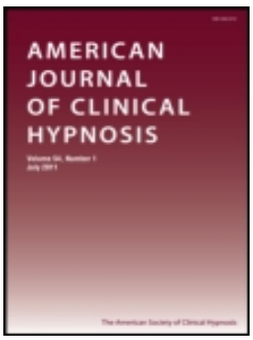

American Journal of Clinical Hypnosis

ISSN: 0002-9157 (Print) 2160-0562 (Online) Journal homepage: https://www.tandfonline.com/loi/ujhy20

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To cite this article: Laurence I. Sugarman , Brian L. Garrison & Kelsey L. Williford (2013) Symptoms as Solutions: Hypnosis and Biofeedback for Autonomic Regulation in Autism Spectrum Disorders, American Journal of Clinical Hypnosis, 56:2, 152-173, DOI: 10.1080/00029157.2013.768197

To link to this article: https://doi.org/10.1080/00029157.2013.768197

American Journal of Clinical Hypnosis, 56: 152–173, 2013

ISSN: 0002-9157 print / 2160-0562 online
DOI: 10.1080/00029157.2013.768197

Routledge
Taylor & Francis Group

Symptoms as Solutions: Hypnosis and Biofeedback for Autonomic Regulation in Autism Spectrum Disorders

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The Autonomic Dysregulation Theory of autism posits that a phylogenetically early autonomic defect leads to overarousal and impairments in language and social engagement. Cognitive rigidity and repetitive behaviors manifest as mitigating efforts. Focusing on the implications of this premise may provide more productive therapeutic approaches than existing methods. It suggests that self-regulation therapy using hypnosis and biofeedback should be highly effective, especially for young people. Hypnotic strategies can utilize restrictive repetitive behaviors in trance as resources for comfort and control. Biofeedback training can be tailored to focus on autonomic regulation. The authors develop this theory and describe methods of integrating hypnosis and biofeedback that have been therapeutic for people with autism. Directions for future research to validate this approach are discussed.

Keywords: autism, autism spectrum disorder, autonomic regulation, biofeedback, hypnosis, repetitive behaviors

The growing prevalence of children being diagnosed with autism spectrum disorders (ASD)[1] and the wide range of phenotypic variation have not yet been explained by a unified theory of causation. Most therapeutic strategies have focused primarily on changing specific core symptoms without considering that they may represent something more common and fundamental: a reach for homeostasis through self-involved behaviors. The authors believe that *all* people, especially young people, who struggle with a chronic disorder, strive in this way. Individuals with ASD, however, manifest an increased intensity of these behaviors that compels two questions: (1) Why is there a more intense need for self-regulation through self-involved behaviors in so many children with ASD? (2) How can we best utilize that need therapeutically? The authors posit that a defect in autonomic regulation may provide that broad theoretical foundation for most features of ASD and their treatment. This treatment focuses on utilizing the self-regulatory efforts of young people with ASD by employing hypnosis and biofeedback strategies. In this article the authors: (1) critically review the nature of ASD as it is typically conceptualized; (2) focus on why an Autonomic Dysregulation Theory may be the most comprehensive of current

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hypotheses with regard to both causation and challenging behaviors; (3) outline treatment strategies based on this theoretical framework and preliminary efforts to employ them; and (4) propose research directions to explore the validity of this theory.

Clinical Vignette

The following vignette describes a boy with ASD whose narrowed interests are utilized therapeutically for self-self-regulation. It presents a child from the first author's practice whose name has been changed. It is included to bring to life the concepts and clinical strategies discussed in this article.

Ten-year-old Stefan manifested social awkwardness and avoidance, repetitive behaviors (including foot tapping and rocking, intensified, narrowed areas of interest), and qualitative language impairments with abnormalities of prosody and fluency (monotone and stuttering). As these symptoms emerged during his early childhood, his primary care pediatrician suspected that he met criteria for a diagnosis of ASD. This was confirmed in consultation with a developmental and behavioral specialist. Comorbid conditions included fine motor delays with dysgraphia, irritable bowel syndrome manifested as abdominal pain with a varied stool pattern, and sleep initiation problems because he worried about "what will happen tomorrow." At home he had "meltdowns" (screaming, shaking, foot-stamping, and withdrawal) when his routine is disrupted. This occurred approximately five times weekly. His parents did their best to prepare him for transitions but unexpected changes in plans could not be avoided. They were particularly concerned about the effect Stefan's rigidity and outbursts had on his 8-year-old sister at whom he "lashed out" when he felt disturbed. At school he frequently needed breaks from the classroom to "calm down" when his repetitive movements distracted his classmates. Even with these interruptions he maintained excellent grades, mastering most subjects with the exception of music, art, and physical education. He found core academic subjects "easy and boring." At school he received occupational and speech therapy and participated in a social skills group that he called "stupid and boring." Over 5 years prior to presentation, he had been treated with sertraline, fluoxetine, methylphenidate, clonidine, and risperidone in increasing dosages and in various combinations for his symptoms of anxiety and intolerance of change. These medications resulted in little significant benefit, limiting side effects, or both. His parents had recently engaged the family in therapy. It was suggested by the family therapist that Stefan receive individual therapy to help him develop coping strategies.

At his initial visit, Stefan began by asking if we were going to talk about "narrow gauge rail in the Southwest." He went on to discuss the various railroad companies, routes, track sizes, and history of this subject. Over the next four visits spanning 6 weeks Stefan learned: (1) how to keep the part of his imagination that "enjoys knowing railroads" and other areas of interest running in the back of his mind "like a radio or TV station" and providing him comfort, especially in novel situations; (2) how to use abdominal breathing and tuning into his "radio or TV station" to change his heart rate

variability and lower his skin conductance with computerized biofeedback; (3) how to link his use of imagination with his biofeedback training so that triggers like his sister's voice or coming into his classroom at school help him feel comfortable and "the boss of his mind" automatically; and (4) how to use those same skills to "smooth out my intestines" and "imagine my tomorrows in advance" at bedtime so that he could experience getting through the unexpected with calm and accomplishment. At school his motor symptoms diminished significantly and he required fewer breaks from the classroom. When he took breaks, he did so to "use my skills." He also shared them with his parents and the social skills group at school. At home his meltdowns grew shorter and his parents sometimes asked him whether he chose to have a meltdown or "use your skills." He had no more symptoms of irritable bowel syndrome. His interests grew broader and included understanding transportation systems.

ASD Characteristics and Anxiety

ASD is defined as a syndrome with varying degrees of impairments in social reciprocity, domains of language, and restrictive interests/repetitive behaviors (American Psychiatric Association, 2013). Social impairments are expressed in a continuum from apparent absolute disregard for others, to lack of shared attention, to difficulty interpreting facial expression and social cues, to an air of aloofness. Communication deficits are characterized by delayed development of verbal skills, problems with prosody, echolalia and stereotyped speech, pronoun reversal, and difficulty with creative expression of thoughts and feelings. Cognitive rigidity manifests by repetitive physical, often inappropriate, behaviors (spinning, twirling, rocking, tapping), narrowed perseverative foci of attention and interests, and resistance to change (Bodfish, Symons, Parker, & Lewis, 2000). This range of behaviors is commonly referred to as "Restrictive Repetitive Behaviors" (RRBs). All symptoms range widely in severity. Relationships that stem from areas of shared interest develop some social reciprocity. Language impairment can be minimal. Narrow areas of interest may be viewed as strengths or talents: domains of depth and expertise. Thus, high functioning individuals with ASD may appear initially to have fewer symptoms but still have functional social impairments that impact their employment, peer and family relationships, and overall quality of life.

Because of the variations in level of impairments of these core areas, ASD has been viewed as a spectrum from "Kanner" autism to high functioning autism and Asperger Syndrome. Ironically, the heterogeneity of ASD has become both defining and problematic. In a recent survey researchers found 47% of children with ASD are diagnosed with Pervasive Developmental Disorder–Not Otherwise Specified (PDD–NOS), referring to the incomplete or atypical expression of core symptoms (Centers for Disease Control and Prevention, 2012). Similarly, in a parental survey, only one of the 11 characteristics (language delay) was met by greater than 75% of the sample of children with ASD. This characteristic also had the largest disparity between ASD types, with autism and PDD–NOS both over 90% and Asperger Syndrome falling below 50% on this measure

(Goin-Kochel & Myers, 2004). This wide variation in phenotype drives the need for a more fundamental and unifying theory of ASD.

While there is great variation in core symptoms, anxiety is very common (Baron, Groden, Groden, & Lipsitt, 2006; Kinsbourne, 2011) and, we argue, more consistent than traditionally cited core symptoms. Neither recognized as a core symptom nor unique to ASD, the high prevalence raises the question of whether anxiety is emergent or represents a core defect in autonomic regulation. Evidence for increased sympathetic tone, even in the "resting state," among subjects with ASD (Ming, Julu, Brimacombe, Connor, & Hansen, 2005; Porges, 2011; Toichi & Kamio, 2003) suggests that anxiety is not merely emergent but part of the causal mixture. We propose that the role of anxiety in ASD is key to understanding the pathogenesis of challenging behaviors.

A Bottom-Up Strategy

If one conceives of the varied symptoms of ASD as deriving from a unifying dysfunction, it appears that most existing theories of ASD have, to some degree, implicated a given branch and identified it as the trunk. However, autonomic dysregulation may be a more basic deficit from which the core symptoms of ASD originate. Table 1 organizes some current theories of ASD by their association with the symptom triad; we add autonomic regulation to account for the ubiquity of anxiety noted earlier. Existing theoretical constructs for the symptoms of ASD overlook the role of anxiety, or acknowledge it only as emergent or comorbid. Our proposed Autonomic Dysregulation Theory includes anxiety as foundational rather than emergent. We propose that focusing on the early-developing autonomic nervous system (ANS) instead of late-developing frontal dysfunctions provides a starting point for cascading effects that branch out to higher-level systems and as so accounts for both core and comorbid ASD symptoms.

TABLE 1
Differing Theories and Their Account for the Major Deficits Found in ASD

	ASD Characteristics			
Theory	*Social Engagement*	*Communication*	*Cognitive Flexibility*	*Autonomic Regulation*
Executive functioning	Secondary	Secondary	Core	Unexplained
Weak central coherence	Secondary	Secondary	Core	Unexplained
Empathizing-systemizing	Core	Secondary	Core	Unexplained
Social motivation	Core	Secondary	Secondary	Unexplained
Mirror neuron	Core	Unexplained	Secondary	Unexplained
Autonomic dysregulation	Secondary	Secondary	Secondary	Core

Note. While other theories overlook the role of autonomic regulation, the Autonomic Dysregulation Theory implicates a core deficit in this system that underlies the defining characteristics in ASD.

Precedent for looking at early autonomic insults comes from animal models, which have implicated early ANS defects in the development of arrhythmias (Cogliati et al., 2002), psychopathology (Beauchaine, 2001; Lang, Davis, & Ohman, 2000), and stress-related phenomena (Sutano & de Kloet, 1994). Recent evidence from childhood abuse victims shows epigenetic effects on autonomic regulation (Klengel et al., 2012), demonstrating lasting effects that stem from early ANS development. Recognizing that the ANS (the primary regulator of stress) is foundational in development provides a more evolutionarily rooted model of this disorder.

Autonomic Dysregulation Theory

We coin the term "Autonomic Dysregulation Theory" as an extension of the Overarousal Theory of ASD (Bergman & Escalona, 1949; Hutt, Hutt, Lee, & Ounsted, 1964). Kinsbourne (2011) and Porges (2011) inform development of the original ideas along with our experience utilizing hypnosis and biofeedback for autonomic self-regulation. We begin by examining RRBs as compensatory for increased sympathetic arousal. Then we explore neurophysiological manifestations of sympathetic hyperarousal observed in individuals with ASD. Finally, positing that hyperarousal is a manifestation of autonomic dysregulation, we describe how autonomic abnormalities can account for the core symptoms of ASD and common comorbidities.

Restrictive Repetitive Behaviors Are Compensatory

Restrictive and repetitive behaviors in ASD can be viewed as part of a continuum of stress reducing activities observed throughout the animal kingdom. Hinde (1970) described displacement behaviors as normal operations that, in response to stress, are " 'displaced' from their usual role in the behavioral repertoire" (Kinsbourne, 2011, p. 374; see also: Kinsbourne, 1980). Most commonly observed in animals and non-human primates in response to confinement (Bachmann, Bernasconi, Herrmann, Weishaupt, & Stauffacher, 2003; Berkson & Mason, 1963; Dickens & Romero, 2009; Floeter & Greenough, 1979; Garner, Meehan, & Mench, 2003; Korda, 1978; Melzack & Burns, 1965), these behaviors "are without perceptible purpose in the context in which they occur" (Kinsbourne, 2011, p. 374) other than to reduce stress. Ritual behavior in humans has been associated with apprehension of dangers and is correlated with anxiety and fearful traits (Boyar & Liénard, 2006; Zohar & Felz, 2001). Troisi (2002) notes that increased displacement behavior provides a better measure of anxiety and negative affect than verbal statements and facial expression.

As manifested by individuals with ASD, RRBs are self-involved, obtrusive, prolonged, and detached from outside reality (though more adaptive in high functioning ASD). Compared to intrusive, ritualistic behaviors indicative of obsessive-compulsive disorder (OCD), many RRBs in ASD (e.g., stereotypy, preoccupations, perseveration) tend to be ego-syntonic and comforting (Gabriels, Cuccaro, Hill, Ivers, & Goldson, 2004;

South, Ozonoff, & McMahon, 2005). As a general statement, repetitive movements serve a homeostatic function for children with ASD (Willemsen-Swinkels, Buitelaar, Dekker, & van England, 1998). Kinsbourne has observed that RRBs tend to be elicited by uncertainty, which is perceived as aversive (Kinsbourne, 2011). While OCD can be concurrent with ASD, we contend that RRBs and compulsions manifest separately in these cases.

Overarousal Is Integral to ASD

If RRBs are effective, though often maladaptive, efforts to relieve anxiety, how and why are people with ASD stressed? It is well established that anxiety is ubiquitous in ASD (Baron et al., 2006; Bellini, 2004; Muris, Steerneman, Merckelbach, Holdrinet & Meesters, 1998; Romanczyk & Gillis, 2006); at least phasically greater than in people without ASD when confronted with novel stimuli (Steingard, Ziminitzky, DeMaso, Bauman, & Bucci, 1997); and increases with environmental novelty (Cohen & Johnson, 1977; Hutt & Hutt, 1965; Hutt et al., 1964; Hutt et al., 1965; Stroh & Buick, 1968). While it can be argued that anxiety emerges from a primary intolerance of novelty, there is evidence that people with ASD have elevated sympathetic arousal even at rest, as if the "autonomic engine" idles high. Toichi and Kamio (2003) reported paradoxical, heightened sympathetic response during "resting conditions" and Ming and colleagues (2005) reported decreased basal parasympathetic tone and unstable cortical networks that alter perception, memory, and motor control leading to aversive reactions to stimuli that are associated with and perpetuate hyperarousal (Critchley, 2005; Goodwin et al., 2006; Hirstein, Iversen, & Ramachandran, 2001; Ming, Julu, Wark, Apartopoulos, & Hansen, 2004; Ming et al., 2005). We submit that this evident sympathetic over arousal results in the conscious, feeling state of anxiety.

There is some controversy about the prevalence of autonomic imbalance in individuals with ASD. A review of the scientific literature revealed little evidence for *tonic* sympathetic hyperarousal (Rogers & Ozonoff, 2005). However, the research on which this is based (1) does not consider that RRBs, by the subjects during the studies, might alter the measured responses, (2) has little ecological validity (occurring inside the laboratory), and (3) lacks valid measures of concurrent central nervous system stress responses. Ecologically valid, longitudinal studies observing individual children will resolve the controversy.

Neurophysiological findings involving the amygdala and oxytocin support our description of the observed over arousal in ASD. Interpersonal stress and fear, such as that stemming from abandonment, child abuse, neglect, and children of mothers with depression, is associated with increased amygdale volume (Pierce, Müller, Ambrose, Allen, & Courchesne, 2001). If the heightened arousal in ASD stems from autonomic dysregulation instead, it is fitting that no increase is seen in amygdale volume of people with ASD. Rather, decreased volume is observed, associated with weakened face processing abilities (Pierce et al., 2001) and greater social impairments (Nacewicz et al., 2006). Animal studies of oxytocin have established it as a modulator of the

stress response (Windle et al., 2003), in part through its efferent activity through the vagus (Dreifuss, Raggenbass, Charpak, Dubois-Dauphin, & Tribollet, 1988), a pathway associated with parasympathetic lowering of arousal (Porges, 2011). Oxytocin is further implicated in "social brain" activation in people with ASD (Gordon et al., 2012). Knowing that social deficits are a primary component in ASD, and taking the view that overarousal is also core, it is expected that oxytocin is measured at lower levels in people with ASD than controls (Modahl et al., 1998). Likewise, oxytocin infusions decrease RRBs in children with ASD (Hollander et al., 2003). With oxytocin affecting both social and autonomic function, we postulate that the social engagement system is intact and therefore responsive when it is not disengaged due to overarousal.

Autonomic Dysregulation Is a Governing Principle

Given the evidence of increased sympathetic tone and autonomic dysregulation in ASD, what may be the basis for autonomic imbalance and how might it provide an explanation for ASD? Porges (2011) implicates impaired functioning in the vagal system—part of the ANS. He proposes that the mammalian vagal system has evolved to include three anatomic and functional branches serving behaviors: (1) immobilization, (2) mobilization, and (3) social engagement. These branches range, in order, from primitive to phylogenetically mature. High vagal tone is seen in immobilization, sympathetic tone is not inhibited for mobilization, while a "vagal brake" balances primitive vagal and sympathetic tone to allow social engagement. The mature vagal system also influences cranial nerves that subserve facial expression, extraction of human voices from background noise, gaze fixation, head turning, and prosody. These cranial nerves (5, 6, 7, 9, 10, 11, and 12), in turn, communicate with the sympatho-inhibitory vagal system that lowers heart rate, lowers blood pressure, and increases emotional regulation.

We propose that the description by Porges (2011) also explains the pervasiveness of anxiety in ASD. Impairment of a phylogenetically mature vagus would leave the primitive systems to: (1) activate and sensitize the periaqueductal grey and basolateral nucleus of the amygdala, (2) increase sympathetic arousal, (3) affect qualitative aspects of language, (4) decrease the ability to modulate sensory input, and ultimately, (5) produce the need for compensatory RRBs.

The comorbidities of disordered sleep (Hoshino, Watanabe, Yashima, Kaneko, & Kumashiro, 1984; Sikora, Johnson, Clemons, & Katz, 2012), gastrointestinal dysfunction (Coury et al., 2012; Horvath & Perman, 2002), and tic disorders (Bodfish et al., 2000) in individuals with ASD may also relate to autonomic dysregulation and thus support its causal role in ASD. The vagal system is implicated in all three of these problems. Sleep problems in children with ASD are associated with abnormal diurnal rhythms and a dysregulated hypothalamic-pituitary-adrenal (HPA) axis as measured by failure to suppress cortisol release in response to dexamethasone (Hoshino, Yokolyama, & Hashimoto, 1987). Since vagal afferents inhibit the HPA (Porges, 2011),

one can infer a failure of the negative (vagal afferent) feedback. The vagus is also a primary gut regulator and involved in the control of gut hormones, satiety, pain perception, and motility (Porges, 2011). It follows that systemic alterations in autonomic regulation could result in problems with both gut function and brain–gut interaction. Finally stress has been implicated as an aggravating factor in tic disorders, in part due to influence of the amygdala (Leckman, 2002). Thus, phasic increases in sympathetic arousal with or without decreased vagal influence can play a role in increased tic prevalence. Significantly, all three of these conditions have been shown to improve with hypnotherapy.

To summarize (see Figures 1, 2, and 3), this time from the bottom up, the Autonomic Dysregulation Theory proposes that there is a defect (as yet uncharacterized) in development of the phylogenetically mature vagus that leads to: (1) impaired development of the social engagement system (hence deficiencies in qualitative language, nonverbal communication, social orienting); (2) impaired sensory modulation for novel stimuli via both intero- and exteroception (hence sensory rejection and sensitivity); (3) increased sympathetic tone with a dysphoric/defensive valence; and (4) anxiety,

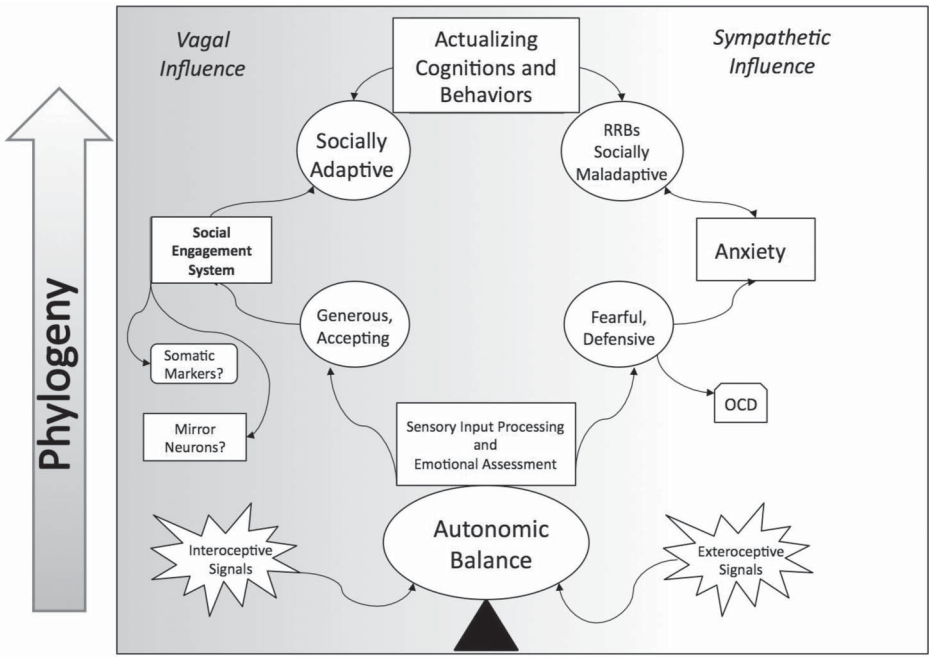

FIGURE 1 The Autonomic Dysregulation Theory: Neurotypical system representing a balanced autonomic regulation.

Note. Schematic shows relationships between autonomic balance, sensory/emotional processing, feeling states, and behaviors across phylogeny (bottom up) and the range of autonomic influence (horizontal).

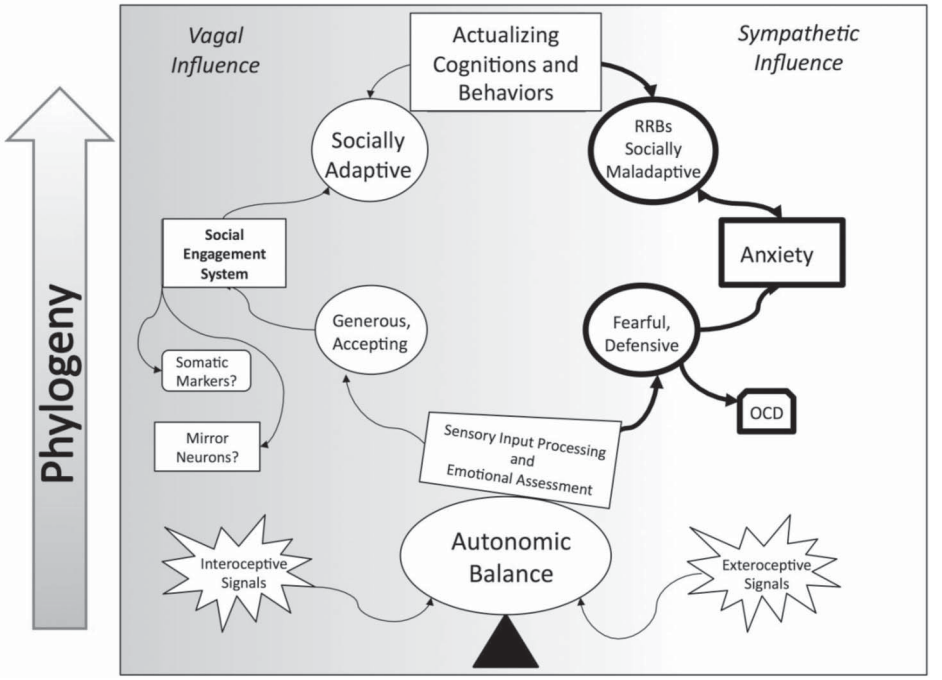

FIGURE 2 The Autonomic Dysregulation Theory: Autonomic apraxia with increased sympathetic influence driving fearful, defensive states, anxiety and compensatory RRBs.

Note. Schematic shows relationships between autonomic balance, sensory/emotional processing, feeling states, and behaviors across phylogeny (bottom up) and the range of autonomic influence (horizontal).

including OCD behavior. Restrictive repetitive behaviors, sensory rejection, and cognitive rigidity are all dearousing and thus signal efforts to limit novelty and self-regulate autonomic dysfunction. Different degrees of impairment in the vagal system account for phenotypic variation.

While the nature of the proposed impairment it is currently unclear, abnormalities in some key cranial nerve and vagal nuclei have been implicated in ASD (Rodier, Ingram, Tisdale, Nelson, & Romano, 1996) and Moebius Syndrome in which one-third of those affected meet criteria for ASD (Gilberg & Steffenberg, 1989).

Therapeutic Implications

Milton H. Erickson famously taught, "the symptom is the solution" (Rossi & Rossi, 1996). How, then, might one utilize the most salient symptoms of autistic behavior: RRBs, sensory sensitivity, and social withdrawal as "solutions"? When viewing the experience of living with ASD through a theoretical lens of autonomic dysregulation

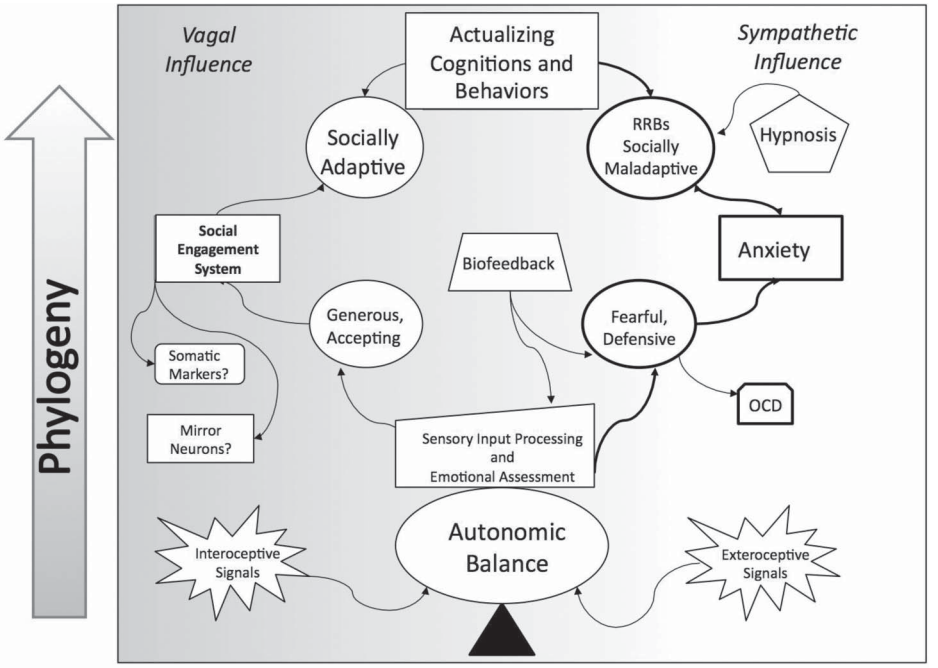

FIGURE 3 The Autonomic Dysregulation Theory: Compensated autonomic apraxia in which hypnosis and biofeedback modulate both anxiety and RRBs.

Note. Schematic shows relationships between autonomic balance, sensory/emotional processing, feeling states, and behaviors across phylogeny (bottom up) and the range of autonomic influence (horizontal).

two compelling, client-centered realms of potential therapeutic interventions emerge. The first is the development of improved autonomic regulation by using biofeedback systems driven by autonomic proxies, for example broadening the base and stability of the system (see Figures 1, 2, and 3). The second is shifting the RRB from content (behavioral expression) to process (the internal experience of competence, comfort, and control) by reframing that experience as private self-care and coping that is also more socially adaptive. These strategies ought to be mutually reinforcing. Improving autonomic regulation should lessen reliance on RRBs for coping and thus improve socially adaptive behavior. Similarly, internalizing the process of RRBs reduces social friction, leading in turn to decreased noxious exteroceptive inputs, increased interceptive comfort, and therefore better autonomic balance. In this section we detail the therapeutic strategies implied by this theoretical frame then note some preliminary experience to support ongoing investigation.

Autonomic Biofeedback Training

Biofeedback is well established as a method for improving autonomic control and has been used extensively with children. Most systems translate physiological measurements into audiovisual effects that users can learn to control. Skin conductance, respiratory rate, peripheral skin temperature, and heart rate are all effective expressions of and proxies for autonomic function (Cacioppo, Tassinary, & Berntson, 2007; Schwartz & Andrasik, 2003). In particular, the power of heart rate variability in the high-frequency range (0.15–0.4 Hz) corresponds with vagal tone (Porges, 2011). Biofeedback that elicits large peaks in the low frequency range (0.1 Hz) can be used to enhance vagal tone through a resonance across multiple systems related to cardiovascular functioning (Vaschillo, Vaschillo, & Lehrer, 2006).

In therapy, biofeedback training induces behavioral change by linking operant conditioning with cognitive anchors. In the same way that looking into a mirror causes us to change our facial expression, audiovisual information that changes with a physiological signal compels us to discern and control the direction of that signal. It is our experience that even with significantly cognitively impaired subjects, biofeedback results in a decreased state of arousal, just as looking in a mirror causes us to make adjustments to look our best.

For individuals with ASD, especially at younger ages, biofeedback presents even greater advantages. Where difficulty with social engagement would limit therapeutic rapport, computer interventions start with a shared focus on the computerized system. The therapist and patient develop a beneficial relationship secondarily. Some researchers suggest that most computer interventions are successful primarily in their ability to facilitate positive teacher–student interactions (e.g., Basil & Reyes, 2003; Heimann, Nelson, Tjus, & Gillberg, 1995). For patients with ASD, the computer decreases the social burden of interacting directly with the therapist while still allowing for the relationship to develop. The aforementioned role of operant conditioning does not require social or cognitive interaction and so can be used with those who have limited verbal ability.

A cognitive component is required to generalize biofeedback training beyond operant conditioning. Especially for individuals with ASD, motivation for cognitive change is critical. Evidence has shown that young people with ASD enjoy computer interventions (e.g., Chen & Bernard-Opitz, 1993). Children with ASD show great receptivity toward virtual-reality hypnosis treatments (Austin, Abbott, & Carbis, 2008) and have made significant improvements in a number of impairments using diverse computer interventions (e.g., Mitchell, Parsons, & Leonard, 2007; Moore & Calavert, 2000; Williams, Wright, Callaghan, & Coughlan, 2002). Individuals with ASD can apply virtual lessons to real world situations (Wallace et al., 2011) likely due to the limits and structure of the role-playing that controls the pace of information to digestible chunks. Specific to self-regulation, Lee (2011) found that visual translations of skin conductance and heart

rate are a useful way for children with ASD to understand and communicate their body's autonomic arousal level.

The therapist's abilities to build rapport and tailor the computer-based intervention to the strengths of the patient may be at least as important as the intervention. A recent systematic review of computer interventions shows mixed overall success (Ramdoss et al., 2012), with customizability identified as one important component for eliciting positive outcomes. Likewise, given the need for positive teacher–student interactions noted above, building upon the appeal of computerized biofeedback with an Ericksonian emphasis on patient strengths (described in detail below) is appropriate.

A number of caveats and questions emerge from this brief overview of biofeedback and computer-based interventions. First, measurements should be valid representations of the individual's deficit. Further characterization of the psychophysiological measures may be required to understand whether the same principles derived from people without ASD apply in this population. Secondly, biofeedback relies on conditioning. It must be reinforcing for users to see their signals move toward adaptive states. Progress is unlikely if users have no motivation or positive expectation beyond the therapist's desire for improvement. Lastly, a major criticism of biofeedback is that there is not always a clear method for users to take the lessons learned outside of the practice sessions or apply them without the computer unless they are simultaneously and specifically taught how to do that. Two relevant solutions to this criticism come from self-regulation training. In heart rate variability biofeedback, patients focus on breathing techniques. This is beneficial because respiration is more noticeable than in other types (blood pressure, heart rate). Secondly, to bring these self-regulation lessons into daily life, patients can be taught to notice when they unintentionally use the skills in their daily routines (e.g., sighing). Similarly, skills can be anchored to daily triggers that serve as reminders (e.g., "When you get off the school bus and find yourself sighing, use that reminder from your inside mind to let your breathing help you . . ."). In these ways, connections between biofeedback training and daily activities can be reinforced with hypnotic suggestion.

Hypnosis

Currently there is neither a consensual definition nor theoretical framework for hypnosis, so a working definition is required for each context. We consider clinical hypnosis an interpersonal interaction that utilizes focused, intensified, and internally-directed concentration (referred to as trance), to cultivate change in maladaptive psychophysiological reflexes. Typically either a naturally-occurring trance (e.g., due to acute pain, anxiety, coming to the therapist's office with expectancy, or RRBs) is utilized or one is induced by ritual (e.g., progressive relaxation, imagery, eye-fixation). Then suggestions, metaphors, recollection of memories, and other internal exploration are used with the hope and intention to change experiences in this intensified state. It is theorized that this

intensified attentional trance state more efficiently and effectively catalyzes changes in synaptic strength. This ultimately leads to changes in psychophysiological reflexes by inducing plasticity of neurophysiological interactions (Rossi, 2002).

Hypnotic and autistic behaviors share commonalities. Both display intensity of attention that subsumes executive function, is internally focused, and serves the purpose of self-regulating non-conscious processes. Whether RRBs and hypnotic trance share common neural patterns is, so far, unknown. But certainly people with ASD rely on this "trance-formative" behavior to cope. We consider this a capacity that can be utilized with hypnotic strategies to increase adaptive behaviors for people with ASD.

The "utilization approach" attributed to Milton H. Erickson is based on the premise that the subject's innate resources and abilities can be hypnotically directed in adaptive ways (Erickson, Rossi, & Rossi, 1976). Thus applied, hypnosis invests in self-mastery. Utilization is particularly germane to a discussion of hypnosis for people with ASD. Broadly, with consideration of the Autonomic Dysregulation Theory, people with ASD are autonomously, intensively, and defensively engaged in the process of self-regulating anxiety through RRBs. Hypnotic strategies that help the person with ASD recognize, distill, and then develop the calming contents and/or processes of those behaviors internally while not manifesting them externally can adaptively employ an inherent strength.

Trance induction with people who have ASD can utilize RRBs either by naturalistically and purposefully experiencing the RRB or a more formal ritualistic induction followed by experiencing the RRB as an intensification (deepening) step. Within this context, the patient is guided to notice their sense of associated comfort, control, and most salient sensory or cognitive elements of the RRB so that they can become entry points ("keys," "signals," "doorways") into the feeling state of the RRB. If the behavior includes a motor component (rocking, tapping, tics) the patient is asked to focus on the feeling and its associated sensory modes while modulating and extinguishing the movement, thereby decreasing the behavior's externalization. Finally the entry points are associated experientially with common daily events that act as triggers for anxiety and RRBs. The patient is guided to virtually experience situations in which they have engaged in RRBs, and practice engaging in the more adaptive "trance version" without externalizing behaviors. Often patients are invited to do this with their eyes open, or in front of a mirror so that they can practice "looking normal" while, privately, knowing that they are comforting themselves. Young people, with and without ASD, commonly engage in these experiences quite actively, with their eyes open and in conversation. The trance states of absorption in active imaginative play and story-telling are examples (Kuttner & Catchpole, 2013).

During this therapeutic process, it is critical that the therapist acts as a coach, facilitator, and "evocateur" of the patient's *own* images, feeling states, and anchors while frequently embedding ego-strengthening observations about how choices and ideas that emerge are "exactly right" because the patient "knows the right way" to help him or herself. Rather than asking the patient to share the content of their experiences, the

author (LIS) asks the patient to choose whether "it will help you better" to share or keep the experiences private. In the author's (LIS) experience, patients with ASD most often choose to keep the images and experiences private, claiming a pride of ownership, and perhaps reflecting a learned defensive posture. This naturalistic, conversational, and evocative approach—utilizing the patient's own resources while guiding them to shift the manifestations internally—is more useful than an approach in which the therapist introduces his or her own metaphors or experiences. As reviewed above, novelty tends to be aversive in ASD. So, while the adaptive re-purposing of patient's RRBs may be a novel *process*, the therapist ought to be careful about novel *content*.

The vignette shared in the opening of this article illustrates this approach. Stefan used his interest in and discussion of railroads as trance induction. Within that experience he found a sense of comfort and mastery in elucidating this complex system. Aspects of that experience, unknown to the therapist, were then used to displace externalized behaviors. This type of approach is a key to imaginal training for children and adolescents with ASD. We hold that when a person copes by relying on intensified, narrowed interests, then utilizing those same interests is the most effective way to provide him or her comfort in change.

In ASD, hypnotic approaches are limited to those who can form reciprocal relationships and have adequate ability to communicate creatively. Hypnosis with some young people with ASD who have relational and communicative limitations can be facilitated using puppets, games, and video games.

Integrating Hypnosis and Autonomic Biofeedback

Combining biofeedback training and hypnosis is synergistic (Culbert, Reaney, & Kohen, 1994). By linking representation of real-time adaptive physiological change with internal states, anchored by common triggers encountered in daily life, this merged approach utilizes both "bottom up" and "top down" neurobiological resources of the person with ASD. This is exemplified in the opening vignette as Stefan uses biofeedback to link breathing with imagery. Not surprisingly, the literal thinking of many young people with ASD—along with their proclivity for computerized games and media—causes them to prefer the learning afforded with biofeedback and eschew the notion of "trance" and "imagery." We have found that the trance of engagement with biofeedback provides the opening for hypnotic learning. The patient is encouraged to lower sympathetic tone and increase vagal tone when internalizing their RRB, then linking this practice to cues encountered in their daily life. While acknowledging the false dichotomy, we have found it useful to explain to patients that "biofeedback starts in the body and hypnosis starts in the mind" (T. Culbert, personal communication, 2011) because, for many young people with ASD, RRBs have a physical component.

Initial Research Efforts

In the Center for Applied Psychophysiology and Self-regulation (CAPS) at Rochester Institute of Technology (RIT), we have initiated two projects that investigate the feasibility and effect of structured computerized biofeedback and hypnotic strategies with college students and children with ASD.

The Minding Anxiety Project engages matriculating college students who have been diagnosed with ASD. These students learn about respiration, heart rate variability, peripheral skin temperature, and skin conductance level as they relate to stress and well-being. Preliminary results have shown correlations between success in physiological control and psychological tests. Participants with the greatest improvement in low-frequency heart rate variability also had the greatest improvement in self-concept and the greatest decrease in anxiety (Sugarman, 2012a, 2012b). While capitalizing on their tendency toward cognitive compensation, participants in this program came away with new insight to their feelings and experiences. One participant's video recorded statement is representative: "What I do is I take a step back—and this where the breathing [subject's term for biofeedback training] is helpful—because it makes your *system* take a step back. You say, 'regardless of how I feel, I'm going to tell my body that I'm in a non-panic scenario.' I am going to simulate the way I breathe when I am not running for my life because, hopefully, then I will look at things rationally and say, 'If I *can't* do everything by Monday, what *can* I do?' " [Italics added from spoken emphasis].

We are also developing a physiologically-controlled, role-playing video game, called MindGamers™. It incorporates self-regulation strategies and is designed to be played collaboratively between a therapist and the patient/player (P/P). The prototype integrates hypnotic and biofeedback strategies with cognitive and narrative approaches while generating real-time physiological data from the P/P's game-play (Jacobs, Rice, & Sugarman, 2012; Rice, Sugarman, & Jacobs, 2012). The physiological controller developed for this video game utilizes a unique Dynamic Feedback Signal Set (DyFSS) that adjusts physiological feedback to each P/P's needs. This type of structured, yet customizable clinical interaction provides a platform for investigating the link between autonomic regulation and RRBs.

Implications for Future Research

Through Autonomic Dysregulation Theory we present two broad directions for future research. The first involves more fully characterizing autonomic reactivity and phenotypes of people with ASD who accept and respond to hypnosis and biofeedback therapies. The second involves how such autonomic self-regulatory therapies affect the manifestations or expressions of characteristics of ASD.

Autonomic Characteristics in ASD

If ASD derives from a phylogenetically early insult, there may be wider psychophysiological variation than seen in controls. Many questions arise from this possibility. Are autonomic responses to stressors across the range of ASD phenotypes different than the responses from people without ASD? Is there a difference in tonic and/or phasic sympathetic arousal and does it correlate with functional status or specific characteristics (especially RRBs)? More specifically, is there disparity between the expected covariance of skin conductance, peripheral temperature, respiratory rate, and heart rate variability? Early investigations characterizing the autonomic measurement of people with ASD indicate that differences do exist (Kushki et al., 2012). Similarly, how do common psychoactive medications commonly affect the range and reactivity of autonomic measurements in ASD? For example, when propanolol is limiting heart rate in a biofeedback user, are adjustments necessary for correct interpretation of the output? Finally and most practically, what is the best "physiological fit" for biofeedback sensors? How can biofeedback be "tuned" so that the most discernable and controllable sensors are selected on an individual basis? As previously noted, DyFSS development and testing begins to explore this clinical question.

Effects of Autonomic Self-Regulation Training on Characteristics of ASD

In teaching self-regulation to people with ASD, we expect a wide range of improvements. Like rebuilding weak muscles, we would expect gradual-but-lasting gains. Primary measures might be best tracked across months or years as patients develop self-regulatory habits. Following the interventions outlined above, we hypothesize that patients will express a decrease in RRBs stemming from lowered arousal and improved autonomic control.

Replacing these RRBs (which we believe to be an often-unconscious, maladaptive attempt at self-regulation) with effective self-regulatory behaviors could lead to further-reaching improvement in the symptoms of ASD. With greater voluntary control of the ANS and decreased arousal, people with ASD may decrease their reliance on RRBs for self-regulation. However, they may continue to use them creatively for self-actualization (as Stefan did in the vignette). Those who exhibit narrow interests might widen their scope of topics, while those who tend toward self-stimulatory behavior may show a decreasing frequency. Biological and behavioral improvements are not expected to be limited to this subset of symptoms. Improving control of the ANS may allow a person diagnosed with ASD to make improvements in attention to faces, emotional receptivity and comprehension, changes in prosody, and motor behaviors.

Better understanding the role of autonomic functioning could provide a basis for further comparisons with anxiety disorders. For example, a decrease in amygdala activation is seen in ASD whereas social phobia exhibits hyperactive amygdala activation

(reviewed in Tyson & Cruess, 2012). However, little is known about autonomic differentiation that may be measured peripherally. Focusing the search on comparisons of autonomic (dys)regulation may highlight specifics on the unique autonomic signature of ASD. This could elucidate the nature of ASD and improve interventions. Genomic studies comparing groups with these similar-but-divergent symptoms may define genetic roles.

The comorbidities of sleep disturbance, gastrointestinal dysfunction, and tic disorders in individuals with ASD may also be targets for investigations of effects of autonomic self-regulation training. Hypnosis has been effective for insomnia and sleep terror disorder, irritable bowel syndrome, and tic disorders in children (reviewed in Kohen & Olness, 2011). Of note, a 5-year follow-up of a prospective, randomized trial of hypnosis for children with irritable bowel syndrome/functional abdominal pain demonstrates continued significant improvement compared to standard medical therapy (Vlieger, Rutten, Govers, Frankenhuis, & Benninga, 2012). In these cases, investing in self-regulation appears to have more persistent effects than standard treatment. While young people with ASD may not necessarily be motivated to change their RRBs, they may want to learn self-regulatory strategies to help themselves sleep, relieve abdominal pain, and decrease their tics. Thus, these comorbidities present the opportunity to initiate self-regulation while relying on its documented "spillover effect" of therapeutic efficacy (Kohen, 2010; Kohen, Olness, Colwell, & Heimel, 1984).

Finally, given phenotypic variation among those with ASD, any study designed to explore autonomic regulation will need to allow for the emergence of multiple variables. We imagine longitudinal cohort studies in which individuals with ASD simultaneously for behavioral change, anatomic and functional neuroimaging, autonomic function, and peripheral stress biomarkers.

Conclusions

Anxiety in ASD is more frequent than the concurrence of the classical core symptoms of language, social, and cognitive impairments. The Autonomic Dysregulation Theory described in this article places these manifestations in a neurodevelopmental context in which restrictive repetitive behaviors compensate for sympathetic arousal and diminished vagally-mediated social engagement. Further studies are needed to elucidate and confirm the effects of teaching young people with ASD more adaptive ways of helping themselves. We predict that associating cortical anatomy, cortical function, and stress markers with RRBs, communication, and social engagement will provide evidence of efficacy. At the same time, this theory directs the etiological research focus to phylogenetically early insults of the ANS.

Perhaps the most profound implication of this model is its potential to shift perspectives regarding people with ASD. Instead of interpreting their social withdrawal,

dyscommunication, and maladaptive behaviors as aloof and antipathic, one can understand these characteristics as manifestations of complex struggles to cope with and compensate for intense and intrinsic defensive/sympathetic tone. Efforts to utilize and repurpose this capacity require a different approach. Rather than attempting to fix symptoms by teaching patients to change their withdrawn and challenging behaviors, this calls on providers to empathically entrain and aid the development of their intense, innate effort to self-regulate.

Note

1. For the remainder of this article, the term "autism spectrum disorder(s)" (ASD) refers to the phenotypic range of autism conditions, unless otherwise specified. The taxonomy of these conditions continues to evolve, but we focus here on unifying traits while not ignoring the important role of clarifying distinctions among phenotypes. This usage is also consistent with the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (*American Psychological Association, 2013) that divides ASD into levels of functioning (high, middle, and low) for all who meet diagnostic criteria, removing subtypes such as Asperger Syndrome.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.; *DSM-5*). Washington, DC: Author.

Austin, D. W., Abbott, J. M., & Carbis, C. (2008). The use of virtual reality hypnosis with two cases of autism spectrum disorder: A feasibility study. *Contemporary Hypnosis*, *25*, 102–109.

Bachmann, I., Bernasconi, P., Herrmann, R., Weishaupt, & M. A., Stauffacher, M. (2003). Behavioural and physiological responses to an acute stressor in crib-biting and control horses. *Applied Animal Behaviour Science*, *8*, 297–311.

Baron, M. G., Groden, J., Groden, G., & Lipsitt, L. P. (Eds.). (2006). *Stress and coping in autism*. New York, NY: Oxford University Press.

Basil, C., & Reyes, S. (2003). Acquisition of literacy skills by children with severe disability. *Child Language Teaching and Therapy*, *19*, 27–45.

Beauchaine, T. (2001). Vagal tone, development and Gray's motivational theory: Toward an integrated model of autonomic system functioning in psychopathology. *Development and Psychopathology*, *13*, 183–214.

Bellini, S. (2004). Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities*, *19*, 78–86.

Bergman, P., & Escalona, S. K., (1949) Unusual sensitivities in very young children. *Psychoanalytic Study of the Child*, *4*, 333–352.

Berkson, G., & Mason, W. A. (1963). Stereotyped movements of mental defectives: III. Situation effects. *American Journal of Mental Deficiency*, *68*, 409–412.

Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism, comparison to mental retardation. *Journal of Autism and Developmental Disorders*, *39*, 237–243.

Boyar, P., & Liénard, P. (2006). Why ritualized behavior? Precaution systems and action parsing in developmental, pathological and cultural rituals. *Behavioral and Brain Sciences*, *29*, 1–56.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (Eds.). (2007). *Handbook of psychophysiology* (3rd ed.). New York, NY: Cambridge University Press.

Centers for Disease Control and Prevention. (2012) Prevalence of autism spectrum disorders—Autism and developmental disabilities monitoring network, 14 sites, United States, 2008. *Morbidity and Mortality Weekly*, *61*, 1–19. Retrieved from: http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6103a1.htm

Chen, S. H., & Bernard-Opitz, V. (1993). Comparison of personal and computer-assisted instruction for children with autism. *Mental Retardation*, *31*, 368–376.

Cogliati, T., Good, D. J., Halgney, M., Delgado-Romera, P., Eckhaus, M. A., Koch, W. J., & Kirsch, H. R. (2002). Predisposition to arrhythmias and autonomic dysfunction in nhih1-deficient mice. *Molecular and Cellular Biology*, *22*, 4977–4983.

Cohen, D. J., & Johnson, W. T. (1977). Cardiovascular correlates of attention in normal and psychiatrically disturbed children: Blood pressure, peripheral blood flow, and peripheral vascular resistance. *Archives of General Psychiatry*, *34*, 561.

Coury, D. L., Ashwood, P. Fasano, A., Fuchs, G., Geraghty, M, Kaul, A., . . . Jones, N. E. (2012). Gastrointestinal conditions in children with autism spectrum disorder: Developing a research agenda. *Pediatrics*, *130*, S160–S168.

Critchley, H. D. (2005). Neural mechanisms of autonomic, affective, and cognitive integration. *The Journal of Comparative Neurology*, *493*, 154–166.

Culbert T., Reaney J., & Kohen D. (1994). Cyberphysiologic strategies for children: The clinical hypnosis/biofeedback interface. *International Journal of Clinical and Experimental Hypnosis*, *42*, 97–117.

Dickens, M. J., & Romero, L. M. (2009). Wild European starlings (Sturnus vulgaris) adjust to captivity with sustained sympathetic nervous system drive and a reduced fight-or-flight response. *Physiological and Biochemical Zoology*, *82*, 603–610.

Dreifuss, J. J., Raggenbass, M., Charpak, S., Dubois-Dauphin, M., & Tribollet, E. (1988). A role of central oxytocin in autonomic functions: Its action in the motor nucleus of the vagus nerve. *Brain Research Bulletin*, *20*, 765–770.

Erickson, M. H., Rossi, E. L., & Rossi, S. I. (1976) *Hypnotic realities: The induction of clinical hypnosis and forms of indirect suggestion*. New York, NY: Irvington Publishers, Inc.

Floeter, M. K., & Greenough, W. T. (1979). Cerebellar plasticity: modification of Purkinje cell structure by differential rearing in monkeys. *Science*, *206*, 227.

Gabriels, R., Cuccaro, M., Hill, D., Ivers, B., & Goldson, E. (2004). Repetitive behaviors in autism: Relationships with associated clinical features. *Research in Developmental Disabilities*, *26*, 169–181.

Garner, J. P., Meehan, C. L., & Mench, J. A. (2003). Stereotypies in caged parrots, schizophrenia and autism: Evidence for a common mechanism. *Behavioural Brain Research*, *145*, 125–134.

Gilberg, C., & Stefenberg, S. (1989). Autistic behavior in Moebius Syndrome. *Acta Pediatrica*, *78*, 314–316.

Goin-Kochel, R. P., & Myers B. J. (2004). Parental report of early autistic symptoms: Differences in ages of detection and frequencies of characteristics among three autism-spectrum disorders. *Journal of Developmental Disabilities*, *11*, 21–40.

Goodwin, M. S., Groden, J., Velicer, W. F., Lipsitt, L. P., Baron, M. G., Hofmann, S. G., & Groden, G. (2006). Cardiovascular arousal in individuals with autism. *Focus on Autism and Other Developmental Disabilities*, *21*, 100–123.

Gordon, I., Bennett, R. H., Vander Wyk, B. C., Leckman, J. F., Feldman, R., & Pelphrey, K. A. (2012). Oxytocin's impact on social cognitive brain function in youth with ASD. *Online Abstract book for the 2012 International Meeting for Autism Research*. Abstract retrieved from https://imfar.confex.com/imfar/2012/webprogram/Paper10197.html

Heimann, M., Nelson, K., Tjus, T., & Gillberg, C. (1995). Increasing reading and communication skills in children with autism through an interactive multimedia computer program. *Journal of Autism and Developmental Disorders*, *25*, 459–480.

Hinde, R. A. (1970). *Animal behavior: A synthesis of ethology and comparative psychology*. New York, NY: McGraw-Hill.

Hirstein, W., Iversen, P., & Ramachandran, V. S. (2001). Autonomic responses of autistic children to people and objects. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *268*, 1883–1888.

Hollander, E., Novotny, S., Hanratty, M., Yaffe, R., DeCaria, C. M., Aronowitz, B. R., & Mosovich, S. (2003). Oxytocin infusion reduces repetitive behaviors in adults with autistic and Asperger's disorders. *Neuropsychopharmacology*, *28*, 193–198.

Horvath, K., & Perman, J. A. (2002). Autism and gastrointestinal symptoms. *Current Gastroenterology Reports*, *4*, 251–258.

Hoshino, Y.,Watanabe, H., Yashima, Y., Kaneko, M., & Kumashiro, H. (1984). An investigation on sleep disturbance of autistic children. *Folia Psychiatrica et Neurologica Japonica*, *38*, 45–51.

Hoshino, Y., Yokolyama, F., & Hashimoto, S. (1987). The diurnal variation and response to dexamethasone suppression test of saliva cortisol levels in autistic children. *Japan Journal of Psychiatry and Neurology*, *41*, 227–235.

Hutt, C., & Hutt, S. J. (1965). Effects of environmental complexity on stereotyped behaviours of children. *Animal Behaviour*, *13*, 1–4.

Hutt, C., Hutt, S. J., Lee, D., & Ounsted, C. (1964). Arousal and childhood autism. *Nature*, *204*, 908–909.

Hutt, S. J., Hutt, C., Lee, D., & Ounsted, C. (1965). A behavioural and electroencephalographic study of autistic children. *Journal of Psychiatric Research*, *3*, 181–198.

Jacobs, S., Rice, R. H., & Sugarman, L. I. (2012). Creating MindGamers™: Building communication, design and development process with clinicians, game faculty and students. Presented at the *Conference on Meaningful Play*, East Lansing, MI, October 2012.

Kinsbourne, M. (1980). Do repetitive movement patterns in children and animals serve a dearousing function? *Journal of Developmental and Behavioral Pediatrics*, *1*, 39–42.

Kinsbourne, M. (2011). Repetitive movements and arousal. In D. A. Fein (Eds.). *The Neuropsychology of Autism* (pp. 367–394). New York, NY: Oxford Press.

Klengel, T., Mehta, D., Anacker, C., Rex-Haffner, M., Pruessner, J. C., Pariante, C. M., . . . Binder, E. B. (2012). Allele-specific FKBP5 DNA demethylation: A molecular mediator of gene-childhood trauma interactions. *Nature Neuroscience*. doi:10.1038/nn.3275

Kohen, D. P. (2010). Long Term Follow-up of Self-Hypnosis Training for Recurrent Headaches: What the Children Say. *The International Journal of Clinical and Experimental Hypnosis*, *58*, 417–432.

Kohen, D. P., & Olness, K. N. (2011). *Hypnosis and Hypnotherapy with Children* (4th ed.). New York, NY: Routledge-Taylor & Francis Group.

Kohen, D. P., Olness, K. N., Colwell, S. O., & Heimel, A. (1984). The use of relaxation-mental imagery (self-hypnosis) in the management of 505 pediatric behavioral encounters. *Journal of Developmental & Behavioral Pediatrics*, *5*, 21–25.

Korda, P. (1978). Locomotor stereotypy visually deprived kittens. *Acta Neurobiologiae Experimentalis*, *38*, 313–351.

Kushki, A., Pla Mobarak, M., Drumm, E., Tanel, E., Chau, T., & Anagnostou, E. (2012). Investigation of the autonomic nervous system response to anxiety in children with autism spectrum disorders. Online Abstract book for the 2012 International Meeting for Autism Research. Abstract retrieved from https://imfar.confex.com/imfar/2012/webprogram/Paper10737.html

Kuttner, L., & Catchpole, R. E. H. (2013). Development matters: Hypnosis with children. In L. I. Sugarman & W. C. Wester (Eds.), *Therapeutic hypnosis with children and adolescents* (2nd ed.) (pp. 27–47). Wales: 700 Crown House.

Lang, P. J., Davis, M., & Ohman A. (2000) Fear and anxiety: Animal models and human cognitive psychophysiology. *Journal of Affective Disorders*, *61*, 137–159.

Leckman, J. F. (2002). Tourette's Syndrome. *The Lancet*, *380*, 1577–1586.

Lee, C. H. (2011). *Externalizing and interpreting autonomic arousal in people diagnosed with Autism.* [Unpublished Dissertation]. Retrieved online from http://web.media.mit.edu/~jackylee/leej-phd.pdf

Melzack, R., & Burns, S. K. (1965). Neurophysiological effects of early sensory restriction. *Experimental Neurology*, *13*, 163–175.

Ming, X., Julu, P. O., Wark, J., Apartopoulos, F., & Hansen, S. (2004). Discordant mental and physical efforts in an autistic patient. *Brain and Development*, *26*, 519–524.

Ming, X., Julu, P. O., Brimacombe, M., Connor, S., & Hansen, S. (2005). Reduced cardiac parasympathetic activity in children with autism. *Brain and Development*, *27*, 509–516.

Minshe, N. J., & Goldstein, G. (1998). Autism as a disorder of complex information processing. *Mental Retardation and Developmental Disabilities Research Reviews*, *4*, 129–136.

Mitchell, P., Parsons, S., & Leonard, A. (2007). Using virtual environments for teaching social understanding to 6 adolescents with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, *37*, 589–600.

Modahl, C., Green, L. A., Fein, D., Morris, M., Waterhouse, L., Feinstein, C., & Levin, H. (1998). Plasma oxytocin levels in autistic children. *Biological Psychiatry*, *43*, 270–277.

Moore, M., & Calavert, S. (2000). Vocabulary acquisition for children with autism: Teacher or computer instruction. *Journal of Autism and Developmental Disorders*, *30*, 359–362.

Muris, P., Steerneman, P., Merckelbach, H., Holdrinet, I., & Meesters, C. (1998). Comorbid anxiety symptoms in children with pervasive developmental disorders. *Journal of Anxiety Disorders*, *12*, 387–393.

Nacewicz, B. M., Dalton, K. M., Johnstone, T., Long, M. T., McAuliff, E. M., Oakes, T. R., . . . Davidson, R. J. (2006). Amygdala volume and nonverbal social impairment in adolescent and adult males with autism. *Archives of General Psychiatry*, *63*, 1417–1428.

Pierce, K., Müller, R.-A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform 'face area' in autism: Evidence from functional MRI. *Brain*, *124*, 2059–2073.

Porges, S. W. (2011) The Polyvagal Theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation. New York, NY: W.W. Norton & Company, Inc.

Ramdoss, S., Machalicek, W., Rispoli, M., Mulloy, A., Lang, R., & O'Reilly, M. (2012). Computer-based interventions to improve social and emotional skills in individuals with autism spectrum disorders: A systematic review, *Developmental Neurorehabilitation*, *15*, 119–135.

Rice, R., Sugarman, L. I., & Jacobs S. (2012). Building MindGamers™, Proceedings of *Games for Health–Europe*. Amsterdam, The Netherlands: Games for Health Europe.

Rodier, P. M., Ingram, J. L., Tisdale, B., Nelson, S., & Romano, J. (1996). Embryological origin for autism: Developmental anomalies of the cranial nerve motor nuclei. *The Journal of Comparative Neurology*, *370*, 247–261.

Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, *46*, 1255–1268.

Romanczyk, R. G., & Gillis, J. M. (2006). Autism and the physiology of stress and anxiety. In M. G. Baron, J. Groden, G. Groden, & L. P. Lipsitt (Eds.). *Stress and coping in autism* (pp. 183–204). New York, NY: Oxford University Press.

Rossi, E. L. (2002). *The psychobiology of gene expression: Neuroscience and neurogenesis in hypnosis and the healing arts*. New York, NY: W. W. Norton.

Rossi, E. L., & Rossi, K. L. (1996). *The symptom path to enlightenment: The new dynamics of self-organization in hypnotherapy: An advanced manual for beginners*. Pacific Palisades, CA: Palisades Gateway Publishing.

Schwartz, M. S., & Andrasik, F (Eds.). (2003). *Biofeedback: A practitioner's guide* (3rd ed.). New York, NY: Guilford Press.

Sikora, D. M., Johnson, K., Clemons, T., & Katz, T. (2012). The relationship between sleep problems and daytime behavior in children of different ages with autism spectrum disorders. *Pediatrics*, *130*, 583–590.

South, M., Ozonoff, S., & McMahon, W. (2005). Repetitive behavior in Asperger syndrome and high-functioning autism. *Journal of Autism and Developmental Disorders*, *35*, 145–158.

Steingard, R. J., Ziminitzky, B., DeMaso, D. R., Bauman, M. L., & Bucci, J. P. (1997). Sertraline treatment of transition-associated anxiety and agitation in children with autistic disorder. *Journal of Child and Adolescent Psychopharmacology*, *7*, 9–15.

Stroh, G., & Buick, D. (1968). The effect of relative sensory isolation on two autistic children. In S. J. Hutt & C. Hutt (Eds.), *Behavior Studies in Psychiatry* (pp. 161–174). New York, NY: Pergamon.

Sugarman, L. I. (2012a, October). *Transforming health and care: Report from the center for applied psychophysiology and self-regulation at Rochester Institute of Technology*. XIX. Internationaler Hypnose Kongress, Bremen. ABSTRACTS (pp. 83–84). Retrieved from http://www.hypnose-tagung.de/

Sugarman, L. I. (2012b, October). *Investing in autonomic balance for students with autism: The minding anxiety project*. Presented at The Autism Spectrum in Higher Education. Cambridge, MA. Retrieved from http://mit.edu/uaap/sds/conference/events.html

Sutano, W., & de Kloet, E. R. (1994). The use of animal models in the study of stress and stress-related phenomena. *Laboratory Animals*, *28*, 293–306.

Toichi, M., & Kamio, Y. (2003). Paradoxical autonomic response to mental tasks in autism. *Journal of Autism and Developmental Disorders*, *33*, 417–426.

Troisi, A. (2002). Displacement activities as a behavioral measure of stress in nonhuman primates and human subjects. *Stress: The International Journal on the Biology of Stress*, *5*, 47–54.

Tyson, K. E., & Cruess, D. G. (2012). Differentiating high-functioning autism and social phobia. *Journal of Autism and Developmental Disorders*, *42*, 1–14.

Vaschillo, E. G., Vaschillo, B., & Lehrer, P. M. (2006). Characteristics of resonance in heart rate variability stimulated by biofeedback. *Applied Psychophysiology and Biofeedback*, *31*, 129–142.

Vlieger, A. M., Rutton, J. M., Govers, A. M., Frankenhuis, C., & Benninga, M. A. (2012). Long-term follow-up of gut-directed hypnotherapy vs. standard care in children with functional abdominal pain or irritable bowel syndrome. *The American Journal of Gastroenterology*, *107*, 627–631.

Wallace, G. L., Case, L. K., Harms, M. B., Silvers, J. A., Kenworthy, L., & Martin, A. (2011). Diminished sensitivity to sad facial expressions in high functioning autism spectrum disorders is associated with symptomatology and adaptive functioning. *Journal of Autism and Developmental Disorders*, *41*, 1475–1486.

Willemsen-Swinkels, S. H. N., Buitelaar, J. K., Dekker, M., & van Engeland, H. (1998). Subtyping stereotypic behavior in children: The Association between stereotypic behavior, mood, and heart rate. *Journal of Autism and Developmental Disorders*, *28*, 547–557.

Williams, C., Wright, B., Callaghan, G., & Coughlan, B. (2002). Do children with autism learn to read more readily by computer assisted instruction or traditional book methods? *Autism*, *6*, 71–91.

Windle, R. J., Kershaw, Y. M., Shanks, N., Wood, S. A. Lightman, & S. L., Ingram, C. D. (2003). Oxytocin attenuates stress-induced c-*fos* mrna expression in specific forebrain regions associated with modulation of hypothalamo–pituitary–adrenal activity. *The Journal of Neuroscience*, *24*, 2974–2982.

Zohar, A. H., & Felz, L. (2001). Ritualistic behavior in young children. *Journal of Abnormal Child Psychology*, *29*, 121–128.