagmann et al. PNAS 2010;107:19067-19072

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Strengthening of circuits involving distant brain regions during adolescence based on white matter measurements

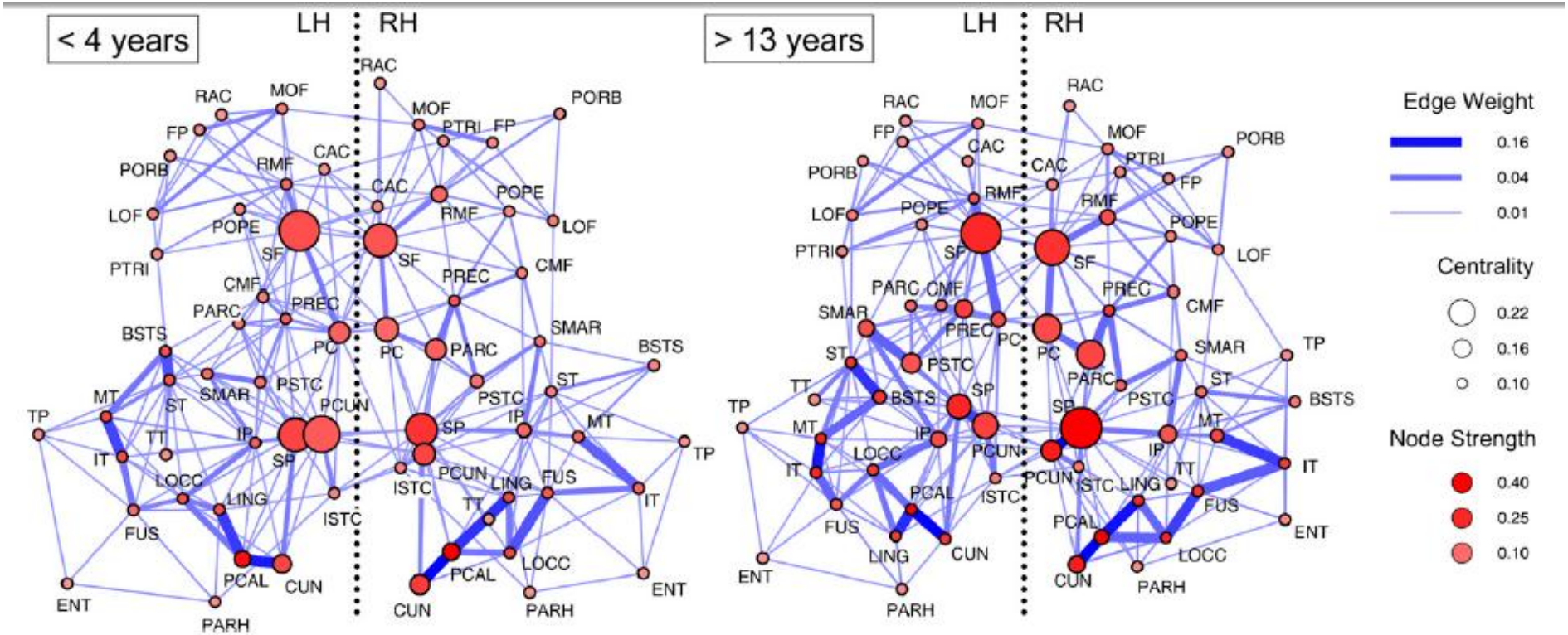

Fig. S4. Developmental changes in structural connectivity. The plots show a Kamada-Kawai force spring layout of the backbone of structural connectivity averaged over younger (<4 y) and older (>13 y) subjects. Plots show node strengths, edge weights, and betweenness centrality. Major hubs in superior frontal cortex, superior parietal cortex, and precuneus are evident in both younger and older brains. BSTS, bank of the superior temporal sulcus; CAC, caudal anterior cingulate cortex; CMF, caudal middle frontal cortex; CUN, cuneus; ENT, entorhinal cortex; FP, frontal pole; FUS, fusiform gyrus; IP, inferior parietal cortex; IT, inferior temporal cortex; ISTC, isthmus of the cingulate cortex; LOCC, lateral occipital cortex; LOF, lateral orbitofrontal cortex; LING, lingual gyrus; MOF, medial orbitofrontal cortex; MT, middle temporal cortex; PARC, paracentral lobule; PARH, parahippocampal cortex; POPE, pars opercularis; PORB, pars orbitalis; PTRI, pars triangularis; PCAL, pericalcarine cortex; PSTC, postcentral gyrus; PC, posterior cingulate cortex; PREC, precentral gyrus; PCUN, precuneus; RAC, rostral anterior cingulate cortex; RMF, rostral middle frontal cortex; SF, superior frontal cortex; SP, superior parietal cortex; ST, superior temporal cortex; SMAR, supramarginal gyrus; TP, temporal pole; TT, transverse temporal cortex.

Hagmann et al. PNAS 2010;107:19067-19072 – supplemental materials

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During adolescence strong drives and still developing cognitive control contributes to risk-taking

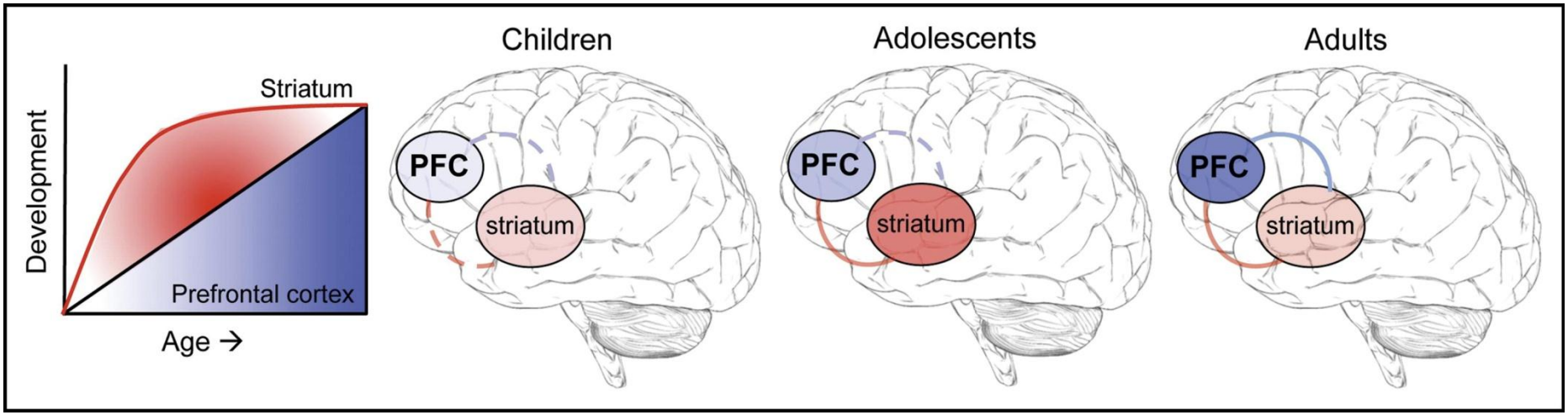

FIGURE 1. Cartoon model of ventral striatal cortex and prefrontal cortex (PFC) interactions across development. Note: Deeper color indicates greater regional signaling. Line represents functional connectivity, with solid line indicating mature connection and dotted line indicating immaturity.

SOURCE: Casey BJ, Jones RM. Neurobiology of the adolescent brain and behavior: implications for substance use disorders. J Am Acad Child Adolesc Psychiatry. 2010 Dec;49(12):1189-201;

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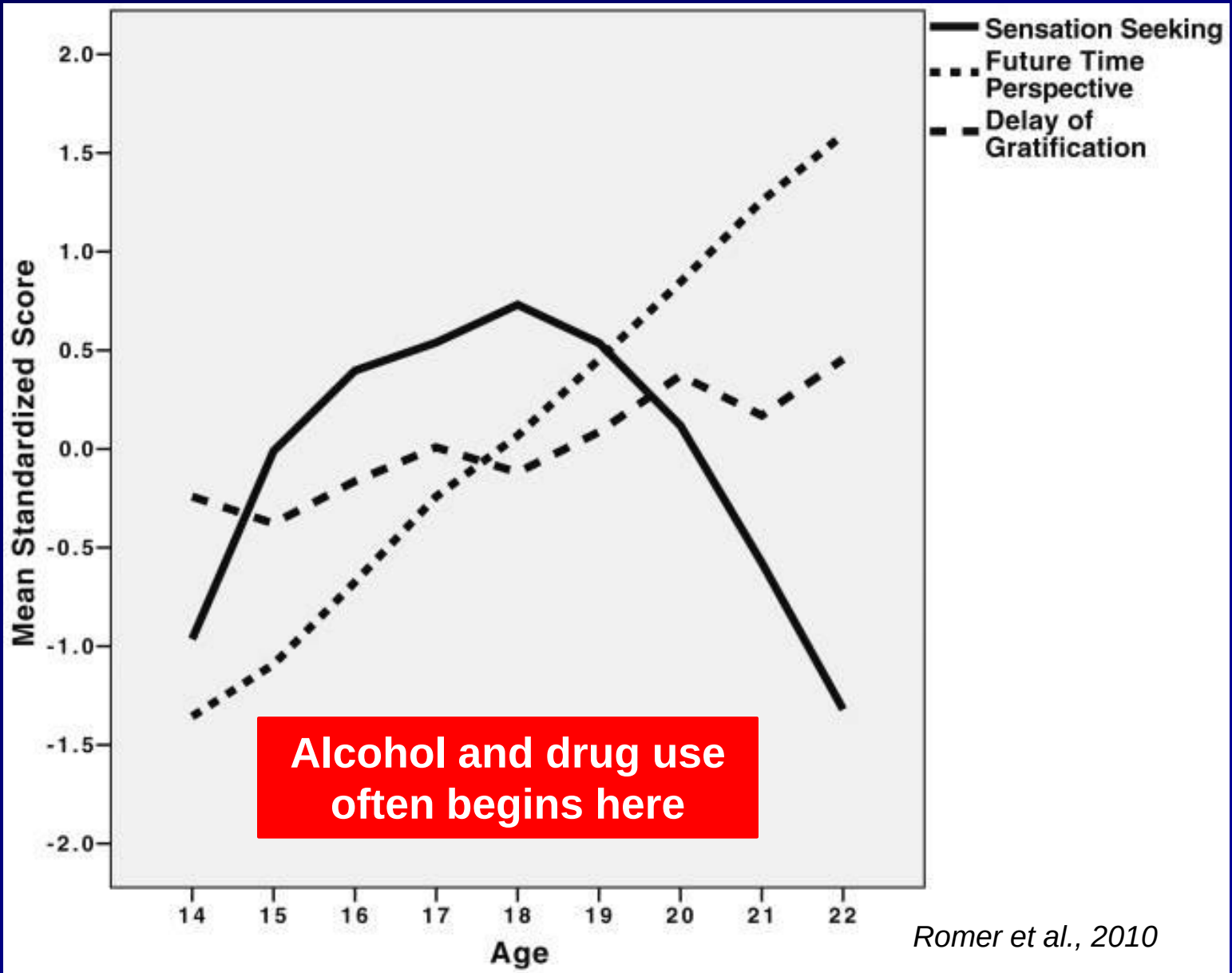
Adolescence is risky business



Romer et al., 2010

Alcohol and other drugs can turn adaptive tendencies into maladaptive behaviors

- Adolescent brains built to learn with ease with an eye toward success during adulthood
- Adolescent brains learn particularly fast when behaviors are rewarded
- Frontal lobes (decision-making, impulse control) are not fully online during adolescence but emotional drive is in high gear
- Alcohol and other drugs produce reward leading to rapid learning and motivating further use
- Alcohol causes both short-term and long-term effects that are bad for adolescents

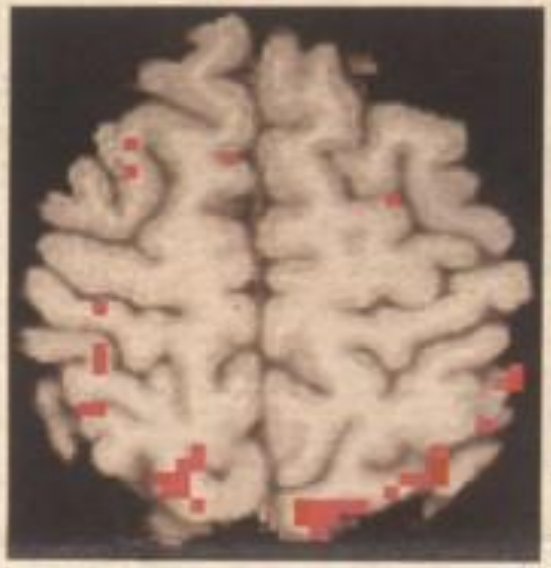
www.usatoday.com

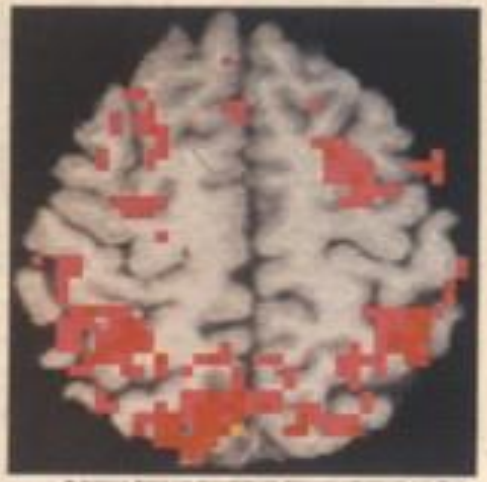
USA TODAY

Life

SECTION D

Wednesday, October 18, 2000





Two 20-year-old women take a memory test. One of them abused alcohol. The MRI scan on the left is her brain, the lack of color indicating a sluggish mind. In contrast, the scan on the right is of the woman who doesn't have a drinking problem. The colors show lots of brain activity. Not surprisingly, she does better on the test.

Cover story

Teen drinking, thinking don't mix

Alcohol appears to damage young brains, early research finds

By Kathleen Sachsenmaier
USA TODAY

Changing youths' drinking habits, 8D

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Alcohol suppresses frontal lobes[1]

- Teens already have problems with decision-making, attention, impulse control
- Alcohol makes the problems worse!

[1]Abernathy et al (2010). Alcohol and the prefrontal cortex. Int Rev Neurobiol 91:289-320.

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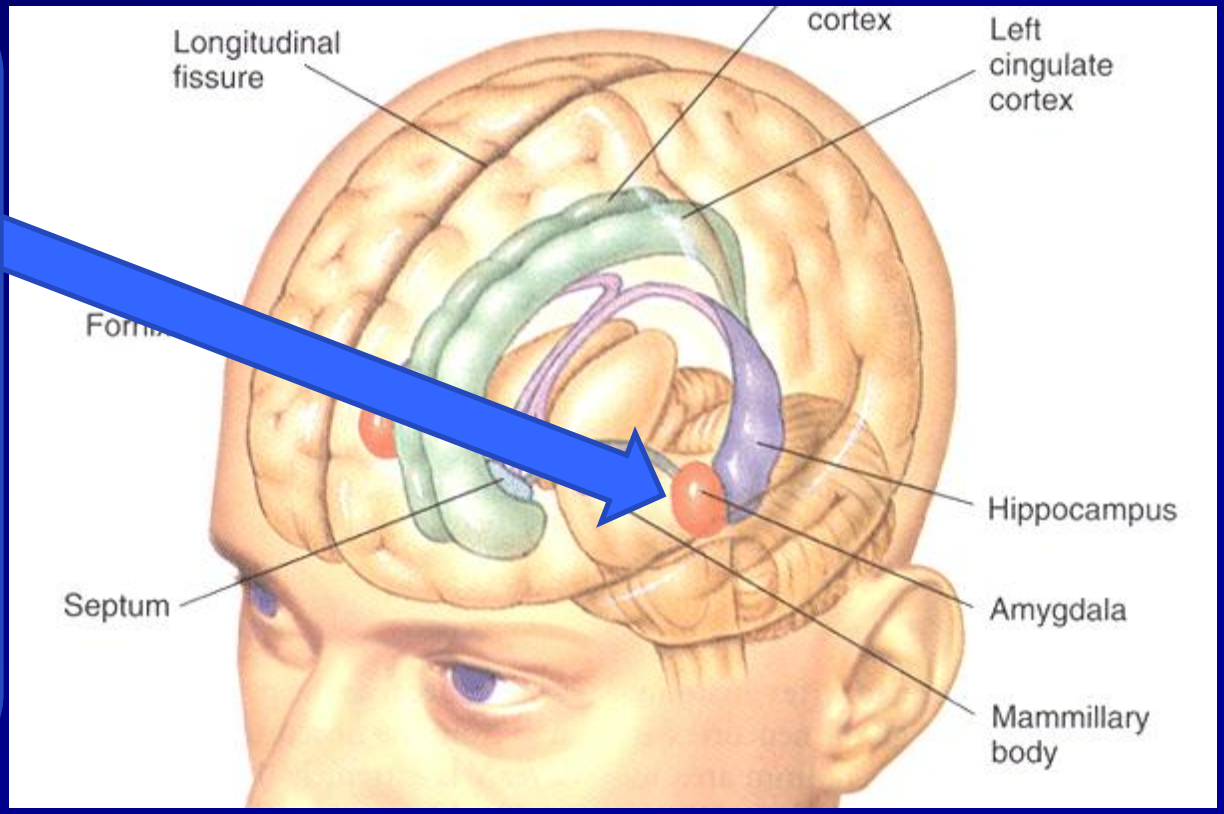
Alcohol – "Shot of courage"

- By suppressing the amygdala alcohol diminishes *psychological* experiences of stress (fear and anxiety)
- Does not help with the *physiological* effects of stress
- Makes it easier to do risky things

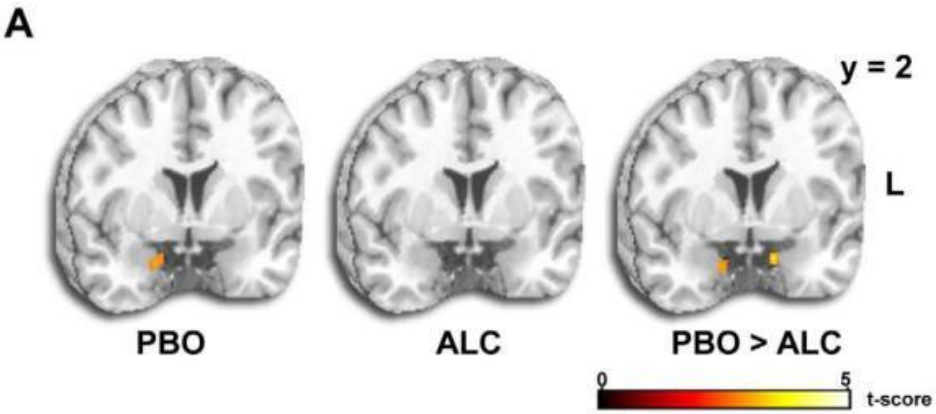
<u>Alcohol</u>

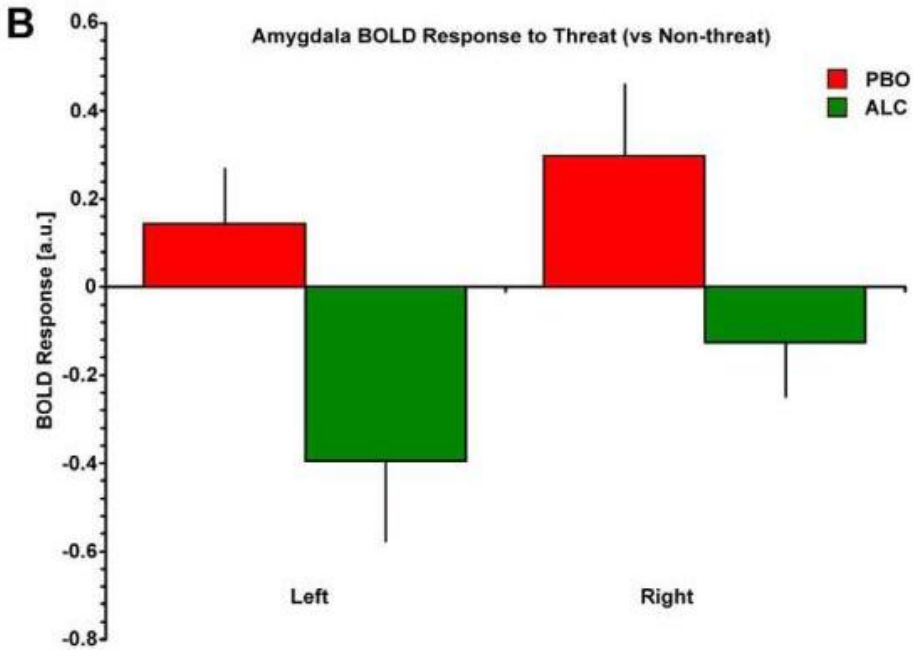
•Quiets the amygdala

•Temporary reduction in *experience* of stress



Alcohol suppresses amygdala response to threatening stimuli





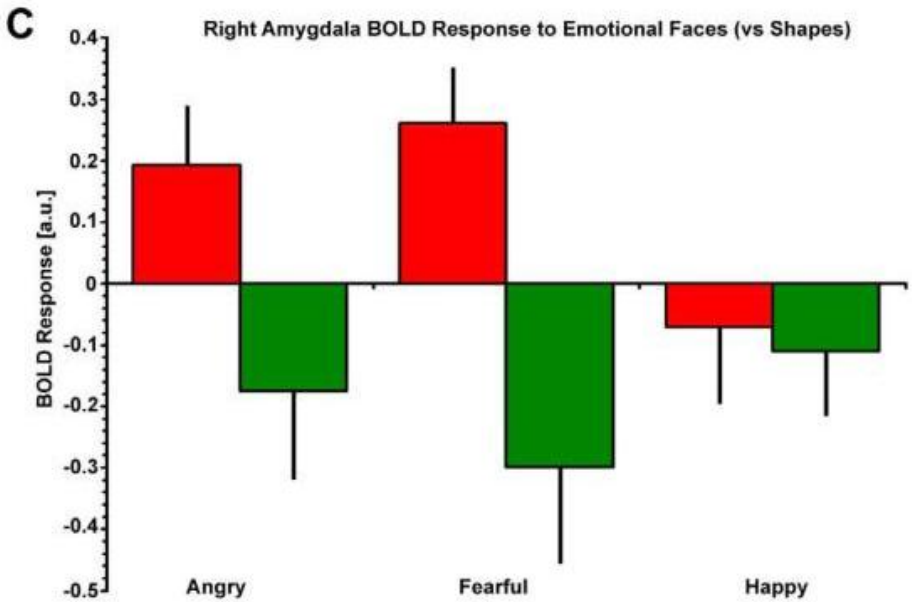


FIGURE. Alcohol effects on amygdala activation to social signals of threat. **A)** Right lateral amygdala activation to Threat (> Non-threat) faces is present during the PBO session but absent during the ALC session. **B)** Mean BOLD Response (β weights ±SEM) extracted from amygdala ROIs showing activation to Threat (> Non-threat) faces in the PBO session but no activation during the ALC session. PBO, placebo; ALC, alcohol. **C)** Mean BOLD Response showing alcohol attenuates (PBO>ALC) activation to Threat (Angry , Fearful) faces but does not affect responses to Non-threat (Happy Faces).

Source: Sripada et al., 2011, Neuroimage, 55, 371-380.

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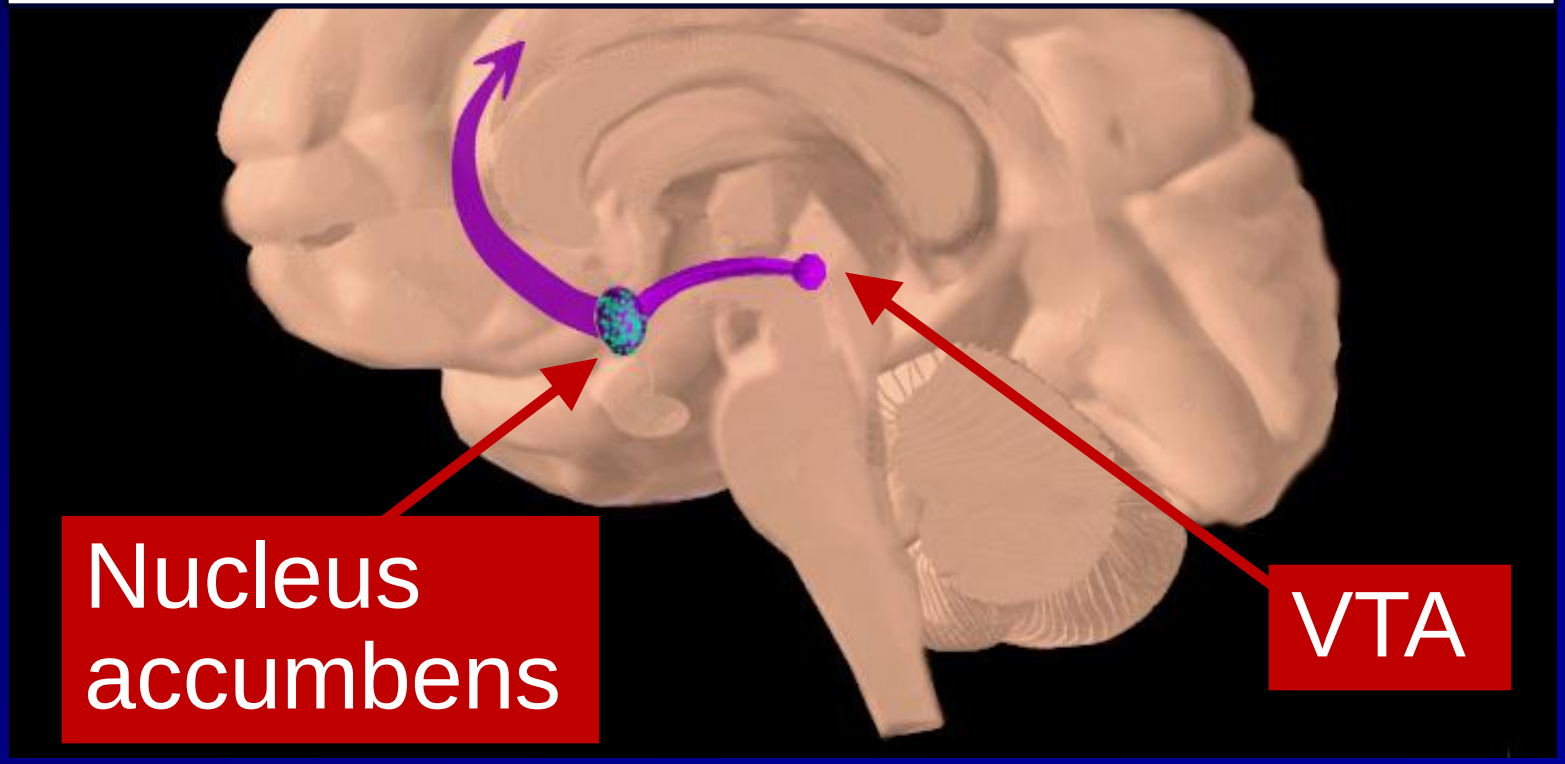
Alcohol activates reward system

Like virtually all drugs used for pleasure and relaxation, alcohol activates the reward system, thus tricking the brain into thinking that something important and worth repeating just happened. Each time the behavior is repeated and reinforced, the odds go up that alcohol will be consumed again. This is a tricky venture for adolescents, whose frontal lobes are still under construction and already have difficulty controlling impulses, and the behaviors they pick up tend to have staying power.



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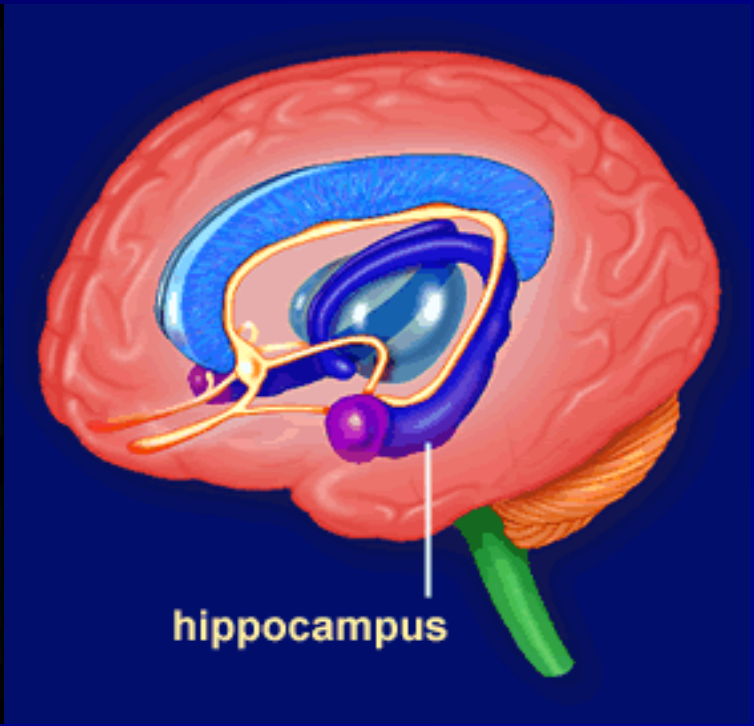
Alcohol blocks new memories



- Adolescent hippocampus more vulnerable to effects of alcohol on hippocampal function
- Effects on hippocampus lead to blackouts

White and Swartzwelder, 2004.

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Make it a night you won't forget,
not one you can't remember.

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Alcohol-induced memory blackouts

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'Drunk and naked' in Cancun

By Gary Strauss, USA TODAY

CANCUN, Mexico — Shane Mahmood woke from a booze-induced fog with a half-inch stainless steel ring pierced through his lower lip.

Shane Mahmood doesn't remember getting his lip pierced.

By Gary Strauss, USA TODAY

"I don't know how I got it or if it hurt when I did," says Mahmood, who was partying at disco La Boom with University of Washington pals when the predawn procedure apparently occurred. "I just remember beer and tequila before waking up and seeing it in the mirror."

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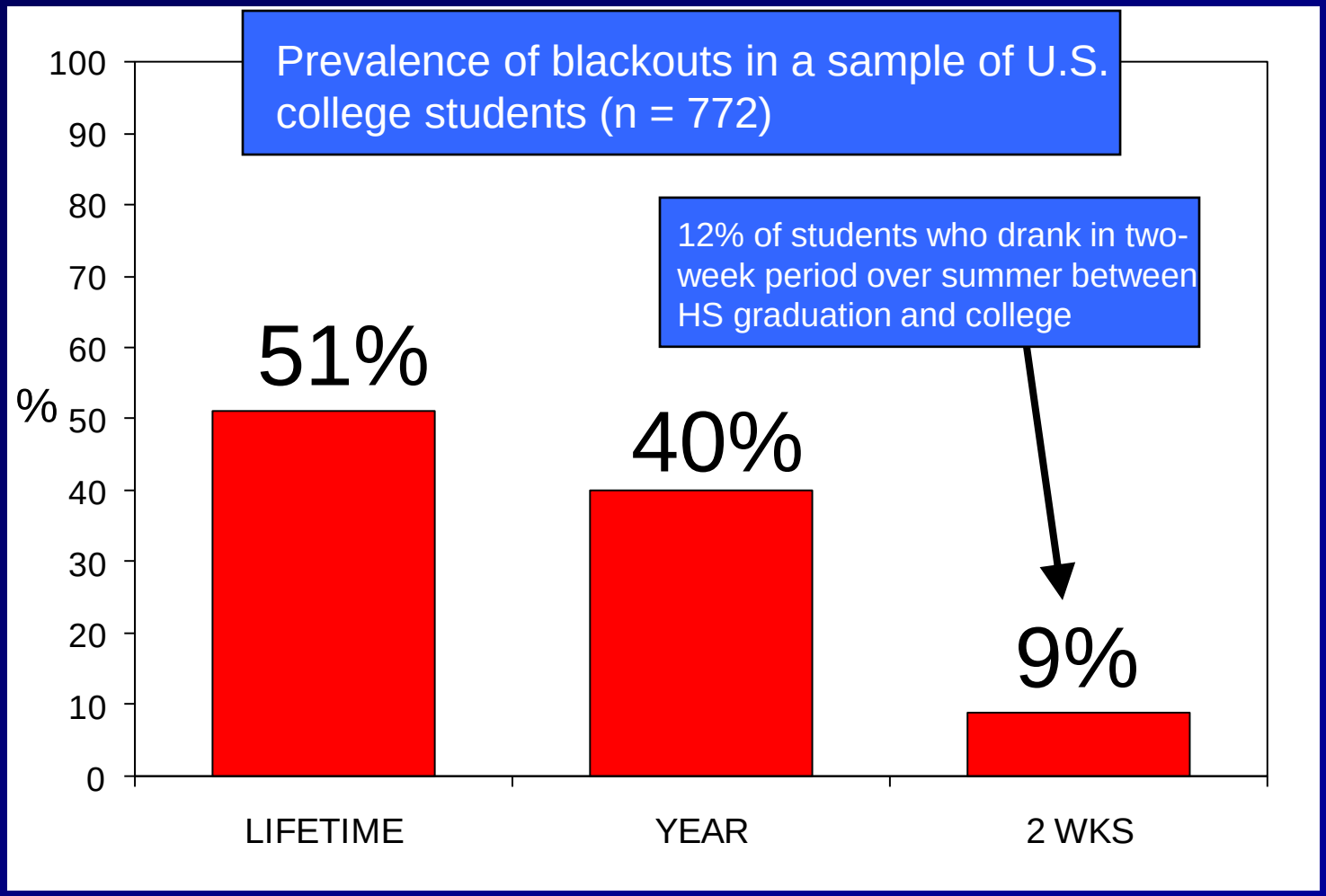
Memory blackouts are common



Source: White et al, 2002, American Journal of College Health

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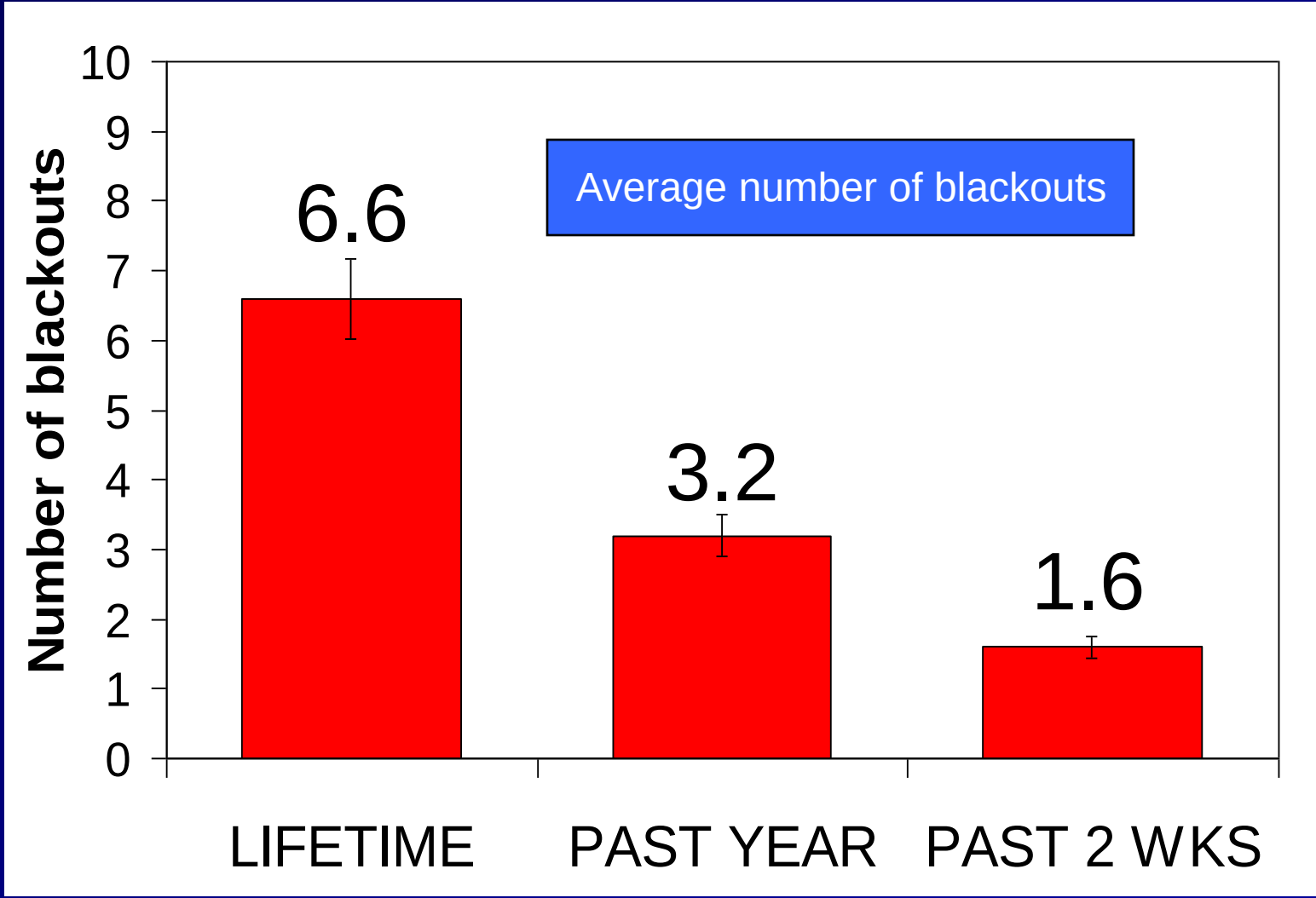
Memory blackouts are common



Source: White et al, 2002, American Journal of College Health

J Forensic Sci, July 2006, Vol. 51, No. 4
doi:10.1111/j.1556-4029.2006.00161.x
Available online at: www.blackwell-synergy.com

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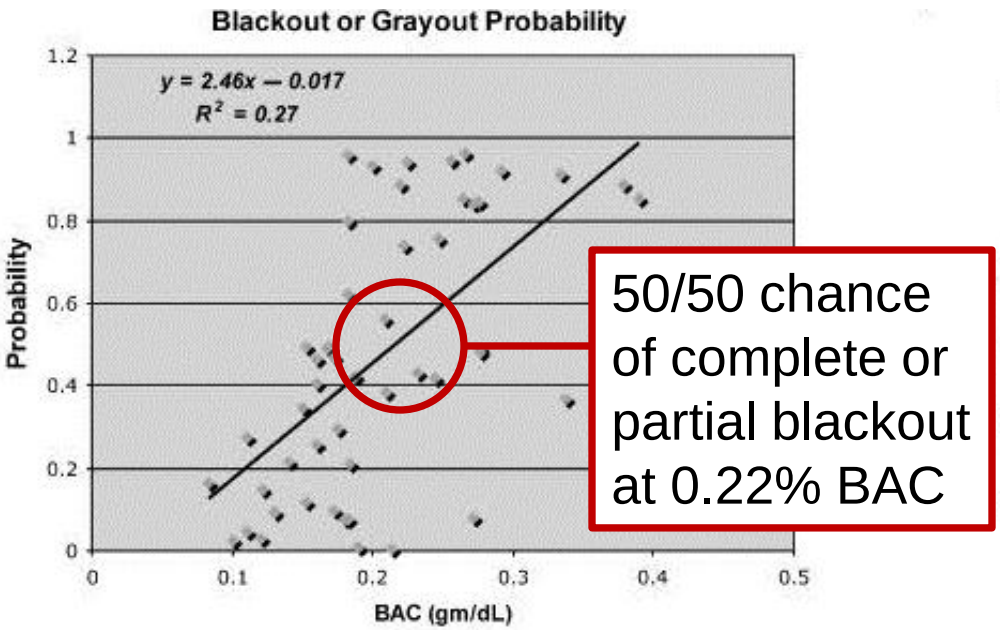
The Association of Alcohol-Induced Blackouts and Grayouts to Blood Alcohol Concentrations

An interview questionnaire was administered to subjects regarding a recent alcohol associated arrest with a documented BAC greater than 0.08 g/dL for either public intoxication, driving under the influence, or under age drinking.



50/50 chance of complete or partial blackout at 0.22% BAC

FIG. 1 *Probability of grayouts or blackouts as a function of the blood alcohol concentration (BAC) (g/dL) where probability = 2.46 (BAC) 0.02 (R^2 = 0.27).*

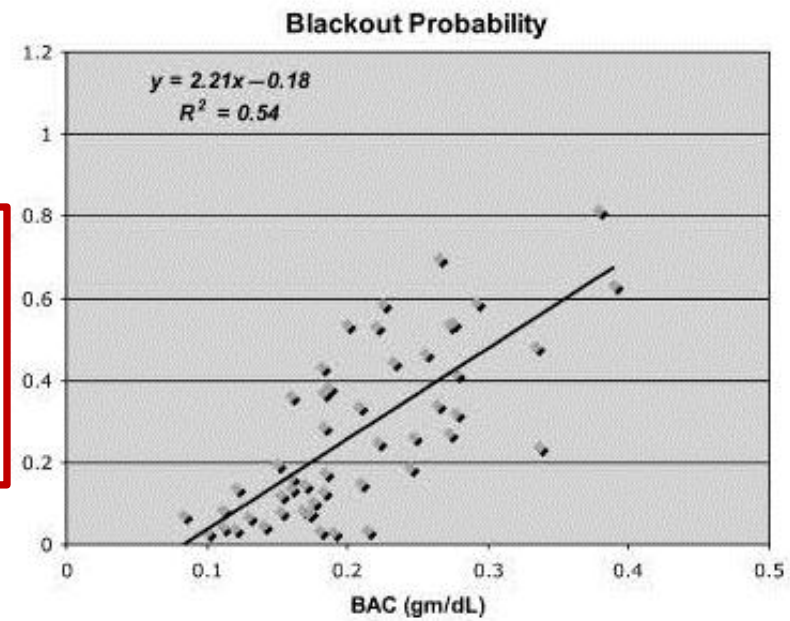


FIG. 2 *Probability of blackouts as a function of the blood alcohol concentration (BAC) (g/dL) where probability = 2.21 (BAC) 0.18 (R^2 = 0.54).*

Fine line between life and death at high BACs

Posted 10/7/2004 12:00 AM Updated 10/6/2004 10:08 PM

Five binge-drinking deaths 'just the tip of the iceberg'

By Robert Davis, USA TODAY

This month has been deadly for binge-drinking college students.

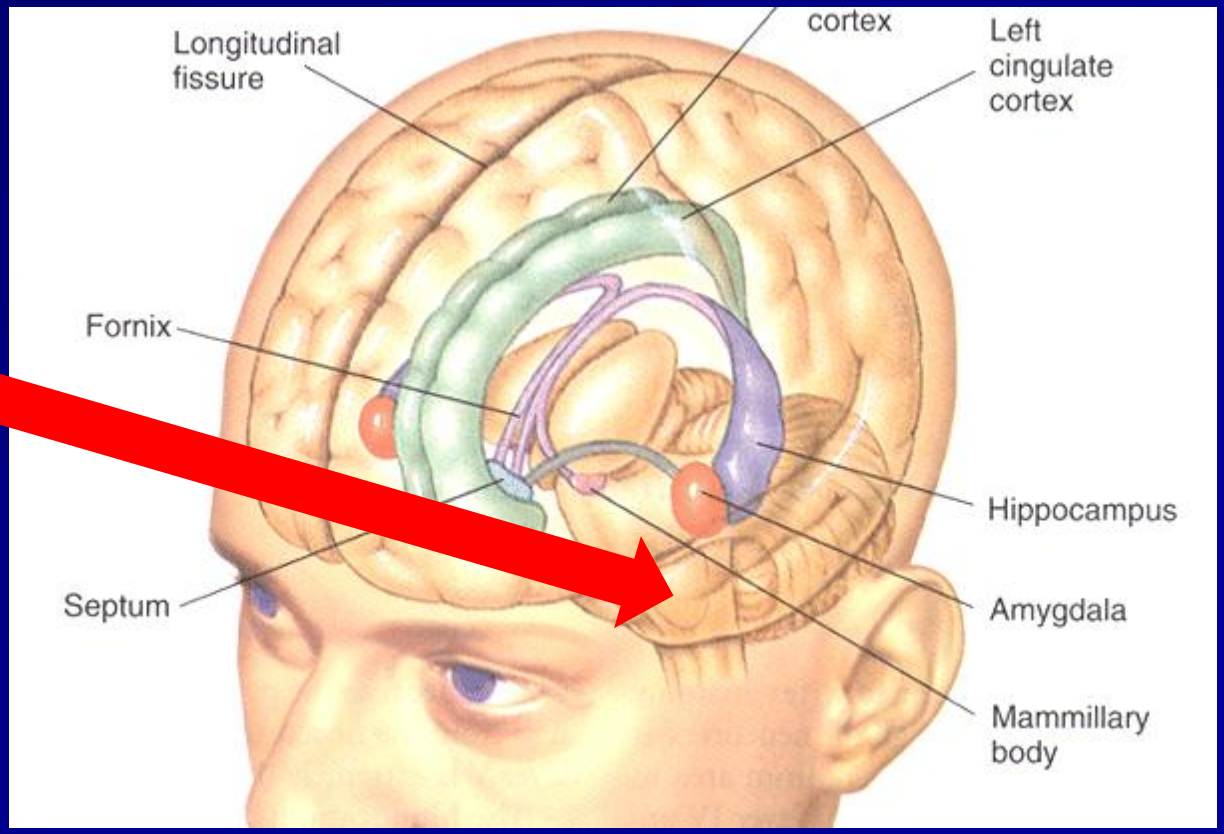
Five underclassmen in four states appear to have drunk themselves to death, police say, after friends sent their pals to bed assuming that they would "sleep it off."

Death by overdose on alcohol/other drug combinations

- Excessive alcohol and/or other CNS depressants can shuts down brainstem areas involved in vital reflexes like breathing, gagging, and heart rate

Alcohol

- Suppresses respiratory areas and other vital reflex centers
- Causes death



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Alcohol overdose/poisoning

- Alcohol has a *very* small therapeutic index (TD_{50}/ED_{50})
- A toxic or deadly dose not much higher than a moderately intoxicating dose

Potentially fatal alcohol overdoses: How much alcohol?

If ED_{50} = ~.08%
And TD_{50} = ~.32%
Then TI = ~4

10 drinks in 2 hrs for a 140 lb female

13 drinks in 2 hrs for a 160 lb male

BAC = 0.32

BAC = 0.43

BAC = 0.44

BAC = 0.48

denverpost.com
THE DENVER POST

Officials say CU student died of alcohol poisoning also had drug in his system

Posted: 09/30/2011 01:00:00 AM MDT

A University of Colorado student who died after a night of partying during the first week of school in August had drugs and alcohol in his system, the Boulder County Coroner's Office said Thursday.

A toxicology test found the narcotic painkiller oxymorphone, and alcohol poisoning caused the death of 21-year-old Michael Alexander Hoffman of Berkeley Heights, N.J.

Hoffman was found unconscious Aug. 26 on the porch of a home at 1429 10th St., two blocks west of the CU campus. The residents there did not know him, the Daily Camera reported at the time.

He never regained consciousness and died at a hospital four days later. A police report said messages on Hoffman's cellphone indicated he had been drinking heavily the night he died.

Drug Abuse Warning Network
The DAWN Report
September 13, 2011

Trends in Emergency Department Visits Involving Underage Alcohol Use: 2005 to 2009

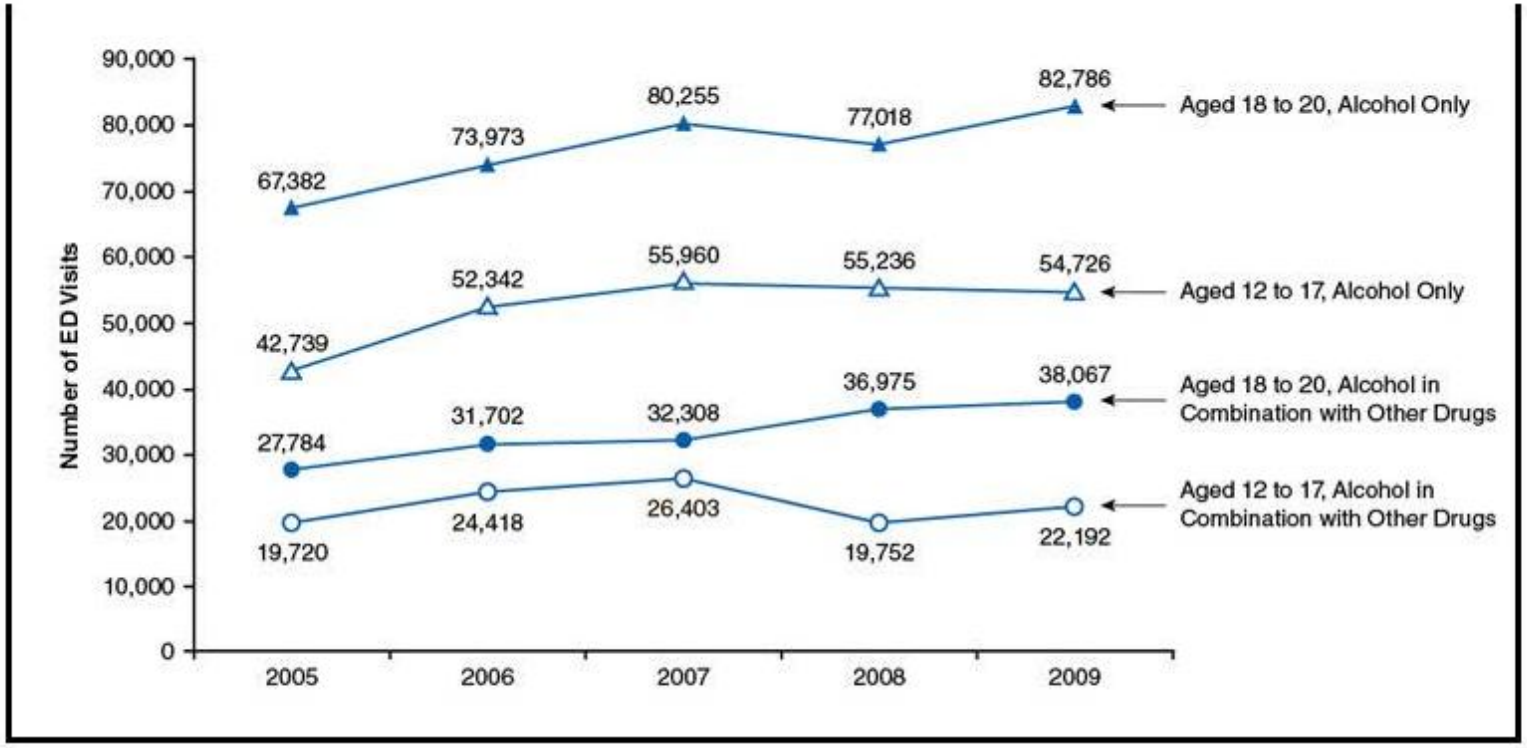


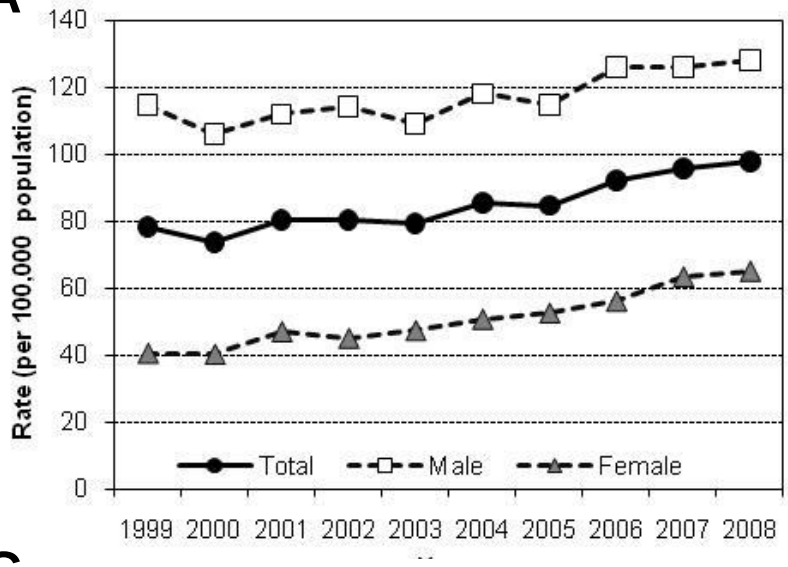
Figure 4 Table. Number of Underage Drinking-Related Emergency Department (ED) Visits, by Age group and Type of Alcohol-Related Visit: 2005 to 2009*

Age Group and Type of ED Visit	2005	2006	2007	2008	2009
Aged 18 to 20, Alcohol Only	67,382	73,973	80,255	77,018	82,786
Aged 12 to 17, Alcohol Only	42,739	52,342	55,960	55,236	54,726
Aged 18 to 20, Alcohol in Combination with Other Drugs	27,784	31,702	32,308	36,975	38,067
Aged 12 to 17, Alcohol in Combination with Other Drugs	19,720	24,418	26,403	19,752	22,192

*ED visits for which age is unknown have been excluded from analysis.
Source: 2005 and 2009 estimates from the 2009 SAMHSA Drug Abuse Warning Network (DAWN).

Changes in rates of alcohol, drug and combined overdoses among 18-24 year olds between 1999-2008

A



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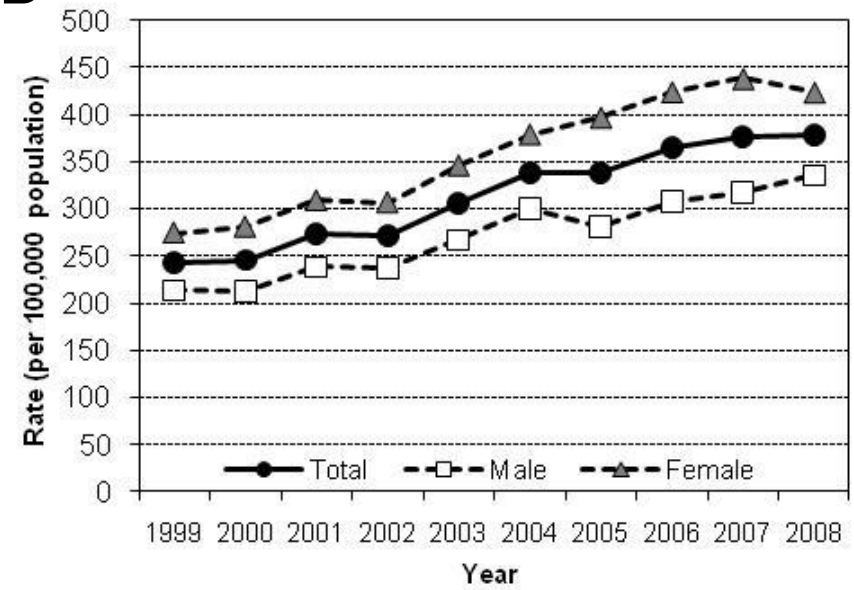

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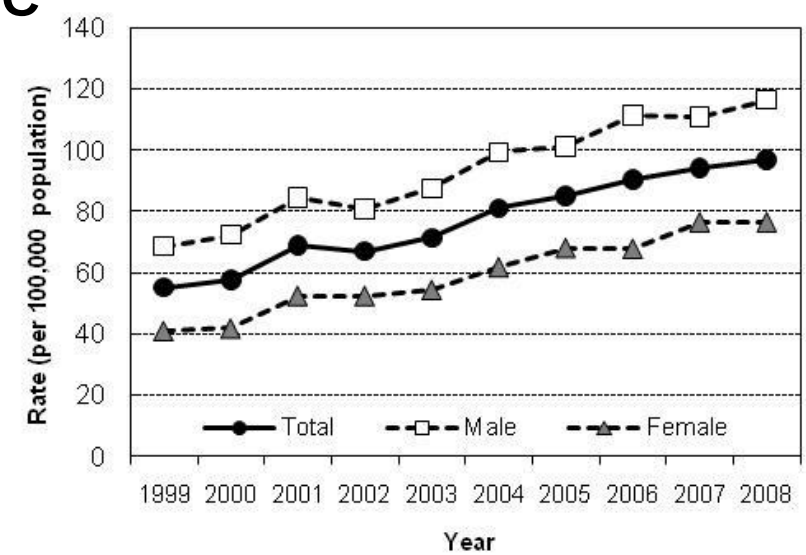

A. Alcohol overdoses ↑**25%**

B. Drug overdoses ↑**56%**

C. Combined alcohol + drug overdoses ↑**76%**

Overdose defined as excessive consumption and/or poisoning based on ICD-9-CM codes

Long-term effects: Starting young is bad news

- Starting early increases risk of dependence
- Brain seems to pick up alcohol habits quickly
- Increased risk of negative outcomes
- Lingering cognitive deficits

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Age of drinking onset and dependence: Risk goes up as age goes down

AGE	FHP (%)	FHN (%)
≤ 13	57	26
14-15	**48**	**32**
16-17	35	21
18-19	**22**	**13**
20	16	9
≥ 21	**16**	**7**

Source: Grant and Dawson, 1997

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Adolescent alcohol abusers show strong reactions to alcohol-related cues reflecting strong associative learning

Functional magnetic resonance imaging (fMRI) results during alcoholic beverage picture trials relative to non-alcoholic beverage picture trials

Greater activation in AUD subjects (red color) in the ventral anterior cingulate and subcallosal, prefrontal, orbital, and limbic regions, areas previously associated with reward and drug craving.

AUD group (N = 15) 6 girls 9 boys average age of 16, DSM abuse or dependence

Control group (N = 15) 6 girls 9 boys average age 16, low levels of previous use

Tapert, Susan. F. et al. Arch Gen Psychiatry 2003;60:727-735.

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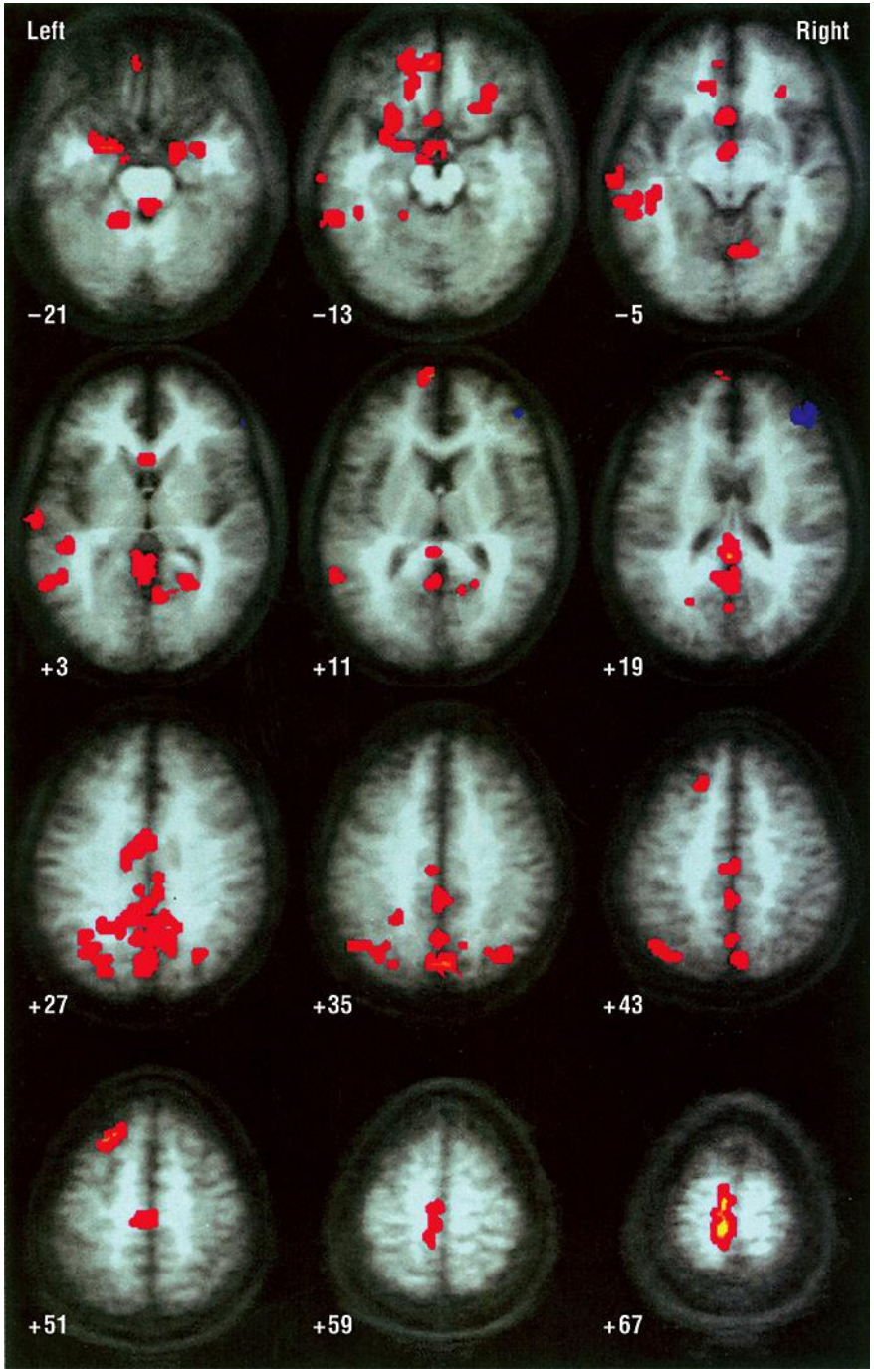
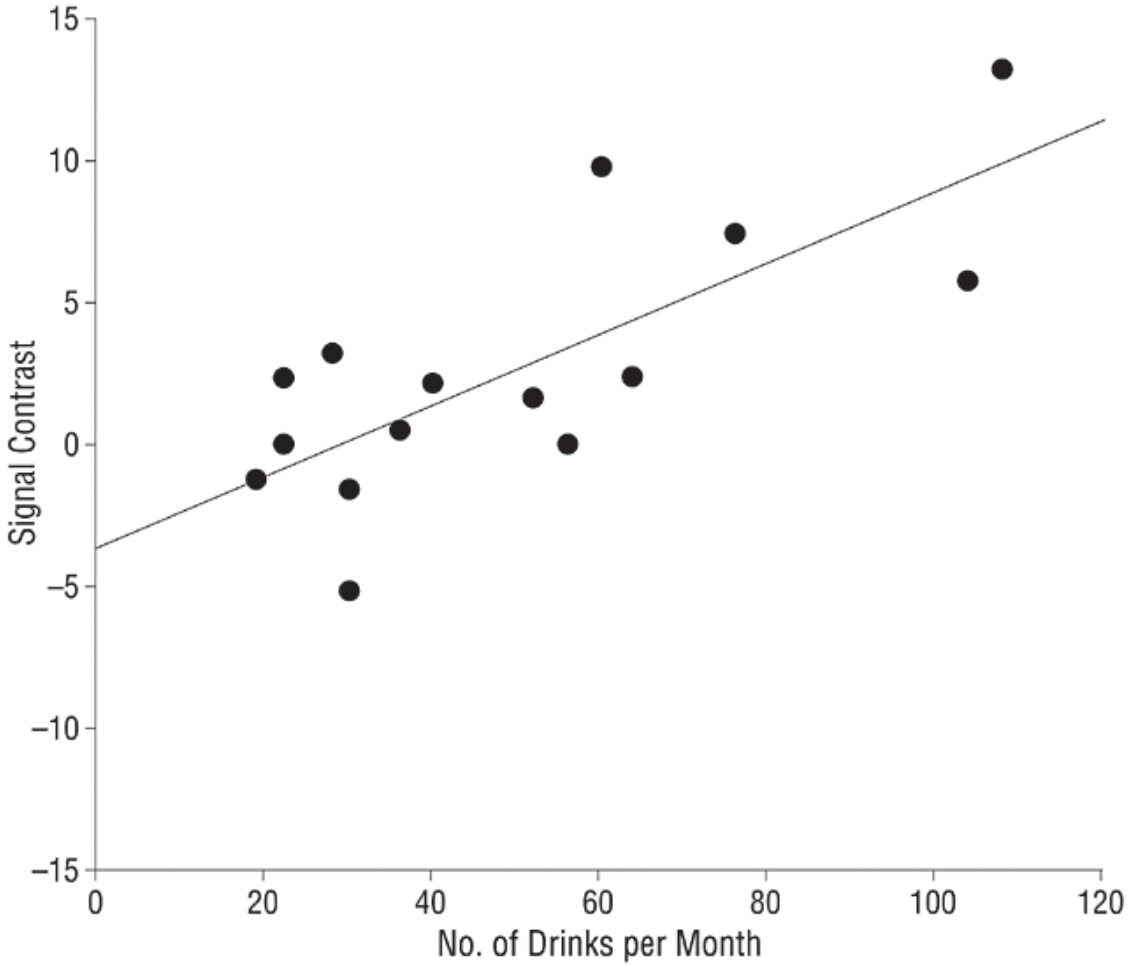


Amount of brain activation in response to alcohol-related cues increases with drinking levels



Blood oxygen level-dependent (BOLD) response signal contrast in the right precuneus/posterior cingulate region during exposure to alcoholic beverage pictures relative to nonalcoholic beverage pictures plotted as a function of drinks consumed per month for adolescents with alcohol use disorder (n = 15)

Are strong associations between alcohol and cues part of why adolescents are at such high risk of developing dependence and of relapsing?

Tapert, Susan. F. et al. Arch Gen Psychiatry 2003;60:727-735.

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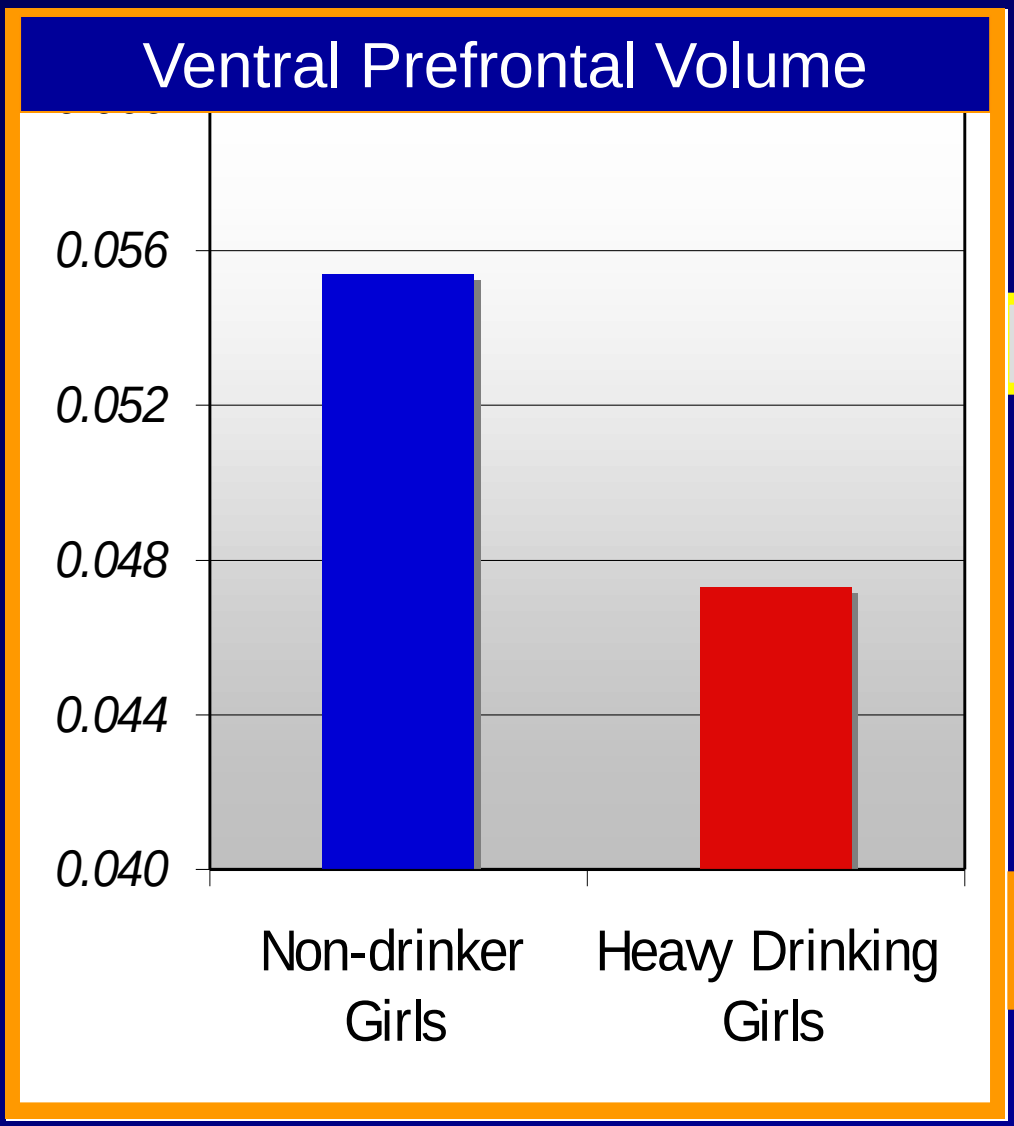
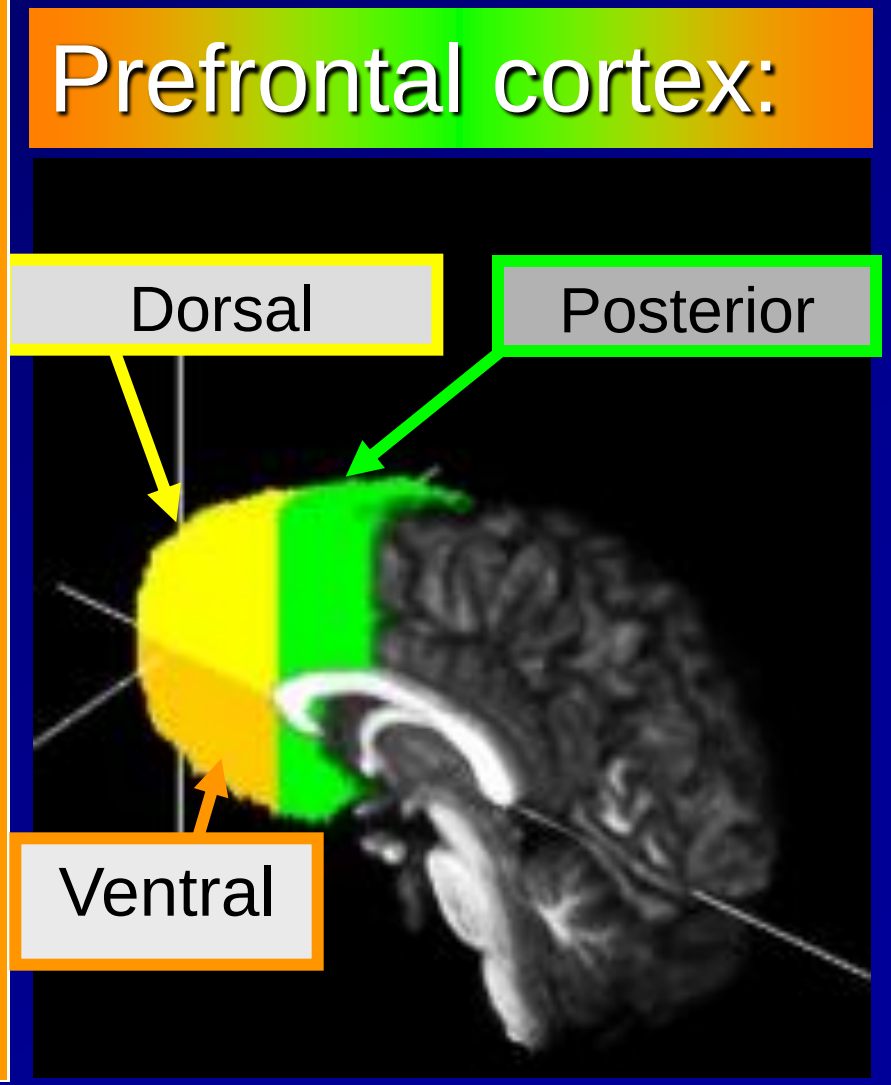
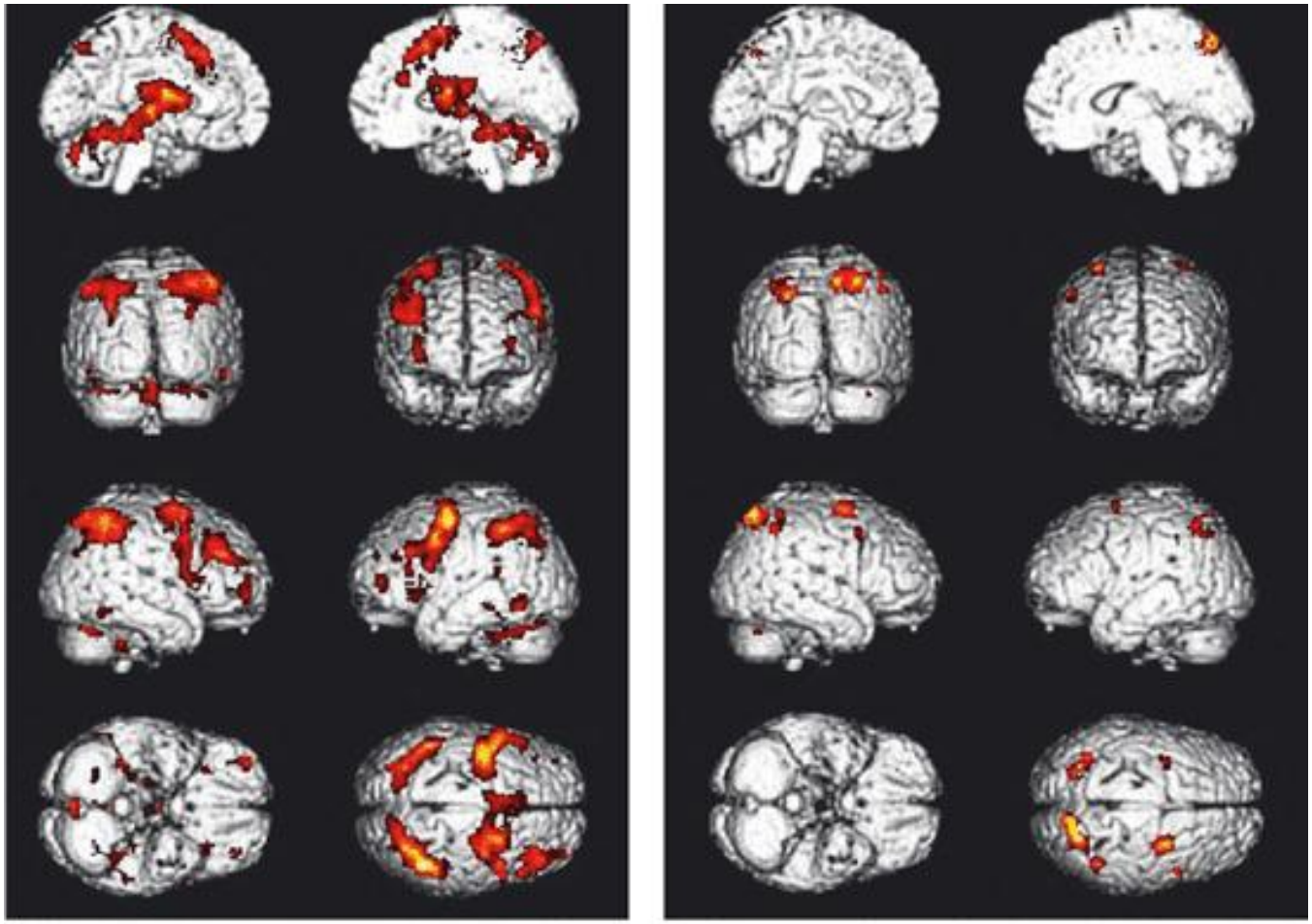
Smaller frontal lobes





Source: Squeglia et al., 2009

Decreased frontal lobe activation during working memory tasks in young males with AUD



The control group

The AUD group

Males 18-26 with DSM abuse or dependence (n = 11) compared to controls (n = 10)

Figure 2. Brain activation areas in the control group (*n* = 10, left) and the AUD group (*n* = 11, right). **Significantly decreased activation was identified in the left frontal circuit including Broca's region (BA 44) and the premotor cortex (BA 6)**. The left frontal circuit is known to be responsible for the articulatory rehearsal process .

From: Park et al (2011) Scand J Psychol. 2011 Feb;52(1):1-7

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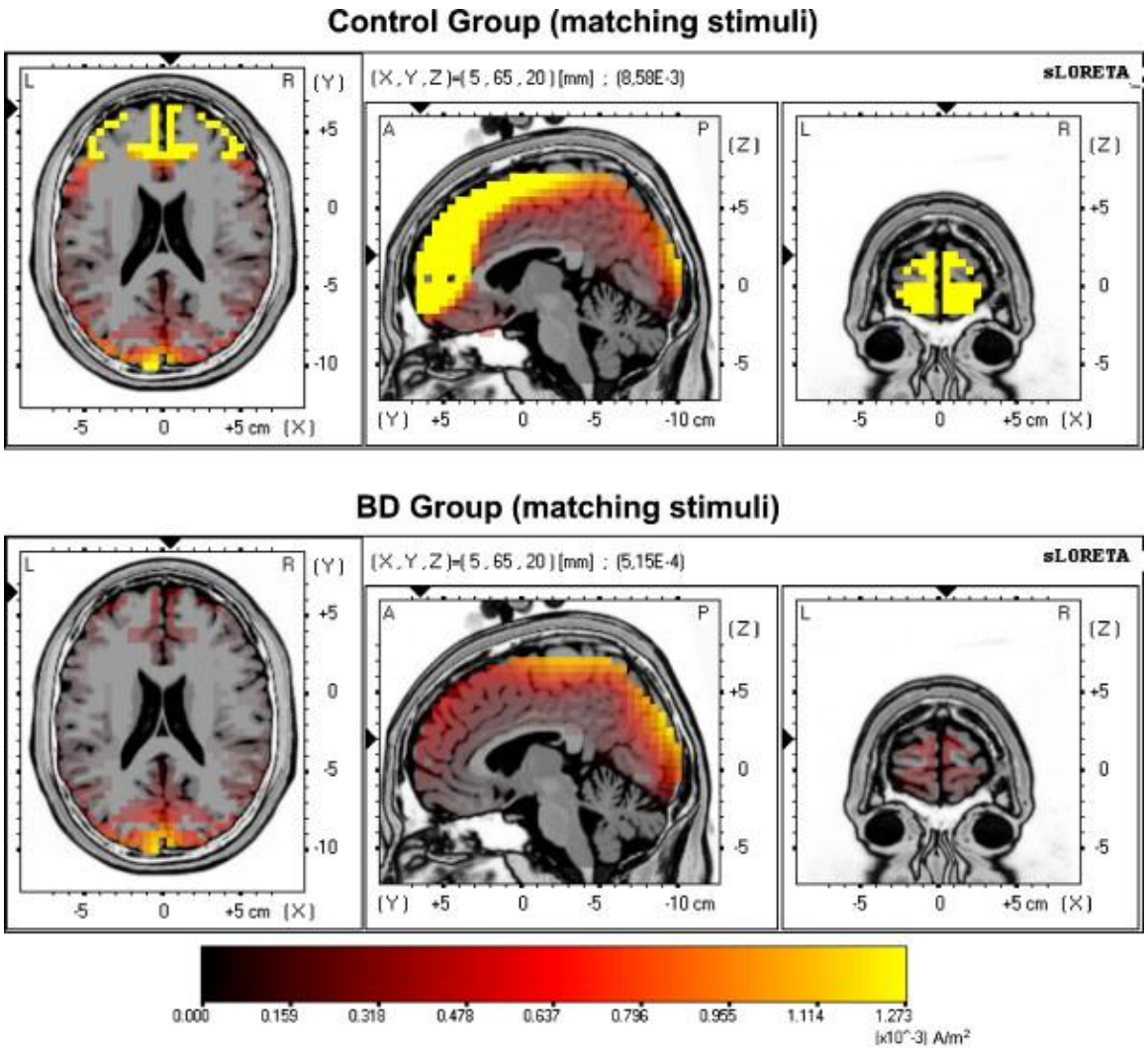
Binge drinkers show less prefrontal lobe activity during visual working memory



Subjects

53 controls
42 binge drinkers (BD)
18-20 years old

Average binge episodes last 2weeks

Controls = 0.2
Binge drinkers = 2

Technique

Low resolution brain electromagnetic tomography

SOURCE: Crego et al. (2010) Reduced anterior prefrontal cortex activation in young binge drinkers during a visual working memory taks. Drug and Alcohol Dependence, 109, 45-56.

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Amount of frontal lobe volume reduction in alcohol dependent adults predicts shorter time to relapse

~ Adolescents have under-developed frontal lobes and alcohol abuse seems to produce damage here – do these factors increase odds of relapse for teens? ~

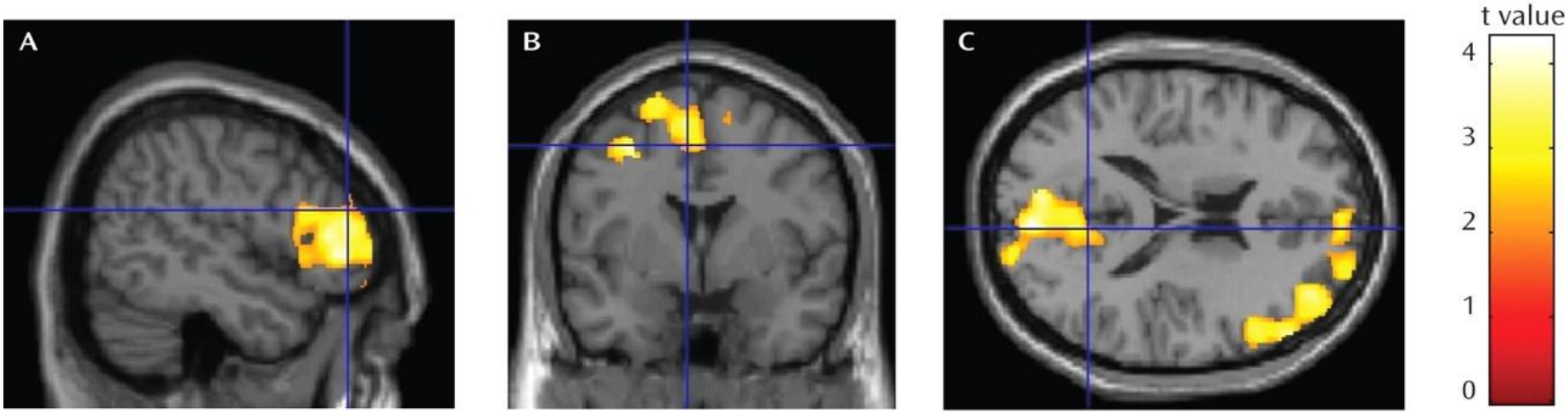

FIGURE 2. Significant clusters of gray matter volume deficit in alcohol-dependent patients (35 men, 10 women, aged 18-50, current DSM-IV alcohol dependence) relative to healthy comparison subjects (28 men, 22 women, aged 18-50)

Rando et al. (2011) Association of frontal and posterior cortical gray matter volume with time to alcohol relapse: a prospective study. Am J Psychiatry;168(2):183-92.

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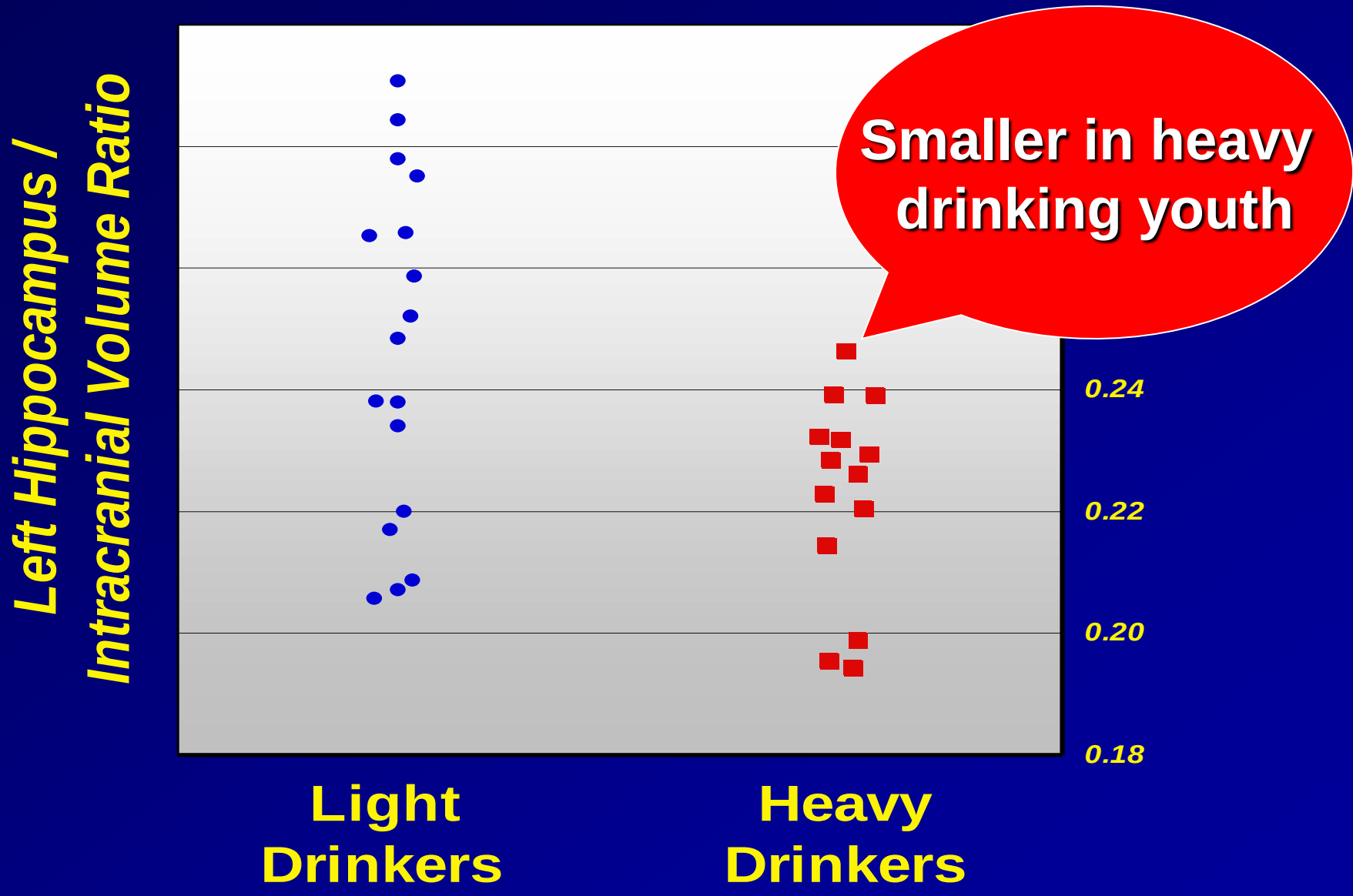
Smaller hippocampus



Sources: Nagel et al., 2005; Medina et al., 2007

Alcohol suppresses hippocampal neurogenesis

- Alcohol suppresses neurogenesis much more so than it kills cells
- Cells born during intoxication abnormal (Nixon et al, 2010)
- Similar amount of suppression of neurogenesis in adolescents and adults but adolescents make more cells, so effect is greater (Nixon et al, 2010)
- In adolescent rats, exercise increases neurogenesis after binge exposure but cells tend to die (Helfer et al, 2009)
- Primates who binge daily for 11 months show impaired neurogenesis 2 months later (Taffe et al, 2010)
- Partial explanation for link between adolescent AUD and memory? depression?

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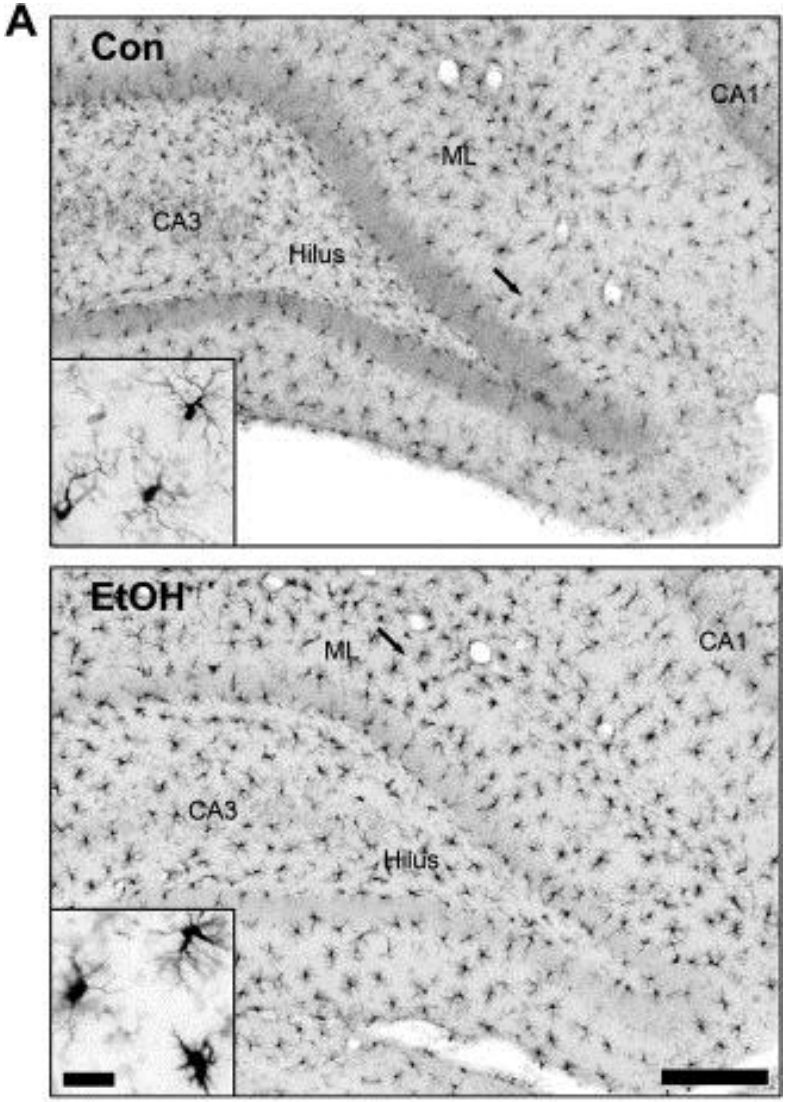
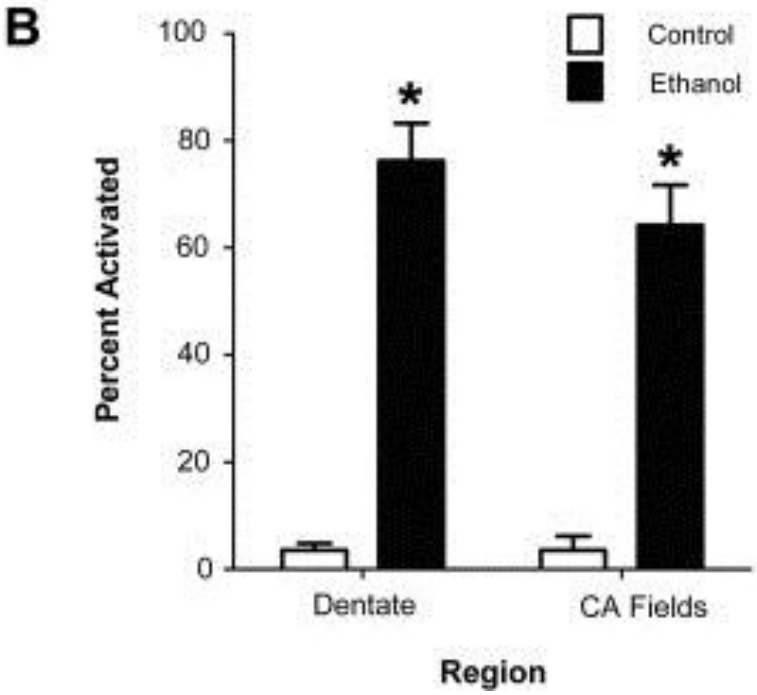
4-day binge alcohol exposure activates microglia in the hippocampus of adolescent rats and activation persists into adulthood





Microglia in control animals had morphological characteristics consistent with resting microglia, while binge ethanol-exposed rats had swollen cell bodies with thicker, less ramified processes, consistent with activation

Could inflammation and immune activity damage brain following binge drinking?

McClain et al. (2011) Adolescent binge alcohol exposure induces long-lasting partial activation of microglia. Brain Behav Immun. 2011 Jun;25 Suppl 1:S120-8.

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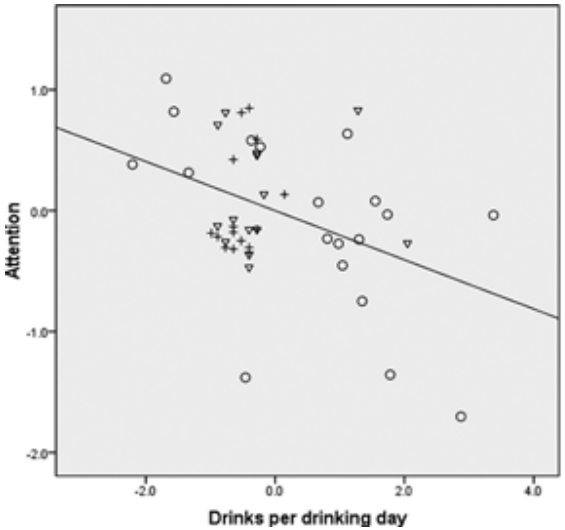
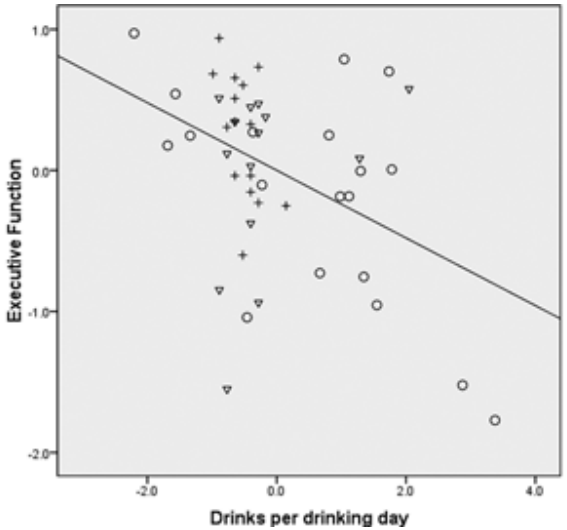
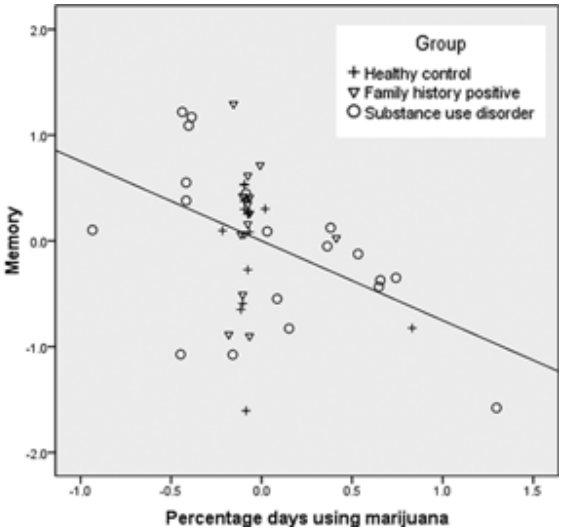
Relationship between levels of alcohol and marijuana use and measures of cognitive function in adolescents

As the number of drinks consumed per day goes up performance on tests of attention, executive function and memory go down. More days smoking marijuana equals poorer memory.







SUBJECTS: 48 adolescents (ages 12 to 18), recruited in 3 groups: a healthy control group (HC, n = 15), a group diagnosed with substance abuse or dependence (SUD, n = 19), and a group with a family history positive for alcohol use disorder (AUD) but no personal substance use disorder (FHP, n = 14).

RESULTS: More DPDD predicted poorer performance on Attention and Executive Function composites, and more frequent use of marijuana was associated with poorer Memory performance. In separate analyses, adolescents in the SUD group had lower scores on Attention, Memory, and Processing Speed composites, and FHP adolescents had poorer Visuospatial Ability.

Thoma et al. (2011), Adolescent Substance Abuse: The Effects of Alcohol and Marijuana on Neuropsychological Performance. Alcoholism: Clinical and Experimental Research, 35: 39–46.

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Summary

- Alcohol risky for adolescents in part because brain is still developing
- Alcohol causes direct effects on adolescent brain that increase risk of accidents and bad decision-making
- Long-term lingering effects on brain including heightened risk of alcoholism and cognitive deficits
- Goal should be to delay onset and model/promote moderate use

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