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Psychobiological Effects of Sexual Abuse

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1. Introduction

Sexual abuse represents a stressor that challenges psychobiological homeostasis. Basic survival requires an ability to maintain internal equilibrium by adjusting physiological processes to match the demands of the external and internal environment. "Stress" is a broad term representing any physical or psychological demand that challenges equilibrium. When outside temperatures rise, physiological systems respond with multiple changes so as to keep internal temperatures from rising to a dangerous level. As nourishment is depleted, hunger is an internal cue that prompts food seeking. If ignored, energy resources will be shut down to non-critical bodily functions and shunted to those most critical for continued survival (e.g., the brain). Threat of physical or psychological harm activates biological systems that facilitate an appropriate response, including aggressive defensive actions ("fight") or avoidance behavior ("flight"). Unfortunately, a protective response is often unavailable or ineffective in the context of sexual abuse. Alternatively, a psychobiological response may prove effective in the short-term, but ultimately lead to a chain of processes that set the stage for long-term mental and physical health risk.

In this chapter, we will: a) describe the biological systems most closely involved in regulating stress responses; b) review existing literature on the psychobiological consequences of child and adult sexual abuse; c) discuss limitations of existing data; and d) introduce a conceptual framework for understanding sexual abuse as it relates to future mental and physical health.

2. The biological response to threat

Stressful experiences bring about a complex and counterbalancing set of hormonal responses in the sympathetic-adrenomedulary (SAM) and the hypothalamic-pituitary-adrenal axis (HPAA) systems, as well as the immune system. The basic components of these systems have been mapped out through animal studies, which allow for scientific control over the timing, frequency, type, and severity of the stress. Widely examined in animal and human research, the HPAA is best known as the regulator of the "fight-flight" response to threat, diverting energy resources needed for protective actions and promoting a subsequent return to homeostasis when the threat is no longer present. Such a system exists in all vertebrates (Lovejoy, 2005). Thus, the HPAA has been a central focus of basic animal research on fear conditioning, general effects of environmental stress, the developing brain, "learned helplessness" models of depression, as well as human studies of anxiety and mood

disorders more generally (Friedman, Charney, & Deutch, 1995). More recently, it has been proposed that stimuli perceived as significant may not only elicit aggressive or avoidance behavior (i.e., fight/flight), but also approach and affiliation behaviors, referred to as "tend and befriend" (Taylor et al., 2000). These behaviors are suggested to be controlled primarily by the neurosubstances of oxytocin and arginine vasopressin (AVP) (known alternatively as vasopressin, argipressin, or antidiuretic hormone (ADH). When triggered by threat, the hypothalamus releases corticotrophin-releasing hormone (CRH/alternatively "factor" or CRF) to the anterior pituitary. CRH, oxytocin, and AVP are all secreted in the paraventricular nucleus (PVN) and the arcuate nucleus (AN) of the hypothalamus. Mutual origination from the PVN and excitatory and inhibitory cross-talk between CRH, OXY, and AVP suggest these may be more interrelated than previously thought (van den Burg & Neumann, 2011). Nonetheless, biological assessment of oxytocin or AVP following sexual abuse has not yet been examined. Thus, we restrict our discussion here to the better known sequelae of the SAM and HPAA.

In humans, the perception and interpretation of stimuli occurs within the limbic system, consisting mainly of the hippocampal formation, amygdala, and entorhinal cortex. When stimuli are identified as potential perturbations to the basic state of equilibrium (i.e., physical or psychological "stressors"), the SAM system immediately (within seconds) releases the catecholamines epinephrine (adrenaline) and norepinephrine (noradrenaline, or NE). The catecholamines facilitate subsequent processes that increase the heart rate, blood pressure, and blood glucose levels in muscles and vital organs in order to allow the body to adapt to the increased demand. Stressors also trigger the HPAA to begin a slower process occurring over minutes. This response begins with the hypothalamus, a cone-shaped brain structure that sits below the thalamus, projecting downward into the pituitary (infundibular) stalk which ends at the roundish-shaped pituitary gland (Afifi & Bergman, 1998). Once triggered, the hypothalamus releases corticotrophin-releasing hormone (CRH/alternatively "factor" or CRF) to the anterior pituitary. The pituitary response to CRH is to release adrenocorticotropic hormone (ACTH). In turn, ACTH stimulates release of glucocorticoids (in humans, "cortisol") and dehydroepiandrosterone (DHEA) from the adrenal glands located above the kidneys. Cortisol and the catecholamines further act on the immune system, which results in changes in the levels and activity of cytokines that are especially relevant for host protection against viral infections like HIV (i.e., CD4 and CD8 T-cells) (Coe & Laudenslager, 2007; Glaser & Kiecolt-Glaser, 2005).

Under normal circumstances, specialized feedback systems are designed to ensure a return to homeostasis once the threat is gone. Once released, cortisol has an important role in shutting down the sympathetic activation of the SAM system and suppressing further release of CRH by a negative feedback mechanism on the pituitary, hippocampus, hypothalamus, and amygdala. The anti-glucocorticoid properties of DHEA are also believed to contribute to an up-regulation of HPAA responses, as well as mitigate possible deleterious effects of high cortisol levels on the brain (Rasmusson, Vythilingam, & Morgan, 2003). Once the perception of threat recedes, the negative feedback mechanisms help restore neurosubstance levels to resting states (allostasis). Thus, the HPAA represents a "negative feedback" loop by which cortisol release is triggered *and* subsequently inhibited.

However, when stress exposure is very intense, frequent, or chronic, the effects of the SAM, HPA axis, and immune system on target cells and organs may be prolonged. The interaction

of the stress hormones (cortisol and the catecholamines) with cell receptors produce both short-term changes in cell processes, as well as changes in gene expression that have long-lasting consequences for cell function. Over time, these effects can lead to tissue damage, and changes in cell functioning and the brain (structural atrophy, suppressed neurogenesis, and synaptic and dendritic remodeling). Such central neurobiological changes may lead to HPAA dysregulation that is opposite to expected patterns. A counter-intuitive *low* level of urinary cortisol was first reported in men diagnosed with Posttraumatic Stress Disorder (PTSD) (Yehuda et al., 1990). PTSD is, by definition, a fear-based psychiatric condition. Diagnostic requirements include: a) exposure to a traumatic event involving perceived threat of death or harm to physical integrity of the self or another person; and b) an acute emotional reaction of fear, helplessness, or horror. Brain imaging studies confirm distinct neurological areas are activated in PTSD patients who show heightened peripheral SAM system reactivity (e.g., increased heart rate) versus those showing blunted reactivity (Lanius et al., 2010) with an explanatory proposal that there are two forms of PTSD. These and subsequent studies in other psychiatric populations (e.g., Major Depressive Disorder: Ahrens et al., 2008; Panic Disorder: Petrowski, Herold, Joraschky, Wittchen, & Kirschbaum, 2010) and otherwise healthy individuals experiencing chronic stress (Juster et al., 2011) demonstrate the bi-directional nature of HPAA output at rest and/or in response to new threat. The general direction of HPAA dysregulation (hyper vs. hypo responsive) is thought to depend upon characteristics of the threat (e.g., the intensity, frequency, and chronicity), the timing of assessment relative to the threat (immediate or minutes/hours versus days or months), or the developmental stage in which the threat occurs (McEwen, 2010; McEwen & Gianaros, 2011). Also, the directional impact of cortisol on other neurotransmitters and systems appears to depend upon the perceived anticipatory immediacy of new and recurring threat (Sapolsky, Romero, & Munck, 2000). Of note, these are environmental, not genetic factors. A study of monozygotic twins discordant for bullying (one with and one without a victimization history) supports the experiential nature of threat effects on HPAA reactivity (Ouellet-Morin et al., 2011). Bullied twins showed a blunted cortisol response to a psychosocial stress test, whereas their non-bullied siblings had a normal cortisol rise. There was no evidence that these group differences were due to genetic makeup, family environment, pre-existing or concomitant individual factors, and the perception of stress or the level of subjective emotional responding to the stress test.

Ultimately, stress effects are associated with poor health outcomes across multiple systems, including cardiovascular, metabolic, and immune systems (McEwen, 2008; McEwen & Gianaros, 2011). Emerging data on telomeres, which are deoxyribonucleic acid (DNA)/protein repeats at the end of chromosomes that regulate cellular replicative life span, indicate stress prematurely shortens telomere length (Epel et al., 2004) and thus may hasten all-cause mortality. Telomere length is receiving widespread attention as a potential biomarker proxy for cumulative stress burden, accelerated aging processes, and expected lifespan for a given individual (Epel et al., 2009).

Sexual abuse, either as a single or re-occurring event, represents a significant threat to the physical and psychological integrity of the individual and thus acts to "perturb" homeostasis. The ability to successfully adapt to this challenge, both immediately and in the long term, is key to psychological and physical health and perhaps even how long one can expect to live.

3. Assessment of biological dysregulation in sexually abused children and adults

In preclinical studies of rodents and primates, neurotransmitters and their effects can be measured centrally in a temporal sequence concurrent with presentation and removal of stress. In humans, we are rarely able to assess immediate post-trauma biology. Sexual abuse remains a terrible and stigmatized secret for many victims who often do not disclose the abuse (London, Bruck, Ceci, & Shuman, 2005; Sorsoli, Kia-Keating, & Grossman, 2008). If they do, they may not receive the emotional support and protection that is needed to avoid further victimization. Childhood sexual abuse (CSA) is particularly unlikely to be revealed until long after it has occurred. It may be revealed to others only as an adult survivor, or may never be revealed. Non-disclosure of adult sexual abuse (ASA) is also common. Thus, treatment seeking in any form (from a psychologist, counselor, religious leader, or other supportive professional) is low, particularly among low-income racial/ethnic minorities.

Widespread under-reporting is likely to result in inaccurate prevalence rates (Pereda, Guilera, Forns, & Gomez-Benito, 2009). As a result, the full effects of sexual abuse on development and long-term physical health across the lifespan are not well known. Studies of biological functioning in sexual abuse victims soon after an incident are limited, with most sequelae being reported retrospectively in adulthood (Burns-Loeb et al., 2002; Glover, Garcia-Aracena, Lester, Rice, & Rothram-Borus, 2010). Thus, most biological data on CSA survivors come from retrospective studies among adult women with self-reported histories of CSA. Studies among sexually abused women are also rarely conducted near the time of an ASA incident. Very little research exists on biological effects of sexual abuse among males, including assessment of CSA effects among boys or adult men reporting retrospectively, or among adult men soon after ASA (Burns-Loeb et al., 2002).

3.1 Biological functioning after CSA among children

CSA victims are often exposed to multiple types of maltreatment (including physical abuse, neglect, and psychological maltreatment) and adversity factors (including variables reflecting interpersonal loss, parental dysfunction, family violence, economic adversity, and life-threatening illness) (Higgins, 2004). Nonetheless, research generally limits investigations to a single type (Kendler et al., 2000), combines different types of adverse exposure into a single group, does not include measures of severity and developmental timing, and rarely includes non-English-speaking participants (Green et al., 2010; McLaughlin et al., 2010; Scott, Smith, & Ellis, 2010). These factors do not have equal effects on outcomes and their joint effects are not additive nor specific to a particular outcome (Green et al., 2010).

Of the few studies reporting biological outcomes in sexually and/or physically abused children, it is clear that physiological disturbances may be widespread. The effects of even a single severe traumatic event involving threat of or actual physical harm to the victim may permanently alter functioning of neurotransmitters from the HPAA and SAM systems. These abnormalities may be detected at rest or only in response to new stressors. Examples of affected systems include autonomic activity, cortisol and catecholamines, and immune system indicators, although findings are not consistent in direction or pattern (Cicchetti & Rogosch, 2001; Gunnar & Quevedo, 2007; McEwen & Gianaros, 2011; McEwen & Wingfield, 2003; Nemeroff, 2004; Putnam, 2003; Turner-Cobb, 2005). Biological studies have largely examined the HPAA, although acute stress is known to affect the limbic system and have

potential effects on gene expression of widespread neurohormonal receptors (DeBellis et al., 1994; DeBellis, Lefter, Trickett, & Putnam, 1994; Heim et al., 2000; Heim & Nemeroff, 2001; Penza, Heim, & Nemeroff, 2003; Wise, Palmer, Rothman, & Rosenberg, 2009).

A recent review of the literature on neuroendocrine dysregulation after CSA showed that of 127 publications from 1990 to January 2007, only 7 studies with biological data soon after a CSA event could be found (Bicanic, Meijer, Sinnema, van de Putte, & Olff, 2008). Of these, several did not report the time since abuse and so are not discussed here. King, Mandansky, King, Fletcher, and Brewer (2001) identified 10 girls who had experienced CSA in the past month (as reported by parents and/or social service agencies). The group was mostly white (70%), and 5 to 7 years of age. Although study eligibility required one CSA incident within the past 2 months, most (80%) also reported multiple incidents occurring over as many as 12 months before study entry. Salivary cortisol was collected on the morning of a physician-scheduled exam designed to identify "physical or emotional" signs to support sexual abuse" (p 72). Morning salivary cortisol was then also collected from non-abused controls matched on age, ethnicity, sex, and socio-economic status. CSA survivors showed significantly lower morning salivary cortisol relative to controls, suggesting HPAA dysregulation can be detected within months of CSA.

In another study reporting assessment soon after CSA, a cohort of sexually abused girls were identified within 6 months of their disclosure of the abuse. In the first assessment, morning plasma cortisol levels were found to be significantly elevated as compared to controls matched for demographics (Putnam, Trickett, Helmers, Dorn, & Everett, 1991). The next assessment of a subsample of this cohort (6 or more months later) collected 24-hour urinary free cortisol and also involved administration of exogenous (ovine) CRH in order to measure plasma ACTH and cortisol reactivity (DeBellis et al., 1994). On this occasion, cortisol secretion detected from urine and plasma following CRH did not differ across abused and non-abused groups, but CSA girls showed reduced ACTH reactivity to the CRH infusion. Results indicate that dysregulation is detectable months and years after the initial abuse and may change over time. Unfortunately, such dysregulation cannot be attributed to the abuse itself because new trauma and/or adverse conditions could have occurred in the period between the initial abuse and the biological assessment.

The findings on ACTH reactivity in CSA (DeBellis, Lefter, Trickett, & Putnam, 1994) preceded a large number of subsequent studies among traumatized adults with PTSD focusing on mechanisms of HPAA dysregulation (for recent updates on mechanisms see Friedman & Pitman, 2007). This focus is reflected in one other study of biomarkers found among CSA survivors during childhood. Duval and colleagues (2004) examined hospitalized adolescents (aged 10 to 14) with PTSD who reported sexual abuse years earlier (mean 5.6 +/- 4 years). ACTH and cortisol levels were measured following administration of an HPAA stimulating hormone, dexamethasone. Compared to controls, these CSA survivors showed ACTH hypersuppression to dexamethasone. Whether dysregulation among these teens was due to PTSD, exposure to child sexual abuse, or both was not possible to determine.

Brain imaging of abused children indicates changes in the size or symmetry of key brain structures (hippocampus, corpus collosum, prefrontal cortex) as well as neuronal quality (density and integrity) of certain brain regions (Bremner et al., 1997; Teicher, Tomoda, & Andersen, 2006). Carrion, Weems, and Reiss (2007) reported brain changes associated with cortisol and PTSD symptoms in 15 children exposed to at least one traumatic event. Most had experienced multiple traumatic events, including sexual, physical, and emotional abuse

as well as witnessing violence. The sample included 6 boys and 9 girls ages 8 to 14 (mean=10.4), and a mixed ethnic composition of primarily whites (n=7) or African Americans (n=6). The children were assessed twice, with assessments separated by 12-18 months. Brain imaging techniques were used to evaluate changes in hippocampal size over time in relation to PTSD symptoms and home-collected cortisol levels across the day. Results showed that participants with the highest severity of PTSD symptoms at Time 1 showed the greatest reductions in the right hippocampus from the first to the second assessment. Elevated evening (pre-bed) salivary cortisol at Time 1 was also related to reductions in hippocampal size at the next assessment.

3.2 Biological functioning among adult survivors of sexual abuse

Most sexual abuse (SA) data relevant for biological functioning come from retrospective studies among adult women who were abused as children. For example, Leserman (2005) reviewed studies of physical health among sexually abused samples, indicating higher than expected associations between SA and headache, gastrointestinal, gynecologic, and panic-related symptoms. Retrospective biomarker assessments with control groups indicate CSA exposure is associated with differences in 24-hour urinary cortisol excretion (e.g., Lemieux & Coe, 1995) and smaller hippocampal volume (e.g., Stein, Walker, Hazen, & Forde, 1997). Retrospective studies of adult women with a history of CSA *or* other types of abuse (e.g., neglect and physical abuse) have shown changes in autonomic activity and ACTH responses to laboratory stress (Heim et al., 2000; Heim & Nemeroff, 2001; Heim & Nemeroff, 2002) as well as smaller hippocampus volume (Bremner et al., 1997; Vythilingam et al., 2002). One study indicated brain volume differences in specific regions were found for women abused at ages 3-5 years (hippocampus) versus ages 9-10 years (corpus callosum), ages 11-13 years (hippocampus), or 14-16 years (frontal cortex) (Andersen et al., 2008). Studies of CSA survivors categorized as having PTSD or not sometimes indicate biological dysregulation occurs only among CSA survivors with current PTSD (e.g., T cell activation of the immune system) (Lemieux, Coe, & Carnes, 2008). Functional brain imaging of CSA exposed adults with PTSD demonstrate changes in activation patterns (Lanius, Bluhm, Lanius, & Pain, 2006). The direction of neuroendocrine differences (higher vs. lower cortisol or ACTH response) has been inconsistent. Trickett, Noll, Susman, Shenk, and Putnam (2010) report data indicating discrepancies may be due to changes in neuroendocrine functioning in the initial period after abuse (associated with elevated cortisol) versus after many years have passed (associated with attenuated cortisol).

Only one study is known to have conducted biological assessments soon after adult sexual abuse. Resnick, Yehuda, Pitman, and Foy (1995) collected plasma cortisol levels from female rape victims within hours of the SA. Those who reported a history of prior assault showed significantly lower cortisol levels in the acute aftermath than those who reported no prior assault histories. Other biological assessment studies among abused adults typically combine SA with other forms of physical and psychological abuse (e.g., Garcia-Linares, Sanchez, Lorente, Coe, & Martinez, 2004; Sanchez-Lorete, Blasco-Ros, Coe, & Martinez, 2010).

4. Problems of interpretation

In the absence of assessment soon after an initial incident of sexual abuse, and periodically thereafter (i.e., longitudinal studies), it is difficult to interpret biological findings in relation

to past events. Numerous intervening events can affect outcomes and the longer the period between CSA and assessment, the less likely scientists can be confident in proper interpretation of findings.

There are two related intervening factors that make interpretation of retrospective studies especially difficult. First, CSA has been linked to a greater likelihood of sexual revictimization both later in childhood and in adulthood (Classen, Palesh, & Aggarwal, 2005; Messman-Moore & Long, 2000; Messman-Moore & Long, 2003). There have been numerous studies conducted with diverse samples of women who were sexually abuse as children and then subsequently revictimized (Messman-Moore & Long, 2003). Studies have included college students (Gidycz, Hanson, & Layman, 1995; Mayall & Gold, 1995; Messman-Moore & Long, 2000; Messman-Moore, Long, & Siegfried, 2000), clinical samples (Briere & Runtz, 1987; Bryer, Nelson, Miller, & Krol, 1987; Shields & Hanneke, 1988), military samples (Merrill et al., 1999), community samples (Fergusson, Horwood, & Lynskey, 1997; Messman-Moore & Long, 2000; Wyatt, Guthrie, & Notgrass, 1992), and lesbian and bisexual female samples (Morris, Balsam, & Rothblum, 2002). Research on sexual revictimization with men, specifically gay and bisexual men is an emerging field (Heidt, Marx, & Gold, 2005; Kalichman et al., 2001). Experiences of CSA have been correlated with negative contributions to mental, physical, and sexual health (Burns-Loeb et al., 2002; Whiffen & MacIntosh, 2005). However, it is not clear why some victims of sexual abuse are at greater risk of revictimization or how singular versus multiple experiences affect mental and physical health outcomes. For example, evidence exists supporting that sexual abuse during certain stages of development, severity of CSA, and additive forms of trauma such as sexual and physical abuse may be important factors in distinguishing survivors of CSA who are revictimized from those who are not (Classen, Palesh, & Aggarwal, 2005). Most of the research on CSA and revictimization is derived from cross-sectional and retrospective studies. As a consequence, it is difficult to distinguish between correlational and causal relationships.

The second related intervening factor that complicates interpretation of retrospective studies is the common comorbidity of mental health disorders, multiple non-sexual trauma types, and concurrent child and adult adversities. Exposure to CSA, as with other childhood trauma and adversities, is associated with increased prevalence of adult DSM-IV mood disorders, anxiety disorders, and substance use disorders in numerous large epidemiological studies (Green et al., 2010; McLaughlin et al., 2010; Scott, Smith, & Ellis, 2010). Abnormal biological assessment profiles in adulthood may be due to: a) child sexual abuse; b) other trauma or adversity occurring in childhood or as an adult; c) the current (adult) presence of a psychological disorder; or d) some combination of these factors.

A landmark longitudinal study by Koenen, Moffitt, Poulton, and Caspi (2007) illustrates this issue. Records for a New Zealand birth cohort (N=1037) were reviewed for childhood neurodevelopment, temperament, behavioral, and family environment characteristics and in-person follow-up assessments at ages 26 and 32 were conducted. Data revealed a cumulative negative effect of exposure to both acute trauma and adversity in childhood (before the age of 11). One set of factors (low IQ and chronic environmental adversity, including poverty) increased risk for PTSD at age 26, but not the number of lifetime trauma exposures. A second set of factors (childhood externalizing characteristics, maternal depression, and loss of a parent) not only increased risk for having a PTSD diagnosis at age 26, but also increased risk

for exposure to more lifetime acute traumas even in the absence of PTSD symptoms. Subsequent victimization could be due to increased risk-taking behaviors, poor cognitive functioning that would normally allow escape from risky situations, the negative impact of early trauma on subsequent educational and vocational achievement, or the impairments associated with PTSD symptoms. If subsequent trauma exposure in adulthood occurred between age 26 and 32, those with the greatest childhood burden of trauma and adversity were also more likely to develop PTSD even if they had not experienced such psychological symptoms before. Thus, a host of developmental factors, including exposure to CSA, contribute to vulnerability for increased trauma exposure and for mental health sequelae.

Among CSA survivors, additional sexual abuse (revictimization), exposure to other trauma and adversities, and the development of mental health disorders are common co-occurrences. Unless a careful history is obtained and these factors systematically controlled (e.g., through research study design or statistical covariance), reliable interpretation of biological outcomes in adulthood is limited.

Studies designed to address these limitations exist. For example, Heim, Mletzko, Purselle, Musselman, and Nemeroff (2008) examined HPAA functioning among healthy men (aged 18-60) recruited into four study groups, including: 1) normal subjects with no childhood abuse history or psychiatric disorder (n =14); 2) men with childhood abuse histories without current major depressive disorder (MDD) (n=14); 3) men with childhood abuse histories with current MDD (n =15); and 4) men with current MDD and no childhood abuse history (n=6). HPAA functioning was examined using the dexamethasone/corticotropin-releasing factor (CRF) test, which is a sensitive measure of HPAA hyperactivity and has been demonstrated to be altered in patients with MDD. Men with childhood trauma histories exhibited increases in ACTH and cortisol responses to dexamethasone/CRF relative to non-abused men with and without depression. Increased response was associated with the severity, duration, and earlier onset of the child abuse. Statistical analyses indicated effects were not due to concurrent PTSD symptoms (Heim et al., 2008).

5. Discussion of limitations of existing studies

Well-designed psychobiological research that includes appropriate comparison groups is surprisingly limited. Timely disclosure of an event is clearly a prerequisite for immediate assessment, but even when reporting has occurred (e.g., to social service agencies or police), psychobiological data are rarely collected. Even when data is collected, researchers now know that the specific form of biological dysregulation after acute or chronic stress is highly variable. As noted earlier, both very high and very low cortisol levels have been found in adults with PTSD (Glover & Poland, 2002) and among physically and sexually abused children (Gunnar & Quevedo, 2007). Bi-directional heart rate and functional magnetic imaging responses in adults with PTSD have also been documented (Lanius, Bluhm, Lanius, & Pain, 2006). Lanius and colleagues (2010) propose there are two types of PTSD based on the pattern of reactivity to new stressors. Some individuals exposed to trauma subsequently fail to react to new stress, showing blunting of the normal cardiovascular and neuroendocrine increases. This pattern is referred to as a "dissociative" type of PTSD and may be related to behavioral tendencies for dissociative amnesia, fantasy proneness, and depersonalization used to cope with extreme stress. Others may react normally, but fail to recover within the expected time frame for a given biomarker. These data are in keeping

with emerging awareness of the fluid and counterbalancing nature of biological systems and their primary output. For example, recent findings show norepinephrine's capacity as both an anxiogenic and anxiolytic (Nemeroff, 2004) and cortisol effects not only reduce immune system activity, but also heighten it depending upon the stress context (i.e., type, duration, imminence of threat) (Sapolsky, 2000). Future research on the psychobiology of sexual abuse must begin to use updated methods, including examining individual biomarkers bi-directionally and multiple biomarkers across systems known to counterbalance each other.

It is now clear that the form of bio-dysregulation may change as a function of the amount of time that has elapsed since the last sexual abuse event or the last traumatic event, if numerous types of traumas are concurrently being experienced (Weems & Carrion, 2007). In a study of mothers of children with life-threatening illness, Glover, Garcia-Aracena, and Mohlman (2008) found that maternal hippocampal volume, known in animal studies to shrink after exposure to stress-related high cortisol, showed a significant positive association with the time elapsed since the child's illness diagnosis. That is, the more time that had passed since the onset of the child's illness, the larger the hippocampus size (after controlling for age of the mother). This cross-sectional study indicates plasticity of the hippocampus, with the greatest negative effect close in time to the stressor and a gradual recovery. The timing of sexual abuse exposure in relation to developmental stage (e.g., child vs. adult, pre- vs. post-puberty, during women's child-rearing vs. post-menopausal years) is also quite likely to influence the psychobiological sequelae of abuse (Heim, Plotsky, & Nemeroff, 2004).

Peri-traumatic factors at the time of sexual abuse may influence mental and physical health effects, including severity (e.g., penetration vs. fondling) and the victim's relationship to the perpetrator (e.g., immediate family member or intimate partner vs. distant acquaintance or stranger) (Glover et al., 2010). The absence of information on specific characteristics of trauma and the socio-cultural context in which trauma occurs is a recognized limitation of large scale epidemiological studies of trauma and adversities (Green et al., 2010). This may be particularly relevant for sexual abuse in the context of racial/ethnic minority cultures that are associated with strong gender roles and expectations (Sciolla et al., 2011).

Finally, perhaps as a result of outside agendas from potential funding providers (e.g., pharmaceutical companies) many existing studies recruit only a narrow band of sexually abused study participants who do or do not meet criteria for a specific mental health disorder (e.g., PTSD or MDD) or physical health problem (e.g., gynecological abnormalities). Ideally, studies should include sexual abuse exposed individuals with and without mental and physical health symptoms and control groups who also vary on abuse and health histories. Although not based on sexual abuse trauma, a series of studies examining multiple biomarkers of health risk (BHR) in highly stressed women with and without PTSD or MDD is informative (Glover, Steele, Stuber, & Fahey, 2005; Glover, Stuber,& Poland, 2006; Glover et al., 2010). Whereas BHR was highest among those with current mental health symptoms, neither PTSD or depression symptoms were a necessary prerequisite for elevations in BHR relative to controls (Glover, Stuber, & Poland, 2006). Furthermore, elevated BHR was linked to lower volume of the hippocampus (Glover, Garcia-Aracena, & Mohlman,2008), independent of any mental health symptoms as determined by clinical interview. Together, these data suggest that psychobiological dysregulation following sexual abuse may be detected even when mental health symptoms as assessed via self-report are absent. Nonetheless, self-reported mental health symptoms, even at sub-clinical levels as measured by conventional psychological assessments, may exacerbate dysregulation and ultimately, pose a risk for health problems.

6. A framework for understanding psychobiological consequences of sexual abuse

Challenges for understanding psychobiological sequelae of sexual abuse include complex, bi-directional, and counterbalancing biological systems, a paucity of studies among child and adult victims, and research design and omission limitations in existing research. As biological investigations of sexual abuse sequelae become more sophisticated, these must also connect with the growing knowledge of personal, interpersonal, and community level moderating and mediating variables, particularly for revictimization (Breitenbecher, 2001; Classen, Palesh, & Aggarwal, 2005; Messman-Moore, & Long, 2003).

6.1 Personal and interpersonal level factors

Age, gender, and race/ethnicity are likely to have significant effects for long term biological outcomes following SA. Research among boys and men is greatly lacking and may show outcome differences than those from females. Sexual abuse during adolescence has been shown to place a woman at greater risk for revictimization in adulthood than that occurring at earlier times (Gidycz, Coble, Latham, & Layman, 1993; Humphrey & White, 2000). Developmental stage of occurrence not only during childhood but also critical stages during the lifespan (e.g., during pregnancy or SA as the mother of young children) may have profound effects on biological health outcomes. The racial/ethnic background of the victim is now understood to be a major influence on the meaning and interpretation of SA for the individual as well as the response from others if/when abuse is revealed (Behl, Crouch, May, & Valente, 2001; Bohn, 2003; Urquiza & Goodlin-Jones, 1994; Wyatt, Guthrie, & Notgrass, 1992). A detailed **sexual history**, including severity, duration, frequency, use of force, number of perpetrators, and relationship to perpetrators are all variables critical to health outcomes and risks for sexual revictimization (Arata, 2000; Bifulco, Brown, & Adler, 1991; Collins, 1998; Kessler & Bieschke, 1999; Koverola, Proulx, Battle, & Hanna, 1996; West, Williams, & Siegel, 2000; Wyatt, Loeb, Solis, Carmona, & Romero, 1999). Recency of abuse in relation to the assessment appears important; the more recent the abuse, the greater the likelihood that it will predict revictimization (Himelein, 1995). A detailed history of **other forms of trauma** including child physical abuse and family adversity have also been linked to negative sequelae (Molnar, Buka, & Kessler, 2001) and sexual revictimization (Arata & Lindman, 2002; Desai, Arias, Thompson, & Basile, 2002; Heidt, Marx, & Gold, 2005; Jankowski, Leitenberg, Henning, & Coffey, 2002). **Family structure and dynamics and interpersonal relationships** should be assessed including the composition of the family unit and of primary social groups, the use of alcohol and drugs by members in these groups, and the methods for resolution of conflict are also relevant issues (Hamilton & Browne, 1999; Kellogg & Hoffman, 1997; Koverola et al., 1996; Long & Jackson, 1991; Nash, Hulsey, Sexton, Harralson, & Lambert, 1993; Nelson et al., 2002; Swanston et al., 2002). **Psychiatric disorders** (e.g., depression and anxiety or substance abuse) as well as **psychological factors** (e.g., shame, self-blame, and resiliency) are critical variables to explore (Neumann, Houskamp, Pollock, & Briere, 1996). **Medical illnesses** prior to, during, or after sexual abuse need to be assessed as they may be associated with and/or sequelae of sexual abuse (Wegman & Stetler, 2009).

6.2 Community level factors

Social Support including relationships with extended family, friends, church, etc., is especially important as it relates to sexual abuse sequelae. Social support has been shown to

have an impact on disclosure and on moderating the psychological impact of sexual abuse (Borja, Callahan, & Long, 2006; Feiring, Taska, & Lewis, 1998). However, very little research has been conducted examining whether such support protects against the biological sequelae of sexual abuse. **Cultural, community and religious norms** need to be evaluated as these may provide scripts which influence how individuals appraise and respond to stress. Sparse research exists on examining how norms influence the way in which individuals interpret and define experiences such as sexual abuse. Also, little is known regarding how appraisal based upon these cultural, community, and religious norms subsequently impact or contribute to psychological or biological/physical manifestations. These factors need to be explored as they may influence health outcomes.

7. Conclusion

There is still much to examine with respect to the psychobiology of sexual abuse. The understanding of moderating and mediating variables for sexual abuse itself is increasing, but these have largely been studied independent of biological assessments. A more holistic model that considers biopsychosocial mediating and moderating variables as complex predictors for and consequential effects of sexual abuse is needed to enhance and accelerate the development of effective prevention and intervention efforts.

8. Acknowledgements

Funding to support this research and to write this chapter was provided from the National Institute of Mental Health (The ES-HIM Project [1 R34 MH077550] and the Center for Culture, Trauma, and Mental Health Disparities [5P50MH073453]).

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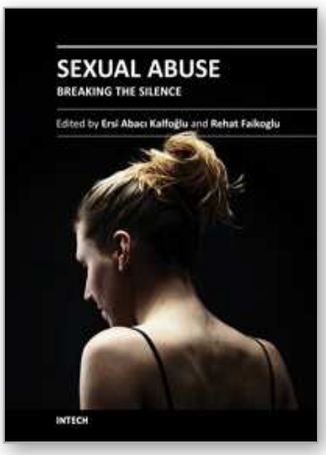

Sexual Abuse - Breaking the Silence
Edited by Dr. Ersi Abaci Kalfoğlu

ISBN 978-953-51-0425-4
Hard cover, 238 pages
Publisher InTech
Published online 23, March, 2012
Published in print edition March, 2012

Sexual assault can be considered as expression of aggression through sex. This, in turn, can have serious negative effects on a survivor's social and occupational functioning. This book has been organized towards that specific approach, by compiling the scientific work of very well-known scientists from all over the world. The psychological victimization of sexual assault, the physiological aspect of sexual abuse and the different attitudes in coping with sexual assault based on different cultural backgrounds are analyzed. Having in mind that one solution may not necessarily be suitable for all cases, we hope that this book will open a debate on sexual assault for future practice and policy and that it will be a step forward to 'break the silence'.

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Dorie A. Glover, John K. Williams and Kimberly A. Kisler (2012). Psychobiological Effects of Sexual Abuse, Sexual Abuse - Breaking the Silence, Dr. Ersi Abaci Kalfoğlu (Ed.), ISBN: 978-953-51-0425-4, InTech, Available from: http://www.intechopen.com/books/sexual-abuse-breaking-the-silence/psychobiological-effects-of-sexual-abuse-

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