Casual Friday Presents

Building Resilient Kids

A BIOGENETIX CLINICAL PRESENTATION

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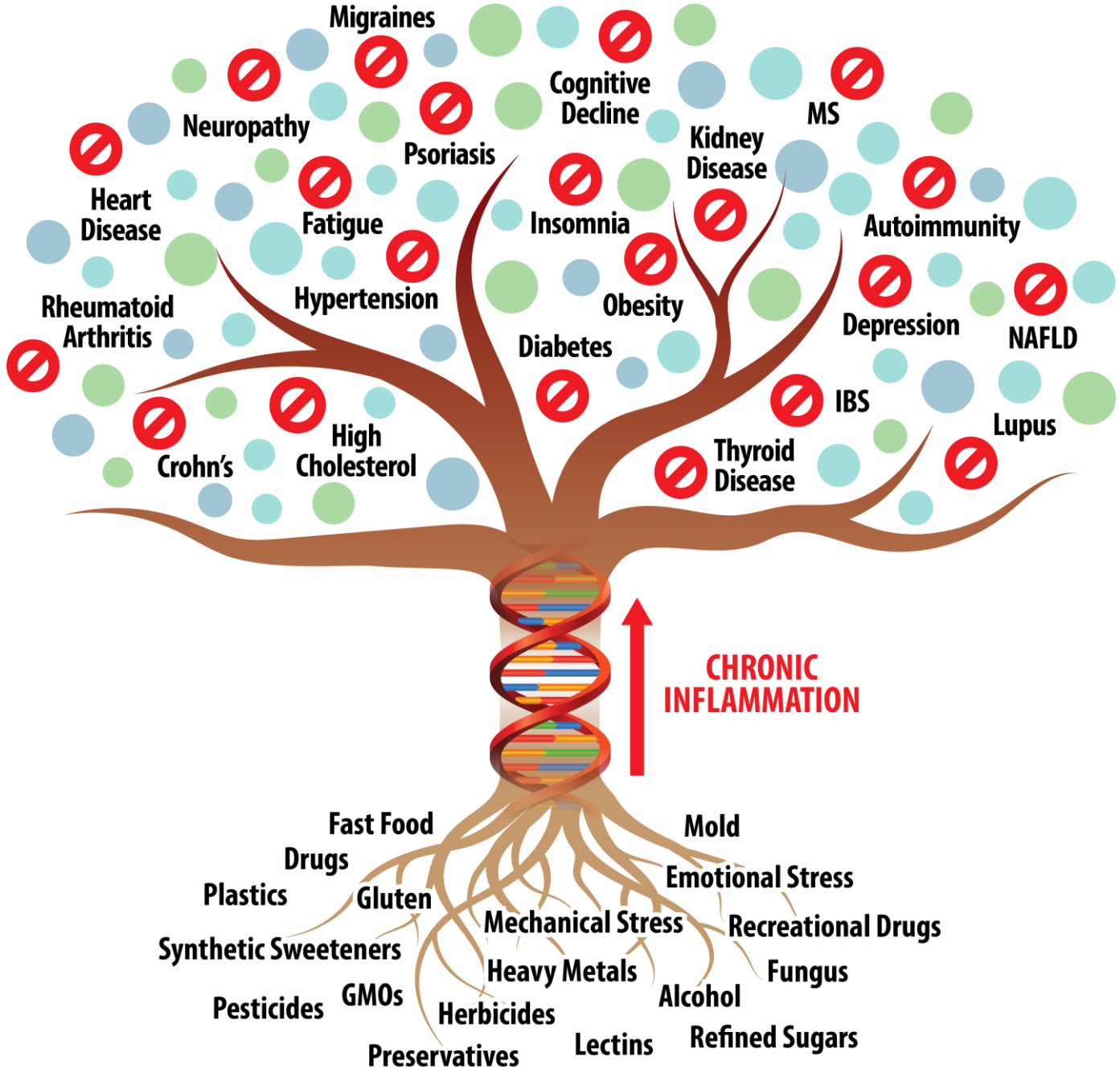
Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Autoimmunity
Rheumatoid Arthritis
Hypertension
Obesity
Depression
NAFLD
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Drugs
Plastics
Gluten
Synthetic Sweeteners
Pesticides
GMOs
Herbicides
Preservatives
Lectins
Refined Sugars
Heavy Metals
Mechanical Stress
Alcohol
Mold
Emotional Stress
Recreational Drugs
Fungus

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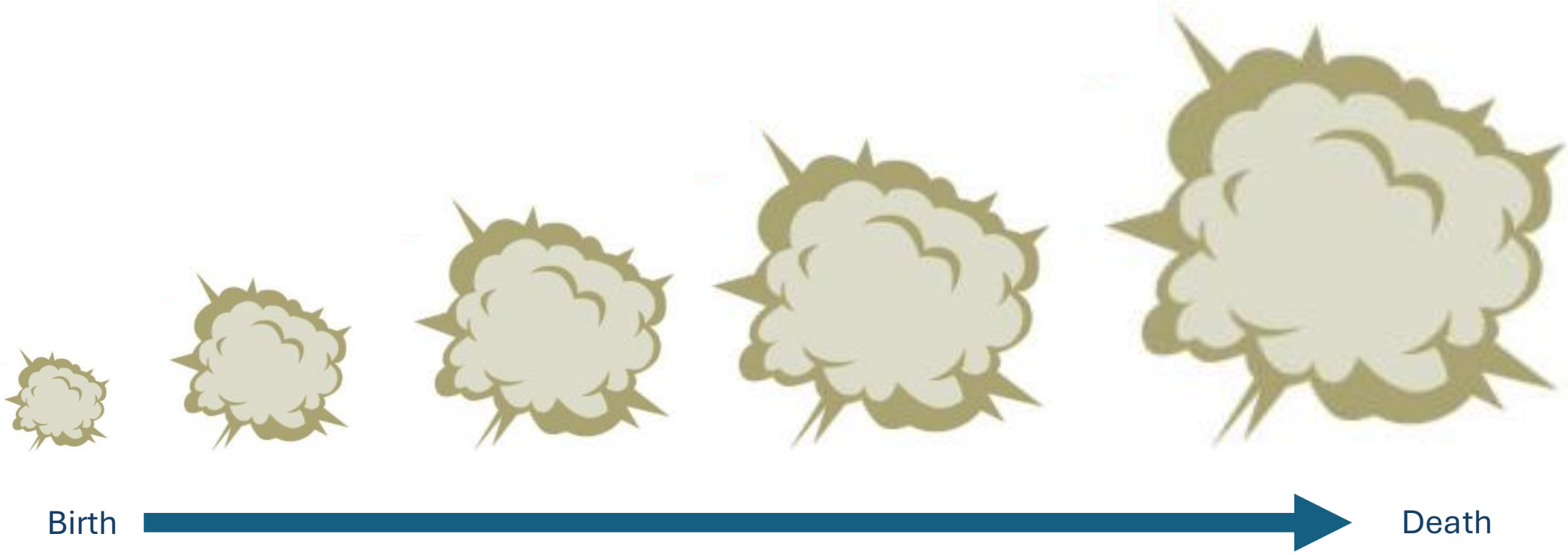
The Antigenic Cloud



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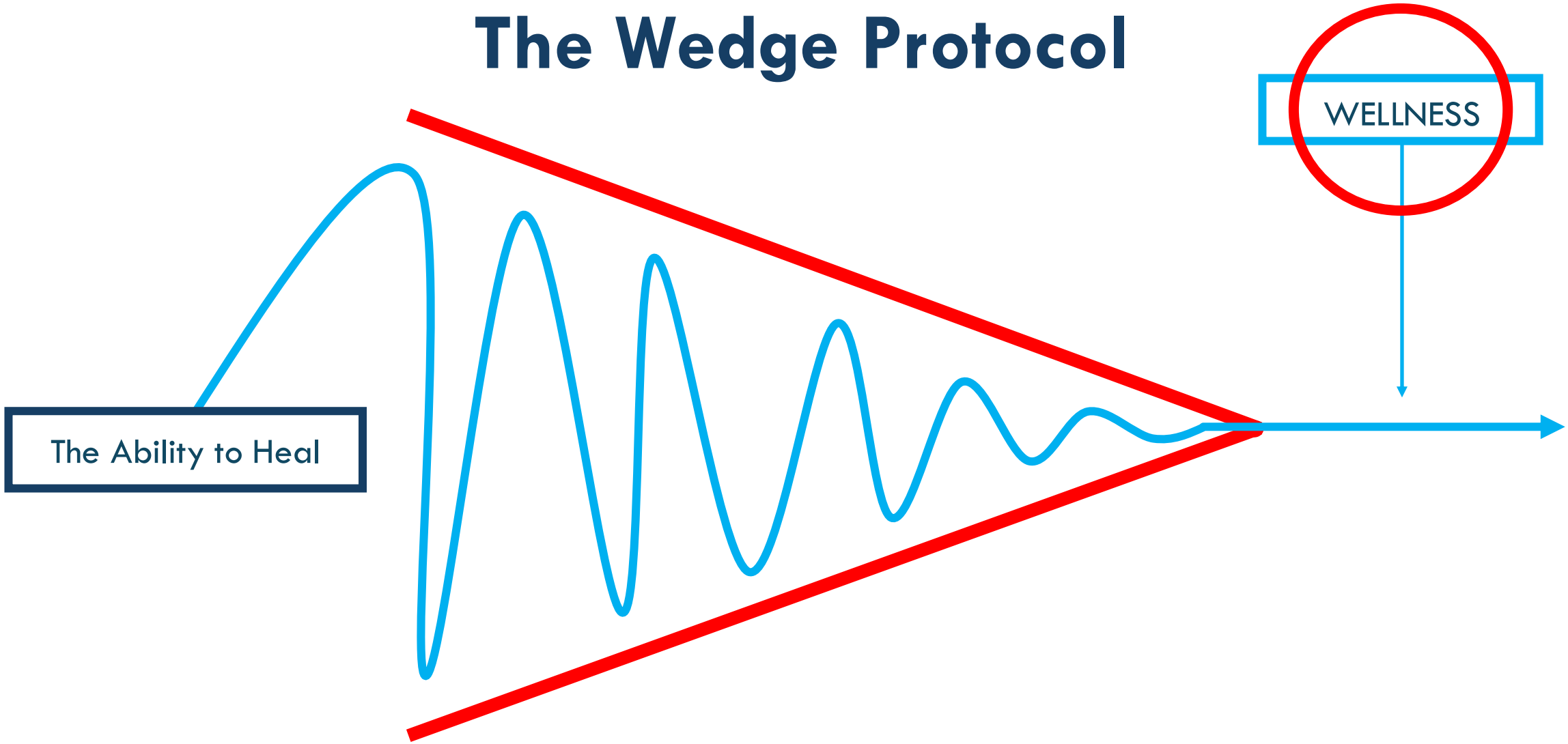
The Wedge Protocol



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Building Resilient Kids

Nervous system regulation

Mitochondrial function

Immune balance

Nutrient sufficiency

Healthy attachment

Environmental inputs

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Children (Basel). 2017 Nov 15;4(11):98. doi: 10.3390/children4110098

Clinical Profile Associated with Adverse Childhood Experiences: The Advent of Nervous System Dysregulation

Jorina Elbers [1,*], Cynthia R Rovnaghi [2], Brenda Golianu [3], Kanwaljeet J S Anand [2,4]

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There is an emerging understanding of intricate links between stress and the development of stress-related medical symptoms. Somatic and autonomic symptoms are commonly reported by combat veterans [1], refugees [2] and abuse survivors [3]. Adults with a history of conversion disorder [4], somatization [5], and migraine [6] have reported high levels of childhood trauma and abuse. Exposure to early life stress can alter neuroendocrine structure and function through neuroplasticity, and may contribute to physical and mental health conditions [7,8]. Early life stressors have been associated with cortisol [9] and autonomic dysregulation [10], in addition to an increased risk of adverse health outcomes ranging from cardiovascular disease and lung disease to cancer [8]. The study of toxic stress is an emerging field of scientific focus, with a recent American Academy of Pediatrics statement calling for science-based efforts in our understanding of childhood adversity [11]. Over a decade of research now suggests that toxic stress causes chronic, dysregulated activation of the stress response system [12,13,14].

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Stress activates a cascade of neural and neuroendocrine responses mediated by the autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA)-axis. Previous studies have identified dysregulation of autonomic and cortisol responses in adults and children with a history of trauma [14,15,16]. Similarly, autonomic and HPA axis dysregulation has been implicated in the pathophysiology of stress-related symptoms such as migraine [17,18], dizziness [19], digestive problems [20], and panic disorder [16]. Medical syndromes of migraine, depression, and post-traumatic stress disorder (PTSD) exhibit a similar list of co-morbid symptoms, suggesting a shared pathophysiology. Despite the critical nature of the ANS and HPA-axis, pediatricians frequently do not assess their function as they fall outside standard laboratory, neurophysiological, and neuroimaging investigations. If ANS and HPA-axis dysregulation is implicated as a cause of medical symptoms, their widespread actions throughout the nervous system would naturally result in symptoms across many of the nervous system's functional domains.

Table 1.

Group A: Symptom profiles of patients with ≥4 medically unexplained symptoms and total adverse childhood experiences (ACE) scores.

Patient (n = 17)	Age (Years)	Sex	Medically-Unexplained Symptoms						Other Medical Problems	Duration of Medical Symptoms	Total ACE Score
			Executive Dysfunction	Sleep Problems	Autonomic Symptoms	Emotional Dysregulation	Digestive/Urinary Problems	Somatization			
1	14	F	Attention problems	Insomnia	None	Anxiety, panic attacks	Constipation, frequent urination	Tremor, weakness	Asthma, chronic fatigue	6 years	3
2	8	F	Poor memory and attention	Nightmares	Dizziness	Cries easily, temper tantrums	None	Headache, blurred vision		4-months	3
3	14	M	Poor attention	Insomnia	Dizziness, syncope	Anxiety, depression, self-harm, suicidality	Diarrhea	Headache weakness, paresthesias	Sinus bradycardia, chronic fatigue	8-months	2
4	15	F	Poor memory and attention	Insomnia, frequent night wakings	Dizziness, syncope,	Anxiety, depression, panic attacks	Constipation	Headache, weakness, paresthesias, abdominal pain	Asthma, cardiac rhythm disturbance, chronic fatigue	4-months	2
5	15	F	Poor concentration	Insomnia	Dizziness	Anxiety, OCD, depression, panic attacks	Reflux, diarrhea	Headache, paresthesias, abdominal pain	Asthma	18-months	1

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Medically unexplained symptoms account for 10–30% of pediatric primary care visits [22] and up to 60% of adult neurology referrals [23]. Comorbidities are common, with many patients exhibiting multiple somatic symptoms, emotional dysregulation, sleep problems, and cognitive complaints, but without a clear explanation or a shared pathophysiology [24]. In our study of 100 consecutive pediatric neurology patients over 5 years of age, 17% patients presented with at least four medically unexplained symptoms for longer than three months. Among these, 29% were being home-schooled, 59% had missed school 10 days or more, and 59% experienced impact on regular school attendance or participation in extracurricular activities. In addition, 65% patients had been seen by at least one other medical subspecialist, and 65% had at least one visit to the Emergency Department within the last year. Patients with multiple medically unexplained symptoms represent a common and fiscally important patient population within our medical and education systems, yet their underlying pathophysiology and most effective treatments are not well understood.

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In children, exposure to early trauma and toxic stress has been associated with learning difficulties, insomnia, eating disorders, asthma, and viral infections [15]. In our study, patients with 4 or more medically unexplained symptoms were similarly more likely to have eating disorders and asthma. Further, patients with this symptom profile were significantly more likely to have a history of adverse childhood experiences and a higher total ACE score. The high prevalence of ACEs in this study suggests that physicians should screen for adverse experiences in children with a clinical profile of multiple medically unexplained symptoms across at least 4 functional domains. The unexplainable nature of these symptoms, and the rising cost of indeterminate investigations and ineffective treatments, suggest that an alternate approach to these patients would benefit both the patient and the health care system. The long-standing and repeated associations between stress and medically unexplained, or psychosomatic, symptoms across decades of research, implicate the stress response system in the generation of these symptoms.

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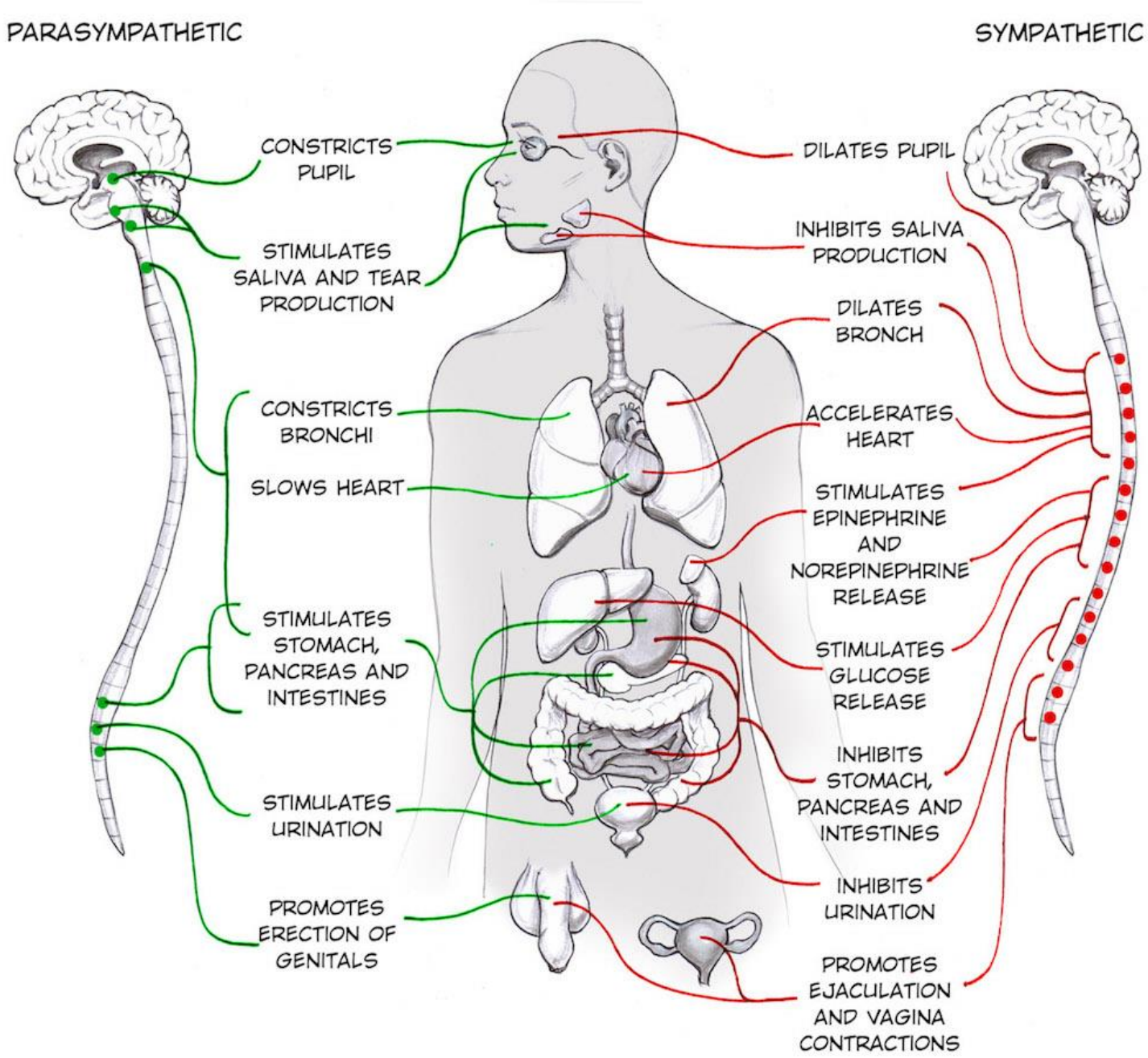
The developing nervous system is particularly susceptible to the effects of extreme or chronic stress. Neuroplasticity adapts to repeated stress responses and establishes a new physiological baseline characterized by structural changes in the brain and dysregulated neural and hormonal responses within ANS, HPA-axis, and sleep/arousal systems [12,25]. Chronic stress leads to elevated levels of corticotropin-releasing hormone, hippocampal cell loss, reduced prefrontal cortical volume, and associated cognitive dysfunction [26]. Children who experience adverse events also develop dysregulated neural responses resulting from chronic or toxic stress. Autonomic testing in adults and children with PTSD demonstrates dysregulated autonomic control, with increased sympathetic and decreased parasympathetic activity [16,27]. Dysregulation of the HPA-axis and ANS is similarly reported in patients with migraine [17,28], dizziness [19], digestive problems [20], sleep disturbance [29], attention deficit hyperactivity disorder [30], and panic disorders [16]. The vagus nerve, traditionally seen as the brake for the sympathetic nervous system, becomes less active during the stress response. This creates an internal system which is unable to effectively maintain its important functions for repair, restoration, and health. When heart rate variability was assessed in the Framingham Heart Study, autonomic dysregulation and decreased vagal function was associated with hyperglycemia [31], risk of new cardiac events [32], and all-cause mortality [33]. Therapeutically, vagal nerve stimulation is a new effective treatment for patients with migraine [34], depression [35] and chronic pain syndromes [36], implicating vagal nerve dysfunction in their pathophysiology. A dysregulated nervous system that has adapted to chronic stress over time with low vagal activity may impair the body's ability to effectively regulate all the functions of the nervous system including sleep, digestion, autonomic function, motor function, and sensory perception. In the absence of appropriate investigations, the clinical consequences of nervous system dysregulation are likely to present as multiple, medically-unexplained symptoms across multiple subspecialty domains.

Autonomic Nervous System



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DRIVERS OF SYMPATHETIC TONE

Things in a kid's world that can turn up the body's "go" system

STRESS & WORRY

Big feelings, worries about school, friends, family, or new situations.

LACK OF SLEEP

Not enough rest makes it harder for the body and brain to reset.

PERFORMANCE PRESSURE

Tests, presentations, sports, or trying to do your best.

More sympathetic tone can help us get ready to act, stay alert, and handle challenges.

LACK OF MOVEMENT

Sitting for too long or not enough active play can keep the body in a higher alert state.

SENSORY OVERLOAD

Loud noises, bright lights, crowded places, or too much happening at once.

UNPREDICTABLE OR UNSAFE ENVIRONMENTS

Conflict at home, feeling unsafe, changes in routine, or not knowing what to expect.

FOOD & DRINKS

Too much sugar, caffeine, or skipping meals can spike energy and stress hormones.

These drivers are normal! Our goal is balance—helping kids find calm, connection, movement, rest, and predictability so their body's "go" system can turn down when it's safe to do so.

Effect of Magnesium Deficiency on Autonomic Circulatory Regulation in Conscious Rats

Yoshinobu Murasato, Yuji Harada, Masaharu Ikeda, Yasuhide Nakashima, Yoshiaki Hayashida

Abstract—A close relationship between magnesium and cardiovascular function has been reported; however, the effect of magnesium deficiency on autonomic cardiovascular regulation has not been clarified. We investigated the effect of magnesium deficiency on the autonomic regulation of oscillations of the R-R interval, arterial blood pressure (BP), and renal sympathetic nerve activity (RSNA) by using the maximum entropy method in conscious rats. Its effect on baroreflex control of RSNA and heart rate were also investigated with a logistic function curve. Mean BP in magnesium-deficient rats was higher than that in control rats (mean±SE, 114.0±4.3 versus 101.6±3.4 mm Hg; $P<0.05$), and urinary excretion of catecholamine was increased by 2.4-fold. The fraction of low-frequency oscillation of RSNA was reduced (31.7±0.9% versus 36.2±1.5%, $P<0.05$) and the correlation between low-frequency oscillations of BP and RSNA was weakened in magnesium-deficient rats. There was no difference in high-frequency oscillation of the R-R interval, which is related to vagal tone, whereas sympathetic tone became dominant (square root of low-frequency/high-frequency ratio of R-R interval, 1.00±0.05 versus 0.67±0.05, $P<0.0001$) in magnesium-deficient rats. The maximal gain in the BP-RSNA relation tended to be reduced in magnesium-deficient rats (−7.7±1.1% versus −12.2±1.9%/mm Hg, $P=0.07$); however, that in the BP–heart rate relation was increased (−8.1±0.7 versus −4.5±0.5 bpm/mm Hg, $P<0.01$). These results suggest that magnesium deficiency induces sympathetic excitation, which results in hypertension but attenuates the baroreflex-related response of sympathetic nerves, whereas magnesium deficiency enhances the sensitivity of the sinus node to autonomic regulation. **(*Hypertension*. 1999;34:247-252.)**

Int J Endocrinol. 2018 Apr 16;2018:9041694. doi: 10.1155/2018/9041694

Magnesium and Human Health: Perspectives and Research Directions

Abdullah M Al Alawi [1,2,✉], Sandawana William Majoni [1,3,4], Henrik Falhammar [1,3,5,6]

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PMCID: PMC5926493 PMID: 29849626

Abstract

Magnesium is the fourth most abundant cation in the body. It has several functions in the human body including its role as a cofactor for more than 300 enzymatic reactions. Several studies have shown that hypomagnesemia is a common electrolyte derangement in clinical setting especially in patients admitted to intensive care unit where it has been found to be associated with increase mortality and hospital stay. Hypomagnesemia can be caused by a wide range of inherited and acquired diseases. It can also be a side effect of several medications. Many studies have reported that reduced levels of magnesium are associated with a wide range of chronic diseases. Magnesium can play important therapeutic and preventive role in several conditions such as diabetes, osteoporosis, bronchial asthma, preeclampsia, migraine, and cardiovascular diseases. This review is aimed at comprehensively collating the current available published evidence and clinical correlates of magnesium disorders.

Children (Basel). 2014 Sep 29;1(3):261–279. doi: 10.3390/children1030261

Iron, Magnesium, Vitamin D, and Zinc Deficiencies in Children Presenting with Symptoms of Attention-Deficit/Hyperactivity Disorder

Amelia Villagomez [1,*], Ujjwal Ramtekkar [2]

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Abstract

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder increasing in prevalence. Although there is limited evidence to support treating ADHD with mineral/vitamin supplements, research does exist showing that patients with ADHD may have reduced levels of vitamin D, zinc, ferritin, and magnesium. These nutrients have important roles in neurologic function, including involvement in neurotransmitter synthesis. The aim of this paper is to discuss the role of each of these nutrients in the brain, the possible altered levels of these nutrients in patients with ADHD, possible reasons for a differential level in children with ADHD, and safety and effect of supplementation. With this knowledge, clinicians may choose in certain patients at high risk of deficiency, to screen for possible deficiencies of magnesium, vitamin D, zinc, and iron by checking RBC-magnesium, 25-OH vitamin D, serum/plasma zinc, and ferritin. Although children with ADHD may be more likely to have lower levels of vitamin D, zinc, magnesium, and iron, it cannot be stated that these lower levels *caused* ADHD. However, supplementing areas of deficiency may be a safe and justified intervention.

Pathway / Reaction	Mg Role	Nervous System Function	Low Mg Effect
ATP → Mg-ATP complex	ATP is biologically active mainly as Mg-ATP	Energy for neurons, ion pumps, synaptic transmission	Fatigue, dysautonomia, poor neuronal firing control
Na^+/K^+-ATPase pump	Mg cofactor for ATPase activity	Maintains resting membrane potential	Tachycardia, palpitations, hyperexcitability
Ca^{2+}-ATPase pumps	Mg required for calcium transport	Prevents intracellular calcium overload	Muscle tension, vasospasm, excitotoxicity
NMDA receptor regulation	Mg blocks NMDA channel at rest	Prevents excessive glutamate activation	Anxiety, panic, sensory hypersensitivity
Glutamate metabolism	Mg-dependent enzymes support balance	Excitatory neurotransmission control	Excitotoxic stress, sympathetic activation
GABA synthesis (glutamate decarboxylase support)	Mg supports enzymatic environment	Main inhibitory neurotransmitter	Anxiety, insomnia, autonomic instability
Tyrosine hydroxylase regulation	Mg indirectly stabilizes catecholamine synthesis	Dopamine → norepinephrine production	Erratic adrenaline output
COMT activity support	Requires Mg for methylation efficiency	Breaks down catecholamines	Adrenaline accumulation
Acetylcholine release modulation	Mg competes with calcium at synapse	Parasympathetic signaling balance	Excess acetylcholine release or instability
Serotonin synthesis pathways	Mg needed for tryptophan metabolism	Mood/autonomic regulation	Depression, vagal dysfunction
Melatonin synthesis	Mg supports serotonin → melatonin conversion	Circadian/autonomic rhythm	Sleep fragmentation
Nitric oxide synthase regulation	Mg affects endothelial function	Cerebral blood flow/autonomic vascular tone	Vasoconstriction, migraines
Adenylate cyclase / cAMP signaling	Mg required for signaling enzymes	GPCR autonomic signaling	Receptor instability
Methylation reactions (SAMe cycle)	Mg needed for ATP-dependent methylation	Neurotransmitter metabolism	Poor stress resilience
Mitochondrial oxidative phosphorylation	Mg stabilizes ATP production enzymes	Neuronal energy generation	Brain fog, autonomic fatigue

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