The Neurobiology of Childhood Trauma

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1. Epigenetics and Neuroplasticity
2. Trauma and Stress Reactivity
3. Mistrust

Review

Neurobiological characteristics of rhesus macaque abusive mothers and their relation to social and maternal behavior

Dario Maestripieri[a,b,*], Stephen G. Lindell[c], Alejandro Ayala[d], Philip W. Gold[d], J. Dee Higley[c]

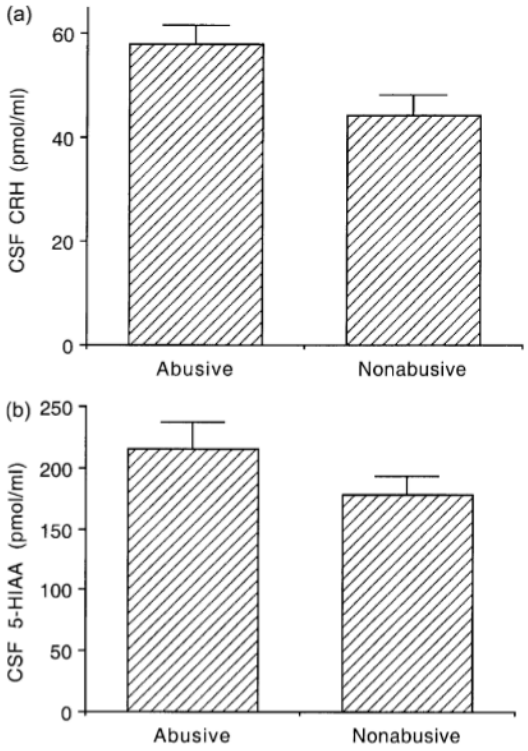


Fig. 1. (a). Mean (±SEM) CSF concentrations of CRH in abusive and nonabusive mothers. (b). Mean (±SEM) CSF concentrations of 5-HIAA in abusive and nonabusive mothers.

1. Observational studies in non-human primates indicate that 5 – 10% of infants are physically abused or neglected

2. Maternal physical abuse does not appear to have adaptive value: abused infants tend not to be neglected, but rather are protected excessively

3. Abusive mothers have mostly normal social behavior, but are characterized by excessive stress reactivity

Neuroplasticity

An NMDA receptor mediated process by which *neurons that wire together fire together*

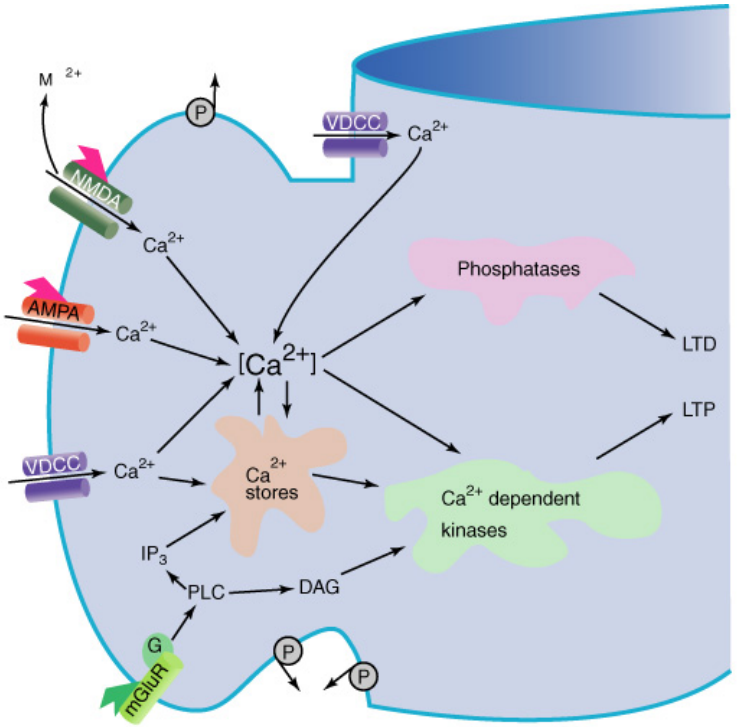
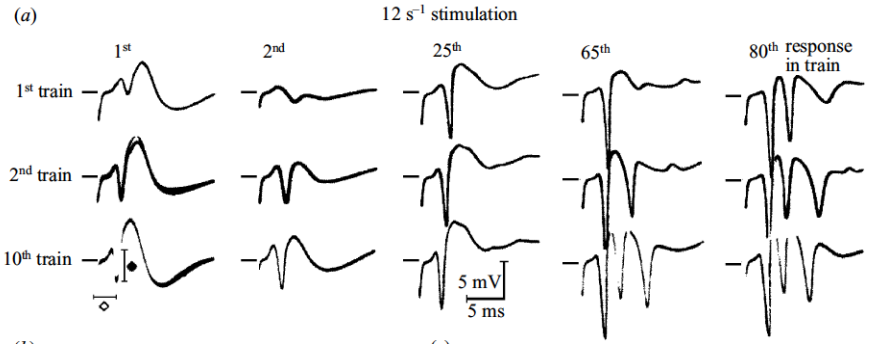
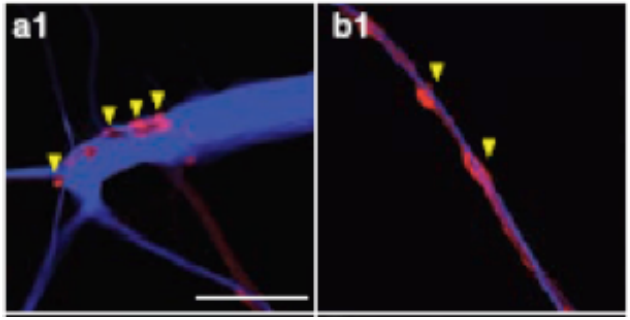
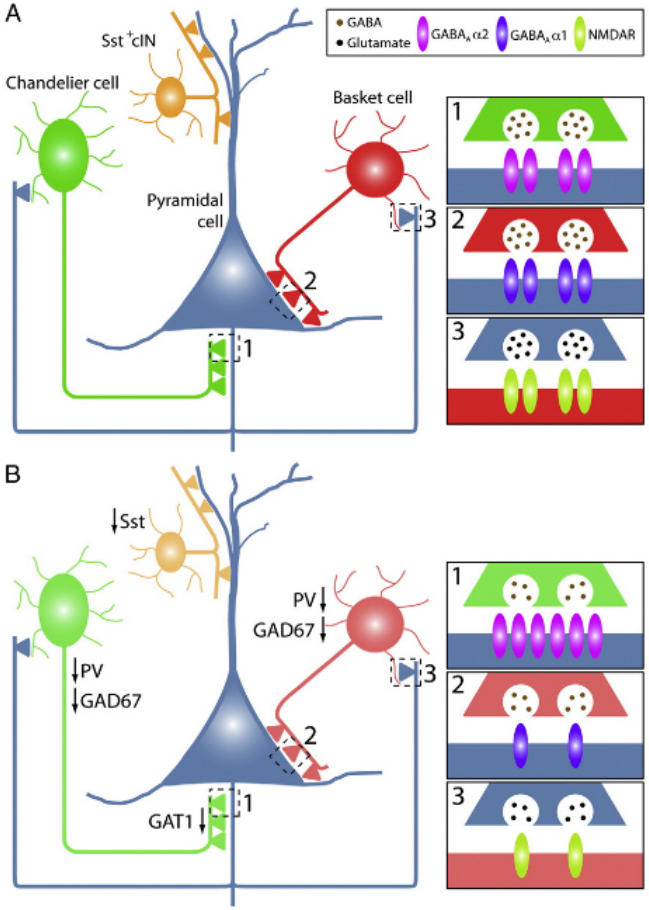


FIGURE 18.10 Events leading to LTP or LTD. The schematic depicts a postsynaptic spine with various sources of Ca^{2+}. The NMDA receptor–channel complex admits Ca^{2+} only after depolarization removes the Mg^{2+} block. Ca^{2+} may also enter through ligand-gated AMPA receptor channel or voltage-dependent Ca^{2+} channels (VDCC), which may be located on the spine head or dendritic shaft. Also, certain subtypes of metabotropic glutamate receptors (mGluRs) are coupled positively to phospholipase C (PLC), which cleaves membrane phospholipids into inositol trisphosphate (IP_3) and diacylglycerol (DAG). Increased levels of IP_3 lead to the release of intracellular Ca^{2+} stores, whereas increases in DAG activate Ca^{2+}-dependent enzymes. Ca^{2+} pumps (P) located on the spine head, neck, and dendritic shaft, are hypothesized to help isolate $[Ca^{2+}]i$ changes in the spine head from those in the dendritic shaft.

FROM MOLECULES TO NETWORKS John H. Byrne, James L. Roberts







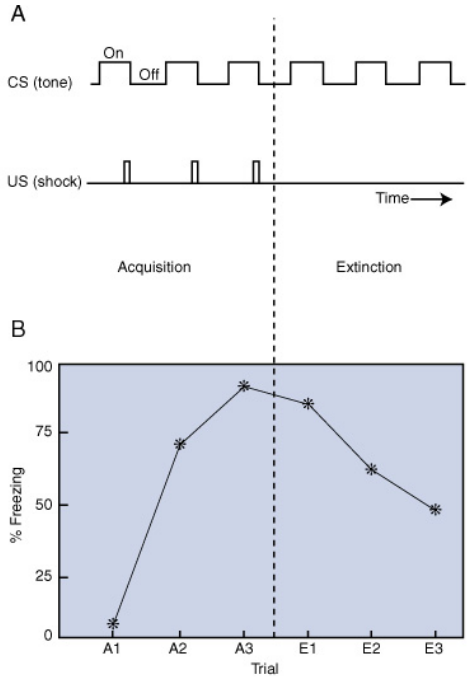
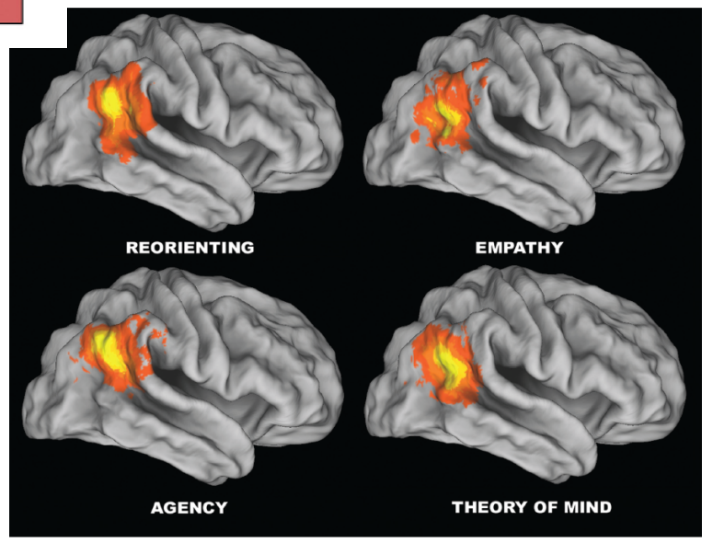


FIGURE 18.31 Fear conditioning paradigm: (A) Typical parametric arrangement of stimuli during the acquisition and extinction phases of a simple delay conditioning task. CS, conditioned stimulus; US, unconditioned stimulus. (B) Hypothetical (but realistic) acquisition and extinction learning curves for defense (freezing) responses conditioned to the CS. Note the rapid increase of freezing during the CS during acquisition trials (A1–A3) and the decline of freezing during extinction trials (E1–E3).

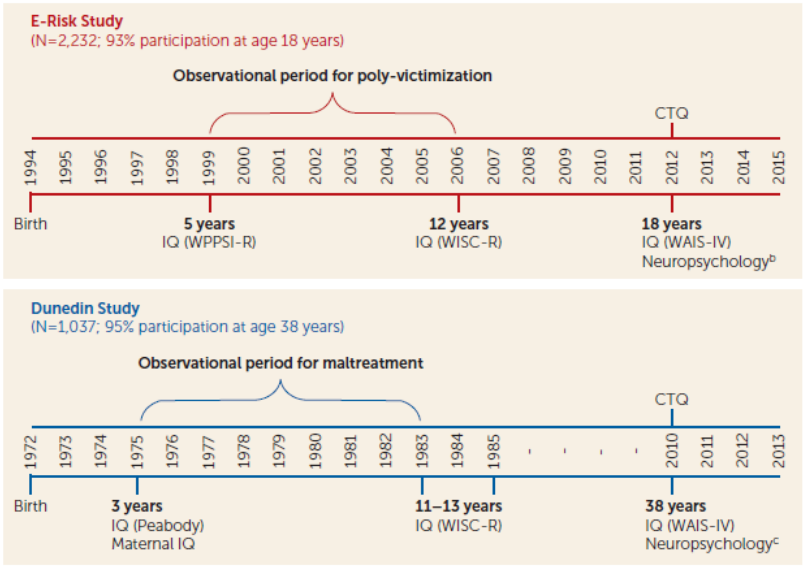
FROM MOLECULES TO NETWORKS John H. Byrne, James L. Roberts



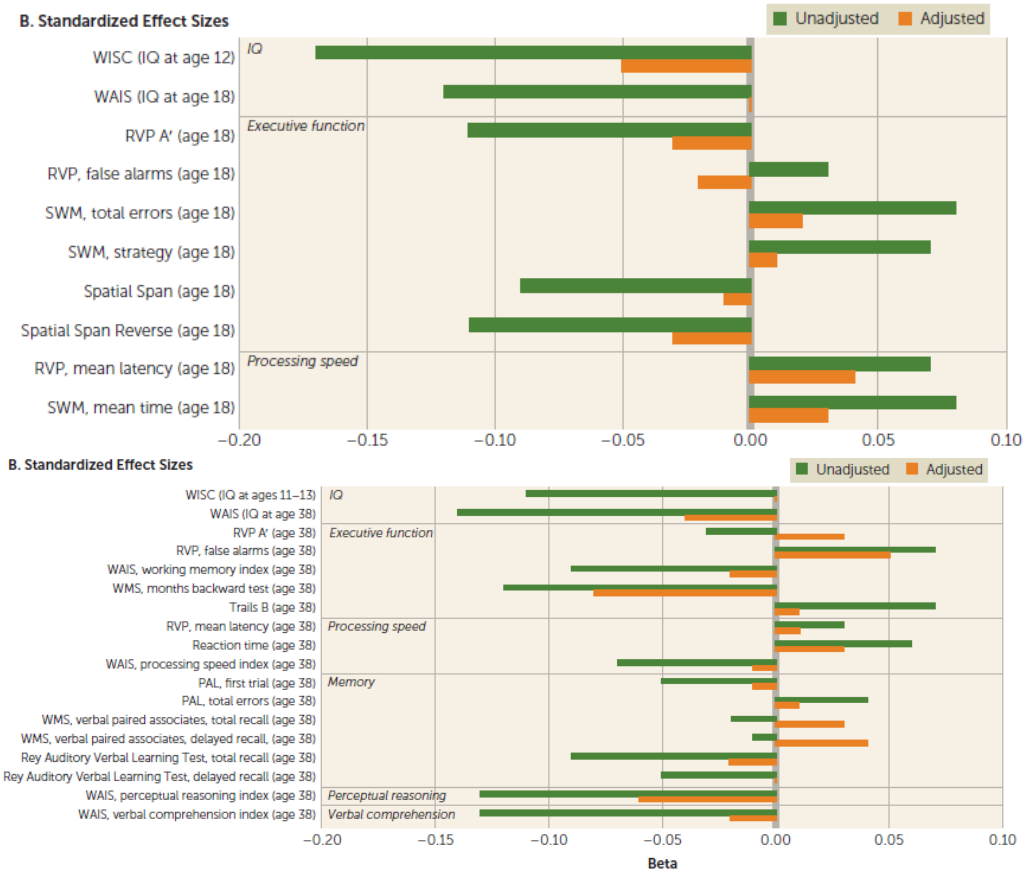
The Origins of Cognitive Deficits in Victimized Children: Implications for Neuroscientists and Clinicians

Andrea Danese, M.D., Ph.D., Terrie E. Moffitt, Ph.D., Louise Arseneault, Ph.D., Ben A. Bleiberg, B.S., Perry B. Dinardo, B.A., Stephanie B. Gandelman, B.S., Renate Houts, Ph.D., Antony Ambler, M.Sc., Helen L. Fisher, Ph.D., Richie Poulton, Ph.D., Avshalom Caspi, Ph.D.

FIGURE 1. Timeline for Assessments of Childhood Victimization and Cognitive Functioning in the E-Risk Study and the Dunedin Study[a]



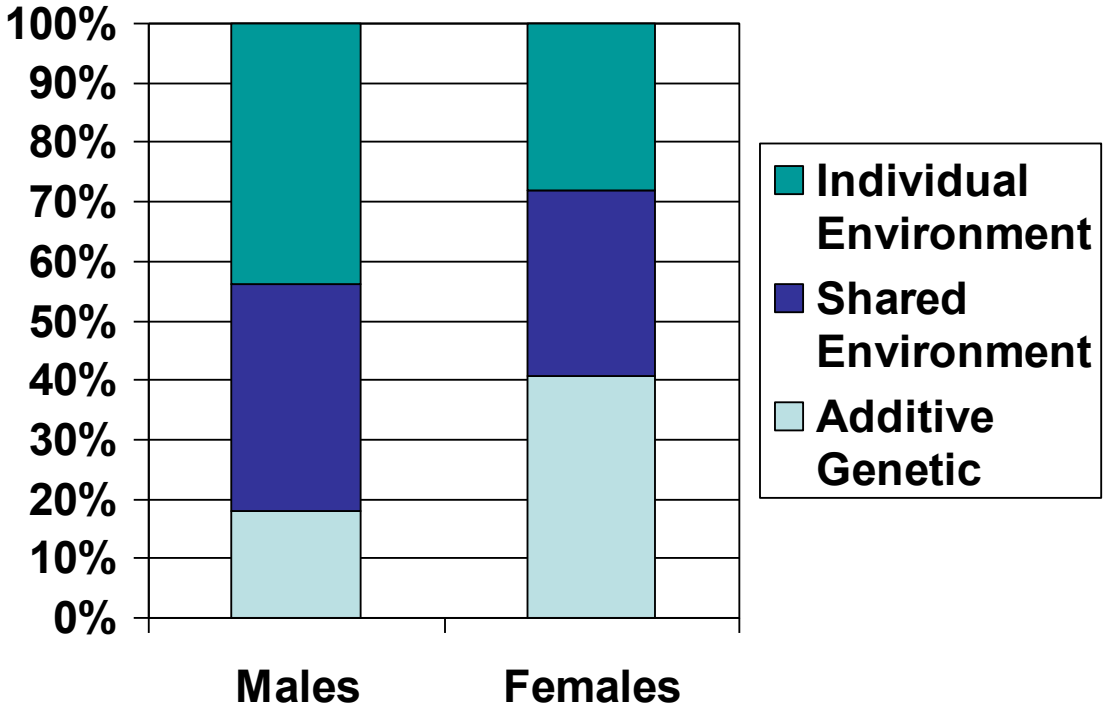
[a] CTQ=Childhood Trauma Questionnaire; Peabody=Peabody Picture Vocabulary Test; WAIS-IV=Wechsler Adult Intelligence Scale, 4th edition; WISC-R=Wechsler Intelligence Scale for Children–Revised; WPPSI-R=Wechsler Preschool and Primary Scale of Intelligence–Revised.
[b] Executive function (Cambridge Neuropsychological Test Automated Battery [CANTAB]) and processing speed (CANTAB).
[c] Executive function (CANTAB, WAIS-IV, Wechsler Memory Scale–III [WMS-III], Trail Making Test, part B), processing speed (CANTAB, WAIS-IV), memory (CANTAB, WMS-III, Rey Auditory Verbal Learning Test), perceptual reasoning (WAIS-IV), and verbal comprehension (WAIS-IV).



The association between childhood violence victimization and adult IQ is probable noncausal

A MZ/DZ Twin Study of the Childhood Trauma Questionnaire

350 twin pairs from the Pennsylvania Twin Registry completed the Childhood Trauma Questionnaire



Males, Additive genetic = .18, Shared environment = .38, Nonshared environment = .44

Females: Additive genetic = .39, Shared environment = .3, Nonshared environment = .27

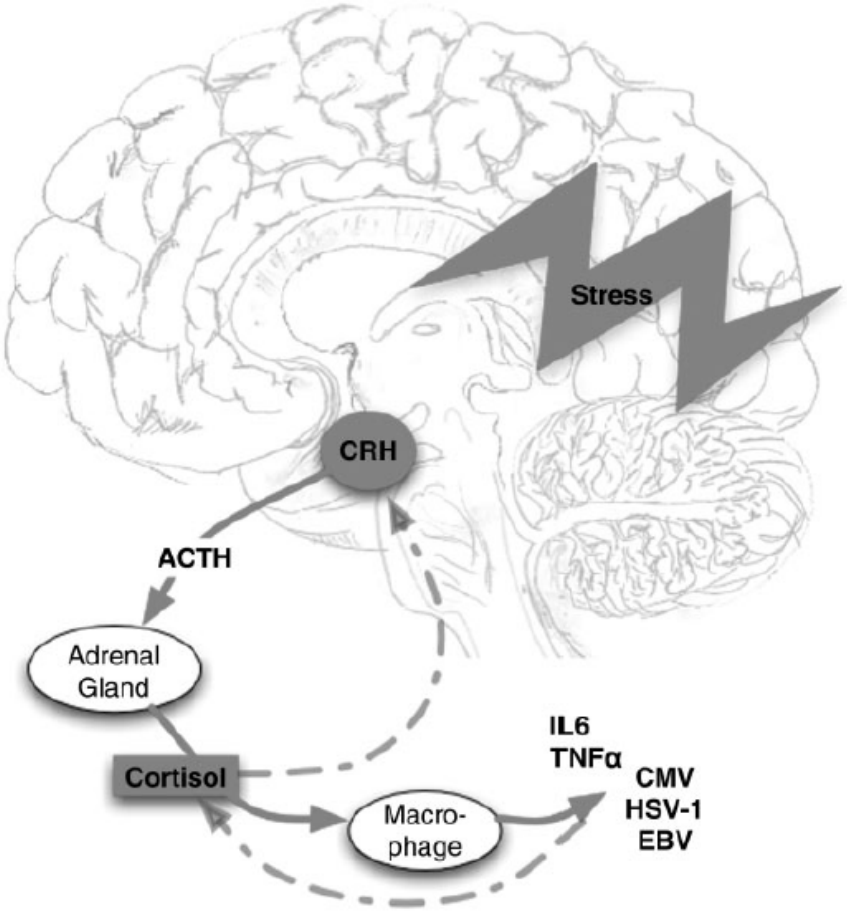


Figure 1. HPA axis and Immune Outcomes. *Note.* In response to stress, the hypothalamus releases corticotropin-releasing hormone (**CRH**). CRH availability is partly regulated by CRH binding protein (**CRHBP**), which can decrease the CRH available for **CRHR1** activation. CRH induces release of adrenocorticotropin (ACTH) and then the secretion of **cortisol** from the adrenal cortex. Cortisol binds to the glucocorticoid receptor (**NR3C1**) which plays important roles in the negative feedback loop of the HPA axis. Cortisol promotes expression of anti-inflammatory proteins while repressing the expression of pro-inflammatory ones (i.e., **IL6**, **TNFα**) and activates/reactivates viral antibodies (i.e., **CMV**, **HSV-1**, **EBV**).

Nugent, Goldberg, Uddin: 2016

The Cortisol-Trauma Story

- In young children, high cortisol response at the time of trauma predicts post-traumatic symptoms
- In older children and adults, low cortisol response predicts post-traumatic symptoms
- The longer the time between the trauma and cortisol measure, the lower the cortisol measured

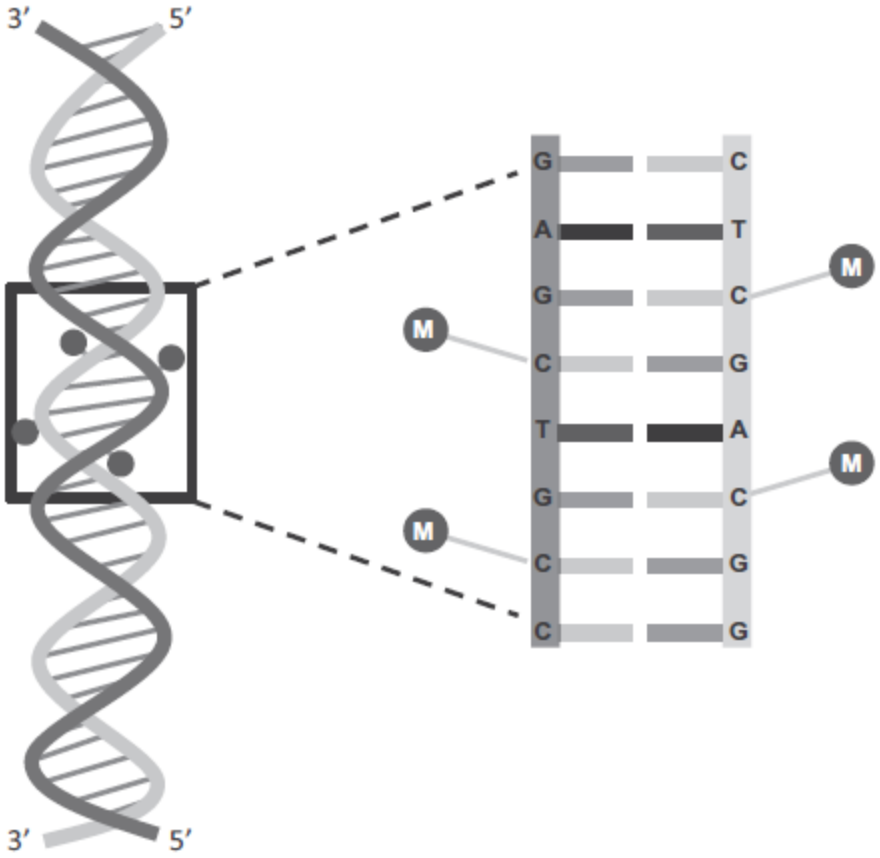
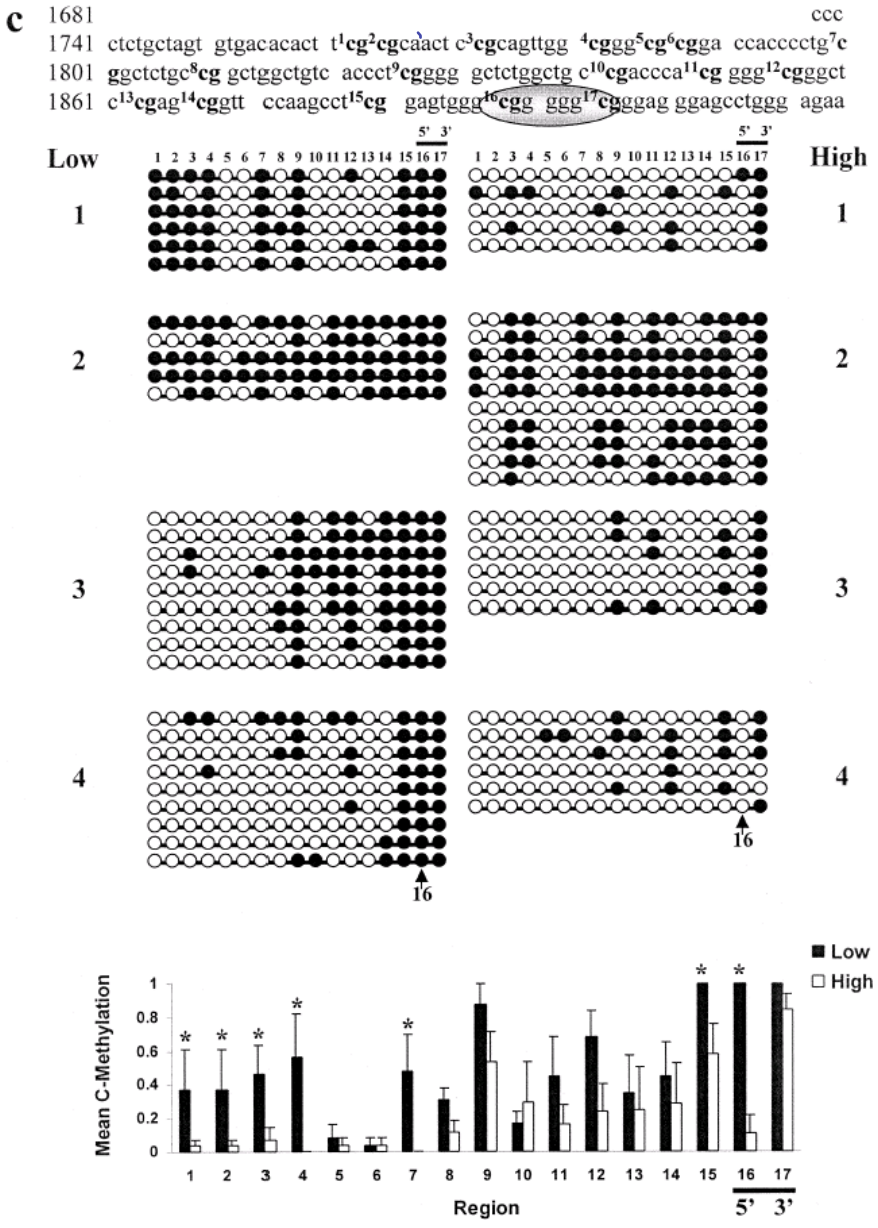
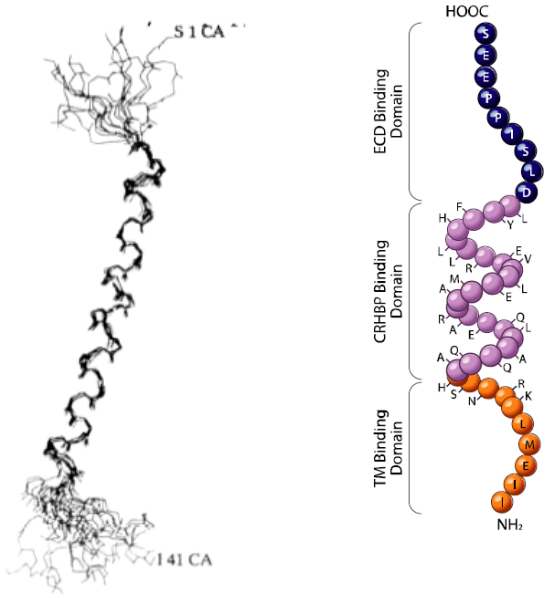


Figure 2. DNA methylation. *Note.* DNA double helix portrayed on the left, showing within the box a segment of methylated DNA (circles). Zooming in on this boxed region shows nucleotide base pairing as well as methylated cytosines (circles with "M").

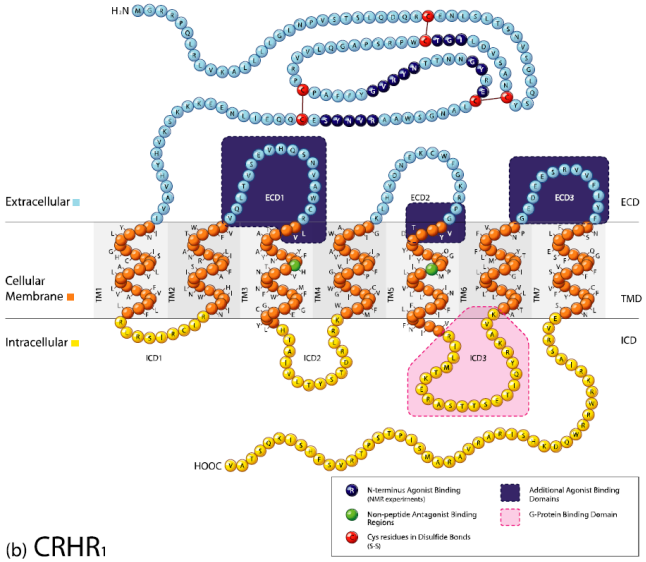
Methyl groups ("M") are covalently bonded to cytosine residues on the DNA double helix. These are stable but reversible inhibitors of gene expression.



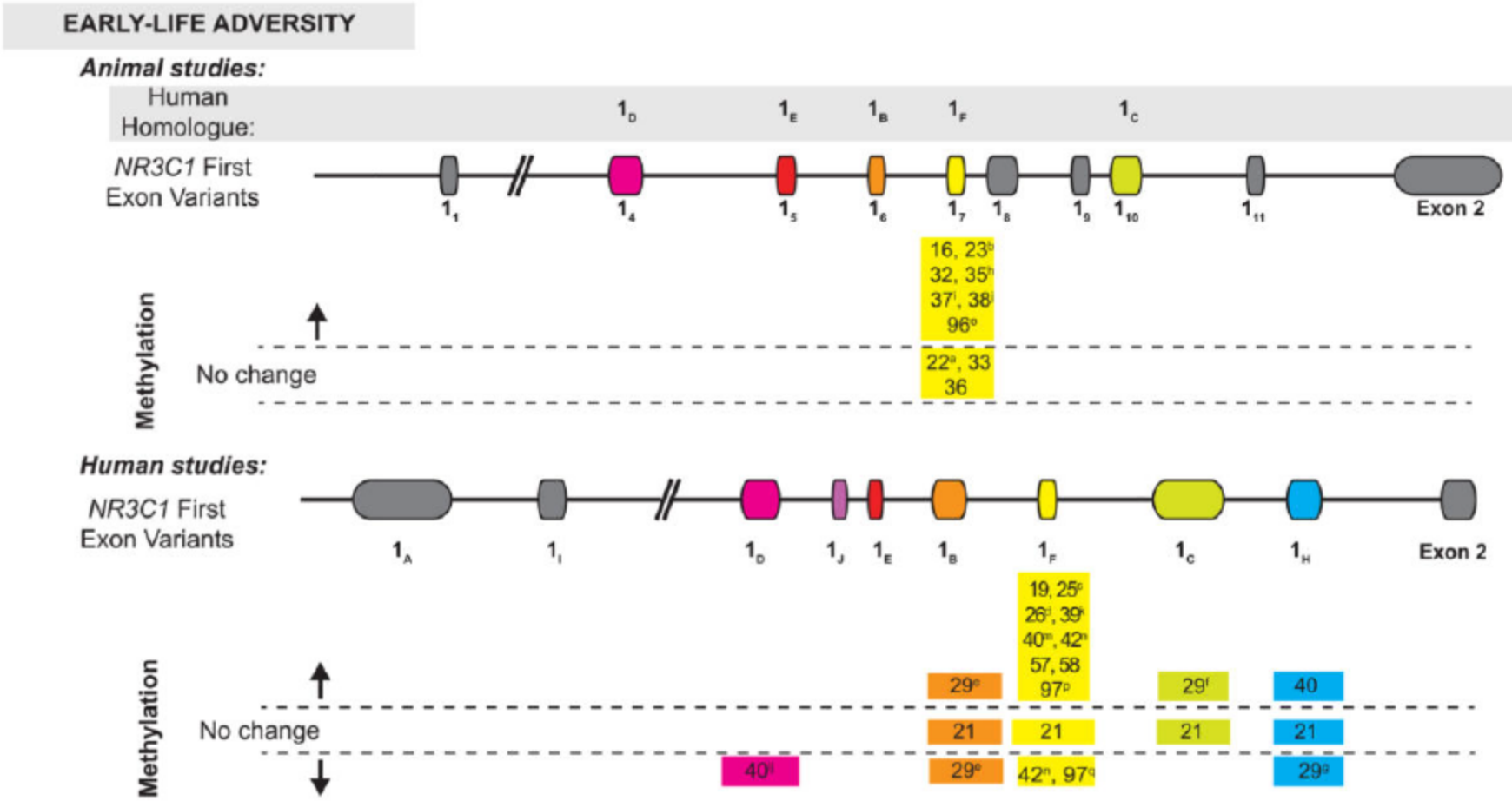
offspring. (**b**) Methylation patterns of the exon 1_7 glucocorticoid receptor promoter in the hippocampi of 1-day-old (within one hour after birth) High and Low LG-ABN offspring (N = 4 animals/group). *Top panel* shows a sequence map of the exon 1_7 glucocorticoid receptor promoter including the 17 CpG dinucleotides (highlighted in bold) and the NGFI-A binding region (*encircled*). *Middle panel* shows bead-on-string representation of the cytosine methylation status of each of the 17 individual CpG dinucleotides of the exon 1_7 glucocorticoid



(a) CRH



(b) $CRHR_1$



- Offspring of mothers with increased maternal behavior (pup licking/grooming) show increased hippocampal glucocorticoid receptor (GR: NR3C1) expression, decreased methylation of the GR 1_7 promoter, and greater negative feedback regulation over hypothalamic corticotropin releasing factor (CRF)
- Humans: GR exon variant $1_{F:}$ 27 studies, 89% positive
- Rats: GR$1_{7:}$ 13 animal studies, 70% positive

Turecki G, Meaney M: Effects of the social environment and stress on glucocorticoid receptor gene methylation: a systematic review. Biol Psychiatry 2016; 79: 87 – 96.

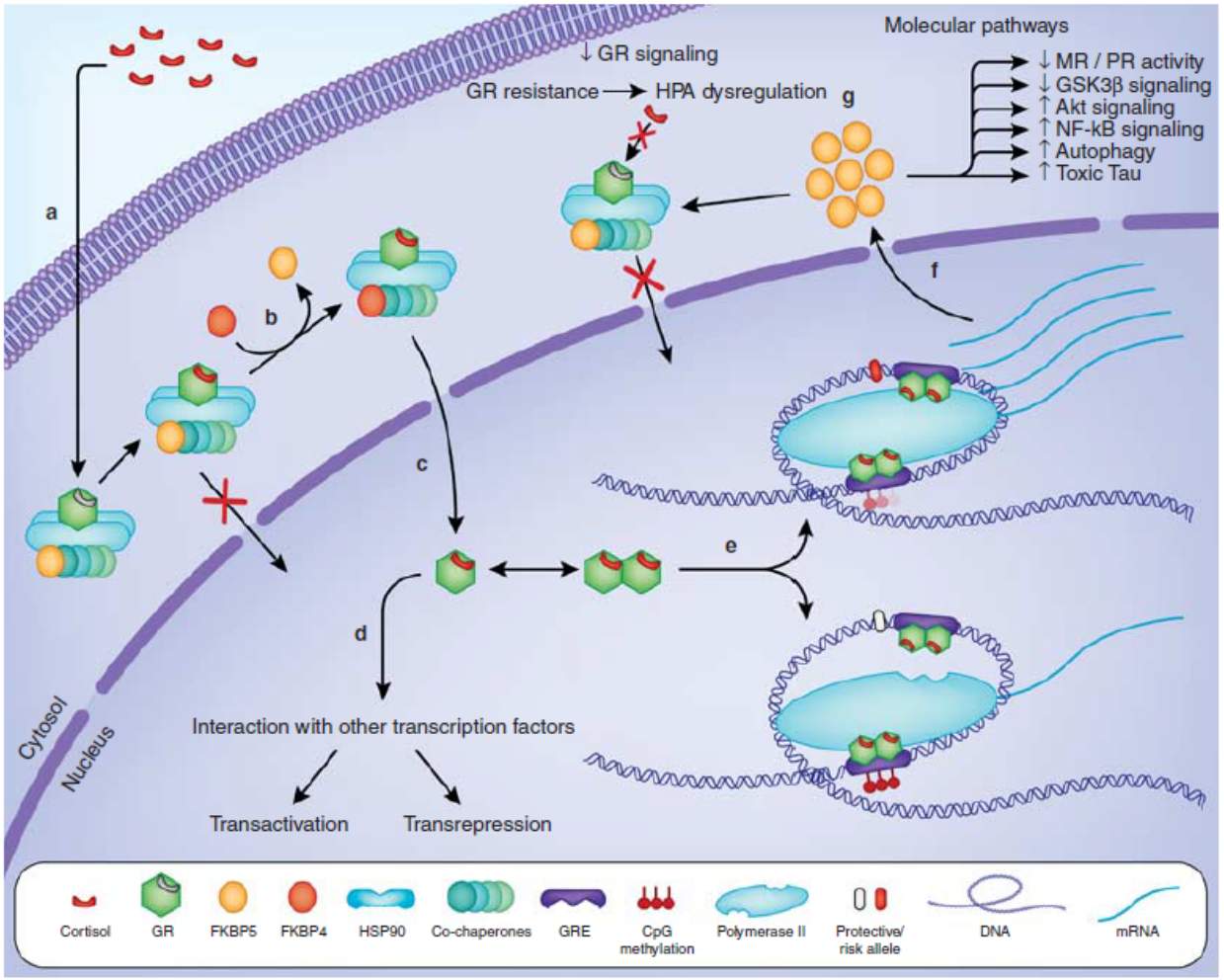


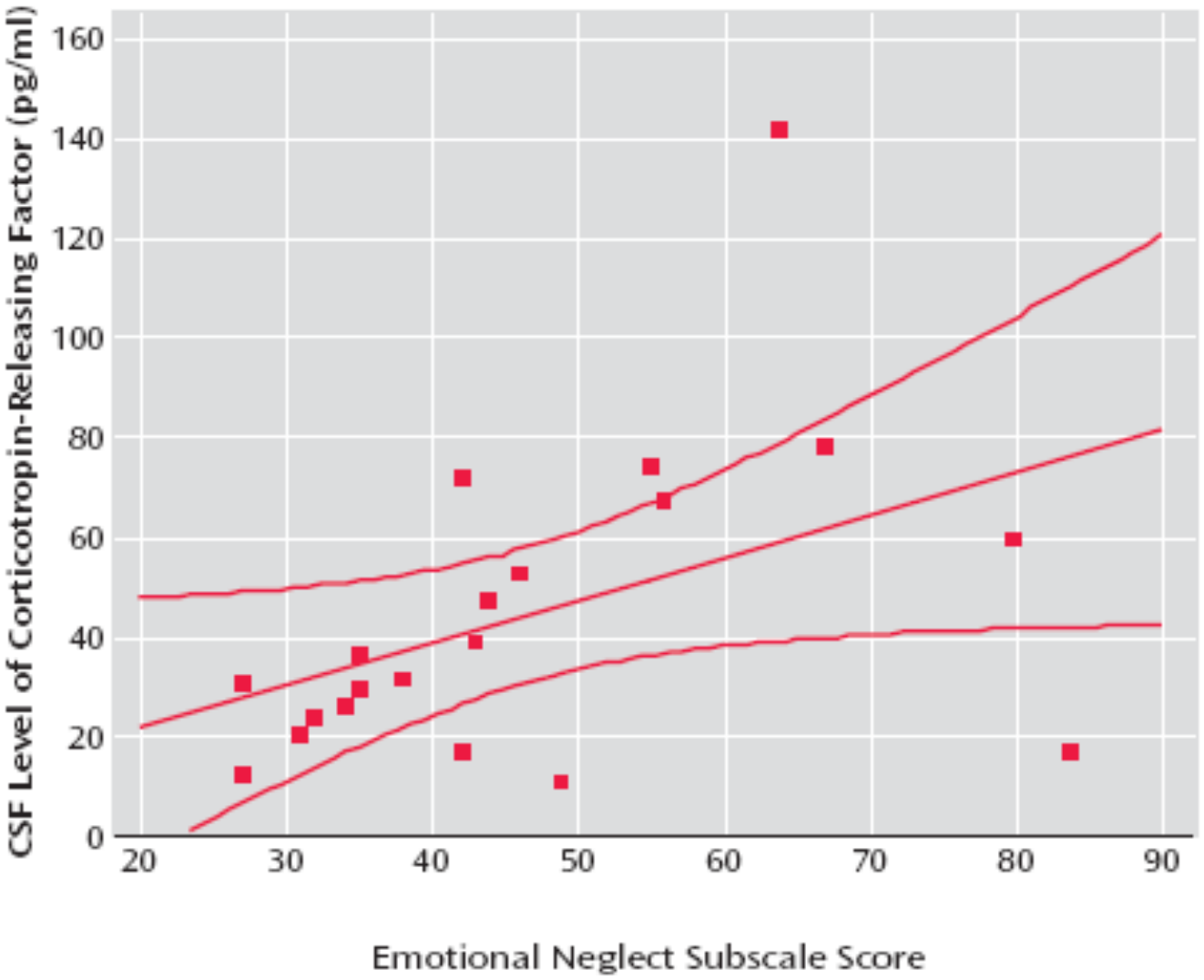
Figure 1. Schematic representation of the molecular events involved in glucocorticoid-mediated FKBP5 induction, the resulting intracellular negative feedback loop, and effects on other biological processes. Glucocorticoids enter the cytoplasm (a) and activate the glucocorticoid receptor (GR) complex. FKBP5 binding to the complex reduces affinity of glucocorticoids to the GR and delays translocation of the GR to the nucleus. However, exchange of FKBP5 for FKBP4 (b) results in GR translocation to the nucleus (c). The GR can either interact as a monomer with other transcription factors (d) or form a homodimer that binds to DNA at glucocorticoid response elements. Overall, GR functions result in transactivation or transrepression of a large number of genes. The *FKBP5* gene is highly responsive to GR, but responsiveness depends on *FKBP5* polymorphisms and methylation status (e). The synthesized *FKBP5* mRNA translocates to the cytoplasm (f) where it is translated into FKBP5 protein. FKBP5 then inhibits GR activity not only forming an ultra-short, intracellular negative feedback loop of GR signaling but also modulating several other biological pathways (g).

FKBP5 inhibits glucocorticoid receptor (GR) signaling via intracellular pathways: it interferes with GR complex interactions within the cell, nuclear translocation, and GR dependent transcriptional activity

Epigenetics of FKBP5

- FKBP5 gene is located on chromosome 6 (6p21.31)
- FKBP5 transcription is induced by GR activation
- FKPB5 is induced by cortisol and steroids
- Td1360780 T allele carriers exposed to childhood abuse have lower methylation of CpG sites located near the functional FKBP5 intron 7 GRE in peripheral blood (Klengel and Binder 2013). Similar effects can be induced by childhood dexamethasone exposure

Zannas AS, Wiechmann T, Gassen NC, Binder EB: Gene-stress-epigenetic regulation of FKBP5: Clinical and translational implications. Neuropsychopharmacology 2016; 41: 261 - 274



- Measured CSF CRH and the Childhood Trauma Questionnaire in 20 men with and without personality disorder

Childhood Trauma and Central CRH Elevations in Adults with Personality Disorder

N = 54

Main measure:
Parental Bonding Inventory

Table 3 Results of Linear Regression Analysis of Model Predicting CSF CRF (pg/ml) ($n = 54$)

Predictor	ΔR^2	F	d,f	p	B	SE	β	t	P
Step 1	0.057	1.532	2,51	0.23					
Sex					0.054	5.809	−0.034	−0.256	0.799
Age					0.522	0.306	0.232	1.706	0.094
Step 2	0.122	2.666	4,49	0.04					
PD			2.589	5.191	0.068	0.499	0.62		
PBI care					−0.689	0.286	−0.337	−2.412	0.02
Step 3	0.057	2.967	5,48	0.02					
PBI × PD					−13.020	6.857	−0.651	−1.899	0.06

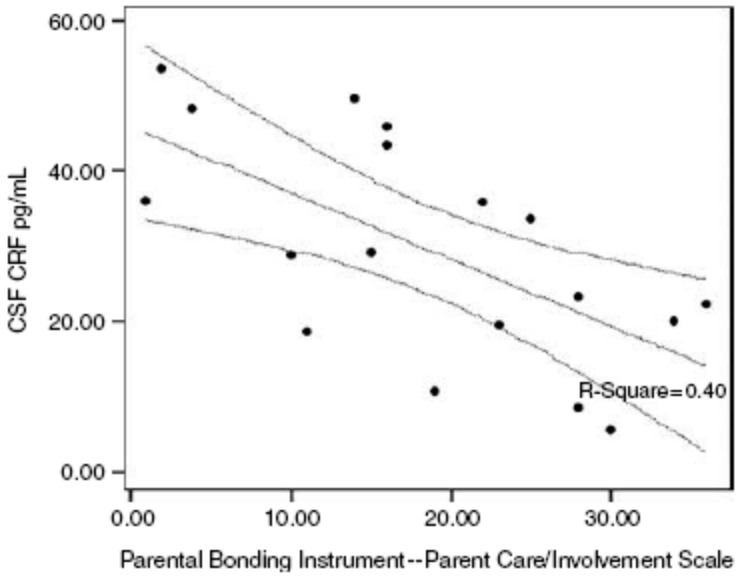


Figure 1 Scatterplot of PBI Care × CSF CRF level (pg/ml) in 18 PD subjects, excluding 19 subjects previously reported on in Lee *et al*, 2005 (Pearson's $r = -0.635$, $p = 0.005$, $n = 18$).

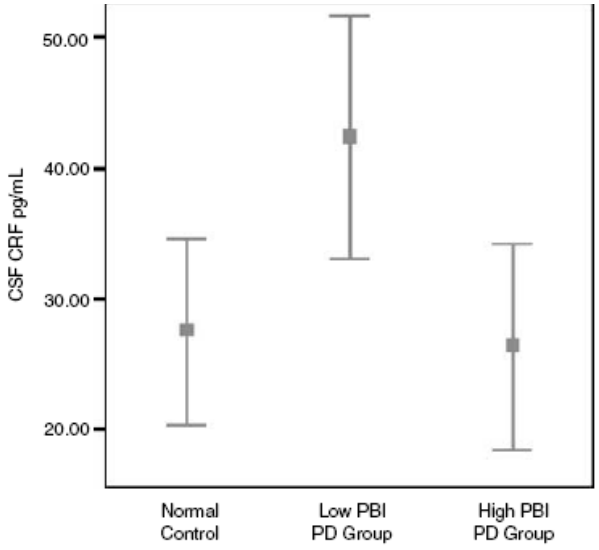
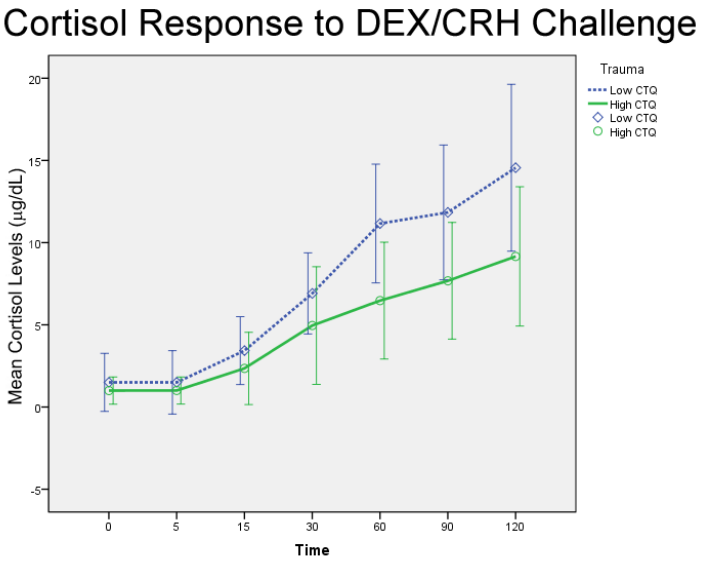
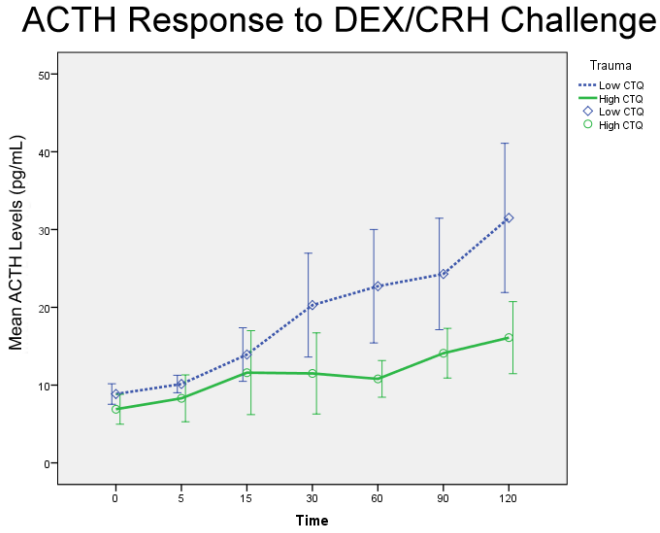


Figure 2 Mean CSF CRF (pg/ml) and 95% confidence interval of the mean in normal controls ($n = 17$), Low PBI care PD Subjects ($n = 18$), and high PBI care PD subjects ($n = 18$). Mean CSF CRF (pg/ml) in the low PBI PD group (44.148, SD = 18.169) was significantly higher than mean CSF CRF (pg/ml) in the normal control (27.587, SD = 13.936) and high PBI PD group (26.366, SD = 15.901). No significant difference in mean CSF CRF was found between the normal control and high PBI PD group.

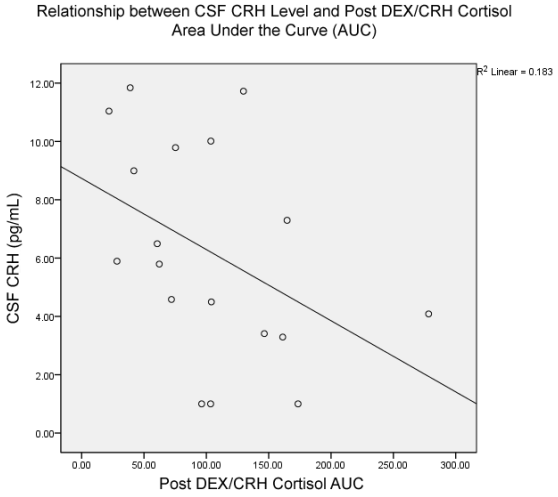
CORTISOL RESPONSE TO EXOGENOUS CRH IS SIGNIFICANTLY BLUNTED IN ASSOCATION WITH CHILDHOOD TRAUMA IN PERSONALITY DISORDER

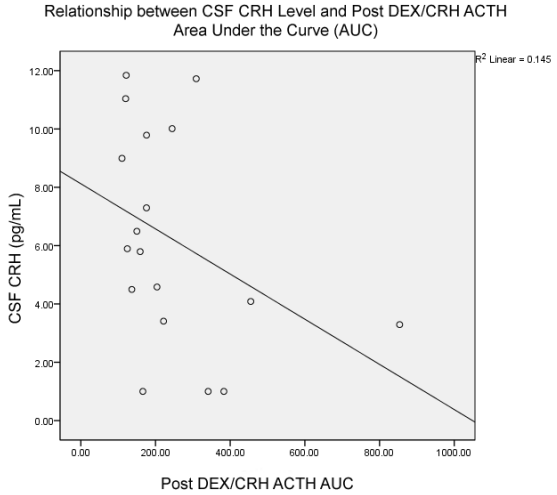


Mean Post DEX/CRH Cortisol Level: Following CRH challenge (Time = 0), cortisol levels in the Low CTQ group (N=14) are significantly higher than the High CTQ group (N=10) at time point +60, +90, and +120 minutes. The error bars indicate the 95% confidence interval of the mean.



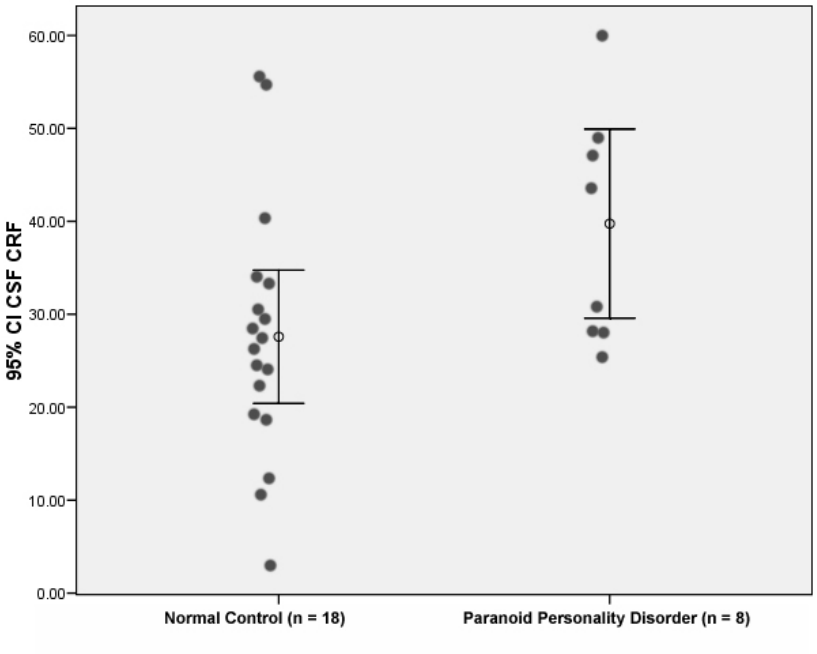
Mean Post DEX/CRH ACTH Levels: Following CRH challenge (Time = 0), ACTH levels in the Low CTQ group (N = 14) are signficantly higher than in the High CTQ group (N = 10) at time points +5, +30, +60, +90, and +120 minutes. The error bars indicate the 95% confidence interval of the mean.





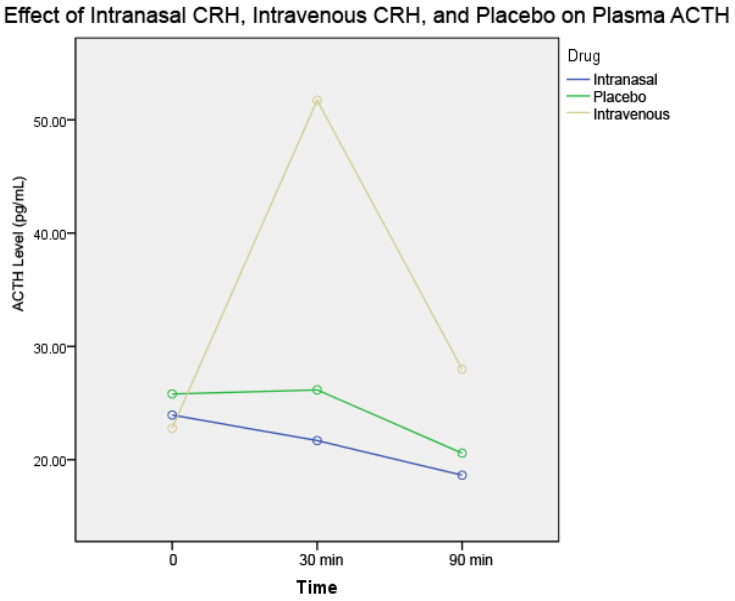
What is CRH Doing?

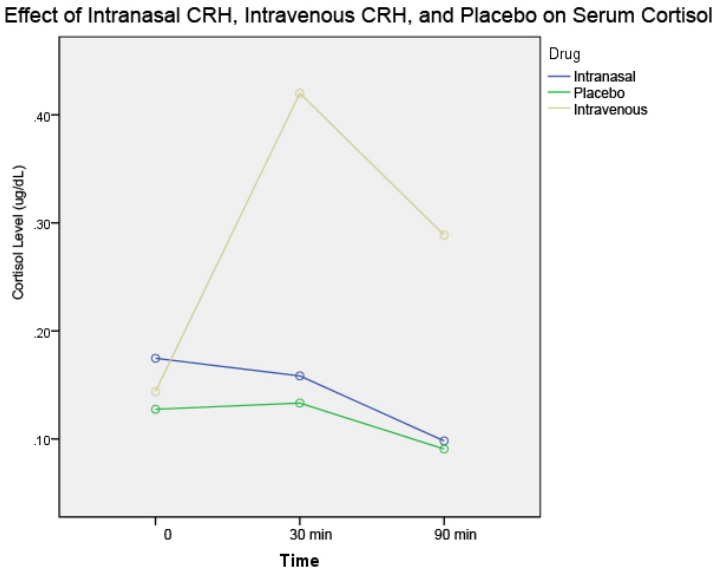
Fig. 2. The behavioral effects of ICV CRF in an adult male rhesus monkey when housed in his normal social group. Note the brown nylon jacket the animal is wearing, which contains the pump used to infuse CRF. The animal has withdrawn from his peers and is exhibiting huddling/wall-facing behavior, one of the depressive-like behaviors induced by ICV CRF in socially housed monkeys.

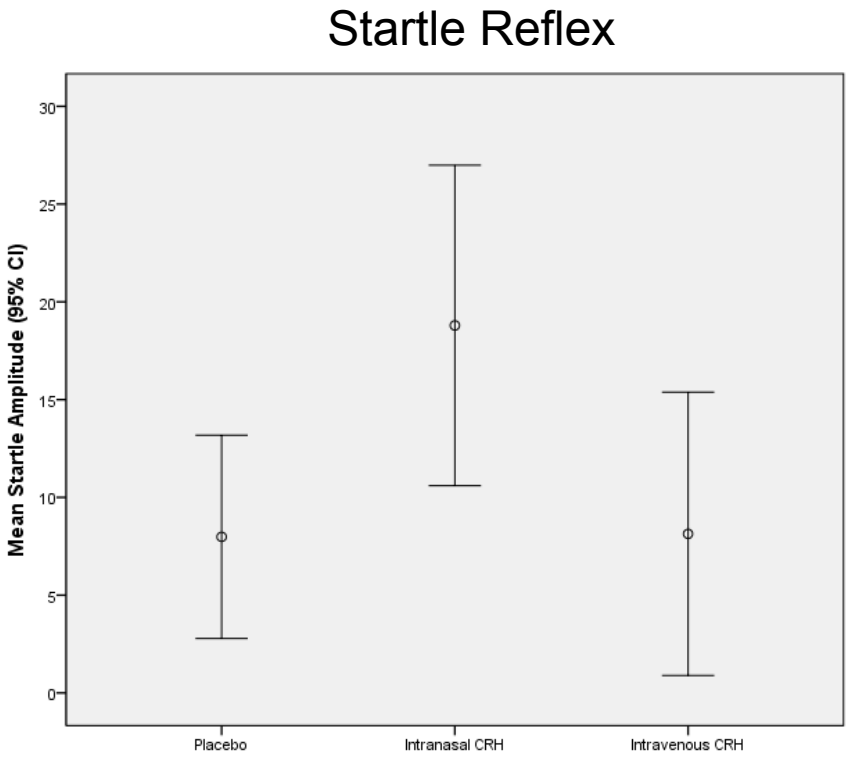


t (25) = -2.313, p = .03

Neuroendocrine Response and Startle Reactivity





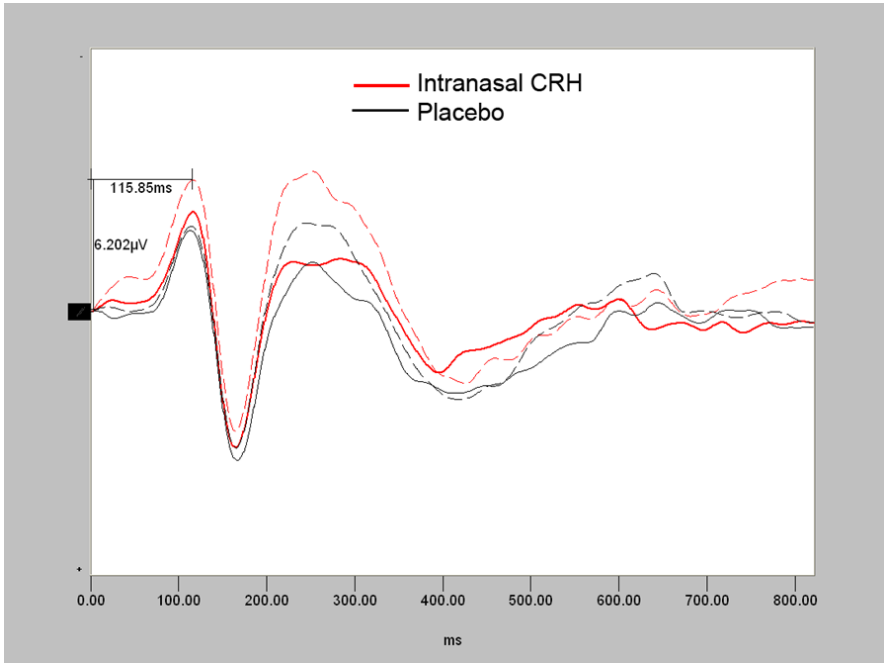


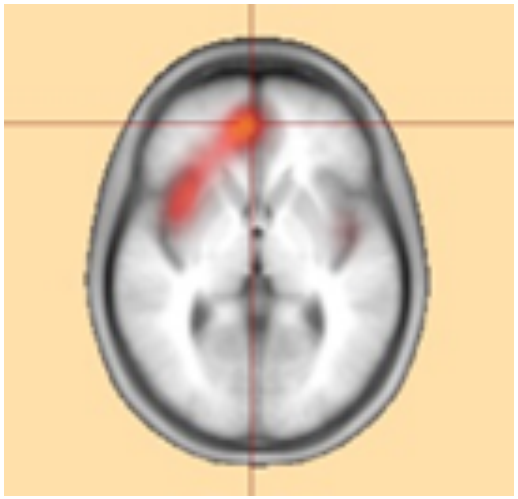
RM-ANOVA Drug: F (2, 26) = 4.462, p = .022.

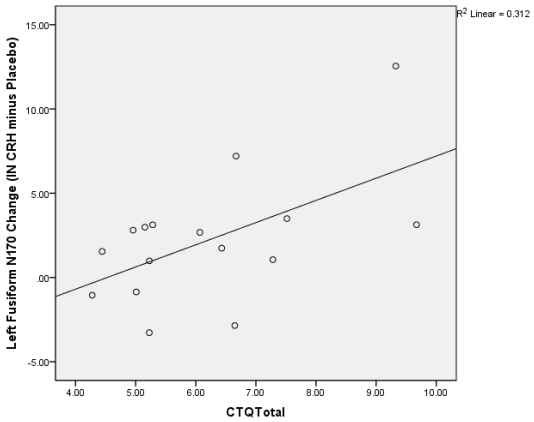
Paired t-tests:

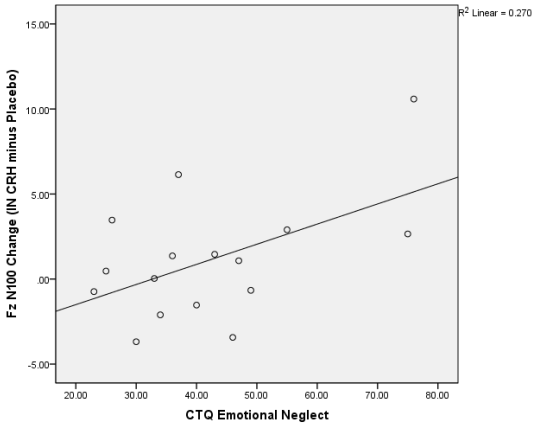
- **IN > Placebo:** t (1, 15) = -2.257, p = .039.
- **IN > IV:** t (1, 14)= 2.016, p = .063.

IN CRH Enhances N100 Amplitude to Neutral Faces









ΔN100 (IN) is correlated with CTQ Total score (r = -.484, p = 0.06)

ΔN100 (IN) is correlated with CTQ Emotional Neglect (r = -.593, p = 0.02)

	PPD + BPD N = 32 Mean F-test p-value	Paranoid PD N = 31 Mean F-test p-value	Borderline PD N = 91 Mean F-test p-value	Normal N = 295 Mean F-test p-value
Emotional Abuse	15.53 (4.77) F = 14.450 p < .001	11.97 (5.34) F = 16.588 p < .001	14.37 (5.0) F = 102.7 p < .001	6.79 (2.84)
Emotional Neglect	14.9 (5.09) F = 7.817 p = .005	12.74 (5.24) F = 10.468 p < .001	13.79 (5.28) F = 39.489 p < .001	8.17 (3.82)
Physical Abuse	10.78 (4.7) F = 21.220 p < .001	10.23 (4.88) F = 7.593 p = .001	11.26 (5.21) F = 35.71 p < .001	6.2 (2.1)
Physical Neglect	9.47 (2.98) F = 6.399 p = .012	8.38 (3.84) F = 10.435 p < .001	8.71 (3.65) F = 3.741 p < .001	5.86 (1.78)
Sexual Abuse	9.16 (6.1) F = 12.185 p = .001	8.35 (5.67) F = 3.017 p = .05	9.43 (6.56) F = 12.185 p < .001	5.28 (1.49)
Minimization	.06 (.25) F = 2.904 p = .09	.26 (.58) F = 1.872 p = .16	.11 (.40) F = 11.195 p < .001	.67 (1.0)

No significant differences between BPD+PPD and BPD, BPD+BPD and PPD, PPD and BPD.

	PPD + BPD N = 6 Mean F-test p-value	Paranoid PD N = 11 Mean F-test p-value	Borderline PD N = 26 Mean F-test p-value	Normal N = 81 Mean F-test p-value
Perspective Taking	16.00 (4.69)	16.80 (3.56) F = 3.84 p = .05	16.9 (4.78) F = 10.86 p = .001	19.10 (4.83)
Empathic Concern	19.67 (4.97)	19.60 (2.30) F = .08 p = 77	19.2 (4.12) F = .96 p = .33	19.3 (4.93)
Personal Distress	13.33 (5.64)	13.60 (3.58) F = 8.01 p = .01	12.60 (5.22) F = 10.203 p = .002	8.71 (4.65)
Fantasy	14.17 (5.31)	15.40 (5.59) F = .00 p = 1.0	15.3 (4.76) F = .14 p = .71	14.33 (5.81)

Due to the limited sample size, power was lacking for a single model incorporating PPD and BPD. For exploratory purposes, two separate Multivariate ANCOVAs were performed for PPD and BPD (shown in the table). Comparisons between the PPD+BPD, PPD, and BPD groups did not result in any significant differences.

Childhood trauma and parental style: Relationship with markers of inflammation, oxidative stress, and aggression in healthy and personality disordered subjects

Jennifer R. Fanning[a], Royce Lee[a], David Gozal[b], Mary Coussons-Read[c], Emil F. Coccaro[a,*]

Path coefficients for model 1: childhood abuse, CRP, state depression, and aggression.

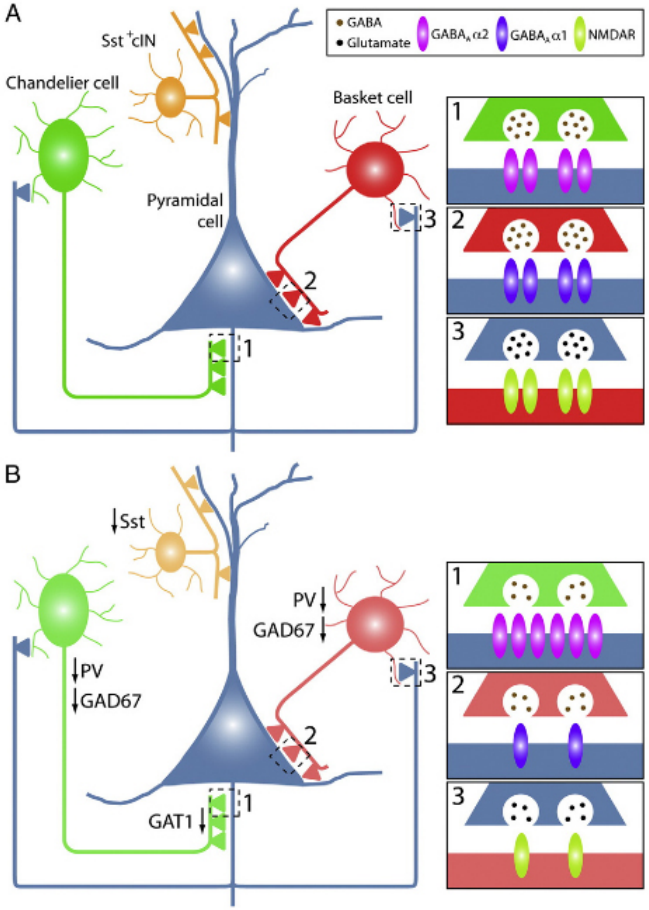
	Consequent								
	CRP(M1)			Current depression(M2)			Aggression(Y)		
Antecedent	Coeff.	SE	p	Coeff.	SE	p	Coeff.	SE	p
CTQ abuse (X)	0.23	0.08	0.006	0.32	0.08	<0.001	0.20	0.08	0.011
CRP(M1)	–	–	–	0.32	0.09	<0.001	0.21	0.08	0.009
Current depression(M2)	–	–	–	–	–	–	0.34	0.08	<0.001
BMI (COV1)	0.22	0.08	0.009	−0.10	0.08	0.245	−0.06	0.07	0.428
Cigarette use (COV2)	0.15	0.08	0.067	−0.10	0.08	0.230	0.05	0.07	0.467
Constant	0.00	0.08	1.000	0.00	0.08	1.000	0.05	0.07	0.428
	$R^2 = 0.17$			$R^2 = 0.22$			$R^2 = 0.36$		
	$F(3,130) = 8.96, p < 0.001$			$F(4,129) = 9.29, p < 0.001$			$F(5,128) = 14.19, p < 0.001$		

CTQ = childhood trauma questionnaire; CRP = C-reactive protein; BMI = body mass index.

Table 3B
Path coefficients for model 2: parental control, IL-6, CRP, state depression, and aggression.

	Consequent											
	IL-6[a](M1)			CRP(M2)			Current depression (M3)			Aggression(Y)		
Antecedent	Coeff.	SE	p	Coeff.	SE	p	Coeff.	SE	p	Coeff.	SE	p
PBI control (X)	0.28	0.09	0.002	0.11	0.08	0.183	0.38	0.08	<0.001	0.13	0.08	0.104
IL-6[a](M1)	–	–	–	0.36	0.08	<0.001	0.05	0.09	0.566	0.12	0.08	0.117
CRP(M2)	–	–	–	–	–	–	0.39	0.09	0.002	0.18	0.08	0.037
BDI-II(M3)	–	–	–	–	–	–	–	–	–	0.34	0.08	<0.001
BMI (COV1)	0.13	0.09	0.156	0.16	0.08	0.059	−0.16	0.08	0.057	−0.08	0.08	0.314
Cigarette use[a] (COV2)	−0.03	0.08	0.701	0.18	0.08	0.019	−0.07	0.08	0.385	0.08	0.07	0.276
Constant	0.00	0.08	1.000	0.00	0.07	1.000	0.00	0.08	1.000	0.05	0.07	0.431
	$R^2 = 0.13$			$R^2 = 0.28$			$R^2 = 0.27$			$R^2 = 0.35$		
	$F(3,130) = 5.78, p < 0.001$			$F(4,129) = 12.33, p < 0.001$			$F(5,128) = 9.25, p < 0.001$			$F(6,127) = 11.63, p < 0.001$		

PBI = parental bonding inventory; IL-6 = interleukin-6; CRP = C-reactive protein; BMI = body mass index.



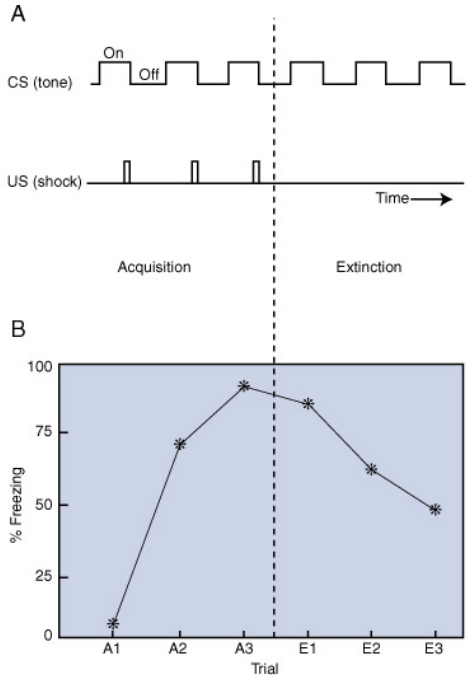
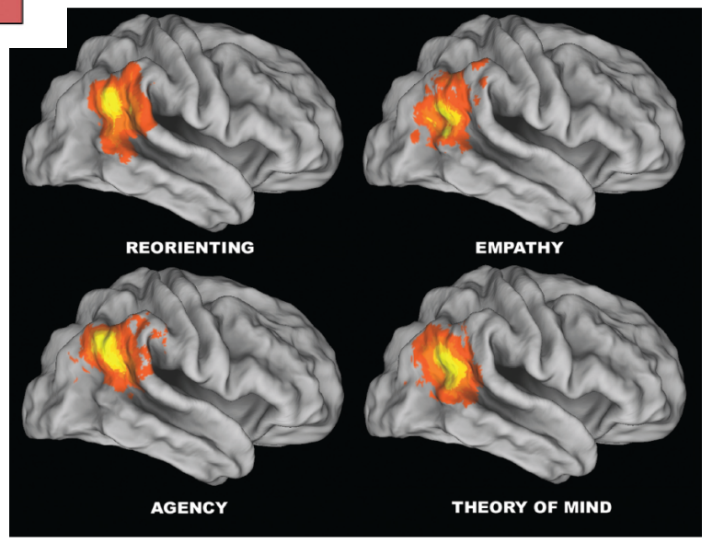


FIGURE 18.31 Fear conditioning paradigm: (A) Typical parametric arrangement of stimuli during the acquisition and extinction phases of a simple delay conditioning task. CS, conditioned stimulus; US, unconditioned stimulus. (B) Hypothetical (but realistic) acquisition and extinction learning curves for defense (freezing) responses conditioned to the CS. Note the rapid increase of freezing during the CS during acquisition trials (A1–A3) and the decline of freezing during extinction trials (E1–E3).

FROM MOLECULES TO NETWORKS John H. Byrne, James L. Roberts



Conflicts and Funding

Azevan Pharmaceuticals

National Institute for Mental Health

American Foundation for Suicide Prevention (AFSP)

National Alliance for Research in Schizophrenia and Depression (NARSAD)

Guggenheim Foundation

Brain Research Foundation

Institute of Translational Medicine

Childhood Trauma: Epidemiology, Health Consequences, and Possibilities for Interventions

Natalie Slopen, ScD
Assistant Professor, Epidemiology and Biostatistics
School of Public Health
University of Maryland, College Park

Rutgers Center on Law and Justice
May 5, 2017

Objectives

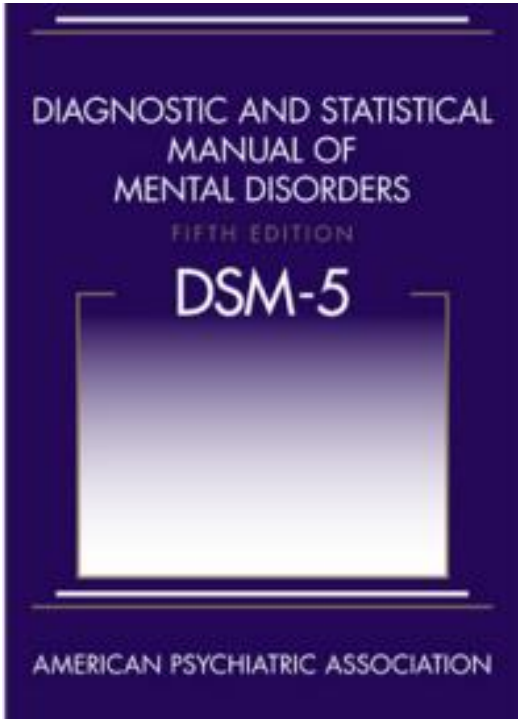
1. Epidemiology of childhood adversity and trauma in the U.S.
2. Childhood trauma and health across the life course.
3. Possibilities for social intervention to improve early life physiological dysregulation.

“Trauma”

Exposure to actual or threatened death, serious injury, or sexual violation.

Can occur via:

- Direct exposure
- Witness event in person
- Learn event occurred to close family/friend



Taxonomy of "Stress"

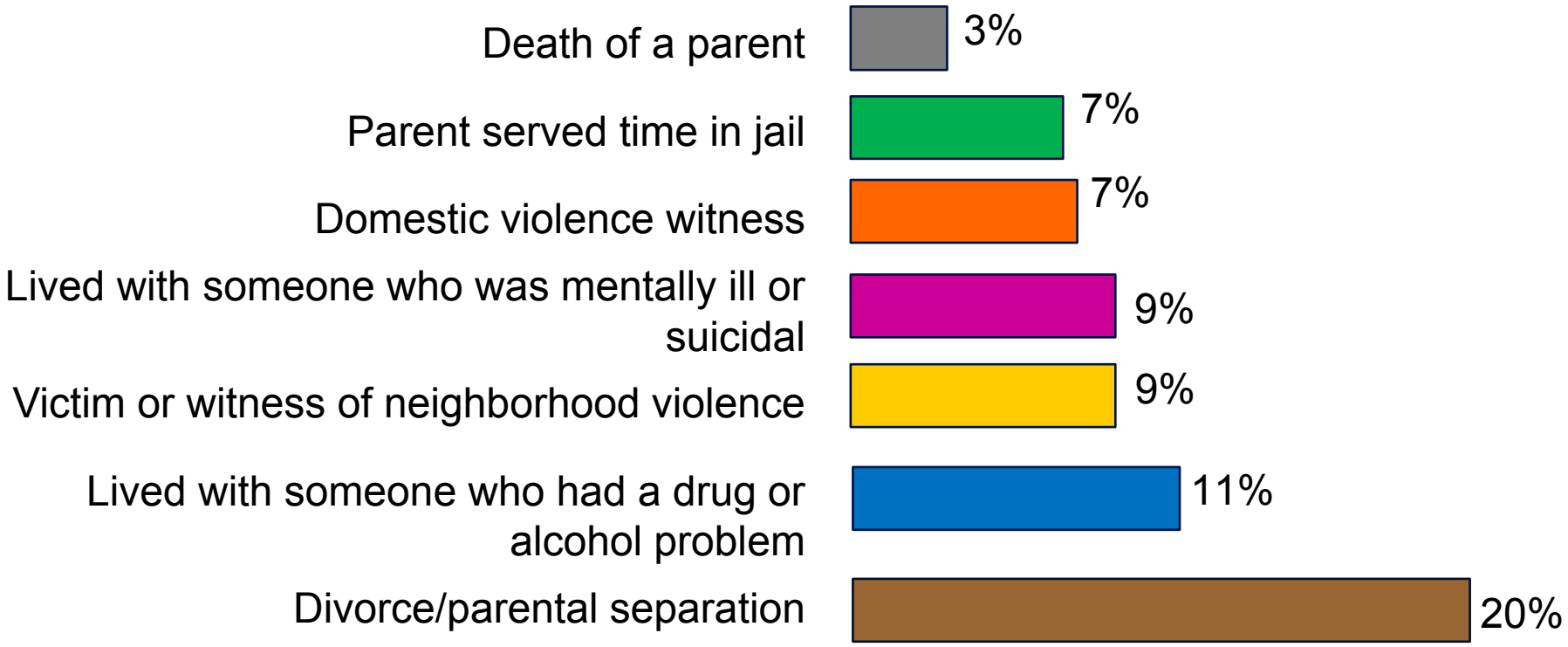
Brief increases in heart rate, mild elevations in stress hormone levels.

Serious, temporary stress responses, buffered by supportive relationships.

Prolonged activations of stress response systems in the absence of protective relationships.

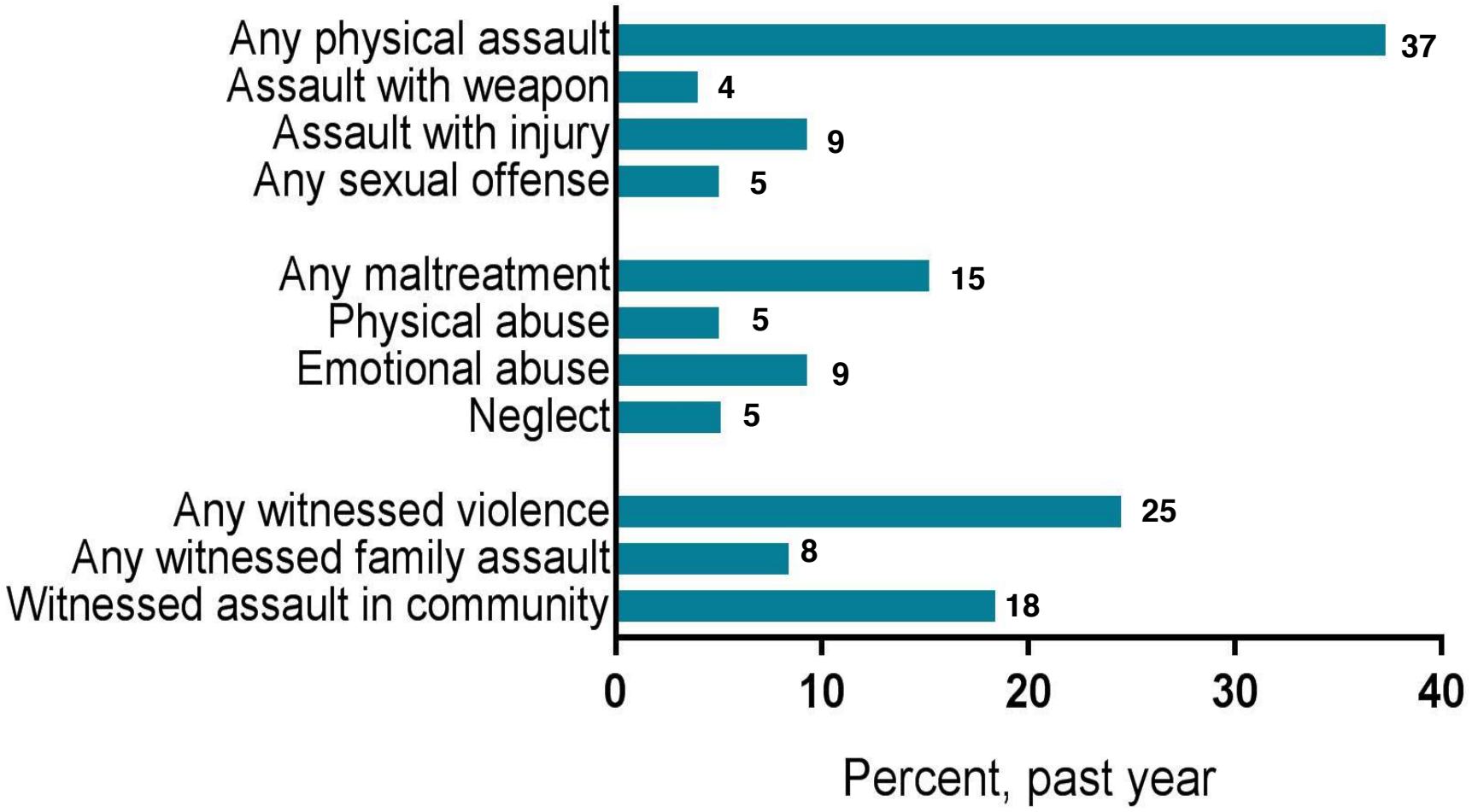
Shonkoff, J.P., Garner, A.S., 2012. Pediatrics 129:E232-E46;
Figure from Center on the Developing Child, Harvard University.

Adverse Child or Family Experiences: Children 0 to 17 years



Source: National Survey of Child Health, 2011-2012 (N=95,677); lifetime reports.
Slopen et al, *Am J Prev Med*, 2016, 50(1): 47-56.

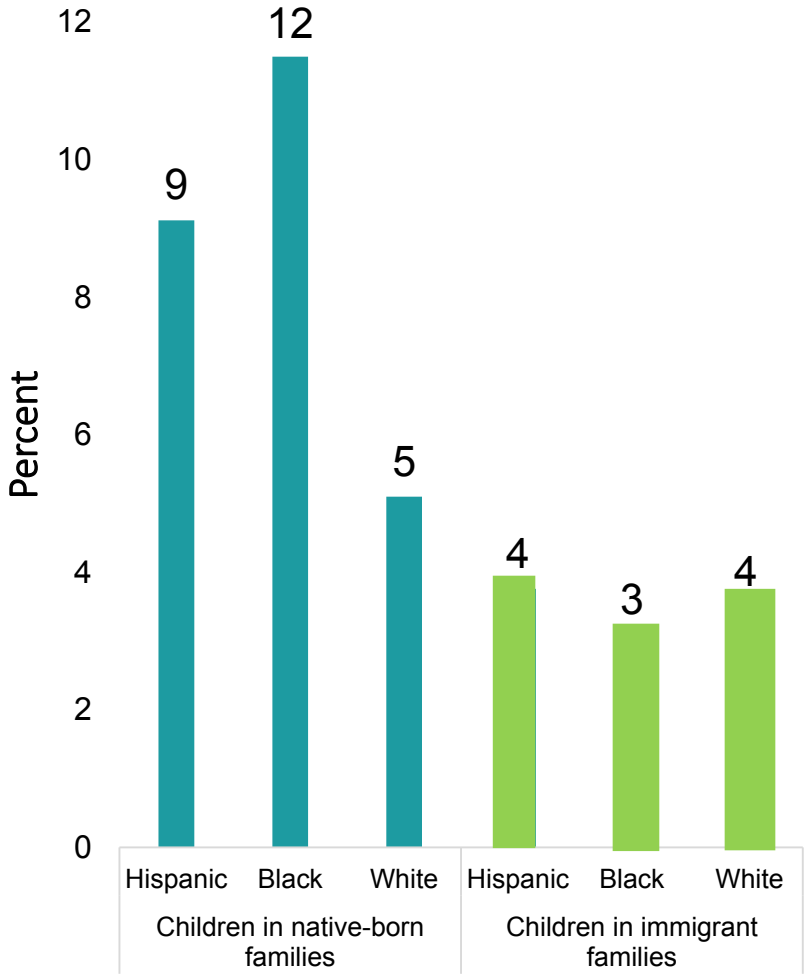
Past Year Violence Experiences: Children 0 to 17 years

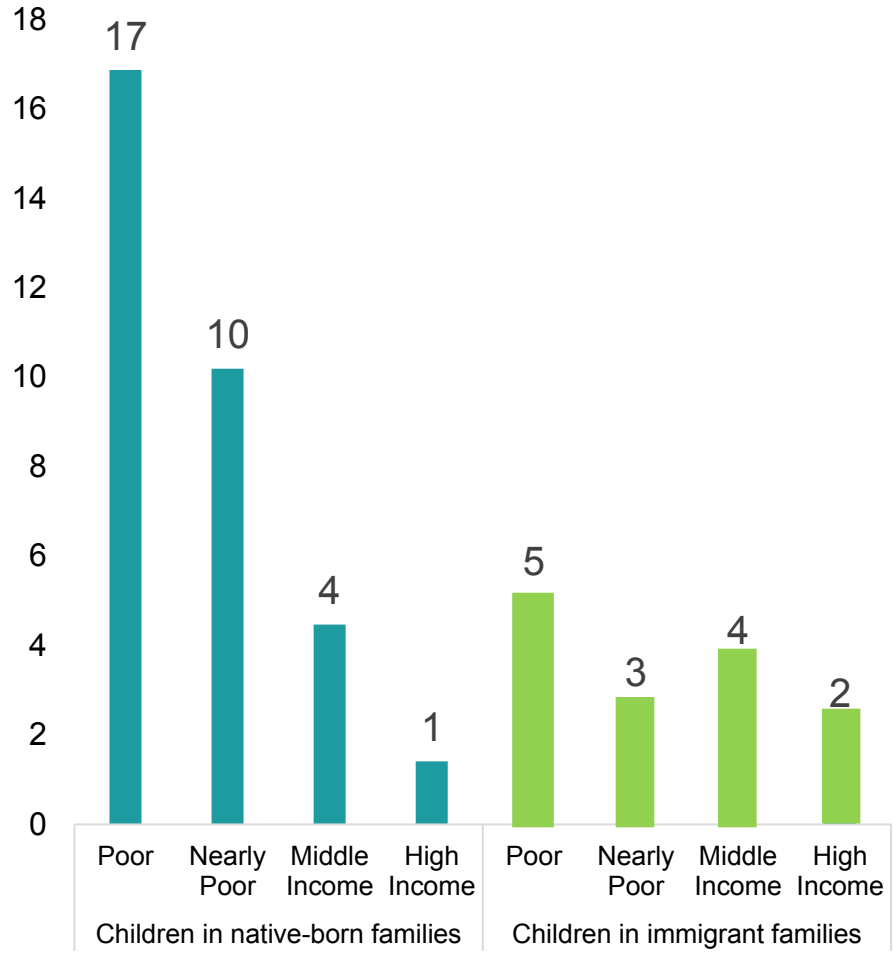


Source: National Survey of Children's Exposure to Violence 2013-2014
Finkelhor D et al. *JAMA Pediatr.* 2015;169(8):746-754.

Patterns by Race, Nativity, & Income

Example: parent served time in jail





Slopen et al, *Am J Prev Med*, 2016, 50(1): 47-56.
Source: National Survey of Child Health, 2011-2012 (N=95,677); poverty categories based on federal poverty thresholds.

Objectives

1. Epidemiology of childhood adversity and trauma in the U.S.
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3. Possibilities for social intervention to improve early life physiological dysregulation.

Adverse Childhood Experiences (ACE) Study

> 17,000 HMO members answered a questions about their childhood experiences.

Responses linked to medical records.

Ground-breaking, and sparked a large body of research with vast public health implications.

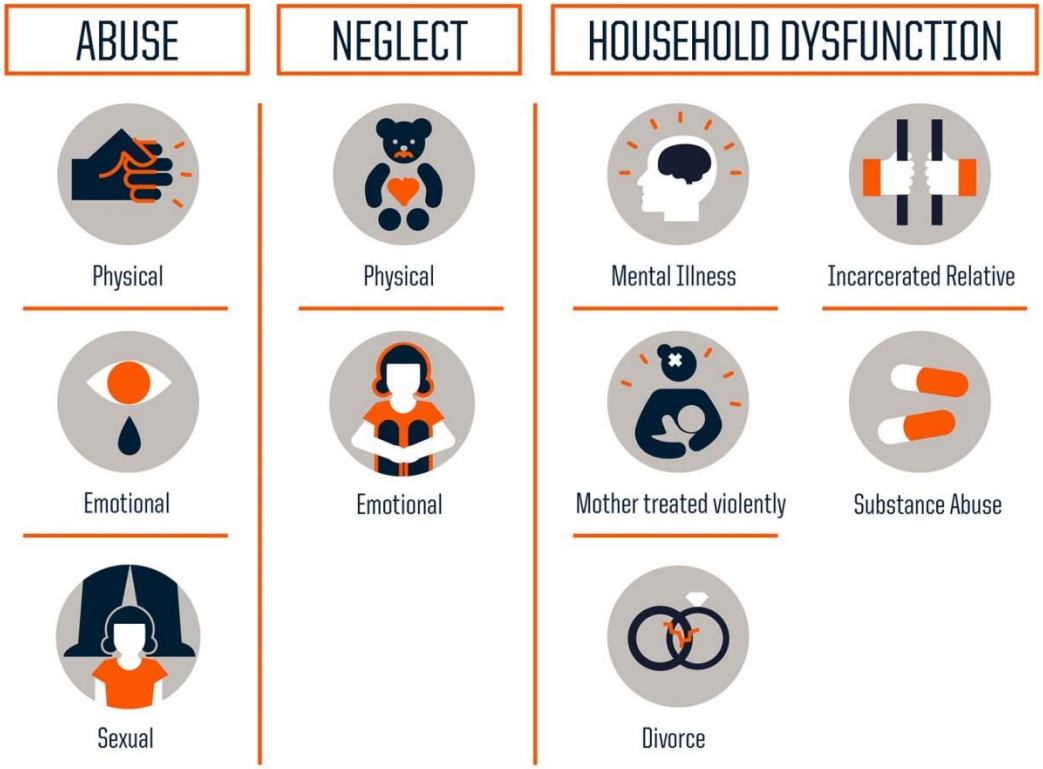
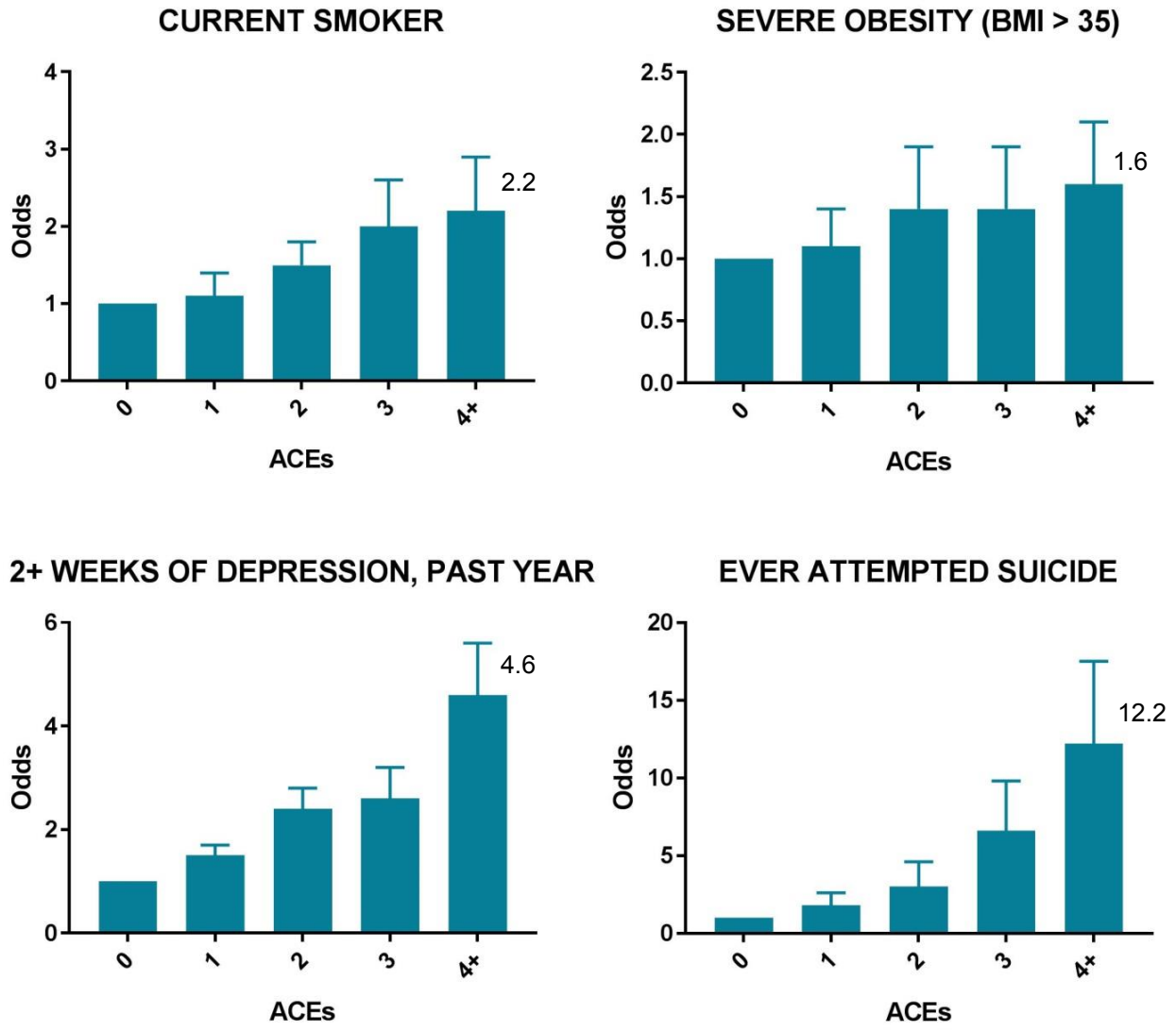


Figure: RWJF: www.rwjf.org/vulnerablepopulations.

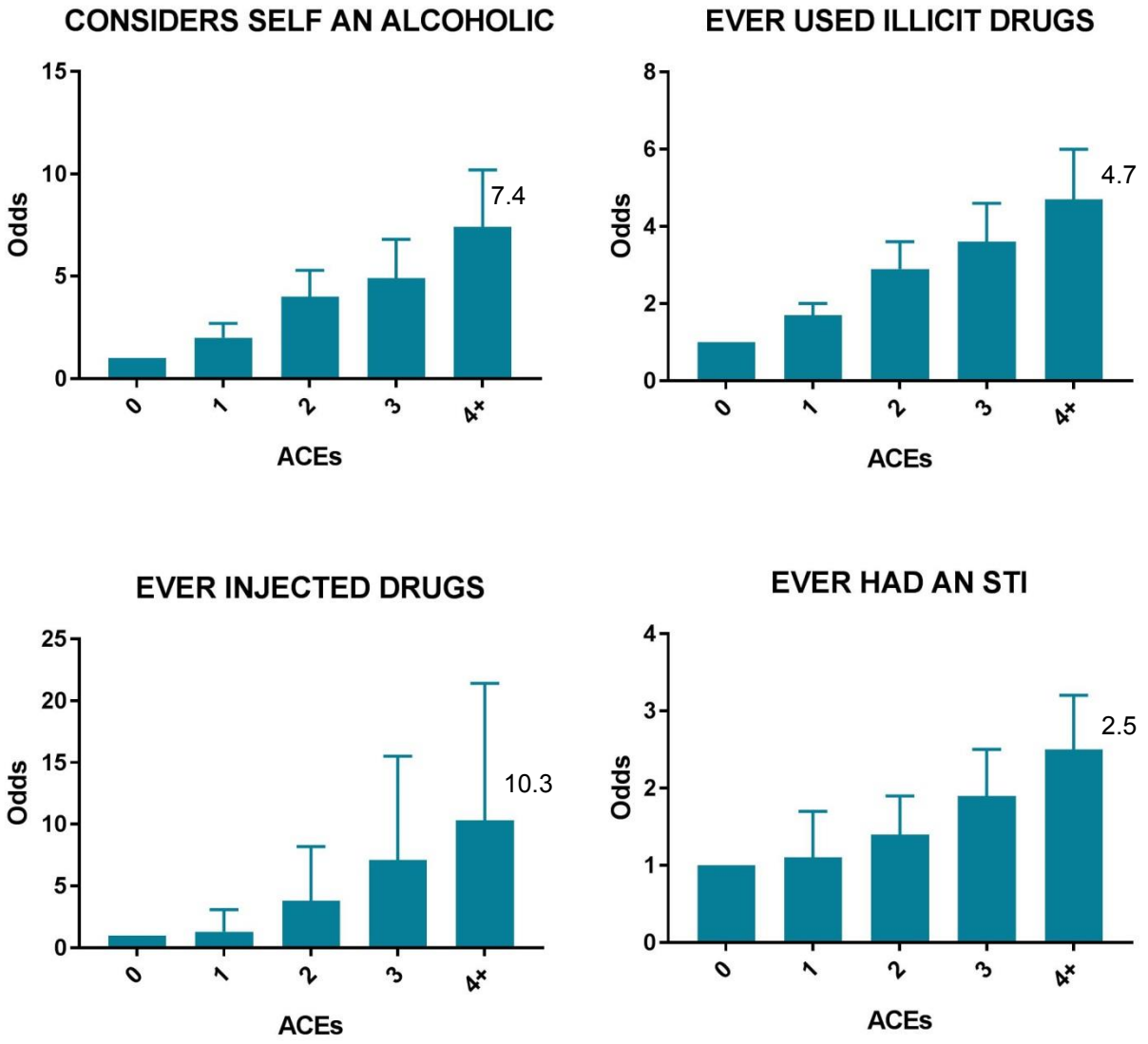
Felitti VJ, et al. Am J Prev Med 1998;14:245–58;

ACEs and Mental Health



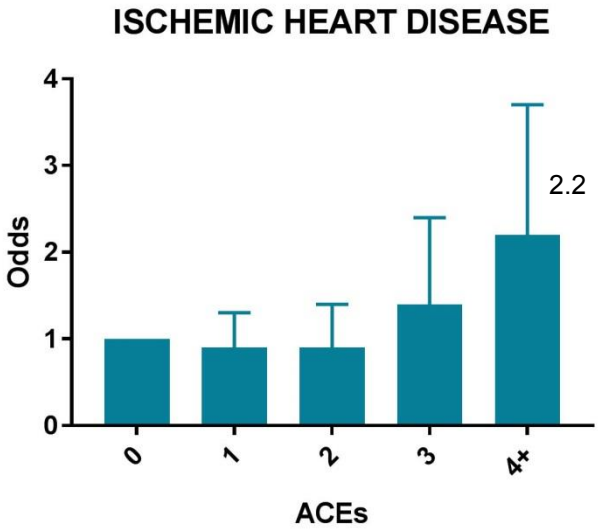
Felitti VJ, et al. Am J Prev Med 1998;14:245–58; Odds ratios adjusted for age, gender, race, and educational attainment.

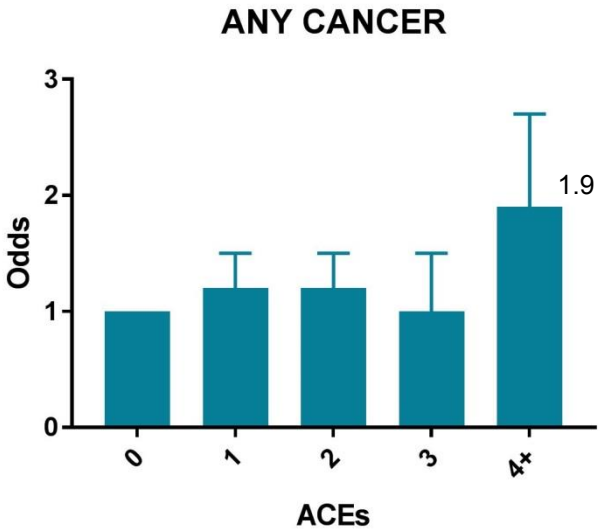
ACEs and High Risk Behaviors

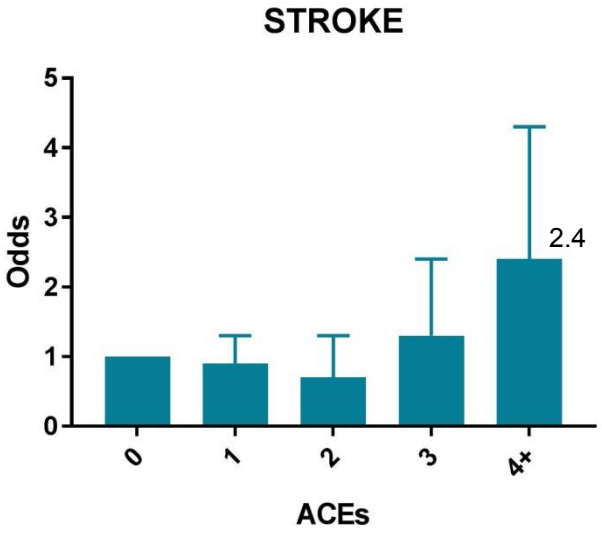


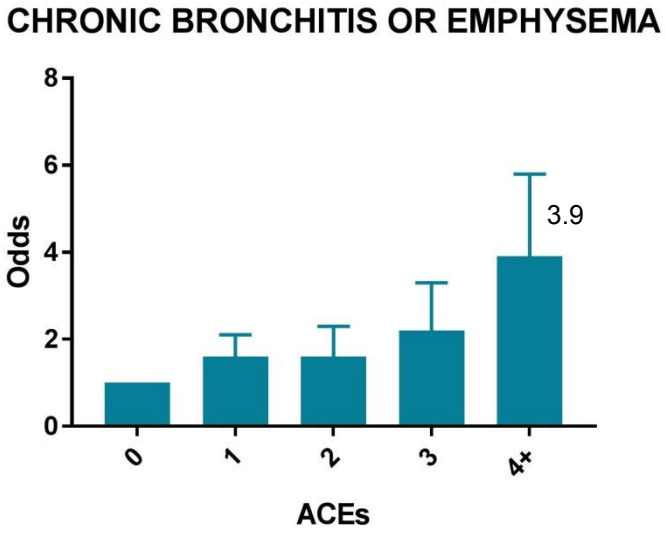
Source: Felitti VJ, et al. Am J Prev Med 1998;14:245–58; Odds ratios adjusted for age, gender, race, and educational attainment.

ACEs and Chronic Diseases









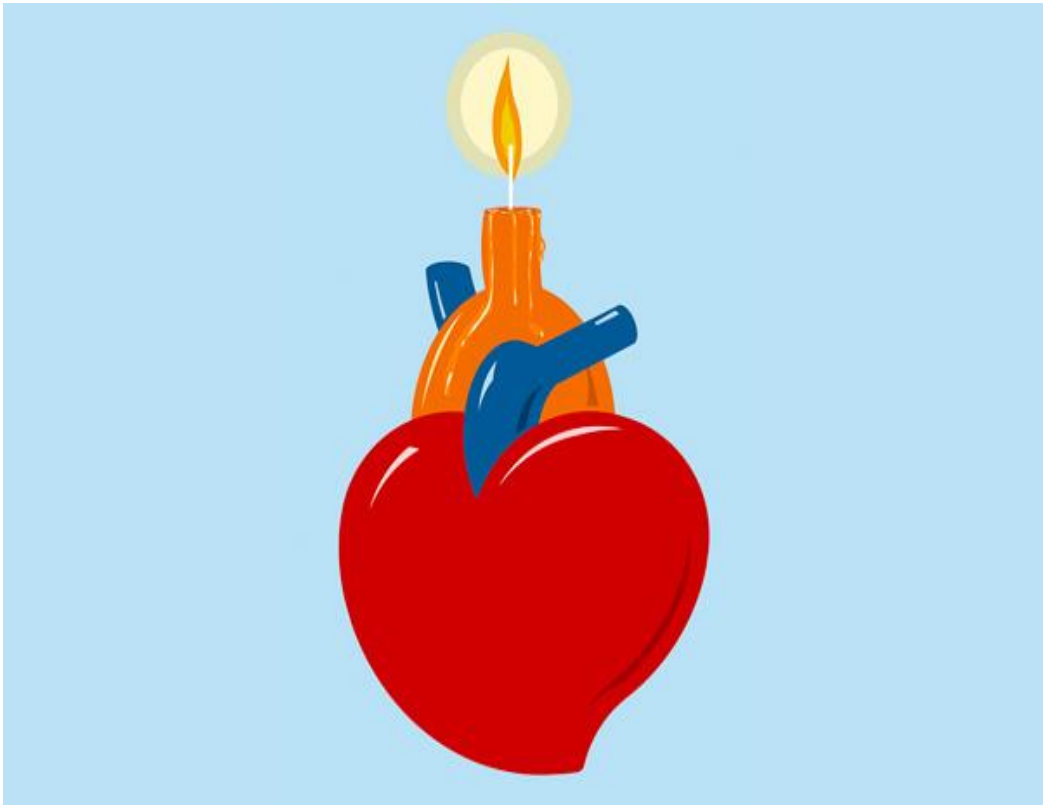
Source: Felitti VJ, et al. Am J Prev Med 1998;14:245–58; Odds ratios adjusted for age, gender, race, and educational attainment.

Where are we now?

Biological Embedding

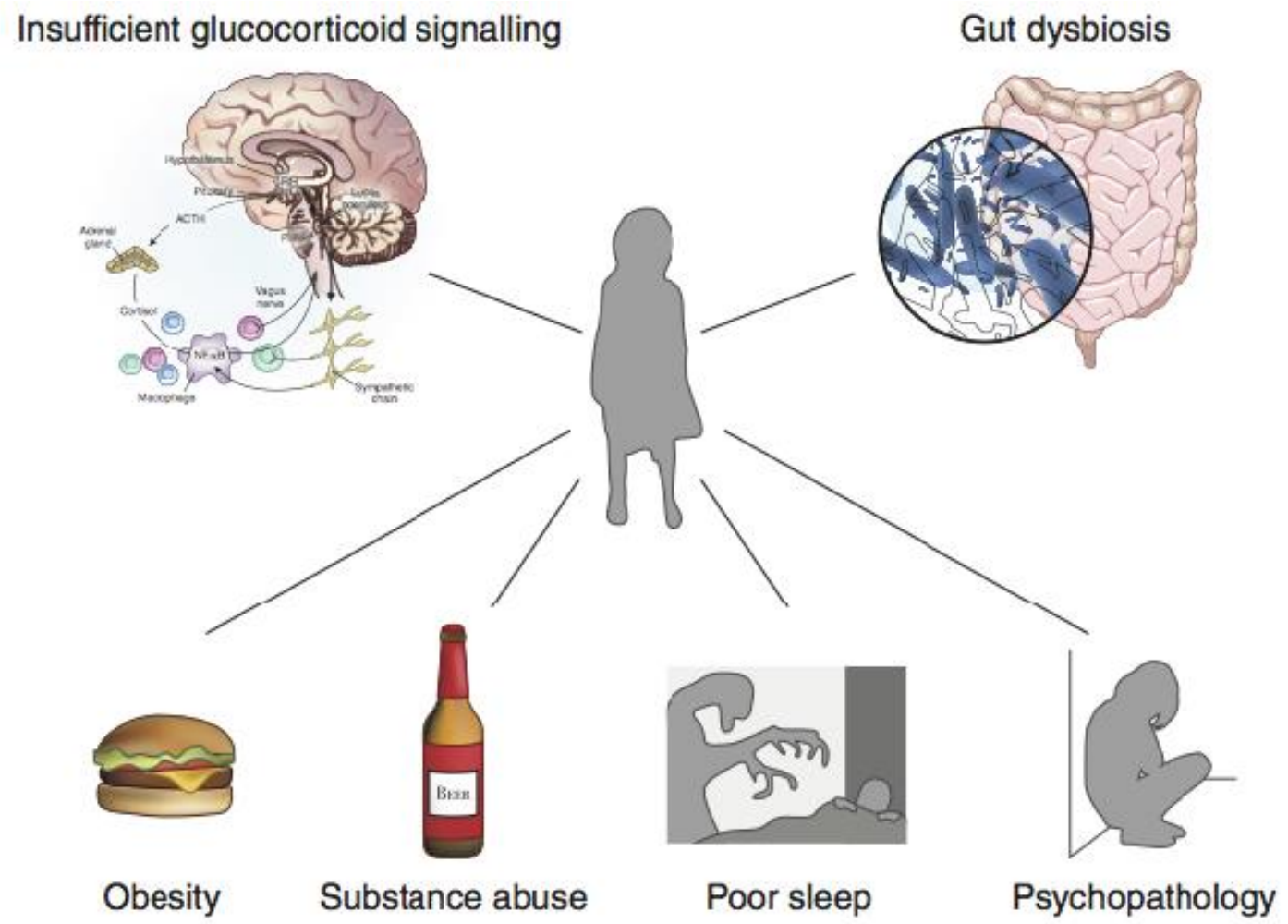
Inflammation

- Part of normal stress response
- Elevated levels adaptive in short term, maladaptive over time
- Physiological mechanism linking trauma to health over the life course?



Danese, A., J Lewis, S., 2017. Neuropsychopharmacology 42:99-114.

Mechanisms linking Trauma to Inflammation



Source: Danese, A., J Lewis, S., 2017. Neuropsychopharmacology 42:99-114.

Trauma-related Inflammatory Dysregulation as early as Childhood

Childhood adversity and inflammatory processes in youth: A prospective study

Natalie Slopen [a,b,c,*], Laura D. Kubzansky [b,1], Katie A. McLaughlin [a,d,e,2], Karestan C. Koenen [f,3]

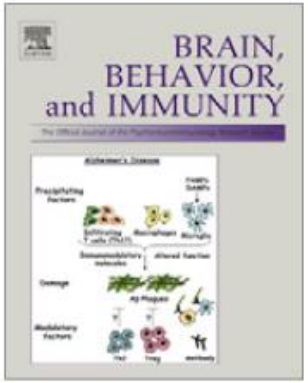
Psychoneuroendocrinology (2013) **38**, 188–200

Review

Childhood adversity and immune and inflammatory biomarkers associated with cardiovascular risk in youth: A systematic review

Natalie Slopen [a,b,c,*], Karestan C. Koenen [a,b,d], Laura D. Kubzansky [b]

Brain, Behavior, and Immunity 26 (2012) 239–250



Objectives

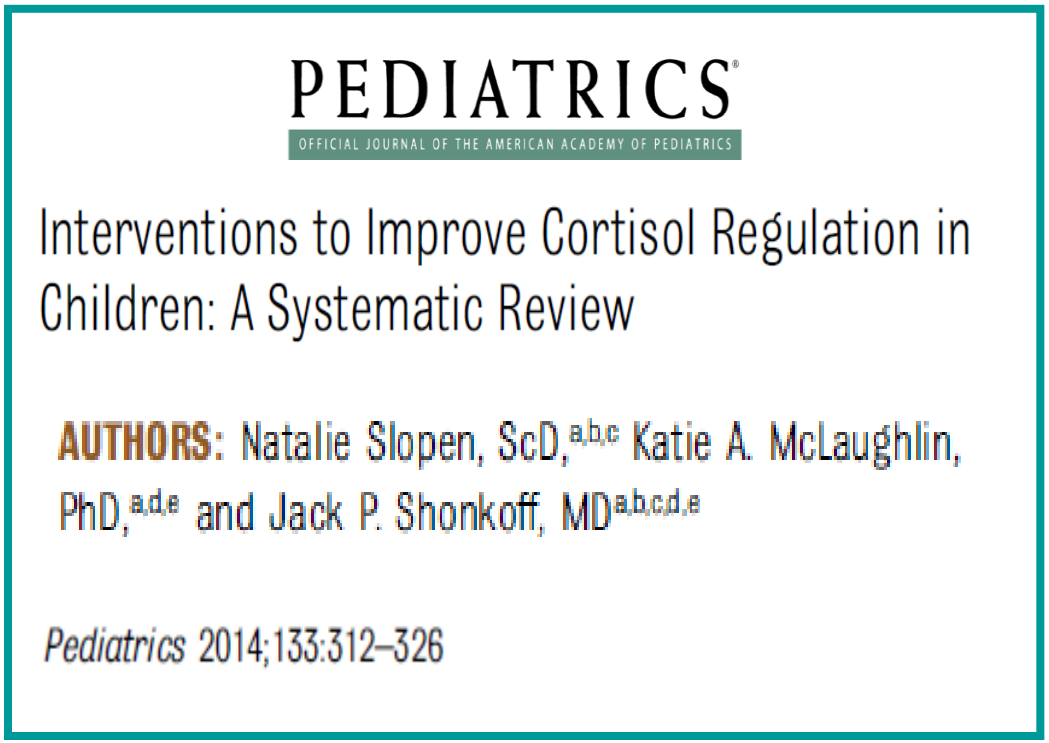
1. Epidemiology of childhood adversity and trauma in the U.S.
2. Childhood trauma and health across the life course.
3. Possibilities for social intervention to improve early life physiological dysregulation.

Interventions to Promote Physiological Improvements in Stress-Response Physiology

Identified 19 articles from 17 randomized or quasi-experimental studies:

1) designed to improve relationships, environments, or psychosocial functioning in children;

2) that examined cortisol as an outcome.


PEDIATRICS

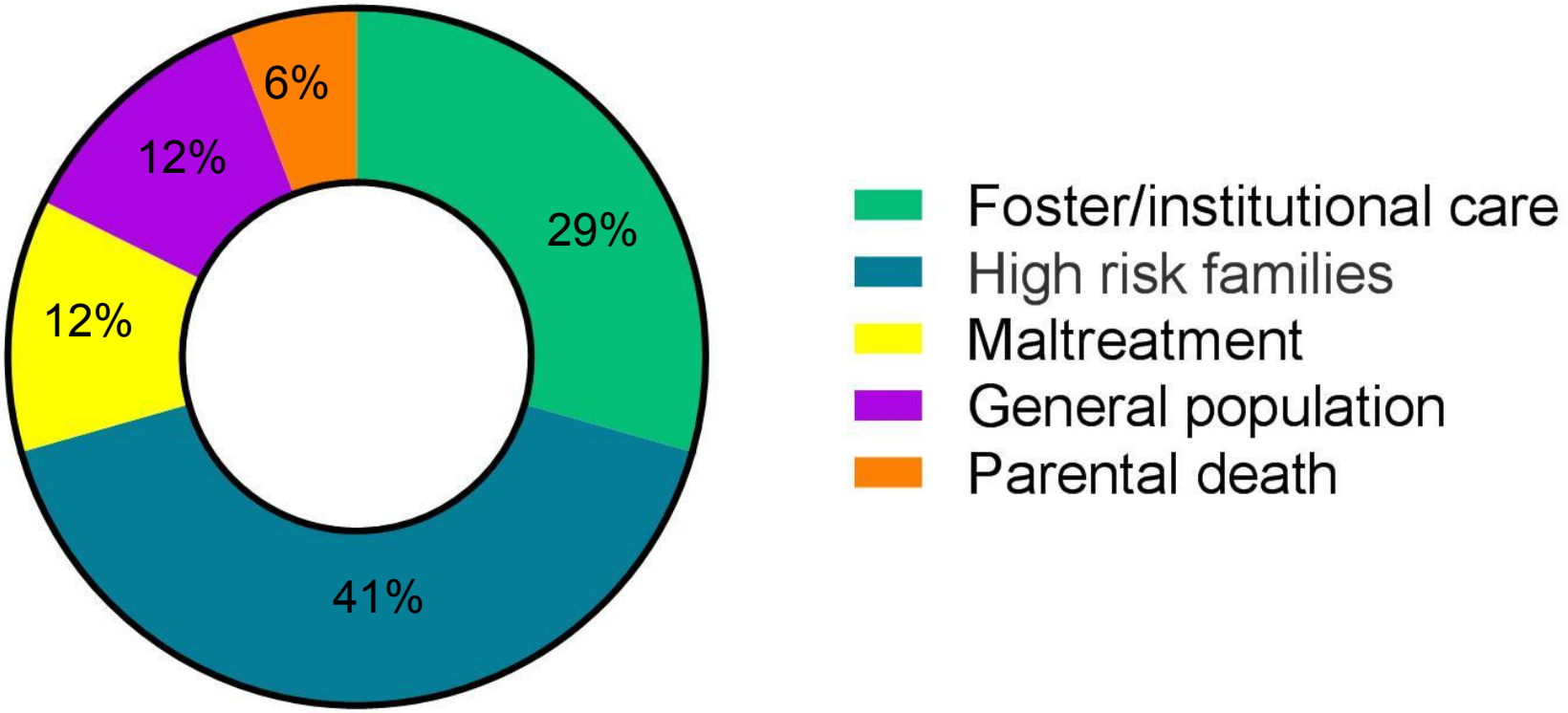
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Interventions to Improve Cortisol Regulation in Children: A Systematic Review

AUTHORS: Natalie Slopen, ScD,[a,b,c] Katie A. McLaughlin, PhD,[a,d,e] and Jack P. Shonkoff, MD[a,b,c,d,e]

Pediatrics 2014;133:312–326

Risk Exposure within Identified Studies

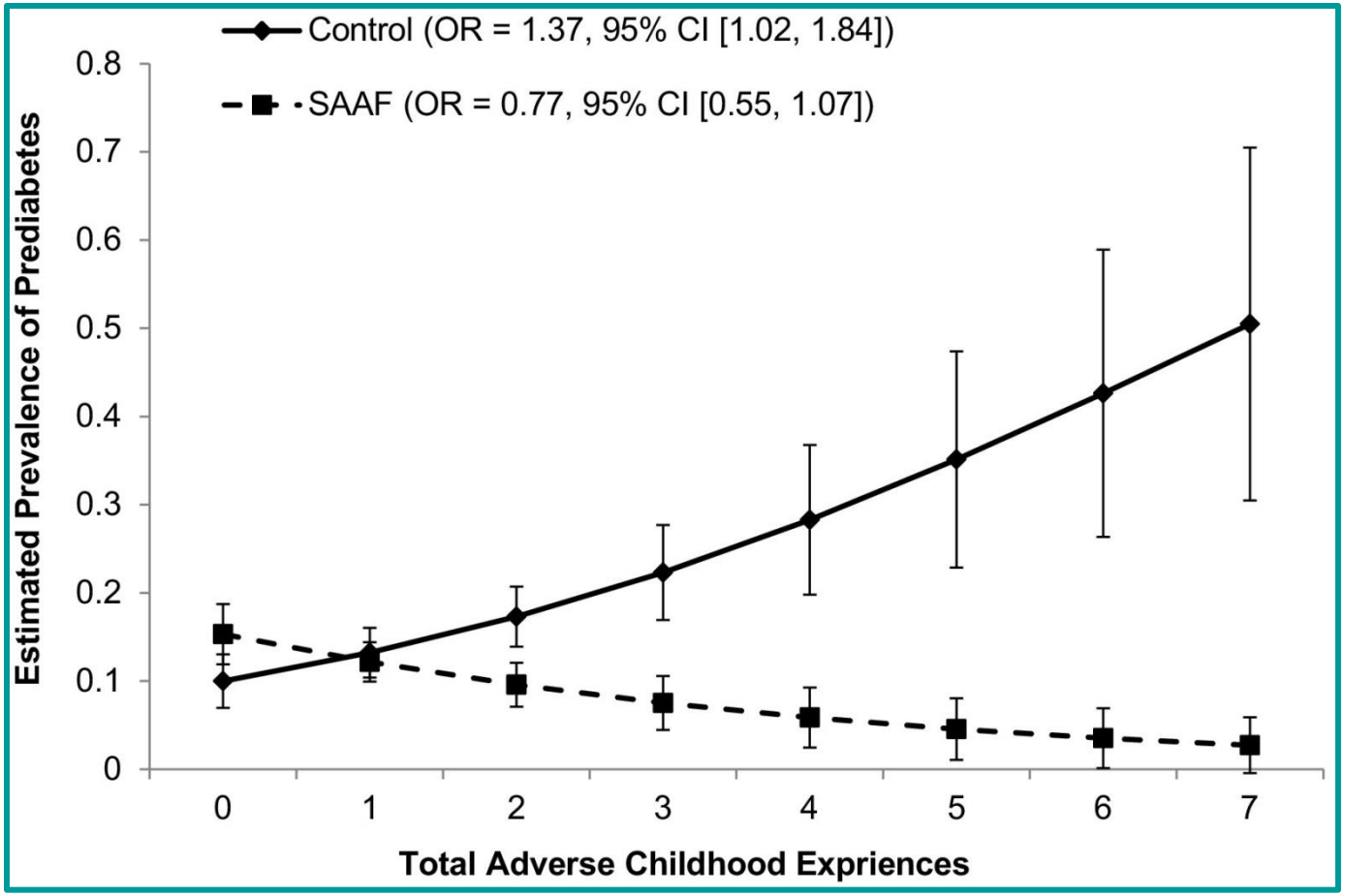


Interventions are Effective

- 18 of 19 studies reported at least 1 difference in cortisol outcome between intervention and control participants
- In all 8 studies that included a low-risk comparison group, post-intervention cortisol in the intervention group approximated low-risk comparison (& differed from usual care).
- Conclusion: cortisol activity can be altered by interventions.
- Promising evidence that it may be possible to repair stress regulatory systems after childhood trauma or other adversity.

Slopen et al, Pediatrics, 133: 312-326 (2014).

Family-centered prevention program ameliorates association between ACEs and prediabetes status in young black adults



Randomized control trial

SAAF = Strong African American Families Intervention program

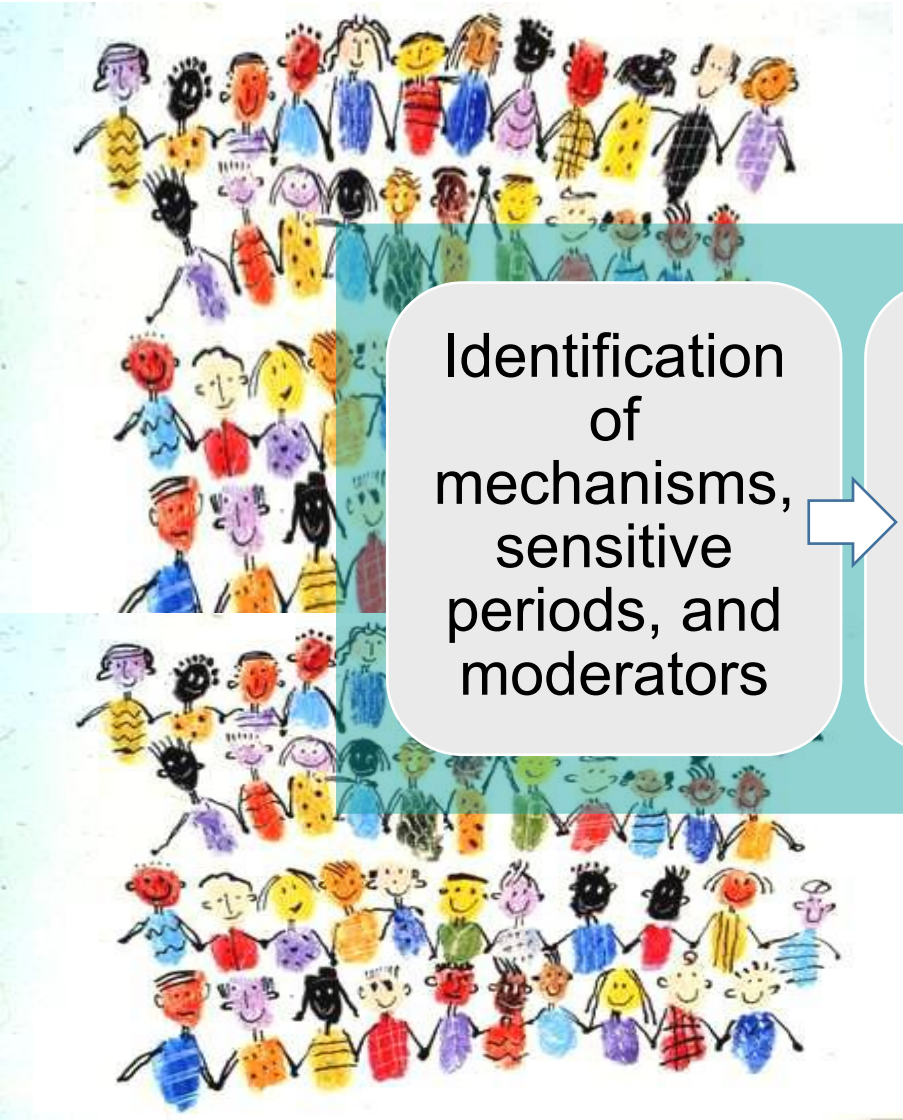
N=390

Rural African American parents and their 11-year old children

Age 25 at follow-up

Brody, G.H., Yu, T., Chen, E., Miller, G.E., 2017. Preventive Medicine 100:117-22.

Future Directions



Identification of mechanisms, sensitive periods, and moderators

Age-appropriate interventions

More effective reduction of disease risk and health disparities.

Contact information:

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E-mail: nslopen@umd.edu

Department of Epidemiology and Biostatistics
University of Maryland, College Park
School of Public Health

Key References

- Brody, G.H., Yu, T., Chen, E., Miller, G.E., 2017. Family-centered prevention ameliorates the association between adverse childhood experiences and prediabetes status in young black adults. Preventive Medicine 100:117-22.
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- Slopen, N., Kubzansky, L.D., McLaughlin, K.A., Koenen, K.C., 2012. Childhood adversity and inflammatory processes in youth: A prospective study. *Psychoneuroendocrinology,* 38, 188-200.
- Slopen, N., McLaughlin, K.A., Shonkoff, J.P., 2014. Interventions to Improve Cortisol Regulation in Children: A Systematic Review. *Pediatrics* 133:312-26.
- Slopen, N., Shonkoff, J.P., Albert, M.A., Yoshikawa, H., Jacobs, A., Stoltz, R., Williams, D.R., 2016. Racial Disparities in Child Adversity in the U.S.: Interactions with Family Immigration History and Income. *Am J Prev Med* 50:47-56.

Psychological Trauma in a Social Context

Susan Cohen Esquilin, Ph.D., ABPP

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Definition

- Trauma: Exposure to death, actual/threatened serious injury, actual/ threatened sexual violation, through direct experience, witnessing event, or having it occur to close relative/friend (must be violent/accidental)—terrifying and overwhelming experience(s) over which the individual has no control or ability to manage
- Multiple possible sources:
  - Intrafamilial
  - Community
  - Natural disasters
  - Accidents
  - Medical procedures
  - War/terrorism

Factors Affecting Impact

- We do not "get used" to trauma. The more events (frequency), the longer they last (chronicity), and the earlier they start (developmental level), the greater the potential damage.
- Interpersonal trauma tends to be more damaging than trauma for other sources.
- Living with chronic stress makes recovery from trauma more difficult.
- The presence of adults and systems that offer support and protection reduces the negative impact of trauma.

Difficulty in Recognizing Impact of Trauma

- Impact of acute trauma looks like anxiety
- Impact of repetitive trauma likely to produce chronic behavioral problems: affect dysregulation, problems with impulse control, problematic self-soothing, flattening of affect, proactive aggression
- These are misinterpreted diagnostically: ADHD, oppositional defiant disorder, conduct disorder, bipolar disorder, antisocial personality disorder
- The central question needs to be: “what happened to this person?” and not, “what is wrong with this person?”

Trauma and Oppression

- Youth from non-dominant/ oppressed groups: increased risk of interpersonal trauma exposure
- Individuals from oppressed groups: higher rates of PTSD in response to similar traumatic events
- Historical trauma: intergenerational traumatic effects
- Chronic non-traumatic stress makes healing from trauma more difficult

Continuous Traumatic Stress (CTS)

- CTS: on-going traumatic experiences that the client is still in the midst of – no safe haven
  - "CTS brings the political and sociological elements of institutionalized racism to the discussion of the effects of living in the midst of trauma."
  - There exist "areas where historical racial trauma interacts with ongoing high levels of violent crime…where there is extremely high, ongoing exposure to violence through crime and social disorganization…"
  - Individuals in these areas may present "a different diagnostic picture for trauma responses, one that is frequently far less sympathetic than the diagnostic picture that gives primary to anxiety. Anger and hostility may be the most prominent feature…"

(Roach, 2013)

Intergenerational (Historical) Trauma

- Responses to trauma experienced by past generations that, through parental behavior impact current generation, even in absence of direct traumatic experience
- Described in descendants of Holocaust survivors
- Directly relevant to descendants of enslaved Africans

(Leary, 2005)

Chronic Stress

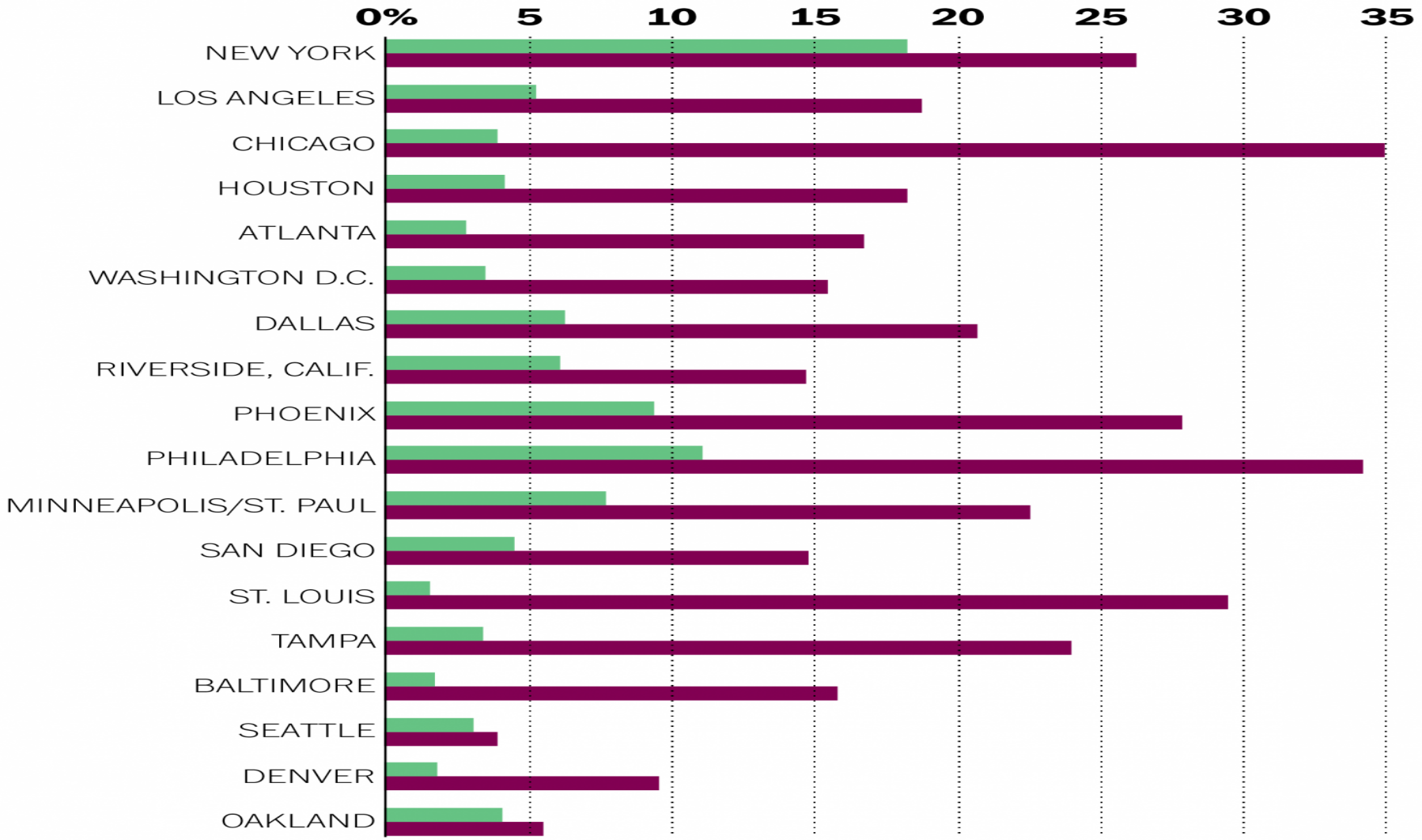
- Stress: events for which we do not have ready made coping strategies; does not necessarily involve immediate threats to physical integrity
- Can result in growth if situation is conquered
- Chronic stress (situation cannot be mastered) creates pressure, and has long-term negative physical and psychological consequences
- Not all stress is traumatic, but chronic stress makes healing from traumatic stress much harder
- Sources of chronic stress: medical problems, poverty, oppression, undocumented immigration status, exposure to family substance abuse and other family dysfunction

Concentrated Poverty

- Areas in which 30% of the population or more are living in poverty (2017 federal poverty line for family of 2 adults and 2 children = $24,600)
- Consequences: fewer resources; higher rates of victimization; higher levels of other sources of trauma (fires, vehicular accidents)
- While there are more poor White people in the US, Black people experience higher rates of poverty generally, and are more affected by concentrated poverty than Whites. Pattern of racial segregation in housing has legal history and is entrenched in American society.

Racial disparities in concentrated poverty

Percentage of poor **blacks** and **whites** living in concentrated poverty, by metro area



WAPO.ST/**WONKBLOG**

Source: The Century Foundation analysis of 2009-2013 American Community Survey data

Racism as a Source of Stress

- Sources of stress:
  - Discrimination, profiling
  - Negative stereotyping/microaggressions: emotional abuse: https://www.youtube.com/watch?v=YWyI77Yh1Gg
  - Undocumented immigration status
- Social and economic marginalization, deprivation, and powerlessness create barriers to service

Hidden Wounds of "Racial Trauma"

- Internalized devaluation: become hypervigilant about gaining respect
- Assaulted sense of self: difficult to know who you really are/ identity formation is developmental task of adolescence
- Internalized voicelessness: wounds are experienced systemically, impairing ability to advocate for self, because youth will appear threatening

=Wound of Rage

(Hardy, 2013)

Resiliency

- People in communities that has long histories of oppression often have developed strengths and adaptive coping skills that can appear problematic to professionals looking at the communities from the outside.
- It is critical that any attempts to address systemic sources of trauma involve members of the communities affected and that professionals hold themselves accountable to those communities.

Summary

- Trauma, stress, and chronic stress all impact poor people and People of Color to greater degrees than they do economically comfortable people and White people
- We must focus on:
  - Decreasing the sources of trauma and stress in poor Communities of Color; this involves increasing the awareness of all people about the role race plays in the incidence of trauma and stress, particularly in those seeking to help
  - The ways in which systems that are supposed to be helpful often create further problems; gatekeepers need to become aware of how they maintain systems that produce racially disparate results
  - In the context of psychological treatment, we must focus both on the experience of race as a core issue for Black people and People of Color, and the lack of attention to race in the treatment and professional training of White people

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Rutgers Center for Law, Inequality and Metropolitan Equity (CLiME)
Trauma, Schools and Poverty Project

A Critical Review of the Psychological Literature

By Alexandra K. Margevich, M.A.

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Abstract

This report provides a critical and comprehensive review of the empirical literature on the sequelae of childhood exposure to potentially traumatic events (PTEs), with special emphasis on low socioeconomic status (SES) populations at disparate risk for exposure to PTEs across the lifespan. First, I will outline the categories and characteristics of childhood PTEs. Second, I will synthesize research on the proximal and distal consequences of childhood PTE exposure. Third, I will identify significant mediators (i.e., how or why PTE-related outcomes occur) and moderators (i.e., when or for whom PTE-related outcomes hold) of children's responses to trauma. Finally, I will provide recommendations for building resilience in children and their social environments.

Keywords: children, potentially traumatic events, trauma, poverty, socioeconomic status, risk, resiliency, education

Contents

A Critical Review of the Psychological Literature

By Alexandra K. Margevich, M.A.

Early life adversity is a pervasive problem in the United States—the majority of the population will be exposed to at least one potentially traumatic event before 18 years of age (e.g., Felitti et al., 1998). Early exposure to potentially traumatic events can interrupt typical developmental pathways and elevate risk for problems with health and functioning in the immediate and distant aftermath of trauma exposure (Heim & Nemeroff, 2001; Heim, Shugart, Craighead, & Nemeroff, 2010; Nemeroff, 2004). Although childhood trauma exposure is a nationwide public health issue, children living in low socioeconomic status (SES), high-crime areas are at disparate risk for developing harmful trauma-related outcomes (e.g., physical, psychological, cognitive) due to the increased sources and frequency of potentially traumatic events (Taylor, Way, & Seeman, 2011). Consistent with research on allostatic load (McEwen & Stellar, 1993), the combination of chronic stressors such as economic hardship (e.g., food insecurity), community violence (e.g., gang violence, property crimes), and social discrimination (e.g., racial, class-based) take a cumulative toll on children's health and functioning (e.g., Body, Yu, & Beach, 2016). Thus, attention should be directed towards addressing this issue in U.S. cities at disparate risk for the sequelae of chronic childhood trauma exposure, like Newark, New Jersey, where approximately 30% of residents currently live in poverty and crime rates are among the highest in the nation (Federal Bureau of Investigation, 2015; U.S. Census Bureau, 2015).

Unfortunately, the observable effects of trauma exposure that present during childhood and adolescence tend either to go unnoticed or to be misattributed to non-trauma-related sources (e.g., juvenile delinquency) by those individuals who are optimally positioned to intervene, including family members, school personnel, and child welfare workers (Aisenberg & Mennen, 2000). For example, adults may overlook external sources of deviant behavior when a child's traumatic response is stereotype-consistent (e.g., an aggressive boy; Burgoon, 2015). This is a major concern because traumatized children in high-risk communities who "fly under the radar" are unlikely to independently seek help, partly due to health service inaccessibility and mental illness stigma (Aisenberg & Mennen, 2000; Pynoos et al., 2008). Moreover, society is more likely to filter traumatized individuals from cities like Newark, New Jersey into the criminal justice system than the mental health system (Sullivan, 2016; Torrey, Kennard, Eslinger, Lamb, & Pavle, 2010). As a result, these children will continue to experience decrements in health and functioning throughout their life course, making it virtually impossible for them to break the cycle of poverty.

Considering the deleterious effects of childhood trauma exposure, which may disproportionately impact children living in poverty, this review paper seeks to unpack the causes and consequences of this major public health issue through a systematic review of the trauma and victimization literatures. After briefly describing the literature search process, I will first outline the categories and characteristics of potentially traumatic events in childhood. Second, I will synthesize research on the proximal and distal consequences of childhood trauma exposure. Third, I will identify significant mediators (i.e., how or why trauma-related outcomes occur; Baron & Kenny, 1986) and moderators (i.e., when or for whom trauma-related outcomes hold; Baron & Kenny, 1986) of children's responses to potentially traumatic events. Finally, I will provide recommendations for a community-based approach to trauma prevention, with an emphasis on building resiliency.

Systematic Literature Search

In order to identify methodologically and statistically rigorous research on childhood trauma exposure and poverty in the United States, I conducted a systematic literature search using the PsycINFO electronic database. In general, empirical articles were selected based on their adherence to the following a priori inclusion criteria: (1) used quantitative research methodology; (2) included a nationally representative U.S. sample; (3) achieved sufficient sample size to power statistical analyses; and (4) measured multiple types of potentially traumatic events. Several exceptions were made regarding sample size and representativeness criteria when research reports were specific to child or adolescent, ethnic-racial minority, and/or low SES samples, given their primary relevance to the current research aims. To this end, PsycINFO search parameters were set to identify articles published in a peer-reviewed journal containing any possible combinations of words with the prefix *trauma* (e.g., trauma, traumatic,

traumatize) and words with the prefix *child* (e.g., child, childhood, children). Poverty (or a related term) was intentionally excluded from the search in order to increase comprehensiveness. This initial search yielded approximately 4,500 potentially relevant articles.

I next eliminated any articles that clearly failed to meet my a priori inclusion criteria based on titles and abstracts, and read approximately 50 literature reviews in full to identify additional relevant articles from their in-text citations. The reduced set of approximately 300 empirical articles were then read, evaluated, and catalogued. Additional articles were eliminated based on inclusion criteria during this phase, resulting in a final database of approximately 200 research reports of varying relevance and quality, only a subset of which I will reference here. As a caveat, the majority of studies included in this paper applied a retrospective, cross-sectional approach using an adult sample, with far fewer studies applying a prospective, longitudinal approach using a child sample.

Categories and Characteristics of Childhood Potentially Traumatic Events

A primary goal of the above literature search was to identify consensually accepted categories and characteristics of childhood potentially traumatic events from the literature. Understanding the scope of events that may be potentially traumatic for a developing child is essential for understanding the magnitude of the problem. For example, using data from the National Survey of Children's Exposure to Violence, Finkelhor, Turner, Shattuck, and Hamby (2015) investigated the past year and lifetime prevalence of exposure to broad categories of traumatic events (or victimizations) in childhood. Among their many findings, their analyses revealed the following lifetime prevalence rates: 51.4% any physical assault (e.g., assault with vs. without a weapon, assault by nonsibling peer) 8.4% any sexual offense (e.g., attempted or completed rape, sexual assault by adult stranger), 24.9% any maltreatment (i.e., physical, emotional and sexual abuse; neglect), 41.3% any property crime (e.g., robbery by nonsibling, vandalized by nonsibling), and 38.3% any witnessed violence (e.g., any witnessed family assault, witnessed assault in community). In an earlier analysis of this dataset, Finkelhor, Ormrod, and Turner (2007) found that 71% of children experienced at least one victimization in their lifetime. This is consistent with data culled from adult samples, namely the Adverse Childhood Experiences Study, which tends to reveal that between two-thirds and three-fourths of the population are likely to experience at least one traumatic event in their lifetime (e.g., Dube et al., 2001; Dube et al., 2009; Felitti et al., 1998).

With these disturbing statistics in mind, I created the below taxonomy by pooling variables included in several widely-used childhood trauma screening and diagnostic tools. Looking at multiple widely-used instruments is advantageous because they noticeably differ in the scope of potentially traumatic events that they measure. I will continue to refer to the sources of trauma as *potentially* traumatic events (PTEs) throughout this paper because there is considerable variability in which events children subjectively experience as traumatic (e.g., Bonanno, 2004; Bonanno & Macini, 2008).

Categories of Childhood PTEs

In the trauma and victimization literatures, childhood PTEs are typically assessed through developmentally appropriate structured diagnostic interviews (e.g., using DSM traumatic stressor criterion) and trauma history questionnaires, including: the Childhood Trauma Questionnaire (Bernstein & Fink, 1998), the Juvenile Victimization Questionnaire (Finkelhor, Hamby, Ormrod, & Turner, 2005), the Conflict Tactics Scale (Straus, 1979), the Past/Recent Exposure to Violence Scales (Singer, Anglin, Song, & Lunghofer, 1995), and the Adverse Childhood Experiences (ACE) Study questionnaire (e.g., Felitti et al., 1998). Across these instruments, the most commonly measured PTEs in the U.S.-based literature are childhood abuse (emotional, physical, sexual) and neglect (emotional, physical), which are often collectively referred to as childhood maltreatment. Other instruments include additional measures of violence exposure in the home (e.g., household dysfunction including witnessing domestic violence, parental marital discord, living with substance abusing, mentally ill, or criminal household members; Dong, Anda, Felitti, Dube, Williamson, Thompson, Loo, & Giles, 2004) and in the community (e.g., Richters & Saltzman, 1990). Mass-exposure PTEs (e.g., war, terrorism, disasters; Chrisman & Dougherty, 2014) are noticeably less prevalent in the childhood trauma literature conducted in developed nations.

Importantly, these different types of PTEs have been further characterized in the literature using one or more of the following

distinctions: (a) individual-level versus community-level; (b) interpersonal versus non-interpersonal; (c) acute versus chronic; (d) one type versus multiple types; and (e) direct versus indirect. These distinctions are important to understand when conceptualizing PTEs because, described later, different characteristics of childhood PTEs in combination with biopsychosocial risk factors (e.g., genetics, poverty) confer differential risk for negative trauma-related outcomes. Additionally, these distinctions will help explain how various terms for specific types of PTEs in the literature overlap, which will aid in conceptual clarity.

Distinctions Between Types of Childhood PTEs

In the literature, researchers may distinguish between individual-level and neighborhood- or community-level PTEs. Individual-level PTEs are typically those experienced within the child's most immediate social contexts (e.g., family violence), others may arise from the child's broader social context (e.g., community/neighborhood violence; Aisenberg & Mennen, 2000). In contrast to individual-level PTEs, community-level PTEs encompass exposure risk based on different base rates of community violence owing largely to systemic factors that maintain social inequalities. Thus, people living in middle- and high-crime areas are statistically more likely to be exposed to community violence than people living in low-crime areas (Aisenberg & Mennen, 2000).

Researchers may also differentiate among PTEs based on whether they result from other people's actions against the child (i.e., interpersonal). Interpersonal PTEs include face-to-face violations such as childhood abuse and neglect, and vary based on the child's relation with the perpetrator (Alisic, Zalta, van Wesel, Larsen, Hafstad, Hassanpour, & Smid, 2014). For example, betrayal trauma refers to an interpersonal PTE that occurs within the context of a close, trusting relationship between the perpetrator and child (Freyd, 1996). By comparison, non-interpersonal PTEs include events such as natural disasters, injuries due to accident, life-threatening disease, and the loss of a loved one. In the latter case, a child may experience childhood traumatic grief (also loss or bereavement) if PTE-related symptoms infringe on the child's ability to grieve (Cohen, Mannarino, Greenberg, Padlo, & Shipley, 2002).

Additionally, researchers have distinguished among firsthand (i.e., direct) and secondhand (i.e., indirect) PTE exposure. For example, whereas childhood abuse qualifies as a direct PTE, witnessing or hearing about another person's PTE exposure qualifies as an indirect traumatic experience. One specific type of PTE, called co-victimization, refers to personally witnessing a violent interpersonal assault (Shakoor & Chalmers, 1991). Moreover, indirect PTEs may be witnessed in the presence of close others (i.e., co-witnessed), such as the child's parent, which has implications for the child's coping resources (Linares, Heeren, Bronfman, Zuckerman, Augustyn, & Tronick, 2001). Additionally, the concept of indirect trauma is related to but distinct from the concept of secondary (or vicarious) trauma, which refers to the transfer of trauma *symptoms* to a person as a result of their close relation with someone who has been traumatized (Motta, 2012). Sadly, this means that even if children are not themselves exposed to PTEs, they may still develop trauma symptoms if their parents, siblings or peers are deeply affected by PTE exposure.

Additionally, researchers often simultaneously distinguish between PTEs based on their (a) frequency and (b) diversity. That is, people may experience a single-incident (i.e., acute) PTE or repeated and prolonged (i.e., chronic) PTEs, and either type of exposure might occur in the context of a single type of PTE or multiple types of PTEs. Other terms have been generated to underscore the particular importance of chronic exposure to multiple PTEs, including poly-victimization, which refers to experiencing multiple types of interpersonal PTEs in childhood (Finkelhor et al., 2005), and complex trauma exposure (see below), which refers to chronic exposure to different types of childhood maltreatment (Cook, Blaustein, Spinazzola, & van der Kolk, 2005). Chronic or complex and acute PTEs map onto Terr's (1991) distinction between Type I and Type II traumas, respectively.

Complex Trauma

Not surprisingly, it is consensually believed (and empirically validated) that there is a dose-response relation between amount and severity of trauma exposure and likelihood of developing negative PTE-related outcomes (e.g., Felitti et al., 1998). As such, much attention has been paid to the construct of complex trauma, which in its broadest conceptualization encompasses both exposure to complex

traumatic events and outcomes resulting from such exposures (Cook et al., 2005; Kliethermes, Schacht, & Drewry, 2014). Unfortunately, Kliethermes, Schacht, and Drewry (2014) noted in their review of the complex trauma literature that "the lack of consensus on a definition of complex trauma has posed challenges for researchers because definitions can have varying emphasis placed on the number of traumatic events, the types of traumatic events, the developmental periods in which they occur, or the resulting symptom profile" (p. 340). These varying definitions in turn make it difficult to reach a consensus on the prevalence of complex trauma. Thus, in order to effectively assess and prevent complex trauma, it is essential that research be guided by comprehensive frameworks that capture the two elements of complex trauma: exposure and outcomes.

Cook and colleagues (2005) from the National Child Traumatic Stress Network's Complex Trauma Task Force proposed the following framework for understanding complex trauma:

"The term complex trauma describes the dual problem of children's exposure to traumatic events and the impact of this exposure on immediate and long-term outcomes. Complex traumatic exposure refers to children's experiences of multiple traumatic events that occur within the caregiving system – the social environment that is supposed to be the source of safety and stability in a child's life. Typically, complex trauma exposure refers to the simultaneous or sequential occurrences of child maltreatment—including emotional abuse and neglect, sexual abuse, physical abuse, and witnessing domestic violence—that are chronic and begin in early childhood. Moreover, the initial traumatic experiences (e.g., parental neglect and emotional abuse) and the resulting emotional dysregulation, loss of a safe base, loss of direction, and inability to detect or respond to danger cues, often lead to subsequent trauma exposure (e.g., physical and sexual abuse, or community violence). Complex trauma outcomes refer to the range of clinical symptomatology that appears after such exposures. Exposure to traumatic stress in early life is associated with enduring sequelae that not only incorporate, but also extend beyond, Posttraumatic Stress Disorder (PTSD). These sequelae span multiple domains of impairment and include: (a) self-regulatory, attachment, anxiety, and affective disorders in infancy and childhood; (b) addictions, aggression, social helplessness and eating disorders; (c) dissociative, somataform, cardiovascular, metabolic, and immune-logical disorders; (d) sexual disorders in adolescence and adulthood; and (e) revictimization". (p. 5)

Research on prevalence of complex trauma exposure as previously defined suggests that nearly a quarter of children (22%) are exposed to complex trauma in a one-year period (four or more victimizations; Finkelhor et al., 2007). Moreover, different populations are at heightened risk for complex trauma exposure, and by extension, complex trauma outcomes. For example, children with high levels of complex trauma exposure (7 or more victimization experiences) were significantly more likely to be Black and low SES. I will return to these sociodemographic disparities later in the paper. Next, I will describe the proximal and distal consequences of childhood PTE exposure.

Proximal and Distal Consequences of Childhood PTEs

Exposure to childhood PTEs has the potential to directly and indirectly affect nearly every facet of a child's current and future existence. Across the trauma and victimization literatures, childhood PTE exposure has been linked to numerous negative life outcomes across the lifecycle. As previously mentioned, most extant rigorous research on the impact of childhood PTE exposure comes from adult samples (18 and older).

Findings in Adult Samples

Perhaps the strongest evidence for the long-lasting consequences of childhood PTE exposure comes from research using data from the Adverse Childhood Experiences (ACE) Survey[1] (Felitti et al., 1998), a study of 17,337 adult health maintenance organization members, and other large nationally representative databases. Collectively, these studies have provided robust support for the role of PTE exposure in the risk of compromised life trajectories. That is, higher cumulative ACE scores predict increased risk for substance use and dependence, posttraumatic stress symptoms, psychiatric illness, suicidal behavior, risky sexual behavior, STDS and adolescent pregnancy, chronic illness, premature death, and homelessness (e.g., Felitti et al., 1998).

Mental health. Research on the relation between childhood PTE exposure and mental health problems has predominately focused

on psychiatric *symptoms* (e.g., depression, posttraumatic stress, anxiety) in the presence or absence of clinical *diagnosis*. Mental health outcomes are indeed most commonly associated with the concept of psychological trauma, which may be defined as "a stress that overwhelms one's ability to cope and reassert equilibrium" (p. 244; Stoddard Jr., 2014).

As a caveat, is important to keep in mind the distinction between the expression of symptoms common in psychiatric disorders like posttraumatic stress disorder (PTSD), such as posttraumatic stress and dissociation, and a clinically significant psychiatric diagnosis of PTSD. For example, to receive a clinical diagnosis of posttraumatic stress disorder (PTSD) based on the American Psychiatric Association's (2013) most recent iteration of their *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*), an individual must meet the following minimum criteria: (Criterion A) at least one exposure (direct or indirect) to a traumatic stressor (i.e., actual or threatened death, serious injury, or sexual violence) ; (Criterion B) at least one symptom of re-experiencing/intrusions (e.g., intrusive thoughts); (Criterion C) at least one symptom of avoidance (e.g., avoiding thinking about the trauma); (Criterion D) at least one symptom of depressed cognitions and mood (e.g., negative affect); (Criterion E) at least two symptoms of hyperarousal/hyper-reactivity (e.g., sleep problems); (Criterion F) symptoms B through E persist longer than one month; (Criterion G) symptoms lead to impaired functioning; and (Criterion H) symptoms are not the result of another cause like medication. Importantly, many people who experience a traumatic event may go on to experience subclinical symptoms without necessarily "qualifying" for a PTSD diagnosis.

In line with the above, considerable research suggests that cumulative childhood PTEs are significantly related to increased risk of both psychiatric symptoms and diagnoses (Anda et al., 2002, Chapman et al., 2004; Dube et al., 2003b; Felitti et al., 1998; Kessler, Davis, & Kendler, 1998; Whitfield, Dube, Felitti, & Anda, 2005). For example, people reporting four or more ACEs (vs. none) were at a 4.6-fold increased odds of experiencing a depressed mood in the past year and a 12.2-fold increased risk of ever attempting suicide (Felitti et al., 1998). Strikingly, Dube et al. (2001) found that people reporting seven or more ACEs (vs. none) were at a 17.7-fold increased risk of ever attempting suicide, even after controlling for self-reported alcoholism, depressed affect, and illicit drug use. Additionally, approximately two-thirds of the risk for lifetime suicide attempts was attributable to childhood PTE exposure (Dube et al., 2001). Finally, research using data from the Collaborate Psychiatric Epidemiology Surveys demonstrated that assaultive/interpersonal violence and childhood maltreatment predicted an approximate 2- and 4-fold increased risk of suicidal ideation in adulthood, respectively (Beristianos, Maguen, Neylan, & Byers, 2016).

Retrospective research using adult samples has also revealed a strong dose-response relation between cumulative ACE scores and increased risk of smoking, alcohol use, and illicit drug use (Anda et al., 1999; 2002; Dube, Cook, & Edwards, 2010; Dube et al., 2003a; Ford et al., 2011). Indeed, people reporting five or more ACEs (vs. none) were at a 2- to 5-fold increased risk of early smoking initiation, ever smoking, current smoking, and heavy smoking (Anda et al., 1999). A similar relation has been found between cumulative ACEs and adult alcohol problems—people reporting four or more ACEs (vs. none) had a 2- to 4-fold increased risk of early alcohol use initiation (before age 14), ever drinking, ever *heavy* drinking, self-reported alcohol problems, personal alcoholism, and marrying an alcoholic (Anda et al., 2002; Dube, Anda, Felitti, Edwards, & Croft, 2002; Dube et al., 2003b; Dube et al., 2006; Felitti et al., 1998).

Physical health. Both individual and cumulative ACEs have been linked to a number of risky health behaviors and history of physical illness. In one of the first published ACE reports, Felitti et al. (1998) demonstrated a strong dose-response relation between cumulative ACEs and physical health—people reporting four or more ACEs (vs. none) had higher odds of having risk factors for leading causes of death (i.e., smoking, severe obesity, physical inactivity, depressed mood, suicide attempt, alcoholism, any drug use, injected drug use) and a 2.2-fold increased odds of having a history of ischemic heart disease, a 1.9-fold increased odds of any cancer, a 2.4-fold increased odds of stroke, a 3.9-fold increased odds of chronic bronchitis or emphysema, a 1.6-fold increased odds of diabetes, a 1.6-fold increased odds of skeletal fractures, a 2.4-fold increased odds of hepatitis or jaundice, and a 2.2-fold increased odds of fair or poor self-rated health (Felitti et al., 1998). For example, Felitti et al. (1998) demonstrated that people reporting four or more ACE events (vs. none) were at a 7.4-fold increased odds of considering self an

alcoholic, a 4.7-fold increased odds of ever using illicit drugs, a 10.3-fold increased odds of ever injecting drugs, a 2.2-fold increased odds of being a current smoker, a 1.3-fold increased odds of physical inactivity, and a 1.6-fold increased odds of severe obesity (BMI greater than or equal to 35; cf. Dube et al., 2010). Moreover, people reporting six or more ACE events (vs. none) were at an increased risk of death from all causes at any age and died nearly 20 years earlier on average (Brown et al., 2009).

Additional support for the relation between childhood PTEs and risky health behaviors comes from research using adult data from the National Survey of Midlife in the United States. This research has further demonstrated that people who reported having experienced frequent childhood physical and psychological violence from parents (vs. none) indicated significantly poorer levels of self-rated health across 10 years of adulthood (b=-.12), more functional limitations (e.g., health-related difficulty lifting/carrying groceries, bathing/dressing oneself; b=.16), and more chronic conditions (e.g., asthma, thyroid disease, diabetes; b=.54; Greenfield & Marks, 2009).

Additional research supports the role of childhood PTE exposure in risk for cardiovascular disease specifically. In a sample of 444 African American adults with Type 1 diabetes present before 30 years of age, Roy, Janal, and Roy (2010) found that cumulative childhood PTEs predicted significantly increased risk for the 6-year incidence of any cardiovascular disease, coronary disease, and stroke, independent of traditional risk factors (e.g., age, BMI). Moreover, Norman, Hawkley, Ball, Bernston, and Cacioppo (2013) found that cumulative childhood PTE exposure was significantly associated with higher pulse pressure (physiological risk factor for cardiovascular disease) in a sample of 229 adults (over 60% African American and Hispanic) from the Chicago Health, Aging and Social Relations Study.

Cumulative ACEs have also been found to show a strong graded relation with risky *sexual* health behavior, history of STDs, and pregnancy outcomes (Dube et al., 2003b; Hillis, Anda, Felitti, Nordenberg, & Marchbanks, 2000; Felitti et al., 1998). Specifically, men and women reporting six or more ACEs were at a 3- and 5-fold increased risk of self-reported history of STDs (Hillis et al., 2000), and those reporting four or more ACEs were at a 3.2- and 2.5-fold increased odds of reporting 50 or more sexual partners and ever having a sexually transmitted disease, respectively (Felitti et al., 1998). Moreover, women reporting four or more ACEs (vs. none) experienced a 2-fold increased risk of unintended first pregnancy, a 4-fold increased risk of having 30 or more sexual partners, a 2-fold increased risk of perceiving self as at risk for AIDS, a 5-fold increased risk of having a first sexual encounter by 15 years of age, and a 3-fold increased risk of adolescent pregnancy (Dietz et al., 1999; Hillis et al., 2001; Hillis et al., 2004). Finally, women reporting 3 or more ACEs were at a 3-, 4-, and 6-fold increased risk of smoking, using alcohol, or using any illicit drug during pregnancy, respectively, and women reporting five or more ACEs were at a 2-fold increased risk of fetal death after first and/or second pregnancy (Chung et al., 2010; Hillis et al, 2004).

Adult social cognitive functioning. Not only can exposure to PTEs affect one's long-term mental and physical health, it can also impair adult social cognitive functioning. Importantly, childhood PTE exposure may lead to memory disturbances and impaired socio-emotional functioning, each of which may predispose children to poorer adult outcomes. For example, cumulative ACEs predicted up to a 9-fold increased risk of child autobiographical memory disturbances (Brown et al., 2007). Using a sample of 782 middle-aged adults, Infurna, Rivers, Reich, and Zautra (2015) also found that cumulative childhood PTE exposure was associated with significantly stronger decreases and increases in well-being in response to negative and positive daily life events, respectively, suggesting that childhood PTE exposure can lead to heightened emotional reactivity and impaired emotion regulation.

Childhood PTE exposure has also been found to increase risk for lower educational attainment, higher unemployment, and housing instability. For example, Porche, Fortuna, Lin, and Alegria (2011) investigated the relation between childhood PTE exposure and high school dropout (vs. completion) at any age in a sample of 2,532 21 to 29-year-old adults from the Collaborative Psychiatric Epidemiology Surveys. Critically, the authors found that 20% of adults reporting any major childhood PTE exposure before age 16 dropped out of high school in comparison to 13% of adults reporting no major childhood PTE exposure (Porche, Fortuna, Lin, & Alegria, 2011). Thus, childhood trauma predicted a 1.65-fold (65%) increased likelihood of high school dropout. This is particularly disconcerting because in this same study, Porche et al. (2011) demonstrated a .83-fold (17%) decreased risk of

high school dropout with each additional year of completed maternal education. This suggests that trauma-related decrements in educational attainment may place future generations at disparate risk for academic underachievement in response to their own childhood PTE exposure. Moreover, using a national probability sample of 92 formerly homeless persons and a comparison sample of 395 never-homeless persons, Herman, Susser, Struening, and Link (1997) revealed that experiencing any childhood adversity (sexual abuse, physical abuse, lack of care/neglect) predicted an 8-fold increased risk of lifetime homelessness. With respect to particular types of childhood adversity, lack of care, physical abuse, sexual abuse, and lack of care plus physical or sexual abuse predicted a 13-, 16-, 2-, and 26-fold increased risk of lifetime homelessness, respectively.

Finally, childhood PTEs may also increase adults' risk for future victimization and perpetration. Research consistently demonstrates that the presence of any one ACE is associated with a 2- to 18-fold increased likelihood of reporting additional ACEs (Dong et al., 2004; Felitti et al., 1998). Moreover, experiencing childhood PTEs may increase one's likelihood of perpetrating potentially traumatic acts against one's own children or in the context of adult interpersonal relationships. Craig and Sprang (2007) demonstrated in a sample of 1,680 child-abusing caregivers that any history of PTE exposure in childhood (M=11.47), adulthood (M=10.65), or both (M=11.41) predicted significantly higher child abuse potential compared to no PTE exposure (M=8.69). Additional research by Milner et al. (2010) investigated the relation between childhood physical abuse specifically and higher child abuse potential in a sample of 5,394 US Navy recruits and 716 college students. In both samples, Milner et al. (2010) demonstrated that greater childhood history of physical abuse predicted significantly higher child abuse potential, above and beyond other types of childhood violence exposure including childhood sexual abuse and interpersonal violence exposure. Related, looking specifically at childhood physical or sexual abuse and growing up with a battered mother, higher individual and cumulative childhood ACEs predicted a 3.5- and 3.8-fold increased risk of being the perpetrator or victim of intimate partner violence in adulthood for men and women, respectively (Whitfield, Anda, Dube, & Felitti, 2003).

Findings in Child and Adolescent Samples

Burgeoning research on child and adolescent samples (under 18-years-old) has extended the above research by demonstrating the early emergence of negative outcomes associated with childhood PTE exposure.

Early mental health. Consistent with the adult literature, research on 12- to 17-year-old adolescents has demonstrated a significant relation between the presence of individual childhood PTE categories and up to a 2-fold increased risk of having substance abuse and mental health problems in adolescence (Lucenko, Sharkova, Huber, Jemelka, & Mancuso, 2015). Moreover, elementary school children's greater frequency of exposure to multiple types of PTEs was significantly related to higher self-reported posttraumatic stress symptom severity (Gonzalez, Monzon, Solis, Jaycox, & Langley, 2016). Finally, research on 11,028 1.5- to 18-year-olds experiencing at least one childhood PTE from the National Child Traumatic Stress Network's (NCTSN) Core Data Set (CDS) demonstrated a strong dose-response relation between childhood PTEs and mental health, with each additional type of PTE exposure accounting for a 1.11- to 1.16-fold (11-16%) and 1.07- to 1.15-fold (7-15%) increased odds of scoring in the clinical range for externalizing and internalizing problems, respectively (except sleep problems; Greeson et al., 2014).

Early physical health. Far less research exists with respect to children's and adolescent's physical health status as a function of childhood PTE exposure. However, research using a sample of 170 3- to 5-year-old children recruited from several early care and education centers in an urban, Northeastern city, demonstrated that children who had been exposed to one or more PTE types had significantly worse health-related quality of life (caregiver reported child physical health, dental health, and psychosocial functioning) and psychosocial health (caregiver reported child internalizing and externalizing symptoms) than children not exposed to PTEs (Roberts, Ferguson, & Crusto, 2013). These early health impairments are likely to have cascading effects well into adulthood.

Early social cognitive functioning. One of the best-demonstrated effects of early PTE exposure on child and adolescent outcomes relates to indicators of cognitive development, namely academic achievement. For example, Graham-Bermann, Howell, Miler, Kwek, and Lilly (2010) investigated the relation between childhood

PTE exposure and verbal ability, as measured by the Wechsler Preschool and Primary Scale of Intelligence, in a sample of 87 pre-school aged children exposed to intimate partner violence (IPV) in the past two years. Graham-Bermann et al. (2010) demonstrated that the IPV exposed sample demonstrated significantly lower verbal ability scores compared to a national sample of 1,700 same-aged children not assessed for IPV exposure. Moreover, within the IPV exposed sample, greater frequency of PTE exposure significantly predicted lower verbal ability (which was mediated by maternal education).

Additionally, Duplechain, Reigner, and Packard (2008) found a significant relation between severity of childhood PTE exposure and reading ability in a sample of 167 children in grades 2 through 5 from the Metropolitan Area Child Study. Reading ability was assessed at three time points using one of three standardized tests. Specifically, they found that children with moderate and high violence exposure (vs. no exposure) demonstrated decrements in reading ability scores over the three-year period (Duplechain, Reigner, & Packard, 2008). This effect did not occur when level of childhood traumatic loss (e.g., death of a family member), as opposed to violence (e.g., witnessing physical assault or gun violence), was used to predict changes in reading ability. Related, Enlow, Egeland, Blood, Wright, and Wright (2012) found that children exposed to interpersonal PTEs (vs. none) in the first two years of life (infancy) scored significantly lower on developmentally appropriate cognitive assessments administered at 24-, 64- and 94-months (Enlow, Egeland, Blood, Wright, & Wright, 2012). The effect of infant interpersonal PTE exposure similarly effected child IQ at all time points, and was not affected by the presence or absence of interpersonal PTE exposure during preschool. Strikingly, this effect occurred even after accounting for the role of known risk factors for impaired cognitive development, including: sociodemographic risk, birth-related risk, maternal IQ, and quality of cognitive stimulation in the home.

Finally, Thompson and Massat (2005) investigated the relation between violence exposure (family violence, witnessing violence, community violence in past year), PTSD symptoms, behavior problems, and academic achievement (as measured by the Iowa Test of Basic Skills, a nationally normed achievement test) in a high-risk sample of 110 11 to 13-year-old African American students from four inner-city Chicago neighborhoods. Thompson and Massat (2005) demonstrated that exposure to family violence (r=-.23), community violence (r=-.18), and witnessing violence (r=-.20) were each significantly related to lower academic achievement. Each type of violence exposure was also significantly related to higher PTSD symptoms (rs=.47-.51), and in turn, higher PTSD symptoms were significantly related to lower academic achievement (r=-.32). These findings suggest that children's PTE exposure directly and indirectly (through PTSD symptoms) places them at disparate risk for academic underachievement and later socioeconomic disadvantage.

As in adults, research using child and adolescent samples has also supported the relation between early PTE exposure and socio-emotional functioning. Indeed, Grasso, Ford, and Briggs-Gowan (2013) found that among a sample of 213 2- to 4-year-old children, children with a history of PTE exposure and current non-traumatic stressful life events (e.g., moving, new children in home) had significantly higher internalizing and externalizing problems compared with PTE-exposed children without current stress and all non-PTE-exposed children. This suggests that initial childhood PTE exposure can increase children's vulnerability to subsequently encountered stressors. Additionally, in a neuroimaging study of 30 children and adolescents with a mean age of 13, Marusak, Martin, Etkin, and Thomason (2015) found that PTE-exposed (vs. not exposed) children showed significantly impaired automatic emotion regulation during an emotional conflict task (similar to a Stroop task) in which they had to categorize children's facial expressions (e.g., fear) in pictures while ignoring the semantic content of an overlying congruent (e.g., fear) or incongruent (e.g., happy) emotion word, which may represent an important risk factor for adult psychopathology.

Finally, early PTE exposure may also exacerbate risk of additional PTE exposure and the likelihood of adolescent problem behaviors. Supporting this relation, in a sample of 4,000 0- to 17-year-olds, Finkelhor, Turner, Shattuck, and Hamby (2015) found that children and adolescents experiencing any one victimization experience (e.g., any physical assault) were at a 2- to 5-fold increased risk for experiencing any other type of victimization in past year. Additionally, in a sample of 3,785 13- to 18-year-old children and adolescents from the National Child Traumatic Stress Network Core Data Set, Layne et al. (2014) found a significant relation between higher childhood PTE exposure and higher adolescent problem behaviors, such that each

additional childhood PTE exposure predicted a 1.22-fold (22%) increased odds for attachment difficulties, a 1.06-fold (6%) increased odds of skipping school, a 1.14-fold (14%) increased odds of running away from home, a 1.13-fold (13%) increased odds of criminal activity, 1.11-fold (11%) increased odds of self-injurious behavior, a 1.08-fold (8%) increased odds of substance abuse, a 1.12-fold (12%) increased odds of suicidality, a 1.11-fold (11%) increased odds of alcohol use, and a 1.18-fold (18%) increased odds of being a victim of sexual exploitation. Importantly, each of these high-risk behaviors may in turn place children at increased risk for future PTE exposure and negative PTE-related outcomes.

Mediators and Moderators of PTE Responses: Risk and Resiliency

While the directly observable consequences of childhood PTE exposure have been well-documented in adults and to a lesser extent, children and adolescents, additional research has attempted to demonstrate the pathways through which PTE exposure exerts its pernicious effects on developing children. This is a critical task because these same pathways represent potential targets for the design and implementation of preventive and treatment interventions. In this section, I review the mediators and moderators of PTE responses in children, which include: PTE characteristics, socio-demographic factors, familial factors, self-resources, and biological pathways. Each of these factors has been proposed to uniquely and interactively influence children's relative risk versus resiliency of both exposure to PTEs and negative PTE-related outcomes.

PTE Characteristics

While I have previously outlined multiple dimensions across which researchers have differentiated between types of PTEs, this section will highlight the PTE characteristics most consistently demonstrated to modify severity of proximal and distal PTE-related outcomes.

Poly-victimization. Considerable research has demonstrated the importance of frequent and repeated exposure to *multiple* types of PTEs (i.e., poly-victimization) as a predictor of health and social cognitive functioning. In fact, the effects of poly-victimization are so strong that accounting for poly-victimization status (dichotomous) significantly predicts major outcomes above and beyond cumulative frequency of PTE exposure. For example, research using data from the National Evaluation of Safe Start Promising Approaches found that only poly-victimization status consistently predicted child behavior problems, PTSD symptoms, and parenting stress, above and beyond cumulative childhood PTE exposure frequency (Hickman et al., 2012). Related research has found that including poly-victimization status as a predictor in analyses significantly reduced the relation between repeated exposures to individual types of victimization on trauma symptoms (anger, depression, anxiety, dissociation; Turner, Finkelhor, & Ormrod, 2010; Finkelhor, Ormrod, & Turner, 2007a; 2007b), and low and high poly-victims demonstrated significantly higher anxiety and depression symptom severity than single and chronic single-type victims (Finkelhor et al., 2007b). Together, these findings suggest that it is exposure to multiple types of PTEs, rather than lifetime frequency, that is key in determining variation in trauma symptoms.

Individual PTE types. In contrast to research demonstrating the importance of poly-victimization status above and beyond frequency of exposure to single or multiple PTEs, other research suggests that particular PTEs or combinations of PTEs (whether acute or chronic) may differentially predict PTE-related outcomes. For example, a meta-analysis of research on the relation between childhood PTE exposure and adult mental health found that emotional abuse and neglect showed the strongest associations with adult depression as compared to other kinds of childhood PTE exposure (Mandelli, Petrelli, & Serretti, 2015). In another meta-analysis by Alisic et al. (2014), people exposed to interpersonal PTEs had significantly higher rates of PTSD (25.2%) than those exposed to non-interpersonal PTEs (9.7%).

Additional research has demonstrated the differential effects of certain types of PTEs on psychiatric symptoms (e.g., conduct problems; Briggs-Gowan, Carter, & Ford, 2010; Ford, Stockton, Kaltman, & Green, 2006), homelessness (Herman, Susser, Struening, & Link, 1997), employment (Sansone, Leung, & Wiederman, 2012), somatic preoccupation (Sansone, Wiederman, Tahir, and Buckner, 2009), pain and psychophysiological disorders (Sansone, Pole, Dakroub, & Butler, 2006), and physical illness (Goodwin & Stein, 2004). Similarly, studies of child and adolescent samples have found that specific PTE types better predict impaired executive functioning (i.e., working memory,

inhibition, auditory attention, processing speed; DePrince, Winzierl, & Combs, 2009), more internalizing versus externalizing problems (Shahinfar, Fox, & Levitt, 2000), and more sleep problems (Spilsbury, Babineau, Frame, Juhas, & Rork, 2014).

Sociodemographic Factors

Research suggests that sociodemographic factors (e.g., age, gender, ethnic-racial identity, socioeconomic status) confer differential risk for both childhood PTE exposure and negative PTE-related outcomes. It is important to keep in mind, however, that this body of research is somewhat weakened by sampling issues and problematic data analytic approaches. Sampling issues are perhaps most evident in the domain of ethnic-racial identity effects on childhood PTE exposure; that is, studies typically include either predominantly White samples with too few members of other ethnic-racial groups to allow for comparison or an all minority sample in absence of a comparison group. Moreover, some researchers control for "potentially confounding" sociodemographic risk factors such as age, gender, and ethnic-racial identity without rigorously testing their direct and indirect effects on PTE-related outcomes. Other researchers simply stratify results by sociodemographic factors in absence of the appropriate statistical support (e.g., Lee, Tsenkva, & Carr, 2014).

Below I will present some of the evidence for the influence of sociodemographic risk factors on the sequelae of childhood PTE exposure. I also draw from the literature on health disparities affecting people living in chronic poverty, particularly ethnic-racial stigmatized group members.

Age. With respect to age, researchers have investigated whether age of first PTE exposure, above and beyond cumulative lifetime PTE exposure, influences the relation between childhood PTE exposure and PTE-related outcomes. For example, Graham-Bermann and Perkins (2010) demonstrated that among a sample of 6- to 12-year-olds exposed to intimate partner violence, earlier age of first exposure was significantly related to boys' externalizing problems and girls' total internalizing and externalizing behavior problems; however, when age of first exposure and cumulative exposure were simultaneously entered into the analysis, only cumulative violence exposure remained a significant predictor. In an additional analysis, the authors demonstrated that cumulative violence fully mediated the effect of age of first exposure on behavior problems. Wamser-Nanney and Vandenberg (2013) similarly found that complex trauma predicted higher levels of behavior problems than experiencing a PTE early in life. However, other research has found the opposite pattern of results; that is, age of first exposure moderated the effect of total violence exposure on trauma symptoms such that symptoms were significantly worse with early age of first exposure (Miller-Graff, Scrafford, & Rice, 2016). Related, some research has demonstrated that trauma symptoms are more severe in adolescents exposed to complex trauma than children exposed to similar levels of complex trauma (Lam, Lyons, Griffin, & Kisiel, 2015). Thus, age may be an important moderator of some PTE-related outcomes but not others.

Gender. Research on the moderating role of gender in PTE-related outcomes generally supports the notion that boys and girls may vary in both their PTE exposure and PTE-related responses. For example, Turner and Butler (2003) found that college-age males were significantly more likely to experience high PTE exposure (seven or more PTEs) than females (37.26% vs. 23.58%). Moreover, Alisic et al. (2014) demonstrated gender-related differences in PTSD rates following childhood PTE exposure, with boys exposed to non-interpersonal PTEs having the lowest PTSD rates (8.4%) and girls exposed to interpersonal PTEs showing the highest rates (32.9%). Additional research has demonstrated that gender moderates the role of early PTE exposure in risk for cardiovascular disease, depression (Batten, Aslan, Maciejewski, & Mazure, 2004), and distress-mediated alcohol problems (Strine et al., 2014).

Ethnic-racial identity. Both theory and research suggest that ethnic-racial identity is uniquely related to differential PTE exposure and PTE-related outcomes (e.g., Beristianos et al., 2016). In general, research has revealed that notable health and achievement disparities exist between the dominant ethnic-racial group (i.e., White) and ethnic-racial stigmatized groups (e.g., African Americans). This is consistent with psychological theories of stigma and health, which suggest that "possessing a consensually devalued social identity (a stigma) increases one's exposure to potentially stressful (identity-threatening) situations" (p. 398; Major & O'Brien, 2005). Chronic experiences with and expectations of discrimination can in turn overwhelm the body's psychological and physiological capacity for coping with stress (see Biological Pathways section below). The chronic stress associated with membership in a stigmatized ethnic-

racial group may be further exacerbated by sociostructural barriers; that is, patterns of systemic discrimination have disparately limited certain ethnic-racial groups (viz. African Americans and Hispanics) to low SES, high-crime communities (e.g., Ross & Turner, 2005). Together, this suggests that ethnic-racial stigmatized group members are doubly at risk for developing negative PTE-related outcomes due to the stress induced by (1) awareness of social stereotypes and prejudicial attitudes against their group and (2) chronic exposure to PTEs that is endemic to low SES communities.

With these disparities in mind, a number of studies have demonstrated that ethnic-racial minority status has been associated with greater exposure to family and community violence, whereas White ethnic-racial identity has been linked with higher exposure to serious accidents and illnesses; however, the majority of this research did not demonstrate a moderating effect of ethnic-racial identity on actual PTE-related outcomes (Benítez, Yen, Shea, Edelen, Markowitz, et al., 2010; Beristianos et al., 2016; Briggs-Gowan et al., 2010; Duplechain, Reigner, & Packard, 2008). By comparison, Porche, Fortuna, Lin and Aleria (2011) found that ethnic-racial identity moderated the relation between childhood PTE exposure, childhood substance and conduct problems (mediator), and likelihood of school dropout. That is, compared to non-Hispanic Whites, African Americans and Hispanics showed a stronger relation between greater childhood PTE exposure and increased likelihood of school dropout via increased substance and conduct problems, whereas Asians showed a relatively weaker mediation effect.

Socioeconomic status. Poverty, which is itself a chronic source of stress, is a strong sociodemographic risk factor for increased PTE exposure and more negative PTE-related outcomes. As is the case with ethnic-racial stigmatized groups, research has demonstrated socioeconomic status related differences in health and achievement (e.g., Reardon, 2011). One potential source of SES-related disparities may be differential exposure to childhood PTEs. For example, Duplechain, Reigner, and Packard (2008) found that among a sample of children from inner-city elementary schools in the Midwest, those from low- (87%) and middle-income (89%) families reported significantly more exposures to violence than those children from high-income (49%) families. Moreover, Klest (2012) found that the relation between childhood and adult victimization was stronger in communities with higher versus lower poverty rates. Importantly, additional research has demonstrated that low socioeconomic status (and higher neighborhood-level crime) can make children more vulnerable to developing depressive symptoms (Lowe et al., 2016) and impaired pre-school age verbal ability (Graham-Bermann, Howell, Miller, Kwek, & Lilly, 2010). These findings are consistent with the idea that low SES limits people's social power and access to resources that may otherwise help them effectively cope with the negative effects of chronic stress and trauma (e.g., Gallo, de los Monteros, & Shivpuri, 2009).

Collectively, findings supporting the moderating role of sociodemographic factors in childhood PTE exposure and PTE-related responses strongly support the need to continue to assess sociodemographic risk in future PTE exposure and victimization research in order to better elucidate the conditions under which it is most likely to affect PTE-related outcomes.

Self-Resources and Social Support

Self-resources. The sequelae of childhood PTE exposure can be meaningfully impacted by individual differences in the child's initial PTE-related reactions, personality traits, coping styles, and attachment beliefs. Importantly, this research is limited in its ability to account for the role of the child's environment in shaping personality.

Initial PTE-related symptoms are one pathway determining risk for later negative outcomes. For example, Briggs-Gowan, Carter, and Ford (2012) demonstrated that the direct effect of 3-year cumulative family and neighborhood violence exposure on higher internalizing and externalizing symptoms as well as lower social competence (social problem-solving, social flexibility, affiliation, consideration of others) in the early elementary school years was significantly mediated by PTE-related symptoms (arousal and avoidance) measured at three years of age, independent of sociodemographic risk and past-year violence exposure. Moreover, levels of psychological trauma symptoms fully and partially mediated the effect of childhood PTE exposure on adult substance use problems (Strine et al., 2012; see also Cross, Crow, Powers, & Bradley, 2015) and child abuse potential (Milner et al., 2010), respectively. Thus, greater childhood PTE exposure was related to significantly more severe trauma symptoms, and more severe trauma symptoms were related to significantly

greater odds of substance use or child abuse potential. Porche et al. (2011) also demonstrated that substance use itself can mediate the relation between childhood PTE exposure and other important outcomes like educational attainment. Indeed, they found that greater childhood PTE exposure was related to childhood onset of substance and conduct disorders, which were significantly related to 2.48- and 2.38-fold increased odds of school dropout, respectively (Porche et al., 2011).

In addition to initial PTE-related reactions, a host of personality traits and coping styles have also been demonstrated to affect relative risk and resiliency. Brodhagen and Wise (2008) found that dispositional optimism partially mediated the direct effect of childhood physical and emotional abuse and fully mediated the direct effect of exposure to PTEs (rape, assault, combat, fire) on current distress symptoms, such that participants with higher levels of optimism had lower levels of PTE-related distress. Research measuring individual differences in overall psychological resilience has demonstrated that higher dispositional resilience mitigates the significant relation between greater childhood PTE exposure and increased lifetime alcohol and illicit drug use (Wingo, Bradley, & Ressler, 2014), depressive symptom severity (Wingo et al., 2010), and likelihood of PTSD (Wrenn et al., 2011). With respect to coping styles and expectancies, in a longitudinal study 3- to 5-year-old children, Jester, Steinberg, Heitzeg, and Zucker (2015) found that the relation between parental violence exposure and peak alcohol use was mediated by psychological distress and coping expectancy (i.e., drinking to cope with negative emotions).

Social resources. The family represents an important social structure affecting children's exposure and reaction to PTEs. Although research has largely focused on parental characteristics associated with children's PTE exposure and outcomes, it is important to think of the family as a dynamic system that is a product of not only individual family members' characteristics, but also their interactions with each other and with the context in which they live. Certain contexts, as I have argued above, confer greater risk for PTE exposure not only in childhood, but throughout the life course. It is therefore important not to interpret the following findings as "finger-pointing" at parents, as toxic family environments may be created because of parents' own past and current PTE exposure, the limits of their environment to support their needs, and the resulting effect on their socioemotional functioning. Thus, larger structural inequalities may ultimately set some families up for failure.

Research has demonstrated several family-related risk and resiliency factors that impact PTE exposure and PTE-related outcomes, including parental alcoholism (Anda et al., 2002), parental psychiatric illness, parental trauma symptoms, and parental victimization (Dulmus & Wodarski, 2000). With respect to PTE exposure, Costello, Erkanli, Fairbank, and Angold (2002) found that children and adolescents with a higher number of family vulnerability factors (i.e., parental psychopathology, family relationship problems, family environment problems) were at greater risk of exposure to more low magnitude (e.g., new child in home) and high magnitude (e.g., death of a loved one) events.

With respect to how specific family factors affect PTE-related outcomes, research has shown that parental trauma symptoms fully or partially mediated the direct effect of family and community violence exposure on pre-school age children's depressive and conduct symptoms (Briggs-Gowan et al., 2010), and on their internalizing and externalizing behavior problems (Linares et al., 2001). High parenting stress may represent another risk factor, as Whitson, Bernard, and Kaufman (2015) found that parenting stress significantly mediated the relation between childhood PTE exposure and internalizing symptoms. Related, in a sample of 5- to 13-year-old children exposed to intimate partner violence, mother's history of violence and maltreatment, maternal depression, and lower maternal social support predicted more traumatic stress symptoms in children (Graham-Bermann, DeVoe, Mattis, Lynch, & Thomas, 2006).

Recent research suggests that it is a constellation of family vulnerability factors, and not necessarily any single factor, that shapes PTE-related responses. In a large nationally representative sample of children 2 to 9 years old, Turner et al. (2012) found that family context and victimization factors that related to *safety* (poor supervision, physical/sexual maltreatment, witnessing family violence, juvenile sibling victimization), *stability* (inconsistent/hostile parenting, family adversity, number of moves in past year) and *nurturing* (mother diagnosed with psychiatric disorder, parent conflict, emotional maltreatment) significantly predicted trauma symptom scores. Inconsistent/hostile parenting and emotional maltreatment were the

strongest independent predictors of trauma symptom severity. Thus, the family represents a critical context for understanding children's relative risk and vulnerability following exposure to PTEs.

A resilient social support network not only stems from the family environment, but also from peer relations and institutional involvement (e.g., religious). Lowell, Renk, and Adgate (2014) found that maternal and peer attachment significantly predicted variance in internalizing and externalizing symptoms above and beyond childhood maltreatment history, suggesting that healthy attachment may protect children against the negative emotional and behavioral effects of childhood maltreatment. Further highlighting the important buffering role of social relations, research has demonstrated that whereas perceived social isolation can exacerbate negative PTE-related responses (e.g., higher pulse pressure; Norman et al., 2013), a strong sense of community and good health habits can prevent negative PTE-related responses (e.g., psychological distress; Nurius, Green, Logan-Greene, & Borja, 2015).

Finally, research on the relation between childhood PTE exposure and religiosity suggests that religious involvement (religious service attendance, use of religion tor comfort and guidance, importance of religion) can protect self-esteem against PTE exposure. However, certain types of religious coping may be harmful. For example, Reinert et al. (2015) found that at high levels of negative religious coping, women's childhood PTE exposure predicted worse physical health than men.

As is true of all risk and resiliency factors, future research is needed to better understand the boundary conditions for the buffering effect of different personality traits, interpersonal relationships and belief systems (e.g., coping expectancies, religion).

Biological Pathways: Epigenetics and the Brain

Accumulating evidence from animal, nonhuman primate, and, more recently, human research suggests that early childhood adversity, such as exposure to PTEs, indirectly increases risk for subsequent PTE-related outcomes (viz. physical and mental illness) via its effects on brain development and functioning, particularly as it pertains to the body's stress response system (for reviews, see Heim et al., 2010; Heim & Nemeroff, 2001; Nemeroff, 2004). The body's stress response system is largely regulated by corticotropin releasing factor (CRF) activity in the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS). Following exposure to a stressor, CRF secreted from the hypothalamus stimulates the release of adrenocorticotropic hormone (ACTH) from the pituitary gland (Heim & Nemeroff, 2001; Heim et al., 2010; Nemeroff, 2004). ACTH then stimulates the release of glucocorticoids (viz. cortisol) from the adrenal glands, which modulate immune system functioning. Additionally, CRF triggers the release of catecholamines (e.g., epinephrine/adrenaline, norepinephrine/noradrenaline) to activate the ANS, which prepares the body for a "fight or flight" response. Such a response is adaptive in the short-term in that it automatically redirects various physiological resources toward preparing the body to either confront or avoid a stressor to ensure survival. Eventually, feedback loops signal the body to "turn off" the stress response and return to its resting state, or homeostasis. Unfortunately, the chronic activation of this otherwise adaptive system during development can ultimately lead to an exaggerated, or sensitized, stress response (Heim & Nemeroff, 2001). Importantly, chronic stress reactivity can ultimately break down the body, leaving people vulnerable to physical and psychological illness (i.e., allostatic load; McEwen & Stellar, 1993).

Early environments can also increase risk for PTE-related outcomes by regulating gene expression (i.e., gene X environment interaction) through epigenetic processes (Liu, Li, & Tollefsbol, 2008). That is, vulnerable (or protective) variants of a gene involved in regulating the body's stress response system may lead to increased (or decreased) risk for an outcome like depression in the presence of moderate to severe childhood PTE exposure, but may be relatively inconsequential in the presence of no to low childhood PTE exposure (Labonte et al., 2014; Liu et al., 2008; McGowan et al., 2009; Tammen, Friso, & Choi, 2013; Weinhold, 2006). Importantly, epigenetic processes may themselves lead to heritable changes in gene expression without changing the DNA sequence itself (Heard & Martienssen, 2014). It is these changes that are thought to largely account for the intergenerational transmission[2] of trauma from one generation to the next, such that some children are at disparate risk to develop traumatic symptoms and impaired functioning in response to their own early PTE exposures due to their parents' trauma history.

The role of brain-mediated outcomes of childhood PTE exposure may be particularly important in the context of poverty. As previously

discussed, chronic PTE exposure throughout the lifecycle is endemic to high poverty, high crime communities, and chronic PTE exposure during childhood is likely to result in prolonged activation (and permanent alteration) of the body's stress response system (i.e., hormones, genes, neural activity). Related, with respect to epigenetic heritability and the intergenerational transmission of trauma, to the extent that generations of a family live within similarly high poverty, high crime environments due to structural barriers that preclude their access to support services and limit their potential for social mobility, epigenetic changes should be more likely to persist through multiple generations.

With the above in mind, I next review empirical evidence for the role of biological pathways in the effect of childhood PTE exposure on illness and impaired functioning.

Stress response markers. One of the primary means by which researchers have investigated the epigenetic and brain-related effects of childhood PTE exposure is through various biomarkers of stress from the neuroendocrine, immunological, metabolic and cardiovascular systems.

In support of this, several studies have indeed suggested that more frequent childhood PTE exposure is associated with elevated stress responsiveness even in absence of a current stressor. For example, Lin, Neylan, Epel, and O'Donovan (2016) investigated the relation between childhood adversity (repeating a year of school, household dysfunction related to parent alcohol or drug abuse, physically abuse by parent), adult adversity (death of child; fire, flood, earthquake or natural disaster; combat exposure; family member addicted to drugs/alcohol; victim of a serious physical attack or assault; self, spouse or child experienced life-threatening illness or accident) and levels of an inflammatory biomarker, high sensitive C-reactive protein (hsCRP), in a population-based sample of 11,198 adults aged 50 or older from the Health and Retirement Study. The authors found that the presence of any childhood or adult adversity (dichotomized) and their greater frequency (continuous) each predicted significantly higher blood levels of hsCRP. Moreover, in a comparison of participants with no adult or childhood adversity, childhood but no adult adversity, adult but no childhood adversity, and childhood and adult adversity, Lin et al. (2016) found that participants reporting both childhood and adult adversity tended to have significantly higher hsCRP blood levels than participants reporting adult adversity alone; however, while the mean hsCRP blood level of childhood and adult adversity participants was higher than for participants reporting childhood adversity alone, this difference was not statistically significant, suggesting that mean differences in hsCRP blood levels between the two samples do not reflect "true" population differences. This finding points to the relation between childhood adversity, even more so than adult adversity, and inflammation, which is a pathway through which chronic stress is thought to increase risk of disease (Lin et al., 2016).

Making a particularly compelling case for the specific role of childhood PTE exposure in the context of poverty in creating biological risk for disease development, Blair, Raver, Granger, Mills-Koonce, and Hibel (2001) investigated the relation between poverty-related early adversity (i.e., length of time in poverty, housing quality, perceived economic strain, perceived economic sufficiency, adult exits from the home) and baseline salivary cortisol levels in a sample of 1,135 children from the Family Life Project (FLP). Poverty-related adversity was measured prospectively at 7 months of age, and children's salivary cortisol was measured at 5 time points between 7 months and 4 years of age. Uniquely, the FLP sample was constructed to allow for the comparison between African American and White children living in high poverty areas in the United States. Results revealed that longer time in poverty and poorer housing quality predicted significantly higher levels of salivary cortisol across the four-year period (Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011). With respect to ethnic-racial identity, African American children had significantly higher levels of cortisol than White children even after statistically controlling for variation in cortisol explained by a number of household risk factors. According to the authors, this finding may suggest that although poverty-related risk influences children of different ethnicities similarly, because African Americans are overrepresented in low-income areas due to a history of social injustice, African American ethnic-racial identity confers additional risk for compromised stress physiology (Blair et al., 2011).

While the aforementioned research did not directly test whether PTE-related changes in concentrations of biomarkers explained the relation between childhood PTE exposure and mental and physical problems, other research has indeed found that altered stress

physiology is in turn predictive of PTE response severity. For example, in a study on the relation between catecholamine (epinephrine/adrenaline, norepinephrine/noradrenaline, dopamine) and cortisol levels and PTSD symptom severity in 82 8 to 18-year-old children admitted to a level 1 trauma center, Delahanty, Nugent, Christopher, and Walsh (2005) found that higher levels of urinary cortisol were associated with higher levels of 6-week PTSD symptoms (r=.31, small effect size). Moreover, even after controlling for significantly related demographic variables and 6-week depressive symptoms, higher urinary cortisol and higher epinephrine/adrenaline each significantly predicted higher 6-week PTSD symptoms, accounting for 10% and 7% of variance in PTSD symptoms, respectively. In a related study on the relation between cortisol and PTSD symptom severity in a sample of 82 8 to 18-year-old children admitted to a level 1 trauma center, Ostroswki, Christopher, van Dulmen, and Delahanty (2007) also found a significant relation between higher levels of urinary cortisol and higher PTSD symptoms (r=.41, small to medium effect size). These findings provide reliable support for the role of altered stress physiology in mental illness severity in response to childhood PTE exposure.

Sadly, the effect of PTE-related elevations in biomarkers that signal chronic stress and cell damage extends not only to children's health, but also to their cognitive development. In a separate report using data from the first 3 years of the FLP dataset, Blair et al. (2011) investigated the relation between parenting (positive and negative) and poverty, salivary cortisol measured at 7 months, 12 months, and 24 months, and two indices of cognitive ability (executive function, intelligence) measured at age 3 in 1,292 children (larger sample than reported above due to less attrition in 3 years compared to 4). Poverty was measured in several ways, including income-to-need (household income/2005 federal poverty level, adjusted for number of residents), maternal education, household risk (density [number of residents/number of rooms], noise and safety), and African American ethnicity. Execute function was operationalized through children's combined performance on a working memory span task, an attention shifting task, and an inhibitory control task; intelligence (IQ) was measured using the Wechsler Preschool and Primary Scales of Intelligence. In a test of their primary hypotheses, the authors found that cortisol levels mediated the direct significant relation between lower positive parenting and lower executive function (but not IQ); that is, lower positive parenting was significantly related to higher cortisol levels, which was in turn significantly related to lower executive function. Further, they found that lower maternal education and African American ethnicity indirectly predicted lower executive function through negative parenting, positive parenting, and the combined effects of positive parenting and cortisol levels. Lower income-to-need, maternal education, and African American ethnicity predicted lower IQ through negative parenting, positive parenting, and household dysfunction. Thus, poverty-related early adversity impairs children's cognitive development by altering the body's stress physiology, and populations who have been historically and presently relegated to living in poverty.

Genes and epigenetics. Research on the structure and function of genes involved in regulating the body's stress response has also provided compelling support for the role of epigenetic and brain-related processes in modulating childhood PTE-related outcomes. For example, Bradley et al. (2008) found a protective effect of variants of the corticotropin-releasing hormone type 1 receptor (*CRHR1*) gene, exposure to childhood abuse (physical, sexual, emotional), and adult depressive symptoms among 476 African American adults living in an urban area. Specifically, the researchers found that although adults experiencing "none to mild" childhood abuse had similar adult depressive symptoms regardless of genotype, among adults experiencing at least one instance of moderate to severe childhood abuse, those with one or more protective CRHR1 variants demonstrated significantly lower adult depressive symptoms than those without this variant.

Additional research has demonstrated the interactive role of HPA axis stress-regulating genes and childhood PTE exposure in predicting risk for suicidal behavior and substance dependence (Enoch, Hodgkinson, Gorodetsky, Goldman, & Roy, 2013; Enoch et al., 2010; Roy, Gorodetsky, Yuan, Goldman, & Enoch, 2010; Roy, Hodgkinson, DeLuca, Goldman, & Enoch, 2012; Roy, Hu, Janal, & Goldman, 2007). For example, Enoch, Hodgkinson, Gorodetsky, Goldman, and Roy 2013) demonstrated a graded relation between number of genetic risk variants in the serotonin transporter gene and risk for suicide attempt in a sample of 474 African American adults (cf. Roy et al., 2007). They found that among those exposed to high levels of childhood trauma

(but not low levels), there was a .25 prevalence of attempted suicide in those with no risk variants, .39 in those with one risk variant, and .56 in those with two risk variants. Further analyses of this database have revealed that among those exposed to high levels of childhood trauma (but not low levels), 20% of people with no FKBP5 genetic risk variants, 36% with one risk variant, and 51% with two risk variants attempted suicide (Roy et al., 2010). Moreover, 58% of African Americans with a risk variant of FKBP5 and CRHPB attempted suicide, compared to 30-35% of those with either variant and 24% among those with neither variant (Roy et al., 2012).

Turning to substance dependence as a childhood PTE-related outcome, in a study of African American men with or without a lifetime history of alcohol, cocaine, and/or heroine dependence, certain variants of the GABRA2 gene moderated the effect of childhood PTE exposure on risk of substance dependence (especially cocaine; Enoch et al., 2010). Consistent with the above reported findings on suicide attempt prevalence, whereas three of the four explored variants predicted significantly higher frequency of any substance dependence among those with high but not low childhood trauma exposure, one variant did not predict significant difference in substance dependence frequency by level of childhood trauma. This latter finding suggests that variants of GABRA2 interact with severity of childhood trauma exposure to predict differential risk for substance dependence.

Interestingly, rather than studying genetic variants, O'Donovan et al. (2011) examined the biological toll of childhood PTE exposure through an investigation of differences in leukocyte telomere length (LTL), one indicator of biological aging. Specifically, they investigated the relation between childhood PTE exposure (number of categories, number of PTEs), PTSD diagnosis, and LTL in a sample of 90 participants with or without a chronic PTSD diagnosis. They found a significant main effect of both PTSD diagnosis and childhood PTE exposure, with shorter LTL among those with versus without PTSD diagnosis and as a function of greater childhood PTE exposure. Importantly, however, analyses comparing the relative contribution of PTSD diagnosis and childhood PTE exposure on shortened LTL revealed that only people with PTSD *and* a history of childhood trauma had significantly shorter age-adjusted LTL (indicator of biological aging) than control subjects. This finding suggests that childhood PTE exposure accounted for the differences in LTL length between PTSD and no-PTSD participants. Indeed, biological changes in response to PTE exposure can lead to premature biological aging, which, in addition to predisposing people to mental illness, can lead to early age-related disease and death.

Neural activation. The effects of childhood PTE exposure on stress reactivity in the brain can also be understood by investigating functional brain changes (e.g., altered patterns of neural activation/inhibition).[4] Using a sample of 67 14- to 18-year-olds, predominately African American adolescents who were part of a longitudinal birth cohort study on disadvantaged youth, Elsey et al. (2015) demonstrated a relation between higher PTE exposure and cortico-limbic activity in response to stress cues. Compared to low-PTE-exposed adolescents, high-PTE-exposed adolescents had increased activation of insula, anterior cingulate, and prefrontal cortex in response to stress cues. Moreover, in the high-PTE-exposed group only, decreased limbic activity (e.g., hippocampus) was associated with higher subjective anxiety in the high- but not low-PTE-exposed group. These findings suggest that more frequent PTE exposure is associated with significant changes in the brain's reaction to subsequent stressors.

In a similar line of research, McLaughlin, Peverill, Gold, Alves, and Sheridan (2015) looked at the relation between brain activation in a sample of 42 13- to 19-year-old adolescents with or without a history of exposure to physical and/or sexual abuse. Specifically, they found stronger activation of the brain's "salience network" (e.g., amygdala) in response to negative (vs. neutral) stimuli among adolescents with a history of PTE exposure compared to those without such a history. Moreover, PTE-exposed adolescents were more likely to show increased activation of brain regions involved in regulating emotional responses (e.g., superior frontal gyrus) when instructed to decrease their emotional response to negative stimuli (vs. passive viewing). The authors interpreted this latter finding to suggest that PTE-exposed adolescents need to recruit more of the brain areas involved in regulating their exaggerated stress response.

Finally, Suzuki et al. (2014) investigated limbic activity in a sample of 115 7 to 12-year-olds with and without psychiatric diagnoses and with varying levels of early life trauma (e.g., physical and sexual abuse, natural disaster, death of sibling) and stress (e.g.,

death of pet, change in school). They found that, regardless of psychiatric diagnosis, greater number of early life trauma was related to greater limbic activation (viz. amygdala) to both positive and negative emotional faces (e.g., sad, happy, fearful) compared to neutral faces, whereas greater number of early life traumas was specifically related to greater limbic activation in response to sad faces. Thus, both early life stress and early life trauma are associated with altered emotion-related brain activity, particularly in response to negative emotional cues. Exaggerated stress-response reactivity may in turn predispose children to develop stress-related disorders like depression.

Recommendations for Building Resilience in Children and their Social Environments

Given the above literature review, childhood PTE exposure is undoubtedly a complicated public health issue for which effective prevention (and intervention) paradigms are needed. This is particularly true for populations that are disparately affected by high levels of family and community violence, namely those in the context of chronic poverty. Disturbingly, a history of social injustice has led to the overrepresentation of African Americans in these high-risk communities (and a conflation of African American ethnic-racial identity with terms like "poverty" and "crime/criminal"). Thus, in the long-term, *preventing* the disproportionate effects of PTE exposure in high risk communities requires major legislative changes aimed at the underlying sociostructural factors that ultimately increase PTE exposure amongst certain demographic enclaves. However, in the interim, it is essential to put systems in place that build resilience[5] in children and in the social environments in which they develop. This will have the dual purpose of helping children who have not yet been exposed to trauma maintain normal functioning when such exposure inevitably occurs in at risk communities, and helping previously PTE exposed children cope with current and future adversity. I therefore end this paper with a series of important recommendations for building resilience in children and their social environments.

Resilience Defined

In their review of the resilience literature, Luthar, Cicchetti, and Becker (2000) defined resilience as a "dynamic process encompassing positive adaptation within the context of significant adversity" (p. 543). Further, they identified three sources of resilience, including (1) the child, (2) the family, and (3) broader social environment(s) (Luthar, Cichetti, & Becker, 2000). Thus, while definitions of resilience vary across the literature, resilience[6] is generally thought of as a constellation of protective factors internal and external to the child. Moreover, resilience is not a stable or global trait, as resilient outcomes may vary across contexts and domains (e.g., academic vs. socioemotional; Luthar et al., 2010). This is critical because even if a child appears resilient in one domain, this does not preclude impaired functioning in another domain. It would therefore be a serious misstep to identify and divert resources away from seemingly "resilient" children. Instead, energy and resources should be focused towards building resilient social environments that positively impact developing children.

In line with this suggestion, Wright, Masten, and Narayan (2013) culled a "short list" of factors associated with resilience among children and adolescents. The list was comprised of four broad categories: (1) child characteristics (e.g., effective emotional and behavioral regulation strategies, positive view of self, faith and a sense of meaning in life); (2) family characteristics (e.g., stable and supportive home environment, socioeconomic advantages, faith and religious affiliations); (3) community characteristics (e.g., high neighborhood quality, effective schools, good public healthcare); and (4) cultural or societal characteristics (e.g., protective child policies, value and resources directed at education, prevention or protection from oppression/political violence; see Wright, Masten, & Narayan, 2013 for the complete list). As is evident from a cursory review of this list, many of the factors underlying individual and environmental resilience are inherently tied to socioeconomic status. Thus, more resources will likely be required to foster resilience in low SES, high crime communities at disparate risk for childhood PTE exposure and its consequences. Importantly, one of the biggest barriers to resilient children and social environments is perhaps a lack of knowledge, suggesting that increased awareness of the sequelae of childhood trauma amongst agents operating in formative social environments should be a primary target of policies aimed at reducing the impact of childhood PTE exposure in at risk communities.

Recommendations for Building Resilience

Building resilience in both children and their social environments (e.g., school, home) is key for enhancing children's ability to maintain normal functioning in the face of chronic PTE exposure, as well as for identifying and supporting vulnerable children. Thus, trauma-informed educational practices must become the standard in educational settings. Such practices are critical for promoting sensitive disciplinary practices, creating knowledgeable and compassionate personnel, and fostering collaboration with mental health professionals (Crosby, 2015). Trauma researchers consensually agree that schools are a primary access point for screening and treating PTE exposed children and adolescents (e.g., de Arellano et al., 2005; Holmes, Levy, Smith, Pinne, & Neese, 2015; Langley, Gonzalez, Sugar, Solis, & Jaycox, 2015). Having access to trauma-informed care within schools is critical because mental health services are often inaccessible to children and parents from low SES communities due to social (e.g., fear of stigma) and structural (e.g., location, costs) barriers (Aisenberg & Mennen, 2000; Pynoos et al., 2008). As a caveat, it is essential to remain sensitive to the fact that mental illness labels are disproportionately stigmatizing in communities at high risk for PTE exposure and its lasting effects. It is therefore essential to ensure that we do not overuse or misuse stigmatizing labels like "traumatized" when referring to children exposed to PTEs, as the labels themselves may be equally or more psychologically damaging than PTE exposure itself.

With respect to specific school personnel, school counselors and psychologists should complete specialized training in effective trauma interventions (Langley et al., 2015; Little & Akin-Little, 2013). However, all school personnel should be routinely trained to recognize and respond to trauma. Otherwise, for example, teachers may misattribute a child's impaired social and academic functioning to a non-trauma-related source, which might lead to a punitive rather than therapeutic response (Aisenberg & Mennen, 2010).

It is also essential to incorporate parents or caregivers in prevention efforts since the family context provides a critical source of risk and resiliency for children. It is not uncommon for parents to underestimate their children's PTE exposure and trauma-related reactions (Aisenberg & Mennen, 2000). Thus, parents must also learn how to recognize and respond to trauma in their children (Fraser et al., 2014; Langley, Santiago, Rodríguez, & Zelaya, 2013). Additionally, schools can serve as sites for organizing activities that foster healthy parent-child attachment (e.g., Swick, Knopf, Williams, & Fields, 2013). This will be essential in promoting consistent and stable family routines, which are important for trauma resiliency (Kiser, 2007; Kiser, Medoff, & Black, 2015). However, policy-makers must be sensitive to the fact that many parents living in low SES communities do not have the luxury of taking time off to participate in collaborative trauma education programs or parent-child bonding activities without additional financial assistance.

Another important aspect of trauma-informed educational practices is the creation of school-wide programs that increase children's and adolescents' personal understanding of trauma and promote effective coping strategies. Such programs will ultimately contribute to the de-stigmatization of trauma, which will in turn encourage children to disclose and process their PTE exposure (Graham-Bermann, Kulkarni, & Kanukollu, 2011). Additionally, school-wide programs will allow school personnel to more discretely identify any particularly vulnerable children and provide them with the extra care that they need. When PTE exposure is endemic to a child's environment, every effort must be made to ensure that when trauma is almost inevitably encountered, the child has a toolbox of effective coping mechanisms to prevent long-term negative consequences. Doing anything else would be turning a blind eye to the very nature of living in poverty.

Finally, other community-settings where children and adolescents spend significant portions of their time, such as religious centers, should also be encouraged to adopt trauma-informed practices. Critically, this should also extend to law enforcement agencies, as police officers are highly likely to interact with children following immediate exposure to a violent event (Murphy, Rosenheck, Berkowitz, & Marans, 2005; Osofsky et al., 2004). Moreover, police officers in contact with delinquent youths must remain aware of the larger factors that influence children's and adolescents' problem behavior in order to respond sensitively and appropriately. Imbuing trauma-informed practices into a number of educational and community-based settings has the added value of increasing children's potential support networks.

Conclusion

It is clear that successful initiatives to combat the toll of childhood trauma require major collaboration across numerous systems (Smithgall, Cusick, & Griffin, 2013). While this certainly poses a major challenge for law- and policy-makers trying to coordinate trauma prevention and intervention initiatives, it is by no means an insurmountable one. By adopting established and emerging trauma-informed practices on a wide-scale, it may be possible to maintain the potentiality of trauma-exposed children.

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Endnotes

[1]The ACE Study consisted of two waves of data collected from adult health maintenance organization members who had received a standard medical evaluation at Kaiser Permanente's San Diego Health Appraisal Clinic between August and November 1995 and January and March 1996 (Wave I) or between June and October 1997 (Wave II; Dietz et al., 1999; Dube et al., 2003; Felitti et al., 1998). Adults were mailed the ACE questionnaire two weeks after their initial evaluation; questionnaires were returned by 70% of Wave I participants and 65% of Wave II participants. The dataset was further reduced by the removal of participants who had mistakenly completed both waves of the ACE Study and who had missing data for their race and educational attainment, resulting in 8,708 and 8,629 participants in Waves I and II (17,337 total), respectively. Research reports generated from this dataset differ in terms of sample size and the types of ACEs included in their analyses. These differences result from changes made to the ACE survey between Waves I and II and researchers' specific exclusion criteria (e.g., only women). The Wave I survey captured three types of childhood abuse (emotional/psychological, physical, sexual) and five types of household dysfunction (household exposure to substance abuse, mental illness, domestic violence, criminal behavior; parental separation or divorce) occurring before the age of 18; however, some reports have excluded parental separation or divorce as an ACE (e.g., Dietz et al., 1999; Felitti et al., 1998). The Wave II survey captured the eight ACEs included in Wave I in addition to emotional and physical neglect. Thus, with few exceptions, ACE reports using Wave II data alone include 10 ACEs, whereas ACE reports using Wave I data alone or both waves include seven to eight ACEs. In general, ACE data is analyzed by summing across ACE categories and/or by looking at the presence (vs. absence) of individual types of ACEs.

[2]Less evidence supports the role of epigenetic inheritance in the transgenerational transmission of trauma across multiple generations far removed from the environment that provoked the epigenetic change (Krauss-Etschmann, Meyer, Dehmel, & Hylkema, 2015).

[3]It is important to note that much extant research on the epigenetic and brain effects of childhood trauma exposure have been conducted with Holocaust survivors and their offspring. For example, van der Hal-Van Raalte, Bakermans-Kranenburg, and van IJzendoom (2008) studied the relation between age during Holocaust and cortisol stress reactivity in a sample of 133 child Holocaust survivors on Holocaust survivors. They found that of the three age groups, the youngest group of males showed the strongest cortisol response to a subsequent stressor (i.e., completing a questionnaire about their experiences surviving the Holocaust and other PTEs), suggesting that PTE exposure during critical early developmental periods is more likely to produce changes in stress physiology than later exposure. Additional research shows that parental Holocaust exposure predicts alterations to Holocaust offspring's HPA-axis stress-regulating genes (e.g., FKBP5; Yehuda et al., 2016), for instance, which may in turn leave Holocaust offspring more vulnerable to developing negative outcomes in response to subsequent life stressors. While this research is outside of this paper's scope, that children exposed to more conventional PTEs demonstrate similar biological changes that predispose them to illness and impaired functioning as children (and their offspring) exposed to one of the greatest human rights violations in history should be a wake-up-call for policymakers and the public alike.

[4]Research linking childhood PTE exposure with altered brain structure (e.g., reduced limbic volume) has provided inconsistent and contradictory results. Moreover, this research tends to look at populations with or without a psychiatric disorder (e.g., PTSD) in the presence or absence of childhood PTE exposure, and therefore typically fails to provide compelling evidence as to whether brain structure changes were a cause or consequence of psychiatric disorder.

[5]According to Bonanno (2004), whereas recovery refers to a PTE exposed person's return to normal functioning following an acute period of impaired physical and psychological functioning, resilience refers to a person's ability to maintain relatively normal functioning throughout the

course of PTE exposure. Thus, although most people do not develop major physical or psychological problems in response to PTE exposure, this does not indicate that most people are "resilient," but rather that most people "recover" (Bonanno, 2004). Moreover, research focusing on discrete PTE-exposure, as opposed to the chronic PTE exposure affecting low SES urban communities, may overestimate the prevalence of recovery when PTEs constantly confront people.

[6]Whereas "resilience" refers to the process of maintaining normal functioning in the face of adversity, "resiliency" refers to an individual trait (Luthar et al., 2000).

The Rutgers Center on Law, Inequality and Metropolitan Equity (CLiME) is committed to studying the role of law and policy in encouraging or inhibiting opportunity based on place. This report is a part of our Trauma, Schools and Poverty Project (TSP), a multi-year effort to understand the relationships between structural inequality and the pervasive experience of complex psychological stress and trauma.

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