A Review of Reviews Examining Neurological Processes Relevant to Impact of Parental PTSD on Military Children: Implications for Supporting Resilience

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In this study, 10 recent meta-analytic and systematic review studies were synthesized on the neurological underpinnings of stress and trauma with implications for the impact of parental post-traumatic stress disorder (PTSD) and resilience among military children. Following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) group guidelines and utilizing a validated quality assessment tool, this systematic review of reviews incorporated results from more than 35,971 individuals with stress exposures, effects, or disorders and healthy controls. This synthesis found support for important gene, physiology, and environment correlations and interactions that predict increased risk for stressful life events and PTSD, but not direct transmission, among military children. Future research is needed to determine if these constitute indirect pathways of intergenerational transmission in military children.

KEYWORDS intergenerational transmission, military, neurobiological susceptibility, secondary traumatization, stress, systematic review, trauma, veterans

As unprecedented repeat deployment cycles characterizing the global War on Terror take their toll on military families (Smith-Osborne, 2009a), evidence of direct and indirect trauma exposure of military children has been observed (Campbell, Brown, & Okwara, 2011). Earlier clinical report and case study work suggesting the possibility of vicarious post-traumatic stress disorder (PTSD) transmission examined the health status of nonmaltreated children of Holocaust survivors and Vietnam War veterans, as well as the health status of therapists and staff exposed to client trauma (Baranowsky, Young, Johnson-Douglas, Williams-Keeler, & McCarrey, 1998; Rosenheck & Nathan, 1985). These studies employed terms such as *vicarious, empathic,* and *secondary traumatization* and *compassion fatigue* (Figley, 1983, 1995) and used attachment, family systems, and social learning theory to hypothesize possible mechanisms of transmission (Baranowsky et al., 1998). Social learning theory has been used to study intergenerational transmission of other phenomena, such as relationship violence, but has explained only a small part of the variance in these associations (Kwong, Bartholomew, Henderson, & Trinke, 2003).

Studies showing higher levels of PTSD and higher rates of other health and mental health problems among children of Holocaust survivors with PTSD compared to those without PTSD (Yehuda, 2007; Yehuda, Bell, Bierer, & Schmeidler, 2008) suggested early rearing environment interaction with genetic or in utero (via maternal hormones) modes of familial transmission of PTSD risk. One of the few early military studies that used a matched control group found an absence of evidence for transgenerational transmission of trauma, finding that parent-veterans' PTSD symptoms (especially parental withdrawal) showed their primary impact via unhealthy family functioning (Davidson & Mellor, 2001; O'Connor, Caspi, DeFries, & Plomin, 2003), paralleling findings regarding spousal secondary traumatization (Nelson & Wright, 1996; Waysman, Mikulincer, Solomon, & Weisenberg, 1993). A recent systematic review of large prospective studies on the association of unhealthy family relationships and development of psychiatric disorders in children found a causal association with PTSD only for direct maltreatment (Weich, Patterson, Shaw, & Stewart-Brown, 2009). Thus, the literature has failed to support a direct mechanism of intergenerational transmission of PTSD to children, either through social learning or compromised attachment.

Concurrently, twin studies were beginning to examine genetic risk factors for primary PTSD, which could also apply to secondary PTSD (Caspi et al., 2002). Earlier twin studies found approximately 12% genetic PTSD risk directly (Saigh & Bremner, 1998) as well as some genetic influences elevating the likelihood of engagement with or exposure to assaultive traumatic events for a combination of approximately 20% (Norrholm & Ressler, 2009). Recent twin studies have found genetic loading of 19% to 25% for child exposure to corporal punishment but not to maltreatment (Jaffee et al., 2004; Lynch et al., 2006; Wade & Kendler, 2000), and that both types of exposure exacerbate

genetic risk for antisocial behavior. Investigation was also begun of genetic variations (in receptors of oxytocin, vasopressin, dopamine, and serotonin) influencing the neurobiology of human social behavior and social cognition that underpin attachment and social learning (Skuse & Gallagher, 2011). This area of inquiry has been supplemented more recently with molecular genetic studies made possible by technological advances in brain imaging and gene sequencing. The current literature suggests that a range of between 30% and 40% of variance predicting anxiety disorders is heritable (Norrholm & Ressler, 2009), indicating the difficulty of disentangling genetic contributors to risk of military children whose parents have PTSD. For example, some recent individual studies of Holocaust survivors have suggested nongenetic neurological pathways, such as parental stress biomarkers affecting fetal environment, associated with child psychiatric risk (Yehuda, 2011; Yehuda et al., 2008). A single study (Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995) found that a history of pre-trauma-exposure sleep disturbance in adults was associated with increased risk of developing PTSD after trauma exposure.

Further, it is important to note contradictory findings of individual studies in the literature. These suggest that there are groups of military and nonmilitary personnel with well-established diagnoses of PTSD, such as former prisoners of war (POWs) and Holocaust survivors (Freeman et al., 2006; Golier et al., 2002; Yehuda, Spertus, & Golier, 2001), who are resilient to effects of trauma or combat exposure and PTSD diagnosis throughout their life course as demonstrated both by neurological indicators (intact hippocampus, unimpaired neuropsychological testing results) and functional indicators (longevity, lower levels of PTSD symptoms, lower psychiatric comorbidity). Brain imaging studies are beginning to identify genetic polymorphisms that contribute to such trauma resilience at the neurological level, and could have implications for protective factors for secondary transmission of PTSD to offspring. There is also an emerging literature on stress threshold differences and brain structure size gradations that might be associated with risk or resilience for nonmaltreated children developing stress disorder symptoms when experiencing family disruption, corporal punishment, and other family or parenting-based stressors (Ellis & Boyce, 2011; Hagan, Luecken, Sandler, & Tein, 2010; Lynch et al., 2006; Van der Plas, Boes, Wemmie, Tranel, & Nopoulos, 2010). Although the bulk of contemporary military trauma literature has examined earlier draft-era cohorts of military personnel and veterans, there is emerging literature on the all-volunteer forces (AVF) troops who have served since the end of the conscription era, in the post-Vietnam conflicts, in the first Gulf War, and in the War on Terror. However, syntheses of secondary transmission studies are not yet available for AVF military families, and recent syntheses and individual studies of Holocaust survivor families have shown an absence of secondary or tertiary traumatization (Sagi-Schwartz, van IJzendoorn, & Bakermans-Kranenburg, 2008; van IJzendoorn, Bakermans-Kranenburg, &

Sagi-Schwartz, 2003). These several areas of advancing knowledge suggest that a review is indicated of recent research syntheses in another area of influence, the neurological etiology of PTSD in terms of relevance for secondary transmission to military children.

METHOD

### Literature Search and Retrieval Process

Approval for the study was obtained from the Institutional Review Board of the University of Texas at Arlington. The first two authors initiated a systematic review of neuroscience meta-analyses and systematic reviews regardless of publication type from two electronic searches of the following databases from 2000 through 2009: Academic Search Complete, Alt HealthWatch, CINAHL Plus with Full Text, Cochrane & Campbell Library for Meta Analysis/SystemicReview, Health Source: Nursing/Academic Edition, ProQuest, PsycARTICLES, PsycINFO, PubMed, and Social Work Abstracts. Two librarian-assisted mediated searches using the same inclusion criteria and MESH forms of the keywords were performed in MEDLINE. Keywords entered in the first search were meta-analysis/meta analysis, systematic review, neuro*, trauma*, and stress*. Keywords entered in the second search were the same, with the added term child*.

These searches resulted in 841 articles found with the use of identified keywords (Search 1 = 688, Search 2 = 153). A final search of PubMed was performed by the first author using the same keywords with the added terms secondary trauma and intergenerational trauma, with 38 results, for a total of 879 studies. A search for newer syntheses that cited the included studies was performed using the forward citation search function in the ISI Web of Science electronic database, yielding no new results. A manual search of the 2010–2011 volumes of the journal *Development and Psychopathology* yielded one new result. To address publication bias, reference lists of identified research syntheses, conceptual overviews, theoretical reviews, and relevant textbooks were scanned, yielding one new result.

Two researchers, working independently, reviewed the 881 abstracts to determine whether inclusion criteria were sufficiently met to indicate retrieval of the full article. Differences were discussed until consensus was reached. Full-text articles were retrieved for those remaining abstracts, and the same independent review process was repeated.

Inclusion and Exclusion Criteria

Search terms were generated and eligibility of retrieved studies was assessed using concept mapping (Kane & Trochim, 2009; Trochim, 1989) and population, intervention, comparison, and outcome (PICO) criteria, as
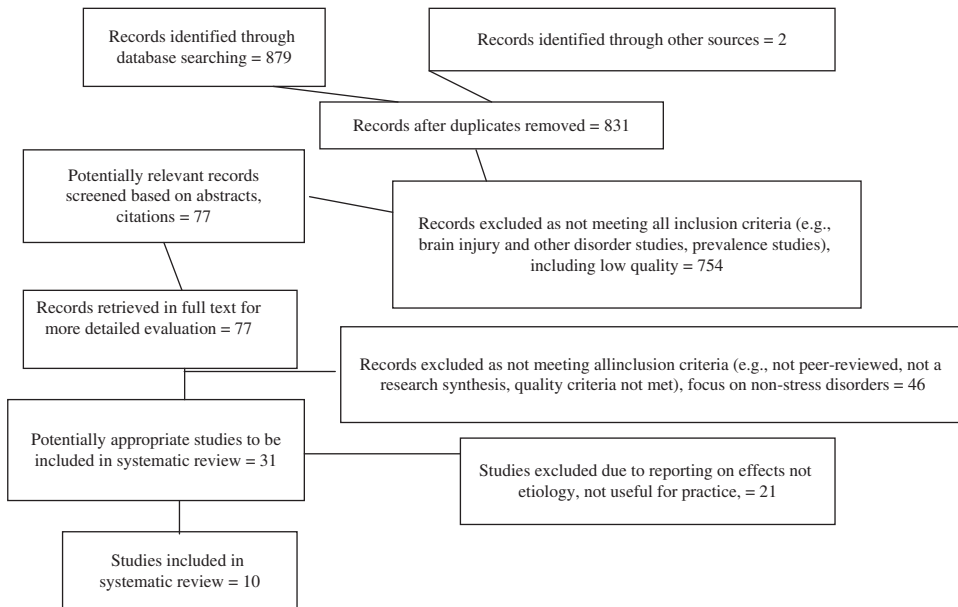
FIGURE 1 Flow chart of resilience instrument validation studies retrieval process following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

recommended by Gambrill (2006). Concept mapping contributes to scientific rigor in maintaining conceptual domain integrity, and the answers to PICO questions define the population under study, the specified condition, the comparison condition, and target outcomes. Inclusion criteria stipulated that the studies be meta-analyses or systematic reviews of neuroscience literature investigating the heritable or transmittable psychobiology of stress and trauma in samples of healthy controls and persons with stress disorders, not simply the characteristics and effects of stress or PTSD on cognition or brain development. Minimum quality criteria for inclusion specified a priori research design, duplicate study selection, and list of included articles. Included studies were published between 2000 and 2009 in indexed peer-reviewed journals and gray literature retrievable in full-text English from electronic databases. A list of excluded articles can be obtained from the first author. Figure 1 summarizes the retrieval process following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher, Liberati, Tetzlaff, Altman, & the PRISMA Group, 2009).

Study Quality Assessment and Data Extraction

Data were extracted following the PICO format as a coding guide following the same process as for search and retrieval. In addition to addressing study quality in the a priori inclusion criteria, we assessed the quality of

the retrieved syntheses using the Assessment of Multiple Systematic Reviews (AMSTAR) tool (Shea et al., 2007), which has established reliability and validity (Shea et al., 2009). See Table 1 for quality assessment of the reviews.

RESULTS

Synthesis of Evidence

Ten reviews met the inclusion criteria for this study, of which eight were meta-analyses and two were nonquantitative systematic reviews (see Table 2 for study characteristics). The included reviews synthesized 1,337 publications and sampled more than 35,971 individuals with stress exposures, effects, or disorders and healthy controls. One synthesis of animal and human studies did not report sample size (Goldstein & Kopin, 2008). No syntheses were located on the etiology of intergenerational transmission of trauma among military families. Evidence for neurological etiological factors relevant to heritability or "transmittability" of trauma vulnerability within military families was found in two or more of the included reviews in the following areas.

GENETIC VULNERABILITY TO MORBIDITY AFTER TYPICAL AND ATYPICAL LEVELS OF STRESS EXPOSURE

Kim-Cohen et al. (2006) synthesized five studies of 3,381 boys that examined the association of a genetic variation (i.e., polymorphism) in one region of the monoamine oxidase A (MAOA) gene with the development of childhood psychopathology after childhood exposure to physical abuse. MAOA is involved in the metabolism of specific stress regulation hormones (i.e., serotonin, dopamine, and norepinephrine). This meta-analysis found that boys with the genotype for low versus high MAOA activity showed the strongest association with a range of childhood onset mental health problems after experiencing physical abuse. Boys with the high MAOA allele showed much less vulnerability after abuse exposure. Authors stated that girls' vulnerability cannot be assessed because girls' MAOA gene expression cannot yet be clearly defined for gene–environment interaction studies.

Kendler and Baker (2007) systematically reviewed 55 genetic studies of 35 environmental measures with established risk for psychiatric morbidity. Their interest was in investigating influences of individual genetics on environmental exposures to contribute to the growing body of evidence on two-way gene–environment interactions in the etiology of illness and health. This line of research (also known as gene–environment correlation or genetic influence on exposure to the environment) has begun to examine community-level variables (e.g., unemployment rate, neighborhood

TABLE 1 Quality of Research Syntheses Found in This Systematic Review of Reviews

Reviews by type	AMSTAR score[a]	No. of studies included	No. control group % of studies	Stressor type and risk factor % of included studies	Biomarker
Meta-analyses					
Chida & Hamer (2008)	10	729	NR	Chronic psychological stress 41.7	HPA/cardio activation
Dickerson & Kemeny (2004)	9	208	NR	Acute psychological stress 11	Cortisol
Goldstein & Kopin (2008)	5.5	60	NA	Physical/social stress 100	Hormone levels
Het, Ramlow, & Wolf (2005)	9.5	16	0	Learning challenge 100	Cortisol
Kim-Cohen et al. (2006)	8	5	NA	Child abuse 100	MAOA genotype
Michaud et al. (2008)	8	140	NR	Psychosocial stress 18.6	Cortisol
Miller, Chen, & Zhou (2007)	9	107	0	Chronic psychological stress 73.8	HPA activation
Woon & Hedges (2008)	8.5	9	0	Child abuse 100	Brain area volume
Systematic reviews					
Kendler & Baker (2007)	7	55	NA	Environmental stress 100	Genetic influences
Kim & Dimsdale (2007)	7	63	28.6	Psychosocial stress 49.2	Sleep characteristics
Overall M (SD)	8.2 (1.36)	139.2 (216.78)	9.2	69.3 (36.31)	

Note. AMSTAR = Tool for Assessment of Multiple Systematic Reviews; NA = not applicable; NR = not reported; HPA = hypothalamic–pituitary–adrenocortical axis; MAOA = monoamine oxidase A.

[a]AMSTAR maximum score is 11; 0–4 = low quality; 5–8 = moderate quality; 9–11 = high quality.

TABLE 2 Research Syntheses Included in This Review

Study design	Sample	Research aim	Stressor/comparison	Measures	Outcome
Chida & Hamer (2008) Meta-analysis of 729 studies	$n = 729$ age = 13–"old age"; gender, race NR	How does acute physiological responsivity change after chronic stress exposure in healthy groups?	Laboratory-induced stress and chronic negative psychological state or trait, life/job stress, psychophysiological fatigue/chronic positive psychological state or trait	HPA, autonomic, and cardiovascular system biomarkers: cortisol, skin conductance, circulatory catecholamines, preejection potential, blood pressure, heart rate	No associations between chronicpsychosocial factor and autonomic system stressor response measures; negative affect with ↓ cardio reactivity/recovery; life stress with ↓ cardio recovery; hostility/aggression with ↑ cardio reactivity
Dickerson & Kemeny (2004) Meta-analysis of 208 studies	$n = 6,153$ gender, age, race NR	What are the physiological effects of various types of stressors in healthy groups?	Laboratory-induced stress with controllable and nonsocial threat/ uncontrollable and social threat	Cortisol	Uncontrollable or social-evaluative threat with ↑ cortisol response and time to recovery
Goldstein & Kopin (2008) Meta-analysis of 60 animal and human studies	NR	What are the responses of the HPA, AHS, and SNS to stress?	AHS stress responses/other systems' stress responses with physiological and social stressors	Plasma epinephrine, corticotrophin, norepinephrine	Differential response of different systems to different stressor categories; AHS/HPA association ↑ AHS/SNS association
Het, Ramlow, & Wolf (2005) Meta-analysis of 16 studies	$n = 563$ age = 18–40; gender, race NR	How do glucocorticoids secreted during stress modulate human memory?	Stress before learning/before retrieval; stress in morning/afternoon	Cortisol	↓ memory morning when cortisol before learning; ↑ memory afternoon when cortisol before learning; ↓ memory when cortisol before retrieval
Kendler & Baker (2007) Systematic review of 55 studies	$n = 3,008$ age = 8–59; gender, race NR	What is the genetic heritability for environmental factors?	General and specific stressful life events or other categories of environmental risk factors	35 environmental measures: life events, parenting, family, social support, peer interactions, marital quality	Heritability ranged from 7–39%; general stressful event heritability averaged 28%; nonassaultive/assaultive traumatic situation heritability averaged 7%/36%; ↑ heritability with age from childhood to adolescence to adulthood

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Kim & Dimsdale (2007) Systematic review of 63 studies	n = 2,293 age = 7–85; M = 679, F = 330; race NR	What is the effect of psychosocial stress on sleep?	PTSD/other stressor types	REM sleep, slow-wave sleep, sleep efficiency, sleep latency, awakening	↓ sleep efficiency and ↑ awakening in PTSD; ↓ slow wave and REM sleep and ↑ sleep efficiency and ↑ awakening with experimental stressors
Kim-Cohen et al. (2006) Meta-analysis of five studies	n = 3,381 age = children and adolescents; all male; race NR	What is the effect of MAOA on childhood psychopathology after physical abuse exposure?	Low/high MAOA activity allele	MAOA polymorphism	Low MAOA allele ↑ psychopathology
Michaud et al. (2008) Meta-analysis of 140 studies	n = 10,976 age = adults, M = 38.6 years (SD = 14); gender = 47% F;race NR	What is the impact of naturalistic stressors on cortisol in health adult humans?	Low/high cortisol release for acute/chronic stress and for medical and sports stressors/job and social stressors	Cortisol	Cortisol release ↑ acute stress and for medical and sports stressors/job
Miller, Chen, & Zhou (2007) Meta-analysis of 107 studies	n = 8,521 age = adults, M = 38.39 years (SD = 16.23); gender = 47% F; race NR	What is the effect of chronic stress on the HPA axis?	Low/high level of HPA activation	Cortisol, glucocorticoid, and other hormones	↑ at stressor onset then ↓ over time, but uncontrollable, trauma, physical stressors show high, flat diurnal pattern; ↑ according to subjective distress; ↓ in PTSD
Woon & Hedges (2008) Meta-analysis of nine studies	n = 347 age = children, M = 11.7 years and adults, M = 37.1 years; M = 151, F = 245; race NR	What are hippocampal and amygdala volumes in persons with PTSD from child abuse and change over development?	Hippocampal and amygdale volume in persons with PTSD/healthy persons	Magnetic resonance imaging of brain volume	↓ bilateral hippocampal volume and left hippocampus no larger than right in adults with PTSD

Note. NR = not reported; HPA = hypothalamic–pituitary–adrenocortical axis; AHS = adrenomedullary hormonal system; SNS = sympathetic nervous system; PTSD = post-traumatic stress disorder; REM = rapid eye movement; MAOA = monoamine oxidase A.

deterioration, crime rate, affordable safe housing), as well as interpersonal variables such as social support (Ellis & Boyce, 2011; Gehlert, Mininger, & Cipriano-Steffens, 2011; Koenen, Amstadter, & Nugent, 2009). The environmental measures in this synthesis evaluated heritability related to general and specific life stressors, parenting (child and parent report), family environment, social support, and marital quality. The heritability of traumatic situation exposure (i.e., associated with PTSD) suggested that the etiologic models for PTSD, despite the diagnostic criterion of a precipitating event, include genetic influences on environmental exposures in addition to gene effects on individual psychophysiological vulnerability. Interestingly, among parenting dimensions, a potential protective factor, parental warmth, showed the highest average heritability across research designs, suggesting that this heritable protective factor might be of greater influence in subsequent generations than the heritable risk factors. See Table 2 for a summary of these research reviews.

TRAUMA RISK BIOMARKERS

Low baseline cortisol levels and dysregulated cortisol amplitude (release and return to baseline levels) have been identified as correlates of certain atypical behaviors and psychiatric disorders, including psychopathy, aggression, and PTSD. Implicated nongenetic cortisol-related mechanisms have been identified as glucocorticoid patterning due to prenatal exposures (i.e., adverse fetal environment) or due to stressful early life experiences (Yehuda, 2007). No research syntheses were found examining cortisol in application to secondary transmission; however, several reviews showed cortisol among possible biomarkers of stress or trauma.

Three reviews (Dickerson & Kemeny, 2004; Het, Ramlow, & Wolf, 2005; Michaud, Matheson, Kelly, & Anisman, 2008) included only adult studies that examined cortisol levels as a stress biomarker, and a fourth (Miller, Chen, & Zhou, 2007) included adult studies with any indicator of hypothalamic–pituitary–adrenocortical (HPA) activations, which included measures of cortisol, glucocorticoid, and other hormones. Another review (Chida & Hamer, 2008) included youth and adult cortisol studies, plus studies that measured sympathetic nervous system biomarkers (e.g., skin conductance, circulatory catecholamines, and preejection period, a heart chamber blood flow measure), a parasympathetic nervous system marker (e.g., heart rate variability), and cardiovascular reactivity markers (e.g., blood pressure and heart rate). These five meta-analyses suggest that HPA reactivity is uniformly reduced in association with positive psychological traits and states, and not only cortisol response patterns but also overall HPA activation (both hypo- and hyperreactivity) vary by type and intensity of stressor. The negative impact of stressor chronicity on cortisol regulation appears to be moderated by these stressor factors, including its level of actual (not

perceived) stressor controllability. One meta-analysis of chronic stress studies using multiple biomarkers (Miller et al., 2007) found that diurnal cortisol output and HPA overall activity is lower in persons with PTSD, consistent with withdrawal, disengagement, and avoidance behaviors found with this disorder. This synthesis also provided further support for the importance of threshold effects, in that levels of subjective distress were positively associated with levels of HPA activation in adults in the absence of a psychiatric diagnosis.

One review (Woon & Hedges, 2008) examined the differences between volume of two areas of the brain, the hippocampus and the amygdala, for persons with PTSD at two different life stages—childhood and nonelder adulthood. Amygdala reductions had been found in a few studies, but most have found no differences between PTSD and control groups. The literature had found that adults with PTSD and trauma-exposed adults without PTSD, but not children with PTSD, showed consistent reductions in hippocampal volume compared to non-trauma-exposed adults (Golier et al., 2002). Those reductions were consistently larger in adults with PTSD than in trauma-exposed controls. Those findings might suggest that an initially smaller hippocampus is a risk factor for developing PTSD after trauma exposure, consistent with the genetic vulnerability findings of Gilbertson et al.'s (2002) twin study.

This 2008 meta-analysis could not rule out this possible risk factor, but did confirm childhood maltreatment as a risk factor for adult hippocampal outcome. The meta-analysis did not find consistent significant effects for children with PTSD, although threshold effects were suggested whereby multiple traumatic exposures or higher severity of PTSD symptoms might be associated with children's hippocampal reduction, but not lower exposure or symptom levels (even given the presence of the diagnosis).

Goldstein and Kopin (2008) examined human (teen and adult) and animal studies of the function of three hormones (i.e., epinephrine, corticotrophin, and norepinephrine) under conditions of a range of types and intensities of physiological and social stressors. Findings supported that stress responses vary by type of stressor as well as intensity of stressor, and are not targeted primarily to "emergency" stressors in fight or flight reactions. In animals and humans, neuroendocrine systems respond differentially based on both the sensory processing of the type and intensity of the stressor and the interpretation of the stimuli in the light of past experience.

Kim and Dimsdale (2007) synthesized sleep characteristics typifying different stress experiences of teens and adults. Their systematic review suggested that short-term or immediate effects of stress exposure on sleep might be similar, whatever the intensity of the stressor. In the short run, stressors, even of trauma intensity, affected sleep by increasing sleep latency and awakening and decreasing sleep efficiency. There were inconsistent findings regarding slow wave sleep, rapid eye movement (REM) sleep, and sleep effects when the stressor was a chronic trauma, and no conclusions were

drawn about pre-trauma-exposure sleep disturbance as a risk factor or bio-logical vulnerability for developing PTSD. A more recent excluded review confirmed that only the single adult retrospective study cited earlier (Mellman et al., 1995) examined sleep disturbance as a risk factor for subsequent PTSD (Babson & Feldner, 2010).

Quality of Evidence

None of the reviews was of low quality (i.e., AMSTAR scores between 0–4), in keeping with inclusion criteria established for study quality. Five were of moderate quality (5–8), and five were of high quality (9–11). The propor-tion of studies in the included reviews in which established neurobiological risk factors for PTSD, acute stress disorder, or traumatization (subthreshold PTSD) were identified as variables was 66%, with the remainder examining lower intensity variables. The overall mean AMSTAR score for the system-atic reviews included in this study was 8.2 on the 11-point scale ($SD = 1.36$). The minimum standards for the research designs of the studies included were specified in all of the reviews. A high proportion of the appropriate research designs in the included reviews utilized control groups. Three reviews that included studies with experimental research designs did not report control groups. In the one review that reportedly included studies with no control group, those studies made up 28.6% of the total.

DISCUSSION

The syntheses examining cortisol and other hormonal biomarkers suggest distinct neurological patterns for stressors other than trauma compared to trauma exposures, whether acute or chronic, in both children and adults. These findings tend to support the conclusion that, on the neurobiolog-ical level, children's experiences of parental trauma are qualitatively, not just quantitatively, different from primary trauma exposure. Those findings are consistent with the literature suggesting that PTSD psychophysiology is distinct from fear conditioning, social learning, and stress response models (Etkin & Wagner, 2007; Hagan et al., 2010; Johnson, Delahanty, & Pinna, 2008; Levin & Nielsen, 2007; Orth & Wieland, 2006).

Kim-Cohen et al. (2006) and Woon and Hedges (2008) examined direct trauma exposure in the form of abuse and potential genetic or neurodevel-opmental protective factors for traumatized children. The 2006 meta-analysis found a polymorphism that might protect traumatized children from devel-oping PTSD. The 2008 meta-analysis found differences between adults and children with maltreatment-related PTSD that might point to a process of hippocampal atrophy in aging over the life span or alternatively, that intact hippocampi in children with PTSD might indicate resilience conferred by the greater level of neuroplasticity in young brains or for those children with

lower levels of exposure or symptoms. Any of these mechanisms underscore the potential value of effective early treatment for those with the diagnosis, and the benefit not only for specific symptom relief but for overall cognitive function and affective regulation. Their findings with regard to hippocampal reduction in trauma-exposed but healthy control group participants, as well as Miller et al.'s (2007) findings of graduated threshold effects of subjective distress on HPA activation in adults, could support the utility of prevention resources in the absence of clear evidence for secondary trauma transmission. Intervention programs like Camp C.O.P.E. demonstrate potential to enhance resilience and protect the brain development of military children who do not have PTSD or subclinical symptoms, but might exhibit distress or behavioral issues that do not require clinical diagnosis and treatment. Cycles of such symptoms associated with deployment cycles, independent of other morbidity, have been noted in military children (Cozza, Chun, & Polo, 2005; Drummet, Coleman, & Cable, 2003; Huebner, Mancini, Wilcox, Grass, & Grass, 2007; Lincoln, Swift, & Shorteno-Fraser, 2008; Mmari, Roche, Sudhinaraset, & Blum, 2009).

The synthesis also points to possible enhancements of military family interventions like Camp C.O.P.E. Pretrauma sleep disturbance, indicated as a possible PTSD predictor, could be emphasized among the target behaviors addressed. More differential stress controllability appraisal skills training could be linked to situation- and stressor-specific active versus alternative coping strategies, preferentially to general skills training. As our knowledge of phenotypes becomes more sophisticated, selected activities within an intervention program can be directed to children in the "best fit" or highest need phenotypes. For example, a recent nonincluded meta-analysis found that dopamine-related alleles might be associated with heightened reactivity to both adverse and enhanced environments (Bakersman-Kranenburg & van IJzendoorn, 2011). Hypothetically, then, in addition to the baseline program, children with that indicated phenotype (e.g., the susceptible dopamine allele plus adverse or less positive environment) could receive complementary or adjunct therapies known to affect emotional regulation such as equine-assisted activities (Smith-Osborne & Selby, 2010; Selby & Smith-Osborne, 2013). Their parents could be provided the positive disciplinary practices and parent affect regulation group, whereas parents of children with the high MAOA activity allele, found resistant to adverse family context (Kim-Cohen et al., 2006), could be provided the relationship enhancement or deployment cycle management group.

FUTURE DIRECTIONS

These reported advances in neuroscience research on the etiology of PTSD have been directed more to both extremes of stress exposure than to the

range that might be most relevant to investigating secondary trauma: parenting and family stressors that do not include direct maltreatment, but are related to the presence of PTSD in the family. For example, in pursuing neurobiological evidence of stress effects on military children, future research can include genetically and neurologically informed designs that examine mechanisms of transmission indicated by the control groups of extant studies of PTSD effects. Research syntheses of studies using both trauma-exposed and non-trauma-exposed healthy controls have suggested that the former have smaller hippocampal volumes compared to nonexposed controls, whether or not they have PTSD (Karl et al., 2006; Woon & Hedges, 2008). Thus, hippocampal volume could be used as a biomarker of potential lower level trauma exposure via secondary transmission for military children without PTSD who have been exposed to parental PTSD, but not to direct trauma. Randomized clinical trial designs to enhance investigation of causal relationships will be necessary for such studies, particularly to assist in teasing out the associated decrements in relationship quality (Lauterbach et al., 2007) or family environment (Caspi & Moffitt, 2006; Yehuda et al., 2008).

Further research should also explore neurological effects of parental PTSD on children in terms of both the hyporeactivity (withdrawal and avoidance) aspects and the hyperreactivity behavioral aspects drawing salient biomarkers from the extant primary literature on PTSD psychophysiology. For example, an excluded meta-analysis of PTSD studies using noncortisol and hormonal measures found PTSD to be associated with elevated psychophysiology (Pole, 2007). One adult prospective study to date (Guthrie & Bryant, 2005) has also found higher levels of some of these same measures pretrauma exposure to be predictive of posttrauma stress severity. These measures—skin conductance, electromyography, and heart rate response to auditory startle stimulus and idiographic trauma cues—could be used in genetically and neurologically informed designs to investigate military children's response to parental trauma cues and children's rates of physiological recovery. Longitudinal designs using these measures will be helpful in elucidating temporal stability of cumulative risk and protective factors across childhood developmental stages (Smith-Osborne, 2007, 2009b) and the changing environments of military children.

Interventions for military children of deployed and injured War on Terror personnel are already being piloted. An example of one that shows some consistency with these neuroscience findings is Camp C.O.P.E., which provides military children with age-appropriate therapeutic interventions in small groups of their peers who have had similar experiences. Eligibility for services is not based on diagnosis. Services are also provided for their parents to help them to help their children at home. Camp C.O.P.E. allows the children to tell their own stories and to put a voice to their feelings and concerns, while providing new ways to cope with their experiences. The Camp

C.O.P.E. curriculum consists of four themed sessions corresponding to the C.O.P.E. acronym:

- C for courage, to embrace and accept the changes in their lives.
- O for optimism, the opportunity to see that it is tough right now but things will get better.
- P for patience, because it will take time to adjust to the changes and see improvements.
- E for encouragement, to help themselves and to help others in similar situations.

Participants are divided into small groups based on age. Each session consists of a large-group discussion, a small-group activity, and an energizing activity called a "COPE-estenic." The therapeutic interventions are specifically designed to teach and encourage healthy coping skills, identification and expression of feelings, grief and loss processes, empathy building, self-esteem boosting, nonthreatening facilitation of debriefing, and therapeutic play. Camp C.O.P.E. puts the emphasis back on the children, sending the message that they are an important part of the family and they have served, too. While the children are attending Camp C.O.P.E., their parents are invited to participate in either a parenting support class or a relationship enrichment class. During the parenting support class, the parents learn to better understand and deal with the different behaviors their children might be exhibiting, and the techniques to help reinforce the skills the children are learning in their groups. Attendees of the relationship enrichment class are provided information and interventions designed to strengthen their interaction with their spouse or significant other.

During the camp process, camper's parents or guardians complete inventories of their overall family functioning and their child's emotional and behavioral states at the time of camp registration. Three months after participation in Camp C.O.P.E., parents are asked to complete posttest inventories. In an initial evaluation of the intervention using Army parents' report ($n =$ 36 child–parent pairs), child participants were not found to meet criteria for PTSD or symptoms. The data indicate children and adolescents experience less distress and improved behavior after being provided the Camp C.O.P.E. curriculum interventions (Ward & Alvarado, 2011).

This exemplar program is consistent with the synthesized neuroscience evidence of this study in several aspects. Children who have been exposed to a specific stressful, but not traumatic, life event are provided with a brief intervention that aims to engage several environmental as well as intrapersonal dimensions in the service of active stress coping with controllable stressors and concomitant dearousal and alternative coping methods for uncontrollable stressors. The intervention activities target stressor appraisal

functions, peer and parental social support, family and unit (defined as other military families) cohesion, and parental and child emotional regulation with particular attention to levels of subjective distress. The synthesized evidence suggests that military personnel might bring increased bidirectional genetic liability for stress exposure, thus supporting the importance of interventions like Camp C.O.P.E, which are offered for the general population of military families, not only for bereaved families or for those who have a parent with PTSD. This evidence supports interventions that attend to threshold effects of stress exposure, to levels of subjective distress, to the differential neurobiological response to different types of stressors as interpreted based on experience, and to the possible interaction of neuroplasticity and differential genetic vulnerability with environment during childhood. The preliminary program evaluation findings for Camp C.O.P.E are consistent with this study's lack of support for direct intergenerational transmission of trauma, but support for important gene and environment correlations and interactions that might predict increased risk for stressful life events and PTSD precipitants among military children, but also increased neuroplasticity to respond to environmental relief of subjective distress (Belsky et al., 2009). The results of this review of reviews might have utility in informing the further development of prevention programs such as Camp C.O.P.E, and in turn the evaluations of such programs (Johnson, 2001) could elucidate the mechanisms of stress effects among families of service members diagnosed with PTSD and comorbid conditions.

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