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 oxytonergic 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 evidencing their own 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, playfull, 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 oxytonergic 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].

Oxytoneric 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 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. Polymorphisms 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 connectivity 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 (Nae), 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 regions most 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 and continuous 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 endocannabinoids 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].

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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, exerts 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 are associated with similar observations. The BDNF gene is a positive regulator of BDNF action on GABAA receptors and regulates emotional behavior, exerting anxiolytic, antidepressant, and sedative effects [72-87].

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 different 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...
The purpose of the CSC is to cluster schemes (YOUNG), maladaptive behaviors, relieving, which presence of states of neuro adaptations of fear, or family stressful events in childhood are correlated, in the Family Alienation Generational Family Disease/ANAAS Syndrome/Chronic polymorphisms that amplify suffering [34-80].

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. Patients 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 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 this can 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 accurately get right [23-56].

The finding of Syndrome Z (initially the empirically 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, adrenoids 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 dysfunctions 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 location and 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) the development of basic movement skills, necessary for healthy living (catching, jumping, swimming, cycling) and helps children 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 of initial 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.

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