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Intersection of Stress, Social Disadvantage, and Life Course Processes: Reframing Trauma and Mental Health

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Abstract

This paper describes the intersection of converging lines of research on the social structural, psychosocial, and physiological factors involved in the production of stress and implications for the field of mental health. Of particular interest are the stress sensitization consequences stemming from exposure to adversity over the life course. Contemporary stress sensitization theory provides important clinical utility in articulating mechanisms through which these multiple levels exert influence on mental health. Stress sensitization models (a) extend understanding of neurobiological and functional contexts within which extreme stressors operate and (b) make clear how these can influence psychologically traumatic outcomes. The value of interventions that are sensitive to current contexts as well as life course profiles of cumulative stress are illustrated through recent treatment innovations.

Keywords

Mental health; Neurobiology; Poverty; PTSD; Stress; Trauma

The field of mental health is currently at the intersection of converging lines of research on the social structural, psychosocial, and physiological factors involved in the production of stress. This convergence holds tremendous implications for the future development and refinement of mental health research and practice. We focus here on a specific stratum in this convergence, perhaps best captured by “stress-sensitization” models. In these models, early life course stress is seen to increase a broad array of subsequent mental health problems, both by increasing vulnerability to stress in later life and through the cumulative erosion of health and stress-buffering capacities. The theories we will focus on reflect efforts to integrate the study and application of psychological processes, societal structures, and biomedical sciences; a perspective contending that individual-level processes and social

structural contexts both need to be addressed to successfully anticipate, prevent, and treat mental health disorders associated with recurrent life course stress.

In this paper, we present the benefits of a cumulative-effect and context-sensitive formulation of the roles of stress in relationship to trauma and pathways to psychopathology. Trauma as manifested by posttraumatic stress disorder (PTSD) is one among only a few psychiatric conditions wherein specific experiences can be explicitly linked to disorder etiology. However, there is remarkable variation in emotional and neurobiological response to specific stressors and cognitive appraisal, coping, and support factors are pivotal to explaining this variation (Olf, Langeland, & Gersons, 2005). Moreover, emerging currents in stress research point to the involvement of relatively distal, chronic, and even diffuse adversities in taxing and eroding stress response capacity, contributing to psychological wounding and setting the stage for what might otherwise be preventable traumatic impact of extreme stress exposure.

Although long appreciated as factors in the etiology of physical and mental health problems, early life stress and chronic conditions of adversity such as poverty, abuse, and discrimination are gaining increasing scrutiny as incremental roots of later health and behavioral health disparities. A scientific consensus is emerging, directing attention to cumulative adversities and disruptions as targets for the prevention of adult disorders, with particular attention to early life exposures (Shonkoff, Boyce, & McEwen, 2009). This view argues that it is not only the dramatic stressful events of life that exact their toll, but also the cascade of chronic psychosocial stress and consequent neurological and physiological dysregulations that act as catalysts of accelerated wear and tear on coping systems and triggers of psychopathology and illness trajectories (Juster, McEwen, & Lupien, 2009).

To contextualize these life course models and their implications for later mental health, we first focus on stress theory as an etiological tool in linking macro forces to individual effects, a bridge toward a more unified view of how physical and mental health are eroded. We build upon advances in understanding biological mechanisms that are theorized to carry risk from stress-producing experiences to later mental illness through allostatic load and key findings regarding the impact of early life stress exposure on subsequent mental illness. *Allostatic load* refers to the strain imposed on multiple intersecting physiological systems when repeated episodes of adjustment are activated in response to repeated, prolonged exposure to stress (McEwen, 1998; Nurius & Hoy-Ellis, in press). The extent of these wear-and-tear or “weathering” effects are demonstrably greater among low income and minority communities, leading to accelerated aging and health disparities, including mood disorders (Geronimus, Hicken, Deene, & Bound, 2006; Kapczinski et al., 2008; Simon et al., 2006).

We next turn our attention to socioeconomic status as a pivotal, sustained contextual factor in the production of both adversity and mental illness. We illustrate creative inroads in new context-sensitive trauma interventions that extend current best practices and conclude with a look at the transdisciplinary collaboration and team science preparation necessary for the next generation of mental health research and practice.

STRESS THEORY AS AN ETIOLOGICAL BRIDGE

Historically, the experience of stress has been linked to subsequent psychopathology via specific acutely stressful events from which clear temporal and causal pathways can be drawn. Stressful events that overwhelm an individual's capacity to cope, such as physical assault, witnessing violence, involvement in natural disasters, and other acutely threatening circumstances can lead to states of psychological trauma and subsequent mental illness. Although resilience and even growth in the aftermath of trauma are more common than not, histories of experiencing discrete, highly stressful life events are common among those burdened with significant mental health and behavioral problems. The construct of psychiatric trauma is explicitly defined in terms of adverse environmental conditions that overwhelm individuals' capacity to cope, yielding stress response syndromes and disorders such as PTSD.

Epidemiologic data suggest that it may take years for trauma-exposed individuals with PTSD and related comorbid conditions to enter treatment (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Effective trauma-focused interventions face the challenge of incorporating both patient-centered supportive care and evidence-based treatment targeting PTSD and related comorbidities (Hobfoll et al., 2007; National Institute of Mental Health, 2002; Roberts, Kitchiner, Kenardy, & Bisson, 2009a; 2009b; Tuma, 2007; Zatzick & Galea, 2007). Intervention models that link exposed individuals to evidence-based services are yet to be widely implemented (Hobfoll et al., 2007; National Center, 2006; National Institute, 2002).

Stress theory provides critical clinical utility in articulating environmental circumstances, factors affecting individual-level neurophysiological "structures" and functional health, and describes the linkages between physical and mental health in the context of cumulative stress (Thoits, 2010). From a biological perspective, psychiatric disorders "are illnesses like any other; they are diseases of the body, specifically the brain" (Schwartz & Corcoran, 2010, p. 82). Results from stress physiology research have enabled researchers to break away from a physical-and-mental-disorders dichotomy that has emphasized different social risk factors and causes with mental health presumably deriving fairly exclusively from psychological phenomena, ending in the mind rather than in the body.

Movement now is toward a more integrated characterization of braided etiological pathways; stress theory reveals ways that psychiatric disorders also have biological representations, and they intersect with etiologies of physical disorders. This advance is spawning consideration of new combinations of generally unrecognized connections between diseases such as coronary artery disease and depression: comorbid, but both linked to common social risk factors. In more general terms, the physiology of stress may suggest types of social risk factors that could be most fruitfully explored for specific constellations of disorders (Schwartz & Corcoran, 2010). Thus, when psychiatric disorders are seen as biological phenomena, new combinations of diseases suggest themselves. For example, coronary artery disease and depression are comorbid, and both have been etiologically linked to at least one common social factor: occupation of a job in which the worker exerts little direction, control or ability to plan (e.g., see Karasek et al., 1988; Link, Lennon, &

Dohrenwend, 1993; 1998). This pattern raises the question: Do adverse occupational conditions lead to negative physiological changes common to both coronary disease and depression?

With anomalous, acute stressful events in which temporal links to subsequent mental health impairment are relatively traceable, clinical interpretations of trauma and pinpointed interventions are well supported, for example, events such as assault or combat and later PTSD. Effects of chronic adversities, particularly those stemming from early life, require additional theorizing regarding carriers of mental health risk and mechanisms of effect. Stress sensitization and stress proliferation processes are two such examples.

Stress sensitization theory posits that repeated stress early in life serves to dysregulate stress response systems, eroding the threshold for reactivity and adaptive responding to later stress and increasing the risk of subsequent mental health disorders (Dienes, Hammen, Henry, & Daley, 2006; Shonkoff et al., 2009). Stress sensitization models have been elaborated through explanations of the impact of early life chronic stress on brain structures and functioning, such as the frontal cortex and limbic system. Early points of development appear to entail windows of vulnerability during which times some brain functions are particularly susceptible to stress, which can interrupt or negatively modify normal brain development, leading to both contemporaneous as well as more subterranean or latent effects (Lupien, McEwen, Gunnar, & Heim, 2009) and negative cascades through interlocking neurobiological systems responsive to stress. (Cohen et al., 2006; Teicher et al., 2004).

EARLY LIFE EXPOSURES AND LATER MENTAL HEALTH

To improve understanding of the effects of stressors and stress sensitization along with the environmental interactions with biology, researchers must adopt some form of a life course perspective that incorporates early childhood and cumulative effects of stress. Increasingly, findings regarding complex forms of biological embodiment of early exposures are pointing to early life experience as pivotal to preventing mental illnesses such as depression, anxiety, substance abuse, aggression, suicidality; personality disorders (Afifi et al., 2011; Anda et al., 2006; Schilling, Aseltine, & Gore, 2008); and chronic physical conditions such as cardiovascular disease and diabetes (Cohen, Janicki-Deverts, Chen, & Matthews, 2010). A focus on single stressors is yielding to multiple stressor analysis—demonstrating moderate to high intercorrelation among stressors and significant graded relationships between number of stressors and likelihood of adverse outcomes (Felitti et al., 1998; Finkelhor, Ormrod, Turner, & Hamby, 2005; Kessler, Davis, & Kendler, 1997). These more comprehensive assessments have documented higher levels of multiple adverse childhood exposures than previously realized (Pirkola et al., 2005).

Cumulative, multiform exposures negatively impact mental health both in adolescence (Dube et al., 2001; Turner, Finkelhor, & Ormrod, 2006) and in adulthood (Afifi et al., 2008; Edwards, Holden, Anda, & Felitti, 2003; Hooven, Nurius, Logan-Greene, & Thompson, 2012; Turner & Lloyd, 1995) with respect to psychiatric problems such as depression, anxiety, anger, aggression, alcoholism, drug use, and trauma spectrum disorders. Based on

discrete time survival models, Benjet, Borges, & Medina-Mora (2010) have provided a unique test of chronic adversities predictive of four classes of psychopathologies (mood, anxiety, substance use, externalizing) over three life stages—childhood, adolescence, adulthood. Adversities included family pathology (parental mental illness, addiction, violence, and criminality), maltreatment (neglect, physical and sexual abuse), interpersonal loss (parental death, divorce, other loss), and physical illness and poverty. In addition to these adult Axis I mental disorders, cumulative childhood adversities have also been demonstrated to show robust association with adult Axis II personality disorders within both clinical and normative samples (Afifi et al., 2011). Although childhood neglect and violence exposure (witnessing, abuse) were most broadly associated with significantly increased odds of having an adult personality disorder, parental history of mental health problems, such as suicide attempt, mental illness, incarceration, and substance abuse, also manifested significantly higher odds of several forms of later personality disorders.

The effect of number of adversities has generally been treated as linear—odds of disorders increase with elevated number of adversities. Some nonlinear relationships are emerging, however, one being a general ceiling effect—once a certain number of adversities are experienced, additional adversities add impact, but at a decreasing rate (Benjet, Borges, & Medina-Mora, 2010). Recent analysis is also arguing for disentanglement of exposure to higher cumulative adversity relative to more severe events, and observations that more “toxic” stress exposure tends to be embedded in conditions of social disadvantage and multiple-form adversity (Benjet et al., 2009; Schilling, Aseltine, & Gore, 2008). In the above-noted research (Benjet et al., 2010), for example, although all adversities were predictive of a mental health disorder, family pathology and maltreatment were the strongest and most consistent predictors of psychopathologies, and these results were found across all three life stages.

Pearlin and colleagues (Pearlin, Schieman, Fazio, & Meersman, 2005) urge attention to the phenomenon of stress proliferation—a process through which individuals and populations exposed to serious adversity are at risk for later exposure to additional adversities—mechanisms through which stress begets stress (Felitti et al., 1998). Proliferation can occur through various factors—among the obvious being that lower socioeconomic status places individuals in conditions (homes, neighborhoods, communities, environments) in which they are more likely to be exposed to dangers and hazards. Individuals from minority populations are more vulnerable to exposure to discriminatory and hate-based experiences as well as to certain unique stress experiences, for example, daily exposure to exclusionary and oppressive practices for racial minorities; coming out or being outed among sexual minorities (Edwards et al., 2006; Hatzenbuehler, 2009). Exposure to initial stressors can lead to secondary effects, such as harmful effects on self-concept, ability to form and sustain healthy relationships, academic achievement, decision making, and a range of other characteristics that leave individuals vulnerable not only to subsequent stressors but also to limited buffering of those stressors and greater impairment relative to individuals without prior significant adversity exposure.

Concern about the mental health–jeopardizing effects of stress proliferation has fueled research linking social disadvantage, early childhood adversity exposure, and physical and

mental health outcomes. We turn next to connections between social disadvantage (lower socioeconomic status) and physical and mental health inequalities, connections that are at least partially mediated by differential profiles of exposure to stressors.

SOCIAL DISADVANTAGE AND MENTAL HEALTH

Studies of psychiatric disorders in the general population have given rise to one of the most persistent findings in psychiatric epidemiology: The highest overall prevalence rates of psychiatric disorders consistently occur among persons of lowest socioeconomic status (Dohrenwend, 1998; Goodman, McEwen, Dolan, Schafer-Kalkhoff, & Adler, 2005; Kessler, Davis, & Kendler, 1997). Individuals with lower socioeconomic status are at greater risk of acquiring emotional disorders compared with their higher socioeconomic status counterparts (Gallo & Matthews, 2003; Lorant, Deliege, Eaton, et al., 2003). The margin is by no means a narrow one: Epidemiological evidence suggests psychiatric illness rates double among low socioeconomic persons relative to their high socioeconomic status counterparts (Holzer et al., 1986), with the highest rates of multiple psychiatric disorders (three or more) found among those in the lowest socioeconomic groups (Dohrenwend, 1998; Kohn, Dohrenwend, & Mirotznik, 1998; Kessler et al., 1995).

Debate regarding the causal nature of this robust association has evolved over time, spanning assumptions of inheritance of mental illness in the pauper class during the mid-1800s, to sobering recognition of environmental effects across class strata in contexts such as the Great Depression and World War II, during which extreme environmental stress produced severe psychopathology in previously normal people, to a more recent Zeitgeist emphasizing the interplay between biological and environmental factors (Dohrenwend, 1998). Social causation explanations hold that disorder rates are higher in lower socioeconomic groups because of greater environmental adversity—such as stressful living conditions, roles, and relationships that overwhelm coping capacity (Horwitz, 2002; 2010). Mental health and mental illness are viewed as at least partially a consequence of basic aspects of social organization (Pearlin, 1989), including degree of social integration and inequality (Horwitz, 2010), reflecting complex relationships associated with social stratification, including gender, race, and life stage (Eaton, Mutaner, & Sapag, 2010).

The health/wealth gradient is most intuitive for those who live in conditions of persistent poverty. However, evidence suggests that disparities are evident across the ranks of social class. That poverty and lack of health care alone cannot account for this distribution has focused attention on understanding how psychosocial factors interact with social class factors and psychological health. The social patterning of psychological disorders is a more variable picture than that of physical health, such as chronic conditions. The clearest pathway from socioeconomic status to mental health status appears to be through exposure to multiple risk factors, the effects of which are often exacerbated by an impoverished fund of protective factors that might otherwise buffer stress (Evans & Kim, 2010).

Lower standing on the social hierarchy tends to place individuals in different settings than their higher status counterparts. Those settings tend to be characterized by higher levels of both physical and social forms of environmental stress and toxins. Examples include

environmental pollutants, crowding, and chronic noise as well as relatively high levels of crime, violence, discrimination, and conflict, with cascading circumstances or events such as job loss, high conflict within families, maltreatment, loss of family members, residential instability, and developmental disruptions. Individuals with lower socioeconomic status encounter more numerous and frequent chronic stressors and negative life events in both adulthood (Lantz, House, Mero, & Williams, 2005; Turner & Avison, 2003) and childhood (Attar, Guerra, & Tolan, 1994; Deater-Deckard, Dodge, Bates, & Pettit, 1998), and are more likely to interpret ambiguous events as stressful (Chen & Matthews, 2001). Moreover, lower socioeconomic status contexts tend to be associated with comparatively fewer opportunities for personal control, autonomy, valued social participation, and positive recognition and affirmation—contributing to social patterning of mental health (Marmot, 2006).

As noted earlier, allostatic load associated with chronic stress progressively distorts stress response mechanisms, catalyzing mental health problems. Running in tandem, many behaviors used to cope with stress (e.g., smoking, drinking, using drugs, overeating, risk taking) contribute to insidious patterns of maladaptation—patterns that converge with neurobiological factors to more deeply entrench psychopathology (Adler, 2009; Jackson, Knight, & Rafferty, 2010). Subjective perception of one's social standing also appears to convey risk. Children with low socioeconomic status, for example, have demonstrated awareness of their lower social standing, which negatively shapes self-perceptions, fostering negative beliefs and evaluative emotionality such as poor self-esteem and shame (Dickerson, Grunewald, & Kemeny, 2004) and depression, even after controlling for objective socioeconomic status (Adler, 2009). These multiple family disadvantages carry forward to predict risk of major adult psychopathology such as depression (Sandowski, Ugarte, Kolvin, Kaplan, & Barnes, 1999).

Socioeconomic status is similarly, although inversely, related to protective factors that might otherwise limit stress exposure and buffer or attenuate its effects. Recent theorizing provides the frame of reserve capacity—referring to the forms and extent of tangible, interpersonal, and intrapersonal coping resource (Matthews, Gallo, & Taylor, 2010). Low socioeconomic status individuals tend to have more impoverished reserve or coping capacity both because their circumstances limit their sources and ability to develop and replenish resources, their exposure to greater stressors requires them to use up what they do have, and persistent stress exposure tends to undermine what may have been previously available resources. A relative paucity of coping reserves leaves lower socioeconomic status individuals more likely to develop more impaired stress reactivity, including more negative emotionality (depression, anger, anxiety) and distorted cognitive responding (Matthews, Raikkonen, Gallo, & Kuller, 2008). Length of exposure to poverty in childhood is significantly predictive of children's experience of behavioral problems (Reid, Macchetto, & Foster, 1999). Integrative models are increasingly demonstrating pathways from early-life adverse experiences, later-life socioeconomic well-being, mental health, and the value of assessing both developmental life course stress and social determinant perspectives (Macmillan, 2009; Nurius, Logan-Greene, & Green, 2012; Zielinski, 2009).

INTERVENTION INROADS: A CASE EXAMPLE FROM ACUTE CARE MEDICAL SETTINGS

We describe below an example of an intervention model that incorporates insights from the stress proliferation literature to effectively treat severely injured adults with PTSD symptoms in the wake of injury. The intervention model was developed by a team of psychiatric and mental health specialists led by coauthor Douglas Zatzick, medical director of a consultation-liaison psychiatry service at a university-based, Level 1 urban trauma medical center in the Pacific Northwest. The trauma center's hospital serves approximately 6,000 adult and pediatric inpatients annually in a four-state region, and it is the only medical center in the region qualified to care for the most severely injured patients. As its Level 1 designation suggests, the trauma center provides intensive, around-the-clock care for injured patients, from resuscitation through rehabilitation, and employs a wide array of medical professionals including emergency medicine doctors, nurses, surgeons, and surgical subspecialists.

Urban trauma center patients include a substantial proportion of economically and socially disadvantaged individuals. One recent nationwide prospective cohort study reports that approximately 30% of injured trauma survivors admitted to urban trauma centers have no insurance and approximately 40% report family household incomes of less than \$30,000 per year (Zatzick et al., 2007). These patients are also highly racially and ethnically diverse: Previous clinical epidemiological investigations found that approximately 1 in 10 randomly sampled injured trauma survivors were monolingual, non-English speaking, with over 40 languages spoken in the cohort (Santos et al., 2008; Stephens et al., 2010).

Severely injured persons are at high risk for developing PTSD, a psychiatric syndrome brought on by exposure to an overwhelming, traumatic life event such as a motor vehicle crash, physical assault, or traumatic experience in a war zone or as a refugee (American Psychiatric Association, 2000). Approximately 20% to 40% of the trauma center's patients are estimated to develop diagnosable symptoms of PTSD during their hospital stays, including intrusive symptoms that impair normal social functioning (e.g., memories or nightmares of the trauma), avoidant symptoms (e.g., avoiding reminders, social isolation), and arousal symptoms (e.g., poor concentration, startle response).

INCLUDING A FOCUS ON ADVERSITY AND LIFE CONCERNS IN TRAUMA INTERVENTION

The Zatzick et al. (2011) intervention was designed to ameliorate historical challenges to successful treatment of severely injured trauma center patients suffering from postinjury PTSD, including and especially those from socioeconomically disadvantaged backgrounds. Those challenges include, first, difficulty in engaging individuals in treatment: Early trials targeting PTSD symptoms consistently describe difficulty engaging and retaining acutely injured patients in psychopharmacological and psychosocial intervention protocols (Roy-Byrne et al., 2004). Epidemiologic data suggest that it may take years for trauma-exposed individuals with PTSD to enter treatment (Kessler et al., 1995). Intervention models that

serve to initially engage and then link injured trauma survivors to evidence-based PTSD services are therefore key in the early mental health response to trauma exposure. The second challenge would be the ineffectiveness of earlier brief interventions in acute care medical settings: Hospital patients receiving some forms of brief, provider-initiated psychosocial interventions in acute care medical settings, such as mandatory psychological debriefings and family participation in end-of-life decision making, have demonstrated worsening PTSD symptomatic outcomes in the days, months, and years following hospital admissions (Bisson, Jenkins, Alexander, & Bannister, 1997; Mayou, Ehlers, & Hobbs, 2000).

Poor treatment outcomes and poor engagement have particularly plagued efforts to treat traumatically injured patients from socioeconomically disadvantaged backgrounds. Moreover, in line with the general evidence regarding the effects of historical and cumulative adversity on psychiatric symptoms, epidemiological evidence suggests that a history of cumulative adversity places an injury survivor at greater risk for the development and prolongation of postinjury PTSD symptoms (Ramstad, Russo, & Zatzick, 2004).

In seeking to develop a treatment model that would effectively engage and treat socially disadvantaged injury survivors, Zatzick's team incorporated insights from the literature on cumulative life stress and adversity in addition to traditional sources on evidence-based treatments for PTSD and other psychiatric syndromes. Indeed, the need for clinicians to consider the association between poverty, cumulative and current adversity, and the individual's experience of psychiatric symptoms is increasingly noted in the trauma literature. Trauma experts in fields ranging from domestic violence to natural disasters relief point out the disproportionate deleterious effects of trauma on economically disadvantaged populations—and call for the assessment and amelioration of social and material concerns of survivors as crucial and a first step in intervention.

In accordance with these insights, one early step taken by Zatzick's team was to develop and field test early posttraumatic care management interventions responsive to social contextual factors, including recurrent traumatic and stressful life events and financial constraints related to enduring poverty (Zatzick et al., 2001; Zatzick, Roy-Byrne, et al., 2001; Zatzick et al., 2007). Care management interventions are tailored to survivor-identified concerns affecting recovery.

The team's methodology for eliciting and testing consumer and survivor-identified life concerns has been described in previous publications (see Zatzick et al., 2007). Here we note two ways in which the introduction of the life concerns methodology has been productive to the intervention model. First, the field test produced a feasible technique for eliciting survivor concerns that was acceptable and easily integrated into the intervention model (Zatzick, Kang, Hinton, Kelly, Hilty, Franz, Le, & Kravitz, 2001; Zatzick, Roy-Byrne, et al., 2001). Second, systematically collected pilot data on consumer and survivor-generated concerns narratives from the point of hospitalization to 6 months postinjury provided insight into the ways in which cumulative adversity and poverty intersect with acute injury, at least from the person's perspective—and led to specific enhancements in the intervention model. The analyses of concern narratives clearly revealed that survivors' stress-related concerns

span multiple life domains, including but extending considerably beyond the realm of PTSD symptoms. Financial worries, exacerbated by the current injury but related to enduring poverty, were among the most often recounted concerns (Zatzick et al., 2001; 2007). Longitudinal pilot data also provided the team with a better understanding of the specific ways in which preexisting financial difficulties and cumulative and continuing life stressors can impact and exacerbate postinjury PTSD symptoms (Uehara & Zatzick, 2002).

Findings from the analysis of concerns were then used to refine the team's care management procedures. Originally conceived as a strategy to assess and treat trauma survivors' co-occurring conditions such as depression, care management was reenvisioned as a strategy to more inclusively assess and target life course and current stressors as well. Intervention was expanded to include tangible support, such as assistance in obtaining public insurance funding to cover injury expenses. Care management interventions were also modified to anticipate recurrent traumatic and stressful life events, including reduction of harm from high-risk behaviors such as alcohol consumption and interventions specifically targeting housing instability, financial deprivation, and domestic violence and other relational conflict and stress.

THE STEPPED COLLABORATIVE CARE MODEL

Zatzick's intervention model is perhaps best described as a stepped, collaborative care model composed of three core elements: a collaborative care platform, evidence based treatment for PTSD and co-occurring psychiatric symptoms, and early identification and case management of adverse life concerns as identified by the trauma survivor. The model's collaborative care strategy ensconces mental health intervention into the hospital setting, allowing for the simultaneous targeting of medical/surgical and psychiatric conditions within the trauma surgical ward. This dual targeting of medical and psychiatric conditions incorporates the trauma center's frontline mental health specialists at an early stage of care and serves to organically link trauma center and outpatient mental health service delivery (Zatzick et al., in press; 2004).

In step one, masters level care managers employ life concerns elicitation and amelioration techniques referenced earlier to engage patients and address immediate issues and needs (Zatzick et al., in press, 2001, 2007). Before evidence-based PTSD interventions are initiated, postinjury care managers focus on ameliorating a patient's concerns. Posttraumatic care management interventions are tailored here to address salient features of patients' social contexts and identified life stressors. For example, an injured patient assaulted by a stranger in the presence of other family members might require more information about the safety of family members before any individually focused PTSD treatment could occur with the patient fully engaged. The care management intervention component is designed to address such key concerns.

In step 2, evidence-based intervention strategies are added to care management in a systematic stepped fashion to directly target psychiatric symptoms. The evidence-based treatment strategies specifically target PTSD symptoms and co-occurring conditions such as depression and alcohol or substance abuse. Among the most commonly employed treatment

methods are cognitive behavioral therapy, motivational interviewing, and pharmacotherapeutic treatment.

Cognitive Behavioral Therapy

Efficacy research suggests that individuals with posttraumatic psychological symptoms may respond to a number of evidence-based cognitive behavioral therapies (CBTs). Across trauma-exposed populations, CBT is the evidence-based treatment modality for PTSD most consistently recommended by best-practice treatment guidelines (Committee on Treatment, 2007; Department of Veterans Affairs, 2003; National Collaborating Centre for Mental Health, 2005; Ursano et al., 2004). CBT intervention explicitly orients patients to the nature and purpose of the intervention, in part toward the goal of teaching patients strategies they can then use themselves, independent of the therapist, and generalize to other problems and situations. The psychoeducational and cognitive restructuring components are specifically designed to provide concrete options for change and obtaining help if needed. A growing body of research now documents that CBT may be an effective early preventive intervention for acutely stress-exposed patients (Roberts, et al., 2009a; 2009b).

Motivational Interviewing

The motivational interviewing (MI) approach emphasizes the belief that people often have the knowledge and skill to do what is best for them, but are ambivalent about doing so and therefore need time to prepare for change by resolving this ambivalence. The clinician using MI focuses on the client's unique perspective toward his or her high risk behaviors and uses skillful questioning, listening, and feedback to elicit the patient's view. MI is based on a foundation of client-provider decision making. Specific person-centered techniques include respecting the person's agenda during the interview and informing him or her about the negative consequences of high-risk behaviors (drinking, etc.) while weighing with the individual in a deliberative fashion the pros and cons of such risk factors (Dunn et al., 2003). Multiple investigations have established the efficacy of motivational interviewing in reducing alcohol consumption and other risky behaviors among injured trauma survivors (Gentilello et al., 1999).

Pharmacotherapeutic Intervention

An emerging evidence base also suggests that pharmacotherapeutic interventions may be effective in managing early as well as chronic trauma symptoms (Agency for Health Care Policy and Research, 1993; Committee on Treatment, 2007; Zatzick & Roy-Byrne, 2006). Evidence-based pharmacotherapy treatments for PTSD include guideline-level therapeutic doses of serotonin-specific reuptake inhibitor antidepressant agents (e.g., sertraline 75–200 mg, paroxetine 10–50mg), and medications targeting insomnia and related symptoms, such as posttraumatic nightmares (e.g., prazosin 1mg, trazadone 50 mg).

Two larger-scale randomized effectiveness trials have now successfully employed the stepped collaborative care intervention model to engage low-income, diverse, injured trauma survivors and reduce PTSD symptoms (Zatzick et al., in press; 2004). In both trials, patients randomly assigned to stepped care intervention demonstrated clinically and statistically significant improvement in PTSD symptoms over the course of the year after injury

compared with injured trauma survivors assigned to a usual care control condition. Injured trauma survivors assigned to the intervention condition have also demonstrated improvement in comorbid alcohol use problems and physical function relative to controls. Results indicate that the model may be associated with both diminished symptoms of PTSD and alcohol consumption over the course of the year after injury (Zatzick et al., 2004; in press).

CONCLUSION

An increasingly persuasive body of evidence is fostering transformative ways of thinking about mental health research and the translational mechanisms between research, policy, and practice. Multi-factor and multi-level models capable of specifying mechanisms of effects between person and environmental forces is reflected in the cells to society vision undergirding the NIH roadmap and related initiatives (Shonkoff & Phillips, 2000; Warnecke et al., 2008). In this vein, Shonkoff et al. (2009) describe a scientific consensus that highlights the childhood roots of adult disease—both physical and mental—and the need for new frameworks that prioritize stress and inequalities within life course models. This call joins many advocating for increased collaborative work that transcends traditional disciplinary, disorder, and population silos. A unifying underlying premise is that promotion of mental health and prevention of disorders should embrace “upstream” factors such as persistent social disadvantage and corollary environmentally stressful and toxic conditions to achieve more complete and durable advances (Pearlin et al., 2005).

The vision of a more unified scientific base is evolving in the interarticulation of advances across multiple fields—such as neuroscience, developmental biology, victimology, medical sociology, and the psychophysiological conduits of stress and social disadvantage. The ascendance of transdisciplinarity as a model for research and translation runs parallel to these aims. Team science is rapidly evolving to accomplish these aims—integrating interdisciplinary groups of scientists and service professionals that transcend individual disciplines as well as the boundaries of social, behavioral, and biological sciences in order to cross fertilize and knit together theories, methods, and results to promote more integrative questions, linkages, and findings (e.g., Adler & Stewart, 2010b). Schwartz and Corcoran (2010) point out that focusing on the biological substrate of psychiatric disorders broadens the range of phenomena we examine in linking social factors to psychiatric disorders:

For example, social class may be related to psychiatric disorder not only through its effect on stressful life events, coping and the like but through increased exposure to viral infections, head injuries, birth trauma, and other physical phenomena. The influence of social structure is no less important if it works through the disparate and unequal distribution of physical risk factors than through psychological ones (2010; p. 86).

Team science builds on a scientific base that has been progressively developing in sophistication, akin to what Adler & Stewart (2010a) refer to as eras of advance. Extending recent gains regarding understanding multiple levels of influence spanning neurons to neighborhoods, we are now poised to make significant gains in assessment of interactions, complex system-to-system relationships, and nuanced pathways of causality. Interaction

effects, for example, are increasingly demonstrated in epigenetic research with growing appreciation of ways that environmental conditions influence gene expression, with significant implications for outcomes of concern for mental health such as aggression, addiction, and depression as well as physical outcomes such as coronary disease, autoimmune disorders, and infectious diseases.

There are, of course, layers of complexity involved—including conceptual, interpersonal, organizational, and institutional factors in addition to scientific methods and translation to stakeholders (Adler & Stewart, 2010b; Shonkoff, 2010; Holmes et al., 2008). Challenges notwithstanding, the fuller understanding achievable through integrative and transdisciplinary collaboration facilitates innovative scientific advances, augmenting societal benefits, including improving the nation's mental health.

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