The Biology of Adolescence

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Summary

Certain adolescent-typical behaviors, including risk-taking behaviors, may be in part biologically driven by a brain that reacts differently to stimuli due to developmental transformations in relatively ancient brain systems

Adolescent risk-taking may be enhanced by:

- Restructuring of key elements of these brain *reward* and *affective* systems beginning early in adolescence, contributing to:
  - Exaggerated reactivity to certain emotional stimuli and rewarding effects, along with sometimes attenuated sensitivity to aversive effects
- Insufficiently countermanded by slowly maturing frontal cognitive control

Adolescence from an evolutionary perspective

- Gradual transition between dependence/ immaturity and relative independence/maturity
- Common goals:
  - to gain skills necessary for survival as adults and reproductive success
- Conserved characteristics:
  - Biological changes: puberty, growth spurt, etc
  - Neural transformations
  - Certain age-typical behavioral features

⟶⟶***IMPORTANT QUALIFIERS***⟵⟵

Adolescent behaviors commonly seen across species

- ***Increases in social interactions/peer affiliations***

  (sometimes assoc. with increase in parent/adol. conflict)

  – **Develop social skills/support**

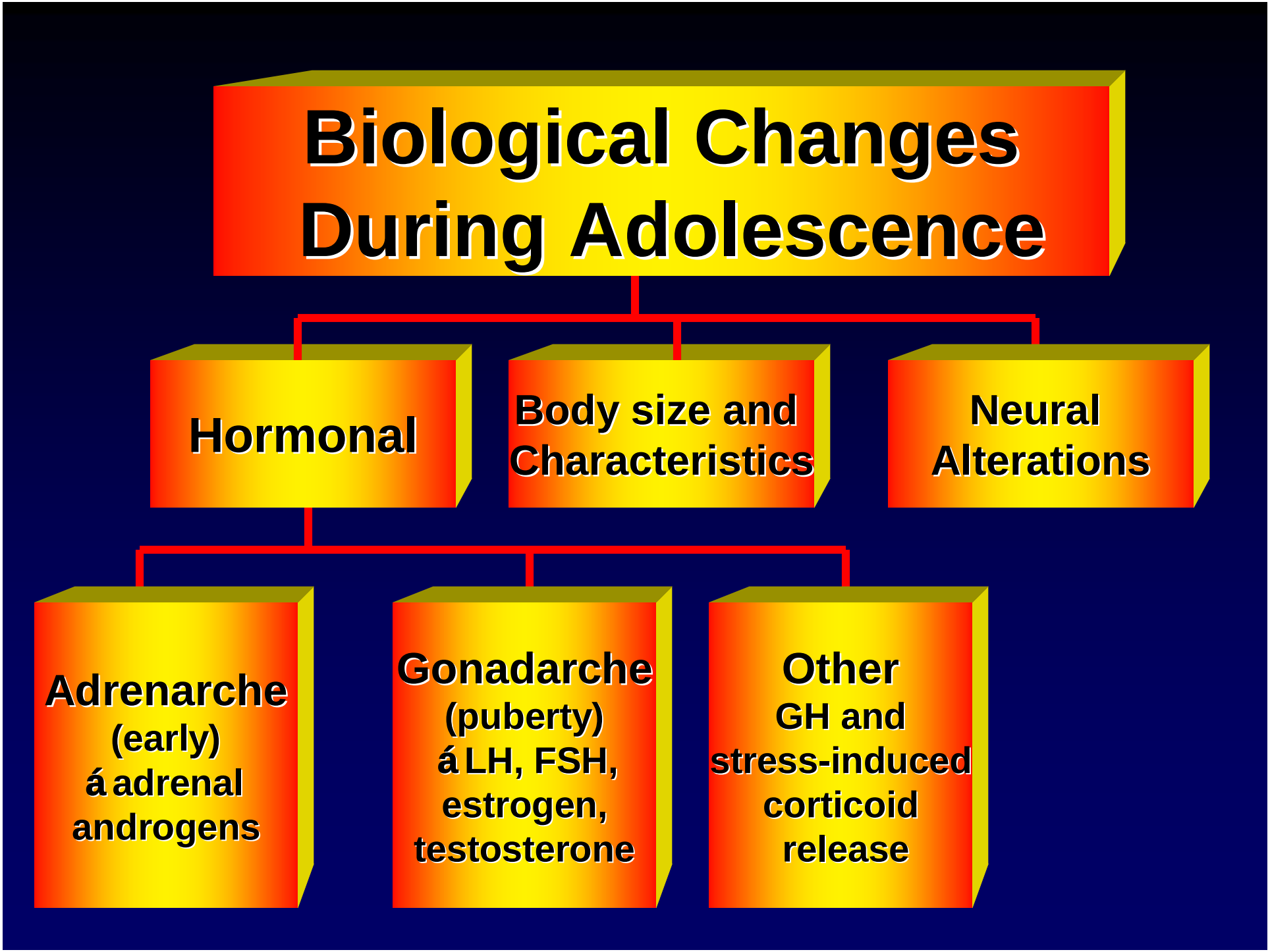
  – **Guide choice behavior**

- Increases in risk-taking, novelty-seeking, sensation-seeking (including drug/alcohol use)

  – Enhance prob. of reproductive success

  – Foster peer acceptance

  – Impetus to explore new territories; aid in emigration

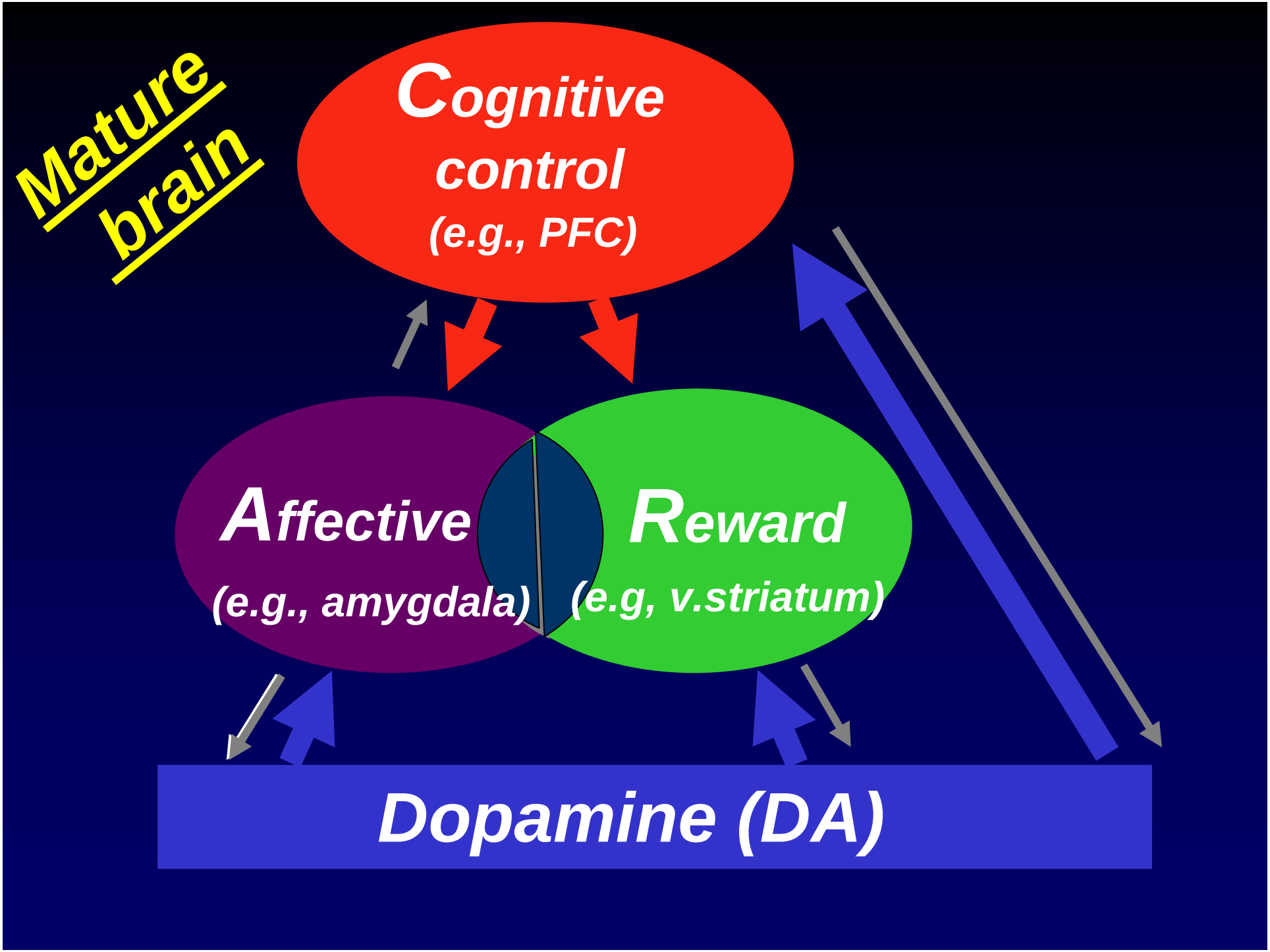

Biological Changes During Adolescence
Hormonal
Body size and Characteristics
Neural Alterations
Adrenarche (early) á adrenal androgens
Gonadarche (puberty) á LH, FSH, estrogen, testosterone
Other GH and stress-induced corticoid release

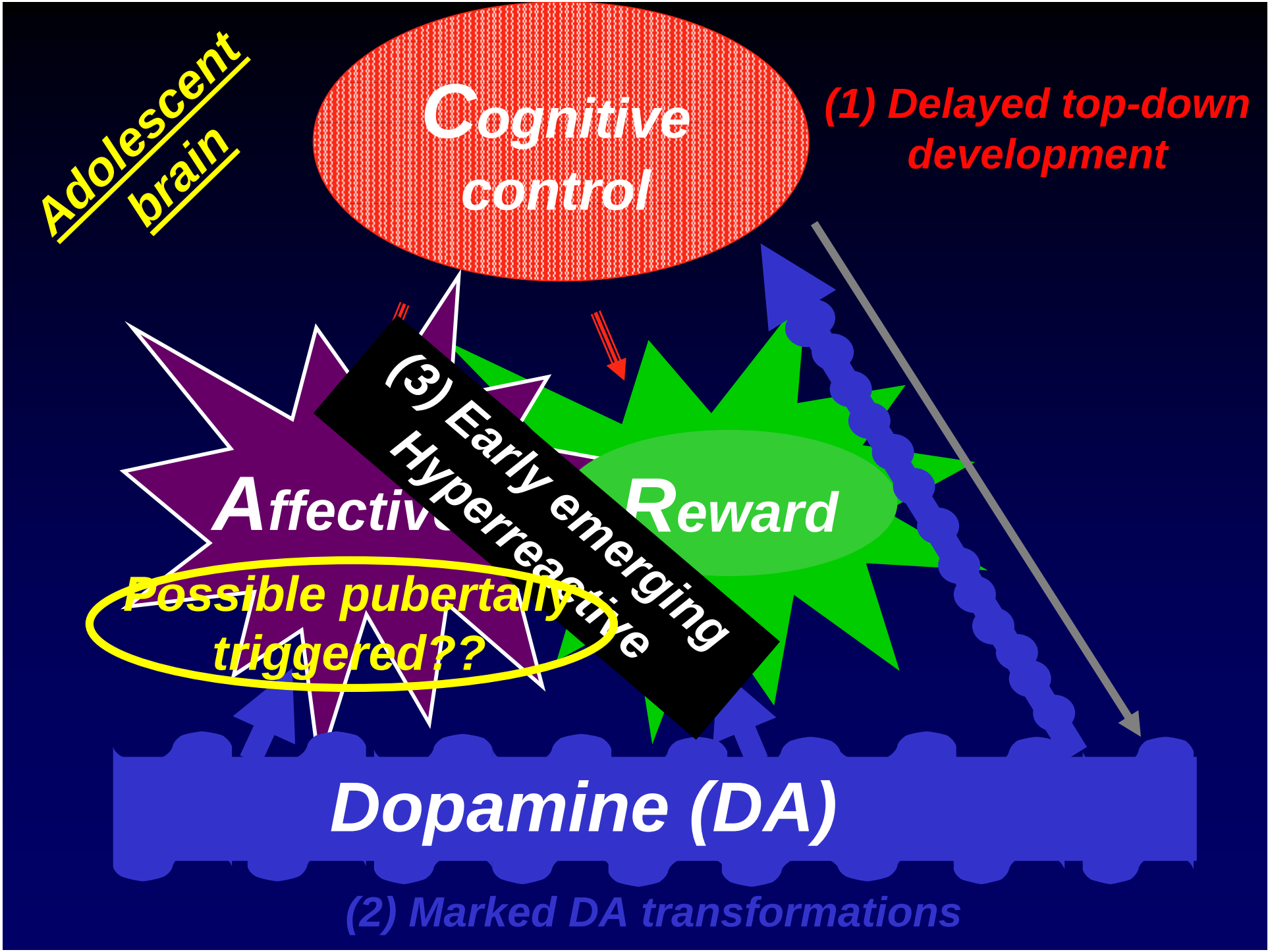
Adolescent brain transformations:

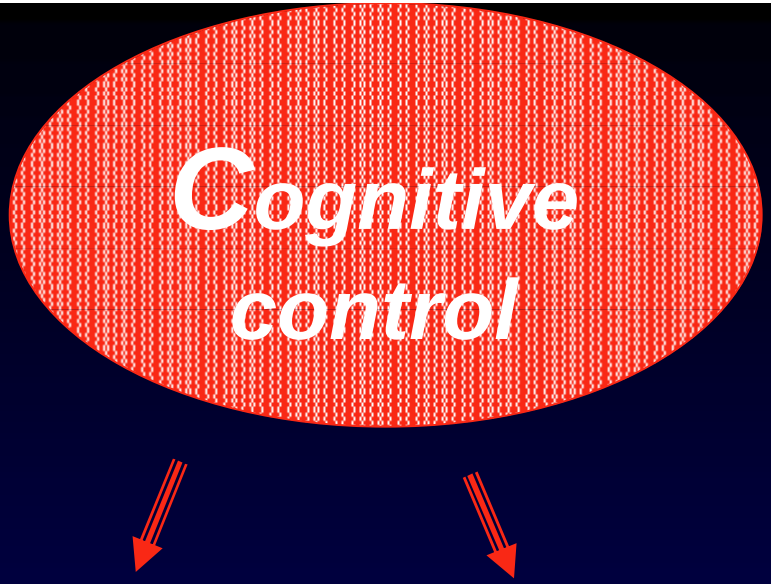
- Regressive changes:
  - Synaptic pruning (experience-influenced?)
  - Regionally-specific apoptosis
  - Declines in gray matter volume
- Increase in white matter volume; myelination
  - Experience-influenced? (activity-dependent component)
- Decline in brain energy utilization
- Region- and system-specific alterations
  - Notable in forebrain cortical and subcortical regions (but seen elsewhere as well – e.g., cerebellum)

Forebrain Changes in Adolescence

- **Prefrontal cortex (PFC)**
  - Reduced excitatory drive (humans, primates, rodents)
  - Decrease in PFC volume (humans & rodents)
  - Peak of DA innervation (humans, primates, rats)
  - High DA turnover early followed by decline (rats)
  - Delayed loss of DA receptors (rats; humans)
- **Ventral striatum (nucleus accumbens); dorsal striatum**
  - Low DA turnover early followed by increase (rats)
  - Marked loss of DA receptors early in adolescence (rats; humans)
- **Hippocampus**
  - **á** sprouting and myelination (humans, rodents)
- **Amygdala**
  - **á** activity (humans, rodents) and PFC connectivity (rodents)


Mature brain
Cognitive control
(e.g., PFC)
Affective
(e.g., amygdala)
Reward
(e.g, v.striatum)
Dopamine (DA)

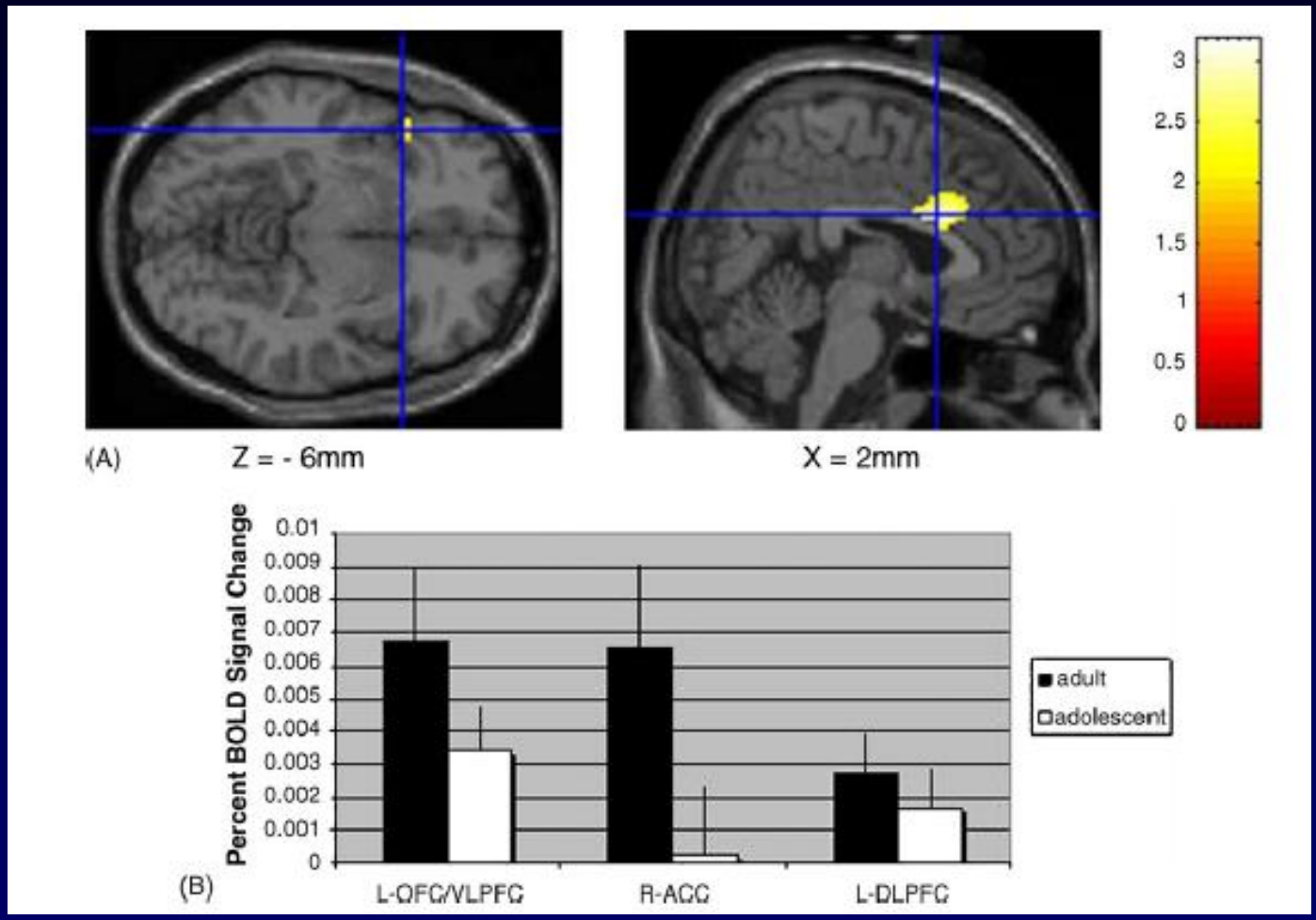

Adolescent brain
Cognitive control
(1) Delayed top-down development
Affective
Reward
(3) Early emerging Hyperreactive
Possible pubertally triggered??
Dopamine (DA)
(2) Marked DA transformations



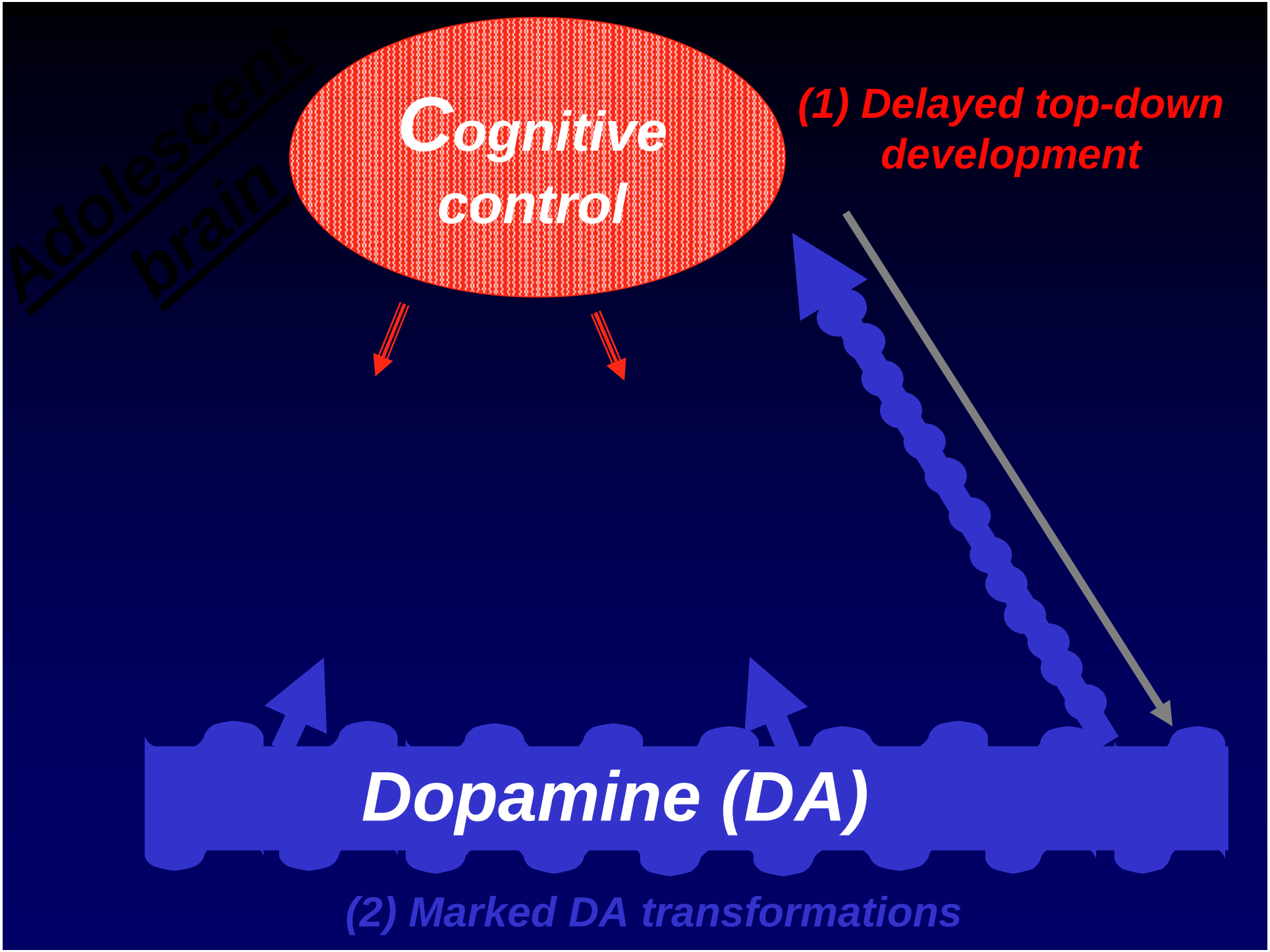
(1) Delayed top-down development

Cognitive control

Signal change: high – low risk conditions



[Eshel et al (2007) 45: 1270-1279]


Adolescent brain
Cognitive control
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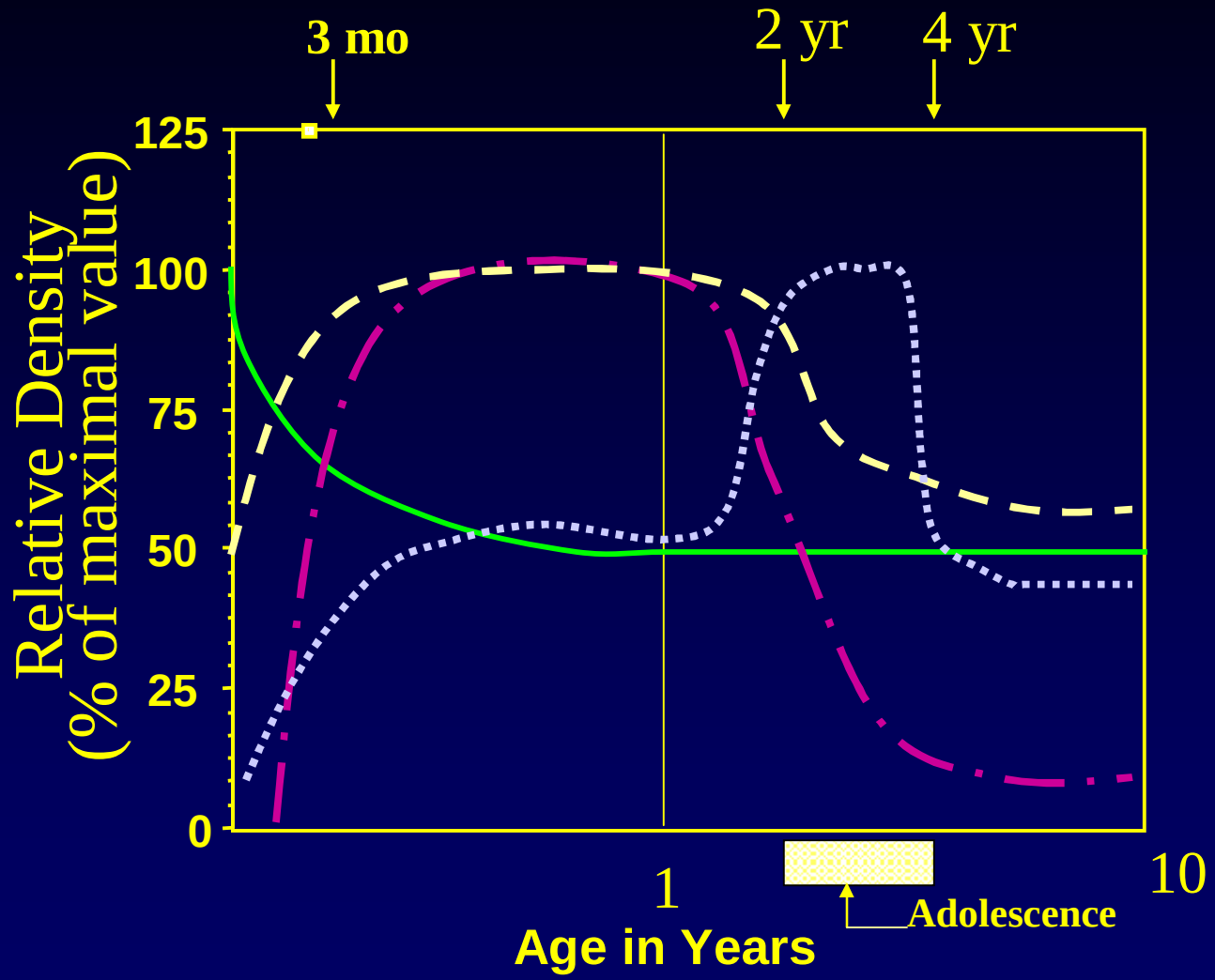
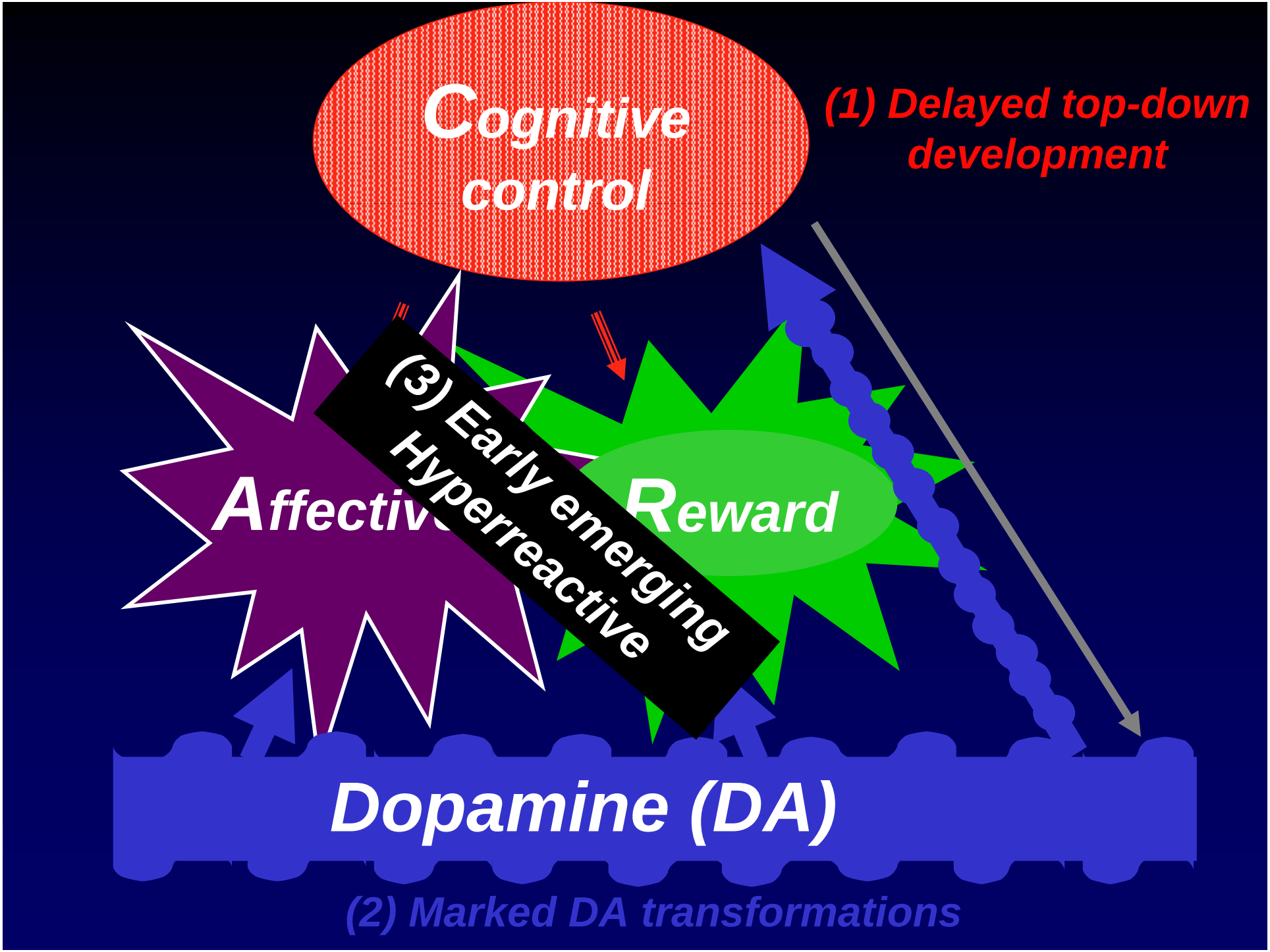
Dopamine (DA)
(2) Marked DA transformations

Ex.: DA alterations in adolescence:



[Lewis, DA (1997) Neuropsychopharmacology, 16:385-398]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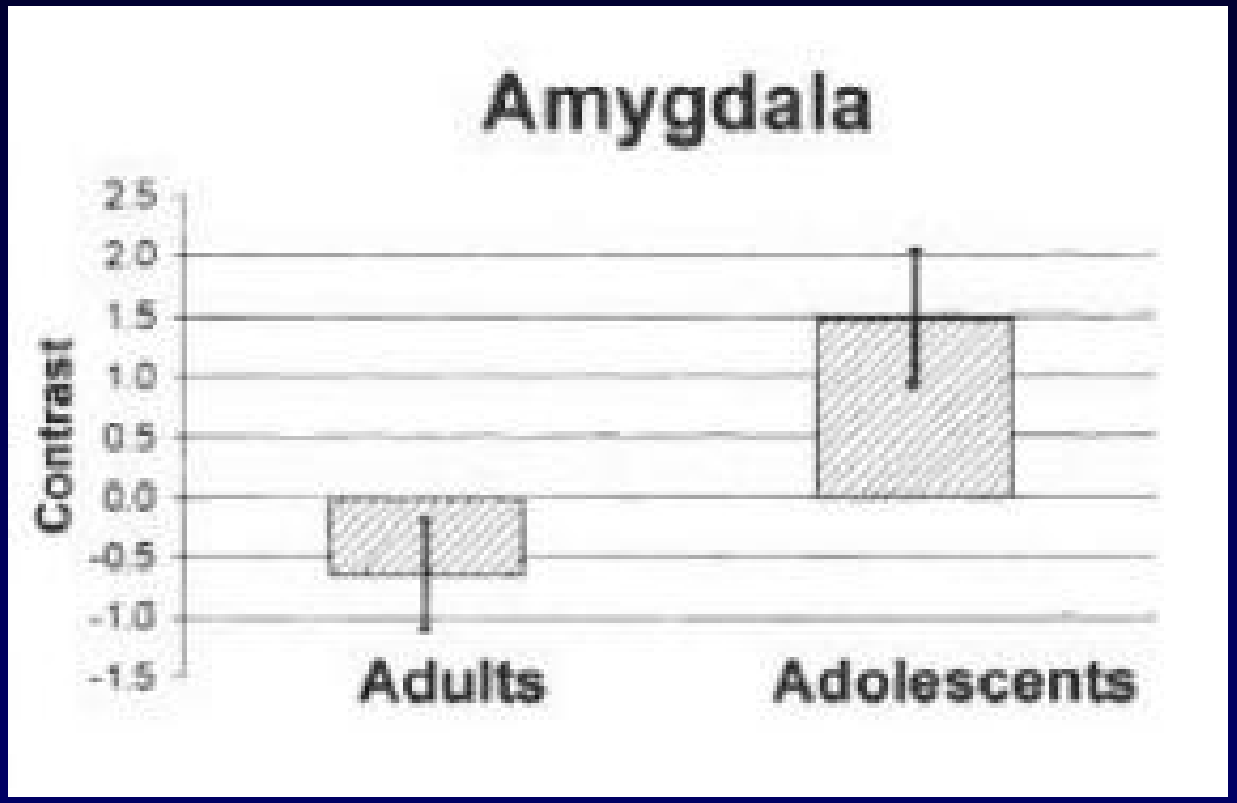
Affective system –
amygdala responding to:

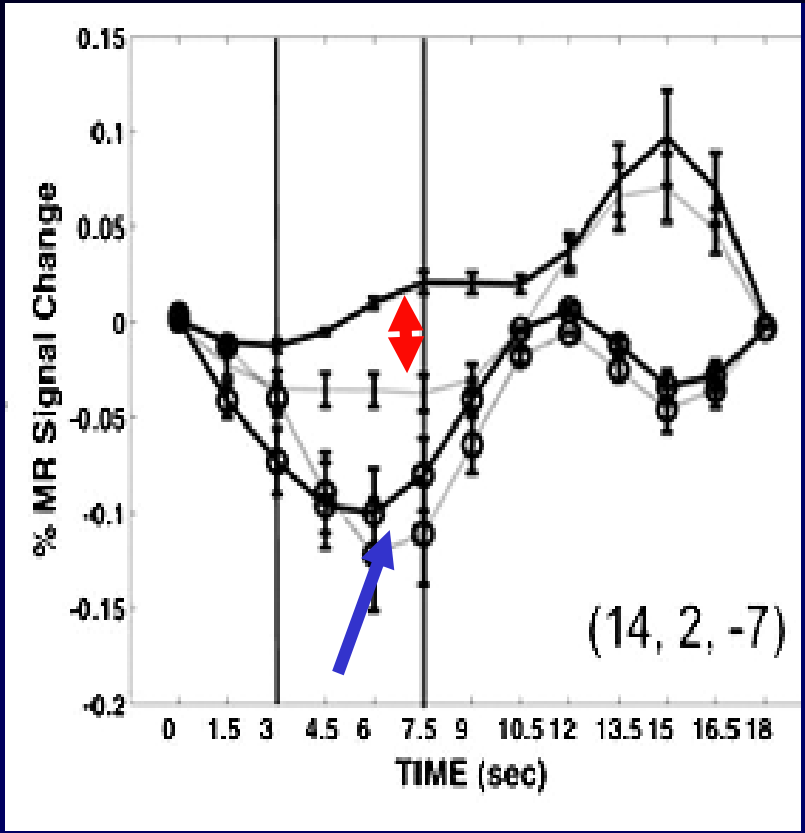
Fearful relative to neutral faces



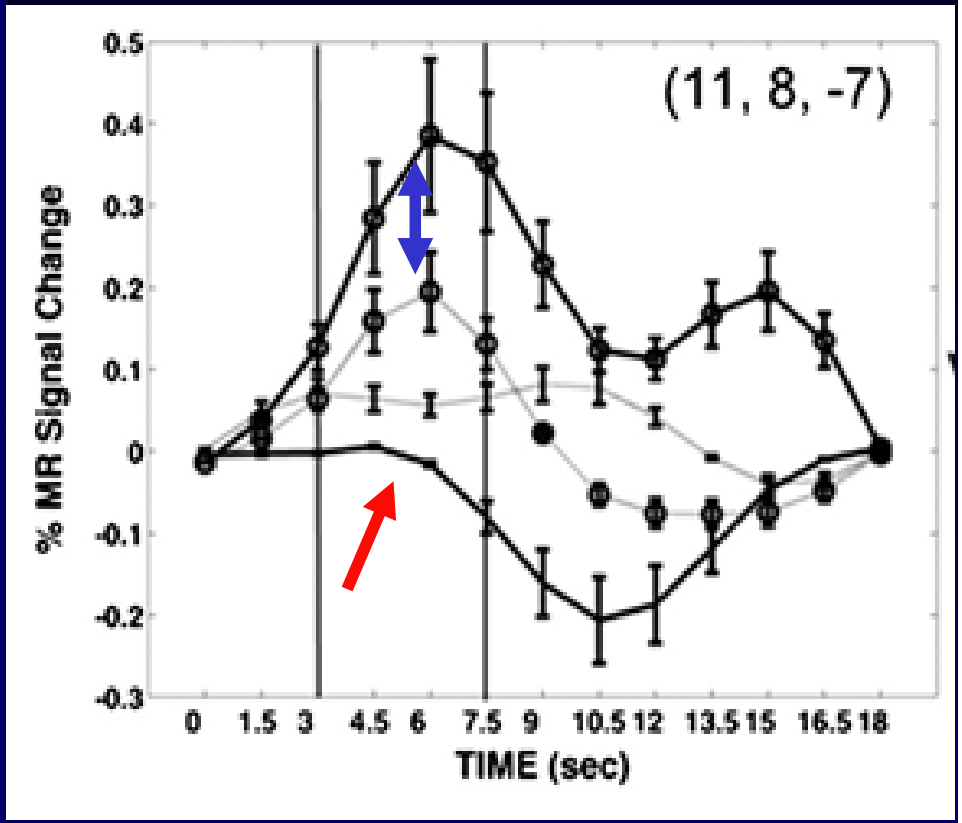
[Monk et al (2003), NeuroImage, 20: 420-429]

Reward system – v. striatum responding to:

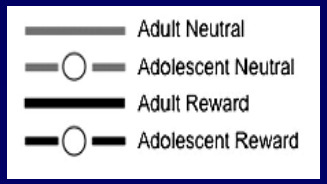
incentive cue



response preparation

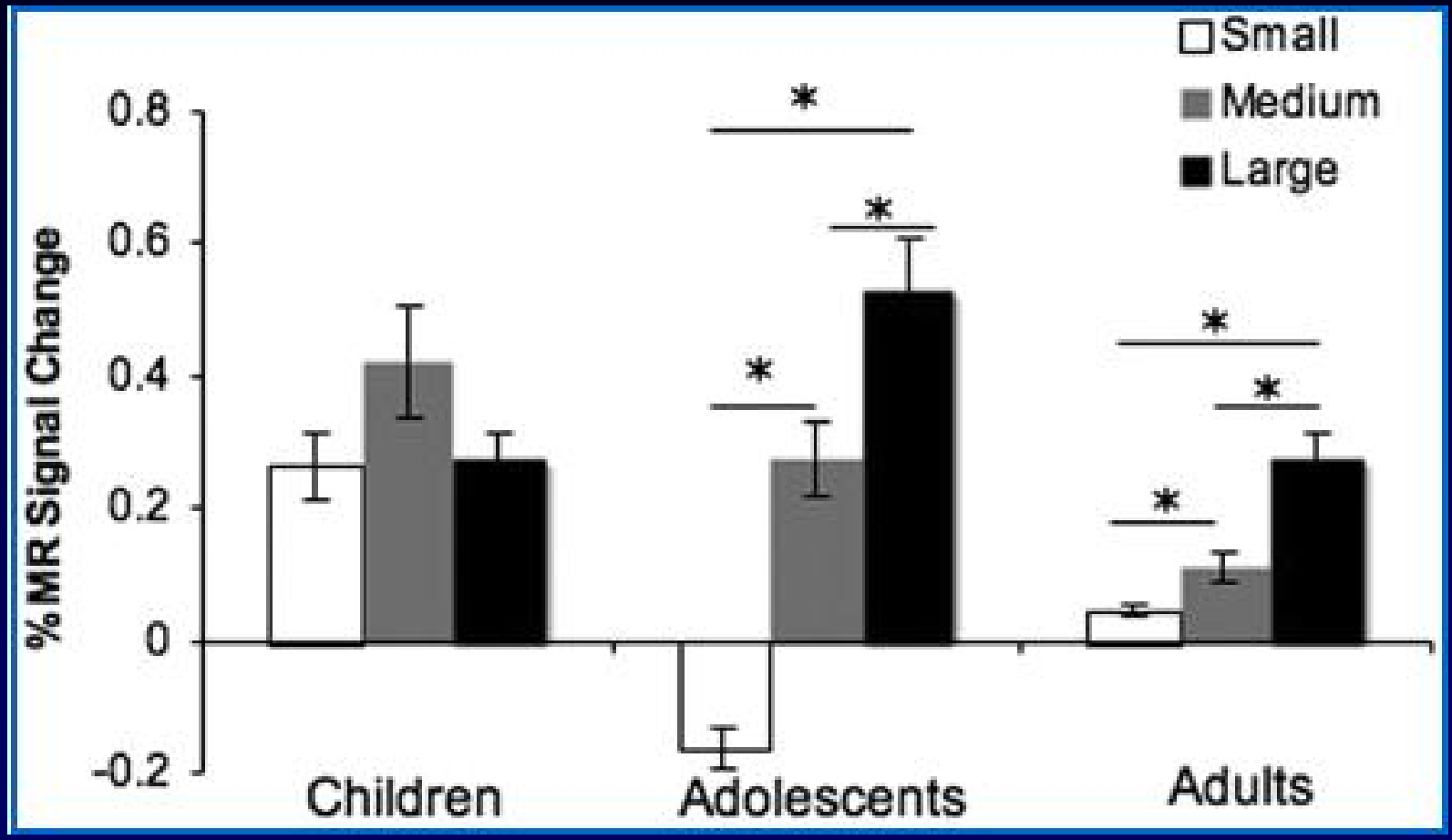


Adult reward
Adolesc. reward



[Geier et al (in press) Cerebral Cortex]

Reward system – v. striatum responding to:
differences in reward magnitude



[Galvan et al (2006) J. Neuroscience, 26: 6885-6892]

Adolescent-typical reward/aversive biases

↑ Rewarding effects of:

adolescents> adults

ETOH:

- Pautassi et al (2008)
- Ristucci & Spear (2008)

NICOTINE:

- Vastola et al (2002)
- Shram et al (2006)
- Torres et al (2008)

COCAINE/AMPHETAMINE:

- Badanich et al (2006)
- Brenthouse et al (2008a,b)
- Zakharova et al (2008a,b,)
  - [but see Aberg et al, 2007; Campbell et al, 2000]

↓Aversive effects of:

adolescents< adults

CTA and CPA for

ETOH:

- Anderson et al (2008)

NICOTINE:

- Wilmouth & Spear (2004)
- Shram et al (2006)
- Torres et al (2008)

AMPHETAMINE:

- Infurna & Spear (1979)

Adolescent-typical reward/aversive biases:

Reflective of more general age-related shifts in sensitivity to stimuli?

In animal studies, adolescents show:

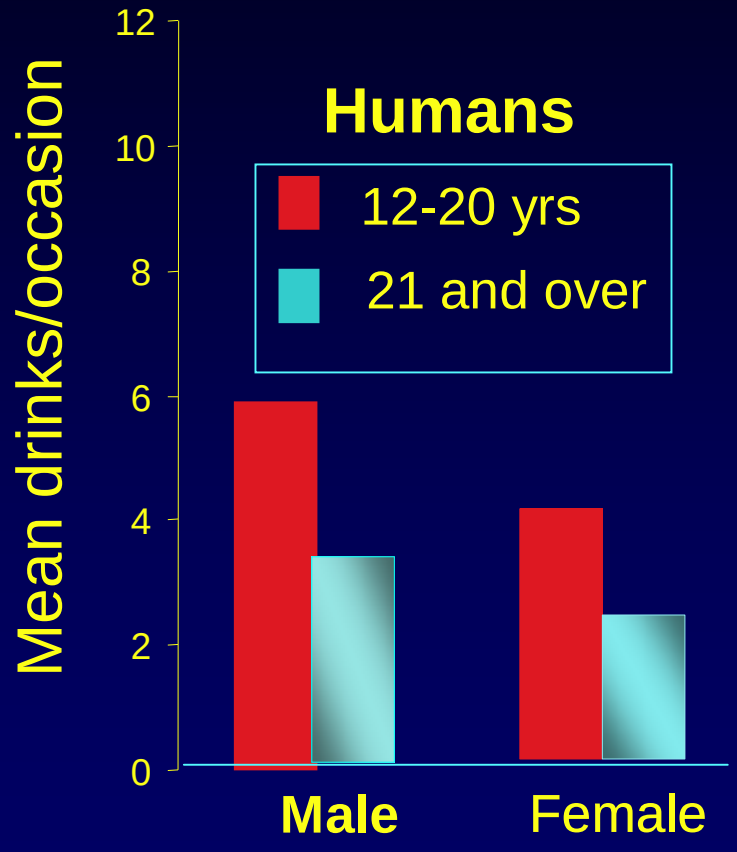
- ***Greater sensitivity to alcohol-induced:***
  - **Rewarding effects**
  - **Social Facilitat**
  - **Impairme LTP + spatial memory tasks**
- ***Less sensitivity to alcohol effects:***
  - **Aversive effects**
  - **Social inhibition**
  - **Sedation**
  - **Motor impairment**
  - **Analgesia**
  - **Hangover effects**

Relevance to human adolescents???

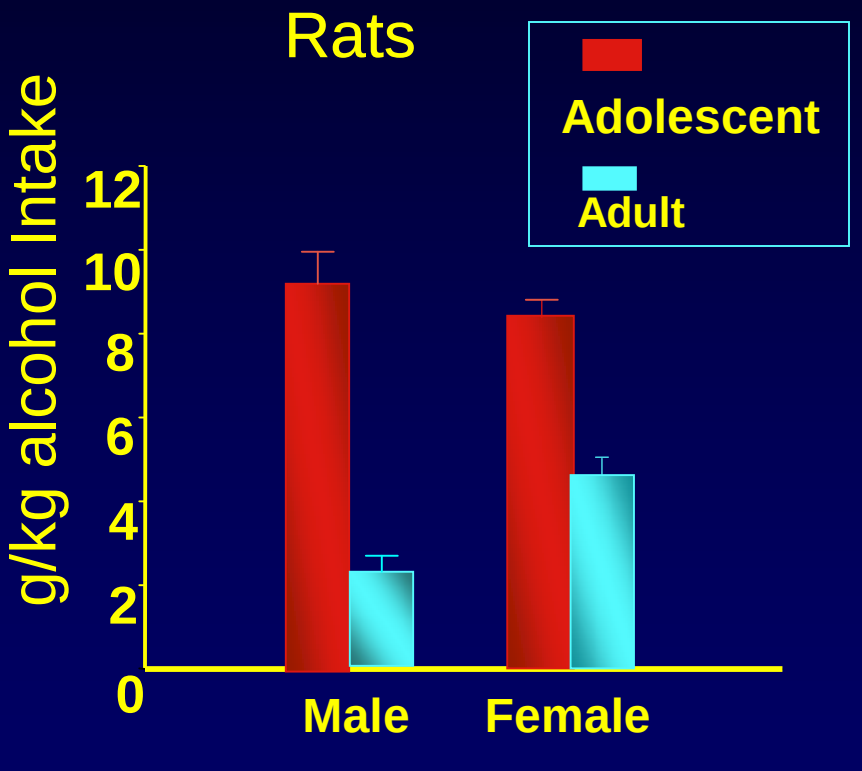
In human adolescents during first intoxicating experience with alcohol:

- 8-15 yrs old; given 0.5 ml/kg; pk. BALs of 34-35 mg%
- In this study of the first intoxicating experience of naïve children, little behavioral change was noted clinically, subjectively, or on a validated objective test of intoxication…"
- "…these children exhibited a smaller behavioral change than expected for their BAL"
- "None behaved grossly 'intoxicated' as our adult…subjects were"
- "We were impressed by how little gross behavioral change occurred in the children…after a dose of alcohol which had been intoxicating in an adult population." ***[Behar et al (1983), ACER, 7:404-410]***

Suggests biological contribution to enhanced alcohol intake in adolescence:



(SAMHSA Survey data, 2003)



(Doremus et al. 2005)

Impact of attenuated alcohol sensitivity for adolescents?

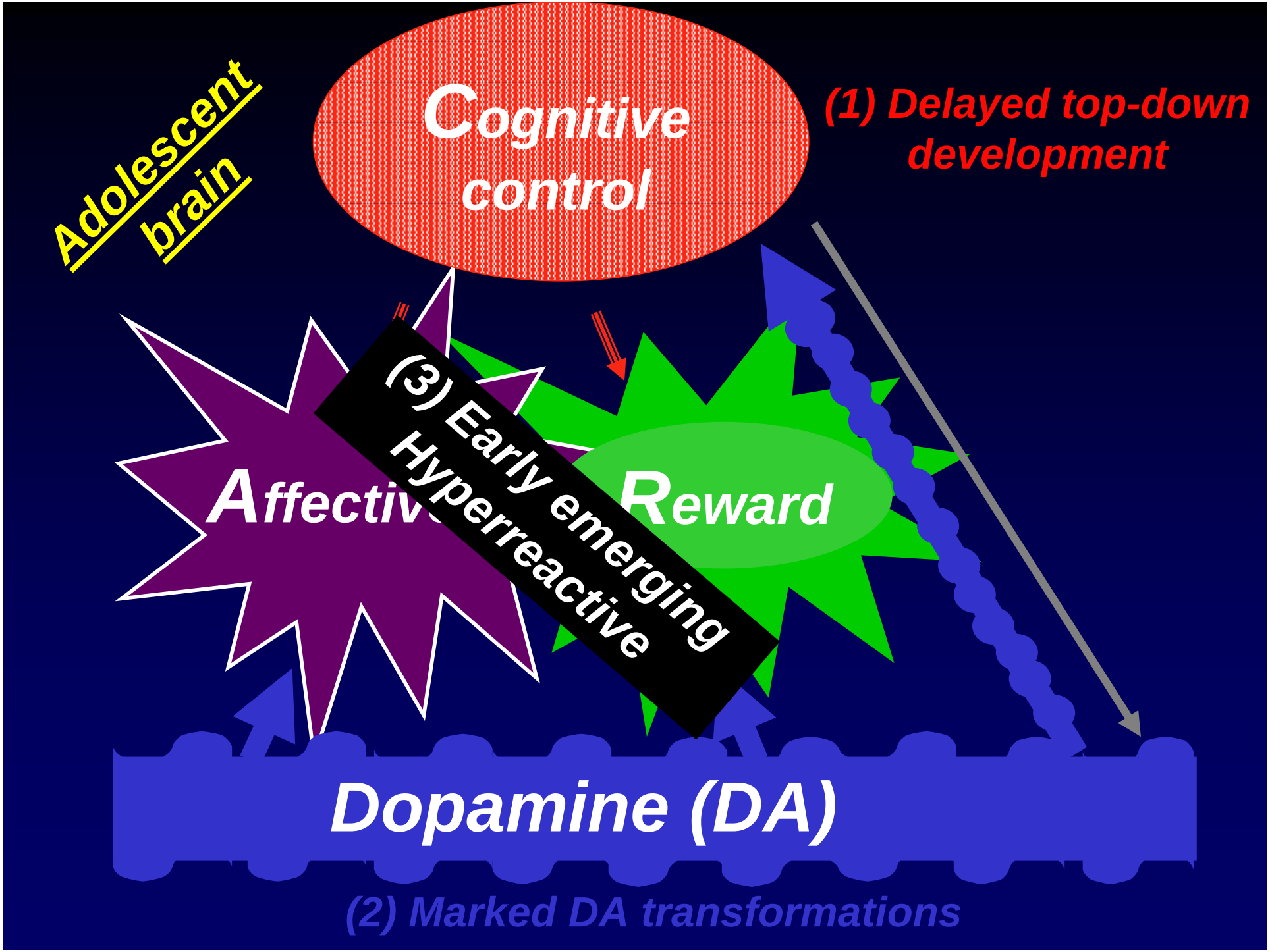
Decreased alcohol sensitivity: known risk factor for problematic EtOH involvement

Lowered sensitivity to EtOH seen:

- In individuals with genetic vulnerability for alcoholism
- Developmentally: in adolescence
- Possibly as a function of prior experience **à** prior ethanol use; stress during adolescence; other exps.

May serve as a permissive factor for elevated intake

- Potentially leading to enhanced long-term effects and an increased susceptibility to abuse disorders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Bottom-Line

Adolescent-typical behaviors may be in part biologically driven by a brain that reacts differently to stimuli due to developmental transformations in relatively ancient brain systems

These adolescent-typical characteristics include:

- Marked transformations in DA systems
- Enhanced amygdala reactivity to emotional stimuli
- Exaggerated reactivity to rewarding effects
- Reduced sensitivity to aversive effects
- All insufficiently countermanded by immature frontal cognitive control, and

Collectively contributing to adolescent risk-taking