Biological Basis of Adolescent Risk-taking:

Evolutionary and Behavioral Genetics Perspectives

I. INTRODUCTION
A. Biology of Adolescent Risk Taking

- Biological basis of adolescence risk-taking focuses on heredity and changes associated with puberty
  - Hereditary: Characteristics that are transmitted genetically from parent to offspring.
  - Puberty: Changes in the body as a function of going through puberty.
- These genetic, neurological and hormonal changes at puberty may promote risk-taking.

I. INTRODUCTION
A. Biology of Adolescent Risk Taking

- Over the next two weeks, we will consider each of these in turn.
  - This week we will focus on two aspects of hereditary
    - Genes make people similar to each other (evolution) and different from each other (behavioral genetics)
  - Next week we will consider three aspects of Pubertal changes
    - Hormonal changes (chemicals secreted by the endocrine glands and carried though the body by the bloodstream), psychological consequences of puberty, and neurological changes.

I. INTRODUCTION
B. The Storm and Stress Model

- G Stanley Hall: Biological changes make adolescence a time of **storm and stress**.
  - Biological changes in adolescence supposedly cause adolescents to:
    - display mood alterations
    - have distressing and unpredictable thoughts
    - show high anxiety and exaggerated defense mechanisms
    - behave impulsively, inconsistently, or inappropriately.
    - manifest inner disturbance.
- Arnett (1999), following up on Hall strongly argues that biological changes makes adolescence a time of conflict with parents, impulsivity, and risky behavior due to biological

I. INTRODUCTION
C. Storm and Stress and Adolescent Health

- According to Arnett, adolescents are more <u>vulnerability</u> to impulsive and risky behavior
  - Accidents: 50% of all 10-19 deaths due to accidents with most involving autos.
    - Risky driving more than a lack of experience (speeding tailgating, DUI)
  - Suicide: Rate of 6% in 10-14 and 12% 15-19
    - Rate has tripled since the 1960's
  - Homicides: Teens three times more likely to die from guns than natural causes.

II. HEREDITY
A. Biology of Adolescent Risk Taking

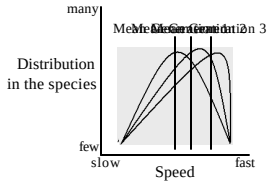
- There are two aspects of the role of hereditary in adolescent risk-taking.
  - Evolutionary Theory: Examines the biological basis of what makes members of the species alike in various characteristics.
    - Is there a biological basis for adolescence being a time of vulnerable to risk-taking?
  - Behavioral genetics: Examines the biological basis of what makes members of the species different in various characteristics.
    - Are some adolescents biologically more vulnerable to risk taking than others?

II. HEREDITY
B. Evolutionary Perspective

- **1. Darwin and Evolution**
  - To Darwin (1872, *The Origin of Species*), all present living things descended from prior forms.
  - **Natural selection**: The explanatory principle.
    - Some organisms produce offspring that are able to survive and reproduce while other organisms of the same species do not.
  - **Survival of the fittest**: The mechanism by which natural selection works.
    - Organisms best suited to exploit environmental resources and defend against predators will reproduce and bequeath their genetic heritage to their offspring.

II. HEREDITY
B. Evolutionary Perspective

- Consider the evolution of the speed of a given species in a given environment and assume that:
  - speed is normally distributed in the species.
  - survival is more likely the faster you are.



II. HEREDITY
B. Evolutionary Perspective

- "Survival of the fittest" does mean **personal survival.**
  - An organism who survives without reproducing is an **evolutionary failure**.
  - More importantly than personal survival of individual organisms is the survival of the **gene pool**: The sum total of the genes of all future parents of the species.
  - In evolution, it is the passing on of genes to offspring which is the critical issue.
    - Who gets to reproduce?
    - What characteristics get passed down?

II. HEREDITY
B. Evolutionary Perspective

- **2. Evolutionary Psychology**: Emphasizes the importance of adaptation, reproduction, and survival of the fittest in explaining behavior.
  - To adopt an evolutionary perspective, one must ask whether a species-related behavior has promotes reproductive success of the species.
  - Consider such universals as:
    - Language
    - Pretense
    - Risk taking in Adolescence.

II. HEREDITY
B. Evolutionary Perspective

- **3. Evolution and Adolescent Risk Taking.** An evolutionary account of risk taking offers a number of "survival of the fittest" reasons for its occurrence.
  - Explore novel and broader areas away from home.
  - Acquire new, perhaps more efficient, behavior patterns and learn new reward patterns.
  - Avoiding inbreeding by the dispersal of men and women before reproduction.
  - Relinquishing child-like and adopting adult-like behavior patterns.

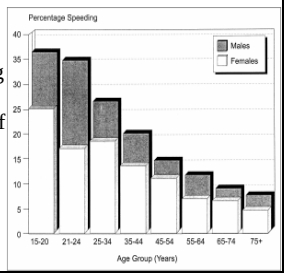
II. HEREDITY
B. Evolutionary Perspective

- Nell (2000) offers an additional perspective on evolutionarily account of adolescent risk taking.
  - Evolutionary theory, he emphasizes, is based on competition between reproductively mature members of the species.
    - Young men who do crazy things are saying … "Look at me! I have so much strength and skill that I am fearless, I will survive no matter how much I drink or how fast I drive"; the handicaps (which include substance use) convey the message that the male can support high costs (Cronin, 1991).

II. HEREDITY
B. Evolutionary Perspective

- Nell (2000) argues that such an account can explain decrease in dangerous driving

Driving represents the most common form of sensation seeking in young men because it bypasses the genetic endowments of strength and speed and makes the demonstration of courage available to all young men, including the slow and the weak.



II. HEREDITY
B. Evolutionary Perspective

- Risk-taking from this perspective is a behavior deeply embedded in our genetic heritage
- Risk taking is a youthful imperative to increase social status by courting danger and demonstrating courage
- It is decidedly not a "stupid" or "meaningless" act but one which promotes reproduction success.
  - As Nell notes: At the individual level, risk is part of life. A life without risk is a life not worth living, empty of mastery, achievement, and social status.

II. HEREDITY
B. Evolutionary Perspective

- Nell further notes that adolescent risk taking is not exclusively a human trait, but one that applies to members of all species
  - As Nell suggests, "Evolution has, on the contrary, so arranged matters that young males of virtually all species are biochemically prepared to fight for territorial advantage and physical dominance."
- Evidence for this claim comes from comparative psychology, which explores the evolutionary basis for similarities and difference between species behavior.

II. HEREDITY
B. Evolutionary Perspective

- Fairbanks et al., (1993) examined risk taking in younger and older Velvet monkeys.
  - Risk taking was ecologically assessed by monkeys approaching a human observer, entering a new area, approaching a novel food container, and coming close to a strange adult male.
  - In all 4 test conditions, risk-taking was an inverted U-shaped function of age.
    - Latency to approach in unfamiliar and potentially dangerous situations declined from birth to age 2 yrs, (age of sexual maturity) then increased with age to adulthood.
  - Risk-taking emerges with sexual maturity

II. HEREDITY

C. Behavioral Genetics

- **Behavioral Genetics:** The field of study devoted to uncovering the contributions of nature and nurture to the diversity of human characteristics.
  - Behavior Geneticists compute **Heritability estimates** which measure the extent to which individual differences in complex traits (e.g., intelligence, personality, risk taking) are due to genetic factors.
  - A characteristic is inherited if unrelated people are different in that characteristic but related people are similar.

II. HEREDITY

C. Behavioral Genetics

- Heritability estimates are obtained from **Twin Studies**, which compare characteristics of various types of siblings.
  - The most well known twin study compares them on IQ.

Relationship	Genetic Similarity	Predicted r for IQ	Actual r for IQ
Unrelated siblings	0%	.0	.15
Related Siblings	50%	.5	.47
Fraternal Twins	50%	.5	.60
Identical Twins	100%	1.0	.86

II. HEREDITY

C. Behavioral Genetics

- Twin Studies of IQ are controversial.

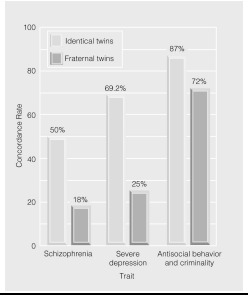
Relationship	Genetic Similarity	Actual r for IQ	Environmental Similarity
Unrelated siblings	0%	.15	low
Related Siblings	50%	.47	medium
Fraternal Twins	50%	.60	medium high
Identical Twins	100%	.86	high

- Adoption studies examine concordance rates…
  - between twins when reared together & apart.
  - between adopted kids are their biological and adopted parents.

II. HEREDITY

C. Behavioral Genetics

- Many characteristics appear to be inherited.



- Limits of research:

  Studies can overestimate the influence of heredity while underestimating the impact of the environment.

  It is difficult to generalize the twin pair study results to the general population.

  They do not address the process of change.

II. HEREDITY

C. Behavioral Genetics

- Stallings et al.(2002) assessed whether risky behavior as smoking and drinking have a genetic component.
  - They distinguished between when these risky behaviors were first tried (age at first used) and the time (latency) it took from when first tied to when it become used regularly.
  - Hypothesized that environmental influences would be more important in determining twin resemblance for age-related milestones, while genetic factors might make a greater contribution to twin resemblance for the latency between first use and regular use.

II. HEREDITY

C. Behavioral Genetics

- Little evidence of a genetic component for age of first use of tobacco and alcohol, except for age at weekly use of alcohol.

Table III. MZ and DZ Twin Correlations for Age-at-Onset Milestones

Onset milestone	r_{MZ}	$(N)^a$	r_{DZ}	$(N)^a$
Alcohol				
Age at first use ever	.57 ± .04	(563)	.45 ± .07	(240)
Age at first intoxication	.43 ± .07	(222)	.41 ± .10	(96)
Age at weekly use[b]	.40 ± .08	(158)	.18 ± .13	(62)
Tobacco				
Age at first use ever	.44 ± .06	(312)	.37 ± .08	(136)
Age at daily use (1 cig/day)[c]	.41 ± .07	(240)	.34 ± .10	(97)
Age at daily use (10 cigs/day)[d]	.35 ± .08	(174)	.50 ± .12	(69)

Note. r_{MZ} = MZ twin (product–moment) correlation ± standard error of estimate; r_{DZ} = DZ twin (product–moment) correlation ± standard error of estimate.
[a] *N* = number of twin pairs with complete data.
[b] Weekly alcohol use—drink alcohol *at least* once per week.
[c] Smoke *at least one* cigarette per day.
[d] Smoke *at least* half a pack (10 cigarettes) per day.

II. HEREDITY

C. Behavioral Genetics

- In contrast, there is a much stronger genetic effect for latency variables.

The results suggest a genetic element to when someone gets hooked after an initial exposure to smoking or drinking

Table IV. MZ and DZ Twin Correlations for Transition Latency Variables

Transition latency variable[a]	r_{MZ}	$(N)^b$	r_{DZ}	$(N)^b$
Alcohol				
Age at first use ever to age at first intoxication	.32 ± .07	(212)	.26 ± .11	(89)
Age at first use ever age at weekly use	.30 ± .08	(151)	−.01 ± .14	(58)
Tobacco				
Age at first use ever to age at daily use (1 cig/day)	.26 ± .07	(235)	.05 ± .10	(97)
Age at first use ever to age at daily use (10 cigs/day)	.37 ± .08	(170)	.12 ± .12	(69)

r_{MZ} = MZ twin (product–moment) correlation ± standard error of estimate;
r_{DZ} = DZ twin (product–moment) correlation ± standard error of estimate.
[a] Latency—difference in years between age at onset for use milestones.
[b] *N* = number of twin pairs with complete data.

II. HEREDITY

D. GxE=P

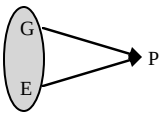
- Genes do not work in a vacuum
  - To be expressed, a gene interacts with the biological, physical, and social environment.
- To express this, geneticists distinguish between:
  - **Genotype**: The genetic basis of a particular trait.
  - **Phenotype**: The actual expression of a trait.
- The following formula is a foundation of modern genetics: **G x E = P**
  - Phenotype (P) is a product the interaction between the Genotype (G) and the biological, physical, and social Environment (E).

II. HEREDITY

D. Gene Environmental Interaction

- Sandra Scarr offered additional clarification of the G X E = P formula.
  - She examined how people with particular genes come to find themselves in particular environments.
  - She presented accounts of these gene (G) gene -(E) environment correlations in the production of (P) phenotypes.

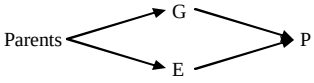
Scarr believes that genes & environments are not so *independent* of each other?



II. HEREDITY

D. Gene Environmental Interaction

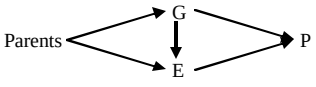
- a. Passive Correlation: In a **passive** correlation, an adolescent has no other control over the environment available to him or her than the one created by parents.
  - Parents create an environment compatible with their own heredity and provide for the adolescents' hereditary.



II. HEREDITY

D. Gene Environmental Interaction

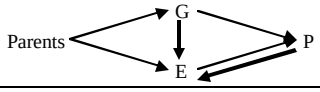
- b. Evocative Correlation: In an **evocative** correlation, the environment is consistent with and generated by the adolescent's heredity.
  - The environment is created in response to the genes from which it is evoked (e.g., temperamentally pleasant and calm adolescents evoke different environments than nervous and anxious ones).



II. HEREDITY

D. Gene Environmental Interaction

- c. Active Correlations: In an **active** correlation, environments are actively sought out which complement genetic tendencies.
  - This tendency to actively choose environments that complement our heredity is called **niche-picking**.
  - With age, genetic factors may become more important in determining the environments we experience and choose for ourselves.



II. HEREDITY
D. Gene Environmental Interaction

- These accounts of gene-environment interactions have been sharpened with better accounts of the environment.
  - "Shared environments" are those commonly experienced by siblings, including their parents personality, social class, and neighborhood.
    - Such environments appear to have little influence on personality or interests.
  - "Non-shared environments" include the person's own unique experiences within and outside the family which is not shared by siblings.
    - Non-shared environments have strong influences on personality and interests, due to active niche-picking.

Comparison of Correlations and Their Interpretations

Comparison in Intraclass Correlations	Interpretation
Magnitude of correlation of monozygotic twins reared apart	Importance of genetic effects
Monozygotic correlations greater than dizygotic correlations	Importance of genetic effects
Twins reared together more similar than twins reared apart	Importance of shared rearing environments
Monozygotic pairs more than twice as similar as dizygotic pairs	Importance of nonadditive genetic effects
No or little difference in monozygotic and dizygotic correlations	Importance of correlated environmental effects
Differences among monozygotic twins (one monozygotic correlation)	Nonshared environmental influences

Malaty, H. M. et. al. Ann Intern Med 1994;120:982-986

Annals of Internal Medicine

II. HEREDITY
D. Gene Environmental Interaction

- Stallings et al., study allows for the analysis of whether smoking and drinking exposure is due to shared or non-shared environmental factors.
  - For 4 of the 6 substance use (first and daily) milestones, twin resemblance could be more adequately explained by a non-genetic model.
    - The evidence points to the role of shared and non-shared environment in the age of adolescents' first exposure to drugs and alcohol and the age of their regular use of them

Table V. Model-Fitting Results: Alcohol and Tobacco Use Milestones

Model	h^2	c^2	e^2	χ^2	df	p	AIC
Alcohol							
Age at first use							
ACE[a]	**.21**	**.35**	**.43**	**0.35**	**3**	**.95**	**−5.65**
(CI_{95})	(.02–.44)	(.14–.53)	(.38–.49)				
AE	.58	—	.42	10.32	4	.04	2.32
CE	—	.54	.46	4.91	4	.30	−3.09
Age at first intoxication							
ACE	.09	.35	.56	1.65	3	.65	−4.36
AE	.65	—	.54	5.31	4	.26	2.60
CE[a]	—	**.43**	**.57**	**1.90**	**4**	**.76**	**−6.10**
(CI_{95})		(.33–.51)	(.49–.67)				
Age at weekly use							
ACE	.43	.00	.57	13.36	3	<.01	7.36
AE[a]	**.43**	—	**.57**	**13.36**	**4**	**.01**	**5.36**
(CI_{95})	(.29–.55)		(.44–.71)				
CE	—	.31	.69	19.78	4	<.01	11.78
Tobacco							
Age at first use							
ACE	.14	.30	.56	1.19	3	.76	−4.81
AE	.46	—	.54	4.74	4	.32	−3.26
CE[a]	—	**.42**	**.58**	**1.96**	**4**	**.74**	**−6.10**
(CI_{95})		(.34–.49)	(.51–.66)				
Age at daily use (1 cig/day)							
ACE	.15	.26	.59	2.43	3	.49	−3.57
AE	.42	—	.52	4.22	4	.38	−3.77
CE[a]	—	**.39**	**.61**	**3.07**	**4**	**.55**	**−4.93**
(CI_{95})		(.30–.48)	(.52–.70)				
Age at daily use (10 cigs/day)							
ACE	.00	.40	.60	3.02	3	.39	−2.98
AE	.41	—	.59	9.94	4	.04	1.94
CE[a]	—	**.40**	**.60**	**3.02**	**4**	**.55**	**−4.98**
(CI_{95})		(.29–.50)	(.50–.71)				

Note. (CI_{95}) = 95% confidence intervals for parameter estimates; A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; h^2 = proportion of variance explained by additive genetic effects; c^2 = proportion of variance explained by shared environmental effects; e^2 = proportion of variance explained by nonshared environmental effects.
[a] Best-fitting model by AIC criteria.

II. HEREDITY

D. Gene Environmental Interaction

- Stallings et al., study also allows for the analysis of whether the time from first exposure to regular smoking and drinking is genetic or environmental factors.
  - In contrast to substance use (first and daily), latency to regular use appears to be more strongly influenced by genetic factors.
    - A model relating genetic and non-shared environmental factors was adequate to explain twin resemblance for three of the four transition latencies.

Table VI. Model-Fitting Results: Latencies from First Use to Regular Alcohol and Tobacco Use

Transition model	h^2	c^2	d^2	e^2	χ^2	df	p	AIC
Alcohol								
Age at first use to age at first intoxication								
ACE	.11	.21	—	.68	0.84	3	.84	−5.16
AE	.33	—	—	.67	1.83	4	.77	−6.18
CE[a]	—	**.30**	—	**.70**	**1.07**	**4**	**.90**	**−6.93**
(CI_{95})		(.19–.40)		(.60–.81)				
Age at first use to age at weekly use								
ADE	.00	—	.31	.69	5.65	3	.13	−.35
AE[a]	**.28**	—	—	**.72**	**7.17**	**4**	**.13**	**−.83**
(CI_{95})	(.13–.43)			(.57–.87)				
Tobacco								
Age at first use to age at daily use (1/day)								
ADE	.00	—	.25	.75	4.05	3	.26	−1.95
AE[a]	**.24**	—	—	**.76**	**4.60**	**4**	**.33**	**−3.40**
(CI_{95})	(.12–.35)			(.65–.88)				
Age at first use to age at daily use (10/day)								
ACE	.37	.00	—	.63	4.28	3	.23	−1.72
AE[a]	**.37**	—	—	**.63**	**4.27**	**4**	**.37**	**−3.73**
(CI_{95})	(.23–.49)			(.51–.77)				
CE	—	.30	—	.70	8.25	4	.08	.25

Note: (CI_{95}) = 95% confidence intervals for parameter estimates; A = additive genetic effects; C = shared environmental effects; D = nonadditive genetic effects; E = nonshared environmental effects; h^2 = proportion of variance explained by additive genetic effects; c^2 = proportion of variance explained by shared environmental effects; d^2 = proportion of variance explained by nonadditive genetic effects; e^2 = proportion of variance explained by nonshared environmental effects.

[a] Best-fitting model by AIC criteria.

II. HEREDITY

D. Epigenetic Model

- Summary
  - Genes and environments are not independent of each other but interact in producing phenotypes (including risk taking behaviors).
  - The interaction depends on:
    - The type of environment; The nature of the genotype; Age; Freedom to choose environments.
- **Epigenetic Model of Development:** Development of the individual results from ongoing, bidirectional exchanges between heredity and all levels of environment.

II. HEREDITY

D. Epigenetic Model

- From evolutionary theory, we have learned that risk-taking may emerge with sexual maturation.
- From behavioral genetics we learn that sexually mature adolescents actively niche-pick.
  - Adolescents are (maybe for the for the first time) able to *seek out* genetically compatible environments and *indulge* in them.
    - Risk-taking may be go hand in hand with indulgence (i.e., next week we will see that teens have difficulty with behavioral inhibition).
  - Some adolescents may have a specific temperament to seek out and indulge in particular kinds of environments.

II. HEREDITY
E. Genes and Environments in Puberty

- Because of their temperament, some may tend towards **sensation seeking**, defined as "the seeking of varied, novel and intense sensations and experiences and the willingness to take physical, social, legal and financial risks for the sake of such experiences."
  - Twin and adoption studies have shown a strong heritability component for Sensation Seeking, which is often characterized as a personality trait (see **Zuckerman 2000**).

II. HEREDITY
E. Genes and Environments in Puberty

- Levenson (2002) examined the role of sensation seeking on risk taking
  - He looked for an effect of sensation-seeking (among other psychological traits), depending on the form of risk in which people engage.
  - He distinguished between the risks sought out by three different groups --
    - Antisocial risk-takers defined as residents in a long-term drug-treatment facility (N=24).
    - Adventurous risk takers, defined as rock climbers (N=18).
    - Pro-social risk takers, or heroes, defined as policemen and firemen decorated for bravery (N=2l).

II. HEREDITY
E. Genes and Environments in Puberty

- Levenson used Zuckerman's SS measure.
  - It includes 4 subscales.
    - **Thrill and adventure seeking**: Defined as a desire for activities involving moderate danger or adventure such as parachute jumping or driving fast.
    - **Experience Seeking**: Defined as looking for new experiences through music, art, drugs, or unconventionality in dress or behavior.
    - **Disinhibition**: Defined as seeking sensation by casting off inhibition through activities such as drinking or sexual variety.
    - **Boredom Susceptibility**: Defined as a dislike for repetitious experience of any kind and a desire for novelty things and people.

II. HEREDITY
E. Genes and Environments in Puberty

- Levenson (2002) found that sensation seeking was different for different groups.

It is interesting that heroes (Gr. 3) were distinct from the other two groups primarily in their low scores on the sensation-seeking scales.
The results suggest that motives for risk taking are not based on thrill seeking.

Table 3
F Tests and Post Hoc Range Comparisons

Scale	*F*	*p*[a]	Scheffé[b] ($p < .01$)
SAP (Form A)	54.40	.000	1 > (2, 3)
Emotionality	33.99	.000	1 > (2, 3)
Depression	46.18	.000	1 > (2, 3)
Psychopathy	12.74	.000	1 > (2, 3)
Independence/Conformity	0.42	.658	—
Empathy	5.66	.006	—
General Sensation Seeking	15.55	.000	3 < (1, 2)
Thrill and Adventure Seeking	7.11	.002	2 > (3)
Experience Seeking	17.32	.000	3 < (1, 2)
Disinhibition	11.17	.000	1 > (2, 3)
Boredom Susceptibility	11.04	.000	1 > (3)
DIT P% score	8.77	.001	2 > (1, 3)
DIT D score	3.03	.056	—

Note. SAP = Substance Abuse Proclivity; DIT = Defining Issues Test. [a] Bonferroni's correction for multiple *F*s was used. Therefore only those *p*s < .004 are considered significant. [b] Group 1 = drug-unit residents, Group 2 = rock climbers, Group 3 = heroes.