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EMOTIONAL SURVIVAL PERSONALITY MICROSTRUCTURE ONCS: THE NEUROBIOLOGICAL ORIGINS OF PERSONALITY DISORDERS AND DYSFUNCTIONAL PARENTING

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Introduction

According to the World Health Organization (WHO), the burden of Mental Disorders (MD) problems increases with the years. According to the WHO, in developed countries, around 50% of patients with MD do not receive treatment effectively.[1]

Currently, in the scientific community, there is little research approach to Personality Disorders (PD), and in professional practice, diagnosis and treatment are two factors of technical failure.[2] PD is defined in the current psychiatric diagnosis system as characterized by generalized patterns, inflexible and stable of thought, feeling, behavior and interaction with others that cause significant suffering to the individual himself and the people in his life.[3]

Problems in personality functioning are defined as how an individual typically experiences him/herself and relates to others: deficiencies in "self" functioning include problems with identity, self-concept, self-direction, and agentic behavior, while deficiencies in "interpersonal" functioning include dysfunctions of intimacy, empathy, and communal behavior.[4] The importance of interpersonal dysfunction in defining PD is fundamental, and currently its descriptive characteristics and diagnostic criteria have not yet been integrated into the accumulated knowledge in neuroscience and human neurobiology.[4]

Currently, there is extensive evidence of multiple social, mental health, and organic impairments that adverse childhood emotions (ACEs) and traumatic situations that generate Posttraumatic Stress Disorder (PTSD) cause in the early childhood window, and unequivocally are situations of interpersonal interaction between biological parents.[5] Although there are few approaches in the family context in an integrative way, with neuroscience and neurobiology, there is a growing body of empirical research that is considering associations between TP, and the quality of functioning in interpersonal relationships (IR) with a focus on emotions.[5-9]

Given the central role of interpersonal dysfunction for PDs, research is developing objective pre-diagnostic constructs. There is still no

comprehensive approach that connects the context of IR, and thus we present a new approach, grouping the evidence of affective temperaments and the current accumulated data from neurobiology and social neuroscience.[5-9]

Through the pathophysiological organization of the neuromaladaptive mechanisms, which were identified in empirical practice, and connected to these data.5-9

They are dysfunctional, as they significantly influence mental health and in clinical medicine, psychiatry and social conflicts involving the family.[5-9]

A set of Eight Common Neuro Dysfunctions (CNDs), identified in clinical practice, presents a nosological hierarchical construction, which represents a microstructure of the initial personality, as it precedes and contributes to the onset of the development of some psychopathologies and some PDs.[13-19]

They are biobehaviors that develop in response to the quality of affective and emotional attention from biological parents, in relation to child neurodevelopment. The following are the clinical and neurobiological elements of ONCs: [13-19]

(1) Neurological Deficit of Family Synchrony (DNSF),

(2) Amygdala and limbic neuromaladjustment (Family Schemas),

(3) Hormonal and immuno-inflammatory dysfunctions,

(4) Dysfunctions of family neuropsychodynamics and neuromirroring,

(5) Neurodysfunction of the reward system and dopamine homeostasis,

(6) Neurodysfunctions of neural networks with emotional engrams, with deficits in primitive and higher mental functions (Neuroschematic Survival Syndrome)

(7) Secondary simultanagnosia1 and cognitive limitation,

(8) Dissociative disorders of neuroadaptive consciousness

1) Secondary simultanagnosia: Inability to effectively identify more than four objects at the same time.

2) Secondary Alexithymia: Inability to effectively self-observe (self-identify) emotional and affective awareness at the same time.

3) Secondary Anosognosia: Inability to effectively identify the emotional and affective state of another person.

4) Anosodiaphoria: Inability to identify one's own health condition, associated with denial of one's state. [13-19]

These are subtle and automatic states such as Alexithymia[2], Anosognosia[3], Anosodiaphoria[4], such as dissociation of Consciousness, deficit and limitation of intellectual cognition, caused by commissurectomies of the anterior fasciculus, which is an interhemispheric disconnection caused by hostile behaviors, mainly in the verbal component. [13-19]

Thus, the limitation of the extension of childhood consciousness is identified by the neuroclinical marker of secondary simultanagnosia, deviant behaviors, produced by neuroactivation of conditioned systems, such as family schemes, which are signatures of the amygdala systems. [13-19] Review studies have shown the orthogonal dimensions of agency (dominance vs. submission) and communion (warmth vs. coldness), and affective temperaments in an objective way, which are allied to the function of emotional survival. [13-19]

Through the identification of the predominance of behavioral activity in pairs, the initial microstructure of ONCs personality, together with family schemes, express with organized clinical practice the Neurodysfunctional Interpersonal Relationships (NIRs) in the family environment, which was initially described as Syndrome Z, due to the intergenerational pattern in the form of a zigzag. [13-19]

Syndrome Z consists of chronically and dysfunctionally activated neurocognitive and behavioral states, which produce NIRs, affective neglect, ableism, and prejudices, involuntarily. [13-19]

ONCs can evolve into internalizing or externalizing states, are interconnected with different types of stress, and depend on dysfunctional neuropsychodynamics and genetic temperament traits, the development of different types of PD. [13-19]

Organizational Concepts Based on Neuroscience

Brain plasticity, particularly neural hodology, brings new understanding through findings of interneuronal connectivity dysfunctions, and dysfunctions of maladaptive neurosystems will be central aspects of all neuroscience research in the coming decades.[20-24]

A better undersding of the presented network can help advance treatments for neuropsychiatric conditions related to aberrant prediction processing and promote improvement.[20-24] Human consciousness presents objective (neurobiological) and subjective components in which each person has a unique narrative of how volition, habituation, performance capacity and the environment interact and influence over time, and thus provide experiences and learning.[20-24] Currently, some concepts considered by current neuroscience that influence the condition of free will are the notion of emotional self-regulation, volition and agency. [20-24]

The neurocognitive variable related to the development of executive attention is called effortful control, representing the ability of an infant or adult to inhibit a dominant response in order to execute a subdominant response. [20-24]

To be responsible for an action is to have the sense, notion and judgment, in the sense of feeling the negative or positive effect of the effects produced by all the events in the causal chains that led to the existence of the conscious intention that determined the action, whether active or passive. [20-24]

Notion is the minimum or deep knowledge that one has of something and is related to perception and sense. 20-24 Sense is the application of reason with the function of judging, understanding or evaluating. Agency is the sense of responsibility for one's own action. [20-24] Judgment is the process that leads to the establishment of significant relationships between concepts and notions, which lead to logical thinking aiming to achieve significant integration, which gives the possibility of a rational attitude and without harm to oneself and other people, in the face of the needs of the moment. It is the intellectual faculty of judging, understanding, evaluating, comparing, appreciating and reaching conclusions. [20-24]

Individual values are personal convictions with a sense of consideration and importance, which generate significant motivation in their search or acquisition, and which have positive reinforcement with their environment, such as culture, belief, beauty, ethics, morals, but also impunity and lack of enlightenment and knowledge. [20-24]

Interests are related to the pleasure of doing something and are reflected in a preference for certain activities over others, and may or may not be congruent with values. [20-24]

Volition is the cognitive and behavioral process by which an individual decides to perform an action that he or she desires. It is defined as a psychological function of mental state, and neurological of deliberate effort. [20-24]

A complex, continuous and standardized volitional process that involves the causality of personal cognitive acts and states, values and interests, which can be neurodysfunctional and even pathological. [20-24] In the determination of a choice to do an activity, this state is called imminent volition, and when we execute it, this act is called immanent, executive, or imperative volition. [20-24]

When an immanent or determined choice controls or governs a series of voluntary acts, we call this state predominant volition, while subordinate volitions are those acts of will that bring into effect the object desired by the predominant or governing volition.[25-28]

Volitional processes can be applied consciously and can be automated as habits over time, therefore, they consist of a state and an act. [25-28] We know that some actions are freely chosen (voluntary) and others are automatic reactions (involuntary). Furthermore, we know that natural phenomena are caused by other natural phenomena, and we should be able to characterize the difference between them. [25-28]

Voluntary or involuntary acts present production of self-identified or conscious effects, and others that are not self-identified by the individual, thus being unconscious or hidden. However, there are behaviors with partial self-identification, of the real will and the real effect, but the opposite behavior predominates. [25-28]

These are habitualized and/or addicted moments. This is an important observation that presents a clinical connection in the hodology of emotional engrams. [25-28]

Several neuroscientists and researchers of the psychic apparatus have highlighted the importance of the defense mechanisms of the human mind, and a new comorbidity has now emerged, exclusive to these, which affects interpersonal relationships and has an elementary function: Emotional Survival. [25-28]

Nosological Hierarchy Construction

Many psychiatric illnesses manifest as symptoms related to social behaviors and present an etiopathogenesis early in life, such as autism spectrum disorder (ASD), schizophrenia, depression, and social anxiety. [25-28] Accurately identifying etiologies and new treatments for social deficit disorders requires a clinical understanding of the underlying neurological elements that affect social behaviors, such as "imprints" or dysfunctional network signatures that are effectively developed in stressful environments and in the absence of attention. [29-33]

Zhu et al. found that physical maltreatment between the ages of 3 and 6 was associated with blunted amygdala reactivity, while emotional abuse among peers during adolescence (ages 13 and 15) was associated with increased amygdala response. [29-33]

McLaughlin et al., evaluating children raised institutionally and later placed in stable family care, suggest that by age 2, the quality of care has unique and determinant effects on the autonomic nervous system and hypothalamic-pituitary-adrenal (HPA) reactivity. [29-33] This age represents a sensitive period for the development of the neurological attachment system, and an environment lacking objective care has been shown to be prone to the dysfunctional development of stress response systems, emotional regulation, and downstream aspects of emotional development. [29–33]

Kim B. et al., evaluated ACEs using dimensional, specificity, aggregation, and child maltreatment-focused approaches, assessing their impact across six domains: biological system dysregulation, neuropsychological impairments, physical health complications, mental health conditions, social and behavioral challenges, and criminal justice involvement, and noted heterogeneity in the impacts of ACEs influenced by type and specific clinical effects. [29–33]

Across all studies, sexual abuse is the most synergistically reactive ACE, and when ACEs were aggregated without distinction of type, considering only their cumulative effects, adverse outcomes were significantly exacerbated. [29–33]

One study showed that approximately 30–40% of the variance in outcomes is explained by additive synergistic interactions between certain pairs of ACEs. Several studies have shown that there is a higher risk (greater than two) of future chronic comorbidities in the presence of four or more ACEs. [29-33]

The relationship between child maltreatment, witnessing violence and household dysfunction and the risk of engaging in violence, engaging in unhealthy behaviors and experiencing mental

health problems. [29-33] A similar study using a large national sample from the USA again explored how ACEs amplify negative effects. [29-33]

Prenatal risk factors

Several findings highlight the role of ACEs in the intergenerational transmission of psychopathology and the importance of considering the prenatal environment in prevention strategies. [29-33]

According to *Malave et al.*, brain development is understood to be particularly malleable during prenatal and early postnatal life, when complex neural circuits underlying cognition and behavior are formed and refined through an interplay of inhibitory and excitatory neural input, synaptogenesis, synaptic pruning, myelination, and neurogenesis. [29-33] Therefore, maternal ACEs and negative life events during the prenatal period may disrupt these processes and influence mental health during childhood. [29-33]

Neuromaladaptive fetal growth has been extensively studied with intrauterine clinical neuromarkers of adversity and may present detrimental prenatal influences on neuromaturation, and is associated with developmental delays with significant risk of developing MD. [29–33]

Fetal growth is influenced by maternal, placental, and genetic factors. Age, socioeconomic status, maternal organic and mental health, harmful habits such as US, malnutrition, and some gestational diseases are considered maternal influences. [29–33]

Placental dysfunction leading to poor nutrient and oxygen supply to the fetus, fetal malformations are also important factors affecting fetal growth. [29–33]

A meta-analysis demonstrated that extremely low birth weight was associated with inattention, hyperactivity, and internalizing problems in childhood and adolescence, which converge to higher rates of social problems, depression, and anxiety in adulthood. [29–33] Being born below gestational age, with a small head circumference, or with a low ponderal index have all been associated with attention difficulties in parents. [35–39]

Several recent studies have implicated that a variety of early life and in utero exposures can cause epigenetic, immunologic, and metabolic dysfunctions. [35-39]

Barker et al. reviewed epidemiological studies and demonstrated the results of these phenomena, that babies born small for gestational age have a greater susceptibility to cardiovascular diseases and metabolic dysfunction. [35-39]

The brain circuits that significantly influence subjectivity overlap, evidencing the clinical notion of inseparable constructs, through structural and functional neuroimaging data that demonstrate extremely specific deviations in brain processes, mainly among the various types of PD and in the family environment. [35-39]

Kocevska et al. showed that adversity during pregnancy (negative life events, contextual, parental or interpersonal stressors) has negative

effects on children's sleep that persist throughout childhood and are exacerbated by the genetic responsibility for insomnia. [35-39]

Affective temperaments

All mammals share homologous emotional feelings/behaviors (except cognitive ones), which allow certain behavioral and psychophysiological measures to be used as indexes of subjective arousal, especially if these states are demonstrably rewarding or punishing. [35-39] The cognitive and social neuroscience of emotion is greatly enriched by seeking its primary affective underpinnings in cross-species investigations of subcortical emotional circuits, in addition to connecting mental health to clinical medicine in an objective manner. [35-39] The issue of affect regulation is one of the greatest challenges in psychiatry, and cross-species affective neuroscience research facilitates the essential neurochemical view from the bottom up, while cognitive neuroscience research from the top down. [35-39]

Thus, it is essential to have a clearer view of how changes following objective treatments focused on clinical neuromarkers, in the higher mental processes of humans, affect the various patterned expressions and regulation of basic affective processes. The affective temperaments (AD) of well-being (positive affect) and discomfort (negative affect) are a continuum in every human being, but in people with PD, they can cause social harm when a situation of discomfort, in addition to corresponding to a clinical marker.[35-39]

AD play a fundamental role in interpersonal abuse, especially child abuse.[35-39] Several studies have shown the effects of child abuse, life events, and temperaments on individual and social well-being.[35-39] Neglect, punishment, and total abuse in childhood increased the effects of negative life events on depressive symptoms. [35-39]

Kanai et al., showed that child abuse, especially neglect, corresponded to an indirect predictive clinical clue, and direct evidence of worsening of the positive and negative effects through cyclothymic, anxious and irritable temperaments, in addition to the negative evaluation of memory well-being reported up to one year before the evaluation, while hyperthymic temperament showed opposite effects. [35-39] *Mazza et al.,* a current study confirms the high prevalence and complex etiology of depressive symptoms during pregnancy and suggests that the assessment of affective temperament appears to be a useful adjuvant instrument for predicting depressive symptoms during pregnancy and postpartum. [35-39]

Carter et al., in a large cohort study, demonstrated the presence of the cyclothymic component through the analysis of the principal components (PCA) of affective temperament, in addition to proving the validity of other EDs such as hyperthymic and depressive in terms of face validity, construction, clinical and family history. [35-39]

The PCAs showed the presence of a global main factor for each subscale, which is a mixture of affect-laden and neuromaladaptive traits. 35-39 Another factorial structure of

a temperament scale is the TEMPS-A (Temperament Evaluation of the Memphis, Pisa, Paris and San Diego - Autoquestionnaire) which assesses the subscales: depressive (D), cyclothymic (C), hyperthymic (H), irritable (I) and anxious (A), presents good reliability and internal consistency, and is reminiscent of neuroticism-extraversion. [35-39]

Nakai et al., showed that hyperthymic temperament inhibited the depressive effects of childhood abuse, while irritable temperament increased and inhibition temperament the depressive effects of negative (stressful) life events in adults. [35-39]

Positive life events in adults had an inhibitory moderating effect on depressive symptoms that was enhanced by cyclothymic and anxious temperaments. [35-39]

Cyclothymic temperament, TC, is the most common affective predisposition in patients with bipolar depressive mood disorder (BDBD), and is often a predictor that precedes the bipolar course. [35-39]

According to *Torrente F et al.,* TC is a fundamental component to be evaluated in patients with ADHD, as it is a strong predictor of occupational deficit, and is associated with other psychiatric comorbidities, in addition to their family members. [35-39]

In a Norwegian study of adults with BD and ADHD, Cyclothymic TC was associated with higher psychiatric symptom burdens, somatic comorbidity, disability, and greater morbidity among first-degree relatives. [35-39] Personality characteristics are linked to dissociable connectivity flows in the human brain. [40-43]

While fiber tracts between a subcortical network, including the hippocampus and amygdala, and the ventral striatum predicted individual differences in novelty seeking, tracts between the prefrontal cortex and the striatum predicted individual differences in reward dependence.[40-43]

Several researchers have suggested that the strength of limbic connectivity

Researchers have investigated the cognitive and neural mechanisms involved in cooperative behaviors. Cooperation is an essential behavioral trait that evolved to facilitate group life. Relevant cognitive processes include motivation, reward coding, action evaluation, and executive functions in the context of social interactions. The neural bases of these cognitive functions have been located in circuits connected to the striatum. The social cognition system processes signals of trust and/or threat that are regulated by the prefrontal cortex. Several studies have shown that the amygdala and orbitofrontal cortex are responsible for processing economic and social rewards, emotions, and motivation.

The anterior cingulate cortex processes the rewards of others versus one's own motivations. The dorsal anterior cingulate cortex processes the intentions and/or mental state of others in social cooperation behaviors. The lateral and dorsal prefrontal cortex process executive functions, such as cognitive control and inhibitory control.

The cognitive control system has modulatory effects on reward

processing through signaling extrinsic incentives (long-term benefits, reputation building, social sanction and norm, fear of punishment), and thus the social cognition system has modulatory effects on reward processing through signaling trust or threat.

Cooperation can be measured with the subscale of the same name using the well-established Temperament and Character Inventory-Revised. People with high scores on cooperation have been considered empathic, tolerant, compassionate, supportive, fair, and principled. Studies have observed that individuals with low scores on cooperation questionnaires are egocentric, selfish, intolerant, critical, unhelpful, vindictive, and opportunistic. [40-43]

Declerck et al., showed that individuals who consider themselves more sensitive to extrinsic cooperative incentives and rely more on cognitive control to make decisions about cooperation. [40–43]

Studies have shown that Cooperation was negatively correlated with fiber connectivity of the cognitive control system (dorsal caudate to rostral cingulate cortex and ventrolateral prefrontal cortex), and not with fiber connectivity of the social cognitive system, which is connectivity with the medial prefrontal cortex and amygdala. [40–43] According to *Lee and Shomstein*, modification of spatial attention through reinforcement learning requires the integration of reward, attention, and execution processes. [40–43]

According to *Haber et al.*,corticostriatal pathways are an ideal neural substrate for this integration because these projections exhibit a globally parallel and underlying topographic organization. [40–43]

The distributed cortical connectivity of these striatal convergence zones was confirmed through follow-up functional connectivity analysis in resting-state fMRI, in which a high percentage of structurally connected voxels also showed significant functional connectivity. [40–43]

Five distinct striatal zones have been linked to distinct brain functions: the anterior caudate for incentive behaviors and the evaluation of different actions, the posterior caudate for executive functions, the posterior putamen for sensorimotor processes, the anterior putamen for social and language-related functions, and the ventral striatum for the representation of stimulus value and motivational states. [44–49]

Studies have reported significant sex differences in reward-related processes and impulse control, which are influenced by striatal-cortical and striatal-subcortical circuits. Studies have found that the putamen showed a higher anticipatory response to reward in men than in women. [44–49] They also showed faster responses and gained greater rewards in risky decision-making, and showed increased activation in the dorsal striatum, including the putamen. [44-49]

When asked to reject immediate rewards in pursuit of a long-term goal, men showed significantly reduced activation than women in the dorsal striatum, subgenual and pregenual anterior cingulate cortex, and posterior orbitofrontal cortex, as well as greater functional coupling between these regions. [44-49]

Previous studies have suggested that the four temperaments of novelty seeking, reward dependence, harm avoidance, and persistence have their respective neurobiological correlates, especially in the subcortical and cortical networks connected to the striatum. [44-49]

Several neurobiological/molecular features differentiate the two harmful and pathological lines of internalizing and externalizing neuropersonalities. [44-49]

Externalizing problems are behaviors directed toward the environment and decrease with age, while internalizing problems are behaviors directed toward oneself and increase with age. [44-49]

Internalizing

The processes of internalizing neuropersonalities are silent, but of intimate suffering, and present few clinical signs that are confused in subjectivity, but are harmful. [44-49]

Examples include: dysthymia, major depressive disorder, disruptive mood dysregulation disorder, agoraphobia, panic disorder, specific phobia, separation anxiety disorder, social anxiety disorder, generalized anxiety disorder, and PTSD. [44-49]

In studies of internalizing, a phenotypic profile of low avoidance, greater sensitivity to threat, anxiety, fear, vulnerability to stress, passive coping style, and greater sensitivity to frustration has been discovered. [44-49] In most studies with preschool-aged children, parental stress and parental mental health are the two environmental factors that have demonstrated the strongest relationship with children's internalizing problems. [44-49]

Multiple dimensions of parenting have been associated with children's emerging internalizing symptoms, including intrusive and unresponsive parenting in early childhood. [44–49]

Parental hostility, one of the two leading predictors of early childhood, is characterized by unsupportive and controlling parenting practices, displays of anger and disappointment in children, punitive discipline, and perceived parental distancing. [44–49]

Parental hostility toward children has been identified as a behavioral driver of many negative child outcomes. [44–49]

Internalizing symptoms in children and adolescents (ages 9–18 years) are predicted by harsh parenting, and child self-regulation and prosocial developmental problems (ages 6–7 years) are associated with hostile parenting in early childhood (ages 2–3 years). [44–49] According to existing research, parental socioeconomic stress may have an indirect influence on child mental health and behavior through parenting and family interactions that are affected by finances. [44-49] *Swartz HA et al.,* demonstrated the bidirectionality between maternal depression and child internalization symptoms, in addition to proving that the parents' mentalization capacity or sensitivity when effectively perceiving the child's suffering from anguish were clues to secure-base behaviors.[30-40]

Fixed mindsets, or beliefs that personal traits are immutable, show significant associations with internalizing symptoms. [30–40]

Studies have shown that fixed beliefs (mindsets) do not present a unique variation in internalizing symptoms, except for hopelessness, which is significantly correlated with internalizing and anxiety. [30–40]

However, these interventions may not necessarily operate by shaping mindsets; instead, they may affect symptom change by shaping maladaptive cognitions that are associated with hopelessness and stronger links to internalizing distress. [30–40]

Suicide prevention among young people is a global priority. Identifying factors and understanding mechanisms for the development of suicide are key suicide prevention efforts. [30–40] The main risk factors identified to combat suicide among young people are social and demographic inequality, personality factors, interpersonal factors, and mental well-being dysfunctions such as depression, anxiety, and substance abuse. [30–40]

Those who believe that emotions are fixed are associated with less active emotion regulation and are less likely to engage in regulatory efforts. Fixed mindsets about anxiety predicted future psychological distress. [30–40]

Young people with fixed mindsets were found to be more likely to have mental health problems. Fixed mindsets about anxiety were associated with less resilience, less laborious treatment, such as counseling rather than medication, and more psychiatric symptoms. [30–40]

Fixed mindsets about depression predicted depressive symptoms, and fixed mindsets about anxiety were more strongly associated with hopelessness and mental health symptoms than general mindsets about emotions. [30–40]

Fixed mindsets play an important role in the suicidal process. Studies have shown that insufficient mindsets to tolerate mental discomfort, and having fixed mindsets, can decrease hope for improvement and lead to avoidance, coping, and hopelessness. [30–40]

Externalizing

Childhood externalizing problems are characterized by aggression, defiance, and hostility. They appear to result from deficits in behavioral inhibition, impaired impulse control, and hyperactivity. [30-40] Given the general motivation for immediate rewards and excitement seeking during adolescence, the likelihood of substance use, which provides immediate gratification and/or sensation, is amplified in those who present deficits in behavioral control, a hallmark of externalizing behavior. [30-40]

Externalizing neuropersonality processes are also detrimental and are pathological clues with more clinical signs, such as ADHD, conduct disorder, and oppositional defiant disorder. This pattern is common in the pre-adolescent period, as its prevalence is higher.[30-40]

Individuals with high avoidance phenotypes present an "externalization and disinhibition"

profile, with greater impulsivity, greater sensitivity to reward, deficits in social behavior, deficits in attentional/cognitive processes, novelty-induced hyperlocomotion, and greater vulnerability to sensitization to psychostimulants and substance dependence. [30-40]

These resemble features of cerebral immaturity such as hypofrontality, disruption of the cortical excitation/inhibition balance, decreased function of the PFC, hippocampus, and amygdala, increased functional tone of the mesolimbic dopamine system, deficit of central metabotropic glutamate-2 (mGlu2) receptors, increased density of serotonin 5-HT2A receptors in the PFC, impaired GABAergic transmission in the PFC, alterations in severalsynaptic markers, and increased density of immature pyramidal dendritic spines in the PFC. [30-40]

Affectively motivated behaviors

Instituting a delay period between the first exploration of an object and when subjects are presented with the same known object and a new object allows assessment of memory for the known object. [30-40] Recognition memory tasks require minimal learning, which allows measurement of hormonal effects on memory without confounding effects of learning. [41–49] However, possible changes in some performance parameters, such as anxiety and motor activity, cannot be ruled out in the performance of recognition tasks. The prefrontal cortex (PFC), especially the medial prefrontal cortex (mPFC) and the orbitofrontal cortex (OFC), have an inhibitory effect on aggression. [30–40]

The dopaminergic system is activated by three types of external stimuli: rewarding stimuli, punishing stimuli, and novel stimuli. [30–40] When activated by rewards or punishers, parts of the DA system discharge in bursts that increase or allow the separate development of long-term potentiation (LTP) and long-term depression (LTD) of learned connections among others. Regarding the functional aspect of the dorsal and ventral striae, the direct striatonigral pathway and the indirect striatopallidal pathway in the dorsal striatum preferentially control movement, whereas their counterparts in the ventral striatum preferentially regulate aversive responses and reward-seeking emotion. [30–40] The basal ganglia striatum regulates a wide range of neurological functions, including movement, reward learning, affect, and cognition, and striatal dysfunction is implicated in neurological and psychiatric disorders. [30–40]

The wide range of striatal functions arises from the fact that parallel pathways from the motor, sensory, associative, and limbic cortices traverse different regions of the striatum. [30–40]

The striatum is a two-layered system comprising architecturally similar but functionally distinct dorsal and ventral striae. [30–40]

Aversion behavior is the opposite of reward, such as avoidance behavior. [30–40] Dopaminergic neurons in the limbic system have been shown to be involved in these processes, and they subserve different neural circuits to coordinate

downstream cognitive structure and control motivational behavior.[30–40]

Serotonin modulates PFC activity, but its role in aggression depends on the receptor subtypes and brain region in which they are expressed and the types of aggressive behaviors (species-typical vs escalated aggression).[30–40]

The activity of the dorsal root nuclei (DRN), which sends dense projections of serotonergic neurons to the PFC and other regions, is critical in aggression.[30–40]

Baseline serotonin level and phasic serotonin change (state) need to be considered separately, as they may play different roles in aggressive behavior. [30–40]

Dopamine in the NAc increases during aggressive encounters, both in aggressors and in defeated animals. Increased levels of dopamine in the NAc are also observed when an animal is anticipating an aggressive encounter.[30–40]

Pharmacological and genetic studies have shown that nearly all subtypes of excitatory glutamate receptors (NMDA, AMPA, kainate receptors, and mGluRs) and inhibitory GABA receptors (GABAA and GABAB) are involved in aggression. [30–40]

Dopamine neurons have a function in detecting errors in reward predictions, and in individuals that show homogeneous responsiveness across their neuronal population, this indicates that the error signal is widely transmitted and provides a teaching signal for synaptic modifications underlying the learning of appetitive, goal-directed behaviors. [30–40]

The responses of these same neurons to conditioned stimuli associated with reward also function as a useful prediction error signal for learning sequences of environmental stimuli that lead to reward. [30-40]

Neurons that exhibit each of these types of activation appear to differentiate between rewarding/rewarding and non-rewarding/non-rewarding outcomes of behavioral acts, and reflect the relative degree of predictability in the magnitude of responses to reward. [30-40]

The risk of an aversive event occurring after a specific action biases future behavior toward other, safer actions, due to fear that has a survival function. [30-40]

Aversive-motivated behaviors characterize dysfunctional/pathological fear conditioning in some situations, which are related to environmental antecedents, but may also have a concurrent effect that punishes behaviors that cause or predict their occurrence. [30-40] The basolateral amygdala (BLA) is a nucleus that has been implicated in the suppression of punishment seeking (revenge), and this structure can modulate goal-directed behavior through projections to subregions of the NAc, and its structural lesions decrease sensitivity to punishment. [30-40]

The NAc plays a key role in aversive learning and learning flexibility. Activation of D1 receptors in the direct pathway significantly controls reward behaviors. [30-40]

Inactivation of D2 receptors in the indirect pathway controls avoidance behaviors. Aversive learning is regulated by a set of receptors involved in the induction of long-term potentiation of cortico-accumbens synapses. [30-40]

A neuroimaging study that evaluated simultaneous effects of aversion, receipt, and expression of punishment showed that BLA inactivation reduced the expression of punishment, specifically in the caudal portion of the BLA, with the punitive effects being influenced by BLA activity according to its aversive value of punishment. [30-40]

If the caudal BLA is responsible for obtaining and carrying out punishment, which are active, directed behaviors, we conclude that clinically, aversion should occur immediately, when in the presence of a state of low alertness or care, or in unplanned behavior, is clearly not a rule, since the three functions of familial psychopathy may be present. [30-40] fMRI studies in people with typical signs and symptoms of psychopathy and sociopathy have shown neuronal hyperconnectivity in the caudal BLA networks, which may be influenced by the PFC, specifically in its medial portion, and the prelimbic and infralimbic regions. [30-40]

Misconduct

In moments of misconduct, they can be situations of minutes that can change a life, or several. Misconduct is a pathological act in people with dissocial PD and can occur in social, family and professional environments. [30-40]

Emotions can be produced both by the memory of reinforcing events and by external reinforcing stimuli; that cognitive processing (conscious or not) is important in many emotions, as very complex cognitive processing may be necessary to determine whether or not environmental events are reinforcing. [30-40]

Emotions typically consist of cognitive processing that analyzes the stimulus and then determines its reinforcing valence. According to Yater and Stone, almost all choices carry some degree of risk, defined as the possibility that an undesirable outcome may arise from a choice and/or course of action that will elicit a reaction from others and oneself. [30-40] We hypothetically conclude, with the help of clinical empirical observation, that individuals with psychopathy and sociopathy present neurodysfunction of neuronal hyperconnectivity in the caudal BLA networks, which may be influenced by the PFC, specifically in its medial portion, and the prelimbic and infralimbic regions. [30-40] Critically, the phase difference between later remembered and forgotten (familiar) emotional stimuli translates into the time delay required for amygdala and hippocampal gamma bursts to achieve transient coherence with a time delay (~25-45 ms). [30-40]

The human amygdala theta phase for which hippocampal broadband gamma activity is the simultaneous moments of neuronal firing to determine subsequent recall of aversive stimuli. The delayed gamma coherence rhythm across the amygdala

theta phase may represent a general mechanism through which the amygdala relays emotional content to distant brain regions to modulate other aspects of cognition, such as attention and decision-making. [30–40]

It is possible that this delay is related to the time required for noradrenergic input, on which emotional memory formation critically depends, to reach the medial temporal lobe, or that the amygdala theta phase-dependent effects on the hippocampus are linked to the optimal conditions required for "emotion tagging" of memory to occur. [30–40]

Furthermore, these findings may represent a general mechanism through which amygdala oscillations influence other brain areas to enable emotion-induced modulation of aspects of cognition, including perception, attention, and decision-making. [30-40]

Normally the mood or affective state is produced by an external stimulus or a reaction, however how many situations have been technically evaluated the depressive mood, which began and maintained after an event of dysfunctional parenting, or the neurological activation of several family schemes simultaneously.

These are cognitive processes related to sensory salience, attentional control, movement planning and psychomotor activity, which do not actually occur, are illusory or self-deceptions. [49-53]

Whittle S et al., showed that the caudal region of the BLA encodes the aversive value of punishers and, therefore, contributes to instrumental aversive learning and decision-making when the punisher is present, but not when he is absent. [49-53]

The dlPFC is also a key area for executive functions, such as inhibitory control, working memory, cognitive flexibility and planning. [49-53] In the case of CC ("hot" versus "cold") motivational control, perhaps the most striking dissociations for human patients with frontal lobe damage have been cases of everyday decision-making in the absence of obvious impairment in IQ or conventional neuropsychological tests of classic "frontal" deficits. [49-53]

By investigating contexts in which people behave as they wish (spontaneous behaviors), we have identified the distinct, volitional dimension of deliberation, largely neglected by previous work due to the focus on other cognitive control processes (regulation/suppression of intuitive responses). [49-53]

People, regardless of ethnicity, time, or social reason, face similar temptations to violate ethical, moral, or health-related rules in order to serve their own interests, which are camouflaged by pride and selfishness, and which can bias a simple volitional cognitive act such as thought or behavior. [49–53]

Individuals may choose to travel freely on public transportation or overstate the cost of a business trip, or in other instances they may choose to undertake complex professional tasks that require significant time and effort, such as performing major surgery or undertaking complex legal defense, unless the value is appetizing. [49–53] When faced with ethical dilemmas

between dishonestly maximizing self-interest and following normative rules of conduct, individuals often seek to maintain a positive image for themselves, their family, and society and restrict their self-interest to a level that allows them to feel and appear honest. [49–53]

Providing first insights into the contextual factors of the intuitive dishonesty effect, our moderation analyses provide suggestive evidence that the relationship between intuition and dishonesty is shaped by interpersonal neurodysfunctions. [49–53]

Consistent with previous theorizing, particularly the social heuristic hypothesis, our data are consistent with the idea that important consequences for others have a substantial impact on people's intuitive decisions. [49–53]

Some work has shown that cooperating with others is an intuitive inclination in many social dilemma situations. [49–53]*Sai et al.,* along with other results in the literature, suggest that the automatic tendency to cooperate can override selfish impulses to dishonesty when knowing that lying has a price to a concrete other.

Recent empirical evidence underscores the external validity of lying in economic games as a proxy for real-life dishonesty. [49–53]

People frequently encounter such situations in daily life, often deciding quickly, without deep reasoning and reflection. As in the experiments included in the current meta-analyses, these temptations mostly involve relatively small (financial) incentives. 49–53 Although each individual act may seem mundane and only harm vague entities, such as "the bus company" when dodging fares or "society as a whole" when falsifying a tax payment, the aggregate costs are immense. [49–53]

Using fMRI studies evaluating decision-making paradigms, when choosing honest decisions, they observed activations in the anterior cingulate cortex (ACC), dorsolateral prefrontal cortex (DLPFC), and ventrolateral PFC (VLPFC), which are regions associated with conflict monitoring, cognitive control, and response inhibition. [49–53]

In other studies, in which people were asked to lie about autobiographical and factual knowledge, they found similar neural activations, but during dishonesty. [49–53]

In response to a deceptive response, people still need to engage in counterfactual thinking to inhibit the truth and create alternative scenarios. [49-53]

Thus, when responding honestly, intuitively honest individuals still.

Functional decoding analyses revealed that pACC activations were associated with negatively valenced emotions, cognitive functioning, volition, and motivation, consistent with an internal conflict during motivated dishonesty. [49–53]

When making self-interested decisions and in interpersonal situations, the neuroadapted human brain automatically produces coping and defense mechanisms. Faced with the temptation to lie for profit, the basic inclination of people's dilemmas is honesty or dishonesty, ethics or unethics, virtue or vice, and the dilemmas evolve

from truth to pleasure of easy reward, into habit, and later to the neuropsychological complex of addiction or rigidity, becoming covert dependence.

Recent fMRI research on prosocial behavior in humans has demonstrated septal activation when making altruistic donations, with the number of donations also significantly correlated with increased neuroactivation of the cavum septum pellucidum (CSP). [49-53]

Aggression

Changes in aggression have been associated with a range of dysfunctions that have affected other domains of functioning, and aggression is only one of the symptoms. [49-53]

A comparative overview of such models suggests that, although the approach still suffers from a number of deficits, they have the important potential to extend our knowledge of aggression control over the pathological domain of this behavior. [49-53]

Escalated and inappropriate levels of aggressive behaviors considered pathological by psychiatry can lead to harmful outcomes and detrimental impacts on the mental health of the family and society, due to epigenetic impairments. [49-53]

ACEs increase the risk of developing pathological aggressive personality traits in adulthood, and the body of evidence for molecular and neurological mechanisms in humans and animals is voluminous. [49-53]

Rawat RS et al., identified that stressful experiences during puberty can trigger behavioral states of aggressionlong-lasting escalation, through the expression of transthyretin (TTR) in the prefrontal cortex and hypothalamus, which generates alterations in circulating thyroid hormones. [49-53] TTR is a known transporter of thyroid hormones and a key regulator of intense escalation aggressive behavior, which is induced by peripubertal stress (PPS) in men. [49-53]

TTR is an important target for reversing escalation aggression that involves psychopathologies. [49-53]

The group of *Rawat et al.,* demonstrated in male mice induced by PPS, specific methylation alterations of the Ttr promoter that were hypermethylated in the hypothalamus and hypomethylated in the prefrontal cortex, corroborating the pattern of neurodysfunctional epigenetics (long-term expression of the brain region) of the Ttr gene and in the availability of thyroid hormone (TH). [49–53]

Increased aggressive behavior, along with reduced Ttr gene expression and hypothalamic TH levels, were also evident in male offspring of the next generation. [49–53]

Although considerable research has been devoted to elucidating the etiology of aggression, the molecular correlates of sex differences remain largely unexplored. [49–53]

Furthermore, little attention has been paid to whether males and females respond differently to the similar causative factor of aggression. In rat studies, while adult PPS males exhibited escalating aggression, females spent the most time in social exploration. [49–53]

c-Fos expression was brain region- and sex-specific. In the adult PPS cohort, only males showed elevated c-Fos expression in the PFC, indicative of their hyperresponsive behavior. [49–53]

MAOA pressure and enzyme activity were reduced in the hypothalamus and increased in the PFC of hyperaggressive male mice. [49–53] Investigation of the underlying mechanisms revealed hypomethylation in the prefrontal cortex and hypermethylation in the hypothalamus of the MAOA promoter negatively correlated with the expression pattern. [49–53] Serotonin modulates PFC activity, but its role in aggression depends on the receptor subtypes and brain region in which they are expressed and the types of aggressive behaviors. [49–53]

The activity of the dorsal horseradish nucleus, which sends dense projections of serotonin neurons to the PFC and other regions, is critical in aggression. [49–53]

Serotonin [5-hydroxytryptamine (5-HT)] and melatonin are important in neurogenesis, synaptogenesis, mood, sleep, and gastrointestinal function. [49–53]

Basal (trait) serotonin levels and their phasic (state) changes need to be considered separately, as they may play different roles in aggressive behavior. [49–53]

Dopamine in the NAc increases during aggressive encounters in both aggressors and defeated animals. Increased NAc dopamine levels are also observed when an animal is anticipating an aggressive encounter. [49–53] The balance between excitatory glutamate and inhibitory GABA is important for maintaining aggressive behavior at the species-typical (adaptive) level. [49–53]

Pharmacological and genetic studies have shown that nearly all glutamate (NMDA, AMPA) and GABA (GABAA and GABAB) receptor subtypes are involved in aggression. [49–53]

However, the role of each receptor may vary depending on the receptor subtype, its location, and the type of aggressive behavior studied. Combinations of newly developed pharmacological agents that can manipulate a specific receptor subtype and genetic models, such as mice conditional or transgenic for a receptor subtype in specific neurons, hold promise for elucidating the roles of these receptors in aggression. [49-53]

Parental Personality Disorder

Parents with personality disorder may also have difficulty expressing appropriate empathic responses, fluctuations in mental well-being, difficulty maintaining a stable and safe environment, confusion of family roles, managing interpersonal conflict, engaging in parenting skills, and demonstrating self-efficacy.[54-60]

Mothers with BPD are considered particularly at risk. Maternal BPD is associated with lower sensitivity, emotion recognition, parental satisfaction and efficacy, and greater parental intrusiveness, overprotectiveness, hostility, and stress/distress, compared with maternal depressive disorder, other personality disorders, and healthy

controls.[54-60] Parental personality disorder also places children at risk for a range of emotional and behavioral problems. Children of mothers with BPD have significantly more emotional and behavioral problems than children of mothers with depression alone. [54–60]

More specifically, research suggests that these children have greater emotional dysregulation, suicidal ideation, insecure attachment styles, depressive symptoms, externalizing problems, and interpersonal difficulties, as well as poorer overall psychopathology and less stable self-image. [54–60]

Studies have shown that maladaptive parenting predicts BPD features and diagnoses in late adolescence and adulthood, with up to 84% of individuals with BPD retrospectively describing experiences of biparental neglect and emotional abuse before age 18. [54–60] Maladaptive parenting contributes to impaired maturation of emotional coregulation neurons and the transmission of social knowledge, which result in deficits in essential neuropsychological processes. [54–60]

Personality Disorders

Beliefs about the malleability of attributes, also known as mindsets, have been studied for decades in social personality psychology and education. [54-60]

Mindset theory for clinical psychology and psychotherapy. Evidence from social psychology and cognitive neuroscience suggests that mindsets and mindset-related messages are largely focused on emotional tolerance. [54-60]

A growth mindset, or the belief that attributes are malleable, broadens awareness and encourages tolerance of anxiety, frustration, and disappointment in healthy, adaptive ways that promote resilience. [54-60] While fixed mindsets and campaigns that discourage the experience of these emotions are often harmful and neglect effective care and treatment, several studies are currently investigating dimensional associations of personality traits with neurobiological influences, including differential and common patterns in specific PDs. [54-60]

Studies have shown that object relations theories, cognitive schema conceptualization, and the interpersonal model have informed the empirical investigations of PD in recent years. [54-60]

Recent work has suggested that all three perspectives have contributed to and are compatible with the dimensional PD frameworks in DSM-5-AMPD and ICD-11 (level of functioning and traits), as well as psychological treatments optimized for PD. [54-60]

Although the majority of psychotherapies have been developed for patients with borderline personality disorder (BPD), there are a growing number of treatment modalities targeting individuals with other forms of PD, such as those with narcissistic or antisocial traits/disorders. [54-60] Recently, a meta-analysis addressed the new Alternative Model for PD, from the current Diagnostic and Statistical Manual of Mental Disorders (DSM-5), and showed that the pathological traits of criterion B

contributed uniquely to the prediction of several psychosocial impairments, which is essential to consider them as an essential criterion in the diagnosis of PD. [54-60] In this approach, the level of moderate or greater impairment of personality functioning (criterion A) is assessed in the sphere of the self (identity and self-direction) and in the sphere of interpersonal relationships (empathy and intimacy).

Identify the pathological personality traits (Criterion B) among the 25 facets organized into 5 personality domains, which are: Negative Affectivity, Detachment, Antagonism, Disinhibition and Psychoticism. [54-60] In logistic regression analyses, Criterion B demonstrated incremental utility in predicting avoidant PD, antisocial PD, and borderline PD. Criterion A predicted the presence of DSM-5 Section II PDs overall. [54-60] Mindsets, or beliefs about the malleability of self-attributes such as intelligence and personality, have been associated with a wide range of outcomes in educational and social psychology. [54-60]

A longitudinal study assessed weekly distress and anxiety mindsets over 5 weeks and demonstrated that a fixed anxiety mindset predicted distress in the following week, even after accounting for distress in the previous week, gender, and socioeconomic status.

For a long time, the etiology and pathogenesis of most personality disorders have not been elucidated, and psychiatrists have therefore tended to rely on phenomenological and descriptive criteria in diagnosing these conditions. [54-60]

Many of the definitions provided in the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the American Psychiatric Association (APA) are established in a format in which an individual must have a subset of several clinical features, or a checklist to be completed in order to be diagnosed. [54-60]

Furthermore, the study of personality disorders leads to areas in which the distinction between health and illness is ambiguous and potentially influenced by social norms and personal values, which facilitates stigma and prejudice, and then the patient and family lose the opportunity and the right to treatment. [54-60]

Classically, the fundamental condition for making the diagnosis of PD depends on whether the personality traits are harmful to society and the people with whom they live. [54-60]

Despite the uncertainty and variation that characterize personality disorders, psychiatrists have the professional responsibility to diagnose and treat them. [54-60]

Personality involves the way a person behaves, interprets themselves, perceives life, other people and situations, it is about the individual's uniqueness, while Personality Disorders (PD) are a set of important changes, which generate difficulties in personal and social functioning, classified asonly the objective or neurological ones. [54-60]

There are personality traits that are universal or common, and most are acquired in childhood, and undergo several changes and influences related to the environment in which they live, such as family, culture, religion,

profession and society, in addition to genetic and epigenetic issues (temperament). [54-60]

Each individual presents traits of different, unique expressions, of varying intensity, as there are multiple internal and external factors, such as beliefs, quality of mental tone, simultaneous mental and emotional states, neuromaladjustments in different intensities, experience, knowledge, concepts of identity and others. [54-60]

Personality characteristics are linked to dissociable connectivity flows in the human brain. While the fiber sectors between a subcortical network, including the hippocampus and the amygdala. These findings suggest that the strength of limbic-striatal connectivity may function underlying human personality traits. PDs are significant dysfunctions of various cognitive states and behaviors that persist for a long time and can be hereditary or acquired through neuroadaptation in childhood. [54-60] They are identified when an individual's personality patterns are expressed in a very rigid or fixed way, resulting in limited ways of thinking and acting, and are harmful to the people around them, such as in marital, family, and professional relationships, and even pose a risk of harm to society, such as crime. [54-60]

Traditionally, PDs are diagnosed in late adolescence or early adulthood, but there are clinical situations that can arise in childhood. [54-60] Most of the negative personality sets studied consist of alexithymia, emotional dysregulation, neuroticism, extroversion, and introversion. [54-60] Individuals with antisocial PD (ASPD) often present symptoms of insensitivity, social anhedonia, irritability, and impulsivity. [54-60] Neuroimaging studies have shown that behavioral abnormalities in ASPD may have a neurobiological basis potentially involving structural, metabolic, and functional dysfunctions related to deficits in the prefrontal cortex, temporal cortex, insula, angular gyrus, parahippocampus, and anterior/posterior cingulate gyrus. [54-60]

Tang Y et al., showed an aberrant topological organization of the functional brain network in individuals with ASPD, with an increase in the clustering coefficient and a decrease in betweenness centrality in the medial superior frontal gyrus, precentral gyrus, Rolandic operculum, superior parietal gyrus, angular gyrus, and middle temporal pole. [55-66] Furthermore, patients with ASPD showed increased functional connectivity located mainly in the default mode network. [55-66]

Borderline Personality Disorder

Borderline personality disorder (BPD) is characterized by emotional instability, impulsivity, and unstable interpersonal relationships. Patients experience intense levels of distress, inducing symptoms such as dissociation, aggression, or withdrawal. [55-66]

Currently, BPD is the most common among the other PDs, with a prevalence of approximately 22% in inpatients and 10% in outpatient settings. 55-66 BPD is characterized by self-injurious and risky behavior, deficits in emotional regulation and poor impulse control, fragile self-images, unstable relationships, and intense fear of abandonment. [55-66]

BPD is also frequently

associated with trauma-related dissociative symptoms. Both alexithymia and dissociation can be conceptualized as dysfunctional interoception. [55–66]

Hyperattention and vigilance toward potential sources of threat in the external environment produce a state of reduced interoceptive awareness, clinically expressed by alexithymia and dissociative symptoms, decreased pain perception, and general deficits in bodily self-awareness in BPD. [55–66]

Patients with BPD often suffer from comorbid mental disorders, most prominently Major Depressive Disorder (MDD) and Posttraumatic Stress Disorder (PTSD). In addition, BPD is associated with a number of medical illnesses, including cardiovascular disease and obesity.[55–66]

Emotional dysregulation, a core feature of BPD, has recently been associated with deficits in the cortical representation of bodily signals. Oxytocin modulates the salience of external social cues. The ability to perceive and regulate one's own emotions has been closely linked to the processing of afferent bodily signals (interoception). [55-66] Thus, disturbed interoception may contribute to the central feature of emotional dysregulation in BPD, as increased levels of depersonalization, body image disturbances, and reduced sensitivity to physical pain suggest low body awareness in BPD. [55-66]

The results indicate state-dependent deficits in cortical processing of body signals in BPD patients, which appear to be associated with central features of BPD. Therefore, emotional awareness can be understood as modulation failures between interoceptive and exteroceptive attention. [55-66] According to *Flasbeck V, et al.,* the perception of internal body signals and emotional awareness are physiologically related to a conceptual framework called the "Somatic Marker Hypothesis". [55-66]

Low interoception may promote the development of unfavorable coping strategies, such as self-injurious behavior, as a dysfunctional attempt to nullify interoceptive deficits through extremely strong sensory stimuli, which frequently occurs in BPD. [55–66]

This idea is indirectly supported by research suggesting that interoception is neuroanatomically linked to the anterior insula region and the anterior cingulate cortex (ACC). [55–66]

With regard to BPD, functional brain imaging suggests that altered activation of the insula and ACC cortex is related to dysfunctional emotion processing and pain processing. The preferential localization of the HEP over frontocentral regions is in line with the involvement of the frontal cortex, somatosensory cortex, ACC, and insula in the cortical representation of cardiovascular signals. [55–66]

However, altered interoception is not specific to BPD, as it is implicated in other psychiatric disorders, particularly PTSD-related disorders, affective disorders, including depressive and anxiety disorders. [55–66]

Social situations are particularly challenging, and acute social stress can reduce patients' cognitive and social functioning. In patients with major depressive disorder or PTSD, who have high comorbidity with

BPD, the endocrine response to stress is characterized by dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis, which affects cognitive functioning, and is plausibly associated with aberrant or deficient neurodevelopment of neurons and enzymes responsible for inhibitory control and emotional regulation. [55–66]

Because patients with BPD are particularly prone to social stress and experience high subjective difficulties in these situations, it seems plausible that HPA axis dysregulation may contribute to the decreased social cognition in BPD. [55–66]

In a study evaluating genetic variants in BPD, they identified only 2 significant genes: dihydropyrimidine dehydrogenase (DPYD) on chromosome 1 and Plakophilin-4 (PKP4) on chromosome 2. They also observed significant genetic overlap between BPD and bipolar disorder, major depressive disorder, and schizophrenia.[55–66]

There was also 1 GWAS study and 1 genome-wide linkage study of BPD traits, with significant results implicating the serine incorporator 5 (SERINC5) gene and chromosome 9, respectively. [55–66]

Some of these found DNA methylation abnormalities associated with BPD and severity of childhood maltreatment. [55-66]

To date, etiological theories of BPD have presented general factors that have been postulated to be critical for BPD across developmental periods, such as mentalizing, invalidation, attachment, and emotional dysregulation, which now have clear, structured and organized origins in clinical neurobiology. [55-66]

The perspective of the onset and development of psychopathology requires the identification of developmental transactions relevant to BPD that occur during each developmental period, as well as period-specific deviations, suggesting family role reversals along with genetics as analytically identified contributors that may set the child on a path toward BPD.

Depressive and BPD features are typical of functional modes of submission or passivity, whereas dissocial PD features are modes of authoritarianism or intrusive behaviors. [55-66]

Several neuroscience data on parent-child RINs are an important etiological contributor to neurobiology that confirms the theories of BPD. [55-66]

Dependent personality disorder

Pervasive and excessive need to be taken care of, dependent on, and submissive to others. [55-66]

Interpersonal theory emphasizes the integral role of interpersonal relationships and experiences with others in broader aspects of psychosocial functioning, and is rooted in the assumption that all interpersonal interactions reflect attempts to establish and maintain self-esteem and avoid the anxiety caused by self-insecurity. [55-66] The principle of complementarity, or reciprocity, posits that an individual's interpersonal behaviors tend to initiate and elicit interpersonal responses from his or her partner that reinforce the individual's original behaviors or replicate

the attachment style experienced with the parent. [55-66] Dominance tends to elicit its reciprocal response of submissiveness (and vice versa, with submissiveness eliciting dominance), while coldness/hostility and warmth/friendliness tend to elicit their corresponding responses. [55-66]

The self-security deficit is noted by the avoidance and fear of assuming responsibilities, especially material and emotional ones. The deficit of Agency (sense of responsibility) and judgment (sense of fear of punishment) and fear (sense of harming the other), are determining clinical neuromarkers, together with the harmful attachment relationship. The second opinion is present all the time, making it difficult to take the initiative in isolation.

In cases that are associated with the pathological attachment relationship, abuse, aggression may occur, and the disadvantaged partner is unable to free themselves spontaneously. [55-66]

Narcissistic Personality Disorder

Patients with narcissistic PD are characterized by a generalized pattern of grandiosity, need for adulation, attention and empathy deficit and egocentrism.[66-70]

They have difficulty regulating self-esteem and, therefore, need praise and affiliations with special people or institutions.They also tend to devalue other people so that they can maintain a sense of superiority. [66-70] They have a habit of overestimating their abilities and exaggerating their accomplishments. This overestimation of their own worth and accomplishments often involves an underestimation of the worth and accomplishments of others. [66-70]

They are arrogant, and exploit others to achieve their own goals. [66-70]

They are sensitive and upset by criticism from others and by failure, which makes them feel humiliated and defeated. They may respond with anger or contempt, or they may retaliate violently. [66-70]

Avoidant Personality Disorder

Eikenaes et al., compared attachment styles in patients with avoidant PD and social phobia (SP). The group with APD had greater attachment-related anxiety than the group with SP, and the anxiety was more pronounced due to fear of abandonment. Fear of abandonment may play an important role in the pathology of APD. [66-70]

Weinbrecht et al., qualify that avoidant PD is being neglected and that more research specifically on symptoms and their treatment is needed, and that components of avoidant symptoms are evidenced by amygdala mechanisms of emotional survival. [66-70]

Lampe L. described the overlap between avoidant PD and social anxiety, and demonstrated key features such as a rigid and negative self-concept, a view of rejection, devaluation, and a sense of not fitting in socially that dates back to early childhood.

Frederick et al., when comparing avoidant PD and Social Anxiety Disorder,

showed from an interpersonal perspective that the relationship between the diagnostic groups is well described by the severity continuum hypothesis, with similar interpersonal problems related to Non-Assertiveness and lower levels of general interpersonal stress in the SAD group compared to both groups. [66-70]

Antisocial Personality Disorder

Antisocial Personality Disorder (ASPD) is the most common psychiatric disorder among formerly incarcerated individuals. A systematic review found that ASPD is present in approximately 47% of currently incarcerated individuals. And in some studies, the prevalence is even higher, reaching 78%. [66-70]

Furthermore, although it is estimated that almost half of currently incarcerated individuals suffer from ASPD, the disorder is much less common in the general population, where lifetime prevalence rates range from 3% to 5%. [66-70]

According to the DSM-V, the classic symptoms of ASPD are disrespect or disregard for social norms, lack of remorse, disregard for and violation of the rights of others, consistent irresponsibility, recklessness in self-safety and the lives of others, aggressiveness, impulsivity, fraudulent manipulation, with interests for gain. [66-70]

Other mptoms of ASPD correspond to behaviors related to difficulties in reintegration, including a "repeated failure to sustain consistent work or honor financial obligations." [66-70]

Habits of justifying or rationalizing one's behavior, blaming the victim for being foolish or helpless, and being indifferent to the exploitative and harmful effects of one's actions on others are still common. [66-70]

Neurological dysfunction of the neural systems of agency, sense, and judgment are pathological, since the individual's system of health ethics (what is good or bad for health) originates in the ONCs complex, which still presents a severe intrusive system. [66-70]

We observe the predominance of the secondary survival system in sociopathic types and of severe emotional survival in psychopathies, but they may be active in both. [66-70]

Psychopathy

In psychopathy, the patient presents response modulation dysfunction, and their attention plays a crucial role in moderating fear and self-regulation deficits. [66-70]

Response modulation involves the "temporary suspension of a dominant response set and produces a rapid, simultaneous shift in the organization's attention and implementation of the response in their interest, playing the victim. [66-70]

In coherent response modulation dysfunction, the patient ignores, does not accept, avoids, or retaliates, and manipulates people or systems to their advantage.

Contextual information helps in the diagnosis. Psychopaths are arrogant and consider themselves superior, despise the world and the people around them, and

exhibit antisocial behavior. They display hatred when they are deceived or defeated, especially when their own grandiosity and ego are used against them. [66-70]

Psychopaths do not feel anxiety, and do not present empathy, a sense of premonition, or consequences in the future. [66-70] They abuse with pathological "manipulative truths." Unconsciously, they always leave clues or traces in pathological crimes. [66-70]

The term "psychopathy" is used for dissocial personality disorders when they manifest a specific set of traits. [66-70]

Psychopaths are described in interpersonal terms as grandiose and self-centered, showing an exaggerated self-esteem and directing blame to others for their omissions or irresponsible behavior. It is common for them to lie and attempt to take advantage. [66-70]

They have an exaggeratedly pathological egocentrism, superficial, theatrical and false emotions, poor or no control of impulsivity, low tolerance for frustration, low threshold for releasing aggression, irresponsibility, lack of empathy with other human beings, absence of feelings of remorse and guilt regarding their behavior. [66-70]

The psychopath not only transgresses the rules, but ignores them, considering them obstacles that must be overcome in achieving their ambitions. The norm does not arouse in the psychopath the same inhibition that it produces in most people.

The psychopath's particular relationship with other human beings always occurs within the framework of ethical alterations. For the psychopath, the other is "a thing", just another work tool, an object of manipulation.

Furthermore, meta-analytic and other empirical findings have suggested strong associations between boldness and happiness, psychological adjustment, self-esteem, self-regulation, and immunity to stress.

Such findings have prompted criticism of the viability of boldness as a necessary and sufficient component in the operationalization of psychopathy, as it reflects its interpretations of descriptions that low levels of anxiety and fearlessness are predominant traits in the disorder. In contrast to some of the aforementioned research, other meta-analytic work has found moderate associations between boldness and measures of psychopathy based on the Non-Psychopathy Checklist. [66-70] Any association between boldness and adaptive outcomes raises significant construct validity concerns (i.e., boldness measures may be capturing personality traits of self-confidence and self-efficacy that are not fully related to psychopathy). [66-70]

Currently accepted definitions of psychopathy as a personality disorder present neurological dysfunctions that comprise in part objective (neurological) to subjective (psychological) behavioral deficits such as core deficits in interpersonal (manipulative, grandiosity), affective (callousness, lack of remorse), and behavioral (impulsivity, irresponsibility) domains. [66-70] Factor analyses of psychopathy scales consistently suggest that psychopathy is multifaceted, and there are two higher-order factors that are moderately

correlated and show similar patterns of relationships with external correlates of psychopathy instruments. [66-70]

The affective-interpersonal dimension factor reflects core personality traits of traditional conceptions of psychopathy, such as empathy, lack of remorse, and manipulation. [66-70]

And the impulsive-irresponsible dimension factor captures socially deviant and impulsive behavior indicative of an antisocial lifestyle. 66-70 The affective-interpersonal dimension is more related to low fear, narcissism, and social dominance, and previous research using psychopathy scales has suggested evidence of fear-avoidance and boldness behavior. [66-70] The impulsive-irresponsible dimension is more related to psychiatric indicators of externalizing problems, such as antisocial behavior and severe addictions, in PD with greater neuroticism and lack of restraint, impulsivity, or low conscientiousness. [66-70]

According to the Hare Psychopathy Scale, starting from a seductive and manipulative way. From an emotional point of view, they tend to be insincere and superficial, lacking empathy and showing a clear emotional coldness, showing a total or partial lack of guilt or remorse when they offend or attack others. [66-70]

Regarding the behaviors they display, they are described as imprudent, impulsive and fearless, seeking out novelty and taking thoughtless risks. [66-70]

One of the typical aspects they demonstrate is the lack of concern about the possibility of punishment, of suffering physical injuries or of being the target of sanctions with social repercussions. [66-70]

Psychopathic traits increase the likelihood of engaging in criminal behavior and substance abuse, although these traits can also be observed in the so-called normal population.[66-70]

Some longitudinal studies have concluded that, in a very specific group of individuals, this construct tends to remain relatively stable in the transition from adolescence to adulthood. As in adults, young people with psychopathic traits tend to engage in more severe and diverse antisocial behaviors than would appear at an earlier age. [66-70]

The neural bases of these cognitive functions have been located in circuits connected to the striatum; however, the amygdala and orbitofrontal cortex are responsible for processing rewards in economically and socially motivated emotions.

The social and cognitive mechanisms involved in cooperation are subserved by circuits projected by the striatum; a positive correlation was found between fiber connectivity to the social cognitive system (medial prefrontal cortex and amygdala). [66-70]

Cooperation is an essential behavioral trait that evolved to facilitate group life, and we hypothetically expect low activity in the Survival neuropersonality. According to a functional neuroimaging study and cooperation questionnaires, individuals with high scores were considered empathetic, tolerant, compassionate, supportive, fair, and ethical. People with low scores were considered self-

appreciative, egocentric, intolerant, critical, vindictive, and opportunistic. [66-70]

Sociopathy

There are endless discussions regarding the differences between the concepts and definitions of "psychopathy" and "sociopathy". 66-70 However, both refer to individuals with antisocial personality disorder, and the difference basically lies in the origin of the disorder. [66-70] Just as sociologists, crime experts and some psychologists believe that the disorder, when originated from the social environment itself, is called sociopathy. [66-70]

The characteristics of sociopaths mainly include disregard for social obligations and the inability to consider other people's feelings, evidencing pathological selfishness, anosognosia, and alexithymia. [66-70]

Individuals with sociopathic characteristics have adapted to living in a family environment with antisocial acts, such as an environment with low socioeconomic status, dysfunctional parenting and authoritarian parents. The psychopath consists of a combination of biological, genetic and socio-environmental factors. [66-70]

Such reasoning believes that psychopaths are born with basic characteristics such as impulsivity and lack of fear and premonition, which increases the chance of engaging in risky and dangerous behavior, often resulting in antisocial attitudes, since they are unable to establish themselves correctly within social norms, as they have an aversion to rules, which can often be a consequence of very authoritarian parents (neuroinversion), or parents lacking authority. [66-70]

Sociopaths have a milder temperament than psychopaths. They still defend the culture of normopathy, and the "adaptive characteristics associated with psychopathy" can "mask" the impairment. [66-70] Individuals who disrespect and violate the rights of others, not conforming to social norms. They are liars, deceivers and impulsive, always seeking to gain advantages over others. [66-70] They are irritable, irresponsible, and completely lacking in remorse, even if they claim to be, once again trying to take advantage. [66-70] They may establish superficial emotional relationships, but are not capable of maintaining deeper, more lasting bonds. [66-70] The dysfunctional construct of boldness has been theoretically controversial, due to its limited convergent associations with other measures of psychopathy. [66-70]

There are few meta-analytic associations and low significance of outcomes linked to psychopathy, general criminality, and violence, and low incremental predictive validity (above and beyond meanness and disinhibition) for antisocial outcomes. [66-70]

Eslinger and Damasio coined the term "acquired sociopathy" to describe the personality changes of a controller in a home construction company who underwent a lasting personality change after surgical removal of an olfactory meningioma. [66-70]

His normal cognitive performance

contrasted with his severe loss of tact and social behavior, which culminated in bankruptcy and abandonment by his wife and friends. [66-70]

In contrast to individuals with developmental psychopathy, who "never learn socially acceptable patterns of behavior," he learned and engaged in such patterns for most of his life; however, after his brain injury, he failed to behave accordingly in real-life situations. [66-70] With renewed interest in antisocial personality changes due to damage to the vm PFC, investigators have documented the emergence of acquired sociopathy in lesions outside the vm PFC, specifically, in the dorsolateral prefrontal cortex, rostral basal forebrain, ventromedial hypothalamus, anterior temporal lobes, anterior cingulate, medial thalamus, and basal ganglia. [66–70]

In most cases, behavioral changes were embedded in a web of more elementary symptoms, such as drowsiness and hyperphagia, which generally reflect damage to functionally heterogeneous neural systems. [66–70] Sociopathy was surprisingly absent in patients with even extensive bilateral prefrontal (PF) damage; yet in others, brain damage had little or no impact on socioоccupational status, even exerting a paradoxically beneficial effect in some. [66–70]

These discrepancies raise the possibility that, at least in some cases, the laterality of the hemispheric lesion may be decisive for the development of sociopathy; that is, damage to one cerebral hemisphere may be sufficient to produce sociopathy in some individuals previously considered normal. [66–70]

Despite the aforementioned clues about a possible asymmetric representation of acquired sociopathy in the cerebral hemispheres, few researchers have pursued this line of investigation. [66-70] The most consistent studies on lesion laterality and acquired sociopathy were carried out by *Tranel et al*., who also investigated a possible interaction between lesion laterality and gender.

Tranel et al., found that acquired sociopathy in a man resulted from damage to the right vm PFC, while in a woman it was caused by damage to the left vm PFC. [66-70]

The anterior temporal lobes encompass a collection of neural structures that support profuse bidirectional connections with the ventromedial, orbitofrontal, and insular cortices, as well as the amygdala. As in the vmPFC cases, sociopathy was associated with left lobectomy in a woman and right lobectomy in a man. Thus, in addition to the laterality of the lesion, studies by *Tranel et al.* imply that gender should also be considered critical to the production of acquired sociopathy. [66-70] Gender asymmetry has been supported by studies that have consistently found higher rates of antisocial behavior among men relative to women. This "gender gap," which is already noticeable at a young age, remains stable from childhood to adulthood (with the exception of a discrete period limited to adolescence), and has been documented across different cultures. [66-70]

Although the lifetime ratio of

antisocial behavior is 10:1, research has also shown that boys and girls with persistent antisocial behavior are identical in terms of poor discipline, family adversity, pattern of cognitive deficits, undercontrolled temperament, hyperactivity and peer rejection. Evidence indicates that men are referred more frequently than women for psychiatric treatment and the justice system because of differences that are intrinsic to gender, the cultural milieu playing an adjunctive role in increasing or containing the overt expression of antisocial acts .[66-70] A representative example of the interaction between gender biology and culture was provided by *Fumagalli et al.,* who studied the performance of 100 right-handed adults (50 men and 50 women) on a moral judgment task; Volunteers were classified into Catholics and non-Catholics according to their declared religious belief systems. [66-70]

They found that only gender predicted the type of moral judgments by men and women, men producing a significantly greater proportion of utilitarian responses than women, religious belief and education played no role in differentiating styles of moral decisions of men and women. [66-70] The authors concluded that cultural factors exerted little or no influence on moral judgments, suggesting that the significant factor was gender-specific differences in neural organization.[66-70] . A study on brain correlates of procedural justice judgments, the overall volume of brain activations in women was asymmetric (total volume of activations = 7,449 mm3) with a leftward preponderance (left hemisphere activations exceeding right hemisphere activations = 1,698 mm3). [66-70]

In recent studies, CSP is related to the interpersonal/affective characteristic of psychopathy, showing that the emotional detachment factor of psychopathy is related to the lack of early maternal and paternal care, and thus gives rise to the hypothesis that psychopathy and antisocial personality throughout life have a basis in neurodevelopmental abnormality in the limbic system (neuroadaptation of fear defense). [66-70] In their etiologies, psychopathic and antisocial behaviors occur at the beginning of maternal deprivation during the bond and attachment development phase.

Lateral septal nuclei (a region rich in oxytocin receptor activity) have been demonstrated in social attachment and synchrony behaviors in mammals. Disruption of the septal system can consequently result in insufficient synchrony among caregivers (even in the absence of maternal deprivation), resulting in antisocial, affectionless, psychopathic behavior. [66-70] The presence of a CSP was more related to the aggressive/life course component of antisocial personality disorder than to deceptive and irresponsible features, as a fused septum pellucidum is expected for normality, whereas a cavum full offluid within both leaflets of the septum pellucidum is indicative of antisocial personality disorder. [66–70]

The septum is critically involved in the regulation of aggression. Septal neurostimulation in a wide range of animals inhibits predatory aggression, whereas lesions of the septum result in

increased aggression and disinhibited behavior. [66–70]

CSP has been associated with aggressive features of antisocial personality disorder but not with nonaggressive features, indicating particular relevance of septal disruption to aggression in humans. [66–70] It is hypothesized that neural maldevelopment of the septum results in increased antisocial, aggressive, and psychopathic behavior through impaired commitment and attachment and the lack of prosocial affiliative behavior in antisocial personality disorder, both of which have been linked to septal functioning.

All of these negative environmental events affect the developing brain, and some of them have been hypothesized to give rise to midline limbic maldevelopment that in turn results in CSP. [66–70]

One advance that the current CSP findings add to previous imaging findings is that because CSP is formed before the first 6 months of life, [66–70] Dysfunctional neurodevelopment precedes the onset of criminal careers and is therefore hypothesized to be less likely to be a product of psychosocial and lifestyle influences that may be confounded in other adult imaging studies. [66–70]

The fact that CSP is related to all of these antisocial constructs suggests that a neurodevelopmental basis for a broad spectrum of antisocial behaviors is shared by these overlapping constructs. [66–70] Factors other than those reflected by CSP must inevitably give rise to the more distinctive features of psychopathy, antisocial personality disorder, and crime. [66-70]

Such preschool programs are not expected to reduce the early development of CSP, but they are expected to improve limbic development, i.e., the neuroadaptation of defense, which is initially associated with family pairs. [66-70]

CSP is a heritable contribution with approximately 50% of the variation in adult antisocial behavior being heritable; genetic influences on limbic development in antisocial personality and psychopathy should also be considered alongside environmental influences. [66-70] The association between CSP and the spectrum of antisocial behaviors supports a hypothesis of neurodevelopment of antisocial personality disorder and psychopathy, in which the first set of neurodysfunctions (ONCs) is responsible for the milder spectrum of survival, and hypothetically associated with CSP, amplification or evolution to psychopathy/sociopathy arises, together with the subjective factors that add up. [66-70]

Objective

The objective is to assist in the initial structuring of psychopathologies and PD, through the organized clinical description of ONCs, associated with the findings of clinical data accumulated from neurobiology studies, as it generates greater understanding of PD, and to highlight promising windows of prevention, based on clinical neuromarkers.

With the expansion of knowledge about neurobiological origins and their

etiopathogenesis, the pre-diagnostic construction of ONCs presents an understanding of the initial development of PDs, since it is a microstructure of the personality that begins to develop in the mother's womb and depends on the IR with the biological parents.

Methodology

This is a mixed review with empirical and theoretical clinical synthesis produced with abstraction of clinical data faithful to practical reality, with scientific articles on functional neuroimaging of human behaviors, chosen for convenience of clinical abstraction. [66-70] It has a reflective and comprehensive character, which aims to contribute to a more refined characterization of the interpersonal relationships of PDs and dysfunctional parenting.[66-70]

Results

Through the quality and quantity of care, the absence of affective attention directly reflects on the development of children and adolescents, who may develop PD. In order to evaluate technically, in a systematic way, without generating embarrassment, the initial complex was constructed that evolves into several PD, which presents clinically with the meeting of biobehaviors, which are objective, and thus we have a clinical notion of this quality of care, without invading subjectivity.

Discussion

According to *Bach B et al.*, recent taxonomic changes in a dimensionally classification system based on ICD-11 and DSM-V of mental disorders, the alternative model of personality disorder includes the requirement of cutoffs to examine clinical results similar to those of hypertension.[60-73]

These new criteria that differentiate neuromaladjustments with the presence of neurodysfunctions, may affect the prevalence of PD where it is comorbid with mental state disorder. [60-73]

It is essential to: organize the cutoff for "pathology", understand whether psychopathology is related to mental state disorder, PD or both, and consider the clinical and real value of a disease. [60-73] According to *Newton-Howes G et al.*, there is overlap between personality pathology and all types of MD. However, the shift to a dimensional structure of personality pathology means that new methods for defining and measuring this comorbidity are needed. Thus, the microstructure of neuromaladaptive personality fits into all dimensions, in addition to generating new understandings and windows of prevention and treatment. [60-73]

Conclusion

For health professionals and professionals who work professionally in human conduct, such as Law, it is essential to understand how emotions and their brain systems, as they are highly neuromaladaptive due to an evolutionary perspective, and also present genetic specification, which can evolve with mutations during life, and produce lasting effects through epigenetic mechanisms.

An organization in Mental Health is

fundamental and mandatory, as issues of Neurodysfunctional Interpersonal Relationships are being a hidden cause of deviant behaviors in family, professional and social environments, in addition to combating crime, domestic violence and MT.

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