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CLINICAL NEUROSCIENCE: LIMITATION OF INTELLIGENCE BY MALADAPTIVE MECHANISMS

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ABSTRACT

Introduction: The COVID-19 epidemic has highlighted another silent epidemic of mental disorders (MD) that present common symptoms and are involved with addictions and mood disorders, such as depression, anxiety, social stress, and behavioral deviations. In addition to the chronic problem with mental health, it is possible that intellectual stagnation also occurs, according to the neuroscientist specialized in human intelligence and neurogenomics. ***Objective:*** The present study aimed to fill a gap by presenting a clinical organization based on the neurobiology of intelligence and pointing out a set of maladaptive mechanisms that limit human intelligence. ***Methods***: After reviewing the literature, we outline the theoretical implications and mechanisms of social predictive perception that limit human intelligence, with clinical and factual insights, indicating its social and mental health consequences. We used the 5-phase perception model, with a generative view of perception and second-person neuroscience, in a "Bayesian" computational method, which was guided by theoretical method and robust clinical empiricism, to explain the errors of perception that limit intelligence. ***Results:*** We outline theoretical implications related to empirical science and found that limitation intelligence by maladaptive mechanismsmay be limited since childhood ***Discussion:*** We carry out a discussion that expands the concept of limitation of intelligence with the functional and executive mechanisms of intelligence. ***Conclusion:***We notify into this concept an improvement in depth attention and correction of systems distorted in childhood through EAI, PTSD, mainly in the issue of evaluating real values, and not learned or conditioned one.

Keywords: Intelligence, Limitation, Conectomic neuro desadaptative, Emotional Intelligence, Neural Connectivity

1. Introduction

The COVID-19 epidemic has highlighted another silent epidemic of mental disorders (MD) that present common symptoms and are related to addictions and mood disorders, such as depression, anxiety, social stress, and behavioral deviations. In addition to the chronic problem with mental health, it is also possible that intellectual stagnation occurs, according to the neuroscientist specialized in human intelligence and neurogenomics, Fabiano Agrela:

´´To enter the "well of ideas'—as" I like to call it—it is necessary to understand that its depth is directly related to the level of intelligence and/or knowledge of the person exploring it. The depth that can be reached depends on these factors, revealing how much it is possible to delve into the essence of ideas. Individuals with high intelligence are able to navigate the subjectivity of ideas, even without prior mastery of the topic, connecting dots through dialogue and logical reasoning. Those with extensive knowledge access memories and information that help to form connections and interpret the idea in a more concrete way. Thus, the depth of the "well" reflects both the intelligence and accumulated knowledge of each person. Furthermore, it is possible to be led into this dive by someone more intellectually capable. Living with highly intelligent and well-informed people allows us to be pulled into this well, expanding our ability to think, connect ideas, and make decisions. The richer and more well-elaborated these ideas are, the better the paths chosen will be. "

Fabiano Agrela's text can be considered a psychological, philosophical, or psychoanalytic theory for many, but it addresses the reason why great thinkers, as well as many individuals, acquire real value from some type of suffering experienced and how this can be associated with intelligence. [7-10]

According to Engle, Efklides, Fernandez-Duque et al., some important processes are interrelated, such as cognitive control mechanisms (CC) and reasoning mechanisms, working memory (WM), metacognitive skills, and executive functions (EF), and thus characterize a fundamental stage of attention that is related to intelligence. [3]

Individuals with high emotional intelligence (EI) in the general population are characterized by an attention bias in relation to emotional information.[5-7]

Duan et al. (2023) evaluated children individually in three information processing tasks: an inspection time task, a choice reaction time task, and an abstract matching task, and showed that IG children showed superiority in all three tasks, presenting shorter reaction times and actions with greater precision. [4]

The term intellectually gifted (IG) refers to individuals with high intelligence, which is classically measured by IQ, which must present a score above 140. Assessments are focused on skills such as logical reasoning, working memory, verbal skills, mathematical calculations, and problem solving. [7-10]

However, cognition is greatly influenced by continuous fluctuations in arousal, since for an individual to perform well, it is necessary to increase their level of arousal. If they are tired or stressed, their performance decreases, or it may be completely displaced towards value

orientation, such as a psychopath who is intelligent to harm people, with their value being strictly emotional, such as large monetary scams in which intelligence has been displaced towards monetary value. [5-7]

2. Objective

The present study aimed to fill a gap, presenting a clinical organization based on the neurobiology of intelligence, and to point out a set of maladaptive mechanisms that limit human intelligence.

3. Methods

To systematically search the literature on the neurobiological basis of human perception, we searched the PubMed and Web of Science digital libraries using the terms "error prediction," "perception," "attention," "synchrony oxytocin," and "inhibitory control processing.".

We reviewed neuroscience articles selected for convenience, presenting experimental findings with clinical data from functional neuroimaging exams, and presenting mechanisms that shape the perception and evaluation of people, behavior, and objects or socially relevant information.

Subsequently, we outlined the theoretical and mechanistic implications of social predictive perception that limit human intelligence, which produces a state of Limitation of intelligence with clinical and factual insights indicating its social and mental health consequences.

Finally, we used the 5-phase perception model, with a generative view of perception and second-person neuroscience, in a Bayesian computational method, which was guided by theoretical method and robust clinical empiricism, to explain the perceptual errors that limit intelligence.

4. Results

4.1 Adverse Childhood Emotions

The term Adverse Childhood Emotions(ACEs) refers to some of the sources of stress to which children can be exposed, including child maltreatment, interparental violence, and fighting. It is now well established that children who have experienced ACEs are at greater risk of unhealthy behaviors and mental and physical illnesses, including cancer, cardiovascular, and immuno-inflammatory diseases. In addition to developing harmful disorders of health and well-being, they are at greater risk of engaging in harmful behaviors such as addictions, antisocial behaviors, abusive relationships, and, most importantly, perpetrating the interpersonal violence that they experienced in adulthood. [74–81]

In fMRI studies, the hallmarks of early brain development are neurogenesis and axonal growth before birth, as synaptic connections are rapidly established after birth with consolidation. Cellular loss and synaptic pruning occur, as well as increased white matter due to improved connectivity through myelination. [74–81]

Changes in synaptic connectivity and axonal myelination are among the major microscopic processes in postnatal development. Changes in cortical thickness and white matter volume are the traditional fMRI markers of gross development. [74–81] The

ventromedial prefrontal cortex is involved in social reasoning and decision-making, the amygdala in social judgment of faces, the right somatosensory cortex in empathy and simulation, and the insula in autonomic response. [74-81]

The "higher-order" cognitive processes that will be critical for successful adult independence, such as planning, problem-solving, and working memory. [74-81] Random assignment of individuals with different genetic polymorphisms has provided evidence that genes interact with the quality of parenting to influence changes in behavior during early life. [74-81]

Parenting behaviors are directly linked in the longitudinal, bidirectional relationships between maternal depression and child internalizing symptoms (depression and anxiety). [74-81]

Studies of the bidirectional relationship between child and maternal internalizing psychopathology are partially explained by the greater psychological aggression of depressed mothers toward their children. 74-81

Altered brain architecture in response to toxic stress partly explains the associations between ACEs as an engram and impulse control, emotion regulation, and other cognitive challenges. [74-81]

There is increased aberrant activity in the amygdala, especially in the centromedial nucleus and insula, during emotional processing in children and adults who have been exposed to ACEs. [74-81]

Several meta-analyses have demonstrated consistent functional changes in the amygdala, hippocampus/parahippocampal gyrus, insula, and DLPFC regions. 74-81

The hippocampus plays a key role in neurobiological stress systems, and several studies have demonstrated positive associations between stress, mental illness, and hippocampal morphology. [74-81]

The reward-based learning mechanism predicts that the simultaneous activation of striatal neurons and the activity of the reward-associated dopaminergic neuron is essential for reinforcement learning. [74-81]

Furthermore, several studies show that ACEs directly affect neurological, hormonal, and immunological development. Thus, ACEs are associated with increased biomarkers of inflammation and shortened telomeres, which is consistent with direct effects on chronic diseases such as cancer, cardiovascular disease, and respiratory disease. [74-81]

A clinical history of childhood adversity should be considered in the differential diagnosis of developmental delay, dyslexia, dermatitis, asthma, recurrent infections, somatization, and sleep disorders, but also for psychological and behavioral functioning similar to ASD and ADHD. [74-81]

The stress system coordinates the body's responses to threats and opportunities, mobilizing physiological and psychological systems to respond to fluctuating environmental conditions and maintain homeostasis. [74-81]

Howland MA et al.

demonstrated that the stress response system can be calibrated to aggressive pre- and postnatal environments and subsequently recalibrated when RINs shift to support (or vice versa), highlighting its potential as a multilevel and multisystem mechanism, whether positive or negative in relation to resilience. [74-81] The first period of neurodevelopment (3 years) is the main moment that will define the regulation of several emotions, mainly fear, self-security, synchronicity of family relationships, emotional regulation control, and inhibitory regulation control.[74-81] In addition to the potential psychological trauma due to the circumstances of suffering a brain injury in childhood, which can significantly alter a child's developmental trajectory, particularly when it occurs during critical periods of brain development. [74-81]

The absence of affection and an insufficient bond in relation to the parents, the child's brain develops the dysfunctional maturation of the primitive emotion system SEEKING, which will condition a chronic and oscillatory hypodopaminergic functioning, along with other alterations in neurodevelopment. [74-81]

The hypodopaminergic state can be potentiated by genetic and epigenetic mechanisms that cause dysfunction of dopamine homeostasis and the Reward Deficiency Syndrome (RDS) described by Blum et al. [74-81]

Such states are responsible for behaviors and habits that generate dopamine (DA), the neurotransmitter of pleasure and well-being, and its basal absence causes displeasure, irritability, boredom, duality and indecision. [74-81]

Several findings and evidence of deleterious effects with lasting neuroplasticity of the HPA axis associated with child maltreatment and early-onset psychiatric disorder, in the development of different subregions of the child's hippocampus, in important periods of neuronal maturation. [74-81]

This experience-induced neuroplasticity is due to changes in the frequency and intensity of stimulation of the sensory systems (olfactory, somatosensory, and gustatory). [74–81]

Animal studies suggest that stress acts primarily to suppress neurogenesis of dentate gyrus granule neurons to cause dendrite remodeling in CA3. [74–81] Teicher et al. found associations between childhood maltreatment and reduced volume of the left hippocampal CA2–CA3 and CA4 dentate gyrus (CA4–DG), as well as the left subiculum and presubiculum, and right CA1 in a community sample of adults. [74–81] Early- and late-onset psychopathology were associated with developmental volumes of the right presubiculum and right cornusammonis 1 (CA1) brain regions, respectively. [74–81]

Altered development of the right CA1 may precede the development of psychopathology in early childhood and later in adolescence. [74–81]

Deficient or ineffective maternal care programs are an abnormal response to stress, encoding lasting molecular changes that may extend into the next generation. [74-81] Thus, brain immaturities combined with unique trauma processing may underlie the long-lasting effects of

abuse that remain largely hidden early in life. [74-81] Studies have demonstrated neuroendocrine and neuroimmune dysfunctions in patients with PTSD, early trauma, and ongoing threat, reflecting lower baseline cortisol levels, increased glucocorticoid receptor (GR) function, and a pre-, peri-, and post-trauma proinflammatory response. [74-81]

4.2 Posttraumatic Stress Disorder (PTSD)

Posttraumatic stress disorder (PTSD) is an acute psychiatric condition resulting from direct or indirect exposure shortly after exposure to severe trauma or moments that generate the memory, similar in the affected mechanism to losses and mourning. [74-81] The pathophysiological process is neuromaladaptive and debilitating, characterized by avoidance and disturbances of emotional oscillations, mainly related to fear, with distortions of negative thoughts, central and peripheral neuronal hyperexcitation, in subsequent months and even years. [74-81]

It involves situations of intrusive re-experiences of the traumatic event in the form of unwanted memories, nightmares, flashbacks, emotional and physical suffering, after exposure to traumatic reminders, and persistent symptoms of avoidance of stimuli related to the trauma. [74-81]

The risk of developing PTSD due to the influence of hereditary genes is up to 40%, and the risk after severe trauma is determined by several factors, such as the presence of ACEs. Several studies have shown that trauma-related disorders are associated with dysfunction of several biological systems, and the severity of PTSD symptoms has a cumulative effect on premature aging of the immune system, telomere length, gene expression dysfunctions, and long-lasting epigenetic alterations. 74–81 The fMRI studies have demonstrated hyperactivation of the amygdala during learning tasks, symptoms of anxiety, fear, stress, and emotional processing among individuals with PTSD and ACEs. In situations of threat, amygdala hyperreactivity plays a causal and/or chronic contributing factor. [74–81]

However, emerging literature in healthy samples shows greater test–retest reliability for amygdala habituation, the change over time in response to repeated stimuli, than for its reactivity to threat. fMRI studies have shown that in temporoparietal regions, associated with morphofunctional changes in amygdala reactivity and volumes, the occipital and frontal lobes subsequently develop atrophy in 30% of cases. [74-81] The central DMN may extend to the temporal subnetwork, as studies have shown that in individuals with PTSD, activities with increased intensity and time (chronic) presented accelerated atrophy of the temporal lobe. [74-81]

They occur due to dysfunctional couplings, which may be pathological, as in cases of family alienation, post-traumatic resilience, and bereavement, in which there is excessive amygdala coupling due to chronic reactivation of the neuronal system, or cases of slow decoupling (neurodysadaptation) of the tracts and fascicles, which are related to the pain mechanism, which are evidenced in some fMRI studies. [74-81]
Typical evidence is the finding after one year of stress exposure in patients with

post-war PTSD, who show decreased amygdala-dACC coupling. [74-81]

The brain regions most consistently associated with PTSD include the amygdala complex, hippocampus, insular cortex, and areas of the dorsolateral prefrontal cortex, including the anterior cingulate, dorsal striatum, thalamus, and sensory areas. These brain regions work together for the initial acquisition and subsequent expression of fear memory. [74-81]

There are numerous studies with evidence of ACE-related disorders + PTSD and long-lasting effects on the structure and function of neural stress regulatory circuits. [74-81] A consensus among trauma researchers, clinicians, and stakeholders is that trauma permanently alters the brain, also known as the neurotoxic stress theory. 74-81

According to Danese et al., impairments in cognitive functions (general intelligence, executive function, processing speed, memory, perceptual reasoning, and verbal comprehension) preceded childhood victimization but were associated with ACEs. [74-81]

4.3 Common NeuromaladaptiveConnectomics

A set of eight common neurodysfunctions (CNDs) is responsible for the neurobiological origin of dysfunctional coparenting and most personality disorders (PD), and the following are their clinical and neurobiological elements: [74-81]

CNDs is a neuromaladaptive clinical connectomics delimited by biobehaviors dependent on family interactions: (1) family synchrony deficit, (2) schematic behaviors, (3) neuromirroring, (4) dysfunctional neuropsychodynamics, (5) dopamine and reward system dysfunctions, (6) emotional dysregulation, (7) inhibitory control deficits, (8) social and family skills deficits. [74-81]

They present influences with disorders of genetic components (epigenetics, gene expression, polymorphisms, absence or presence of variant genes) and hormonal dysfunctions (cortisol, oxytocin, vasopressin, and melatonin dysfunctions) and immunological inflammation. [74-81]

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

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

3) Reward system dysfunction (RDS)

4) Navigation mode in the family environment, with predominance of activity in the hippocampus and amygdala system, due to the formation of engrams of increased interneuronal connectivity strength of these systems of the amygdala and limbic systems with predominance of their activity with the hippocampus, forming an exclusive navigation mode in the family environment. Chronic aberrant and sudden amygdala inputs occur, generating maladaptive states expressed as family schemas (Young) that produce neurodysfunctional interpersonal relationships (NIRs) such as misconduct, neglect, and psychological abuse among family members. The navigation mode with predominance of amygdala

systems and automatic behaviors generates neurocognitive states with Alexithymia: the inability to effectively self-observe (self-identify) emotional and affective awareness at the same time, and Secondary Anosognosia: the inability to effectively identify the emotional and affective state of another person. **5) Commissurectomy: Interhemisphericdisconnection of the ungual and arcuate fascicles,** secondary to toxic parental behaviors, mainly verbalizing, which causes secondary simultanagnosia (inability to identify more than 2 objects at the same time), causes cognitive inflexibility, decreased processing speed, and limits the extent of consciousness with dense intelligence. **6) Neuromirroring dysfunction of mirror neuron networks:** Occurs through the inversion or increase or decrease of the functional roles of the family system. The inversion of a mode has an important cumulative effect on the SEEKING system and the RDS. Replication of the experienced effect occurs, in an increased, decreased or inverted form of these functional modes of neuropsychodynamics, and involves the entire circuitry of attachment with neuromirroring.

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

8) Neuroenzymatic deficit of inhibitory control: impulsivity, behavioral deviation. 9) Neurological deficit of social skills (extradomiciliary): social isolation, preferences for habits without social contact, introversion, shyness. **10) Neuronal deficit of family skills: absence of mentalism and parental incapacity.**

11) Underdevelopment of the PFC: The PFC is 'hijacked' by the subcortical structures of the midbrain. It occurs due to a deficit in myelination secondary to stress. Myelination, which is involved in regulating processing speed, especially in pre-developmental periods.74-81

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. [74-81]

Genetic alterations involved in the limitation of intelligence indirectly:

1-(rs2254298, rs1042778) = reciprocity in social bonding (synchrony)

2- oxytocin receptor polymorphism rs53576 = decreases anxiety and fear regulation

3-OXT receptor gene (OXTR) or CD38 (cluster of differentiation 38), a gene involved in OXT release

4-methylation in serotonin transporter genes (SLC6A4) = depression and anxiety

5-brain-derived neurotrophic factor (BDNF) = depression and anxiety

6-glucocorticoid receptor (NR3C1) = depression and anxiety

7-FK506 binding protein (FKBP5) = depression and anxiety

8-DNA

Binding Protein Inhibitor ID-3 (ID3) = adverse events and depression

9-Tubulin Polymerization Promoting Protein (TPPP) = adverse events and depression

10- Glutamate Receptor, Ionotropic N-methyl- D-aspartate (NMDA) 1 (GRIN1) = adverse events and depression.

11- high-risk FKBP5 polymorphisms = stress and childhood abuse 12- increased methylation (NR3C1) : suicide with history of child abuse - low-risk CD38 alleles = increased parental care - OXT rs2740210 and rs4813627 = many sensitive to ACEs. - OXTR gene polymorphism rs53576 = emotional dysregulation, problematic attachment, PTSD symptoms, depression symptoms, lower levels of perceived social support, and internalizing behavioral problems.

13- COMT-Val/Met = hypodopaminergic trait and disorganization attachment style

14- DRD2 TaqA1 and DAT1 alleles 10 = avoidant attachment 15- DRD2 A1 allele = controlled eating if parents control themselves emotionally

16- dopamine receptor D2 gene (DRD2) = novelty seeking (emotions)

17- any A1 allele of the DRD2 gene = novelty seeking, if punished

18- GABRA6 (Pro385Ser) = Mentalism and paternal rejection

19- the short alleles (two or five repeats) of the DRD4 gene = novelty seeking 20-TaqA1 = brain preference for known or experienced attachment 21- DNA methylation in the OPRM1 gene = , stress regulation, motivation, and potentially all subtypes of RDS.

22-MAOA-H allele (high activity) = high risk of committing serious, recidivist, impulsive and violent crimes and physical abuse in childhood 23- haplotypes rs1360780, rs9296158, rs3800373 and rs9470080 =PTSD with history of rape, sexual violence

24- polymorphism of the brain BDNF gene Val66Met (rs6265) = attention deficit, depression associated with child abuse

25- nuclear receptors NR4A: Nur77 (NR4A1), Nurr1 (NR4A2) and Nor1 (NR4A3) = dysregulation of the stress response

26- polymorphisms in OXTR and AVPR1A = individual variation in social behavior and cognition

27- SNP OXT rs139832701 and rs11131147 = depression and anxiety related to parental care in childhood

28- GRIN2B = childhood adversity and depression

29-Deletion CD38= social amnesia

4.4 Intelligence

The psychological functions are superior to the processes of memory, attention, and voluntary recall; active memorization; imagination; the ability to plan; establishing relationships; intentional action; the development of will; conceptual elaboration; the use of language; symbolic representation of purposeful actions; deductive reasoning; abstract thinking; and effective action.[7-12]

These psychological activities are considered "higher" because they differ from more elementary mechanisms of biological origin present in all human beings (common in humans), such as reflex actions, automated reactions, and instincts.[13-15] Modern neuroscience thinking on intelligence, which shows the relationship between working memory capacity and fluid intelligence, generates a convincing clinical mechanism to explain the differences in intelligence, one being fluid and the other crystallized.[16-19]

Fluid capacity has the character of a purely general capacity to discriminate and perceive relationships between any foundations, new or old. It increases until adolescence and then slowly decreases.[20-23]

Crystallized ability consists of long-established discriminatory habits in a specific domain, originally through the operation of fluid ability, but no longer requiring insightful perception for their successful operation.[24-29]

4.4.1 Fluid Intelligence

Tsukahara et al. argue that working memory capacity and fluid intelligence are related by task utilization, and thus when measuring these constructs, they require executive control of attention to organize processing around task demands. Executive control of attention directs thoughts and behaviors in a goal-relevant manner, which is the basis of synthesis power and pragmatism.[29-31]

Controlling attention determines how effectively one deals with complex and changing environments. That is, during situations of distraction, interference, and stress, being able to attend to useful information and tune out information that is no longer relevant is critical.[29-31]

Thus, "attentional control" (or executive control of attention) overlaps with terms such as executive functions, inhibition, cognitive control, binding, and central executive.[29-31]

However, there are cognitive states that present neuromaladaptive dysfunctions that impair attentional control, especially when they are in situations of automatism, and are related to crystallized or dense intelligence, but mainly with errors of perception or cognitive penetrability.[32-33]

Several theories and debates about human intelligence have presented theories such as single regions or overlapping specific networks, but with the discoveries of neural hodology and the reconnection of interhemispheric neurons, they can help in a better understanding.[32-33]

The emphasis on multi-network connectivity of the whole brain and the inclusion of dysfunctions of weak connections in some regions and intense in others motivate the prediction that intelligence will depend on functional connections that are globally distributed throughout the connectome.[32-33]

Mental state, attention, and calculation are types of crystallized intelligence, which is formed through the accumulation of knowledge and experience. As people age and gain new knowledge and understanding,

crystallized intelligence tends to increase first and decrease more slowly.[32-33]

As a channel for acquiring knowledge and skills, education is expected to have a substantial effect on crystallized abilities. They described two pathways through which genes could influence social and health outcomes.[32-33]

Genes contribute to both educational attainment and cognition independently due to underlying biological and latent genetic mechanisms. [32-33]

A person's genetics can influence brain development to affect non-cognitive self-control, interpersonal skills, preferences, and behaviors, leading to differences in both educational attainment and cognition. Intelligence tests test at all ages the combined results of fluid and crystallized abilities, but in childhood the former is predominant, while in adulthood, due to the recession of fluid ability, peak performance is determined by crystallized abilities.[32-33]

´´Hebb (1941c, 1942) independently and very clearly stated what constitutes two-thirds of the current theory, for he says that "intellectual power may be necessary for the first appearance of the qualitatively superior response, but not necessarily for its persistence" (Hebb, 1942), and "in any test performance there are two factors involved, the relative importance of which varies with the test: one factor is the enduring changes in perceptual organization and behavior induced by the first factor during the period of growth."

Modern thinking about intelligence is strongly influenced by the discovery that individual differences in working memory capacity are highly correlated with fluid intelligence.[12-16]

Episodic memory—the ability to encode and retrieve personally experienced events that occurred at a specific place and time—involves the ability to think and reason abstractly; it is a type of fluid intelligence. [12-16]

Mental state, attention, and calculation make up the mechanisms of thick intelligence, which is formed through the accumulation of knowledge and experience.[12-16]

4.4.2 Attention

In IG individuals, the ability to switch between different attentional focuses simultaneously, as well as their depth perception, is regulated by several shared attentional networks in the states of alertness, orienting, and executive control. [14-19] The Posner and Petersen framework identified three attentional networks that are functionally and anatomically distinct: alertness, orienting, and executive control. Alertness allows an optimal state of vigilance to be applied and maintained during a task. [14-19]

The alerting network has two basic cognitive mechanisms: tonic alerting, which is the ability to self-control the alert state without any external cue, and phasic alerting, which is the ability to increase an individual's reaction after an external cue for a short period of time.[20-23]

The precueing mechanism sets the values of some parameters of the transformation rules in feedforward processing and thus defines the

parameters that highlight some information in the visual scene, increasing the activation of the neurons that encode this information[20-23].

Regions and Sub-regions Involved in Care:

Dorsolateral (dlPFC): essential for executive control, such as planning and redirecting attention.

Ventromedial (vmPFC): related to emotional processing and controlling irrelevant distractions.

Anterior Cingulate (ACC): acts as a "supervisor" that detects conflicts between competing stimuli, allowing attention to be redirected.

Salience Network: Includes structures such as the anterior insula and anterior cingulate, which detect environmental changes and help prioritize the most relevant stimuli.This network is more active in people with greater cognitive flexibility.

Attention Network: Dorsal Attention Network: Involves areas such as the superior parietal lobule and the premotor cortex, responsible for maintaining attention on specific tasks.

Ventral Attention Network: Includes the inferior parietal cortex and the temporal lobe, which help redirect attention to new stimuli.

Amygdala and Hippocampus: These regions contribute to filtering emotional stimuli and prioritizing those that require immediate attention**.**

4.1.4 Working Memory

Working memory consists of multiple stores of domain-specific information and a central executive, which oversees and coordinates attention so that only relevant information occupies working memory.[25-28]

It is also considered a workspace in which representations are actively maintained in consciousness and manipulated to influence information processing. Working memory in primates, the lateral frontal cortex already well known in the spatial delayed response task, presented cells that exhibited activity in delay periods in response to stimuli in various sensory modalities.[25-28]

4.4.5 Perception

Studies have shown that the development of social conformity is equivalent to the principles of reinforcement learning and that adaptations to the group norm are linked to so-called prediction error signals in the ventral striatum in adapting individuals. [1-6]

Based on a perceptual model focusing on elicitation mechanisms, we propose different types of cognitive penetration depending on the level of processing at which penetration occurs and depending on where the penetrating influence comes from. [1-6] The hierarchical order of sensory integration in the human brain can be seen as a Bayesian model, with five phases of perception that were constructed with guidance from a "theory-based model" and clinical empiricism: Phase 1: Preperception, Phase 2: Registration perception, Phase 3: Elaborative perception, Phase 4: Self-observation and CC, and Phase 5: Execution perception. [1-6]

Orientation is the ability to prioritize the processing of any stimulus by selecting salient information in a sensory input. Executive control is the ability to voluntarily focus attention to resolve a simple conflict.

Intelligence
↓
Working Memory
↓↓↓↓
Phase 1 □ Phase 2 □ Phase 3 □ Phase 4 □ Phase 5
Pre-Perception Perception Self-Perception Perception Elaborative Recording Executive Observation
↓↓↓↓↓
Cognitive Penetrability with positive or negative modulation
Phase1 Phase2 Phase 3 Phase4 Phase 5
↓↓↓↓↓
-Pre -Initial Vision -Reasoning -Intelligence -Cue Making -Identification -Emotional Reflection Decision -Synchronization -Encoding -Choice -Agency -Judgment -Covert Attention -Attention -Notion -Action -Processing -Understanding – Speech

4.5 Limiting Emotion with Rationalization

The information that the brain processes as emotional content is influenced by limbic and paralimbic structures, which are then sent to certain regions of the cerebral cortex and provoke an involuntary (automatic, sudden) response, whether it be a thought or a behavior.[34-39]

Autonomy is the voluntary decision-making process and depends on the function of the frontal or prefrontal cortex. In this context, the use of reason would be initiated medially by the action of the anterior cingulate cortex (executive attention), which has the function of focusing perceptual and cognitive attention, modulating the activity of the corresponding areas.[34-39]

The dorsolateral regions of the prefrontal cortex would be responsible for comparing new and old information. The final adjustment, taking into account the objectives of the individuals and the social contexts, is made by the ventromedial prefrontal cortex. [34-39]

The objective, intention, will, and motivation are guided by the individual's real value, in which changes due to the influence of previously learned stimulus-reward associations occur, the phenomenon of "value-driven attentional capture" (VDAC). The VDACs are representations of stimuli associated with reward and undergo plasticity in the sensory cortex, automatically capturing attention during the initial processing of any lived experience. [34-39]

Images certainly provoke, for the most part, activation of the occipital visual cortex (occipital gyrus and fusiform gyrus), but the amygdala also receives a substantial amount of stimuli from the temporal areas associated with vision, participating in the formation of memories through the hippocampal circuits or striatal circuits. [34-39] This fact results from the specialized role of the amygdala in processing visually relevant emotional cues, signaling fear and aversion, or other evidence. [34-39]

Activation of the amygdala interferes with the emission of alerts for threats arising from the perception obtained by the occipital cortex.[34-39]

Sensory impressions (vision, hearing, and other somatosensory information) converge through the OFC to the VMPFC, from where the synthesized information is

taken to the regions of the dorsomedial prefrontal cortex and inferolateral prefrontal cortex for decision-making.[34-39]

Lesions in the VMPFC cause impairment in the ability to make decisions, generally characterized by an inability to adopt behavioral strategies appropriate to the consequences of actions taken, leading to impulsivity.[34-39] The VMPFC and the OFC have an important relationship with the amygdala and both contribute to decision-making, although the mechanisms by which this occurs are distinct. [34-39]

These cortical regions receive inputs from the amygdala, which represent the motivational value of stimuli, integrating them and promoting an evaluation of the behavior to be performed.[34-39]

Although the amygdala does not establish a direct connection with the lateral prefrontal cortex, it communicates with the anterior cingulate cortex and the orbital cortex, which are involved in memory circuits, a process that the amygdala participates in the modulation of memory and in the integration of emotional and cognitive information, attributing an emotional charge to them, enabling the transformation of subjective experiences into objective emotional experiences (neurological predominance). [34-39]

According to these investigations, the medial PFC is involved in the association of the cognitive aspect with the emotional aspect, being responsible for the evaluation and/or cognitive interpretation of emotions. [34-39]

The results show that the cognitive processes are modulated by the ACC in the same hemisphere.[34-39]

The DLPFC participates in executive situations, such as coordination of concurrent cognitive processes and selective attention to information from relevant tasks.[34-39]

Another important structure in the integration of emotion/reason is the insula, which is activated during the induction of memories of moments experienced by an individual, which provoke a specific sensation, whether positive or negative.[34-39]

However, the insula is not activated when the same sensation is provoked in the same individual by a film, for example, showing that this region participates in the aspects of evaluation, experimentation, or expression of a deeper emotion.[34-39]

4.6.1 Value Encoding/Processing

A key aspect of contextual modulation is that information is encoded in a relative manner, with contextual inputs controlling the transformation between boosting input and boosting output, producing an encoding of input information dependent on the motivating or interesting spatiotemporal environment. [33-40]

Similar adaptive value coding occurs in the monkey anterior cingulate cortex and midbrain dopamine nuclei, similar to reward-related processing in the human brain.[33-40]

A key step in examining neural value coding is

distinguishing issues related to the representation of decision value from other forms of reward-related activity. Various neural responses may appear closely correlated with value but instead encode other forms of related information. [1]

Midbrain dopamine neurons reflect reward prediction error, a quantity that encodes the difference between expected and received reward value. [33-40]

Although value coding has been examined in several brain regions, including the orbitofrontal cortex, dorsolateral prefrontal cortex, and basal ganglia, we focus here specifically on value representation in the lateral intraparietal area. [33-40]

4.8 InteligenceLimitation

However, it is a common phenomenon, but it presents harmful components to human relationships and human health, which is silent and evidently limits human intelligence.[41-47]

The state of Limitation cognitive was identified through empirical observation, and after connection to the accumulated fMRI data, a neuromaladaptiveconnectomics was constructed that presents a pathophysiological and clinical causal. [41-47] This limitation is characterized by a deficit in emotional intelligence, a deficit in mentalism and empathy, a predominance of dense intelligence, cognitive penetrability, or perception errors, which are the main limiting factors, in addition to the displacement of the sense or orientation of the functional set of intelligence through the habituation of the VDAC, in which monetary and emotional values are the most evident and common. The limitation of recognition to single objects in simultanagnosia may suggest reduced ability to identify objects, but other alternative concepts suggest preserved object identification, in which patients can recognize single objects, coupled with impaired spatial attention, which restricts the number of objects that can be perceived.[41-47] In addition to inattention to whole objects, there is evidence that patients with simultanagnosia perceive individual objects in a fragmented manner, indicating another manifestation of damage to an object-based attention system.[41-47] According to Karnath et al. (2000), "local capture" demonstrates this mechanism, as patients identify the local components of an object but fail to see the global aspect of the object, even with unlimited presentation.[48-49] Poort et al. showed that distinct response suppression mechanisms are performed by inhibitory and excitatory neurons in the visual cortex, associated with top-down input models, and thus generate improved sensory processing due to learning and attention.[38-45]

The social brain in a neuromaladaptive state promotes dysfunctional inattention, with the aim of avoiding threats of emotional suffering, and indirectly generates neglect effects and low diligence processes, which will limit the activity of intelligence, as in the cases of dense intelligence. [38-45]

If gifted individuals with high EI are characterized by a preference for emotional information without negative cognitive penetrability, they should be

more "captured" by emotional stimuli compared to neutral ones.[38-45]

4.8.1 Value-driven intelligence

Through the 5-phase multimodal model of perception, the set of intelligence (working memory, deep attention, response inhibition) may appear to direct intelligence according to the VDAC, which justifies high intelligence in emotional values in individuals who perform behaviors motivated by emotion. [38-45]

Individuals with high work skills may have a shift in the entire set involved in executing intelligence towards monetary gains and business objectives, and the value initially captured may be directed solely towards monetary acquisition or satisfaction from the work effect, or both.[38-45]

However, it is possible that there are simultaneous activities of the emotional VDAC and monetary acquisition, which may generate errors of perception, limitation of intelligence, and produce irrational behaviors. [1]

Successful inhibition of responses to irrelevant stimuli (response inhibition) may depend on sustained attention to relevant information (attention) and working memory.47

According to fMRI studies, states of cognitive and behavioral automatisms, such as amygdala activity, whether due to the function of maintaining rationality or emotional defense inputs, simultaneously and mechanically, are states of alexithymia that, in emotional cases, have high intensity. [38–45]

However, it may also be that processing stimuli at two locations requires some expansion of the narrowed attentional window, which has a consequent reduction in the spatial resolution of attention as in simultanagnostic individuals, allowing binding errors to occur. [38–45]

One possibility is that with cortical fatigue, the attentional window closes completely. Another explanation is that information processing within the attentional window may be fatigued. [38–45]

Thus, visual stimuli outside the boundaries of the narrow spatial window of attention are subject to partial or complete failures of representation and thus diminish the executive capacity of intelligence.

Working memory is the central intrinsic regulator that orchestrates the phases of perception, as well as simultaneously stimulating adaptive processes such as the reward system and other types of memories. Working memory may also be oriented according to the central value obtained in attentional capture. [47]

4.9.2 Perceptual Errors by Monetary Acquisition Value

The brain processes three sources of motivation: extrinsic rewards, moral values, and image concerns.[37-43]

According to decision neuroscience, when choosing whether to accept or reject an offer that weighs two types of attributes (moral values and money), the brain assigns a value to each option and compares them, calculating their difference.[37-43]

Current models of predictive processing suggest that there may be many different brain states that give rise

to feelings of (dis)pleasure or arousal. Although default mode areas often show reduced overall activity during certain cognitive and attentional performance tasks, they show increased activity during social cognitive tasks.[37-43]

This framework has been applied successively in the field of value-based decision-making regarding various types of benefits (money) and costs (waiting for a long delay).37-43

Relevant cognitive processes include motivation, reward coding, action evaluation, and executive functions in the context of social interactions. The prefrontal cortex and striatum reflect individual differences in reward dependence.

Several studies on the neurobiology of addiction have shown that at the time of monetary acquisition, there is an intense spike in dopamine release, which can generate an experience of well-being. [37-43]

Several studies have shown that the amygdala and orbitofrontal cortex are responsible for processing economic, social, emotional, and motivational rewards. What we pay attention to is influenced by reward learning. Humans automatically attend to stimuli previously associated with reward and to stimuli that have been experienced and conditioned during visual search, even when it is disadvantageous in current situations.[37-43]

Several studies have shown that associative reward learning alters the brain's processing mechanisms of visual stimuli in the face of learned reward cues that are difficult to ignore. [37-43]

Studies using magnetoencephalography have investigated modulations by reward learning and have shown that VDAC is supported by learned value signals that modulate spatial selection throughout the visual and posterior parietal cortex, which can still occur in the absence of changes in visual processing in the cortex.1 Several neuroscientists have proven that value signals automatically guide attention to new association situations, which may or may not be beneficial, depending on their congruence with current goals. [37-43]

However, we draw attention to the fixation of VDAC on monetary acquisition through habituation and engrams, which simultaneously activates the reward system and traumatic emotions in childhood with dopaminergic release and subjectively generates greater survival security. This set of mechanisms, in addition to representing errors of perception, justifies several behaviors of exclusive interest in monetary gain and may hypothesize the pathogenesis of addiction to monetary acquisition. [37-43]

5. Discussion

The assessment of the presence of emotional intelligence by the presence of metacognition, mentalism, empathy, resilience, cognitive flexibility, and phases of perception that present less maladaptive interference, is assessed by the presence of alexithymia, anosognosia, history of Adverse Childhood Emotions (ACE), Post-Traumatic Stress Disorder (PTSD) in childhood, presence of family coping,

intrusive behaviors, cognitive rigidity, inability to correlate several pieces of knowledge simultaneously, absence of subjective creative process, which are all clinical neuromarkers of cause and effect, related to cognitive limitation in childhood, which prevents the development of fluid intelligence, and maintains dense intelligence.

In both the gifted individual and the average IQ, the first is due to neurobiological reasons, and the second is limited by the demands of capitalist savagery because the environment, stress, and social factors are not stimulating. On the contrary, they are limited, as their intelligence system may have a shift towards the real value that the individual is concerned about or seeks in practice.

The second presents the limitation of the absence of metacognition and emotional intelligence, which must be taught. Intelligence begins with the stimuli that are captured, or not captured, which processes the information.

1. Value-oriented attentional capture (VDAC), attention and capture of information, and processing with the captured data. This data may be limited by the selective capture of value-oriented information or due to the limitation of attention in its depth. In the processing that builds the notion or percept, the abstraction force of the real value captured and not captured interferes in this construction.

Let's assume that a profoundly gifted individual has 70% attentional capacity. And the gifted individual has 50% attentional capacity. And the average IQ individual has 50% capacity.

Next step 2: Working memory that regulates the processing of information processed by the attentional capture data. Working memory works with the received data, which were processed into a percept or notion.

Therefore, if the attentional system does not capture all the information correctly, the processing that forms the percept or notion will be limited. Just as processing errors can occur due to the Stroop effect, it will limit working memory that will work with the information substrates that it received.

Working memory is the executive conductor that is influenced by a greater conductor that is intelligence. Working memory can be interrupted by anticipating the perception phases with anticipatory judgment before reasoning, evaluation, and choice. The gifted individual has 80% capacity. Working memory without interference and with information received with less interference produces greater fluidity, working with all five stages of perception, which will lead to effective decision-making and will check the outcome of the decision with emotional intelligence preserved from agency. Individuals who do not present emotional intelligence and metacognition do not present the fourth stage of perception and thus present only 4 stages, with an absence of agency, which can be a mechanism that explains irrationality.

It is the one that makes the correlations and ends in the last instance, which is decision-making, creating, speaking, and judging. It also has limits. An average IQ is, obviously, less than 80%.

Processing speed measures this. It facilitates all stages of understanding and choice, but it can be limited by belief and when the whole set is shifted towards an interest (value).

So, at different times, these percentages will decrease, and thus intelligence, which is the functional set, will evidently not perform to its full potential. In a gifted individual, if attention was 80% and working memory 80% = intelligence 80%, but if in a moment of tiredness or perceptive errors, the most common of which are emotional, attention may decrease, since it is what provides tone and is dependent on hydration, nutrition, dopamine, and norepinephrine.

So, at a specific moment, attention drops to 40%, for example, and memory may remain at 80% = intelligence drops. 60%, for example. We use numerical percentages only to demonstrate the flexibility of each domain that makes up the entire fluidity of intelligence; that is, if an attentional window captures 3 pieces of information superficially, intelligence will result in a conclusion X. If the window captures 3 pieces of information in depth, it will result in a conclusion of greater perception, greater than X.

However, emotional intelligence is the great limiter of the quantity and depth of attention and, therefore, of intelligence.

The process of synthesis and creation needs an enhanced attentional window, and all processing steps cannot have interference in order to have what is missing to be seen or insight.

The perception processes need to be in sync and without interference in order to reach the maximum limit that an individual has. This involves attention and final judgment without judging emotionally and without judging by the learned rational.

Abstraction of the same object and value in different knowledge is an attention-switching skill associated with the assessment of the correct measure of the strength of the object's value.

By training executive attention through mindfulness, there is an expansion of attention and working memory, since neurologically these are cracked mechanisms, and which are impaired by the right parietotemporal exclusion in childhood associated with commissurotomy and the predominance of amygdala activity through chronic activation of family schemes.

Systemically, the barriers that block human intelligence can be divided into microsystem (neuromodulated complex/ONCs), mesosystem (communication between parents, teachings, and acquired beliefs), exosystems (occupational), and macrosystem (politics and society).

Finally, we conclude that individuals who go through severe emotional crises, who feel suffering in their skin, can break the cycle of orienting intelligence towards monetary or emotional value and acquire a better notion of moral values and suffering.

6. Conclusion

We integrated into this concept an improvement in depth attention and

correction of systems distorted in childhood through EAI, PTSD, mainly in the issue of evaluating real values, and not learned or conditioned ones, which can represent the state of limitation of Intelligence.

We demonstrated a functional and executive model of intelligence that controls working memory and depends on perception substrates and perception phases without cognitive penetrability or perception errors.

This work demonstrates functional morphology through clinical neuroscience, constructed by theoretical guidance and robust empiricism, but presents the limitation of the experimental method component, as it is a concept in the childhood phase but unprecedented.

7. Declaration of conflict of interest

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

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