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Physiological Correlates of Childhood Abuse: Chronic Hyperarousal in PTSD, Depression, and Irritable Bowel Syndrome[1]

Kathleen A. Kendall-Tackett, Ph.D.
Family Research Laboratory, University of New Hampshire, Durham

Objective: *During the past ten years, researchers have documented how trauma—especially severe trauma—can alter the functioning of the brain. In some cases, these alterations create a state of chronic hyperarousal. The present review serves as an introduction to this research.*
Findings: *Persons who have experienced traumatic events are often "primed" to over-react to subsequent stressors, making them more vulnerable to these events.*
Conclusion: *Chronic hyperarousal underlies three common and often co-occurring sequelae of childhood abuse: post-traumatic stress disorder (PTSD), depression, and irritable bowel syndrome. Knowledge of these physiological correlates can affect treatment decisions as well as our theories about the mechanisms underlying the development of symptoms.*

Key Words: Chronic stress, PTSD, Depression, Irritable Bowel Syndrome

The 1990's have been dubbed "the decade of the brain." During these past ten years, there has been a veritable explosion of information on how the brain works. We have also learned a great deal about how experience, including traumatic experience, can alter the brain (Perry & Pollard, 1998).

Research on how traumatic events may change the brain is exciting, and highly relevant to our work. Yet by and large, it has not penetrated the child abuse field. Several recent reviews (including ours) have either omitted physiological variables, or have only covered them briefly (Briere & Elliot, 1994; Kendall-Tackett, Williams & Finkelhor, 1993). Some child abuse researchers may not be aware of this work because it is generally published in neuroscience. Others may know about these studies, but find them to be difficult to understand—or even "boring."

The purpose of the present review is to provide an introduction to research on the physiological correlates of past victimization. This review is not meant to be comprehensive, but rather to stimulate interest and broaden our thinking to include the

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physiological variables. I organize these recent studies around three manifestations of chronic hyperarousal: PTSD, depression and irritable bowel syndrome.

The sequelae considered in the present paper are by no means the only possible consequences of past abuse. Nor is abuse the only mechanism by which these syndromes occur. Nevertheless, they provide a vehicle by which we might understand the consequences of past abuse in the broader context of chronic or extreme stressors. Without at least a working familiarity of biological variables, our theories for sequelae of abuse are incomplete, and we may be hampered in our ability to treat patients (Thase, Dube, Bowler, Howland, Myers, Friedman, & Jarrett, 1996).

Hyperarousal and Sensitization

Chronic hyperarousal is an abnormal state of activation that occurs in the wake of traumatic or highly stressful events. A normal stress response is adaptive, meant to preserve the life of the organism. The stress response becomes a problem when the stressor is an extreme one-time occurrence, which floods the system with stress hormones, or a chronic stressor, in which the response that is supposed to be to an acute emergency becomes a frequent occurrence.

Chronic hyperarousal can manifest itself as abnormal levels of stress hormones such as norepinephrine and cortisol, or as a change in the number or sensitivity of receptors to these substances (Yehuda, 1998). Chronic hyperarousal may also alter certain brain structures, such as the hippocampus, with exposure to abnormal levels of stress hormones (Bremner, 1999; Lovallo, 1997; Southwick, Bremner, Krystal, & Charney, 1994).

Hyperarousal can make victims of past traumatic events more vulnerable to current life stressors through a process called sensitization (McCarty & Gold, 1996). In the aftermath of traumatic events, the brain becomes threat-sensitized. The body "remembers" the traumatic event and over-reacts when faced with a new stressor (Schwarz & Perry, 1994). In this paper, three common sequelae of child abuse are described. Each is accompanied by physiological hyperarousal. In post-traumatic stress disorder, there are abnormally high levels of norepinephrine. In depression, cortisol is abnormally high. Finally, in irritable bowel syndrome, there appears to be a heightened sensitivity to physical pain. The first sequelae described is posttraumatic stress disorder.

Posttraumatic Stress Disorder (PTSD)

Symptoms of PTSD are common among adult survivors of childhood abuse and other traumatic events. Hyperarousal may manifest as sleep difficulties, hypervigilance, startle response, and intrusive thoughts (Briere & Elliot, 1994).

The endocrinology of PTSD has been the focus of many recent studies (Bremner, 1999; Yehuda, 1999). To date, researchers have found that people with PTSD have abnormally low levels of cortisol and abnormally high levels of norepinephrine (Yehuda, 1999; Yehuda, Levengood, Schmeidler, Wilson, Guo & Gerber, 1996). PTSD, particularly chronic PTSD, appears to be a dysregulation of a normal stress response. In

a normal response, cortisol and norepinephrine operate in concert. In PTSD, they appear to operate separately (Bremner, 1999). The separate actions of these two hormones has led some researchers to speculate that each may be involved in different aspects of PTSD symptomatology (Yehuda, Resnick, Schmeidler, Yang, & Pitman, 1998). For example, high levels of norepinephrine (NE) have been linked to intrusive thoughts and severity of PTSD symptoms (Lemieux & Coe, 1995).

Much of our understanding of PTSD is based on studies of male combat veterans (Bremner, Staib, Kaloupek, Southwick, Soufer & Charney, 1999). Similar findings have emerged for female adult survivors of child sexual abuse (Stein, Yehuda, Koverola, & Hanna, 1997). Yet at least one study of veterans and one of child sexual abuse survivors found that *both* norepinephrine and cortisol levels were elevated in subjects with PTSD (Lemieux & Coe, 1995; Pitman & Orr, 1990). In discussing the results of these two studies, Yehuda (1998) points out that the discrepancy may be due to other variables, such as patient obesity. Overall, however, the most consistently found pattern is low cortisol and high NE.

Childhood abuse also appears to make its victims more susceptible to PTSD in reaction to current life stressors. In one study, Bremner and colleagues (Bremner, Southwick, Johnson, Yehuda & Charney, 1993) compared rates of childhood abuse in Vietnam veterans with and without combat-related PTSD. They did indeed find higher rates of childhood physical abuse among veterans with PTSD compared with those without PTSD (26% vs. 7%). This association held even after controlling for level of combat exposure. The authors note, however, that childhood abuse does not account for all PTSD among veterans. Seventy-four percent of those with PTSD did not report a history of abuse.

A similar finding was made among female rape survivors. In this study, women with a history of either physical or sexual assault had significantly lower cortisol response to a current rape than did women who did not have such a history (Resnick, Yehuda, Pitman, Foy, 1995). Low cortisol levels predicted greater risk of developing PTSD from the current rape (Yehuda, McFarlane & Shalev, 1998).

In an effort to refine these results, the authors, using a sub-group from the original study (Resnick et al., 1995), examined the relationship between past abuse, rape characteristics, and levels of cortisol and MHPG (a metabolite of NE). They found that cortisol was related to a prior history of rape or assault, but not with either rape or symptom severity. In contrast, MHPG was associated with rape severity and symptoms of avoidance, but not related to prior history. The results of this study provide further evidence that prior traumatization may have affected the coordination between HPA and catecholamine systems (Yehuda, Resnick et al., 1998).

PTSD also affects the quality of sleep, whereas intrusive thoughts may manifest as disturbing dreams. Even though sleep disturbances in PTSD have been observed clinically, surprisingly few studies have examined the sleep patterns of those with PTSD. In one recent study, female sexual assault survivors with PTSD had severe sleep disturbances that were equal to, or greater than, depressives or insomniacs. They especially had problems with insomnia and chronic nightmares. These chronic

nightmares could also indicate a higher percentage of REM throughout the course of the night. Other problems included sleep latency, sleep efficiency, and use of sleep medication (Krakow, Tandberg, Sandoval, Cutchen & Schrader, 1998).

Yehuda (1999) concludes a recent review by noting that trauma survivors with PTSD may be hypersensitive to environmental and external events, and may hyperrespond, even in the face of non-dangerous stimuli. In the next section, depression, another manifestation of hyperarousal is considered. Although it is opposite of PTSD in many ways, it is frequently a co-morbid disorder.

Depression

"Depression" is a term that is commonly used to describe a wide variety of psychiatric illness. But it is major depressive disorder (MDD) that is the most commonly studied with regard to neurobiological alterations that occur in the aftermath of traumatic events (Levitan, Parikh, Lesage, Hegadoren, Adams, Kennedy, & Goering, 1998). While depression is generally not considered a disorder of hyperarousal, researchers studying it from a physiologic standpoint have noted signs of hyperarousal in some depressed patients. And this may impact the effectiveness of treatment. In one recent study, depressed patients with hyperarousal were less responsive to psychotherapy than were those without this type of activation (Thase et al., 1996).

Patients with major depressive disorder have abnormally high levels of cortisol and low levels of NE (DeSouza, 1995; Owens & Nemeroff, 1993; Shively, 1998; Shuchter, Downs, & Zisook, 1996; Thase et al., 1996). Further, the system that normally keeps these levels in check breaks down with an insensitivity to cortisol feedback (Yehuda, 1998). Low levels of the neurotransmitters NE and serotonin have both been implicated in depression (Bear, Connors, & Paradiso, 1996; Preston & Johnson, 1997; Shuchter et al., 1996). Further, high levels of cortisol are related to severe more depressions (Plotsky, Owens, & Nemeroff, 1998). Older tricyclic antidepressants work on both NE and serotonin levels. The newer class of antidepressants, the selective serotonin reuptake inhibiters (SSRI's), work exclusively on serotonin. SSRI's appear to also increase the responsiveness of the glucocorticoid receptors, perhaps lowering levels of circulating cortisol. Indeed, all types of antidepressants improve HPA function in both human and animal studies (Plotsky et al., 1998).

The sleep patterns of some depressed patients also demonstrate a pattern of hyperarousal. One recent study examined the strength of the relationship between EEG sleep measures and depression symptoms (Perlis, Giles, Buysse, Thase, Tu, & Kupfer, 1997). Using canonical correlation, the authors identified that sleep and depression were essentially and intimately related. Severe depression drastically reduces the amount of time spent in Stage 4 (delta) sleep. Further, depressed patients have more REM sleep, and REM sleep occurs earlier in the night (reduced REM latency), indicating increased arousal (Perlis et al., 1997; Ware & Morin, 1997). Antidepressants, particularly tricyclics and MAO inhibitors, decrease percentage of REM sleep and prolong the latency to first REM sleep. Cognitive-behavioral therapy may also produce these changes (Ware & Morin, 1997). Unlike PTSD, however, there does not appear to be an increase in intrusive

thoughts and nightmares. As noted earlier, intrusive thoughts appear to be related to high levels of NE (Lemieux & Coe, 1995), which are low in depressed patients.

Sensitization has been raised as an explanation for susceptibility to depression observed in those who have experienced trauma in early life. This model holds that early, painful experiences may sensitize persons who subsequently develop depression. Their vulnerability to depression persists and worsens over time, resulting in recurrent dysphoria even when the original stimulus is gone (Kramer, 1993; Post, Rubinow & Ballenger, 1986; Shuchter et al., 1996).

The mechanism for this increased vulnerability has a strong mind-body component, and may be found in the patient's appraisal of events. Adult survivors of childhood abuse have been found to have cognitive distortions about the world (Briere & Elliot, 1994). The abuse experience may have altered their "internal working model," causing them to perceive the world as a dangerous place. They may be more likely to make negative appraisals of the world than they would have if they had not been abused. Not only does this increase their vulnerability to depression, but negative cognitive appraisals lead to the release of cortisol (Lovallo, 1997).

PTSD vs. Depression

Depression and PTSD are often co-morbid disorders, and have some symptoms, such as disturbed sleep, that overlap (Krakow et al., 1998; Perlis et al., 1997). However, even with these areas of overlap, many of the physiological symptoms are exact opposites. These are summarized below (Yehuda, 1998).

Table 1
Comparison of Endocrinology of Major Depression and PTSD

Major Depression	**PTSD**
• Increased cortisol	• Decreased cortisol
• Decreased responsiveness of glucocorticoid receptors	• Increased responsiveness of glucocorticoid receptors
• Decreased sensitivity of HPA to negative feedback	• Increased sensitivity of HPA negative feedback system
• Progressive desensitization of entire HPA axis	• Progressive sensitization of entire HPA axis

One challenging question is how a patient can have both conditions when many of the physiological symptoms are opposites. The answer may lay in our deepening understanding of neuroendocrinology. In one recent study, two groups of patients with PTSD were studied: patients with co-morbid depression, and those who were not depressed. Lower levels of NE were associated with more severe depression, and elevated NE levels were only found in those who were not depressed (Yehuda, Siever, Teicher, Levengood, Gerber, Schmeidler, & Yang, 1998).

In summary, the study of the physiological correlates of PTSD and MDD is still in its infancy. For example, Yehuda (1998) notes that PTSD is something that researchers can describe but not fully explain. The same could also be said for depression. As research progresses in this field, we may be able to answer these questions more thoroughly. In the next section, irritable bowel syndrome, a physical manifestation of hyperarousal, is described.

Irritable Bowel Syndrome (IBS)

IBS is another manifestation of hyperarousal, this time focusing on the perception of pain. Pain is a symptom with a strong mind-body component, making its link to childhood abuse quite plausible. Further, increased somatic complaints tend to co-occur with PTSD (Beckham, Moore, Feldman, Hertzberg, Kirby, & Fairbank, 1998; Shalev, Bleich & Ursano, 1990), and major depression (Shuchter et al., 1996). Of all pain syndromes described in the medical literature, the link between childhood abuse and IBS is perhaps the clearest.

Irritable bowel syndrome is a disorder of the lower gastrointestinal tract whose cause is currently unknown. Traditionally, IBS is a diagnosis of exclusion, made after ruling out possible organic causes of the symptoms. Symptoms include abdominal pain or cramping; diarrhea or constipation; and bloating or abdominal distention. The symptoms can be continuous or recurrent, and must be present for at least 3 months (American Gastroenterological Assocation, 1997). Because IBS is functional (i.e., no laboratory findings confirm its existence), occurs mostly in women, and tends to co-occur with psychiatric illness, some have considered it predominantly a psychological problem (Blanchard, 1993; Roy, 1998; Whitehead & Crowell, 1991). However, some intriguing neuroendocrine and cerebral abnormalities have also been observed.

One theory of the cause of IBS is visceral hypersensitivity (Wingate, 1991). The viscera appear to be exquisitely sensitive to internal and external stressors (Scarinci, McDonald-Haile, Bradley & Richter, 1994). Patients with IBS have been shown to have increased awareness of bodily sensations and lowered pain thresholds (Drossman, Whitehead & Camilleri, 1997; Munakata, Naliboff, Harraf, Kodner, Lembo, Chang, Silverman & Mayer, 1997). Further, IBS patients have demonstrated an increased sensitivity to life stressors (Levy, Cain, Jarrett, & Heitkemper, 1997). Researchers have also observed the relationship between hypersensitivity and cerebral activity. Following delivery of a painful stimuli, the healthy subjects, in one recent study, had activity of the anterior cingulate cortex. The patients with IBS, on the other hand, had an aberrant brain activation pattern, with activation of the left prefrontal cortex, and no activity of the anterior cingulate cortex (Silverman, Munakata, Ennes, Mandelkern, Hoh, & Mayer, 1997).

Gastroenterologists have conducted a number of studies on the relationship between past abuse and current IBS. A recent position statement by the American

Gastroenterological Association (1997) identified a history of physical or sexual abuse as having a "strong effect" on the outcome of treatment for IBS (p. 2118).

IBS was significantly related to abuse in child or adulthood in two recent studies. Those who had an abuse history were twice as likely to have IBS as the remainder of the sample. Those who reported abuse in both child and adulthood were three times as likely to have IBS (Talley, Fett, & Zinsmeister, 1995; Talley, Fett, Zinsmeister & Melton, 1994). Moreover, patients with IBS were more likely to report a history of threatened sex, incest, forced intercourse and frequent physical abuse than were patients in treatment for organic gastroenterological illness (Drossman, Leserman, Nachman, Li, Gluck, Toomey & Mitchell, 1990). Similarly, patients with IBS had higher rates of lifetime sexual victimization (54% vs. 5%), *severe* lifetime sexual trauma (32% vs. 0%), and severe child sexual abuse (11% vs. 0%) than did those with organic gastrointestinal illness (Walker, Katon, Roy-Byrne, Jemelka & Russo, 1993). Finally, women with a history of sexual abuse had more pain, more overall somatic complaints, bed disability days, surgeries, psychological distress and functional disability compared with those who were not sexually abused. But physically abused women had the worst health outcome. Interestingly, patients whose abuse first occurred in childhood did not have worse health outcomes than those whose abuse first occurred as adults (Leserman, Drossman, Li, Toomey, Nachman, & Glogau, 1996).

Some researchers have dismissed somatic complaints, and reports of gastrointestinal symptoms, as somatization, or as being due a depression. But one recent study (Scarinci et al., 1994) found that IBS patients with a history of abuse had altered sensations of pain. Relative to the non-abused patients, abused patients had significantly lower pain threshold levels in response to finger pressure and significantly lower cognitive standards for judging a stimuli as noxious. *These results held even after controlling for psychiatric disturbance.*

Summary

The previous sections described three manifestations of chronic hyperarousal: PTSD, depression, and IBS. Each of these syndromes produce amplified somatic complaints, and high rates of health care utilization (Beckham et al., 1998). Sleep disturbances were present in PTSD and depression, but not in IBS (Perlis et al., 1997; Spielman & Glovinsky, 1997). Interrupted delta sleep can cause soft-tissue pain and may be responsible for somatic complaints in PTSD and depression. Recall, that heightened sensitivity to pain was still occurring even after controlling for psychiatric co-morbidity (Scarinci et al., 1994).

The overlap between these conditions suggest that these may be different expressions of the same underlying process (Veale, Kavanaugh, Fielding, & Fitzgerald, 1991). Some have suggested that the amygdala is involved and becomes hypersensitive (Bear et al., 1996; Southwick et al., 1994). In reviewing the literature, Lovallo (1997) describes how the most important effects of traumatic stress are its effects on the central nervous system. He notes that changes appear to be related to long-term alterations in frontal-limbic connections and alterations in feedback to the central nervous system from the brainstem aminergic nuclei, including the locus ceruleus and raphe nuclei. The raphe

nuclei synthesizes serotonin, which is involved in pain perception, sleep and depression. The locus ceruleus also has a role in sleep and modulating arousal level, and is thought to be responsible for the "fear memories" that are observed (Bear et al., 1996).

Individual variations in manifestations of symptoms are another facet that may further illuminate the processes involved in response to trauma. In the next section, research on two possible explanations for these different manifestations of symptoms are presented.

Individual Differences in the Expression of Symptoms

The human response to overwhelming or chronic stress is diverse and varied. There is not a unidirectional pattern of how stress affects the brain and no one system is likely to be responsible for the diversity of contrasting symptoms in the face of severe trauma (Wright, Master & Hubbard, 1997). We are a long way from a taxonomy of responses to traumatic stress.

Coping with traumatic stress is a process that affects both mind and body. Herman (1992) eloquently describes the effects of trauma in childhood from a perspective of psychological adaptations that a child has to make:

> Repeated trauma in childhood forms and deforms the personality. The child trapped in an abusive environment is faced with the formidable task of adaptation. She must find a way to preserve a sense of trust in people who are untrustworthy, safety in a situation that is unsafe, control in a situation that is terrifyingly unpredictable, power in a situation of helplessness. Unable to care for or protect herself, she must compensate for the failures of adult care and protection with the only means at her disposal, an immature system of psychological defenses. (p. 96)

Yehuda and colleagues also identify many of these same variables as being related to the biological response to stress. They acknowledge that there an interaction of several factors such as the nature of the stressor (type, chronicity, severity, controllability and predictability), and modifiers of stress such as previous stress history, genetic vulnerabilities, social factors and subsequent environmental stressors (Yehuda et al., 1996). In this next section, I describe two potential modifiers of the response to traumatic events: maturation and severity of the experience.

Age of Onset, Brain Maturation and Appraisal of Events

Age of onset of abuse has the potential to influence the types of sequelae that survivors manifest. This can be discussed both in terms of the physiology of the brain, and the development of the ability to evaluate events. Childhood trauma puts children at risk for a variety of difficulties while they are still children, and increases the risk of adult psychopathology. This is due, in part, to the relative plasticity of children's brains. Children also manifest a more global, persistent, and generalized hyperarousal, and appear to respond to a variety of stimuli rather than those specifically related to the trauma itself (Schwarz & Perry, 1994).

Although many assume that abuse that occurs at an earlier age is more harmful than abuse that occurs later, there is not a linear relationship between age and vulnerability to abusive experiences (Finkelhor & Kendall-Tackett, 1997; Maccoby, 1983). Each stage of development has its own vulnerabilities and its own protections.

According to Rutter and Rutter (1993), the human brain is at its most vulnerable in the first five years of life. During this time, many maturational events take place. The main structure of the brain is formed, followed by the development of nerve cells. After the nerve cells are formed, they migrate to their final destination within the brain. Simultaneous to these processes, the neuronal network in the brain is increasing in complexity and number of synapses. Finally, there is selective cell death that acts as a pruning and fine tuning process.

Alterations in the brain can take many forms. Very early damage may lead to overall generalized problems and a general lowering of intelligence. This has been observed in studies of children raised by depressed mothers (O'Hara, 1987). Depression affects the quality of the mother-infant relationship. Poor mother-infant interactions have been observed to be stressful for infants (Cicchetti & Toth, 1998), even resulting in EEG abnormalities (Jones, Field, Fox, Lundy & Devalos, 1997). Children raised with chronically depressed mothers have been found to have lowered IQ scores (O'Hara, 1987) and may be at risk for developing depression as they mature (Cicchetti & Toth, 1998).

Difficulties may also arise when the brain tries to "fix" itself following a damaging event. Faulty "re-wiring" may result in permanent changes that can be as serious as the actual loss of brain substance (Rutter & Rutter, 1993). We must also take into account "neural plasticity," where parts of the brain may take over the functions normally performed by damaged sections. Symptoms may be more pronounced if abuse takes place at a later age because the brain is not as able to have other sections take over for the damaged portion (Bremner, Randel, Vermetten, Staib, Bronen, Maquire, Capelli, McCarthy, Innis, & Charney, 1997).

Assessing specific damage can also be difficult. The same part of the brain may serve different functions at different phases of development because of cell migration and the changing pattern of organization of nerve cells. This means that the effects of early damage to the brain may not appear until several years after the damage occurred. These delayed effects may also account for different symptoms at different ages and children who are asymptomatic at their original assessment (Kendall-Tackett et al., 1993; Rutter & Rutter, 1993).

Early trauma may set into motion the pattern of hypersensitivity to stimuli described earlier. According to van der Kolk (1994), early onset of trauma may interfere with semantic memory and may render an adult particularly vulnerable to post-trauma flashbacks, physiological arousal and kinesthetic memories of the abuse. In a recent study of 77 female victims of child sexual abuse (ages 6 to 16), early age of onset is associated with dissociative and "hyperactive" behavior, bizarre destructive behaviors and anxious depression (Trickett, Reiffman, Horowitz & Putnam, 1997).

The role of appraisal is also crucial in terms of understanding the appearance of symptoms. The cognitive ability to evaluate events is key in the body's response to threat. Cortisol is not released until the event is perceived as noxious (Lovallo, 1997). The cognitive ability to evaluate the motives of the abuser, or to realize that abuse is not part of everyone's life experience, is not likely to occur until children have gained the ability to "de-center" their thinking—around ages 5 to 7. De-centering is the ability to take into account the perspective of another person. Especially with sexual abuse, young children may be protected by their naiveté when the abuse is not physically painful. (Even very young children can understand that a physically painful event is noxious.). Because of cognitive development and the appraisal process, children are also vulnerable after age five (Finkelhor & Kendall-Tackett, 1997).

The vulnerability of the brain in childhood cannot explain all reactions to traumatic events. We must also keep in mind that overwhelming stresses can have similar effects on adults, as seen in the reactions of combat veterans (Yehuda et al., 1996), rape survivors (Resnick et al., 1995) and earthquake survivors (Goenjian, Yehuda, Pynoos, Steinberg, Tashjian, Yang, Najarian & Fairbanks, 1996). One study that specifically compared those whose abuse started in childhood and those whose abuse started in adulthood found no significant difference in symptoms (Leserman et al., 1996).

Severity of Abuse

Severity of abuse appears to influence severity of the symptoms that adult survivors experience. From a biological perspective, it appears to make little difference whether the abuse is physical or sexual, but greater exposure to traumatic events has been related to higher levels of symptoms (Wright et al., 1997). In a study of 73 female sexual abuse survivors, the authors found that PTSD symptoms were related to whether the abuse experience included intercourse (Briggs & Joyce, 1997).

Severity of abuse experience also impacted symptomatology for irritable bowel syndrome. More severe sexual acts were associated with more severe gastrointestinal symptoms (Drossman, et al., 1990; Leserman et al., 1996; Walker et al., 1993). Life-threatening physical abuse and rape led to more severe symptoms than less severe physical or sexual abuse (Leserman, et al., 1996). Further, among those with organic gastrointestinal illnesses, past abuse was related to increased pelvic and abdominal pain (Drossman et al., 1990). An additive effect was also apparent in IBS. Those with a history of abuse in childhood and adulthood were three times more likely to have IBS compared with the rest of the sample (Talley et al., 1994). Those who experienced all three types of abuse (physical, sexual and emotional) had the highest odds ratios for IBS symptoms (Talley et al., 1995).

How Shall We Then Treat?
Implications for Clinical Practice

In previous sections, research was presented that demonstrated the relationship between past abuse and chronic hyperarousal. My focus on physiological variables was in no way meant to minimize psychosocial factors. Indeed, only by considering both can we offer the most effective treatments. For example, depression can be explained both psychologically and physically. As described earlier, traumatic events during childhood can distort a survivor's "internal working model," shaping how they view the world. Survivors may develop a negative attributional style. And negative appraisals increase cortisol levels (Lovallo, 1997). However, negative cognitions (and subsequent depression) respond well to cognitive therapy, thus lowering levels of cortisol.

In treating children, the focus should be on preventing a traumatic-stress reaction from turning into a more chronic form of PTSD, using a mind-body approach. Psychopharmacologic agents can decrease physiological arousal including hyperactivity, distractibility, elevated blood pressure and nightmares. Treatment can also help the child regain a sense of mastery over each aspect of the traumatic experience. Multiple treatment modalities can be used including art and play therapy, desensitization and flooding, and cognitive therapy (Schwarz & Perry, 1994). Practitioners need to also be sensitive to the fact that children will manifest different symptoms at different developmental ages (Kendall-Tackett et al., 1993).

Treatment for adult survivors should also address both psychological and physical reactions. More typically, treatment of adult survivors involves mental health services that are completely separate from medical services. A more holistic approach is called for. Interestingly, it is the gastroenterologists that have led the way in this, in their position statement on the treatment of irritable bowel syndrome (American Gastroenterological Association, 1997).

In this statement, the AGA recommended that practitioners include a mental health professional on the team for the treatment of psychiatric disturbances, such as major depression and a history of abuse, that may interfere with the patients' adjustment to their illness. They went further by stating that the mental health professional should be considered an integral part of the treatment team for the patient's overall care. The authors of this statement noted that dynamic psychotherapy, cognitive-behavioral therapy, hypnosis, relaxation techniques, and biofeedback are effective in reducing abdominal pain and diarrhea, and giving IBS sufferers more effective coping strategies (van Dulmen, Fennis & Bleijenberg, 1996).

Another issue is "health locus of control." This constuct refers to the sense of agency people have in terms of health and health behaviors. People who believe that they have control over their health are more likely to engage in health enhancing behavior and less likely to engage in harmful ones (Norman, Bennett, Smith & Murphy, 1998). Adult survivors have been found to be more likely to engage in unhealthful behaviors including smoking, substance abuse, high-risk sexual behaviors and eating disorders (Briere & Elliot, 1994; Kendall-Tackett, Marshall & Ness, 1999). Further, adult survivors sometimes lack the ability to act self-protectively and may up in harmful

or antagonistic doctor-patient relationships. Doctors may begin to view them as "difficult" and be less likely to offer assistance.

In conclusion, past abuse can influence not only mood states and emotions, but physiological processes as well. The three sequelae described all have an underlying state of hyperarousal and hypersensitivity. Recognizing this fact may help both care providers and adult survivors themselves develop effective treatment plans that address all their concerns.

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