



**Beyond Mental Health:
Child Development
In
Evolutionary Perspective**

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of
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Central Claim:

By viewing human development principally from a mental-health perspective, we misconstrue it by failing to view it in evolutionary perspective.

2



Most developmental theory, inquiry and practice presumes (too often) that there is something commonly referred to as “optimal development.”


Freud: to love and to work

Maslow: self actualization

Erikson: basic trust, autonomy, identity, intimacy, generativity



Attachment theory: security, competence, intimacy

3

In light of this way of viewing so-called optimal development, it is no wonder that we view adversity as “undermining” or “compromising” development—and so use terminology emphasizing dysfunction, dysregulation and disorder.


4

But from an evolutionary perspective, we need to appreciate and distinguish diverse developmental *strategies* that serve the ultimate goal of ALL living things under varying contextual conditions:

PASSING GENES ON TO THE NEXT GENERATION

5





I will illustrate the utility of an evolutionary perspective— for leading to new developmental insights by considering two current topics in developmental science whose origins are sometimes decades old, yet remain underappreciated:

- Accelerating effect of contextual adversity on biological development;
- Differential susceptibility to developmental experiences and environmental exposures.


--As we will see, the second topic will qualify the first in important ways.

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**Accelerating Effect
Of
Adversity on Biological
Development:**
*--why should adversity accelerate
development?*



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**The basic
evolutionary-developmental
logic here is very simple:**

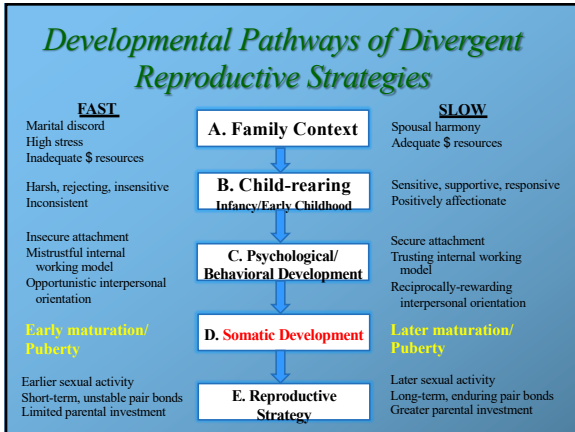
--If the fundamental goal of all living things is to pass on their genes to the next generation,
--then if, due to contextual adversity, the risk of becoming developmentally compromised or dying before reproducing, the organism should speed up development so as to reach sexual maturity sooner than would otherwise be the case
--and thus be in position to reproduce.

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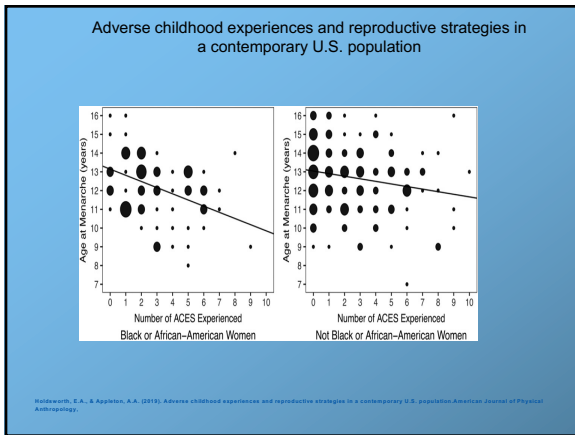


**Developmental Adversity
Accelerates Biological Aging**
--Pubertal Timing
--Cellular Aging
--Brain Development

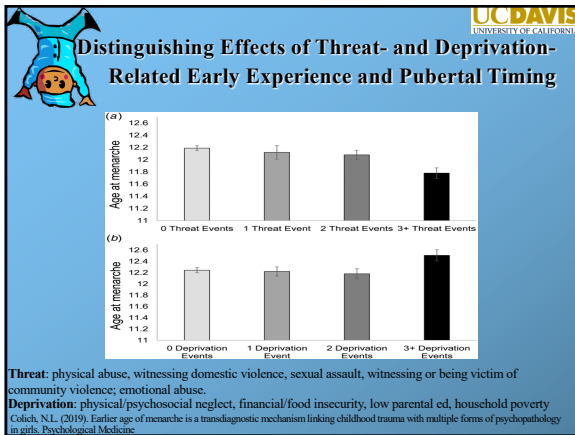
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**META-ANALYTIC STUDY OF
(43 studies with 46 independent data sets)
ADVERSE CHILDHOOD EXPERIENCES
AND
FEMALE PUBERTAL TIMING**
(Age at menarche, Tanner staging scores, Pubertal Development
Scale (PDS) and relative perceived timing)

Table 2. Pooled effect size of specific types of ACEs.

Types of Adversity	K	Cohen's d (95% CI)	I ² (%)	p Value
Sexual abuse	12	-0.14 (-0.18, -0.11)	72.4	<0.001
Physical abuse	8	-0.03 (-0.07,0.01)	91.5	<0.001
Neglect	8	0.02 (-0.10,0.10)	72.0	<0.001
Low SES	26	0.07 (-0.02,0.16)	97.4	<0.001
Father absence	12	-0.40 (-0.63, -0.16)	98.2	<0.001
Family dysfunction	11	-0.08 (-0.11, -0.04)	66.9	<0.001

(Data in parentheses are 95% CIs, ES = effect size (Cohen's d), SES = socioeconomic status.)

Zhang, L., Zhang, D., & Sun, Y. (2019). Adverse childhood experiences and early pubertal timing among girls: A meta-analysis. *International Journal of Environmental Research and Public Health*, 16, 2887.

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**DO EFFECTS ON PUBERTAL DEVELOPMENT
EXTEND TO SEXUAL/REPRODUCTIVE
BEHAVIOR?**

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Developmental Psychology 2010, Vol. 46, No. 1, 120-128
**The Development of Reproductive Strategy in Females:
Early Maternal
Harshness → Earlier Menarche → Increased Sexual Risk
Taking**

Jay Belsky
Birkbeck University of London

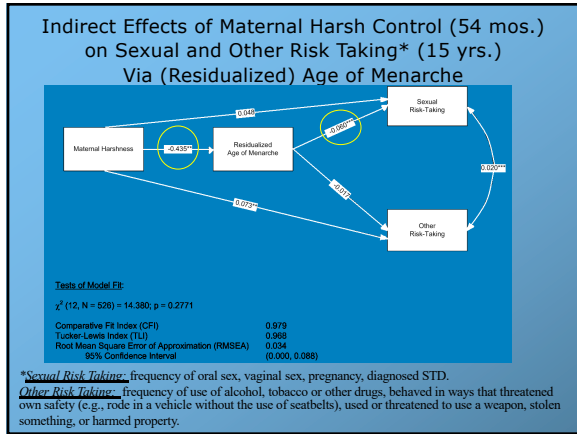
Laurence D. Steinberg
Temple University

Renate M. Houts
Duke University

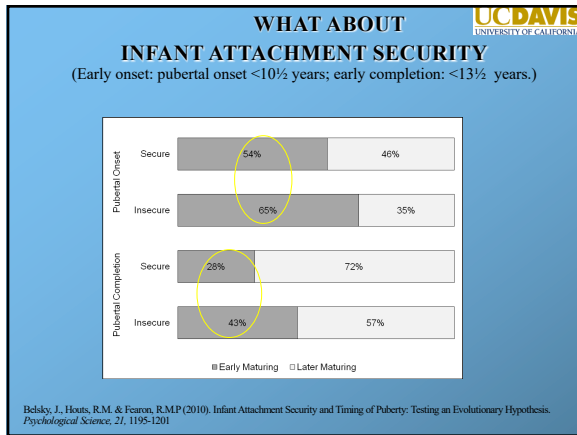
Bonnie L. Halpern-Felsher
*University of California, San
Francisco*

the NICHD Early Child
Care Research Network

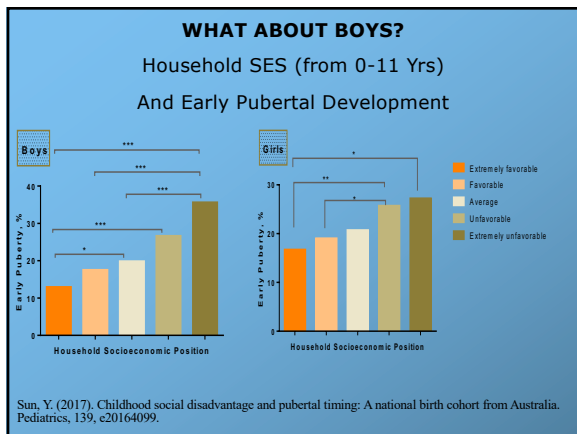
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**Developmental Adversity
Accelerates Biological Aging:**

- Pubertal Timing
- Cellular Aging
- Telomeres
- Epigenome
- Brain Development



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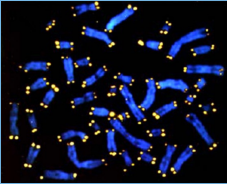



Telomeres

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Telomeres

Ends of linear chromosomes
Shorten Every Time Cell Duplicates
And So Decrease with Age

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Maternal psychosocial stress during pregnancy is associated with newborn leukocyte telomere length

Sonja Entringer, PhD; Elissa S. Epel, PhD; Jue Lin, PhD; Claudia Buss, PhD; Babak Shahbaba, PhD; Elizabeth H. Blackburn, PhD; Hyagriv N. Srinhan, MD; Pathik D. Wadhwa, MD, PhD

Entringer et al, 2013, Am J Obstet Gynecol

Scatterplot of association between maternal pregnancy-specific stress and newborn cord blood telomere length ($r^2 = 0.25$). TS ratio is adjusted for covariates (preterm gestational age at birth, weight, sex, and exposure to antepartum obstetric complications).

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Meta-Analytic Associations Between Type of Adversity And Telomere Length (138 Studies, 402,116 individuals)

type of exposure	median sample size	k	m	r (95% CI)
physical disease	308.5	94	53	-0.15 (-0.22, -0.09)
environmental hazard	86	11	8	-0.26 (-0.51, -0.01)
nutrition (poorer)	224	5	3	-0.17 (-0.42, -0.07)
psychiatric illness	556	87	10	-0.08 (-0.13, -0.03)
smoking	193	61	5	-0.05 (-0.08, -0.02)
alcohol	200	81	34	-0.13 (-0.19, -0.07)
sleep (worse)	280	52	22	-0.08 (-0.14, -0.02)
physical activity (less)	341	43	34	-0.07 (-0.11, -0.02)
psychosocial	124	35	27	-0.08 (-0.09, -0.08)
parental care (poorer)	283	15	11	-0.03 (-0.08, 0.02)
socioeconomic status (lower)	284	14	10	-0.03 (-0.08, 0.03)
other	1454	20	5	-0.02 (-0.08, 0.04)
		10	4	-0.01 (-0.04, 0.02)
		16	11	-0.02 (-0.04, -0.01)
		12	7	-0.03 (-0.04, -0.03)
		100	43	-0.16 (-0.23, -0.10)
		67	25	-0.06 (-0.10, -0.03)
		8	4	-0.11 (-0.23, -0.01)
		4	2	-0.13 (-0.22, -0.03)
		45	21	-0.04 (-0.07, -0.01)
		22	18	-0.01 (-0.06, -0.04)
		23	12	-0.02 (-0.10, 0.07)
		7	5	0.03 (-0.07, 0.14)

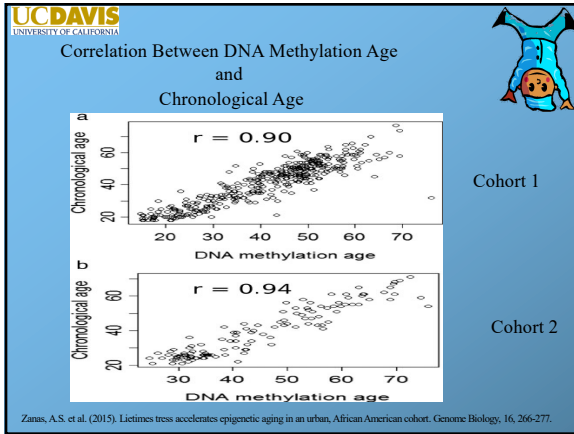
Overall, exposure to adversity associated with shorter telomeres (r = -.15, CI: -.18 to -.11)

Pepper, G.V., Bateson, M., & Nettle, D. (2018). Telomeres as integrative markers of exposure to stress and adversity: A systematic review and meta-analysis. *Royal Society Open Science*, 5, 180744.

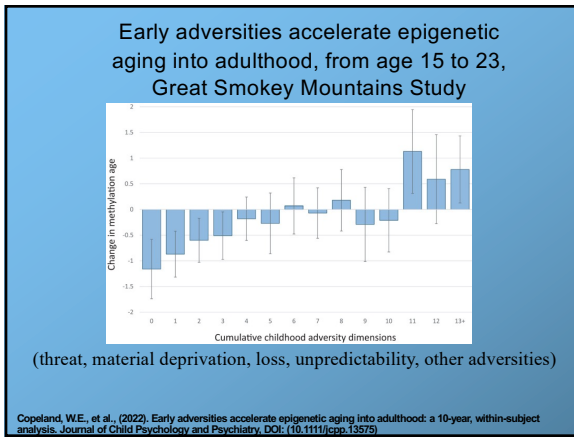
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EPIGENOME: As we age, the epigenome changes

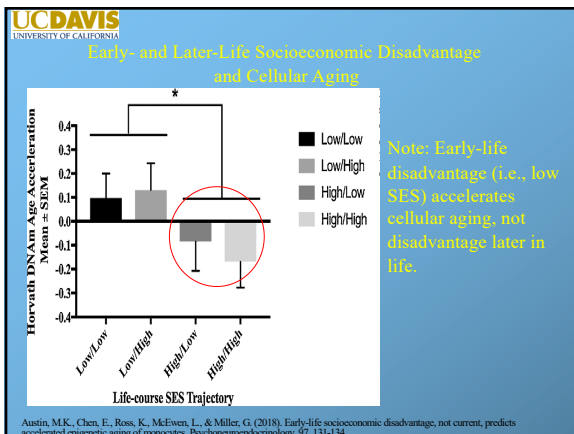
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Developmental Adversity Accelerates Biological Aging:

- Pubertal Timing
- Cellular Aging
- Telomeres
- Epigenome
- Brain Development**



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CONNECTING THE BRAIN BRAKE AND ACCELERATOR?

Development and Psychopathology 29 (2017), 505–518

Insensitive parenting may accelerate the development of the amygdala–medial prefrontal cortex circuit

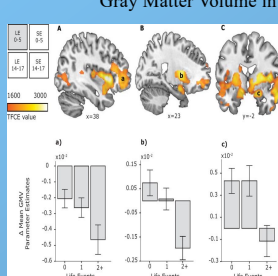
SANDRA THUISSEN, RYAN L. MUETZEL, MARIAN J. BAKERMANS-KRANENBURG, VINCENT W. V. JADDOE, HENNING TIEMEIER, FRANK C. VERHULST, TONYAWHITE, AND MARINUS H. VAN IJZENDOORN

Abstract: stronger amygdala–mPFC connectivity in children with less sensitive mothers ... Amygdala–mPFC resting-state functional connectivity has been shown to increase from age 10.5 years onward, implying that the positive association between age and amygdala–mPFC connectivity in 6- to 10-year-old children of less sensitive parents represents accelerated development of the amygdala–mPFC circuit.

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Early-life Stress (Before Age 5) Accelerates Typical Maturational Decrease in Gray Matter Volume in Adolescents

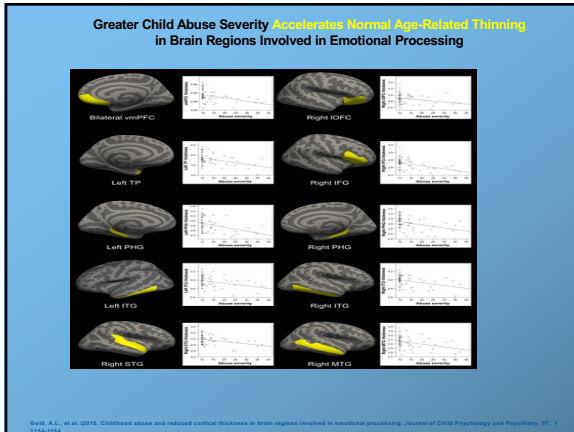


The more adverse life events, the smaller the GMV

Prefrontal Cortex Insula Amygdala

Tyborowska, A. et al. (2018). Early-life and pubertal stress differential modulate grey matter development in human adolescents. Scientific Reports, 8, 9201.

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Differential Susceptibility to Environmental Influences:
Why should children vary in their susceptibility to environmental influences?

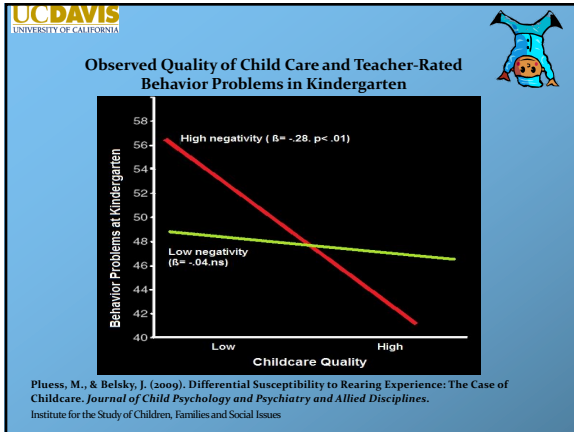
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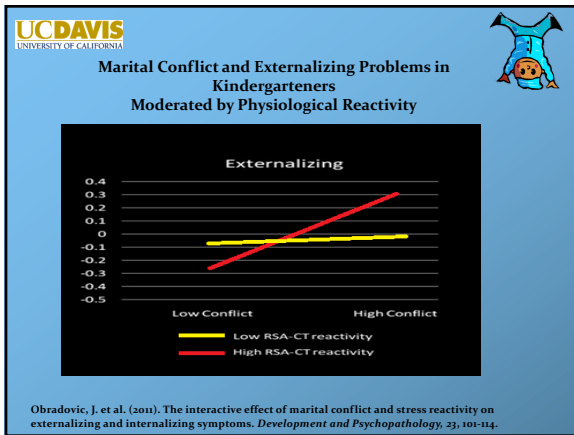
Again, the basic evolutionary-developmental logic here is very simple:

- Because the future is **INHERENTLY UNCERTAIN**
- there must have been times and places in human evolutionary history when being shaped by early developmental experiences proved counter-productive, undermining rather than enhancing the chance of passing on genes to future generations;
- thus, to "hedge bets" about an uncertain future, evolution should have led to the bearing of children who vary in their developmental plasticity, that is, their susceptibility to developmental experiences and environmental exposures.

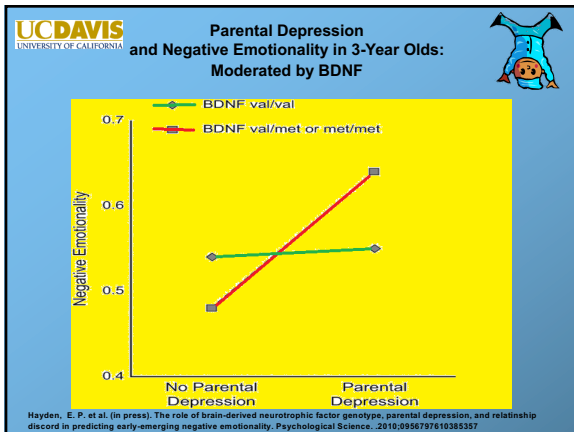
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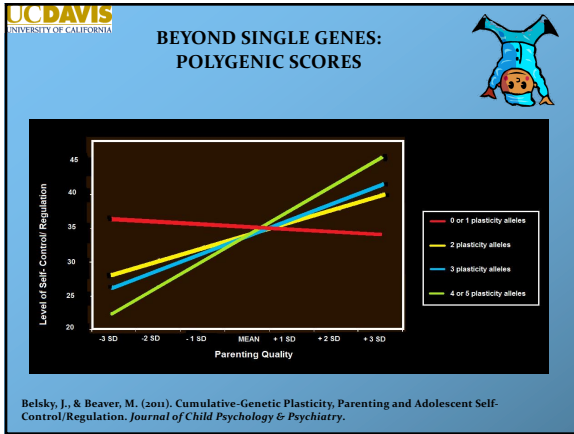
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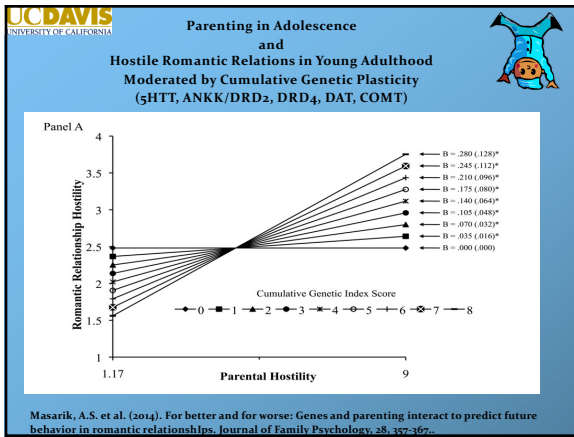
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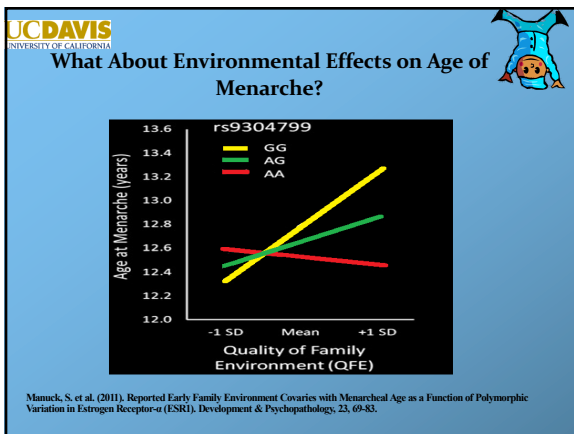
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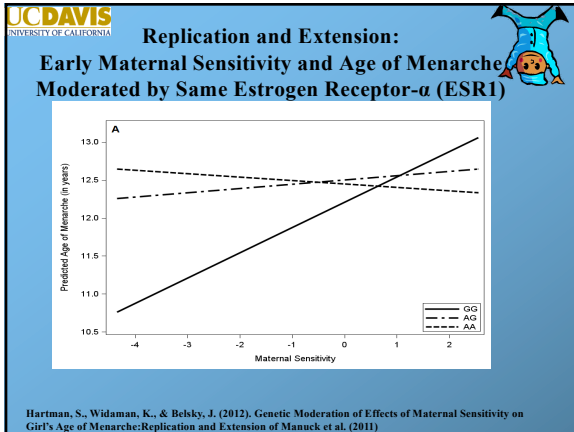
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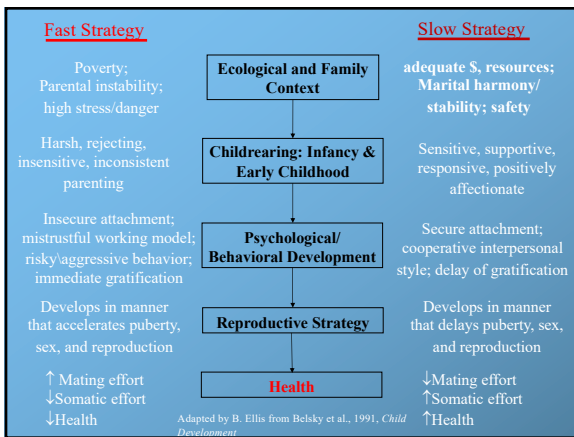
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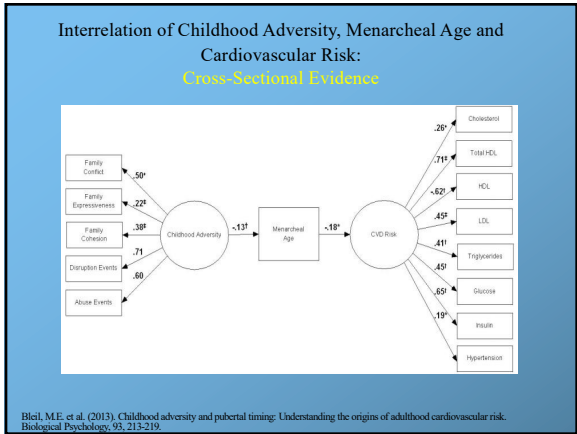
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**Toward an Evo-Devo
Model of
Reproductive
Strategy, Health and
Longevity**

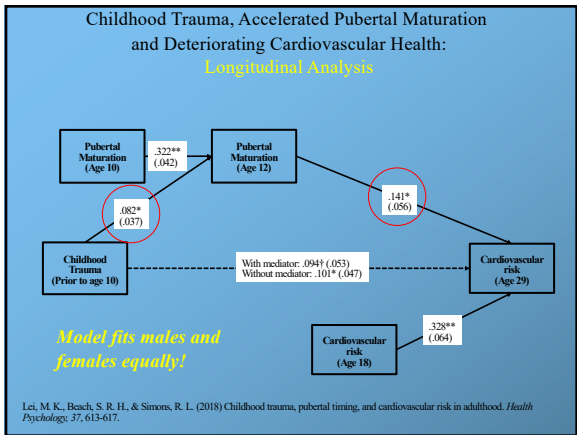
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