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Stakekold ZXSY Brazil, Precision Medicine in Pediatric Clinic: Reward Deficiency Syndrome (RDS) Is Surprisingly Evolving and Found Everywhere: Is It "Blowing in the Wind"? Yes, but how Syndrome Z.

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Introduction

Much neurobiological and psychological knowledge about brain development and maturation in pregnancy and early childhood should be evaluated simultaneously, due to several underlying pathophysiological findings, because with proven clinical evidence, with time greater than twenty years, such as situations of Adverse Emotions in Childhood (ACE), Post Traumatic Stress Disorder (PTSD), and recently the absence/ deficit of oxytoninergic neurons responsible for family synchrony, cause a series of aberrant pathophysiological effects, with inflammatory, behavioral, cognitive, affective and social impairments [1].

Functional neuroimaging studies, associated with evidence of common and specific family behaviors, together with genetics and neurobiologies, have broadened the approach of endophenotypes, and the expression of simultaneous phenotypes, which have been objectively described, and are equivalent in empirical clinical practice [2].

Since 1995, Blum et al. have studied dopamine-dependent behaviors associated with epigenetic changes, the presence of polymorphisms, and several other mechanisms, which produce the hypodopaminergic state or syndrome (Sd) of the dysfunctional reward system (RDS) [3-6]. Converging evidence from epidemiological and neurobiological studies that ACE and PTSD as sexual and physical abuse, are closely related to lasting brain dysfunctions that affect physical and mental health, producing traumatic signatures, tonsillar neuroadaptations, which by activation of these neurons of "defense" and mirror neurons, unconsciously reproduce what they received from their parents (family psychodynamics) and reproduce in many cases a chronic child neglect or chronic unconscious parental alienation [7- 9].

In addition to contributing to secondary mental disorders such as addictions, psychosomatic disorders, cardiovascular diseases, colorectal adenocarcinoma, immunoinflammatory diseases , parents and health professionals themselves continue to maintain unconscious neuroadaptation without the concern of neuromaladaptation "of their children, and without any curiosity or motivation, and yet deny, with irony or arrogance, the presence of the facts, clinically evidencingtheirown family schemes (Young) [10-33].

For what mother would not want her child to develop without neuromaladaptation, or with the presence of fluid intelligence? But first there is a need for psychoaction and awareness. Any automatic behavior of the human being, mainly of tonsillar origin, produces a momentary state of mild *alexithymia* (inability to observe oneself effectively) associated with *anosognosia* (inability to observe others effectively), thus causing a picture of chronic unconscious parental alienation [10-13].

In addition to automatic relieving behaviors from parents to children and vice versa, children are currently growing up and suffering deactivation of the neuronal systems of the arched and uncinate fascicles by loud voice command of the parents, producing infantile simultanagnosia (inability to identify more than four objects in the same second) [10-14]. When a family member has a workaholic (neurological and psychological dependence on work work) unconsciously, it produces the

effect of asynchrony, reproducing false effectiveness and still activating a family scheme, it unconsciously evidences its effective disconnection or family asynchrony [15-17].

We clinically update with hierarchical sequence the etiopathogenesis of RDS Spectrum Disorders, adding descriptions of the unconscious effects of psychoanalysis that neurobehavioral neurobiology produces, without theory [3-18].

As a clinical finding, we empirically observed the neurobiological and clinical causal link changes that the Neurological Deficit of Familial Asynchrony (DNAF) triggers in a cascade, tonsillar neuroadaptation of fear, hyperstimulation of the basic primitive neuronal system SEEKING, presence and dysfunctions of hypodopaminergic RDS genes (GWARS), which further increase the effects by the quantities and intensities of episodes of ACE and PTSD [18-23].

Blum *et al.*, used the term Generational Family Disease in studies of RDS families, as other authors have already used the same term of generational disease, we classified it as infantile Z Syndrome, because it is possible to identify in up to three family generations clinically, and organizes a clinical "*stakehold*", in precision medicine, of dysfunctional neurobehavior of RDS that evolves with the metabolic syndrome (Sd X) [31- 34].

RDS children present emotional and cognitive intelligence, speech delay, are pragmatic, intense, perform hyperfocus in areas of interest, playful, empathetic, perceive the emotional state of another child, exchange looks with the environment, perceive three to four objects in the same instant, present subitization with geometric figures, difficulty in mathematics and grammar, hyperkinetic, difficulty concentrating in the classroom with mental fatigue, have deficits in inhibitory control, and emotional dysregulation, primary insomnia, usually lose their reason in family conflicts [3-6].

They have gutted imaginations, hypersensitive to pain, changes in anti-saccadic eye movements, and the minds of geniuses or survivors, according to Blum. *et al.*, because they are hypodopaminergic babies and are surviving without family synchrony, and without interventional medical plans [7-45].

They present disturbances in cognitive processing, affective fear, insecurity, duality, greater attention to threatening stimuli, phobias, and especially it is about Chronic Unconscious Family Alienation, the disease is always bilateral, never only of the child or only of the parents [33-39].

Childhood obesity, the presence of diabetes with childhood insulin resistance, should be reassessed if there is presence of chronic unconscious hypodopaminergic behavior associated with asynchronous parental habits [40-44].

A window of prevention and therapeutic plan emerged, observing signs of hypodopaminergic babies, observing prenatal causes, we have factors that influence family synchrony and cause stress to the fetus, such as maternal hypoxia, restricted intrauterine growth, maternal chemical dependence, prematurity, placental diseases, and other causes already evidenced [45-48].

Poor fetal growth has been widely used as a marker of an adverse intrauterine environment and extensively studied when researching prenatal influences on brain development and behavior. Fetal growth is influenced by maternal, placental, and genetic factors [48-54].

A meta-analysis demonstrated that extremely low birth weight was associated with inattention, hyperactivity, and internalization problems in childhood and adolescence, which converge to higher rates of social problems, depression, and anxiety in adulthood [45-55].

Objective

The objective of this qualitative mini-review, for convenience, and empirical clinical description, is to present a consistent overview of the present literature on the lasting impact on child health by ACE, PTSD, DNAF, Sd Z infantile with real neurological, psychological, social changes, which are subtle, which can be observed with clinical training, subtle behaviors of child suffering, in adolescence, and later in adulthood. Therefore, we searched the PubMed, Web of Science, PsycINFO databases for literature and included manuscripts based on original research as well as reviews and meta-analyses and brought results to an objective, robust evidence-based clinical medical view.

Neurological Dysfunction of Familial Asynchrony

Physiological familial synchrony is developed through the neurostimulation of peripheral oxytoninergic neurons mainly in the NB through maternal touch, and other physical senses, which must interact by the reaction of the NB. The family synchrony depends on these neurons, being a directly mechanical dysfunction, in which we evaluate in the relationship of parents and children, bilaterally at first, and then in a multidimensional way [56-59].

Oxytoninergic dysfunction runs during the window of early childhood, and it is not about the amount of affection, love, time, quality, quantity of attention, presence, but rather establishing an empathic compression, or a perfect bond, to the point of seeing what the child needs at the moment, instead of being in doubt, using societal standards, cultural, or that we have fears that we lack what we have lacked [59-63].

Oxytocin(*OT*) and Vasopressin (*VP*), the hormonal neuropeptides, are responsible for the functioning of familial synchrony, maternal behavior. Oxytocin is involved in social recognition, as well as in the preference of similar individuals such as family members, because its signaling alters the reward properties of social stimuli [64-69].

They present central release and act on the cortico-limbic systems, and are associated with behaviors related to

attachment, stress regulation, social communication and emotional reactivity (perception of internal states and emotional states of other individuals) [70-73].

Attachment can be broadly defined as the process by which an NB has an innate biological need to stay close. Intimate contact creates safety experiences and impacts the relieving systems [75-79].

Cortisol levels increase in the child when separated from the parents briefly (child stressed by chronic exposures), while endogenous opiates, glutamate, and dopaminergic systems are responsible for the development of emotional regulation capacity [80-85].

Attachment-related behaviors are primarily sustained by *OT* and *AVP*, tonsillar neuroadaptations. Work in the brain areas responsible for controlling emotions and motivation (anterior cingulate gyrus, amygdala and orbitofrontal cortex) Individual asynchrony is observed by the individual who is performing his activities, but only in his rigid internal reality with the presence of alexithymia, anosognosia, has the false feeling that you are in the right direction, because you are not violating any laws, you are fulfilling your life goals and beliefs, but you are with the reality produced, different from the behavior and the illusory speech [85-89].

OT expression would allow animals to overcome their natural avoidance of proximity and inhibit defensive behavior while AVP modulates typical male behaviors, including aggression, bonding[84- 89].

In other regions, OT was less expressed, such as the basal ganglia and the amygdala. In the hypothalamus, there is an increase in gene expression, by the observation of neuronal products of the OT-VP system, which they express in their receptors, in which single-nucleotide polymorphisms are the most common causes of variability in the system, when low parental contact, in the brain regions, orbitofrontal cingulate cortex, and anterior [89-93]. Polymorphisms in *AVPRs* are linked to variability in social behavior. Polimorphisms in the *AVPR1B* receptor gene were associated with levels of aggressiveness in children, or empathy and prosociality in adults, have a higher level of methylation in the first intron of *OTR*, which was associated with hip conectivity in cortical areas [94 -99].

Blocking CB1 receptors after the formation of pair bonds increases the occurrence of a specific form of social rejection – vertical defensive response – which is displayed towards the partner, but not towards a new individual [99].

Studies have shown that the action of oxytocin on the nucleus accumbens (Nac), which regulates by social experience the trajectory of social interactions as the relationship with the partner progresses, and promotes the maintenance of the pair bond, inhibiting aggressive responses [100].

It is a subtle connection, it is still unconscious, unprecedented and difficult to accept, because it is necessary to have specific neurons to perform, which is only acquired with cognitive training [100].

The DNAF triggers a series of chronic neurohormonal dysfunctions in early childhood, common to every human being regardless of ethnicity [8-9]:

1. neuroadaptation of fear defense by the tonsillar and limbic neurological systems (YOUNG's Family Schemes) [56 to 59];
2. Dysfunctions of the family roles of psychodynamics, with **inversion**, **excess** or **absence** of function of the roles in the family system, which simultaneously reproduce the reality of family asynchrony inherited from the parents, through mirror neurons and activation of maladaptive schemes and behaviors [56 to 59];
3. **Chronic hypodopaminergic state** due to hyperstimulation of the primitive emotional system SEEKING, associated with genes of the (GWARS) Reward System Dysfunction Syndrome (RDS) described by Blum et al [115-119].
4. Worsening of the hypodopaminergic state, with specific factors such as specific obstetric diseases, ACE, PTSD, stressful situations (Covid-19 for example) [88-98].

The DNAF is subtle, and clinically observed by the dialectic (conscious and unconscious) of the family relationship, identifying if there is unconscious neglect, as a secondary gain, usually with denial, avoidance, aversion or rationalization through behaviors, discourses, all unconscious [60-89].

We are faced with the problem with neurobiological, clinical neurobehavioral, and genetic background (genes that worsen family asynchrony) of chronic and unconscious parental alienation [56-78].

Currently we observe only at the moment of trauma, which becomes conscious, sharpening the pathological behavior, usually of infantile background, which is not aware of the neurological and psychological injuries to the child [56-67].

Other key functions in the assessment of neurological familial asynchrony are resilience, empathy, enforcement function, semantic capacity.

Neurobiology: ACE and PTSD

The brain regionsmost consistently associated with PTSD include the complexes of the amygdala, hippocampus, insular cortex, and areas of the dorsolateral prefrontal cortex, including the anterior cingulate, dorsal, striatum, thalamus, and sensory areas. . These brain regions work for the initial acquisition and subsequent expression of fear memory [72-87].

There are numerous studies with evidence of disorders related to ACE + PTSD and lasting effects on the structure and function of neural stress-regulating circuits. Stressful factors such as ACE and PTSD alter the maturation of the hippocampus, amygdala and ACC, causing changes in control and response

to stress, emotional dysregulation, which with the increase of glucocorticoids, also occur dendritic atrophy and suppression of neurogenesis, as they have high density and effects of glucocorticoid receptors [72-87].

Neuroendocrine and neuroimmune findings in PTSD include lower baseline cortisol output, increased glucocorticoid receptor function, and a pre-, peri- and post-trauma pro-inflammatory response while early trauma andcontinuous measurement are associated with similar observations. The endocannabinoid system is related to the pathophysiology of stress-related psychiatric disorders such as PTSD [72-87].

The main endocananinoids are: anandamide and 2-arachidonoylglycerol that act on type 1 (CB1) and (CB2) receptors. The CB1 receptor is widely expressed in the prefrontal-limbic system and, together with anandamide, positively modulates the extinction of fear, acting mainly on the hippocampus and basolateral amygdala [66 -98].

The acidergic gamma-aminobutyric (GABAergic) neurosteroid system in the biosynthesis of allopregnanolone, which is a positive allosteric modulator of GABA action on GABAA receptors and regulates emotional behavior, exerting anxiolytic, antidepressant, and sedative effects [72-87].

There are several studies with classical alterations of single nucleotide polymorphisms in the brain-derived neurotrophic factor (BDNF) gene, which is the substitution of valine (Val) for methionine (Met), which interacts in a three-dimensional way, in the promoter protein linked to the serotonin transporter (5-HTTLPR) [78-89].

These changes are correlated with decreased brain volume in the hippocampal areas and its subfields, amygdala and PFC and ACC [78-89]. Studies using specific genes have shown the neurotrophic factor of the brain (BDNF) that has neuroplasticity function in the entire brain system, and is altered when the effects of childhood emotional adversity, encoded by the BDNF gene, are present and present different BDNF genotypes. Polymorphisms in the variations in Val66Met (rs6265) influence attention problems in adopted young people, which depends on the duration, intensity of institutional care, as they interact or add to the effects of child maltreatment. Changes in the Met gene evidences right amygdala volume increases in methylated cytosine-phosphate-guanine [72-87].

The evaluation of the confirmatory model indicated that the interaction effect involving the ‘’maternal warmth’’ reasoning was in accordance with the differential susceptibility rather than the diathesis-stress model of the person-X-environment interaction. Thus, Val’s patients had less depressive symptoms than Met homozygotes when motherhood was more present (Synchrony), and worse symptoms when maternity was less present (Asynchrony) [45-68].

DNA methylation is one of the most studied epigenetic mechanisms in relation to psychiatric diseases. DNA methylation, defined as the addition of the methyl group on the fifth carbon of cytosines 5-methylcytosine. Studies have shown that methylations of the *FKBP5*, *NR3C1* and *SLC6A4* genes are associated with dysfunctional connectivity and decreased brain volume, facilitating depression in adolescents [72-87].

The increased transcriptional response to gene expression of ACE has been observed in low socioeconomic status, chronic stress, post-traumatic stress, and its expression increases the immune system response with pro-inflammatory cytokines, IL-2, IL-6, and inhibition of interferon Gamma (IFN-γ) formation required in the responses of virus-infected defense cells [45-92].

The neurobiology of Pavlovian threat memory acquisition is well characterized. This process is particularly relevant to understanding PTSD, as exposure to PTSD-inducing trauma is often considered an example of human naturalistic fear conditioning [72-80].

Consequently, the individual will exhibit fear-related behavior in response to the conditioned stimulus, regardless of whether or not it is accompanied by the aversive unconditioned stimulus. Evidence from neuroimaging, injury, and pharmacology studies in species suggests that information about the conditioned stimulus and the unconditioned stimulus converges in the lateral and basolateral amygdala through afferent pathways of the thalamus and cortex, inducing synaptic plasticity at the level of the basolateral amygdala [54-80].

Subsequent activation of the central amygdala,through the entry of the basolateral amygdala, elicits conditioned fear responses triggered by stimuli, including freezing, increased heart rate, activating downstream brain areas such as the hypothalamus, locus coeruleus, and other brainstem nuclei [72-87].

Laboratory studies have shown that individuals with PTSD increased fear conditioning, extinction deficits, and physiological signs (sympathetic responses) associated with current functional neuroimaging findings of fear response (hyper-reactive amygdala and anterior cingulate) when compared with healthy control participants [72-78].The negative health outcomes that follow later in life reflect the physiological, epigenetic, and cognitive consequences that the brain and body pay to adapt to stressful and traumatic experiences [72-98].

A systematic review of eighteen studies showed that fibromyalgia has a significantly important correlation with physical and sexual abuse in childhood, more frequently in children with ACE [31-65].

Protective factors such as the social support of a nurturing adult, living in a safe community, and resilience play a protective role in adaptive development [120].

Built on a wealth of neuroimaging, neurostimulation, and neuropsychology data, such as these new clusters we are

presenting, one "*cluster*" already studied is *controlled semantic cognition* (SCC). The purpose of the CSC is to assess the sustaining of semantic cognition a. The 'semantic control' system, which features activity in the prefrontal cortex, dynamically monitors and modulates the 'semantic representation system. The SSC provides that unfamiliar and demanding semantic tasks intensify communication between the systems of 'control' and 'representation' in relation tofamiliar tasks. Although various evidences have shown that both cerebral hemispheres participate in semantic processing, there is an activation asymmetry, in which the left side is disproportionately more activated by written words and speech production, while nonverbal representations or spoken words generate bilateral activations [121].

Severity

Many scientific evidences and clinical *insights* of quantity and quantity of how the impacts of traumas, causes of ACEs, significantly interfere in chronic health conditions throughout life, as they potentiate the chronic hypodopaminergic state, along with epigenetic changes, can be added to other hypodopaminergic genes [35-67].

Felitti *et al*., showed that children with three or more ACEs had a higher risk of various health conditions, such as ischemic coronary heart disease, cancer, obesity, alcoholism, lung disease and depression. Higher exposure to ACE s (≥3 ACEs), in a "dose-response" relationship, is associated with a higher level of mental clinical complexity with worsening of the hypodopaminergic state [87- 90].

ACE and PTSD and somatic consequences

Several studies have shownthat ACE and PTSD are associated with the development of a variety of somatic disorders, such as obesity, diabetes, inflammatory bowel diseases such as ulcerative colitis and Crohn's disease, by dysregulation of the innate immune system as a possible biological mediator between ACE and the disease of adulthood [72-87].

Inflammatory Bowel Diseases are driven by the interaction of gut microflora and environmental factors in a genetically susceptible host. Childhood is an important phase in the development of neurobiological systems, the mucosal immune system, the gut microbiota and immune tolerance in the gut, ACE can contribute to dysfunctions and this effect may depend on the type and timing of ACE [72-90].

Dissociative disorders and symptoms (depersonalization and derealization) were associated with trauma intensity at 13 to 14 years of age, with emotional neglect being the main causeof emotional abuse. Currently there are already risk polymorphisms that amplify suffering [34-80].

Generational Family Disease/ANAAS Syndrome/Chronic Family Alienation

Most stressful events in childhood are correlated, in the presence of states of neuro adaptations of fear, or family schemes (YOUNG), maladaptive behaviors, relieving, which are activated neuronal systems automatically, clinically expressed among family members, because there was a reaction to a stimulus, such as a voice, a word or the simple presence in the family environment [72-87].

According to brain neurobiology, every emotion is a reaction, and it is felt in the physical body. Emotions are primitive neuronal systems, which have a survival function, such as pain, fear. Clinically, distinguishing first a rational behavior from an emotional one, the emotional one is the starting point for evaluation [44].

The neuroadaptation of fear when in activity, consumes much of the basal dopamine, like any automatic behavior, relieving a state of discomfort or pain, (in this case a family scheme) which is the central mechanism of Sd ANAAS (The Asynchrony, Neuro maladaptation, The Alexithymia, Anasagnosia and Simultanagnosia) which is description of the unconscious internal reality [72-87].

The Sd ANAAS is the reason for the worsening of human relations, which we collectively evidenced by the Asynchrony of family, marital and professional relationships. When more severe, by fixing the strength of neuronal connection of the tonsillar systems, and worsens limiting beliefs, reinforces the behavior of denial, avoidance, aversion, generating a vicious cycle [72-120].

Without motivation, without synchrony, with anosognosia and complicating with simultanagnosia in the presence of cortical blindness, and bilateral parietal temporal dysfunction (Sd Antom Babinsky), the treatment becomes very difficult, without motivation. The first steps of treatment are cadenced, but after the intimate effort is individual. The reflection of the attempt of collective exposure of the neurological disease of familial asynchrony should be evaluated [20-21].

Findings of selective atrophy of the anterior parts of the right and left temporal lobes to verify the current cognitive models of identification of familiar people. According to these models, the information coming from the modality-specific "face", "voice" and "name" recognition units converges to "Personal Identity Nodes" [34-79].

Children with Z spectrum have genes of familial asynchrony and RDS, have specific neurological mechanisms and different clinical, cognitive and behavioral signs and symptoms among the child ADHD, which has different genes, pathophysiology of absence of dopaminergic production, unique clinical characteristics, which is also individualized from ASD, with other genes, and other clinical characteristics [45-87].

Disturbiums in the recognition of family members can be observed in patients with lesions in the anterior areas of the temporal lobes, and that these disorders can be multimodal, simultaneously affecting the visual, auditory and linguistic channels that allow the identification of the person. Pacients with right anterior temporal atrophy are more impaired

in assessing familiarity and retrieving specific semantic information from familiar faces than from names, while the opposite pattern of performance can be observed in patients with left temporal lobe atrophy [72-103].

Voice recognition disorders are primarily due to right temporal lesions, similar to facial recognition disorders [72-87]. Fear-related modulations of facial processing driven by amygdala signals may imply not only spindle cortex, but also anterior visual areas in the occipital cortex and other distant regions involved in social, cognitive, or somatic responses (superior temporal sulcus, cingulate, or parietal areas). In the temporal domain, evoked potentials show a generalized temporal course of emotional perception of the face, with some increases in the amplitude of responses recorded in the occipital and frontal regions to fearful faces in relation to neutral faces (amygdala and orbito frontal cortex) [72-100].

The first emotional responses may arise around 120ms, before a full stage of visual categorization indexed by the face-selective N170 component, possibly reflecting the rapid processing of emotions based on raw visual cues in the faces [99-120].

Neuroinflammation

Microglia comprise about 5 to 12% of the total CNS glia population in adults, depending on the brain region. In resting states, microglia are involved in synaptic remodeling, maintenance, and monitoring of the CNS environment with cell surface receptors that bind to antigens, antibodies, cytokines, and hormones [72-87].

Microglia release anti-inflammatory cytokines and cellular debris from cellular defense activity, which causes insults, especially when dysregulated by microglial inflammation are harmful to the tissue environment and can kill healthy neurons [72-87].

Aberrant microglial activation has been associated with epigenetic dysregulation in anxiety spectrum disorders, mood, ACE, autism as well as neurodegenerative diseases [72-80].

In the human cerebral cortex, the glial population includes approximately 20% astrocytes and 75% oligodendrocytes. Astrocytes are responsible for maintaining homeostasis through ion damping, immune signaling, maintenance of the blood-brain barrier, regulation of neuronal synaptogenesis. Epigenetic dysregulation of astrocytes and their reduction in size are correlated with ACE disorders and major depressive disorder [89-120].

ACE and PTSD inhibit the neurodevelopment of microglial synapses leading to an increase in the number of excitatory synapses in neurons expressing corticotropin-releasing hormone [45-60].

Source analysis of potentials related to high-density events is an ideal means to examine both the precise temporal profile and spatial location, of the brain's early electrical activity in response to emotionally salient stimuli in the(high-functioning) usually during explicit and implicit processing of emotions from images showing happy, angry, afraid, sad, and neutral facial expressions [90-113].

Children with autism showed normal patterns of behavioral responses. Dipole source analysis revealed that ERP responses related to face detection (visual cortex) and face configuration processing (fusiform gyrus), as well as mental status decoding (medial prefrontal lobe), were significantly weaker and/or slower in autism during slower and higher amplitude ERP source activity in the parietal somatosensory cortices [72-87].

A possibly reflected more effortful compensatory analytical strategies used by the autism group to process facial gender and emotion. This aberrant neurophysiological processing of facial emotion observed in children with autism within the first 300 ms of stimulus presentation suggests abnormal cortical specialization within social brain networks, which would likely disrupt the development of normal sociocognitive skills [24-87].

Childhood Obesity

Childhood obesity in preschoolers is a global phenomenon in both developed and non-developed countries, where nutritional deficiencies and environmental factors of parents' lifestyles may explain part of their children's health, but where other direct lifestyle factors such as diet and physical activity may also play a critical role in promoting higher energy expenditure [25-87].

Children with obesity have breathing difficulties, increased risk of thiscan also play a critical role in promoting higher energy expenditure. Recent studies have implied that a variety of exposures early in life and in the womb can change metabolism [22-87].

Barker *et al.*, made observations of this phenomenon with several epidemiological studies showing that babies born of short stature, for gestational age have a greater susceptibility to cardiovascular diseases and metabolic dysfunction. From observations of increased adiposity and decreased fat mobilization after fetal malnutrition are insults that during early development can influence cellular plasticity, thus increasing the risk of chronic diseases later in life, including obesity and its comorbidities [34-79].

Currently the etiologies that lead to obesity can be identified in the context of the struggle between nutrition, nature, genetics and epigenetics, environmental and microenvironment. The key race that is central to some chronic diseases, how food cravings are neurologically regulated, how gut hormones, adipose tissue or the gut microbiota regulate appetite and satiety in the hypothalamus, as well as the roles of gut dysbiosis played in the development of obesity and how dysfunction of glucose and lipid metabolism. Genetic factors play critical roles in determining an individual's predisposition to weight gain [35-79].

Evidence has been found with a higher incidence of negative weight-related health problems, such as high triglycerides and twice the chances of developing metabolic syndromes. These problems are compounded in certain individuals who possess a genetic susceptibility to fat accumulation, which can be caused by significant interactions between homeostatic circuits and brain reward [23-79].

Accumulation of lipid metabolites, inflammatory signaling, or other hypothalamic mechanisms that harm neurons may also lead to obesity, which may explain the biological defense of elevated body fat mass [34-58].

Obesogenic marketing to promote beverages or foods high in sugar and fat negatively modulates human behavior, as there are few explorations of the neurocognitive cause of obesity, which we believe we accurately get right [23-56].

The finding of Syndrome Z (initially thebempirically served as a chronic hypodopaminergic state, associated with worsening in family relationships) were identical to patients addicted to substance use, with or without secondary obesity [72-87].

Unwanted foods high in fat and sugar that stimulate the reward centers of the brain, the same part of the brain that is stimulated by cocaine, heroin, and other addictive drugs, meaning these products are specifically designed according to the will of the population. If we look at the tasting tests, and the preferences of double-blind studies of sugar versus stevia, sugar is clearly victorious because people are unconsciously dependent, and this VIES is not considered, and the lack of sugar, is not rationalized by the different Stevia, which does not activate the brain reward pathway [72-87].

The brain reward offers a plausible mechanism to explain the high body fat mass, however, it seems that only certain individuals exhibit this characteristic according mechanism, which is proven by the ZX syndrome [34-54].

The genetic causes of obesity can be broadly classified as: 1) monogenic causes that result from a single genetic mutation, located primarily in the leptin-melacortin pathway. Many of the genes, such as AgRP (agouti-related peptide), PYY (orexogenic) or MC4R (the melanocortin-4 receptor), have been identified for monogenic obesity by disrupting the appetite and weight regulatory system, hormonal signals (ghrelin, leptin, insulin) [72-87].

Paternal obesity has also been associated with inhibited methylation levels in regions of IGF2 (insulin-like growth factor 2), which promote the division and growth of various cell types. Other genes investigated in the context of metabolism and obesity include: tumor necrosis factor (TNF), hypoxia-inducible factor 3a (HIF3A), neuropeptide Y (NPY), insulin receptor substrate 1 (IRS1), mitochondrial transcription factor A (TFAM), and IL-6 [34-87].

Histones are functions of proteins in DNA packaging and histone modifications are associated with epigenetic regulation of adipogenesis and the development of obesity Five key regulatory genes in adipogenesis, CCAAT β (C/EBP β) binding protein, pre-adipococyte-1 factor (Pref-1), adipocyte protein 2 (aP2), PPARγ and C/EBPα, are modulated by histone modifications during differentiation from a Enzymes play roles in the modification of Histones and also work in obesity [72-87].

Chronic inflammatory diseases, such as hypertension, hypercholesterolemia, and insulin resistance, which is the prelude to Type II diabetes, are strongly related to the development of obesity and contribute to the onset of the well-known metabolic syndrome. Insulin receptors are widely distributed throughout the central nervous system [23-89].

They are very abundant in the hippocampus, cortex and cerebellum, protecting neurons from neurodegeneration and cell death, hence memory and learning processes. Insulin resistance associated with obesity can cause an imbalance in neural metabolism [67-88].

Biological pathways related to adult BMI involved in neuronal developmental processes, hypothalamic expression and regulation, cyclic AMP, WNT signaling, membrane proteins, monogenic obesity/energy homeostasis, glucose homeostasis, and cell cycle likely influence adiposity from early life, (Sd ZX) [23-67].

Infant Sleep Apnea

Both obstructive sleep apnea (OSA) and obesity are the main health problems that contribute to the increase in systemic inflammation in children and adults, and have underlying pathophysiology in common simultaneously [72-87].

Differences in the levels of interleukin-6, interleukin-9, basic fibroblast growth factor, platelet-derived growth factor-BB, as well as regulated in activation, normal T cells expressed and secreted are significant. In a study of children who were in the 3-month postoperative period of adenectomy, differences in these inflammatory markers decreased along with a decrease in OSA severity while obesity persisted [78-90]. Previous studies have shown that obesity is one of the most important risk factors for OSA, along with increased age, male sex, and abnormalities of craniofacial structure [72-87].

IL-6 is a myokine produced and released from muscle fibers in response to exercise, and has been shown to have extensive anti-inflammatory functions. IL-6 has been shown to be involved in inflammatory processes in a wide range of diseases, including metabolic, cardiovascular, neurological, and autoimmune cancers and diseases [72-98].

IL-9 secretes by CD4+ helper cells and stimulates cell proliferation and prevents apoptosis. It is encoded by the human IL-9 gene, a candidate gene for asthma, diseases in the amygdala, adenoids and lymphoid tissue, and an important factor in airway hyperresponsiveness. The literature reports

bronchial asthma as an important bidirectional contributing factor to pediatric OSA. When comparing subgroups with the same OSA severity, non-obese patients had higher levels of IL-9 [32-87].

Systemic inflammation is one of the most commonly proposed mechanisms, and has been suggested to influence the severity of OSA and associated comorbidities [67-78].

Discussion

Instead of excluding the law of parental alienation, therapeutic justice, the judicialization of medicine, are fundamental movements, because we have a systematic reorganization, which allows prevention and therapeutic plans, in primary health care. And we observe the difficulty of demonstrating alone to large institutions [72-87].

The distinction of self refers to the self-ability to identify one's physical and mental states, and of other people (actions, perceptions, emotions). Both the right temporo-parietal junction and the brain areas associated with the human mirror neuron system significantly influence the intimate distinction of an emotional state, of a thought and behavior [72-87].

The degree of exact intimate dexterity of oneself and what one feels in reaction to others will vary according to the situation of emotional, environmental, social, and family quality, and how useful or unbothered one feels or remains separate from another person's mental state [44-67].

To start self-observation, it is necessary to have the patient motivated, with the presence of basal dopamine. The automatic, and neuroadaptive, states consume all the basal dopamine, and in a state of hypodopaminergia worsen the control of fear, insecurity, duality, doubt [43-67].

Variation in dysfunction occurs according to the intensity, quality, and sum of causal or strengthening factors, such as ACE and PTSD. The fear of the pain of Familial Asynchrony (MDAF) is the repression of all human beings, associated with other factors that potentiate [89-120].

These are processes harmful to child neurodevelopment, which shape perennially, and with subtle clinic among family members, with avoidance, denial, aversion, avoidance, all unconscious, and produce typical pictures of 'childish' fights of families, such as tantrums, toxic relationships, and severe cases of domestic violence, family alienation, emotional dependence and domestic violence [45-65].

The emotional resonance we can share with others offers the gift of empathy, but oversharing must be individualized if it is not attachment emotion, or pain and mirror [45-68].

The various signs and symptoms associated with the problematic distinction of self-healing comprise maladaptive and adaptable (compensatory) responses to dysfunction within a common underlying neuropsychological mechanism, in which each practitioner presents a different theoretical concept, and so it will always be a maze without systematization. If we always focus on motivated behavior, in relation to speed and response time, it technically organizes the distinction of an automatic or schematic neurobehavior [33-68].

The RDS study was started by Blum around age 27, and we still have no therapeutic or preventive plans, while only worsening and increasing the number of somatic mutations, such as familial asynchrony. In the middle of 2023 super experts cannot identify a behavior, purely dependent on biology, and others cannot [56-100].

The clinical sense that the lack of brain dopamine, brings suffering to the child, adolescent and elderly, is the first step to broaden the clinical view. These people do not know the real reason they suffer, have anxieties, and survive with dopamine-producing behaviors such as smartphone use, overwork, producing asynchrony effect as a secondary gain [45-68].

In children in an educational setting, perpetuation of ACE and PTSD, neglect of Sd Z, and several factors may occur, including the proportion of boys and girls in the classroom, the content and location of the class (ball games, fitness, dance) teachers' decisions about lesson content, time management (the amount of time spent explaining an assignment or the amount of time before moving on to a different assignment), delivery (enthusiastic verbal promotion). This new attention to children's mental health is fundamental, if the educator, teacher, pedagogue, occupational therapist, are not attentive, will neglect an act of family scheme, which is fact, not yet observed [121-123].

Pedagogy is defined as interdependent elements of curriculum design, learning, and teaching, important with physical educators possessing higher levels of pedagogical content knowledge and positive attitudes toward promotion concerns (which often include generalist classroom teachers) not having the necessary level of pedagogical content knowledge to support learning and promote specialized attention that a child with TEA, ADHD, SD Z need [121-123].

Physical education has as its key objective with young children (5 to 7 years of age) thedevelopment of basic movement skills, necessary for healthy living (catching, jumping, swimming, cycling) and helpschildren to develop physically. But for children who have increased frequency of thoughts, and other predominantly biological or psychological characteristics, martial arts exercises, team sports, yoga, are fundamental from the point of view of neurobiological and medical [121-123].

The trained professional has learned to interpret the semantic language of children, which they must learn to develop their emotional intelligences, semantics, corporais, identification of thoughts, basic neurological states family, *mindfulness*, Music therapy, play therapy, effective family skills training. The pedagogy of Rudolf Steiner presents a philosophy that stimulates neurodevelopment, without simultanagnosia,

without tonsillar neuroadaptation and with fluid intelligence, and therefore there will be real chances of prevention against chemical dependence, chronic diseases, domestic violence [121-123].

Conclusion

We believe that some empirical studies like this one, and several others like Blum *et al.*, are enough for us to think with clinical reasoning, and to start studies or to start without waiting remotely the empirical prevention of our children of simultanagnosia, or tonsillar neuroadaptation, because we will have children with development of free thought, free consciousness, with fluid intelligence, in addition to the prevention of family violence, and various diseases, and free of psychological suffering unraveled by the lack ofinitial basal dopa [23-67].

Out of respect, admiration, I dedicate this humble work to the true neuroscientist Kenneth Blum, with the Music "*Logical Song*" by *Supertramp*.

Conflict of Interest

I declare that there is no conflict of interest, I have no affiliation with pharmaceutical companies, I have no political and religious interests.

References

1. Antunes M, Biala G. The new memory of object recognition: neurobiology, test procedure and its modifications. Cogn process. 2012 May;13(2):93-110. DOI: 10.1007/s10339-011-0430-z. Epub 2011 December 9. PMID: 22160349; PMCID: PMC3332351.
2. Heinrich H, Grunitz J, Stonawski V, Frey S, Wahl S, Albrecht B, Goecke TW, Beckmann MW, Kornhuber J, Fasching PA, Moll GH, Eichler A. Attention, cognitive control and motivation in ADHD: Linking brain potentials related to DNA methylation events and patterns in boys of early school age. Sci Rep. 2017 Jun 19;7(1):3823. DOI: 10.1038/s41598-017-03326-3. PMID: 28630479; PMCID: PMC5476641.
3. Duquette LL, Mattiace F, Blum K, Waite RL, Boland T, McLaughlin T, Dushaj K, Febo M, Badgaiyan RD. Neurobiology of KB220Z-Glutaminergic-Dopaminergic Optimization Complex [GDOC] as a Liquid Nano: Clinical Activation of Brain in a Highly Functional Clinician Improving Focus, Motivation and Overall Sensory Input Following Chronic Intake. Clin Med Rev Case Rep. 2016;3(5):104. DOI: 10.23937/2378-3656/1410104. Epub 2016 May 11. PMID: 29214221; PMCID: PMC5714519.
4. Blum K, Badgaiyan RD, Braverman ER, Dushaj K, Li M, Thanos PK, Demetrovics Z, Febo M. Hypothesizing that, A Pro-Dopamine Regulator (KB220Z) should optimize, but not hyper-activate the activity of Trace Amine-Associated Receptor 1 (TAAR-1) and induce anti-craving of psychostimulants in the Long-Term. J Reward Defic Syndr Addict Sci. 2016;2(1):14-21. DOI: 10.17756/jrdsas.2016-023. Epub 2016 Jun 29. PMID: 28317038; PMCID: PMC5351297.
5. Blum K, Thanos PK, Oscar-Berman M, Febo M, Baron D, Badgaiyan RD, Gardner E, Demetrovics Z, Fahlke C, Haberstick BC, Dushaj K, Gold MS. Dopamine in the Brain: Hypothesizing Surfeit or Deficit Links to Reward and Addiction. J Reward Defic Syndr. 2015;1(3):95-104. DOI: 10.17756/JRDS.2015-016. Epub 2015 Oct 23. PMID: 27398406; PMCID: PMC4936401.
6. Blum K, Phoebe M, Fried L, Li M, Dushaj K, Braverman ER, McLaughlin T, Steinberg B, Badgaiyan RD. Hypothesizing that neuropharmacological and neuroimaging studies of Glutaminergic-Dopaminergic Optimization Complex (KB220Z) are associated with "dopamine homeostasis" in reward deficiency syndrome (RDS). Subst Use Misuse. 2017 Mar 21;52(4):535-547. DOI: 10.1080/10826084.2016.1244551. Epub 2016 Dec 29. PMID: 28033474; PMCID: PMC5589271.
7. Lin X, Li H. Obesity: Epidemiology, Pathophysiology and Therapeutics. Frontal Endocrinol (Lausanne). 2021 Sep 6;12:706978. DOI: 10.3389/fendo.2021.706978. PMID: 34552557; PMCID: PMC8450866.
8. Monnereau C, Vogelezang S, Kruithof CJ, Jaddoe VW, Felix JF. Associations of genetic risk scores based on adult adiposity pathways with childhood growth and adiposity measures. BMC Genet. 2016 Aug 18;17(1):120. DOI: 10.1186/s12863-016-0425-y. PMID: 27538985; PMCID: PMC4991119.
9. Wesseldijk LW, Dieleman GC, van Steensel FJA, Bartels M, Hudziak JJ, Lindauer RJL, Bögels SM, Middeldorp CM. Risk factors for parental psychopathology: a study in families with children or adolescents with psychopathology. Eur Child Adolesc Psychiatry. 2018 Dec;27(12):1575-1584. DOI: 10.1007/s00787-018-1156-6. Epub 2018 Apr 11. PMID: 29644474; PMCID: PMC6245117.
10. Gilbar O, Taft C, Dekel R. Male intimate partner violence: Examining the roles of childhood trauma, PTSD symptoms, and dominance. J Fam Psychol. 2020 Dec;34(8):1004-1013. DOI: 10.1037/fam0000669. Epub 2020 April 30. PMID: 32352814.
11. Hamza M, Halayem S, Bourgou S, Daoud M, Charfi F, Belhadj A. Epigenetics and ADHD: Towards an Integrative Approach to the Pathogenesis of the Disorder. J Atten Disord. 2019 May;23(7):655-664. DOI: 10.1177/1087054717696769. Epub 2017 Mar 9. PMID: 28665177.
12. Brown CL, Halvorson EE, Cohen GM, Lazorick S, Skelton JA. Addressing Childhood Obesity: Opportunities for Prevention. Pediatr Clin North Am. 2015 Oct;62(5):1241-61. DOI: 10.1016/j.pcl.2015.05.013. Epub 2015 Jul 16. PMID: 26318950; PMCID: PMC4555982.
13. EA Network, Young LJ. Oxytocin, vasopressin, and pair binding: implications for autism. Philos Trans R Soc Lond B Biol Sci. December 29, 2006;361(1476):2187-98. DOI: 10.1098/RSTB.2006.1939. PMID: 17118932; PMCID: PMC1764849.
14. Dick KJ, Nelson CP, Tsaprouni L, Sandling JK, Aïssi D, Wahl S, Meduri E, Morange PE, Gagnon F, Grallert H, Waldenberger M, Peters A, Erdmann J, Hengstenberg

C, Cambien F, Goodall AH, Ouwehand WH, Schunkert H, Thompson JR, Spector TD, Gieger C, Trégouët DA, Deloukas P, Samani NJ. DNA methylation and body-mass index: a genome-wide analysis. Lancet. 2014 Jun 7;383(9933):1990-8. DOI: 10.1016/S0140-6736(13)62674-4. Epub 2014 Mar 13. PMID: 24630777.
15. Ding X, Barban N, Tropf FC, Mills MC. The relationship between cognitive decline and a genetic predictor of educational attainment. Soc Sci Med. 2019 Oct;239:112549. DOI: 10.1016/j.socscimed.2019.112549. Epub 2019 September 13. PMID: 31546143; PMCID: PMC6873779.
16. Middeldorp CM, Akingbuwa WA, Jami ES, Bartels M. Genetisch onderzoek in de kinder- en jeugdpsychiatrie [Genetic research in childhood and adolescent psychiatry]. Tijdschr Psychiatr. 2022;64(5):295-300. Dutch. PMID: 35735040.
17. Oztan O, Garner JP, Constantine JN, Parker KJ. Neonatal CSF vasopressin concentration predicts later medical record diagnoses of autism spectrum disorder. Proc Natl Acad Sci U S A. 2020 May 12;117(19):10609-10613. DOI: 10.1073/pnas.1919050117. Epub 2020 Apr 27. PMID: 32341146; PMCID: PMC7229671
18. Marquez C, Poirier GL, Cordero MI, Larsen MH, Groner A, Marquis J, Magistretti PJ, Trono D, Sandi C. Peripubertal stress leads to abnormal aggression, altered amygdala and orbitofrontal reactivity and increased expression of the prefrontal MAOA gene. Transl Psychiatry. 2013 Jan 15;3(1):e216. DOI: 10.1038/TP.2012.144. PMID: 23321813; PMCID: PMC3566724.
19. Gilbar O, Dekel R, Hyland P, Cloitre M. The role of complex post-traumatic stress symptoms in the association between exposure to traumatic events and the severity of intimate partner violence. Child Abuse Negl. 2019 Dec;98:104174. DOI: 10.1016/j.chiabu.2019.104174. Epub 2019 September 26 PMID: 31563703.
20. King SE, Skinner MK. Transgenerational Epigenetic Inheritance of Obesity Susceptibility. Trends Endocrinol Metab. 2020 Jul;31(7):478-494. DOI: 10.1016/j.tem.2020.02.009. Epub 2020 March 24. PMID: 32521235; PMCID: PMC8260009.
21. Vuilleumier P, Pourtois G. Distributed and interactive brain mechanisms during emotional face perception: evidence from functional neuroimaging. Neuropsychology. 2007 Jan 7;45(1):174-94. DOI: 10.1016/j.neuropsychologia.2006.06.003. Epub 2006 July 18. PMID: 16854439.
22. Minnebusch DA, Suchan B, Köster O, Daum I. A bilateral occipitotemporal network mediates the perception of the face. Behav Brain Res. 2009 Mar 2;198(1):179-85. DOI: 10.1016/j.bbr.2008.10.041. Epub 2008 November 11 PMID: 19041896.
23. Rossion B, Caldara R, Seghier M, Schuller AM, Lazeyras F, Mayer E. A network of areas sensitive to the occipito-temporal face beyond the right middle fusiform gyrus is required for normal processing of the face. Brain. 2003 Nov;126(pt 11):2381-95. DOI: 10.1093/brain/awg241. Epub 2003 July 22. PMID: 12876150.
24. Sorger B, Goebel R, Schiltz C, Rossion B. Understanding the functional neuroanatomy of acquired prosopagnosia. Neuroimaging. 2007 Apr 1;35(2):836-52. DOI: 10.1016/j.neuroimage.2006.09.051. Epub 2007 February 14. PMID: 17303440.
25. Voith LA, Logan-Greene P, Strodthoff T, Bender AE. A Paradigm Shift in Batterer Intervention Programming: A Need to Deal with Unresolved Trauma. Trauma Violence Abuse. 2020 Oct;21(4):691-705. DOI: 10.1177/1524838018791268. Epub 2018 July 30. PMID: 30060720.
26. Machisa MT, Christofides N, Jewkes R. Structural pathways between child abuse, poor mental health outcomes, and intimate partner violence (IPV) perpetrated by men. PLoS One. 2016 Mar 17;11(3):e0150986. DOI: 10.1371/journal.pone.0150986. PMID: 26986056; PMCID: PMC4795913.
27. Oyekunle V, Gibbs A, Tomita A. Assessing the role of depression in reducing the perpetration of intimate partner violence among youth living in urban informal settlements using a mediation analysis of the Stepping Stones and Creating Futures intervention. Glob Health Action. 2023 Dec 31;16(1):2188686. DOI: 10.1080/16549716.2023.2188686. PMID: 36927500; PMCID: PMC10026746.
28. Gibbs A, Jewkes R, Willan S, Washington L. Associations between poverty, mental health and substance use, gender power, and intimate partner violence among young women and men (18-30) in urban informal settlements in South Africa: A cross-sectional study and structural equation model. PLoS One. October 3, 2018;13(10):E0204956. DOI: 10.1371/journal.pone.0204956. PMID: 30281677; PMCID: PMC6169941.
29. Mannell J, Minckas N, Burgess R, Chirwa ED, Jewkes R, Gibbs A. Does experiencing a traumatic life event increase the risk of intimate partner violence for young women? A cross-sectional analysis and structural equation model of data from the Stepping Stones and Creating Futures intervention in South Africa. BMJ Open. 2022 Apr 29;12(4):E051969. DOI: 10.1136/bmjopen-2021-051969. PMID: 35487735; PMCID: PMC9058688.
30. Thomson G, Flacking R, George K, Feeley N, Haslund-Thomsen H, De Coen K, Schmied V, Provenzi L, Rowe J. Parents' experiences of emotional closeness with their babies in the neonatal unit: a metaethnography. Early Hum Dev. 2020 Oct;149:105155. DOI: 10.1016/j.earlhumdev.2020.105155. Epub 2020 August 14. PMID: 32829240.
31. Borie AM, Young LJ, Liu RC. Sex-dependent and sex-dependent oxytocin-endocannabinoid interactions in the nucleus accumbens: implications for social behavior. Philos Trans R Soc Lond B Biol Sci. 29 Aug 2022;377(1858):20210057. DOI: 10.1098/RSTB.2021.0057. Epub 2022 July 11. PMID: 35858094; PMCID: PMC9272148.
32. Stevens AL, Herrenkohl TI, Mason WA, Smith GL, Klevens J, Merrick MT. Effects of the development of child domestic adversity, transitions, and relationship

quality on adult outcomes of socioeconomic status: Effects of reasoned child maltreatment. Child Abuse Negl. 2018 May;79:42-50. DOI: 10.1016/j.chiabu.2018.01.031. Epub 2018 February 6. PMID: 29407855; PMCID: PMC6134210.

33. Khoury JE, Bosquet Enlow M, Plamondon A, Lyons-Ruth K. The association between adversity and hair cortisol levels in humans: A meta-analysis. Psychoneuroendocrinology. 2019 May;103:104-117. DOI: 10.1016/j.psyneuen.2019.01.009. Epub 2019 Jan 11. PMID: 30682626; PMCID: PMC6450779.
34. Oh DL, Jerman P, Silvério Marques S, Koita K, Purewal Boparai SK, Burke Harris N, Bucci M. Systematic review of pediatric health outcomes associated with childhood adversity. BMC Pediatrician. 2018 Feb 23;18(1):83. DOI: 10.1186/s12887-018-1037-7. PMID: 29475430; PMCID: PMC5824569.
35. Yee AZ, Lwin MO, Ho SS. The influence of parenting practices on the promotional and preventive behaviors of infant food consumption: a systematic review and meta-analysis. Int J Behav Nutr Phys Act. 2017 Apr 11;14(1):47. DOI:10.1186/s12966-017-0501-3. PMID: 28399881; PMCID: PMC5387370.
36. Jaswetz L, de Voogd LD, Becker ES, Roelofs K. No evidence of disruption of reconsolidation of conditioned threat memories with cognitively demanding intervention. Representative of Science. 22 Apr 2022;12(1):6663. DOI: 10.1038/s41598-022-10184-1. PMID: 35459769; PMCID: PMC9033821.
37. Cirillo A, Diniz E, Gadelha A, Asevedo E, Axelrud LK, Miguel EC, Rohde LA, Bressan RA, Pan P, Mari JJ. Population neuroscience: challenges and opportunities for psychiatric research in low- and middle-income countries. Psychiatry: Braz, J. 2020, Aug;42(4):442-448. DOI: 10.1590/1516-4446-2019-0761. Epub 2020 April 3. PMID: 32267341; PMCID: PMC7430393.
38. Anker JJ, Kushner MG. Alcohol Use Disorder and Anxiety: Connecting Psychiatric, Psychological, and Neurobiological Perspectives. Alcohol Res. December 30, 2019;40(1):arcr.v40.1.03. DOI: 10.35946/arcr.v40.1.03. PMID: 31886106; PMCID: PMC6927748.
39. Ramírez S, Haddad-Tóvolli R, Radosevic M, Toledo M, Pané A, Alcolea D, Ribas V, Milà-Guasch M, Pozo M, Obri A, Eyre E, Gómez-Valadés AG, Chivite I, Van Eeckhout T, Zalachoras I, Altirriba J, Bauder C, Imbernón M, Garrabou G, Garcia-Ruiz C, Nogueiras R, Soto D, Gasull X, Sandi C, Brüning JC, Fortea J, Jiménez A, Fernández-Checa JC, Claret M. Hypothalamic pregnenolone mediates recognition memory in the context of metabolic disorders. Cell Metab. 2022 Feb 1;34(2):269-284.e9. DOI: 10.1016/j.cmet.2021.12.023. PMID: 35108514; PMCID: PMC8815774.
40. Vlachos II, Papageorgiou C, Margariti M. Neurobiological Trajectories Involving Social Isolation in PTSD: A Systematic Review. Brain Sci. March 18, 2020;10(3):173. DOI: 10.3390/brainsci10030173. PMID: 32197333; PMCID: PMC7139956.
41. Ceban F, Ling S, Lui LMW, Lee Y, Gill H, Teopiz KM, Rodrigues NB, Subramaniapillai M, Di Vincenzo JD, Cao B, Lin K, Mansur RB, Ho RC, Rosenblat JD, Miskowiak KW, Vinberg M, Maletic V, McIntyre RS. Fatigue and Cognitive Impairment in Post-COVID-19 Syndrome: A Systematic Review and Meta-Analysis. Immune Brain Behavior. 2022 Mar;101:93-135. DOI: 10.1016/j.bbi.2021.12.020. Epub 2021 December 29. PMID: 34973396; PMCID: PMC8715665.
42. Turkey's Association for Child and Adolescent Psychiatry, 2020). r J Psychol Med. 2020 Sep;37(3):222-225. DOI: 10.1017/ipm.2020.57. Epub 2020 May 21. PMID: 32434621; PMCID: PMC7276500.
43. Geugies H, Mocking RJT, Figueroa CA, Groot PFC, Marsman JC, Servaas MN, Steele JD, Schene AH, Ruhé HG. Impaired reward-related learning signals in non-drug patients with recurrent depression. Brain. 2019 Aug 1;142(8):2510-2522. DOI: 10.1093/brain/awz167. PMID: 31280309; PMCID: PMC6734943.
44. Hornor G. Attachment Disorders. J Pediatr Health Care. 2019 Sep-Oct;33(5):612-622. DOI: 10.1016/j.pedhc.2019.04.017. Errata in: J Pediatr Health Care. 2020 Jan - Feb;34(1):82. PMID: 31447013.
45. Bellis MA, Hughes K, Ford K, Ramos Rodriguez G, Sethi D, Passmore J. Life course health consequences and associated annual costs of adverse childhood experiences across Europe and North America: a systematic review and meta-analysis. Lancet Public Health. 2019 Oct;4(10):e517-e528. DOI: 10.1016/S2468-2667(19)30145-8. Epub 2019 Sep 3. PMID: 31492648; PMCID: PMC7098477.
46. Tough H, Siegrist J, Fekete C. Social relationships, mental health and wellbeing in physical disability: a systematic review. BMC Public Health. 2017 May 8;17(1):414. DOI: 10.1186/s12889-017-4308-6. Erratum in: BMC Public Health. 2017 Jun 16;17 (1):580. PMID: 28482878; PMCID: PMC5422915.
47. Yee AZ, Lwin MO, Ho SS. The influence of parenting practices on the promotional and preventive behaviors of infant food consumption: a systematic review and meta-analysis. Int J Behav Nutr Phys Act. 2017 Apr 11;14(1):47. DOI:10.1186/s12966-017-0501-3. PMID: 28399881; PMCID: PMC5387370.
48. BL Mah. Oxytocin, Postnatal Depression, and Parenting: A Systematic Review. Harv Rev Psychiatry. 2016 Jan-Feb;24(1):1-13. DOI: 10.1097/HRP.0000000000000093. PMID: 26735320.
49. Boyda, D., McFeeters, D., Dhingra, K., Rhoden, L. (2018). Childhood maltreatment and psychotic experiences: Exploring the specificity of early maladaptive schemes. Journal of Clinical Psychology, 74(12), 2287–2301. https://doi.org/10.1002/jclp.22690.
50. Tabbaa M, Paedae B, Liu Y, Wang Z. Regulation of the Neuropeptide of Social Attachment: The Prairie Vole Model. Compr Physiol. 2016 Dec 6;7(1):81-104. DOI: 10.1002/cphy.c150055. PMID: 28135000; PMCID: PMC5308882.
51. Mitchell, I. J., & Beech, A. R. (2011). Towards a neurobiological model of offending. Clinical Psychology

Review, 31(5), 872–882. DOI:10.1016/j.cpr.2011.04.001
52. Caldiroli A, Capuzzi E, Affaticati LM, Surace T, Di Forti CL, Dakanalis A, Clerici M, Buoli M. Candidate biological markers for Social Anxiety Disorder: A Systematic Review. Int J Mol Sci. 2023 Jan 3;24(1):835. DOI: 10.3390/ijms24010835. PMID: 36614278; PMCID: PMC9821596.
53. EA Network, Young LJ. Oxytocin, vasopressin, and pair binding: implications for autism. Philos Trans R Soc Lond B Biol Sci. December 29, 2006;361(1476):2187-98. DOI: 10.1098/RSTB.2006.1939. PMID: 17118932; PMCID: PMC1764849.
54. Borie AM, Agezo S, Lunsford P, Boender AJ, Guo JD, Zhu H, Berman GJ, Young LJ, Liu RC. Social experiment alters oxytocinergic modulation in the nucleus accumbens of female prairie rats. Curr Biol. 2022 Mar 14;32(5):1026-1037.e4. DOI: 10.1016/j.cub.2022.01.014. Epub 2022 February 1. PMID: 35108521; PMCID: PMC8930613.
55. Beery AK, Lopez SA, Blandino KL, Lee NS, Bourdon NS. Social selectivity and social motivation in rats. Elife. 2021 Nov 2;10:e72684. DOI: 10.7554/eLife.72684. PMID: 34726153; PMCID: PMC8594915.
56. Thompson R, Lewis T, Proctor LJ, Isbell P, English DJ, Dubowitz H, Litrownik AJ, Black MM. Childhood abuse, emotional distress and sexual relations in early adolescence: multi-informant perspectives on parental monitoring. J Fam Psychol. 2011 Dec;25(6):885-94. DOI: 10.1037/a0025423. Epub 2011 September 19 PMID: 21928888; PMCID: PMC3874382.
57. Andrzejewski, T., DeLucia, E.A., Semones, O. et al. Adverse childhood experiences in autistic children and their caregivers: examining intergenerational continuity. J Autism Dev Disord (2022). https://doi.org/10.1007/s10803-022-05551-w
58. Oberlander SE, Wang Y, Thompson R, Lewis T, Proctor LJ, Isbell P, English DJ, Dubowitz H, Litrownik AJ, Black MM. Childhood abuse, emotional distress and sexual relations in early adolescence: multi-informant perspectives on parental monitoring. J Fam Psychol. 2011 Dec;25(6):885-94. DOI: 10.1037/a0025423. Epub 2011 September 19 PMID: 21928888; PMCID: PMC3874382.
59. Wong TK, Fung PC, Chua SE, McAlonan GM. Abnormal spatiotemporal processing of emotional facial expressions in childhood autism: dipole source analysis of event-related potentials. Eur J Neurosci. 2008 Jul;28(2):407-16. DOI: 10.1111/j.1460-9568.2008.06328.x. PMID: 18702712.
60. Batty M, Meaux E, Wittemeyer K, Roger B, Taylor MJ. Early processing of emotional faces in children with autism: A potential event-related study. J Exp Child Psychol. 2011 Aug;109(4):430-44. DOI: 10.1016/j.jecp.2011.02.001. Epub 2011 April 1. PMID: 21458825.
61. Akechi H, Senju A, Kikuchi Y, Tojo Y, Osanai H, Hasegawa T. The effect of gaze direction on the processing of facial expressions in children with autism spectrum disorder: an ERP study. Neuropsychology. 2010 Aug;48(10):2841-51. DOI: 10.1016/j.neuropsychologia.2010.05.026. Epub 2010 May 31. PMID: 20546762.
62. EM, Baruth JM, Sears L, Sokhadze GE, El-Baz AS, Casanova MF. Prefrontal neuromodulation using rTMS improves the function of monitoring and error correction in autism. Apply Biofeedback from Psychophysiol. 2012 Jun;37(2):91-102. DOI: 10.1007/s10484-012-9182-5. PMID: 22311204.
63. Akechi H, Senju A, Kikuchi Y, Tojo Y, Osanai H, Hasegawa T. The effect of gaze direction on the processing of facial expressions in children with autism spectrum disorder: an ERP study. Neuropsychology. 2010 Aug;48(10):2841-51. DOI: 10.1016/j.neuropsychologia.2010.05.026. Epub 2010 May 31. PMID: 20546762.
64. Jaworska-Andryszewska P, Rybakowski JK. Childhood trauma in mood disorders: neurobiological mechanisms and implications for treatment. Pharmacol Rep. 2019 Feb;71(1):112-120. DOI: 10.1016/j.pharep.2018.10.004. Epub 2018 October 11. PMID: 30544098.
65. Vlachos II, Papageorgiou C, Margariti M. Neurobiological Trajectories Involving Social Isolation in PTSD: A Systematic Review. Brain Sci. March 18, 2020;10(3):173. DOI: 10.3390/brainsci10030173. PMID: 32197333; PMCID: PMC7139956.
66. Oh DL, Jerman P, Silvério Marques S, Koita K, Purewal Boparai SK, Burke Harris N, Bucci M. Systematic review of pediatric health outcomes associated with childhood adversity. BMC Pediatrician. 2018 Feb 23;18(1):83. DOI: 10.1186/s12887-018-1037-7. PMID: 29475430; PMCID: PMC5824569.
67. Kim YJ, van Rooij SJH, Ely TD, Fani N, Ressler KJ, Jovanovic T, Stevens JS. Association between posttraumatic stress disorder severity and amygdala habituation to fearful stimuli. Depress Anxiety. 2019 Jul;36(7):647-658. DOI: 10.1002/da.22928. PMID: 31260599; PMCID: PMC6943827.
68. Flanagan JC, Sippel LM, Santa Maria MMM, Hartwell KJ, Brady KT, Joseph JE. Impact of oxytocin on the neural correlates of fearful facial processing in PTSD related to childhood trauma. Eur J Psychotraumatol. 2019 May 7;10(1):1606626. DOI: 10.1080/20008198.2019.1606626. PMID: 31105906; PMCID: PMC6508045.
69. Oh DL, Jerman P, Silvério Marques S, Koita K, Purewal Boparai SK, Burke Harris N, Bucci M. Systematic review of pediatric health outcomes associated with childhood adversity. BMC Pediatrician. 2018 Feb 23;18(1):83. DOI: 10.1186/s12887-018-1037-7. PMID: 29475430; PMCID: PMC5824569.
70. Azulay H, Guy N, Pertzov Y, Israel S. Empathy modulates the effect of stress reactivity on generous giving. Neurosci of the Front. 2022 Apr 25;16:814789. DOI: 10.3389/fnins.2022.814789. PMID: 35546889; PMCID: PMC9081844.
71. Tidmarsh LV, Harrison R, Ravindran D, Matthews SL, Finlay KA. The Influence of Adverse Childhood Experiences on Pain Management: Mechanisms, Processes, and Trauma-Informed Care. Frontal Pain Res (Lausanne). 2022 Jun 10;3:923866. DOI: 10.3389/fpain.2022.923866. PMID: 35756908; PMCID: PMC9226323.
72. Gonzalez SL, Reeb-Sutherland BC, Nelson EL. Quantifying Motor Experience in the Infant Brain: EEG

Power, Coherence, and Mu Desynchronization. Front Psychol. 2016 Feb 18;7:216.
DOI: 10.3389/fpsyg.2016.00216. PMID: 26925022; PMCID: PMC4757680.

73. Bellis MA, Hughes K, Ford K, Ramos Rodriguez G, Sethi D, Passmore J. Life course health consequences and associated annual costs of adverse childhood experiences across Europe and North America: a systematic review and meta-analysis. Lancet Public Health. 2019 Oct;4(10):e517-e528. DOI: 10.1016/S2468-2667(19)30145-8. Epub 2019 Sep 3. PMID: 31492648; PMCID: PMC7098477.
74. Hughes K, Ford K, Bellis MA, Glendinning F, Harrison E, Passmore J. Health and financial costs of adverse childhood experiences in 28 European countries: a systematic review and meta-analysis. Lancet Public Health. 2021 Nov;6(11):e848-e857. DOI: 10.1016/S2468-2667(21)00232-2. PMID: 34756168; PMCID: PMC8573710.
75. Houtepen LC, Heron J, Suderman MJ, Fraser A, Chittleborough CR, Howe LD. Associations of adverse childhood experiences with educational attainment and adolescent health and the role of family and socioeconomic factors: A prospective cohort study in the UK. PLoS Med. 2020 Mar 2;17(3):e1003031. DOI: 10.1371/journal.pmed.1003031. PMID: 32119668; PMCID: PMC7051040.
76. Reichman NE, Corman H, Noonan K, Jimenez ME. Infant Health and Future Childhood Adversity. Matern Child Health J. 2018 Mar;22(3):318-326.
DOI: 10.1007/s10995-017-2418-5. PMID: 29285632.
77. Brown NM, Brown SN, Briggs RD, Germán M, Belamarich PF, Oyeku SO. Associations Between Adverse Childhood Experiences and ADHD Diagnosis and Severity. Acad Pediatr. 2017 May-Jun;17(4):349-355.
DOI: 10.1016/j.acap.2016.08.013. PMID: 28477799.
78. Adaralegbe NJ, Okobi OE, Omar ZT O, Segun E, Evbayekha EO, Abolurin A, Egberuare EO, Ezegbe HC, Adegbosin A, Adedeji AG, Angaye EG, Izundu IC, Oyelade BO. Impact of adverse childhood experiences on resilience and Cureus. 2022 Nov 26;14(11):e31907. DOI:10.7759/cureus.31907. PMID: 36579285;
PMCID: PMC9792122.
79. Kim YK, Ham BJ, Han KM. Interactive effects of genetic polymorphisms and childhood adversities on brain morphological changes in depression. Prog Neuropsychopharmacol Biol Psychiatry. 2019 Apr 20;91:4-13. DOI: 10.1016/j.pnpbp.2018.03.009. Epub 2018 March 10. PMID: 29535036.
80. Kim JM, Stewart R, Kim SW, Yang SJ, Shin IS, Kim YH, Yoon JS. Interactions between life stressors and susceptibility genes (5-HTTLPR and BDNF) in depression in Korean elderly. Biological Psychiatry. 2007 Sep 1;62(5):423-8. DOI: 10.1016/j.biopsych.2006.11.020. Epub 2007 May 4. PMID: 17482146.
81. Lucas T, Wegner R, Pierce J, Lumley MA, Laurent HK, Granger DA. Perceived Discrimination, Racial Identity, and Multisystem Stress Response to Social Evaluative Threat Among African American Men and Women. Psychosom, Med. 2017, Apr;79(3):293-305.
DOI:10.1097/PSY.0000000000000406. PMID: 27806018; PMCID: PMC5374002.
82. Laurent HK, Lucas T, Pierce J, Goetz S, Granger DA. The coordination of cortisol response to social evaluative threat with autonomic and inflammatory responses is moderated by stress and affect assessments. Biol Psychol. 2016 Jul;118:17-24. DOI: 10.1016/j.biopsycho.2016.04.066. Epub 2016 May 4. PMID: 27155141;
PMCID: PMC4956552.
83. Bendezú JJ, Calhoun CD, Vinograd M, Patterson MW, Rudolph KD, Giletta M, Hastings P, Nock MK, Slavich GM, Prinstein MJ. Exploring joint HPA inflammatory stress response profiles in adolescent girls: Implications for developmental models of neuroendocrine disruption. Psychobiol Dev. 84. 2022 Mar;64(3):e22247.
DOI: 10.1002/dev.22247. PMID: 35312047;
PMCID: PMC8944282.
84. Kaufman J, Yang BZ, Douglas-Palumberi H, Grasso D, Lipschitz D, Houshyar S, Krystal JH, Gelernter J. Brain-derived neurotrophic factor-5-HTTLPR gene interactions and environmental modifiers of depression in children. Biological Psychiatry. April 15, 2006;59(8):673-80. DOI: 10.1016/j.biopsych.2005.10.026. Epub 2006 February 3. PMID: 16458264.
85. Parel ST, Peña CJ. Genome-wide signatures of stress in early life: influence of sex. Biological Psychiatry. 2022 Jan 1;91(1):36-42. DOI: 10.1016/j.biopsych.2020.12.010. Epub 2020 December 19. PMID: 33602500;
PMCID: PMC8791071.
86. Holmes L Jr, Shutman E, Chinaka C, Deepika K, Pelaez L, Dabney KW. Aberrant Epigenomic Modulation of the Glucocorticoid Receptor Gene (NR3C1) in Early Life Stress and Correlation of Major Depressive Disorder: Systematic Review and Synthesis of Quantitative Evidence. Int J Environ Res Public Health. 2019 Nov 4;16(21):4280. DOI: 10.3390/ijerph16214280.
PMID: 31689998; PMCID: PMC6861987.
87. Rahman MF, McGowan PO. Cell-type-specific epigenetic effects of early life stress on the brain. Transl Psychiatry. 2022 Aug 10;12(1):326. DOI: 10.1038/s41398-022-02076-9. PMID: 35948532; PMCID: PMC9365848.
88. Rahman MF, McGowan PO. Cell-type-specific epigenetic effects of early life stress on the brain. Transl Psychiatry. 2022 Aug 10;12(1):326. DOI: 10.1038/s41398-022-02076-9. PMID: 35948532; PMCID: PMC9365848.
89. Chuang HH, Huang CG, Chuang LP, Huang YS, Chen NH, Li HY, Fang TJ, Hsu JF, Lai HC, Chen JY, Lee LA. Relationships between and predictive values of obesity, markers of inflammation, and disease severity in pediatric patients with obstructive sleep apnea before and after adenotonsillectomy. J Clin Med. 2020 Feb. 20;9(2):579. DOI: 10.3390/jcm9020579. PMID: 32093397;
PMCID: PMC7073666.
90. Giotakos O. Neurobiology of emotional trauma. Psychiatriki. 2020 Apr-Jun;31(2):162-171.
DOI: 10.22365/jpsych.2020.312.162. PMID: 32840220.

91. Hakamata Y, Suzuki Y, Kobashikawa H, Hori H. Neurobiology of early life adversity: A systematic review of meta-analyses towards an integrative account of its neurobiological trajectories to mental disorders. Neuroendocrinol Front. 2022 Apr;65:100994. DOI: 10.1016/j.yfrne.2022.100994. Epub 2022 Mar 21. PMID: 35331780.
92. Blair C, Raver CC. Child development in the context of adversity: experiential canalization of brain and behavior. Am Psychol. 2012 May-Jun;67(4):309-18. DOI: 10.1037/a0027493. Epub 2012 Mar 5. PMID: 22390355; PMCID: PMC5264526.
93. Miguel PM, Pereira LO, Silveira PP, Meaney MJ. Early environmental influences on the development of children's brain structure and function. Dev Med Child Neurol. 2019 Oct;61(10):1127-1133. DOI: 10.1111/DMCN.14182. Epub 2019
94. Ramírez S, Haddad-Tóvolli R, Radosevic M, Toledo M, Pané A, Alcolea D, Ribas V, Milà-Guasch M, Pozo M, Obri A, Eyre E, Gómez-Valadés AG, Chivite I, Van Eeckhout T, Zalachoras I, Altirriba J, Bauder C, Imbernón M, Garrabou G, Garcia-Ruiz C, Nogueiras R, Soto D, Gasull X, Sandi C, Brüning JC, Fortea J, Jiménez A, Fernández-Checa JC, Claret M. Hypothalamic pregnenolone mediates recognition memory in the context of metabolic disorders. Cell Metab. 2022 Feb 1;34(2):269-284.e9. DOI: 10.1016/j.cmet.2021.12.023. PMID: 35108514; PMCID: PMC8815774.
95. Blair C, Raver CC. Child development in the context of adversity: experiential canalization of brain and behavior. Am Psychol. 2012 May-Jun;67(4):309-18. DOI: 10.1037/a0027493. Epub 2012 Mar 5. PMID: 22390355; PMCID: PMC5264526.
96. Miguel PM, Pereira LO, Silveira PP, Meaney MJ. Early environmental influences on the development of children's brain structure and function. Dev Med Child Neurol. 2019 Oct;61(10):1127-1133. DOI: 10.1111/DMCN.14182. Epub 2019
97. Ruggieri V. El autismo a lo largo de la vida [Lifelong autism]. Medicine (B Aires). Aug. 30, 2022;82 Supplement 3:2-6. Spanish. PMID: 36054849.
98. Sokhadze E, Baruth J, Tasman A, Sears L, Mathai G, El-Baz A, Casanova MF. Potential study related to novelty processing abnormalities events in autism. Apply Biofeedback from Psychophysiol. 2009 Mar;34(1):37-51. DOI: 10.1007/s10484-009-9074-5. Epub 2009 February 6 PMID: 19199028.
99. Senju A, Tojo Y, Yaguchi K, Hasegawa T. Deviant gaze processing in children with autism: an ERP study. Neuropsychology. 2005;43(9):1297-306. DOI: 10.1016/j.neuropsychologia.2004.12.002. Epub 2005 January 24. PMID: 15949514.
100. Akechi H, Senju A, Kikuchi Y, Tojo Y, Osanai H, Hasegawa T. Does gaze direction modulate facial expression processing in children with autism spectrum disorder? Child development. 2009 Jul-Aug;80(4):1134-46. DOI: 10.1111/j.1467-8624.2009.01321.x. PMID: 19630898.
101. Senju A, Kikuchi Y, Hasegawa T, Tojo Y, Osanai H. Is anyone looking at me? Direct eye detection in children with and without autism. Brain Cogn. 2008 Jul;67(2):127-39. DOI: 10.1016/j.bandc.2007.12.001. Epub 2008 January 28 PMID: 18226847.
102. McDonald JJ, Green JJ. Isolating potential components related to events associated with voluntary control of visuospatial attention. Brain Res. 2008 Aug 28;1227:96-109. DOI: 10.1016/j.brainres.2008.06.034. Epub 2008 June 20. PMID: 18621037.
103. Sokhadze EM, El-Baz AS, Sears LL, Opris I, Casanova MF. rTMS neuromodulation improves electrocortical functional measures of information processing and behavioral responses in autism. Neurosci Frontal System. Aug. 6, 2014;8:134. DOI: 10.3389/fnsys.2014.00134. PMID: 25147508; PMCID: PMC4123734.
104. Choenni V, Hammink A, van de Mheen D. Association Between Substance Use and the Perpetration of Family Violence in Industrialized Countries: A Systematic Review. Trauma, Violence, Abuse. 2017 Jan;18(1):37-50. DOI: 10.1177/1524838015589253. Epub 2015 Aug 20. PMID: 26296740.
105. Madruga CS, Viana MC, Abdalla RR, Caetano R, Laranjeira R. Pathways from witnessing parental violence during childhood to involvement in intimate partner violence in adult life: The roles of depression and substance use. Drug Alcohol Rev. 2017 Jan;36(1):107-114. DOI: 10.1111/dar.12514. PMID: 28134495.
106. Claypool N, Moore de Peralta A. The Influence of Adverse Childhood Experiences (ACEs), Including the COVID-19 Pandemic and Toxic Stress on the Development and Health Outcomes of Latinx Children in the U.S.: A Literature Review. Int J Child Maltreat. 2021;4(3):257-278. DOI: 10.1007/s42448-021-00080-y. Epub 2021 July 11. PMID: 34278229; PMCID: PMC8272684.
107. Klengel T, Mehta D, Anacker C, Rex-Haffner M, Pruessner JC, Pariante CM, Pace TW, Mercer KB, Mayberg HS, Bradley B, Nemeroff CB, Holsboer F, Heim CM, Ressler KJ, Rein T, Binder EB. Allelé-specific FKBP5 DNA demethylation mediates gene-childhood trauma interactions. Nat Neurosci. 2013 Jan;16(1):33-41. DOI: 10.1038/nn.3275. Epub 2012 December 2. PMID: 23201972; PMCID: PMC4136922.
108. Choudhury S, Yeh PG, Markham CM. Dealing with adverse childhood experiences during the COVID-19 pandemic: Perceptions of mental health care providers. Frontal Psychotic. 2022 Sep 8;13:975300. DOI: 10.3389/fpsyg.2022.975300. PMID: 36160597; PMCID: PMC9493451.
109. Mannell J, Minckas N, Burgess R, Chirwa ED, Jewkes R, Gibbs A. Does experiencing a traumatic life event increase the risk of intimate partner violence for young women? A cross-sectional analysis and structural equation model of data from the Stepping Stones and Creating Futures intervention in South Africa. BMJ Open. 2022 Apr 29;12(4):E051969. DOI: 10.1136/bmjopen-2021-051969. PMID: 35487735; PMCID: PMC9058688
110. Vlachos II, Papageorgiou C, Margariti M. Neurobiological

Trajectories Involving Social Isolation in PTSD: A Systematic Review. Brain Sci. March 18, 2020;10(3):173. DOI: 10.3390/brainsci10030173. PMID: 32197333; PMCID: PMC7139956.

111. Seitz, Benjamin M et al. "Higher-Order Conditioning and Dopamine: Charting a Way Forward." Frontiers in Behavioral Neuroscience vol. 15 745388. October 4, 2021, DOI:10.3389/fnbeh.2021.745388
112. Jung H, Herrenkohl TI, Skinner ML, Lee JO, Klika JB, Rousson AN. Gender Differences in Intimate Partner Violence: A Predictive Analysis of IPV for Child Abuse and Exposure to Domestic Violence During Early Childhood. Violence Against Women. 2019 Jun;25(8):903-924. DOI: 10.1177/1077801218796329. Epub 2018 August 30. PMID: 30160199; PMCID: PMC6395526.
113. Gainotti G. Confront feelings of familiarity, the right temporal lobe, and the possible underlying neural mechanisms. Brain Res Rev. 2007 Nov;56(1):214-35. DOI: 10.1016/j.brainresrev.2007.07.009. Epub 2007 August 3 PMID: 17822771.
114. Bridgemohan C, Cochran DM, Howe YJ, Pawlowski K, Zimmerman AW, Anderson GM, Choueiri R, Sices L, Miller KJ, Ultmann M, Helt J, Forbes PW, Farfel L, Brewster SJ, Frazier JA, Neumeyer AM. Investigating Potential Biomarkers in Autism Spectrum Disorder. Front Integr Neurosci. 2019 Aug 2;1:31 PM. DOI: 10.3389/fnint.2019.00031. PMID: 31427932; PMCID: PMC6687766.
115. Takahashi A, Miczek KA. Neurogenetics of aggressive behavior: studies in rodents. Curr Top Behav Neurosci. 2014;17:3-44. DOI: 10.1007/7854_2013_263. PMID: 24318936; PMCID: PMC4092042.
116. Rawat RS, Bhambri A, Pal M, Roy A, Jain S, Pillai B, Konar A. Stressful experiences in early life increase aggressive behavior in adulthood through changes in transthyretin expression and function. Elife. 2022 Oct 13;11:e77968. DOI:10.7554/eLife.77968. PMID: 36226913; PMCID: PMC9633068.
117. Blum, Kenneth et al. "Reward Deficiency Syndrome (RDS) Surprisingly Is Evolutionary and Found Everywhere: Is It "Blowin' in the Wind"?." Journal of personalized medicine vol. 12.2 321. 21 Feb. 2022, DOI:10.3390/jpm12020321
118. Blum K, Bowirrat A, Braverman ER, Baron D, Cadet JL, Kazmi S, Elman I, Thanos PK, Badgaiyan RD, Downs WB, Bagchi D, Llanos-Gomez L, Gold MS. Reward Deficiency Syndrome (RDS): A Cytoarchitectural Common Neurobiological Trait of All Addictions. Int J Environ Res Public Health. 2021 Nov 2;18(21):11529. DOI: 10.3390/ijerph182111529. PMID: 34770047; PMCID: PMC8582845.
119. McLaughlin T, Blum K, Oscar-Berman M, Febo M, Demetrovics Z, Agan G, Fratantonio J, Gold MS. Using the Neuroadaptagen KB200z™ to Ameliorate Terrifying, Lucid Nightmares in RDS Patients: the Role of Enhanced, Brain-Reward, Functional Connectivity and Dopaminergic Homeostasis. J Reward Defic Syndr. 2015;1(1):24-35. DOI: 10.17756/jrds.2015-006. PMID: 26065033; PMCID: PMC4459746.
120. Pardo-Moreno T, Mohamed-Mohamed H, Rivas-Dominguez A, Garcia-Morales V, Garcia-Lara RA, Suleiman-Martos S, Bermudez-Pulgarin B, Ramos-Rodriguez JJ. Poor conservation of cognitive agility in obese and aging people. Biomedicines. 2023 Jan 5;11(1):138. DOI: 10.3390/biomedicines11010138. PMID: 36672646; PMCID: PMC9855664.
121. Chiou R, Humphreys GF, Jung J, Ralph Lambon MA. Controlled semantic cognition relies on dynamic and flexible interactions between the executive systems of hub-and-spoke 'semantic control' and 'semantic representation'. Cortex. 2018 Jun;103:100-116. DOI: 10.1016/j.cortex.2018.02.018. Epub 2018 March 9 PMID: 29604611; PMCID: PMC6006425.
122. Crotti M, Rudd J, Roberts S, Fitton Davies K, O'Callaghan L, Utesch T, Foweather L. Physical activity promoting teaching practices and children's physical activity within physical education classes supported by motor learning theory (SAMPLE-PE). PLoS One. August 1, 2022;17(8):e0272339. DOI: 10.1371/journal.pone.0272339. PMID: 35913904; PMCID: PMC9342796.
123. Bonawitz E, Shafto P, Gweon H, Goodman ND, Spelke E, Schulz L. The double-edged sword of pedagogy: instruction limits spontaneous exploration and discovery. Cognition. 2011 Sep;120(3):322-30. DOI: 10.1016/j.cognition.2010.10.001. Epub 2011 January 8 PMID: 21216395; PMCID: PMC3369499.