

SPECIAL ISSUE

Are You Sure It's AD(H)D?

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Top-down procedures for diagnosing and treating conditions such as Attention Deficit Hyperactivity Disorder are, simply stated, logically flawed. The symptoms that form the basis for the diagnosis can be caused by myriad of other factors unrelated to bona fide (i.e., neurologically based) Attention Deficit Hyperactivity Disorder. The diagnostic power of the clinically normed, bottom-up electroencephalographic assessment is remarkable and facilitates treatment of the actual causes, neurological and experiential, of children's challenges. Neurotherapeutic treatments of these conditions are often suboptimal because symptom-based protocols are often inaccurate leading to treating the wrong disorder, not the least of which is trying to mollify normal children's behavior.

Introduction

Bessel van der Kolk, keynote speaker at the 2015 International Society for Neurotherapy and Research Conference, reminded us of a statistic that is sobering. He pointed out that several studies have indicated that over 50% of children have been exposed to levels of emotional stress that are in the "trauma" sphere (Van der Kolk, 2015). Although there are socioeconomic and gender variations in these statistics, the 50% threshold appears ubiquitous.

McLaughlin et al. (2013) reported that in a sample of over 6,000 adolescents, a startling 61% reported exposure to at least one potentially traumatic event (PTE), including rape, physical abuse, domestic violence, death of a close friend or family member, and natural disasters. Almost 20% of these children reported three or more such exposures. Of this 61%, 7.3% of females and 2.2% of males fit diagnostic criteria for Post-Traumatic Stress Disorder (PTSD), a rate similar to that of U.S. adults.

We know from the twin studies (e.g., Arseneault et al., 2005) that bullying affects children's mental health and certainly their ability to function in school settings. Data from the U.S. Department of Justice (2011) indicated a bullying frequency of about 25% and cyberbullying of

about 9%, from a survey of about 24 million students. Both victims and perpetrators of bullying are at a higher risk for suicide than their peers. Children who are both victims and perpetrators of bullying are at the highest risk (Hay & Meldrum, 2010).

Children with past and current bullying are most at risk for worse physical and mental health, followed by those children with only present bullying (Bogart et al., 2014). In particular, these children show greater depression symptoms, a classic condition often misdiagnosed as Attention Deficit Hyperactivity Disorder (ADHD) and unsuccessfully treated (P. G. Swingle, 2015a). Bullies and victims and those who are both also have higher frequencies of being victims and/or perpetrators of criminal activity.

In a study of 18,834 adolescents, almost 20% had experienced cyberbullying in the past year. Of that number almost 20% reported depression; almost 5% reported suicide attempts; and about 6% prescription drug abuse (Elgar et al., 2014).

Yehuda et al. (2015) and Siklenka et al. (2015) also pointed out that exposure to traumatic stress can be transmitted epigenetically to one's offspring. Children of Jewish survivors of the Holocaust, for example, show genetic variations not shown in children of Jews who escaped.

So, are you sure it's AD(H)D? Maybe the explosion in the diagnosis and "treatment" of ADHD has to do with factors that have nothing to do with neurological dysfunction affecting attention but rather because we (parents and health and education professionals) are using top-down methods for diagnosis and simply routinely missing the actual causes of children's problems with focus and attention.

And yes, we can identify children who may be experiencing bullying and other forms of trauma. They show markers for exposure to emotional stress (P. G. Swingle, 2013), emotional volatility (P. G. Swingle, 2015b), and reactive depression (Henriques & Davidson, 1991) on

Genetic predispositions	
Monozygotic twins	30-50%
Dizygotic twins	15%
Siblings	15%
General population	1%
Adopted-biological relatives with schizophrenia	
Adoptee with schizophrenia	13%
Adoptee without schizophrenia	2%

the ClinicalQ electroencephalogram (EEG; P. G. Swingle, 2015a) assessment.

Bottom-up Assessment: The ClinicalQ

For clinicians, the most accurate databases are clearly clinical. Normative databases are far less accurate. The fundamental organizing concept of the normative database for the clinical practitioner is, simply stated, logically incorrect.

The organizing concept for normative databases is that one can identify a group of individuals who are symptom-free and therefore have "normal" functioning neurology. This group of symptom-free individuals then serves as the comparative database to identify those who are statistically discriminant. The statistical departures from the normative database define the anomalous neurological condition that is associated with the client's clinical condition. This concept is also logically incorrect.

The reason that normative database treatment recommendations are so often incorrect is because the fundamental premise is wrong. Symptom-free individuals may well have predispositions to conditions that have not manifested. The data are quite clear and we have definitive evidence for this that spans decades.

Let us simply take the example of heritability data for schizophrenia (similar data are available for other conditions as well, such as vulnerability to PTSD and bipolar disorder). As the data in the Table (Ginsberg & Cancro, 1985; Gottesman, 1991; Gottesman & Shields, 1972) indicate, if one monozygotic twin has diagnosed schizophrenia, the probability that the second identical twin will have schizophrenia is about 50%. So, the schizophrenic ends up in the ClinicalQ database. But, the interesting statistic is that 50% will not! Where do we find the 50% without manifested schizophrenia, but

obviously with the same genetic load? In the normative databases. So clearly the organizing concept for normative databases, at least for clinicians, is incorrect. Normative databases so constituted ignore basic psychopathology and basic biology. Every person has predispositions—predispositions to anxiety, depression, emotional volatility, and the like. However, many of these predispositions are not manifest at any particular time. In general, clinicians understand that one needs a trigger to "turn-the-key" to manifest a neurological predisposition.

These logic considerations are well known and, surprisingly, ignored by the developers of the normative databases. If in the normative database one has subjects with nonmanifested predispositions, then statistically one can expect very poor discrimination. The normative databases are going to be statistically blind to manifested predispositions that bring clients into our offices. Related to this problem is that the data collection procedures ignore conditions that expose clinically relevant information. An example of this is ignoring the Alpha blunting response that is the cardinal marker for exposure to severe emotional stress (P. G. Swingle, 2013).

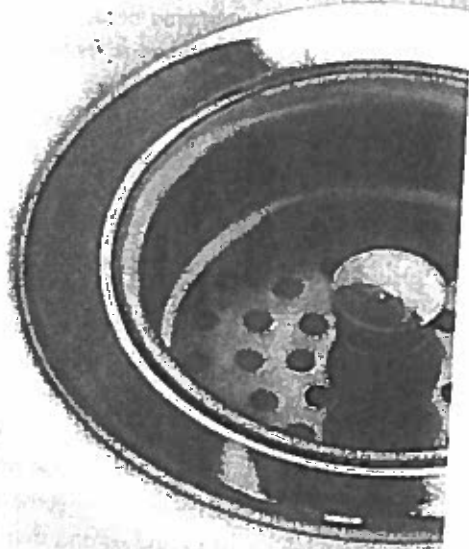
Clinical databases, on the other hand, permit quite remarkable accuracy in revealing the fundamental causes and exacerbating factors contributing to a client's morbidity. Clinical databases compare an individual's quantitative EEG (QEEG) patterns to aggregated data from the brains of patients with a variety of well-diagnosed clinical disorders. Instead of providing a summary of how different this individual's EEG is from some hypothetical "normal" brain, a clinical database highlights similarities in cortical activation patterns to groups of patients with known clinical disorders.

We, of course, ask clients about their condition, but these reports are often quite inaccurate as to causality. A good example is the client report of depression that is more accurately despair associated with debilitating anxiety. Treating depression instead of anxiety with antidepressant medications or left frontal Alpha brain-wave amplitude suppression is therefore likely to be marginally effective because the wrong condition is under treatment.

The ClinicalQ database is a clinical database developed by Paul G. Swingle with a present sample of over 1,500 quantitative five-site EEGs. The database is managed by the Swingle Clinic: Supporting data and clinical application is available to clinicians in P. G. Swingle (2015a).

The accuracy of the ClinicalQ database is nicely captured in the following excerpt:

"...combines the visceral force of lived experience with the nuance and narrative drive of the best fiction." Nina Ricci



PATHOLOGIES

A Life in Essays

SUSAN OLDING

Figure 1. Susan Olding's book, *Pathologies* (2008).

Desperate, determined, undeterred by cost or lack of insurance coverage, undismayed by the doubts of conventional physicians ... I switched off my cell phone at the threshold of Dr. Swingle's office and carried my daughter into the office, ...

I had brought a medical and developmental history—the long history of concerns that had brought us to this doctor—but Dr. Swingle waived the papers as he would never look at them. Instead, he ushered Maia toward a computer screen. ...

my response. I nodded. "And here," he continued, "too much Theta. This is the hyperactivity people associate with ADHD ... There was more extreme stubbornness, a tendency to perseverate, lapses of short-term memory, attachment disorder, inability to read social cues, emotional reactivity, tantrums, explosions. One by one he read the ratios, divining my daughter's character more quickly, more accurately than any professional I'd yet encountered.

—Susan Olding, 2008, pp. 169–173, reprinted with permission

The assessment described by Susan Olding is the *ClinicalQ* that is based on a few minutes of recording time. The clinical database then offers direction for the clinician's probing the client regarding anomalies in functioning that may be the bases for the clinical condition.

The Case of the Kelly Family

Mrs. Kelly brought in her two children, Jane who was seven years of age and Martin who was nine years of age, for treatment of what her family physician thought was ADD with both children. Fortunately for Mrs. Kelly, her family physician was strongly opposed to medicating children for ADHD, unless absolutely necessary. It may well have been that this very vigilant physician was suspicious that the problem with the children resided in problems with the family and that medicating this problem would be totally inappropriate.

The *ClinicalQ* showed that both children had minor excesses of Theta/Beta ratio at location Cz indicating very mild ADHD. However, both showed blunted Alpha responses at locations O1 and Cz, indicating exposure to severe emotional stress (the "trauma" markers, P. G. Swingle, 2013).

We often find that children who have severe attention problems show the trauma marker. It is possible that the trauma is associated with fear of failure and humiliation in school associated with their attention and/or learning problems. However, whenever we see this pattern in children, we always determine if the child is being exposed to marked emotional stressors. This could be bullying, it could be family strife, or it could be some form of abuse. So in addition to neurotherapeutic treatment for the ADHD, we have to determine the cause of the Alpha blunting.

There were several important issues to consider here. First, given that we saw this marker with both children, it is possible that we were dealing with a genetic factor. Although Alpha blunting is highly correlated with exposure to severe emotional stressors (P. G. Swingle, 2013), nonetheless, although rare, we do find it in

situations in which there is no apparent present or historical exposure to emotional trauma.

The second issue is how we would approach the mother in a manner that would not make her bolt from the therapeutic situation or make her severely distraught about her children's well-being. If there were no context in which this parent was aware of severe emotional stress, this kind of information could be clearly distressing. Parents immediately think about bullying, sexual predators, and other forms of abuse to which children might be exposed. It is extraordinarily important for the therapist to be able to deal with this situation in a manner that is rational and systematic.

The third issue is that healthcare providers have an obligation to report to the authorities any potential harm to a child. However, we had no direct evidence of this other than the EEG data. Recognizing that the parent might be the perpetrator, careful and prudent probing of the parent regarding the various conditions under which the emotional stress may occur, or have occurred, was required.

When I broached the subject of the children showing signs of being exposed to severe emotional stress, Mrs. Kelly broke down and admitted that there were severe problems in the family. According to Mrs. Kelly, her husband vacillated between severe depression and severe emotional abuse. He "flew off the handle" with minimal provocation, was heavily medicated, and she felt that the children were severely disturbed by her husband's behavior. Mrs. Kelly agreed to let me measure her brainwave activity.

Mrs. Kelly's EEG also showed the marker for exposure to severe emotional stress, just as her children's. Her brain assessment also showed mild markers for problems with attention, again, just as her children, so she may be the source of the ADD markers that we find in both of her children. There were several other features of Mrs. Kelly's EEG that are important to note. The first was that she had a major marker for predisposition to depressed mood states. The amplitude of Beta activity was markedly greater in the right prefrontal cortex relative to the left. Whether the depression of Mrs. Kelly contributed to the family dynamic issue or whether it was the result of her exposure to the abusive behavior of her husband, nonetheless, children whose mother is severely depressed are profoundly more likely to have emotional/behavioral problems.

It seemed obvious that we were dealing with a family in crisis. Both of the children and Mrs. Kelly showed markers for exposure to severe emotional stress (the blunted Alpha trauma markers). Mrs. Kelly also showed a major marker for predisposition to depressed mood states and, on her

intake self-report assessment, she described herself as being one who falls into depression easily. Mrs. Kelly's description of her children's behavior, likewise, suggested that these children had some emotional difficulties. She described Jane as easily upset, quick to anger, and unable to engage in cooperative play because she always must win. Mrs. Kelly described Martin as being very anxious, unresponsive to others' feelings and, importantly, she described him as having behaviors associated with Internet addiction (addiction to video games). Internet addiction is an extraordinarily serious problem that is largely unrecognized by parents. This problem will be discussed later.

Our ability to diagnose the problem with the Kelly children as being primarily a problem with family strife testified to the remarkable facility of the ClinicalQ EEG as a diagnostic instrument. Most importantly, however, it pointed out that other therapeutic strategies had to be put in place to assist this family. Changing the neurology of the situation was important, but it was a minor component associated with the treatment of these children. It was extremely important to understand that family therapy and treatment of the parents would be equally as important as any kind of neurological work that we might do with the children.

We were most fortunate that Mr. Kelly not only recognized that he had serious problems but he also willingly came in for the ClinicalQ EEG assessment.

Mr. Kelly described himself as follows: "I fly off the handle at minor problems. I'm anxious, depressed and fatigued. I am on major medications including Wellbutrin, Cipralex, and Ativan that are not very effective. And I've been on other mixes of medications, all of which may have helped somewhat, but eventually lost their effectiveness. I know that my behavior has seriously affected my marriage, my children, and my wife."

Mr. Kelly's ClinicalQ showed that he had an ADHD condition and a particularly nasty form, at that. He had marked elevation of Alpha amplitude in the front part of the brain (locations F3 and F4). The high frontal Alpha form of ADHD is characterized by problems with planning, organizing, sequencing, and following through on things (P. G. Swingle, 2010). However, more importantly in this case is that high amplitude frontal Alpha is associated with emotional dysregulation. These individuals can have marked emotional volatility, problems with emotional impulse control, and difficulty sustaining emotional stability. Clients with this neurological condition are often diagnosed with bipolar disorder, personality disorder, and anxiety disorders in addition to ADHD (P. G. Swingle, 2015a).

Although this situation was severe and complex, I am really tempted to take out the "Cured" stamp at this point! Whenever one has clients who are willing to present themselves for treatment and are open and candid about their problems and their potential influence on other individuals, the prognosis is extraordinarily good for a favorable outcome. We will have continued challenges in dealing with this situation, of course. The challenges are not only neurological but behavioral in nature. Martin, for example, had developed a dependency on video games. This provided an escape and stimulation for this child and it would be very difficult to wean him from this addictive behavior. We will discuss the problem of Internet addiction later in this article.

The ClinicalQ identified that the cause of the Kelly children's academic difficulties were, if not caused by, then certainly markedly exacerbated by, not their ADHD, but Mr. Kelly's ADHD! Mr. Kelly was being pharmaceutically treated for the wrong condition. Hence proper treatment for Mr. Kelly turned out to be the effective treatment for his children's academic difficulties. The children did have some neurotherapy to correct the minor excesses of slow frequency amplitude associated with the inattentive form of ADD (Swingle, 2001). And weaning Martin off of his addiction to Internet gaming was difficult and required some parenting assistance.

As the above case indicates, for those of us who actually treat patients/clients, QEEG statistical discriminations based on normative databases are not adequate for clinical practice. Discriminations based on the normative databases are simply statistically blind to many of the important neurological features associated with the clinical condition of clients. Clinical databases, such as that used in the ClinicalQ, are far more efficient for identifying manifested predispositions and experiential factors that are fundamental to the efficient neurotherapeutic treatment of our clients. Clinical databases are also far more efficient in identifying conditions that require therapies other than neurotherapy. The normative databases for QEEG provide very useful and important information. For efficient clinical practice, however, it must be augmented by discriminative comparisons with clinical norms.

Returning to the role of screen-based technologies on ADHD symptoms, research over the past two decades has unequivocally shown strong alliances of excessive or addictive usage of screens to both emotional distress and the maintenance or development of ADHD, or ADHD-like symptoms (M. K. Swingle, 2015a). Earlier in this article, we reviewed research showing that emotional distress is often an unidentified source of ADHD symptomatology. So too i-

technologies contribute significantly to ADHD and ADHD-like symptoms. Here we may indeed have the makings of the perfect storm for the development of ADHD.

At the turn of the 21st century, maladaptive i-tech usage was typically associated with gaming and male children and was rarely on the clinical radar as a cause or contributor to educational or behavioral difficulties (Block, 2008; M. K. Swingle, 2015a, 2015b). In the case of Martin, above, it is highly likely that excessive gaming was first used as a retreat from family dynamics. In turn, said excessive exposure to gaming triggered the expression of liabilities common to those with ADHD. Following epigenetic theory, two such environmental triggers were conceivably the catalysts for the symptoms for which the family originally sought psychological services (see M. K. Swingle, 2015a).

Today, these problems are relatively gender equal and, with the exception of juvenile addiction to pornography viewing, choice of content has little or naught to do with levels of experiential distress or alliance with specific disorders such as ADHD. Children deregulate equally with excessive viewing or searching on *YouTube* as they once did with videogames. Similarly incessant texting affects arousal cycles much the same as gaming, present and past. In sum, the development of an attentional difficulty is related to the medium or process itself not the content it delivers.

The central reason excessive usage of i-tech becomes aligned with the development and maintenance of ADHD is directly related to the effects of screen-based activities on the development of inhibitory process—or lack thereof. Almost all activities mediated by screens have varied reinforcement schedules, opportunities for increasingly stimulating content, and the ability to switch content instantaneously when no longer sufficiently entertained (see multiple early works of Greenfield, 1999a, 1999b). In sum they propagate the development of reduced attention cycles on that which is not immediately stimulating or arousing (M. K. Swingle, 2015a).

The result is that the brain becomes entrained to require higher stimulation to engage. By proxy, the brain then becomes increasingly incapable of maintaining focus on the standard or arguably mundane, such as the learning of school lessons (e.g., learning to read, scribe, and do math equations) and routine (e.g., organizing one's backpack for school, remembering to write down homework assignments, and turn them in once done).

Part B to this can be the development of emotional regulation issues wherein i-tech has become a source of emotional "brain fuel" without which a child, just like an

adult, becomes emotionally deregulated, if not overtly distraught with its absence. For example, when children get what they deem as insufficient screen time, they protest, tantrum, beg for more, or walk around aimlessly, agitated, and bored. Here, due to previous excessive exposure and entrainment, the brain, and hence the child, has lost the ability to self-stimulate or self-entertain (M. K. Swingle, 2015a).

A caveat of the digital era is that as consistent applications of i-tech become more and more of a societal norm, negative and excessive use directly associated with emotional and cognitive challenges, are increasingly hard to differentiate from healthy or integrated usage and ailments or disorders that a child would develop regardless (M. K. Swingle, 2015a). In the face of this challenge, many, for example Gold (2015), suggest modified i-tech parameters for children who are already diagnosed with ADHD, anxiety, or depression. Others state that children in clinical classifications are not proper measures for the general population, being significantly more susceptible (See Weiss & Schneider, 2014). Yet others classify children in titles such as "orchids" versus "dandelions," implying that some children will be significantly more sensitive to deleterious effects of standard usage than others (see Dobbs, 2009). Duly noted, however, there are also years of prior research revealing the importance of predisposing environments, predisposing personality traits, and risk factors that arguably apply to many, rather than few (e.g., isolation and family dynamics, sensation seeking, impulsivity, reduced attentiveness, introversion, shyness and low self-esteem, need to escape from family discord, loneliness, guilt and need to detract from or seek to ease anxiety, depression, social phobias). Newly on the radar is now the classification of "gifted" (see Young & Nabuco de Abreu, 2011, or M. K. Swingle, 2015a, for syntheses on the supporting literature).

This brings us full circle to the primary thesis of this article: that ADHD symptoms can have multiple emotional/functional rather than solely cognitive sources as can be seen on the EEG (when compared to a clinical database). To this list one should add process: for many children, increasingly, attentional difficulties, and true blue ADHD can be due to overexposure to i-mediated screen activities that alter developmental processes regulating inhibitory mechanisms. This altered development, due to environmental influence rather than solely biological inheritance, is implicated in the development or maintenance of ADHD symptomatology.

References

- Arseneault, L., Milne, B. J., Taylor, A., Adams, F., Delgado, K., Caspi, A., & Moffitt T. E. (2008). Being bullied as an environmentally mediated contributing factor to children's internalizing problems: A study of twins discordant for victimization. *Archives of Pediatric and Adolescent Medicine*, *162*(2), 145-150.
- Block, J. J. (2008). Issues for DSM-V: IA. *American Journal of Psychiatry*, *165*, 306-307.
- Bogart, L. M., Elliott, M. N., Klein, D. J., Tortolero, S. R., Mrug, S., Peskin, M. F., Davies, S. L., et al. (2014). Peer victimization in fifth grade and health in tenth grade. *Pediatrics*, *133*, 440-447.
- Dobbs, D. (2009). The science of success. *Atlantic Monthly*. Retrieved from <http://www.theatlantic.com/doc/200912/dobbs-orchid-gene>
- Elgar, F. J., Napolitano, A., Saul, G., Dirks, M. A., Craig, W., Poteat, V. P., Holt, M., et al. (2014). Cyberbullying victimization and mental health in adolescents and the moderating role of family dinners. *JAMA Pediatrics*, *168*(11), 1015-1022.
- Ginsberg, G. L., & Cancro, R. (1985). Schizophrenia: The epigenetic puzzle. *The Psychoanalytic Quarterly*, *54*, 305-306.
- Gold, G. (2015). *Screen-smart parenting*. New York: Guilford Press.
- Gottesman, I. I. (1991) *Schizophrenia genesis: The origin of madness*. New York: Freeman.
- Gottesman, I., & Shields, J. (1972). *Schizophrenia and genetics: A twin study vantage point*. Boston: Academic Press.
- Greenfield, D. N. (1999a). Psychological characteristics of compulsive Internet use: A preliminary analysis. *Cyber Psychology and behavior*, *2*(5), 403-412.
- Greenfield, D. N. (1999b). The nature of internet addiction: Psychological factors in compulsive internet use. Paper presented at the 1999 American Psychological Association Convention, Boston, MA.
- Hay, C., & Meldrum, R. (2010). Bullying victimization and adolescent self-harm: Testing hypotheses from general strain theory. *Journal of Youth and Adolescence*, *39*(5), 466-459.
- Henriques, J. B., & Davidson, R. J. (1991) Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, *100*, 535-545.
- McLaughlin, K. A., Koenen, K. C., Hill, E. D., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2013). Trauma exposure and posttraumatic stress disorder in a national sample of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*(8), 815-830.
- Olding, S. (2008) *Pathologies*. Calgary, AB: Freehand Press.
- Siklenka, K., Erkek, S., Godmann, M., Lambrot, R., McGraw, S., Laffleur, C., Cohen, T., et al. (2015). Disruption of histone methylation in developing sperm impairs offspring health transgenerationally. *Science*, *350*(6261), aab2006. doi:10.1126/science.aab2006
- Swingle M. K. (2015a). *i-minds: How cell phones, computers, gaming, and social media are changing our brains our*

- behavior, and the evolution of our species. Portland, OR: Inkwater Press.
- Swingle, M. K. (2015b). What is i-technology really doing to our brains? *Psychophysiology Today*, 10(1), 5-8.
- Swingle, P. G. (2001). Parameters associated with rapid neurotherapeutic treatment of common ADD (CADD). *Journal of Neurotherapy*, 5, 73-84.
- Swingle, P. G. (2010). *Biofeedback for the brain*. Piscataway, NJ: Rutgers University Press.
- Swingle, P. G. (2013). The effects of negative emotional stimuli on alpha blunting *Journal of Neurotherapy*, 17(2), 133-138.
- Swingle, P. G. (2015a). *Adding neurotherapy to your practice*. New York: Springer.
- Swingle, P. G. (2015b) *When the ADHD diagnosis is wrong*. Santa Barbara, CA: Praeger.
- U.S. Department of Justice. (2011). *National Crime Victimization Survey (NCVS): School Crime Supplement (SCS)*. Bureau of Justice Statistics. Retrieved from <http://www.icpsr.umich.edu/icpsrweb/RCMD/studies/33081>
- Van der Kolk, B. (2015, October). *Developmental trauma*. Keynote address to the annual meeting of the International Society for Neurotherapy and Research, Denver, CO.
- Weiss, R., & Schneider, J. (2014). *Closer together, further apart*. Carefree, AZ: Gentle Path Press.
- Yehuda, R., Daskalakis, N. P., Bierer, L. M., Bader, H. N., Klengel, T., Holsboer, F., & Binder, E. B. (2015). Holocaust exposure induced intergenerational effects on FKBP5 methylation. *Biological Psychiatry*, (in press). doi:10.1016/j.biopsych.2015.08.005
- Young K. S., & Nabuco de Abreu, C. (2011). *Internet addiction: A handbook and guide to evaluation and treatment*. Hoboken, NJ: Wiley Press.



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