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SYSTEMS MEDICINE: DIAGNOSTIC MODEL OF PSYCHOPATHOLOGY AND CLINICAL MEDICINE

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ABSTRACT

Introduction: Systems medicine is an approach to understanding disease through the integration of large patient data sets, integrating data with clinical measurements, which have the potential to impact our understanding of the molecular basis and management of a disease. ***Objective:*** To present a new model of Systems Medicine, which is based on neuroscience and human neurobiology, which presents the robustness of empiricism, and which addresses groups of diagnoses of chronic comorbidities, which are linked to neuropsychological symptoms, such as stress, psychosocial factors and family conflicts in an objective manner. ***Methods:*** The clinical neuroscience of family interactions and childhood adversities develops a three-level neuromaladaptive connectome through common biobehaviors, which represents a structure delimited in personality traits, which were identified in empirical observation and constructed a new model. ***Results:*** A clinical synthesis associated with organic pathophysiology, we organized a diagnostic systems medicine model, with grouping of individuals that addresses objective neuropsychological alterations associated with changes in chronic comorbidities of clinical medicine. ***Discussion:*** This method organizes the clinical approach to the family factor, which is neurodysfunctional and represents a promising tool, but is in its infancy of theoretical basis, which requires experimental evaluation. ***Conclusion:*** The model is a new instrument for new scientific research and assists in the scientific advancement of family medicine, mental health policies, and the creation of mental health care plans in primary care, which is currently lacking.

Keyword: Mental health, diagnosis, systems medicine, neuroscience

1.Introduction

According to the World Health Organization (WHO), mental health (MH) systems have not yet responded effectively to the burden of mental disorders (MD), which significantly influence social conflicts and several chronic diseases such as obesity, systemic arterial hypertension (SAH), and cardiovascular diseases (CVD).[1]

Also according to the WHO, the availability and need for treatments in this area are precarious, since in low- and middle-income countries, people with MD who do not receive treatment are around 76% to 85%, while in high-income countries, the situation is between 35% and 50%.[1]

In the field of science, it is often reported that diversity increases excellence and innovation due to the variety of thoughts and concepts and the breadth and scope of research questions and discoveries, which arise from lived experiences.[2] The phronetic scientific approach has been highlighted in several public health care (HC) plans, as it contains content from various effective practical and theoretical knowledge, which prioritize functionality.[3]

The HC also involves medical combinations that aim at clinical functionality, which are aware of the professional reality and the real needs of health system users.[4] The Systems medicine is an approach to understanding disease through the integration of large patient data sets, which integrate data with clinical measurements, which have the potential to impact our understanding of the molecular basis and management of a disease.[5]

Currently, there is increasing and voluminous accumulated evidence of the association of chronic comorbidities in relation to exposure to Adverse Childhood Emotions (ACE). However, with the advancement of positron emission tomography (PET) magnetic resonance imaging studies, it is possible to visualize a central and objective structure.[6]

The psychodynamic factors of the family environment, associated with genetic factors and their neurobiological substrates, are responsible for the formation of a personality microstructure, composed of the integration of biobehaviors, albeit in a maladaptive way, which precedes traditional educational and socioeconomic factors and directly contributes to the development of some psychopathologies such as addictive disorders and personality disorders. [7-12]

2. Objective

To present a new systems medicine model, which is based on neuroscience and human neurobiology, which presents the robustness of empiricism, and which addresses groups of diagnoses of chronic comorbidities, which are linked to specific mental health symptoms, such as stress, psychosocial factors, and family conflicts in an objective manner. Thus, it allows for new measures and organization of mental health care, in primary care and family medicine, in addition to new approaches to population research.

3. Methods

A comprehensive literature review was conducted in the PubMed and Web of Science digital libraries, using the terms neurobiology, adverse childhood emotions, reward dysfunctional syndrome (RDS), and stress. Only functional neuroimaging articles that presented clinical data based on neurobiological mechanisms of maladaptive processes, which, however, corroborated empirical practice, were selected.

4. Results

Clinical neuroscience of family interactions and childhood adversities develops a three-level neuromaladaptive connectome through common biobehaviors, which represents a

delimited structure of personality traits. [13-30] The first level represents individual characteristics, where adverse (toxin exposure, physical abuse) and protective exposures are assessed from the prenatal period onwards, along with individual moderators such as subjective experience and protective metacognitive processes, perceived predictability, and emotional intelligence.[31-43]

The second level represents family and peer characteristics, where beneficial and risk-associated influences are captured (type and frequency of social contacts, family environment, bullying events). [44-50]

The third level represents characteristics of extra-home environments and violent crimes, such as socioeconomic status, social organization, and criminality. [44-50]

The model begins with the formation of the first group of the system, which is essentially composed of clinical alterations of objective neuropsychological factors, which are attributed to common neuromaladaptive elements that develop through social interactions among family members early in life.

Patient selection is performed through the Assessment of Neuromarkers of Neuromaladaptive Inference, which is systematized and presents an identification methodology focused on clinical neuromarkers, which are derived from the set of eight common neurodysfunctions (ONCs).

For this grouping of clinical symptoms of mental health, we use the letter Z. After the initial grouping, we will select the second group of comorbidities that present a pathophysiological and clinical causal link and significantly influence group Z, such as stress, for example.

For comorbidities of metabolic mechanisms, the letter X was used. Thus, a system of patients ZX was formed. For comorbidities of immunological and inflammatory mechanisms, the letter S, which forms the ZS system. For psychiatric comorbidities that present a clinical and neurobiological connection with group Z, we use the letter Y.

4.1.1 Group Z

The set of neuromaladaptive connectomics is delimited by biobehaviors dependent on family interactions: (1) family synchrony deficit, (2) schematic behaviors, (3) neuromirroring, (4) dysfunctional neuropsychodynamics, (5) dopamine and reward system dysfunctions, (6) emotional dysregulation, (7) inhibitory control deficits, and (8) social and family skills deficits. They present influences of genetic components (epigenetics, gene expression, polymorphisms, and absence or presence of variant genes) and hormonal dysfunctions (cortisol, oxytocin, vasopressin, and melatonin dysfunctions) and immunoinflammatory disorders.[51-70]

1) Deficit/absence of peripheral oxytocin and dopamine neurons that are responsible for synchrony, depth attention, and family social skills.

2) Positive feedback dysfunction of the primitive emotional system of the dopaminergic SEEKING system. It develops in the presence of childhood adversity and expresses chronic and oscillatory hypodopaminergic states.

3) Reward system dysfunction (RDS)

4) Navigation mode in the family environment, with predominance of activity in the hippocampus and amygdala system, due to the formation of engrams of increased interneuronal connectivity strength of these systems of the amygdala and limbic systems with predominance of their activity with the

hippocampus, forming an exclusive navigation mode in the family environment. Chronic aberrant and sudden amygdala inputs occur, generating maladaptive states expressed as family schemas (Young) that produce neurodysfunctional interpersonal relationships (NIRs) such as misconduct, neglect, and psychological abuse among family members. The navigation mode with predominance of amygdala systems and automatic behaviors generates neurocognitive states with Alexithymia: the inability to effectively self-observe (self-identify) emotional and affective awareness at the same time, and Secondary Anosognosia: the inability to effectively identify the emotional and affective state of another person.

5) Commissurectomy (interhemispheric disconnection) of the ungual and arcuate fascicles, secondary to toxic parental behaviors, mainly verbalizing, which causes secondary simultanagnosia (inability to identify more than 2 objects at the same time), causes cognitive inflexibility, decreased processing speed, and limits the extent of consciousness with dense intelligence.

6) Neuromirroring dysfunction of mirror neuron networks due to inversion or increase or decrease in the functional roles of the family system. The inversion of a mode has an important cumulative effect on the SEEKING system and the RDS. Replication of the experienced effect occurs in an increased, decreased, or inverted form of these functional modes of neuropsychodynamics and involves the entire attachment circuitry with neuromirroring.

7) Neurological and enzymatic deficit in emotional regulation: chronic and acute stress, behavioral deviation.

8) Neuroenzymatic deficit in inhibitory control: impulsivity, behavioral deviation.

9) Neurological deficit in social skills (extradomiciliary): social isolation, preferences for habits without social contact, introversion, shyness.

10) Neuronal deficit in family skills: absence of mentalism and parental incapacity.

11) Underdevelopment of the PFC: The PFC is 'hijacked' by the subcortical structures of the midbrain. It occurs due to a deficit in myelination secondary to stress. Myelination, which is involved in regulating processing speed, especially in pre-development periods. In practice, it is the presence of family schemes, which function as hidden triggers, and chronic stress in the home environment that induce reward behaviors, exacerbating dysfunctional mechanisms of immunological, inflammatory, and hormonal orders.

4.1.2 Family Synchrony Bio-Behavior

Family synchrony bio-behavior develops through the stimulation of maternal touch and through other physical senses between biological parents and the newborn (NB), which is involved in interpersonal emotional and affective behaviors. These dyadic processes observed in early childhood contribute to the development of self-regulation and general socio-emotional outcomes in children.[71-89]

Family Synchrony Biobehavior is an important proximal component because when deficient, it contributes to the development of many maladaptive responses early in life, such as the formation of alleviating behaviors (amygdala) or Family Schemas (FSs). [71-89]

The peripheral oxytocin and dopamine neurons (NODP) are responsible for the mechanisms of effective or deep attention, which are essential for identifying the emotional and affective

state of a family member, which is currently called mentalism.[71-89]

4.1.3 Reward System Dysfunction Syndrome (RDS)

The brain's dopaminergic and opioidergic reward pathways are critical for survival because they provide the pleasure drives to eat, love, and reproduce; they have been called "natural rewards" and involve the release of dopamine in the nucleus accumbens and frontal lobes.[90-95]

Deficiencies in reward neurotransmission (dopamine) interfere with the pleasure derived from powerful human physiological drives that serve the function of satisfying or relieving.[90-95]

The mesolimbic pathway, the "reward pathway," is a dopaminergic pathway in the brain. This pathway connects the ventral tegmental area (VTA) in the midbrain to the ventral striatum (nucleus accumbens (NAc) and olfactory tubercle) of the basal ganglia in the forebrain.[90-95]

In 1996, Blum et al. described Reward Deficiency Syndrome (RDS), which encompasses a wide range of addictive, compulsive, and impulsive behaviors, such as obesity. RDS refers to the breakdown of reward neurotransmission and destructive behaviors initiated by a combination of environmental (epigenetic) influences and DNA-based neurotransmission deficits that interfere with the usual fulfillment of human physiological drives (food, water, sex).[90-95]

RDS is a polygenic trait with implications suggesting impaired crosstalk between different neurological systems, including the known reward pathway, neuroendocrine systems, and motivational systems.[90-95]

Objectively associated with it are substance use disorder (SUD), major depressive disorder, early life stress, immune dysregulation, attention deficit hyperactivity disorder (ADHD), posttraumatic stress disorder (PTSD), compulsive gambling, and compulsive eating disorders, which are subtypes of overlapping and interrelated neurochemical dysfunctions.[90-95]

These disorders recruit underlying mechanisms of reward deficiency in multiple brain centers. This variety of associated and overlapping behavioral manifestations has hypodopaminergia in common, and the basic endophenotype recognized as RDS is likened to a behavioral octopus. [90-95]

The chronic hypodopaminergic state, in the vast majority of situations, is associated with RDS, with behaviors and habits that produce dopamine to alleviate displeasure, irritability, boredom, duality, and indecision due to dopamine deficit.[90-95] Evidence from human studies and fMRI supports the hypothesis that multiple brain circuits are disrupted in obesity and SUD and implicates the involvement of reward circuits, modulated by DA, in pathological eating behaviors.[90-95] An essential feature of RDS is the lack of integration between cognition, perception, and emotions that occurs due to substantial dopaminergic increases in the reward, motivation, and learning centers that lead to neuroplasticity in the striato-thalamic-frontal cortical loop, with subsequent top-down dissociation of subcortical activity. Associated with the hypofunctionality of the excitatory glutamatergic afferents of the amygdala-hippocampal complex, there is a failure to produce bottom-up restriction of the striato-thalamic-frontal cortical loop. [90-95]

4.1.4 Family Schemas

In the human brain, a basic pattern built from past experiences occurs

through learning or conditioning of adaptation, which are called schemas.88-93 In primary interpersonal relationships with biological parents, there is a deficit of affective attention, affective and emotional neglect, and situations of adverse emotions in childhood (AEC), which can be cumulative to traumatic situations, such as post-traumatic stress disorder (PTSD); the child's brain develops maladaptive neurocognitive and behavioral states, which in clinical practice are identified through family schemas, described by Jeffrey Young.[96-107]

Family schemas (FSs) develop due to maladaptive reasons; therefore, they are dysfunctional, and currently eighteen types of FSs are described. An individual can present activation of more than one schema at the same time and can be activated twenty-four hours a day.[96-107]

EFs are automatic, sudden, and function as non-self-identified or non-perceptible (unconscious) mechanisms that affect behaviors, cognitive states (distorted and biased perceptions and beliefs), and physiological states such as hormonal dysfunctions and neurodysfunctions of emotions, which begin in children and persist into adulthood.[96-107] EFs occur unconsciously between peers in the family environment (bidirectional), through activation of the amygdala neurological systems and also through a process of neuromirroring (unconscious neuroactivation) of "mirror neurons.´´[96-107]

Typical EFs include avoidance, denial, aversion, escape, punishment, ableism, prejudice, devaluation, and distortion of beliefs, which result in toxic relationships, chronic family fights, and cases of dysfunctional parenting. [96-107]

1) Disconnection/Rejection: inability to form secure bonds, with experiences of negative social experiences. In general, people tend to present characteristics of instability, abuse, coldness, rejection, or isolation from the outside world. Five schemes are linked to this domain: Abandonment, Distrust, Emotional Deprivation, Defectiveness/Shame, and Social Isolation. [96-107]

2) Impaired Autonomy and Performance: Presents dysfunctional expectations about themselves and the world, which interfere with their ability to differentiate themselves from paternal or maternal figures and function independently. The family of origin is overprotective and cannot stimulate them to perform competently. The schemes in this domain are Dependence/Incompetence, Vulnerability to harm or illness, Enmeshment, and Failure. [96-107]

3) Impaired Limits: There is no development of internal limits and responsibility towards others. People may be selfish and spoiled, and most often they grew up in permissive families. The schemas associated with this domain are grandiosity/arrogance and insufficient self-control/self-discipline.[96-107]

4) Other-Orientation: excessive emphasis is placed on meeting the needs of others rather than one's own. The emotional needs and desires of the parents are valued more than the needs and feelings of the child. The EFs developed are subjugation, self-sacrifice, and seeking approval/recognition. [96-107]

5) Overvigilance and Inhibition: excessive emphasis on suppressing feelings, impulses, and spontaneous personal choices, reinforcing compliance with rigid internalized rules regarding one's own performance (perfectionism and self-demand). The family is severe, demanding, and punitive. [96-107]

The EFs associated with this domain are negativism/pessimism, emotional inhibition, inflexible standards, and posture.[96-107]

According to Mason, Platts, and

Tyson, EFs have a self-perpetuating character with resistance to change. They end up constituting the core of the self-concept and the worldview of the individual.[96-107]

4.1.5 Dysfunctional Neuropsychodynamics

The human brain, since the primate era, has been programmed to develop the functional elaboration of family roles during the first window of neurodevelopment (the first 3 years) according to the explicit and implicit reality of the parents, due to cortical, frontal, inferior, and left mirror neurons (Broca's area) and the arcuate fasciculus (Korsakoff's area).[108-114]

Through 40 years of observational studies, attachment research has codified profiles related to parent-child interactions in the form of disorganized, controlled, submissive, and authoritarian attachment behavior.[108-114]

The assessment of attachment relationships has now progressed well beyond infancy so that changes in the quality of attachment can be tracked from infancy to early adulthood.[108-114]

The expectation of the child's brain is that the father will be dominant, with real control of limits, and the mother will provide nurturing.[108-114]

Newborn boys' brains block out pain (repression), disaffection, absences, and traumas from their mothers, and girls' brains do the same in relation to their fathers. The father's role, in particular, is to provide order within the group, teach limits, validate tasks, introduce new environments and novelties, explore, and provide a functional survival experience. [108-114]

When there is intense or repetitive trauma or pain, an unconscious brain mechanism of fear causes an unconscious inversion of the functional role due to the neurobiology of attachment formation.[108-114]

The presence of attachment figures can block the activation of stress physiology, even when the infant is expressing strong behavioral distress, and this mechanism is known as parental social buffering.[108-114]

The neural mechanism for this inhibition of fear learning is the blocking of maternal social buffering of amygdala plasticity. [108-114]

4.1.6 Neuromirroring

A neural network highly relevant to self-other distinction is the Mirror Neuron System (MNS), which was first identified in primates.[115-131] The MNS includes the inferior frontal gyrus, the superior parietal lobe, and the inferior parietal lobe.[115-131]

This network develops very early in infancy and may be automatically activated before the second network, the mentalizing system that includes the medial prefrontal cortex, the precuneus, and the right temporal parietal junction (rTPJ).[115-131] The mentalizing network supports more conscious reasoning about mental states. [115-131]

The rTPJ is of particular importance in self-other distinction, given its role in multisensory integration and activation during tasks in which different beliefs about self and other are salient, allowing switching between their related representations. [115-131] Different lines of evidence support the claim that the mirror mechanism may contribute to the understanding of the actions of others, facilitating the identification of the outcome towards which these actions are directed.[115-131]

It is involved in the activation of family schemas among peers, associated with amygdala activation.[115-131]

4.1.7 Assessment of Neuromarkers of Neuromaladaptive Inference

Identifying acute events or exacerbations of chronic diseases triggered by chronic stress, acute stress, or social conflicts derived from family patterns are the main clinical requirements for selection into group Z.[132-140]

Signs and symptoms of chronic hypodopaminergia:

- Constant duality
- Constant insecurity
- Boredom
- Intensity
- Irritability in routines
- Deficit in executive function
- Emotional instability
- Anxiety
- Deficit in attention and concentration
- Accelerated pace of thought
- Indisposition
- Nighttime tiredness after activities involving automatism
- Disorganization
- Excesses
- Impulsivity
- Affinity for sugar and carbohydrates
- Procrastination
- Hyperactivity/hyperkinesia
- Constant search for novelty
- Hypersensitivity and intolerance to pain
- Shyness
- Social isolation
- Hypersexuality
- Concern for others, but not for oneself
- Initiation of several activities or projects simultaneously
- Affinity for caffeine and energy drinks
- Sensory hyperstimulation
- Addictions
- Tics
- Adrenaline-dependent habits
- Attraction to extreme sports and complex and challenging activities

- Insomnia

Assessment of adverse childhood emotions and PTSD (Krauser Questionnaire)

Greater exposure to adverse childhood effects or mild aggravating events (≥4 ACEs) has a "dose-response" relationship and increases the risk of presenting chronic diseases by around two times or more, as does the presence of PTSD.[132-140]

Emotional Intelligence/Mentalism Inference Assessment

- *Assess the presence of Alexithymia;*

After explaining the concept of an emotion, ask what emotion the individual is feeling at that moment (precise identification). If the immediate response involves rationalization or prediction, a state of alexithymia is configured. After several attempts after training, and if the individual does not present technical success, we can consider significant alexithymia.[132-140]

- *Anosognosia Assessment:*

Ask the individual to try to identify or perceive their emotion, without predicting. Assess whether they actually observe it.

- *Assessment of Amygdala Neuroschemas*

Ask questions about their family members and observe whether there are sudden cognitive and behavioral changes, which are negative in nature, or with speech incongruent with the behavior and reality produced.[132-140]

- *Assessment of Family Synchrony/Mentalism.*

Check whether the individual can perceive the will or emotional state of a family member and fulfill the demand of the moment. Check for routine, automatic habits that are the individual's beliefs.[132-140]

- *Anasodiaphoria Assessment*

After the assessment, provide psychoeducation and identify whether the individual has the ability to accept their condition. They usually present denial, avoidance, escape, aversion, and even punishment. The presence of this clinical neuromarker indicates marked neuromaladaptive activity.[132-140]

4.2 Neurobiology of Stress

Stress is an important part of modern life. Humans, like other species, have developed adaptive mechanisms to limit the physiological or psychological

impact of stress. However, exposure to traumatic or cumulative stressors can contribute significantly to the development of several comorbidities. [141-155] Indeed, the stress response is fundamentally an adaptive phenomenon directed at the reallocation of physiological resources in response to an external or internal stimulus that has threatened homeostasis. This process of active adaptation through the mobilization of neuroendocrine and immunological mechanisms has been called allostasis.[141-155]

Allostatic load refers to the cost of this rebalancing process for the organism. In situations of acute or sporadic exposure to stress, the cost is low and transient. However, in situations where the stressor is persistent or the organism is weakened, prolonged involvement or overstimulation of allostatic systems causes a physiological burden that can lead to disease. [141-155]

This chronic involvement of the stress response system has been associated with a number of health disorders, including cardiovascular, immunological, and reproductive dysfunction and an increased incidence of stress-related psychiatric disorders. [141-155]

Thus, the current view is that stress-related pathologies develop from unnecessary, excessive or long-lasting activation of the stress response system that affects physical and mental physiology.[141-155]

Investigation of the mechanisms of adaptive and maladaptive stress response, as well as interpersonal interactions, is fundamental and includes effects on cognitive and translational function.[141-155]

Acute stress can be defined as a temporary real or perceived challenge to the organism's ability to maintain homeostasis and can be physiological or psychological in nature. [141-155]

The organism responds to acute stress by rapidly mobilizing the autonomic and neuroendocrine systems, producing physiological changes that facilitate the response to the threat and the return to homeostasis. Activation of the autonomic system releases epinephrine (E, secreted by the adrenal medulla) and norepinephrine (NE, from the adrenal medulla and sympathetic nerves) that act on peripheral adrenergic receptors. Catecholamines are released into the brain, where they activate receptors in the central nervous system (CNS).[141-155]

The acute effects of catecholamines are short-lived, disappearing within 1 h, and include cardiovascular actions, allocation of metabolic resources, and sustained alertness. [141-155]

The neuroendocrine response is under the control of the hypothalamic-pituitary-adrenal axis (HPA axis), which is the system where glucocorticoids (cortisol) are released by the adrenal cortex in response to circulating adrenocorticotropic hormone (ACTH) released by the anterior pituitary. In contrast to catecholamine-induced responses, glucocorticoid effects can be rapid (within minutes of stimulation) and long-lasting.[141-155]

Long-term effects develop over several hours and include transcriptional effects from activated glucocorticoid receptors (GRs) and epigenetic effects, such as methylation changes in target genes. [141-155]

With prolonged and/or intense exposure to stress (chronic stress), the physiological burden of reestablishing

allostasis can produce detrimental consequences for the organism.[141-155]

Chronic secretion of glucocorticoids decreases GR expression in the brain, which results in reduced negative feedback and dysregulation of the HPA axis. Due to reduced GR levels, CRH levels increase, and the balance between MR and GR expression is altered; these changes affect the function of other brain areas, mainly the prefrontal cortex (PFC) and the hippocampus, and may underlie the emotional and cognitive impairments produced by chronic stress. [141-155]

Disturbances in glutamatergic transmission have been linked to depression in clinical studies and in animals analyzed with molecular markers of glutamatergic transmission suggest that similar adaptations may also underlie HPA axis dysfunction. [141-155]

4.3 System X: Psychoneuroendocrinology

Recognizing and identifying the various neuropsychological mechanisms in relation to eating habits is essential to understanding the relationship between the psychological factor and obesity and thus increasing therapeutic success in cases of obesity treatment failure.[156-170]

An imbalance between energy intake and expenditure is considered the main cause of obesity. Numerous distinct characteristics and conditions can contribute to obesity, and exposure to very high levels of stress-related glucocorticoids leads to a phenotype characterized by abdominal obesity and other features of the metabolic syndrome, such as increased blood glucose and decompensated diabetes mellitus (DM)[156-170]

Altered appetite hormone signaling plays a crucial role in obesity, as it can lead to uncontrolled eating due to reward. Such disorders can be induced not only by weight gain itself but also by overexposure to glucocorticoids due to chronic stress. Cortisol affects reward pathways and appetite centers in the brain, with a role for insulin, leptin, neuropeptide Y (NPY), endocannabinoids, orexin, and gastrointestinal hormones.[156-170]

A recent meta-analysis demonstrated that adversity also correlates with long-term GC levels and found robust evidence for an association between hair cortisol and obesity.[156-170]

Food cravings are regulated in the brains of individuals with obesity, as gut hormones and adipose tissue regulate appetite and satiety in the hypothalamus, play a role in the development of obesity, and cause dysfunction of glucose and lipid metabolism.[156-170]

These problems are exacerbated in certain individuals who have a genetic susceptibility to fat accumulation, which may be caused by significant interactions between homeostatic circuits and brain reward.[156-170]

Accumulation of lipid metabolites, inflammatory signaling, or other hypothalamic mechanisms that impair neurons in the hypothalamic region may also lead to obesity. Numerous data indicate that social, behavioral, neuroendocrine, and metabolic factors can encourage compulsive eating behaviors, thus increasing the risk of obesity.[156-170]

Milano W et al., found a clinical connection in the neurological circuits underlying dysfunctional eating behaviors after reviewing more than 100 publications that presented neuroendocrine alterations, emotional homeostatic factors, and the reward circuit.[156-170]

In binge eating,

food addictions, and in individuals who present the ingestion of pleasurable foods as an emotional regulation mechanism (food coping), the main signs and symptoms of exposure to highly palatable foods are loss of control, increased impulsivity, inability to change eating behavior, and deficits in the sense of premonition and negative consequences.[156-170]

Vainik U. et al. analyzed the behavioral similarity linking the personality profiles of each phenotype in 28 phenotypes extracted from 22 studies and showed that obesity had a moderate to high behavioral similarity with addictions, and compulsive eating was underlying most psychiatric phenotypes, while obesity was interconnected with mood disorders and certain personality disorders. [156-170]

A current study demonstrated in rats that 5-HT 1A receptors in the left BLA are more responsible for anxiety-like behaviors and metabolic changes in responses to stress, with modifications in weight gain. In such environments where maintaining homeostatic goals of energy and nutrient balance does not present a challenge, overconsumption is believed to be driven by a more hedonic form of eating.[156-170]

The term hedonic eating refers to intake driven not by metabolic need but by the reward experienced from consuming food, particularly relevant for highly palatable and high-calorie foods, which have an emotional regulation or coping function. Although the vast majority of cases of childhood obesity are exogenous, a small proportion may have endogenous causes. In many studies, parental stress is associated with obesity in children.[156-170]

In both adult and child obesity, the main clinical component to be observed is the presence of family schemas, which function as hidden triggers, and chronic stress in the home environment and induce reward behaviors.[156-170]

4.4 System S: Psychoneuroimmunological

The neurobiological mechanisms of stress resilience and of physiological and transcriptional adaptations of specific brain circuits are interconnected and influence the role of cellular and humoral factors of the immune and inflammatory systems and changes at the interface of the peripheral nervous system.[156-170]

Neuroimmunology is one of the fastest-growing fields in science and helps to bridge the gap between the nervous system and the immune system. Although both systems affect each other through bidirectional interactions, we focus on the direction of nervous system effects on immunity.[156-170]

Our cognitive and social abilities depend on a highly sensitive and finely tuned balance of immune responses involving innate and adaptive immunity. Autoimmunity, chronic inflammation, infection, and psychosocial stress have bidirectional effects. Innate immunity produces resistance by sustaining reactivity to pathogens and by sustaining long-term nonspecific responses. Innate immunity may contribute to human diseases characterized by an uncontrolled increase in inflammation, even sustaining a neuroinflammatory cycle. Innate immunity plays a key role in the growth and progression of brain tumors and in several neurological diseases.[156-170] According to Slavich GM, social cues derived from the family and occupational environment influence the brain and transmit downstream signals to the peripheral immune pathway through several mechanisms, such as the hypothalamic-pituitary-adrenal (HPA) axis, adrenergic signaling, and the vagus nerve.[156-170]

These processes alter different body functions and metabolism, such as increased levels of cortisol, epinephrine, norepinephrine, vasopressin,

oxytocin, interleukins (IL), and interferons (INF), which in turn modify gene expression and immune cell function. 104–122 Recently, the gut microbiota has emerged as a key regulator of brain and behavior, especially those related to social function.[156-170]

Obese individuals have a different microbial profile than lean individuals, and this may lead to transient immune dysfunction. These findings are particularly relevant for individuals with impaired immunity, including the elderly and individuals with obesity.[156-170]

The examining the influence of the gut microbiota on neurobehavioral functioning related to social processes across the animal kingdom. [156-170]

Obesity-related inflammation originates in the intestinal lumen, where bacteria-derived substances leak into the bloodstream and are believed to initiate inflammation.[156-170]

Ritz et al. demonstrated that the microbiota in individuals with SAD may generate increased social fear that is associated with impaired peripheral immune activation and neuronal oxytocin.[156-170]

The distinct and increased social fear response was associated with changes in central and peripheral immune function and oxytocin expression in the bed nucleus of the stria terminalis.[156-170]

Diseases associated with social isolation are typically characterized by elevated inflammation, and the accumulated results have provided an early indication of the molecular pathways through which they lead to increased systemic inflammatory activity. [156-170]

Inflammatory biomarkers:

- Neurotrophin 4 (NT-4), Vascular Endothelial Growth Factor (VEGF), Epidermal Growth Factor (EGF), Fibroblast Growth Factor (FGF), Transforming Growth Factor β1 (TGF-β1), Interleukin (IL)-6, IL-8, IL-10, and IL-12p70, and changes in BDNF DNAm in blood23

4.4.1 Neuro-oncology System

Adiponectin production is, however, reduced in individuals with obesity in response to increased production of the pro-inflammatory cytokine tumor necrosis factor-alpha (TNF-a), contributing to a state of 'hypoadiponectinemia' and increased tumorigenesis. [171-198]

Pro-inflammatory cytokines, including interleukin-6 (IL-6), IL-1β, and TNF-a, released by adipocytes themselves, increase the production of C-reactive protein (CRP) and serum amyloid A (SAA) and may contribute to tumorigenesis.[171-198]

Adipose tissue is a metabolic organ composed primarily of adipocytes that secrete a variety of bioactive signaling molecules, including pro-inflammatory adipokines and cytokines that may stimulate cancer development.[171-198]

Altered DNA methylation patterns, considered a hallmark of cancer for their regulation of tumor suppressor genes and oncogenes, continue to be studied as a possible link between obesity and cancer risk.[171-198]

There are several biologically plausible mechanisms that explain why Sd X may increase the risk of high-grade prostate cancer. Sd X conditions are associated with a proinflammatory state, with elevated levels of C-reactive protein, TNF-a, interleukin 8 [IL-8], IL-6, and IL-1β, which have been associated with prostate cancer risk.[171-198]

However, hyperinsulinemia, a common condition among men with Sd X, has been associated with an increased risk of death from prostate cancer. Sd X conditions may also alter circulating levels of IGF-1, leptin, and adiponectin, all of which are associated with prostate

cancer risk.[171-198]

The implication of the same complex, the NLRP3 inflammasome, in different diseases, such as cardiovascular, neurodegenerative, psychiatric and metabolic diseases.[171-198]

Between the years 2007 and 2020, 65 case-control and cohort studies were identified. In addition to general cancer, 12 types of cancer have been associated with psychological stress, with the majority for breast, colorectal, and lung/prostate/pancreatic cancer.[171-198]

Evidence on the mechanisms is still scarce; the development of cancer in relation to stress may be due to interactive and combined effects of different types of stressors, but this interaction has not yet been truly tested.[171-198]

The stress pathway to cancer incidence consists of a biological pathway with endocrinology and immunology, as well as stress-induced behavioral pathways including smoking, alcoholism, sleep disruption, unhealthy diet, and low physical activity, along with the related phenomenon of obesity. [171-198]

4.4.2 Neurodermatological System

The skin actively responds to psychological stress, with involvement of skin immune cells, hormones, and neurotransmitters. [199-206]

Skin immune cells actively regulate tissue inflammation with their pro-inflammatory and anti-inflammatory effects.[199-206]

Stress-induced skin reactions mainly include secretion of cytokines (e.g., interleukin-6, interleukin-1, interferon-γ) and activation of the corticotropin-releasing hormone (CRH). -Proopiomelanocortin (POMC)-adrenocorticotropic hormone (ACTH)-peripheral skin corticosteroid axis, which leads to acute/chronic secretion of corticosteroids in the skin. [199-206]

Skin cells themselves can secrete these hormones and participate in skin inflammation. Thus, the local skin CRH-POMC-ACTH-corticosteroid axis plays a prominent role in stress-induced responses.[199-206]

In addition, keratinocytes and fibroblasts produce and express receptors for hypothalamic and pituitary signal peptides (CRH receptors and POMC degradation peptides melanocortin receptors), which allow them to respond to CRH by activating the POMC gene, which is then followed by the excretion of ACTH and subsequently corticosteroids.[199-206]

Keratinocytes can express receptors for neurotransmitters (e.g., adrenaline, noradrenaline, dopamine, histamine, acetylcholine), neurotrophins, and neuropeptides (e.g., substance P, nerve growth[199-206]

Psychoneuroimmunology provides an understanding that the skin is both a target and a source of stress mediators[199-206] .

This complex, locally expressed, stress-induced network has been confirmed to be active in many skin diseases (psoriasis vulgaris, atopic dermatitis, chronic urticaria, human papillomavirus infections/warts, hair loss, and acne).[199-206] Skin reactions to stress and their influence on skin diseases may have implications for disease severity and exacerbation frequency, given the effect of locally secreted corticosteroids and other mediators that affect skin integrity, inflammation, and healing potential.[199-206]

Studies have shown that the introduction of psychiatric treatment (medication or psychotherapeutic methods) may have positive effects on dermatological

diseases influenced by exposure to psychological stress.[199-206]

Skin barrier dysfunction in AD may be due to downregulation of epidermal barrier components such as filaggrin, acylceramides, cornified envelope precursors, and claudin-1. 104-122 T helper cell-derived (Th2) cytokines, such as interleukin (IL)-4 and IL-13, and keratinocyte-derived cytokines, such as thymic stromal lymphopoietin (TSLP) and IL-33, contribute to Th2-mediated cutaneous inflammation in atopic dermatitis. 104-122 IL-31, TSLP, IL-4, and IL-13 are capable of directly activating primary sensory neurons to induce itch in AD, and the increased susceptibility to itch (termed allocnesis) is partly due to epidermal hyperinnervation.[199-206]

Importantly, the three main factors (skin barrier dysfunction, immunologic abnormalities, and itch) interact with each other, creating a positive feedback loop that leads to the induction and maintenance of atopic dermatitis.[199-206]

Atopic dermatitis is a complex disease that traditionally involves an interaction of genetic, environmental, and immunological factors.[199-206]

The observations that internal (bacterial infections) or external (psychological) stressors can induce flare-ups of atopic dermatitis are explained by studies showing that stress impairs skin barrier function and favors a shift in immunity toward an allergic/T helper cell type 2 response.[199-206]

Furthermore, those with atopic dermatitis appear to have an inherited hypothalamic deficiency that impairs normal function of the hypothalamic-pituitary-adrenal axis. Neuropeptides released into the skin may also mediate neurogenic inflammation, including mast cell degranulation.[199-206]

Atopic dermatitis causes significant stress and impaired quality of life in patients and their families. Psychological and stress-reduction interventions have recently been shown to improve patient well-being and significantly improve cutaneous manifestations.[199-206]

In response to stress, upregulation of neuropeptide mediators in the brain, endocrine organs, and peripheral nervous system directly affects immune and skin-resident cells. Lesional and non-lesional skin of patients with atopic dermatitis demonstrates increased mast cells and mast cell-nerve fiber contacts. [199-206] In the setting of stress, sensory nerves release neuromediators that regulate inflammatory and immune responses, as well as barrier function. [199-206]

4.4.3 Neuropneumology System

Trachtenberg E et al. analyzed the link between the social environment, the immune system, and health outcomes and identified neuroimmunological mechanisms, highlighting the promotion of a 'safe' state by the vagus nerve, oxytocin circuits, and the additional contribution of reward pathways. [207-218]

They showed that individuals who experienced social isolation exhibited greater expression of pro-inflammatory immune response genes, which fight bacterial infections and are involved in inflammatory diseases (the pro-inflammatory cytokine genes IL1B, IL6, IL8, and TNF), in addition to decreased expression of antiviral immune response genes (IFN-A and IFN-B), which are adaptive immune genes and target intracellular pathogenic threats.[207-218]

These changes in the expression of immune response genes were linked to increased activity of the transcription factors NF-κB and AP-1, which promote decreased activity of interferon response factors, which generates innate antiviral resistance, and

promote decreased activity of the glucocorticoid receptor, which regulates inflammation by acting as a transcription factor.[207-218]

Current evidence suggests that exposure to stressors (at the individual, family, and community levels) or stress (acute and chronic) is associated with asthma and worse asthma outcomes, but such evidence should be interpreted with caution due to limitations in the design or analytical approach of the published studies.[207-218] Lung function results demonstrated that airway hyperresponsiveness (AHR) was increased by psychosocial stress compared with allergy exposure alone. 104–122 Furthermore, the inhibitory effect of corticosterone on IL-6 and TNF-a in LPS-stimulated splenocyte cultures in vitro was decreased in the asthma-RDS group compared with the asthma group.[207-218]

The CCK-8 assay revealed that the inhibitory effect of corticosterone on LPS-induced splenocyte proliferation was significantly impaired in the RDS and asthma-RDS groups, whereas no significant effect was observed in the control and asthma groups. GR mRNA and GR protein expression were significantly reduced in the lung tissues of the asthma-RDS group. [207-218]

Social interruption stress can promote anxiety-like behavior, activate the hypothalamic-pituitary-adrenal (HPA) axis, increase AHR and inflammation, and also impair glucocorticoid sensitivity and function in a murine model of asthma. 104–122 Social interruption stress-induced downregulation of GR expression is in part associated with glucocorticoid insensitivity, which leads to asthma exacerbation.[207-218] The immune system responds to stressors and communicates with the central nervous system through several mechanisms, including cytokine signaling, vagal innervation, and the lymphatic system. Stressful life experiences have been reported to be associated with elevated proinflammatory cytokines in childhood and also with a high risk of mental illness in adulthood.[207-218]

Prenatal stress can cause not only abnormalities in the HPA axis but also epigenetic alterations in the fetal glucocorticoid receptor gene (NR3C1), leading to impaired glucocorticoid metabolism.[207-218]

Maternal stress can alter fetal cytokine balance, favoring TH2 (allergic) immune responses characteristic of atopic asthma: interleukin 6 (IL-6), which has been associated with preterm birth, can promote TH2 responses by stimulating the production of IL-4 and IL-13. 104-122 Given a link between stress, prematurity, and asthma, future research should include birth cohorts with the goal of confirming and further characterizing "premature asthma."[207-218]

There was a significant linear trend for allergy risk from good stress management skills without stressful events to poor stress management skills with stressful events with significant interaction in additive models.[207-218] One useful intervention involves mindfulness techniques, which allow the individual to put their life situation into context for better personal management.104-122 There are both independent and antagonistic combined associations of stressful life events and stress management skills with allergy risk.[207-218]

4.5 System Y: Neuropsychiatric

4.5.1 Social Anxiety

Social Anxiety Disorder (SAD) is characterized by discrete behaviors, such as deficits in social interaction, restlessness, and emotional regulation behaviors. In moderate intensity, it is characterized by intense fear, anxiety in social situations, and escape and/or avoidance.[219-232]

Understanding the

biological basis of SAD is essential because it is one of the most disabling and common MDs, which basically represents a significant neurodysfunction of common social mechanisms. The worst stage is considered to be social phobia and episodes of panic syndrome.[219-232]

The onset of SAD occurs early in life and often has a lifelong impact, being associated with considerable functional impairment and reduced quality of life. This distinct deficit in normal social fear responses is combined with disturbances in brain immunity.[219-232]

The typical personality profile of patients with SAD is characterized by a tendency to respond to perceived aversive stimuli through inhibition, in light of an inability to change behavior to achieve personal goals, a reduced tendency to respond to novelty through exploration, and a lack of cooperation in the social context.[219-232v] In the family environment, these states become chronic due to the presence of chronic activation of family schemas, aiding in the refractoriness process of anxious and depressive MDs, in addition to significantly influencing chronic diseases. [219-232]

4.5.2. Depressive Disorders

Major depressive mood disorder (MDD) is a devastating neuropsychiatric disorder that encompasses a wide range of cognitive and emotional dysfunctions, which lead to work limitations and low quality of life, in addition to presenting a significant risk of suicide. [233-240]

The etiology of depression is multifactorial and is characterized by the interaction of genetic and environmental factors and structural alterations of the brain, in addition to psychosocial factors, when there is some physical disability, obesity, or bullying.[233-240]

fMRI studies have documented neurodysfunctional alterations in mesocorticolimbic circuits and revealed a general decrease in subgenual anterior cingulate cortex and striatal reactivity to rewards in depression.[233-240]

Several neurobiological studies have shown an association between maternal depression and ACE, in addition to causing harm to child neurodevelopment, which in the presence of adversity has a 60% chance of developing depression over the lifetime.[233-240]

A study of cortical thickness in adolescents of mothers with recurrent depression found impaired development in cortical thickness and atrophic gray matter in the anterior cingulate were associated with difficulty managing sadness. In healthy conditions, we are able to process events with apropriate top-down regulation and optimism governed by the prefrontal cortex (PFC).[233-240]

Symptoms of major depressive disorder include psychic pain and anhedonia, which is a neuropsychological state in which negative events are prioritized, neutral stimuli are experienced as sad or pessimistic, and pleasure is lost in rewarding events. However, with exposure to uncontrollable stress with emotional exhaustion, such as burnout, the orchestration of brain circuitry changes in such a way that we can lose perspective and experience our universe through the "aversive lens." [233-240]

Recent research in nonhuman primates has begun to illuminate the neural basis for this phenomenon, where activation of Brodmann Area 25 (BA25), a major PFC output to subcortical structures that mediate emotion, increases threat responses and induces anhedonia.[233-240]

These mechanisms occur due to overactivation of BA25 in patients with depression, and

when BA25 activity is normalized, symptom relief occurs. Activation of the subgenual cingulate (BA25) induces the "aversive lens" state, and like higher prefrontal cortical (PFC) areas, it provides top-down regulation of BA25 but is weakened by excessive dopamine and norepinephrine release during stress exposure and loss of dendritic spines with chronic stress exposure.[233-240]

Bidirectional connectivity between BA25 and the basal amygdalar complex may create a positive feedback loop that contributes to its overactivation and the generation of anxious behaviors. One of the core symptoms of major depressive disorder is anhedonia, an inability to experience pleasure.[233-240] In patients, greater anhedonia was associated with lower temporal difference activation in the ventral tegmental area, whereas in healthy controls.[233-240]

5. Discussion

Kelly and Barker point to the need for new solutions to treat chronic disease problems:241-254

''The number of people in the world with type 2 diabetes is expected to increase from 366 million currently to 552 million by 2030, and while an estimated 17 million people died from cardiovascular disease in 2008, an estimated 23 million are expected to do so by 2030. Responding to and understanding these epidemics must involve human behavior. However, it is not just individual behavior, of course, that drives these epidemics.´´

Behavior occurs in social settings, and efforts to change it must therefore take into account the social context and the political and economic forces that act directly on people's health, independently of any individual choices they may make about their own conduct. This method organizes the clinical approach to the family factor, which is neurodysfunctional and represents a promising tool, but is in its infancy in terms of theoretical basis, which requires experimental evaluation.241-254 Currently, there are not many studies that address psychological factors as moderators in the analyses due to the lack of universally accepted definitions to apply in the studies.241-254

Currently, medical clinic specialists use the CaReMe syndrome model, which is the direct association of cardiorenal-metabolic comorbidities in the form of heart failure, chronic kidney disease and DM, in which they carry out new research approaches with the association of comorbidities. This model has the same design and objective. The investigation of common neuropsychological factors should be considered for chronic public health diseases, since some comorbidities are currently increasing in prevalence, such as young obese individuals, who currently have an increased incidence of colorectal adenocarcinoma. 241-254

This system model presents a mechanistic organization based on neurobiology in the family environment, which addresses only objectivity and preserves the individual and family integrity of the patient. It does not interfere with professional assistance work and also aims to assist in understanding and directing a more assertive flow to specialized MH services. 241-254

Practical example: An adult patient with obesity, hypertension, and a smoking habit, who presents the objective neuropsychological clinical criteria of group Z and who has clinical events with a pathophysiological causal link with obesity and/or smoking, we will use the ZXY Sistem classification. The letter X for obesity and hypertension + Y for smoking.

6. Conclusion

We present an unprecedented clinical model that objectively addresses the mental health of the family in a comprehensive manner, with a joint approach of clinical medicine, through traditional empirical science, which were connected to the accumulated data of clinical neuroscience.

It can be used as a tool for new scientific research, updating and advancing family medicine, mental health policies, and creating mental health care plans in primary care, which is currently lacking. It can be used in multifunctional machine learning platforms for extraction, aggregation, management, and analysis of clinical data to support physicians and researchers, efficiently stratifying subjects to understand specific scenarios and optimize decision-making. 241-254

The limitation of this study is that it is a review with a synthesis of a theoretical systems medicine model, which was extracted from empiricism, and does not present experimental methodology but directs future population research, with the opportunity to methodologically address the objective neuropsychological component, simultaneously with chronic diseases in clinical medicine.

7. Declaration of conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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