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/ /	³ اسم الجامعة والدولة (للثالث) باللغتين العربية والإنجليزية	Formation of Human Emotions
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يهدف هذا البحث إلى دراسة عميقة للأسس البيولوجية والكيميائية العصبية التي تُشكل المشاعر الإنسانية، والتي تُعرف أيضًا بالسلوك الوجداني. فبدلًا من اعتبار المشاعر مجرد ردود أفعال عابرة، يسعى البحث إلى فهمها كقوى مُحرّكة تؤثر في إدراكنا للعالم وعملياتنا المعرفية وتفاعلاتنا الاجتماعية. ويستكشف البحث ما يُطلق عليه مجازًا "معادلة كيمياء السلوك الوجداني"، وهو مصطلح يُشير إلى فهم الآليات البيولوجية والكيميائية العصبية المُعقّدة التي تكمن وراء نشأة المشاعر وتعبيراتها المختلفة، مع التركيز على دور النواقل العصبية (مثل الدوبامين والسيروتونين)، والهرمونات (مثل الكورتيزول والأوكسيتوسين)، والمناطق الدماغية المُتخصّصة (مثل اللوزة والجهاز الحوفي وقشرة الفص الجبهي)، وكيفية تفاعلها معًا لتحديد طبيعة المشاعر الإنسانية وتنوعها.

كلمات مفتاحية: (السلوك، السيروتونين، الدوبامين، التفاعلات البيوكيميائية ، النواقل العصبية)

Abstract:

This research aims to conduct an in-depth study of the biological and neurochemical foundations that shape human emotions, also known as affective behavior. Rather than considering emotions as mere fleeting reactions, the research seeks to understand them as driving forces that influence our perception of the world, our cognitive processes, and our social interactions. The research explores what is metaphorically termed the "equation of affective behavior chemistry," a term referring to the understanding of the complex biological and neurochemical mechanisms underlying the. Emergence and expression of various emotions, with a focus on the role of neurotransmitters (such as dopamine and serotonin), hormones (such as cortisol and oxytocin), and specialized brain regions (such as the amygdala, the limbic system, and the prefrontal cortex), and how they Interact to determine the nature and diversity of human emotions.

Keywords: (Behavior, Serotonin, Dopamine, Biochemical Interactions, Neurotransmitters)



Introduction:

Emotions, or what is known as affective behavior, represent a fundamental dimension of the human experience. They are not merely transient reactions but driving forces that shape our perception of the world and profoundly influence our cognitive processes, from making simple daily decisions to forming complex judgments. They also determine the nature of our social interactions and relationships with others, coloring our life experiences with a wide spectrum of sensations. But what exactly are the biological and neurochemical mechanisms behind these driving forces? And how do these complex mechanisms interact to produce the vast diversity of human emotions? Based on this importance, this research aims to explore the "Equation of Affective Behavior Chemistry," a term that refers to the endeavor to understand the complex biological and neurochemical mechanisms underlying the emergence and various expressions of emotions. It is important to note that the term "equation" here is used metaphorically to describe these complex interactions, not as a precise mathematical equation. Can this complex human experience actually be reduced to neurochemical interactions? What are the limits of such reduction? And how can we better understand the complex interaction between biological and chemical factors on one hand, and life experiences and environmental influences on the other, in shaping affective behavior? The research will provide a detailed analysis of biochemical interactions in the nervous system, focusing on the role of neurotransmitters (such as dopamine, serotonin, norepinephrine, glutamate, and GABA), the impact of hormones (such as cortisol, oxytocin, and sex hormones), and the role of specialized brain regions (such as the amygdala, limbic system, and prefrontal cortex). Most importantly, the research will focus on understanding the dynamic and integrative interaction between these elements and how they collectively form a complex network that determines the nature and diversity of human emotions. But how can we better understand this dynamic interaction? Can we identify specific "neurochemical fingerprints" for certain emotions? And how can this understanding contribute to the development of effective treatments? • Research Importance: o Clarifying the importance of studying affective behavior from a biological and neurochemical perspective for a deeper understanding of human emotions. o Indicating that human emotions are the result of a complex interaction between biological, neurochemical, and psychological factors. • Research Objectives: o Analyzing the biological and neurochemical foundations that shape affective behavior. o Presenting a conceptual model (metaphorical equation) to understand the interaction of these factors. o Exploring the practical applications of this knowledge in improving mental health. • Research Questions: o What are the main biological and neurochemical factors that influence the formation of human emotions? o How do these factors interact with each other to produce affective behavior? o What are the practical applications of understanding these interactions in treating affective disorders? Previous Studies

Study by Berger et al. (2009) Study Title: "The expanded biology of serotonin" The study was conducted in the United States. One of the most important findings of the study is that serotonin plays a key role in regulating mood and anxiety, and that low levels of it are closely associated with the onset of depression and anxiety symptoms. It also showed that drugs that work to increase serotonin levels (such as SSRIs) demonstrate effectiveness in treating depression. Among the key recommendations is the need for further research to understand the precise mechanisms by which

serotonin affects the brain, and to develop more effective drugs targeting the serotonin system to treat affective disorders.

Study by Nestler et al. (2009) Study Title: "Molecular neuropharmacology: A foundation for clinical neuroscience" The study was conducted in the United States. One of the most important findings of the study is that dopamine plays a major role in the feeling of happiness and reward, and that dysfunction in the dopamine system is linked to depression and bipolar disorder. It also showed that drugs that modify dopamine levels can help improve mood. Among the key recommendations is studying the effect of environmental factors (such as stress) on the dopamine system, and developing drug treatments that target the dopamine system more precisely.

Study by Heinrichs et al. (2009) Study Title: "Oxytocin, vasopressin, and human social behavior" The study was conducted in Germany. One of the most important findings of the study is that oxytocin enhances feelings of trust and social bonding, and can reduce anxiety symptoms and enhance emotional stability. It also showed that low levels of oxytocin are associated with an increased risk of depression. Among the key recommendations is studying the effect of oxytocin in treating social disorders such as autism, and developing oxytocin-based treatments to promote mental health.

Study by Sapolsky (2004) Study Title: "Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping" The study was conducted in the United States. One of the most important findings of the study is that elevated cortisol levels are associated with chronic stress and depression, and that cortisol negatively affects brain areas responsible for regulating emotions. It also showed that reducing cortisol levels can improve mental health. Among the key recommendations is studying the effect of stress management techniques (such as meditation) on cortisol levels, and developing treatments targeting the HPA axis to reduce the impact of stress on mental health.

Study by Caspi et al. (2003) Study Title: "Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene" The study was conducted in New Zealand. One of the most important findings of the study is that individuals with a dysfunction in the 5-HTT gene are more susceptible to depression when exposed to life stressors, and that the interaction between genes and environment plays a key role in the emergence of affective disorders. Among the key recommendations is studying the effect of other environmental factors (such as social support) on genes associated with depression, and developing preventive interventions for individuals genetically predisposed to depression.

Study by Teicher & Samson (2016) Study Title: "Annual research review: Enduring neurobiological effects of childhood abuse and neglect" The study was conducted in the United States. One of the most important findings of the study is that psychological trauma during childhood leads to permanent changes in brain structure (such as increased amygdala activity), and that these changes are associated with an increased risk of depression and anxiety in adulthood. Among the key recommendations is developing intervention programs to support children who

have experienced psychological trauma, and studying the effect of psychotherapy on reorganizing the brain after trauma.

Study by Kandel (1998) Study Title: "A new intellectual framework for psychiatry" The study was conducted in the United States. One of the most important findings of the study is that the brain has the ability to adapt and reorganize itself in response to life experiences, and that neural plasticity plays a key role in learning, memory, and recovery from psychological injuries. Among the key recommendations is studying the effect of psychological interventions (such as cognitive-behavioral therapy) on neural plasticity, and developing treatments based on enhancing neural plasticity to improve mental health. Benefits from Previous Studies • Developing theoretical models: The results of these studies can be used to develop a conceptual model (metaphorical equation) to understand the complex interactions between biological and psychological factors in shaping affective behavior. • Guiding current research: Previous studies provide a reference framework for identifying research gaps, such as the need for more studies on the effect of environmental factors on neurochemistry. • Practical applications: The results can be translated into therapeutic interventions, such as developing more effective drugs or therapy programs based on understanding the interactions between biological experiences.

Differences between previous studies and the current study • Focus on the conceptual model: While previous studies focused on analyzing individual factors (such as the role of serotonin or cortisol), our current study aims to present a comprehensive conceptual model (metaphorical equation) that illustrates the interaction between all these factors. • Emphasis on practical applications: Our study aims to translate theoretical findings into practical applications in the field of mental health, such as developing integrated treatments based on understanding complex interactions. • Integration between disciplines: Our study relies on the integration of neuroscience, psychology, and neurochemistry, providing a comprehensive and integrated vision of affective behavior. Differences from previous studies

Comprehensive conceptual model: o Our study presents a conceptual model (metaphorical equation) that illustrates the interaction between biological, psychological, and social factors, while previous studies focused on individual factors.

Focus on practical applications: o Our study aims to translate theoretical findings into practical applications in the field of mental health, such as developing integrated treatments based on understanding complex interactions.

Integration between disciplines: o Our study relies on the integration of neuroscience, psychology, and neurochemistry, providing a comprehensive vision of affective behavior. Research Methodology: An analytical study based on reviewing scientific literature and previous research. Analysis tools: Theoretical analysis of previous studies in the fields of neuroscience, biological psychology, and neurochemistry. Using conceptual models to illustrate the complex interactions between different factors. Theoretical Framework Chapter One: Biological Foundations of Affective Behavior Affective (emotional) behavior is an essential part of the human experience and includes a wide range of emotions such as joy, sadness, anger, fear, and love. To understand these emotions, it is necessary to study the biological foundations behind them, which include the nervous system, the hormonal system, and chemical interactions in the brain. This chapter reviews the biological foundations of affective behavior, focusing on the role of the nervous system and

hormones in regulating emotions. The Nervous System and Affective Behavior The nervous system plays a central role in regulating affective behavior. The nervous system consists of the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system (PNS), which includes the nerves that connect the central nervous system to the rest of the body.

The Brain and Emotions: o The Amygdala: The amygdala is considered a primary center for processing emotions, especially fear and anxiety. Studies have shown that damage to the amygdala leads to a decrease in emotional responses to frightening situations (LeDoux, 2000). o Prefrontal Cortex: Plays a role in regulating emotions and decision-making. Damage to this area can lead to personality changes and emotional control disorders (Davidson, 2002).

Neurotransmitters: o Serotonin: Associated with mood and anxiety levels. Low serotonin levels are associated with depression and anxiety (Berger, Gray, & Roth, 2009). o Dopamine: Associated with feelings of happiness, reward, and pleasure. Disorders in the dopamine system can lead to conditions such as schizophrenia and depression (Nestler, Hyman, & Malenka, 2009). The Hormonal System and Affective Behavior Hormones play an important role in regulating emotions and affective responses.

Cortisol: o Known as the stress hormone, it is secreted in response to stress. High levels of cortisol can lead to increased anxiety and depression (Sapolsky, 2004).

Oxytocin: o Known as the love hormone, it plays a role in enhancing social bonds and trust. Studies have shown that oxytocin can reduce anxiety and enhance feelings of attachment (Heinrichs, von Dawans, & Domes, 2009). Interaction Between the Nervous System and the Hormonal System The interaction between the nervous system and the hormonal system is essential for understanding affective behavior. For example, in stressful situations, the hypothalamic-pituitary-adrenal (HPA) axis is activated, leading to the secretion of cortisol. This interaction affects brain areas such as the amygdala and prefrontal cortex, which in turn affects emotional responses (McEwen, 2007). Affective behavior is considered the result of complex biological interactions in the brain and nervous system. Neurotransmitters, hormones, and brain regions intertwine in an integrated network that contributes to generating and modulating emotions. Neurotransmitters play a crucial role; serotonin regulates mood, anxiety, and depression, and it is believed that low levels contribute to the appearance of depressive symptoms. Dopamine is associated with motivation, reward, and pleasure. Norepinephrine plays a role in responding to pressure, attention, and alertness. Glutamate is the primary excitatory neurotransmitter in the brain, and GABA is the primary inhibitory neurotransmitter that helps calm the nervous system and reduce anxiety and stress (Bear, Connors, & Paradiso, 2016). The impact does not stop at neurotransmitters but includes hormones secreted by the endocrine glands. Cortisol, secreted by the body in response to stress, can lead to anxiety, stress, and depression. Oxytocin enhances social bonding, trust, and positive feelings. Sex hormones (such as testosterone and estrogen) play a role in emotions related to sex and aggression (Carlson, 2013). These chemical factors integrate with the activity of specific brain regions. The amygdala is a central hub for processing fear and anger. The hippocampus contributes to forming memories associated with emotions. The prefrontal cortex plays a critical role in regulating emotions and decision-making (Kandel et al., 2012). The researcher concludes that the biological foundations of affective behavior include complex interactions between the nervous system and the hormonal system.

Understanding these interactions helps explain how emotions are regulated and how disturbances in these systems can lead to psychological disorders such as depression and anxiety. Future research in this field can provide deeper insights and lead to the development of more effective treatments for affective disorders. Chapter Two: Interactions Between Biological Factors and Psychological Experiences Affective behavior cannot be understood in isolation from the complex interactions between biological factors and psychological experiences. Life experiences affect brain chemistry, where psychological stress and trauma can lead to changes in neurotransmitter and hormone levels. Continuous exposure to stress can lead to elevated cortisol levels. Social relationships and social support contribute to enhancing positive feelings (LeDoux, 2015). The interaction between genes and the environment is considered a decisive factor. Genetic factors play a role in predisposing individuals to psychological disorders, but genetic influence interacts with environmental factors to determine the final outcome. Life experiences, such as abuse or neglect during childhood, affect gene expression and increase the risk of developing psychological disorders (Meaney, 2001). The amygdala participates in forming emotional associations, where environmental stimuli are linked to specific emotions. The hippocampus contributes to remembering emotional contexts. The interaction between biological factors and psychological experiences is a fundamental axis for understanding human behavior and mental health. Biological factors include genes, neurochemistry, and brain functions, while psychological experiences include life experiences, the social environment, and cognitive processes. This chapter reviews how these factors interact together to shape behavior and emotions, focusing on theories and research linking biology and psychology. Biological Factors Influencing Psychological Experiences

Genes and Heredity: o Genes play an important role in determining predisposition to psychological disorders such as depression, anxiety, and schizophrenia. Twin studies have shown that there is a strong genetic component to these disorders (Plomin et al., 2013). o Gene-Environment Interaction: Genes do not operate in isolation from the environment. For example, individuals with a genetic predisposition to depression may be more likely to develop it if exposed to significant life stressors (Caspi et al., 2003).

Neurochemistry: o Neurotransmitters: Such as serotonin, dopamine, and norepinephrine, play a key role in regulating mood and behavior. Imbalances in these neurotransmitters are associated with disorders such as depression and anxiety (Berger et al., 2009). o Hormones: Such as cortisol and oxytocin, affect emotional and social responses. For example, high cortisol levels are associated with chronic stress, while oxytocin enhances social bonds (Heinrichs et al., 2009).

Brain Structure and Functions: o Amygdala: Plays a role in processing emotions such as fear and anxiety. Damage to the amygdala can lead to a decrease in emotional responses (LeDoux, 2000). o Prefrontal Cortex: Responsible for regulating emotions and decision-making. Damage to this area can lead to emotional control disorders (Davidson, 2002). Psychological Experiences and Their Impact on Biological Factors

Early Life Experiences: o Attachment: Early relationships with caregivers affect brain development and emotional responses. Children who suffer from emotional neglect may show changes in brain structure and function (Schore, 2001). o Psychological Trauma: Exposure to trauma during childhood can lead to long-term changes in the stress response system, such as increased activity of the hypothalamic-pituitary-adrenal (HPA) axis (Teicher & Samson, 2016).

Psychological and Social Stress: o Chronic Stress: Can lead to biological changes such as elevated cortisol levels, negatively affecting mental and physical health (McEwen, 2007). o Social Support: Having a strong social support network can enhance mental health and reduce the impact of stress on the body (Cohen & Wills, 1985). Interaction Between Biological Factors and Psychological Experiences

The Biopsychosocial Model: o This model proposes that mental health and mental illness are the result of a complex interaction between biological, psychological, and social factors. For example, depression can result from a neurotransmitter imbalance (biological factor), negative thinking patterns (psychological factor), and life stressors (social factor) (Engel, 1977).

Neuroplasticity: o Neuroplasticity refers to the brain's ability to adapt and reorganize itself in response to life experiences. Learning, psychotherapy, and even exercise can lead to changes in brain structure and function (Kandel, 1998). Chapter Three: Affective Disorders from a Neurochemical Perspective Affective disorders, such as depression, anxiety, and bipolar disorder, can be understood from a neurochemical perspective, where they are considered disorders in the functioning of neurotransmitters, hormones, and brain regions. Depression is associated with a deficiency in serotonin and norepinephrine levels. The effectiveness of antidepressant medications depends on increasing the levels of these neurotransmitters in the brain, either by preventing their reuptake or increasing their release (American Psychiatric Association, 2013). Generalized anxiety disorder is associated with a deficiency in GABA and increased amygdala activity. Anti-anxiety medications work by enhancing the effect of GABA or reducing amygdala activity (Stahl, 2013). Affective disorders, such as depression and bipolar disorder, are among the most common and impactful psychological disorders on quality of life. To understand these disorders, it is necessary study the neurochemical foundations behind them, which include imbalances in to neurotransmitters, hormones, and brain functions. This chapter reviews affective disorders from a neurochemical perspective, focusing on the role that neurochemistry plays in the emergence and persistence of these disorders. Neurotransmitters and Affective Disorders

Serotonin (Serotonin): o Depression: Low serotonin levels are associated with depression. Antidepressant medications such as selective serotonin reuptake inhibitors (SSRIs) work to increase serotonin levels in the brain, helping to improve mood (Berger et al., 2009). o Anxiety: Serotonin also plays a role in regulating anxiety. Low levels can lead to increased anxiety symptoms (Gordon & Hen, 2004).

Dopamine (Dopamine): o Bipolar Disorder: Dysfunction in the dopamine system is associated with bipolar disorder. During manic episodes, dopamine levels are high, while they are low during depressive episodes (Berk et al., 2007). o Depression: Low dopamine levels can contribute to symptoms of depression such as loss of interest and pleasure (Nestler et al., 2009).

Norepinephrine (Norepinephrine): o Depression: Low norepinephrine levels are associated with depression. Medications that increase norepinephrine levels can help improve mood (Moret & Briley, 2011). o Anxiety: Norepinephrine plays a role in the stress and anxiety response. Elevated levels can lead to increased anxiety symptoms (Southwick et al., 2005). Hormones and Affective Disorders

Cortisol (Cortisol): o Depression: High cortisol levels, resulting from chronic stress, are associated with depression. The hypothalamic-pituitary-adrenal (HPA) axis is overactive in many cases of

depression (Pariante & Lightman, 2008). o Anxiety: High cortisol levels can also lead to increased anxiety symptoms (Sapolsky, 2004).

Oxytocin (Oxytocin): o Depression: Low oxytocin levels are associated with an increased risk of depression. Oxytocin enhances social bonds and can help improve mood (Heinrichs et al., 2009). o Anxiety: Oxytocin can reduce anxiety symptoms and enhance feelings of attachment (Neumann & Landgraf, 2012). Changes in Brain Structure and Function

Amygdala: o Depression: Increased amygdala activity is associated with depression. This excessive activity can lead to increased emotional responses to negative situations (Drevets et al., 2008). o Anxiety: The amygdala plays a key role in processing fear and anxiety. Increased activity can lead to increased anxiety symptoms (Etkin & Wager, 2007).

Prefrontal Cortex: o Depression: Decreased prefrontal cortex activity is associated with depression. This region is responsible for regulating emotions and decision-making (Mayberg, 2003). o Bipolar Disorder: Dysfunction in prefrontal cortex activity can contribute to mood swings in bipolar disorder (Phillips & Swartz, 2014). Behavioral Chemistry Interaction Equation (Practical Aspect) This chapter presents a metaphorical conceptual model to understand the complex interactions that shape affective behavior, emphasizing that the term "equation" here is used symbolically, not as a mathematical equation. Conceptual Model of Behavioral Chemistry Interaction Equation: Behavior (B) = [Genetics (G) \times Environment (E)] + [Neurotransmitters (NT) + Hormones (H) + Brain Regions (Bas)] + Psychological Processes (PP) Behavior (B): The final outcome, i.e., the actions and feelings exhibited by the individual. Genetics (G): Genetic factors that influence predispositions to behaviors and personal traits. Environment (E): All external upbringing, life experiences, social relationships, influences, such as and culture. Neurotransmitters (NT): Chemical substances that transmit signals between neurons, such as serotonin, dopamine, and norepinephrine. Hormones (H): Chemical substances secreted by endocrine glands that affect bodily functions and mood, such as cortisol and oxytocin. Brain Regions (Bas): Different parts of the brain involved in processing information and regulating behavior, such as the amygdala and prefrontal cortex. Psychological Processes (PP): Internal mental processes, such as thinking, perception, memory, and emotions. Behavior is influenced by a set of interacting factors:

Interaction between Genetics and Environment ($G \times E$): Genetics determine some predispositions, but the environment shapes how these predispositions are expressed. For example, an individual may have a genetic predisposition to depression, but the onset of depression depends on exposure to specific environmental conditions, such as psychological stress or trauma.

Direct impact of biochemical factors (NT + H + Bas): Neurotransmitters, hormones, and brain regions directly affect mood and behavior. For example, serotonin deficiency is associated with depression, and increased amygdala activity is associated with anxiety.

Role of psychological processes (PP): Internal mental processes affect how events are interpreted and responded to, thus influencing behavior. For example, negative thinking patterns contribute to worsening depressive symptoms. Example Application 1: Suppose a person suffers from social anxiety. Their behavior can be explained through the following model: Genetics: They may have a genetic predisposition to anxiety. Environment: They may have been exposed to negative social experiences in the past. Neurotransmitters: They may have a deficiency in GABA or an increase in norepinephrine. Brain Regions: They may have excessive activity in the amygdala. Psychological Processes: They may have negative thoughts about social interactions. Detailed Interaction Model: Where: Stimulus (S): Any external or internal trigger that elicits a response, such as seeing a joyful scene, hearing sad news, or feeling hungry. Sensory Perception (P): The process by which the stimulus is received and interpreted by the senses and nervous system. Electrochemical Neurological Interactions (ECNs): The heart of the "equation," which includes: Ion Transition (I⁺/I<sup>): Movement of ions across neuronal membranes, leading to action potential generation. Neurotransmitter Release (NT): Secretion of neurotransmitters (such as adrenaline if the dog poses a threat) in synaptic junctions. Chemical Reactions (CR): Interactions of neurotransmitters with receptors on target neurons. Changes in Electric Potential (ΔV): Changes in electric potential across neuronal membranes. It can be represented metaphorically as follows: ECNs = $f(I < sup > + </sup > - </sup >, NT, CR, \Delta V)$ where f means "a function of." Emotional Behavioral Response (BAR): The final outcome, including: Observable Behavior (B): Actions and observable behaviors. Emotional Response (AR): Internal feelings and sensations. Example Application 2: A person sees a barking dog (the stimulus). This scene is perceived by the senses (sensory perception). This leads to a series of electrochemical interactions in the brain, including ion movement and neurotransmitter release (such as adrenaline if the dog poses a threat) and changes in neuronal electric potential. These interactions lead to an emotional behavioral response, such as fear and moving away from the dog (observable behavior) and feeling tension and anxiety (emotional response). Sub-conclusions: The Comprehensive Interaction Model: The proposed model for behavior (B) = [Genetics (G) × Environment (\in)] + [Neurotransmitters (NT) + Hormones (H) + Brain Areas (Bas)] + Psychological Processes (PP) is a crucial starting point for understanding the complexity of emotional behavior. This model rejects reductionism to a single factor, instead emphasizing the dynamic interaction between genetic and environmental factors, which together form the biological basis of behavior, in addition to the direct impact of biochemical factors in the brain, and finally, the important role of psychological processes in interpreting events and responding to them. This holistic perspective aligns with modern trends in neuropsychology and neuroscience, which emphasize the importance of complex interactions between genes, environment, and the brain in determining behavior. Interaction Between Genetics and Environment (G \times E): The model highlights the importance of the interaction between genetics and environment, where genetics determine biological predispositions, while the environment shapes how these predispositions are expressed. This concept is known as geneenvironment interaction and is essential for understanding mental illnesses. For example, having a genetic predisposition for depression does not necessarily mean developing it; rather, it depends on exposure to triggering environmental factors, such as psychological stress or childhood trauma. This interaction explains why some individuals develop mental illnesses while others with the same genetic predisposition do not. The Central Role of Biochemical Factors (NT + H + Bas): The model sheds light on the crucial role of neurotransmitters, hormones, and brain regions in regulating mood and behavior. This aspect is essential for understanding emotional disorders, where depression is associated with a deficiency in serotonin and norepinephrine, and anxiety with a deficiency in GABA and increased activity in the amygdala. This understanding justifies the use of psychiatric medications that aim to modify the levels of these chemicals in the brain. However, the model emphasizes that these biochemical factors are not solely responsible for behavior but interact with other factors, explaining why medications are not always effective for all individuals.

The Importance of Psychological Processes (PP): The model emphasizes the role of psychological processes, such as thinking, perception, memory, and emotions, in shaping emotional behavior. The way events are interpreted and responded to affects mood and behavior. For example, negative and pessimistic thoughts contribute to worsening depressive symptoms, while negative self-assessments lead to social anxiety. This highlights the importance of psychotherapy, which aims to modify these negative psychological processes, in addition to pharmacotherapy. The Detailed Model of Electrochemical Neurological Interactions (ECNs): The detailed model (ECNs = $f(I < sup > + < /sup > / I < sup > , NT, CR, \Delta V)$ provides a deeper understanding of the biological mechanisms that lead to emotional behavioral responses. This model focuses on the complex electrochemical interactions that occur in the brain upon receiving a stimulus, starting from the movement of ions across neuronal membranes, through the release of neurotransmitters and their interaction with receptors, to changes in the electrical potential of neurons. This model provides a framework for understanding how external and internal stimuli are translated into emotional behavioral responses. General Conclusion: The proposed model provides a comprehensive framework for understanding the complexity of emotional behavior, focusing on the dynamic interaction between genetic, environmental, biochemical, and psychological factors. This integrated perspective is essential for developing effective strategies for preventing and treating emotional disorders, as these strategies should take into account all these interacting factors. It should be emphasized that the proposed "equation" is a metaphorical conceptual model, not a precise mathematical equation, but rather a tool for understanding the complex interactions that shape emotional behavior. Conclusion: This research demonstrates the close connection between the biochemical makeup of the brain and the emotional experience of humans. We have explored how neurotransmitters and hormones form the biological basis of emotions, and the pivotal role of brain regions in processing and regulating emotions. We have also highlighted the importance of complex interactions between these biological factors and psychological experiences. This research presents a framework for understanding the "Equation of Affective Behavior Chemistry," shedding light on the dynamic interaction between biological and psychological factors. Nevertheless, this field still requires further research. Fully understanding these interactions will enable us to develop more effective treatments for affective disorders and improve mental health.

المصادر والمراجع

المراجع الأجنبية

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.).
- Bear, M. F., Connors, B. W., & Paradiso, M. A. (2016). Neuroscience: Exploring the brain (4th ed.). Lippincott Williams & Wilkins.
- Berger, M., Gray, J. A., & Roth, B. L. (2009). The expanded biology of serotonin. *Annual Review of Medicine*, 60, 355–366.
- Berk, M., Dodd, S., Kauer-Sant'Anna, M., Malhi, G. S., Bourin, M., Kapczinski, F., & Norman, T. (2007). Dopamine dysregulation syndrome: Implications for a dopamine hypothesis of bipolar disorder. *Acta Psychiatrica Scandinavica*, 116(s434), 41-49.
- Carlson, N. R. (2013). Physiology of behavior* (11th ed.). Pearson.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., ... & Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301(5631), 386-389.
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98(2), 310-357.
- Davidson, R. J. (2002). Anxiety and affective style: Role of prefrontal cortex and amygdala. *Biological Psychiatry*, 51(1), 68-80.
- Drevets, W. C., Price, J. L., & Furey, M. L. (2008). Brain structural and functional abnormalities in mood disorders: Implications for neurocircuitry models of depression. *Brain Structure and Function*, 213(1-2), 93-118.
- Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, 196(4286), 129-136.
- Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A metaanalysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry*, 164(10), 1476-1488.
- Gordon, J. A., & Hen, R. (2004). Genetic approaches to the study of anxiety. *Annual Review of Neuroscience*, 27, 193-222.
- Heinrichs, M., von Dawans, B., & Domes, G. (2009). Oxytocin, vasopressin, and human social behavior. *Frontiers in Neuroendocrinology*, 30(4), 548-557.
- Kandel, E. R. (1998). A new intellectual framework for psychiatry. *American Journal of Psychiatry*, 155(4), 457-469.
- Kandel, E. R., Schwartz, J. H., Jessell, T. M., Siegelbaum, S. A., & Hudspeth, A. J. (2012). Principles of neural science (5th ed.). McGraw-Hill Medical.
- LeDoux, J. (2015). Anxious: Using the brain to understand and treat fear and anxiety. Viking.
- LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23(1), 155-184.

- Mayberg, H. S. (2003). Modulating dysfunctional limbic-cortical circuits in depression: Towards development of brain-based algorithms for diagnosis and optimised treatment. *British Medical Bulletin*, 65, 193-207.
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87(3), 873-904.
- Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. Annual Review of Neuroscience, 24, 1161–1192.
- Moret, C., & Briley, M. (2011). The importance of norepinephrine in depression. *Neuropsychiatric Disease and Treatment*, 7, 9-13.
- Nestler, E. J., Hyman, S. E., & Malenka, R. C. (2009). *Molecular neuropharmacology: A foundation for clinical neuroscience* (2nd ed.). McGraw-Hill Medical.
- Neumann, I. D., & Landgraf, R. (2012). Balance of brain oxytocin and vasopressin: Implications for anxiety, depression, and social behaviors. *Trends in Neurosciences*, 35(11), 649-659.
- Pariante, C. M., & Lightman, S. L. (2008). The HPA axis in major depression: Classical theories and new developments. *Trends in Neurosciences*, 31(9), 464-468.
- Phillips, M. L., & Swartz, H. A. (2014). A critical appraisal of neuroimaging studies of bipolar disorder: Toward a new conceptualization of underlying neural circuitry and a road map for future research. *American Journal of Psychiatry*, 171(8), 829-843.
- Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral Genetics* (6th ed.). Worth Publishers.
- Sapolsky, R. M. (2004). Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping. *Henry Holt and Company*.
- Schore, A. N. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, 22(1-2), 7-66.
- Southwick, S. M., Vythilingam, M., & Charney, D. S. (2005). The psychobiology of depression and resilience to stress: Implications for prevention and treatment. *Annual Review of Clinical Psychology*, 1, 255-291.
- Stahl, S. M. (2013). Stahl's essential psychopharmacology: Neuroscientific basis and practical applications (4th ed.). Cambridge University Press.
- Teicher, M. H., & Samson, J. A. (2016). Annual research review: Enduring neurobiological effects of childhood abuse and neglect. *Journal of Child Psychology and Psychiatry*, 57(3), 241-266.