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Aristotle “Youth are heated by Nature as drunken men by wine”
Shakespeare “I would that there were no age between 10 and 23, for there is nothing in between but getting wrecks with child, wronging the ancenetry, stealing, fighting…..” The Winter’s Tale, Act. III.

80% of adolescents navigate through ADOLESCENCE TRANSITIONS (from puberty to adult responsibilities) without major problems.

END OF ADOLESCENCE?: Stabilized sleep chronotypes closing the typical delayed pattern: around 19.5 years old in women and 21 years old in men.
**FACTORS MADURATIUS I ENTRADES AMBIENTALS**

- Genes
- Hormones
- Ambient
- Desenvolupament
- Comportament adolescent conflictu

**The Adolescent Brain**

*Risky choices*: blending of higher sensitivity-maturation of limbic structures with delayed prefrontal maturation

*Impulsiveness*: prefrontal protracted development alone


**Key brain regions mediating Aggressive Emotions**

- Prefrontal regions (yellow)
- Limbic regions (red)
- Posterior Hypothalamus (blue)
- Dorsolateral prefrontal cortex (violet)
- Ventromedial (brown) and orbital prefrontal cortex (green)

*Davidson RJ et al (2000), Science, 289, 591-594*
Puberty/adolescence main regulators: Hypothalamic-Hypophysis-Gonadal interactions


Mean youth values of steroid hormones in each sex

<table>
<thead>
<tr>
<th></th>
<th>Females (N=81)</th>
<th>Males (N=102)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Follicular</td>
<td>Luteal</td>
</tr>
<tr>
<td>DHEAS (ng/ml)</td>
<td>1.32 (0.64)</td>
<td>1.3 (0.62)</td>
</tr>
<tr>
<td>AD (ng/ml)</td>
<td>3.6 (2.56)</td>
<td>5.99 (2.76)</td>
</tr>
<tr>
<td>DHEA (ng/ml)</td>
<td>6.24 (5.5)</td>
<td>3.2 (3.3)</td>
</tr>
<tr>
<td>LH (mIU/ml)</td>
<td>15.06 (6.17)</td>
<td>30.57 (17.6)</td>
</tr>
<tr>
<td>T (ng/ml)</td>
<td>0.6 (0.4)</td>
<td>0.73 (0.4)</td>
</tr>
<tr>
<td>TEBG (nmol/l)</td>
<td>57.8 (21.7)</td>
<td>67.4(33.9)</td>
</tr>
<tr>
<td>FTI</td>
<td>0.04 (0.04)</td>
<td>0.04 (0.03)</td>
</tr>
<tr>
<td>P (ng/ml)</td>
<td>1.08 (1.23)</td>
<td>4.53 (5.73)</td>
</tr>
<tr>
<td>E (pg/ml)</td>
<td>0.001 (0.001)</td>
<td>0.019 (0.015)</td>
</tr>
</tbody>
</table>

DHEA= Dihydroepiandrosterone; AD: Androstenodione; LH: Luteinizing hormone; T= Testosterone; TEBG: Testosterone binding globuline; FTI: Free testosterone; P: Progesterone; E: Estradiol.

From Udry (1990).

The "trouble" with salivary testosterone

Psychoneuroendocrinology, 29, 1229-1240

Mandela’s kids study in South Africa:

STAGES OF HUMAN BRAIN DEVELOPMENT


Normal cortical maturation

Insel Th (2010), Nature, 468, 187-193

Development of synaptic density in sensory and frontal regions. The left-hand graph shows the mean synaptic density in the primary auditory cortex (red circles), in the primary visual cortex (green circles) and in the prefrontal cortex (PFC: middle frontal gyrus, crosses) in postmortem human brains at different ages. The x-axis shows the conceptual age in days from 200 postconception to 10,000 days postconception (approx. 27 years). Synaptic density increases in all three regions in early childhood but synaptogenesis is most prolonged in the PFC. This is further demonstrated on the right graph which shows the difference in synaptic density between the auditory cortex and PFC for each year postconception plotted against conceptual age. The line represents a linear regression which suggests that peak synaptic density in the auditory cortex occurs early (approx. three months after birth) while the peak synaptic density in the PFC occurs significantly later.
Scanning adolescent’s brains

EXPLORING BRAIN CHANGES WITH MAGNETIC RESONANCE IMAGING (MRI) SCANS

Changes on cortical thickness (gray-matter density) in both hemispheres across lifetime.

Points correspond to regional measures taken on 176 individuals with ages ranging from 7 to 87 years old.

CHANGES IN GRAY MATTER DENSITY ON CORTICAL REGIONS ACROSS AGES


Saguenay Lac St. Jean (Quebec) study of adolescent brains

Pauss T et al (2010), Hormones and Behavior, 57, 63-75.
Why do many psychiatric disorders emerge during adolescence?


Subjects:
- 16 children (7-11 years old; seven females)
- 13 adolescents (13-17 years old; six females)
- 12 adults (23-29 years old; six females)

Payment: 50 $ for participating + 25 $ in variable earnings!!
Earlier Development of the Accumbens Relative to Orbitofrontal Cortex Might Underlie Risk-Taking Behavior in Adolescents.


Figure 2: Localization of adverse reactions in all and right frontal cortex (dotted lines represent T < 0.001; lateral ventricle seen as a white area). The left and right lateral ventricles were removed to show the white matter. The right lateral ventricle is visible in the right hemisphere, and the left lateral ventricle is visible in the left hemisphere.

Figure 3: Comparison of adolescent and adult amygdala activity. The amygdala is located in the ventral portion of the temporal lobe, just anterior to the thalamus. The amygdala is involved in the processing of emotional stimuli and is thought to play a role in the development of risk-taking behavior.


Risky choices: blending of higher sensitivity/maturation of limbic structures with delayed prefrontal maturation

Impulsiveness: prefrontal protracted development alone

Adolescent agressiveness: Madurative and Environmental Inputs: from early maltreatment to drug/toxic insults
Pathways to individual aggressiveness


The UNESCO Courier, 1993 (p.40).

RELATIONSHIPS OF ANDROGENIC STEROIDS WITH REPRODUCTIVE FUNCTION, SEXUAL BEHAVIOR AND AGGRESSION.
The discrete character of high-lethality of youth violence
