I. INTRODUCTION

A. Puberty and Risk Taking

- Dahl agrees with Arnett’s revision of Hall’s view of the impact of puberty on adolescent risk taking
- He sees adolescent as unique period of time and risk taking as an essential feature of what makes it unique.
- Theory: Biological and social-cognitive-cultural aspects of puberty’s impact on adolescent risk taking
- Research: Importance of interdisciplinary research on puberty
  - Integrating biological with cognitive, social, and cultural research
  - Clinical and Basic research; Experimental, developmental, and correlational research

- Dahl’s point is that puberty makes adolescents vulnerable to risk taking.
- Increase in emotional and motivational tendency towards risk taking and sensation seeking.
- Increase appetite for emotional intensity, excitement and arousal.
- Forces are exacerbated by the greater length of adolescence and the freedom they experience.
- Greater amount of time and less control between sexual maturity and adopting adult responsibilities (job, marriage, etc.).

- Last week: Genetic basis for risk taking
- Shared and unique genetic heritage interact with environments to produce risk taking behavior in adolescence.
  - The interaction depends on the type of environment; The nature of the genotype; Age; Freedom to choose environments.
- Risk taking also affected by biological changes associated with puberty (Latin for to grow hairy)
- Dahl’s definition of Adolescence corresponds with Amsel’s definition
  - That awkward period between sexual maturation and the attainment of adult roles and responsibilities.
I. INTRODUCTION

B. Biological Vulnerabilities to Risk Taking

- Dahl claims that the puberty (not age) specifically results in adolescents’ inclination toward novelty, arousal, and excitement.
  - Recognizes that puberty creates opportunities
  - Harness emotions in the service of positive goals
  - Also recognizes that it opens up vulnerabilities.
  - Increasing appetite for intense emotions.
  - Notes that changes associated with puberty creates trajectories for life time problems.
  - Sounds like the now familiar exploration vs. problem behavior defined in terms of adolescent-limited vs. life-course-persistent patterns of risky behavior.

- Dahl offers a general explanations of why puberty creates vulnerability to risk tasking.
  - He notes that puberty is not a single event, but multiple, somewhat independent processes.
  - There are two fundamental changes: Hormonal Changes and Brain Changes.
  - Dahl characterizes the forms of relations between these two changes.
    - Certain brain changes CAUSE hormone changes (upstream changes)
    - Certain brain changes are the CONSEQUENCE of hormonal changes (downstream changes)

I. INTRODUCTION

C. Lecture

- Lecture will address two aspects of puberty:
  - Hormonal changes
    - Physical Maturation
    - Sexual Maturation
  - Hormone behavior relations
    - Sussman
    - Garber
  - Brain changes
    - Aspects of Brain Development
    - Neurotransmitters and risk taking vulnerability
      - Fairbanks
    - Neuro-cognitive development and risk taking vulnerabilities
      - Overman
II. HORMAL CHANGES IN PUBERTY

A. Endocrine system

- Hormonal changes during puberty begins when Hypothalamus in the brain causes the Pituitary Gland to release or increase production of certain hormones (upstream changes)
  - The Hypothalamus regulates basic biological functions as eating, sleeping, and the experience of pleasure.
  - It is also in direct contact with the Master gland of the Endocrine System (click here for details) the Pituitary gland.
  - Together the Hypothalamus and Pituitary regulate homeostasis.

- There are a number of steps in the process of becoming sexually mature.
  - Hypothalamus releases GnRh (gonadotropin releasing hormone)
  - This stimulates the pituitary gland to produce two gonadotropins -- FSH (follicle stimulating hormone) and LH (luteinizing hormone).
  - The gonadotropins cause the gonads (testes in males, ovaries in females) to produce sex hormones.

B. Sexual Maturity

- The sex hormones include:
  - Males: Testes (males) produce androgens. The androgens (most importantly testosterone) cause muscle growth, body and facial hair, and other sex characteristics in boys.
  - Females: Ovaries (females) produce estrogens. The estrogens (most importantly estradiol) cause girls' breasts, uterus, and vagina to mature, the body to take on feminine proportions, fat to accumulate, and menstrual cycle to regulate.

Levels of sex hormones are regulated by a negative feedback system. If the level of sex hormones rises too high, the Hypothalamus reduces GnRH and the Pituitary reduces LH and FSH. If the hormone level is too low, the Hypothalamus increases GnRH and the Pituitary increases LH and FSH.
In addition to regulating sex hormones, the Pituitary Gland is also involved in the regulation of growth hormones.

The pituitary releases hormones to produce:
- **ACTH** (Adrenocorticotropic hormone) which stimulates adrenal glands to make androgens.
- **Adrenal androgens** influence the girl’s height spurt and stimulate growth of underarm and pubic hair.
- **Growth hormone** (GH) and **thyroxine**, regulated by the thyroid gland, contributes to the gains in body size and skeletal maturation during puberty.

II. HORMONAL CHANGES IN PUBERTY

C. Growth

- Sussman et al. (1987) assessed the relation between hormone levels in adolescence on some risk-taking relevant behaviors.
- Her analysis used hormonal level and emotional disposition to predict such risk-related characteristics as delinquency and rebelliousness.
- Delinquent behavior in boys was related to being less calm, lower levels of Estrodiol, Testosterone/Estrodiol ratio, TeBG (a measure of free testosterone) and DEAHS (Adrenal steroid) and higher levels of Delta 4A (Adrenal steroid).
- Rebellious attitude in boys was related (independently of emotional dispositions) to higher levels of LS, DHEAS and lower levels of FSH.

D. Puberty: Hormones \( \rightarrow \) Behavior

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Sussman et al.’s (1987) assessment included an accounting of pubertal level based on Tanner. Tanner assessments are line drawings of the bodies of prepubescent and immature children (stage 1) to fully mature young adults (stage 5).

Sussman found that hormone levels were correlated with Tanner Score, suggesting an alternative explanation for the data.

She dismissed that explanation by finding that Tanner Scores accounted for no more of the variance in various run regressions (p. 1129).

Was this adequate?

II. HORMONAL CHANGES IN PUBERTY
D. Puberty: Hormones ➔ Behavior

- Sussman et al.’s (1987) concludes the following

  In conclusion, relations between hormones and some emotional dispositions and aggressive attitudes were found for boys but not for girls. This pattern of findings indicates that puberty-related hormone changes may not be as important in the development of aggression in adolescence as previously speculated.

- What do you think of the conclusion?

  Other research explores interactions between hormonal levels and secondary sex characteristics on social behavior.

Hormonal changes cause physical changes in adolescents:

- Height and Weight
  - The growth spurt is the rapid gain in height and weight that is the first outward sign of puberty.
  - Growth in body size is complete for most girls by age 16 and for boys by age 17½.
  - During puberty, accelerated growth occurs first in the hands, legs, and feet, and then torso.

- Body Proportions
  - Boys broaden shoulders relative to the hips and girls broaden hips relative to the shoulders and waist

- Sexual Maturation
  - Presented separately for girls and boys.

II. HORMONAL CHANGES IN PUBERTY
D. Puberty: Hormones ➔ Behavior

- Sexual Maturation in Girls
  1. Female puberty usually begins with the budding of the breasts and the growth spurt.
  2. Menarche is a girl’s first menstruation.
  3. It typically happens around 12½ years for North American girls.
  4. Following menarche, pubic hair and breast development are completed and underarm hair appears.
  5. Nature delays menstruation until the girl’s body is large enough for successful childbearing.
II. HORMONAL CHANGES IN PUBERTY
D. Puberty: Hormones ➔ Behavior

- Individual Differences in Menarche
  - Timing is partly **genetically** controlled
  - Identical twins generally reach menarche within a month or two of each other, whereas fraternal twins differ by about 12 months.
  - Also controlled **biologically** by weight and fat
  - A sharp rise in body weight and fat may trigger sexual maturation in females.
  - A trigger point may also come into play: 106 lbs
  - **Home life** affects menarche
    - Conflict promotes early, affection later menarche.
  - **Health, SES, and world region** affects timing
    - Menarche delayed in regions where malnutrition and infectious disease are widespread.

- Garber et al., (2004) examine early and late maturing boys and girls experience of risk-related psychopathology (antisocial behavior, drug abuse)
  - Early maturing women were more likely than other women to engage in disruptive behavior.
  - Late maturing men were more likely than other men to engage in disruptive behavior and substance use.
  - The findings suggest that hormonal influences on behavior may be mediated by secondary sex characteristics.
Brain development in adolescence is hot news!

Consider this Washington Post article from 2 years ago:

Brain Immaturity Could Explain Teen Crash Rate; Risk-Taking Diminishes At Age 25, NIH Study Finds

As we work through the neuropsych work, please remember correlation is not causation,

Just because risky behavior is associated with a biological phenomenon, that does make the biology the cause of risk taking.

Also, remember, biological accounts do not rob us of our ability to freely make and be morally responsible for our choices.

IV. NEURONAL CHANGES IN PUBERTY
A. Brain Development

- Review: Two mechanisms of brain growth.
  - Non-specialized mechanism involve in changes in the brain that are dependent on experience or learning and applies to infants and adults alike.
  - Non-specialized mechanism called Long Term Potentiation (LTP) which involves new connections between neurons.
  - It accounts for the neural changes much like how your floppy disk changes when saving a file.
    - Just as new patterns of magnetic connections are formed on the floppy disk to represent your paper, so new synaptic connections between neurons are formed in your brain to represent your experience.

- Specialized mechanisms affect the growth of the brain from childhood through adolescence make brains operate
  - Faster: Neurons fire faster.
    - Myelinization.
  - More efficiently: Neural firing activates a more relevant set of neurons.
  - Synaptogenesis (the blooming and pruning of synapses)
  - With greater control and organization: Neurons organize in ways promoting better control.
    - Laterization and Corticalization. Greater influence of the prefrontal cortex in the operation of other brain functions.

- There are many aspects of brain changes related to emotions and emotional regulation
  - Increase in Dopamine (neurotransmitter) in the prefrontal cortex (planning and evaluation, click here) and the limbic system (center of emotions).
    - May make adolescents more vulnerable to impulsivity and risk taking.
    - Prefrontal cortex make need extended period to override emotional responding.
  - Emotional information is processed by adolescents more extensively in the limbic system (Amygdala, source of flight or flight) than the frontal cortex.
    - Adults showed the precise opposite.
IV. NEURONAL CHANGES IN PUBERTY
A. Brain Development and Risk Taking
- Spear (2000) argues that these neurological changes provides a basis for an account of adolescent risk taking
  - The dopamine system plays a role in novelty-seeking and in assessing the motivational value of stimuli.
  - The changes in dopamine levels in the prefrontal and limbic systems of the brain can altered many aspects of the emotional processing of information.
  - Alterations in the incentive value may make adolescents more sensitive to peers and peer pressure.

IV. NEURONAL CHANGES IN PUBERTY
B. Neurotransmitters and Risk Taking
- Fairbanks (2003) further examines the role of serotonin in risk taking using animal models
  - She notes that in monkeys, the movement from childhood to adolescence is marked by lower levels of serotonin.
  - Serotonin pathways in the brain regulate emotions, sleep, appetite among other functions.
  - Low levels are associated with impulsive aggressiveness and risk taking.
  - Dopamine facilitates typical adolescent behavior, higher levels of serotonin provide inhibitory control.
    - High Dopamine ➔ activates reward seeking, sex, and addictions
    - Low Serotonin ➔ release of inhibitory effects of serotonin neurons
    - Worst case is a low serotonin/dopamine ratio.
IV. NEURONAL CHANGES IN PUBERTY
C. Neurocognition and Risk Taking

- Overman et al. examined prefrontal cortical system as a neurological basis for adolescent risk taking
- Noted that Iowa Gambling Task (IGT) measures prefrontal cortex function
  - IGA thought to assess the ventromedial prefrontal cortex which taps the myopia associated with poor decision making
  - Poor IGT task performance suggests poor decisions regarding the evaluation, prediction, anticipation, and response to reward
- Measured by choosing a deck of cards to play a gambling game
  - 2 decks: small + payoff but smaller – payoffs
  - 2 other decks: large + payoffs but larger – payoffs.

- Also noted that the Wisconsin Card Sorting Task (WCST) measures prefrontal cortex function
  - WCST thought to assess the dorsolateral prefrontal cortex
  - Poor WCST task performance suggests greater number of perseveration errors
- Measured by a task in which participants categorized multi-dimensional cards on a single dimension a
  - Participants not told which dimension is active in categorization, only that they made a correct or incorrect choice.

Overman examined adolescents (11-18) on the IGT and WCST.
- Looked for differential performance.
  - Higher WCST and lower IGT suggests a localized in the vmPFC
- Assessed relationship of each task to substance abuse (frequency and amount) of alcohol, tobacco, and drugs
- They also looked at each task and impulsivity and excitement seeking.

- Found age and sex (no interaction) on IGT.
- Found no age and sex effect on the WCST
- No correlation between the IGT and WCST tasks
- WCST did not correlate with any risk behavior or tendency.
- IGT negatively related only to poly-drug use.

Surprised by the findings? Any thoughts on why???