



RESEARCH ARTICLE

Toward an integrative approach to mental health: Clinical and ethical challenges in ADHD treatment through the lens of ‘Top-Down’ and ‘Bottom-Up’ models, and psychosocial and lifestyle factors

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ABSTRACT

Human adaptation depends on the increasing complexity of neural processes. Nevertheless, contemporary stressors and health determinants frequently undermine this capacity. In psychiatry and psychology, the diagnosis of attention deficit hyperactivity disorder demonstrates an ongoing debate between reductionist approaches, viewing the brain as the primary executive organ, and the multidimensional realities encountered in clinical settings. This article examines limitations within current diagnostic paradigms and introduces a more comprehensive conceptual model. It allows us to differentiate between mental disorders of neurodevelopmental origin and problems that are similar but associated with other issues, such as relationship problems, risk factors or harmful lifestyle habits. The discussion underscores that the prevailing use of psychostimulants tends to mask underlying causes such as systemic inflammation, intestinal dysbiosis, or affective deprivation. Evidence indicates that both cerebellar function and the ‘social nervous system’ are integral to cognitive regulation, suggesting that medication alone is inadequate. Additionally, long-term pharmacological treatment may present risks including reduced bone density, metabolic dysfunction, and potential psychotic adverse effects, thereby necessitating careful ethical consideration. Effective clinical management should prioritize a strong therapeutic partnership and a thorough differential diagnostic strategy. Accordingly, an integrative approach to positive mental health is recommended, emphasizing lifestyle modifications, emotional stability, and the combined use of Bottom-Up mechanisms with cognitive and medical interventions to promote patient autonomy and resilience.

Keywords: ADHD, differential diagnosis, neuroscience, gut-brain axis, clinical ethics, therapeutic alliance, integrative medicine

Introduction

All organisms must adapt to changing environments for survival, a capacity found in plants, insects, birds, and animals. In mammals, the evolution of the nervous system and brain enabled more complex behaviors, fostering learning and interaction. Human neural complexity has advanced affective, cognitive, and social abilities, but adaptation remains difficult due to modern challenges. Genetic and epigenetic factors may not always mitigate psychosocial risks and health threats, leading some individuals to struggle with resilience. If necessary, psychiatry and psychology offer therapies to support or restore adaptive abilities needed for daily functioning. However, some significant challenges constrain the effectiveness of educational and therapeutic interventions, like observed in *attention deficit hyperactivity disorder* (ADHD), as well as likely mood disorder, anxiety disorder, and a few other psychiatric illnesses.¹⁻¹²

The lack of objective biological markers requires a thorough evaluation of behavioral indicators associated with mental disorders, including careful interpretation of psychometric test results. However, these assessments may be overly reductive and are frequently subjective, despite initial perceptions to the contrary¹⁻⁶. Standardized evaluations may be useful in urgent situations, but they are fundamentally limited in their ability to accurately assess the anatomical and functional status of patients' nervous systems.^{2,3,7-11}

Then, collaboration with other medical disciplines prompted psychiatry, beginning in the 1950s, to investigate diseases by examining the impact of pharmacological treatments.¹²⁻¹⁶ This approach has resulted in an agreed-upon definition of psychological normality, which is occasionally viewed as arbitrary, particularly due to the influence of psychotropic medications.^{1,6,8,10,11,17} Consequently, the application of psychotropic medications has expanded beyond the management of pronounced symptoms in individuals with psychotic disorders to include the regulation of psychological issues even prior to a confirmed psychiatric diagnosis.^{1,4,5,17}

Although there is a broad array of therapeutic intervention strategies available, it is essential to address the associated ethical considerations.^{1-4,6,8,11,15,24} Firstly, therapeutic orientation may be insufficient if the diagnosis is founded on an inaccurate understanding of the patient's psychological functioning. Furthermore, providing effective care becomes challenging without comprehensive attention of all risk factors, individual choices, and adjustment difficulties. While certain disorders can be attributed to genetic vulnerability, it should be noted that not all symptoms are necessarily related to such factors.¹ Secondly, the administration of a psychotropic medication is typically determined by the necessity to rapidly manage maladaptive behaviours, which may result in reduced attention to potential adverse effects these substances may have on brain function. Furthermore, particularly regarding children and adolescents, it is important to critically assess the underlying rationale for prescribing such medications.^{2,11}

Third, most prevailing theories concerning mental disorders focus on neurological dysfunctions affecting the processing of sensations and thoughts within specific life contexts.¹⁹⁻²¹ This reflects the conventional '*top-down framework*', wherein the brain is regarded as the primary center for affective and cognitive functions. Nonetheless, accumulating research demonstrates that disturbances in additional physiological systems—including hormonal, cardiovascular, and enteric pathways—may also contribute to the onset of psychiatric symptoms. This supports a '*bottom-up framework*' that recognises the influence of these physiological systems on individuals' affective and cognitive skills.²²⁻²⁴

Clinicians should systematically assess all relevant factors to confirm or reconsider psychiatric diagnoses. Using differential diagnosis and reflective practice improves symptom interpretation and treatment choices. Overlooking potential contributors can lead to inappropriate care, as seen with the global increase in ADHD diagnoses linked to reliance on select indicators and dominant theories.^{4,6,24}

Clinical and Ethical Considerations in ADHD Treatment

Achieving optimal understanding of clinical manifestations and informing therapeutic decisions requires an ongoing alignment between empirical evidence gathered through systematic observation of symptoms, and theoretical knowledge that is consistently updated considering scientific progress. Since 1996, there has been consensus on the definition of ADHD.²⁵ However, establishing a comprehensive theoretical framework grounded in behavioral indicators interpreted as manifestations of a genetically influenced neurodevelopmental disorder has necessitated extensive research and ongoing refinement over many years.²⁶ Currently, it is recognized that the clinical profile presents a greater degree of complexity than previously anticipated, and the approach to management may not always align directly with the underlying etiology of the symptoms.^{1,2,6,10,17,24} While a '*top-down*' approach is valid, it often results in the prescription of psychostimulants to mitigate symptom severity without first confirming that the mental disorder cannot be attributed to alternative clinical factors.^{5-6,8,24} This situation presents a significant ethical concern, particularly as side effects are often downplayed to mitigate potential social and academic setbacks.

Pharmacological management of symptoms commonly linked to ADHD originates from Bradley's initial observations, wherein Bzedrine® was administered to 30 adolescents presenting with severe behavioral disturbances.²⁷ He noted that 14/30 individuals demonstrated behavioral improvement; however, the emerging theory did not adequately address outcomes for the remaining 16 participants, for whom treatment yielded unsatisfactory results. Subsequently, Bradley²⁸ conducted a study involving 470 children—primarily those experiencing anxiety or presenting with behavioral or adjustment disorders—treated with Bzedrine® and/or Dexedrine®. Symptom improvement was observed in 60~75% of cases, no significant change was

reported in 15~25%, and an unfavorable response occurred in 10~15% of the subjects. Subsequently, clinicians referred to the condition as 'hyperkinetic impulse disorder'²⁹, and later, Conners and Eisenberg³⁰ utilized Ritalin® for the management of learning difficulties. Furthermore, Douglas³¹ demonstrated that this psychostimulant enhanced concentration and regulated impulsivity. The characterization of ADHD then evolved to emphasize its basis as a neurodevelopmental disorder of genetic origin, typically treated with pharmacological agents that inhibit dopamine reuptake or enhance its synaptic release.

Currently, the prevalence of ADHD among students is estimated to be between 3% and 6%^{2,4,32}, although certain countries have reported substantially higher rates.^{2,5,34} Swanson and colleagues¹ have long questioned the rigor of clinical evaluations, noting that only approximately 1% of students are genuinely affected by ADHD. However, multiple studies indicate that the prevalence of ADHD diagnoses varies depending on students' birth months, which challenges the hypothesis of a genetic origin and raises concerns regarding the validity of psychometric assessments.^{2,4,6,12,24,25} In Quebec, the rate is 17% for students born in the fall and 22% for those born during the summer³⁴. Similar effects of birth month on diagnosis rates and psychostimulant prescriptions have also been documented in France³⁵. Given the observed trend in France and Quebec, where the youngest students in a class are disproportionately identified as having ADHD³⁴⁻³⁵, it is pertinent to critically assess the conceptual framework that categorizes ADHD primarily as a genetically based disorder^{5-6,24}.

To enhance the effectiveness of psychosocial and medical interventions, we have recently advocated for distinguishing between ADHD and ADHD-like.^{6,24} A review of scientific literature corroborates our clinical observations: various physical or mental health challenges may result in behaviors resembling ADHD without constituting the disorder itself. The absence of this differentiation poses both ethical and clinical concerns, as it may prevent patients from receiving appropriate treatment aimed at restoring their health. Additionally, there is reason to believe that unsuccessful clinical differentiation has contributed to the pervasive view of ADHD as a condition of strictly genetic and permanent origin.

Conversely, other researchers have observed that modifications in educational environment, career direction, or independence from familial influences are frequently associated with significant reductions in the intensity and frequency of ADHD-related symptoms.³⁶ This underscores the necessity of evaluating environmental risk factors, including those present at home and in school, during diagnostic assessments.^{3,6,24,37} Furthermore, guidelines stipulate that an ADHD diagnosis should not be made if symptoms are attributable to another mental disorder.^{10,37} Notably, over 75% of students diagnosed with ADHD also present with comorbid conditions, such as anxiety, learning disabilities, or behavioral disorders.³⁸ It is therefore plausible to suggest that insufficient clinical rigor may have

contributed to the persistent rise in psychostimulant prescriptions over the past 25 years.^{1,4-6,34-35,39}

In the absence of a thorough differential diagnosis, clinicians may prescribe psychostimulants to students who do not necessarily present with neurological dysfunction.^{6,34,35,39} This insufficiently targeted therapeutic approach is particularly concerning given the limited understanding of the side effects associated with psychotropic medications. Notably, a meta-analysis reviewed 1563 studies, of which 1499 were excluded due to significant methodological biases.⁴⁰ Additionally, most assessments of adverse effects have been conducted in populations over 16 years of age, whereas most psychostimulant users are younger individuals. Furthermore, while 33/64 studies reported serious adverse events, only 5/33 examined side effects lasting longer than 12 weeks, even though psychotropic drug use frequently continues over several years.⁴⁰

Several studies indicate that an initial reduction in growth of 0.9 cm/year was observed over the first 14 months, followed by a further decrease of 1.03 cm/year ten months later, relative to children of comparable age.⁴¹⁻⁴² Among children commencing treatment at preschool age, this decrease reaches 1.38 cm/year.⁴³ Adolescents receiving psychostimulant therapy present an elevated risk of long bone fractures, associated with diminished bone mineral density that may contribute to early onset osteoporosis and prolonged bone fragility.⁴⁴⁻⁴⁵ This effect is influenced by dosage and biological sex.⁴⁶ Additionally, research identifies the occurrence of weight loss and sleep disturbances in treated populations.^{33,47-48}

In addition, the risk of developing psychosis doubles in adolescents using methylphenidate, but it is increased fourfold in the case of Adderall®. In addition, the likelihood of developing psychosis or bipolar disorder increases by a factor of 5.3.⁵⁰ A meta-analysis of sixteen studies highlights the association between stimulant use and the occurrence of psychosis or bipolar disorder in patients, whose average age is 12.6 years; this risk appears to be more marked with Adderall®.⁵¹ Several studies have identified cardiac symptoms, including bradycardia and tachycardia.^{10,33,47,48,52-53} Furthermore, Health Canada withdrew Adderall® from the market for six months in 2005, following FDA reports of 20 deaths and 12 strokes in adolescents. However, two studies have concluded that these phenomena are extremely rare. Finally, a large study has shown that psychostimulants do not act on attentional functions, but rather on the reward system, like cocaine, to increase the student's availability for less affordable learning, but also modify the functioning of cortical areas, the effectiveness of which is interdependent on quality sleep.⁵⁶

Furthermore, the risk of developing psychosis in adolescents is doubled with methylphenidate use and increased fourfold with Adderall®. The probability of experiencing psychosis or bipolar disorder rises by a factor of 5.3.⁵⁰ A meta-analysis of sixteen studies underscores the association between stimulant use and the incidence of psychosis or bipolar disorder, with an average patient age of 12.6 years; this risk is notably higher with Adderall®.⁵¹ Multiple studies have also

reported cardiac symptoms such as bradycardia and tachycardia.^{10,33,47,48,52-53} In 2005, Health Canada temporarily withdrew Adderall® from the market following FDA reports of 20 deaths and 12 strokes among adolescents. However, two studies concluded that these adverse events are rare.⁵⁴⁻⁵⁵ Finally, a large-scale study demonstrated that psychostimulants primarily affect the reward system, like cocaine, thereby increasing students' engagement with more challenging learning tasks and altering cortical area function, which is closely linked to quality sleep.⁵⁶ Somewhere, Ritalin® addresses reduced motivation for unengaging tasks as well as sleep deficits that may result from academic challenges and detrimental lifestyle practices, such as evening screen exposure.^{4,6,56}

A recent meta-analysis found that while 14 studies indicated an improvement in patient autonomy, 22 studies reported contrary results.⁵⁷ The positive effects of psychostimulants appear to persist for approximately 14 months before gradually diminishing or disappearing entirely, potentially resulting in additional psychosocial issues. These findings are consistent with previous studies.^{1,40,47} Nonetheless, symptoms may remit abruptly, for instance, following relocation, enrollment in a new academic pathway or career, or the initiation of corrective eyewear.^{24,36} This highlights the necessity of thoroughly considering all potential triggers of ADHD-like.⁶ It is noteworthy that more than fifty medical or psychosocial conditions can produce symptoms resembling those of ADHD, emphasizing the need for comprehensive evaluation prior to diagnosis.^{2,4-6,10,11,24}

Although public awareness may be limited, ongoing scientific debate persists regarding the genetic basis of ADHD and the validity of psychometric assessment tools. These discussions have significant implications for therapeutic practice and management.^{3-5,8,34-35,47-48} Current guidelines recommend a multimodal approach that integrates psychosocial interventions with pharmacological treatments.^{6,24,58-60} Selection of drug therapy deliberates efficacy, tolerability, duration of action, affordability, and potential interactions with other medications.^{40,47} Nevertheless, clinicians often face insufficient resources. For instance, while regulations mandate a differential diagnosis prior to the prescription of psychotropic drugs⁵⁸, they lack specifications regarding the processes required to distinguish symptoms, relevant risk factors, or indicators necessary to inform clinical decisions. Furthermore, there are concerns regarding the limited time and resources available to physicians for conducting comprehensive differential analyses. Consequently, numerous alternative causes of ADHD-like symptoms may be overlooked, resulting in an incomplete behavioural profile and frequent reliance on extended psychotropic medication prescriptions.^{4-6,24}

The 'Top-Down' Approach

Determining the origin of symptoms and thereby establishing an appropriate treatment pathway for psychiatric illnesses and mental disorders is a highly complex clinical undertaking, as exemplified by the case of ADHD. The tendency to focus primarily on the frequency and intensity of behavioral indicators, while

sometimes understandable, can inadvertently obscure various medical and psychosocial issues that may manifest as ADHD-like symptoms. Renaud⁷ previously noted similar concerns in the diagnosis of depression, often resulting in prolonged prescription of antidepressants, despite evidence that at least nine different brain regions may produce symptoms resembling those of depression. Likewise, conditions such as bipolar disorder, schizophrenia, and anxiety disorders involve multiple dysfunctional brain areas and psychotropic medications prescribed for these disorders may generate significant adverse effects. A reflective and critical approach should therefore guide clinical decision-making to ensure that therapeutic strategies are optimally tailored to available information and focused on alleviating patient suffering and facilitating recovery opportunities. While Faraone and colleagues³⁹ highlight apprehensions regarding diminished state oversight, this very oversight remains crucial for maintaining effective public financial control.

Cognitive Models

Explanatory models for skills and abilities are commonly classified according to four main theoretical perspectives, with recent developments in neuroscience promoting greater integration in our comprehension of human behaviour. From the cognitivist standpoint, psychopathology is attributed to impairments in information processing, as opposed to unconscious conflicts or biological irregularities.⁶⁴⁻⁶⁶ This theoretical model asserts that individual responses are shaped by subjective interpretations rather than objective events. Mental disorders are conceptualized through a three-tiered hierarchy. At the primary level are cognitive schemas, which are fundamental, rigid, and unconditional beliefs about oneself, the external environment, or the future.⁶⁷⁻⁶⁸ These schemas, typically formed during childhood, remain dormant until reactivated by stressors reminiscent of prior experiences. The secondary level involves cognitive distortions; when memory is engaged, perceptions of reality are modified to reinforce pre-existing beliefs, often manifesting as logical errors such as dichotomous thinking, overgeneralization, or selective abstraction. The tertiary level encompasses automatic thoughts, characterized by reflexive, immediate, and negative patterns that contribute directly to psychological distress and maladaptive behavioral manifestations.

This theoretical model, widely utilized in North America, has gained increasing traction in Europe as a response to the scientific limitations of psychoanalysis. Nevertheless, the cognitivist approach is not exempt from certain reductionist tendencies that may affect the efficacy of cognitive-behavioral therapies. The quantitative research process, for example, often necessitates standardization to control various variables, simultaneously assigning a significant role to the clinician. Furthermore, such standardization frequently aligns with administrative requirements within healthcare organizations. While these intentions are legitimate, they risk compromising the therapeutic process if insufficient adaptation is made to the unique needs of individual patients. Additionally, the inclusion and exclusion criteria established for research protocols tend to prioritize research objectives over

patient interests. As a result, individuals presenting comorbidities or complex behavioral profiles are seldom selected, despite their prevalence in clinical practice.⁶⁹⁻⁷⁰

Consequently, unsuccessful interventions may diminish patients' confidence in their own resilience and perpetuate reliance on medication based on the assumption of a genetic origin for the condition.¹¹ While genetic inheritance plays a role in influencing behavior, prevailing scientific understanding does not support a strictly deterministic genetic model.^{18,71} Brain plasticity and the extended period of normal brain maturation suggest that educational or therapeutic approaches, despite requiring time and facing resistance from some individuals, remain crucial in mitigating symptom intensity and frequency. However, these interventions can also potentially lead to psychophysiological imbalances. Cyrulnik⁷¹ advocates for viewing humans as integral components of an ecosystem, navigating various sensory and emotional environments that shape development and challenge models linking psychopathology solely to the dysfunction of specific genes.

When applied to ADHD, the cognitivist framework emphasises enhancing executive functions and restructuring automatic thoughts. Nevertheless, this approach may be insufficient if it overlooks the attentional demands faced by patients; specifically, when the neurophysiological substrate is characterised by persistent hyperarousal or chronic fatigue, the cognitive effort required for inhibitory control becomes significantly taxing. As a result, cognitive remediation may serve only as a temporary solution unless it is complemented by interventions that address the deeper biological processes involved.

Cerebellar and Emotional Influences on Cognitive and Affective Functioning

Several conceptual and methodological biases constrain the effectiveness of cognitivist models. For instance, while advances in brain imaging offer valuable technological resolution, they may obscure epiphenomena and cannot reliably distinguish between neuronal activation and inhibition. Furthermore, subcortical processes can be challenging to identify, particularly when the structure remains continuously active. Neurophysiological research utilizing animal models supplements our understanding, especially concerning emotional, cerebellar, and brainstem functions.⁷² Additional methods, such as transient inhibition of neuron groups and the application of retrograde or anterograde transported dyes, enhance the identification of neural pathways and clarify structural interconnections. Lastly, behavioral analysis of patients with highly localized lesions provides further insights into the functional roles of specific brain regions within activation sequences.

This body of research enhances our understanding of the sequential organization underlying neurological processes that contribute to the development and adaptive modification of human skills. The data are particularly relevant for examining the behaviors of children and adolescents whose prefrontal cortex is not yet fully mature. Holstege and colleagues⁷³ have biochemically distinguished the emotional circuits that

influence behavior from an anatomical perspective, identifying highly specific fibers referred to as the 'third sensorimotor system'. At the anatomical-functional level, Allen and Tsukahara⁷⁵, along with Paillard⁷⁴, have documented the activation sequence across various cortical areas and basal ganglia during the transition from intention to observable behavior.⁷² Furthermore, we have redefined the role of sensory feedback and integrated feedback and feedforward loops to clarify how bodily sensations and the cerebellum modulate behaviors during the neurological process^{72,76-77}, which may be crucial in regulating affect and, potentially, cognition.²⁰

Historically, the cerebellum's involvement in affective and cognitive functions has been largely underestimated. Nonetheless, evidence of its role in emotional and attentional processes has emerged through electrophysiological protocols, temporary inactivation of cerebellar nuclei, and observations in patients with cerebellar disorders.^{72,78-82} Recent findings suggest that the cerebellum, particularly through the lateral cortical regions and the dentate nucleus, may play a significant role in modulating and integrating emotional and attentional regulation as it relates to behavioral efficiency.^{20-21,72,82-83}

In the context of ADHD or ADHD-like symptoms, dysfunction within cerebellar feedforward loops may account for difficulties some individuals experience in anticipating the outcomes of their actions or sustaining consistent attention. The third sensorimotor system is probably 'under tension' because of amygdala and cerebellar activity. Impulsivity, therefore, should not be regarded merely as a lack of willpower, but rather as a manifestation of the nervous system's impaired capacity to simulate and adjust behavior prior to execution. The cerebellum thereby represents a crucial structure in elucidating the relationship between motor coordination and attentional regulation.

This hypothesis is further supported by the research of Porges⁸⁴, who systematically identified the activation sequence of subcortical structures and the vagus nerve that modulate affective and emotional behaviors. Emotional security influences amygdala activity and facilitates the development of positive emotional and social competencies.^{20,84} This perspective is consistent with clinical approaches such as '*mindfulness-based stress reduction*' (MBSR), which utilizes experiences of bodily calm to regulate emotional intensity and mitigate specific psychopathological symptoms.⁸⁵⁻⁸⁶ Furthermore, a meta-analysis of approximately twenty studies demonstrates that MBSR increases the efficacy of therapeutic approaches for cancer, pain, and psychological distress.⁸⁷ In addition, perinatal care practices frequently highlight the value of touch and bonding in regulating amygdala activity for both mothers and infants.²¹

Pharmacological treatment may be useful, but interventions should systematically address lifestyle changes. For example, chronic stress related to school failure or any other unstable family situation can create a vicious cycle of relationship insecurity. Parents and clinicians should prioritise student safety to regulate

emotions and calm the amygdala, freeing prefrontal attention and sometimes reducing the need for medication.^{8,20-21,84}

The 'Bottom-Up' Approach

The classical cognitivist approach posits that cognitive processes are dominant over emotional factors, often downplaying the impact of social, systemic, and environmental influences that significantly contribute to psychological distress and psychiatric conditions. However, recent findings indicate that gut-microbiota dysfunction could potentially trigger ADHD-like.^{6,24} Thus, a neurophysiological dysfunction or trauma can disturb the homeostasis of the enteric, cardiorespiratory, or hormonal systems, which may, in turn, adversely affect affective, cognitive, and social functioning. Furthermore, could metabolic disease, via a retrograde signal in the vagus nerve, or the blood system, create neurological dysfunction leading to ADHD-like?

Excessive intestinal permeability allows endotoxins from the digestive tract to enter the bloodstream, potentially leading to systemic inflammation.⁸⁸ For instance, a meta-analysis of 183 studies indicates that dysbiosis may negatively impact mental health, particularly due to the depletion of essential bacteria observed in individuals with mood disorders, bipolar disorder, or schizophrenia.⁸⁹ Furthermore, there are risks associated with diminished absorption of nutrients vital for optimal brain function, including amino acids, vitamins, polyunsaturated fats, and antioxidants, as well as an increased likelihood of synthesizing harmful substances such as indoles, ammonia, and sulfides.⁸⁹

Objective markers were recently identified in a recent meta-analysis⁹⁰ assessing microbial health among 2758 patients and 1847 healthy individuals:

- elevated zonulin levels have been identified in four studies related to bipolar disorder and depression;
- increased concentrations of lipopolysaccharides were documented in two studies, both addressing chronic fatigue and depression;
- elevated endotoxin antibody levels were reported in seven investigations examining bipolar disorder, depression, schizophrenia, and chronic fatigue;
- six studies noted increased scd-14 levels in subjects with bipolar disorder, depression, schizophrenia, and chronic fatigue;
- an increase in low back pain was observed; additionally, two studies included findings on chronic fatigue and depression;
- higher concentrations of alpha-1-antitrypsin were recorded in six studies focusing on bipolar disorder, depression, and schizophrenia.

Recent research suggests that the homeostasis of the microbiota may influence individual personality traits and decision-making processes.⁹¹⁻⁹³ These findings highlight the significance of promoting a healthy diet, as certain additives are associated with an increased risk of type 2 diabetes and mood disorders.⁹⁴⁻⁹⁵ Systemic inflammation has the potential to impair cognitive functions, while

cytokines can both disrupt neurotransmitter synthesis and cross the blood-brain barrier. Additionally, tryptophan may be diverted toward the production of toxic molecules rather than supporting serotonin synthesis, which can manifest in symptoms of depression.⁹⁶ Therefore, the investigation of metabolic psychiatry, including the role of the enteric system and dietary lifestyle habits, in mental illnesses should be considered to enhance patient treatment plans. Furthermore, it is essential to account for the impact of hormonal homeostasis on mental health to achieve a 360-degree comprehensive evaluation of symptoms.²⁴

This 'bottom-up' perspective presents a significant reconsideration of ADHD diagnoses that presently rely exclusively on behavioral observation, supporting the ADHD-like theory. If symptoms such as restlessness and distractibility are manifestations of systemic inflammation or dysbiosis, the ethical implications of prescribing psychotropic drugs become more complex. In such scenarios, there is a possibility that, in addition to the precedingly described side effects, psychostimulants may conceal underlying metabolic distress, specifically within the gut-brain axis, instead of addressing the root biological cause of dysfunction.

Impacts from the Quality of the Therapeutic Alliance

There exists a notable challenge within psychiatry and psychology to integrate two seemingly contradictory realities. Jeannerod⁹⁷⁻⁹⁸ describes the concept of the '*neural subject*' arising from sequential brain region activation. Although neurophysiology provides valuable insights, its reductionist approach is debated. The '*clinical subject*' refers to patients who present their own suffering and retain decision-making autonomy, requiring nuanced understanding. However, the limits of generalizing clinical findings can reduce confidence in empirical results. Ultimately, it is important to consider whether drug prescriptions serve patient needs or are influenced by external factors, such as the physician's desire for quick symptom relief.

A primary challenge in the field of '*affective, cognitive, and social neuroscience*' is establishing a shared framework for communication across different levels of analysis. The overarching aim is to achieve a comprehensive understanding of psychophysiological phenomena while bearing in mind each patient's unique phenomenological experience. Recent studies indicate that similar symptomatology may conceal diverse biochemical, psychosocial factors, or lifestyle habits as underlying causes.⁹⁹⁻¹⁰¹ This insight facilitates the identification of clinical strategies both for prevention of mental disorders and for promoting patient engagement in mitigating symptom severity. Consequently, there is an ongoing question regarding how best to balance patient needs with administrative requirements, all while adhering to regulatory standards for intervention.

Although concerns exist, artificial intelligence resources are anticipated to assist clinicians in identifying indicators of psychiatric disorders, as well as in selecting appropriate pharmacological and psychosocial

treatment strategies. Nevertheless, it is posited that qualities such as therapeutic alliance, empathy, and attentive listening will remain distinguishing factors between ‘machines’ and ‘clinicians’. Furthermore, standardized protocols for psychotherapy and psychopharmacology have already been subject to significant critique.

Henry and collaborators¹⁰² observe that cognitive-behavioural therapies enhance clinicians' sense of competence; however, they may simultaneously impact practitioners' levels of empathy and authenticity, potentially diminishing the quality of the therapeutic alliance. Thus, the therapeutic relationship remains the primary predictor of successful outcomes. A related instance pertains to ‘Eye Movement Desensitization and Reprocessing’ (EMDR). It was previously assumed that eye movements provided relief to patients.¹⁰³ Nonetheless, research indicated no significant difference in outcomes between groups treated by the same therapist with or without EMDR practice.¹⁰⁴ A subsequent meta-analysis encompassing 34 studies affirmed the therapeutic alliance as a critical factor in symptom alleviation.¹⁰⁵ EMDR exercises serve to concentrate patients' thoughts, thereby facilitating the psychotherapeutic process during trauma recollection.

It is essential to critically evaluate the practice of prescribing psychotropic medications without first engaging in respectful, attentive dialogue with patients experiencing distress and thoroughly considering all potential explanations for their symptoms. Clinicians are advised to foster a therapeutic alliance and maintain a high level of trust with patients.¹⁰⁶ Bowlby's ‘attachment theory’ is relevant in this context, as it posits that infants possess an innate biological mechanism prompting them to seek proximity to a protective figure.¹⁰⁷⁻¹¹⁰ The initial relationship not only meets basic physical needs but also significantly affects neurological development, with the quality of attachment shaping brain maturation. A meta-analysis of 79 studies demonstrates that recurring interactions with attachment figures influence neural circuitry, particularly within the limbic system (responsible for emotion regulation) and the prefrontal cortex (involved in cognitive control).¹¹¹ Secure attachment is associated with increased oxytocin secretion and reduced cortisol levels, supporting optimal synaptic development; conversely, chronic emotional deprivation can impede hippocampal growth and disrupt hormonal responses to prolonged stress.¹¹¹⁻¹¹³ Bowlby¹¹⁰ additionally introduced the concept of internal working models, mental representations of self and others stored in procedural memory, which emerge from repeated relational experiences and guide individuals in regulating emotions and managing social relationships throughout adulthood.

Thereafter, Schore proposed that attachment constitutes a process of interactive affect regulation that actively shapes neural development.¹¹⁴⁻¹¹⁵ The initial two years of life represent a critical period during which the right hemisphere—particularly its capacity for non-verbal processing—undergoes rapid growth. Subsequent brain

maturation is contingent upon empathic communication between caregiver and infant. Mechanisms such as eye contact, facial cues, and vocal prosody enable the attachment figure to modulate the infant's emotional states. This attunement facilitates transitions in the child's nervous system from stress to calm, thereby supporting the maturation of neural circuits essential for affective self-regulation. Furthermore, secure attachment serves not only as psychological reassurance but also as a biological imperative for the proper organization of the limbic system and orbitofrontal prefrontal cortex.^{21,84,113-114} Conversely, an inadequate or inconsistent relational environment may disrupt synaptic development and diminish resilience to stress in later life.^{4,6,116}

In 1998, Seligman introduced a new psychological orientation to move beyond traditional definitions of mental disorders.¹¹⁷ His perspective aligns with psychodynamic theories of child development.^{2,8,20,21,84,110,113,119-121} Nonetheless, terminology and concepts have often been misinterpreted, particularly in French-speaking communities, and recommendations have frequently failed to adequately address children's needs.^{2,4,8,122-123} Initially, some individuals value emotions only as resources for guiding behavior, yet emotions themselves are neither inherently positive nor negative; rather, they may be experienced as pleasant or unpleasant, but not ‘good’ or ‘bad’. Additionally, there is a misconception that kindness equates to non-intervention, resulting in insufficient education regarding consistency and clear boundaries for children.¹²² Therefore, we propose a model to clarify these concepts and to articulate educational and clinical interventions for ‘positive mental health’ (PMH), emphasizing the essential role of adults and the importance of emotional security for children.¹²⁴⁻¹²⁵ PMH interventions refer to the capacity to feel, think, and act in ways that promote enjoyment of life and resilience in the face of challenges.

A compromised therapeutic alliance may trigger recollections of adverse childhood experiences, potentially intensifying trauma and reducing patient resilience.¹²⁶ Conversely, a strong therapeutic alliance, characterized by empathy, integrity, and respectful listening, can foster affective resonance that soothes the patient. This supportive environment enables individuals to feel secure as they explore various strategies for enhancing well-being within an authentic therapeutic relationship. Over time, such conditions can improve adherence to pharmacological or psychotherapeutic treatment plans and facilitate greater emotional autonomy. It is important to recognize that resistance to change frequently stems from past trauma or excessive stress levels. Thus, the therapeutic alliance provides opportunities for patients to experience healing and increased well-being. Ultimately, this partnership establishes circumstances conducive to transforming thought patterns, affect regulation, and lifestyle habits in multiple spheres of life, thereby supporting optimal mental health despite psychosocial adversities or neurobiological challenges.

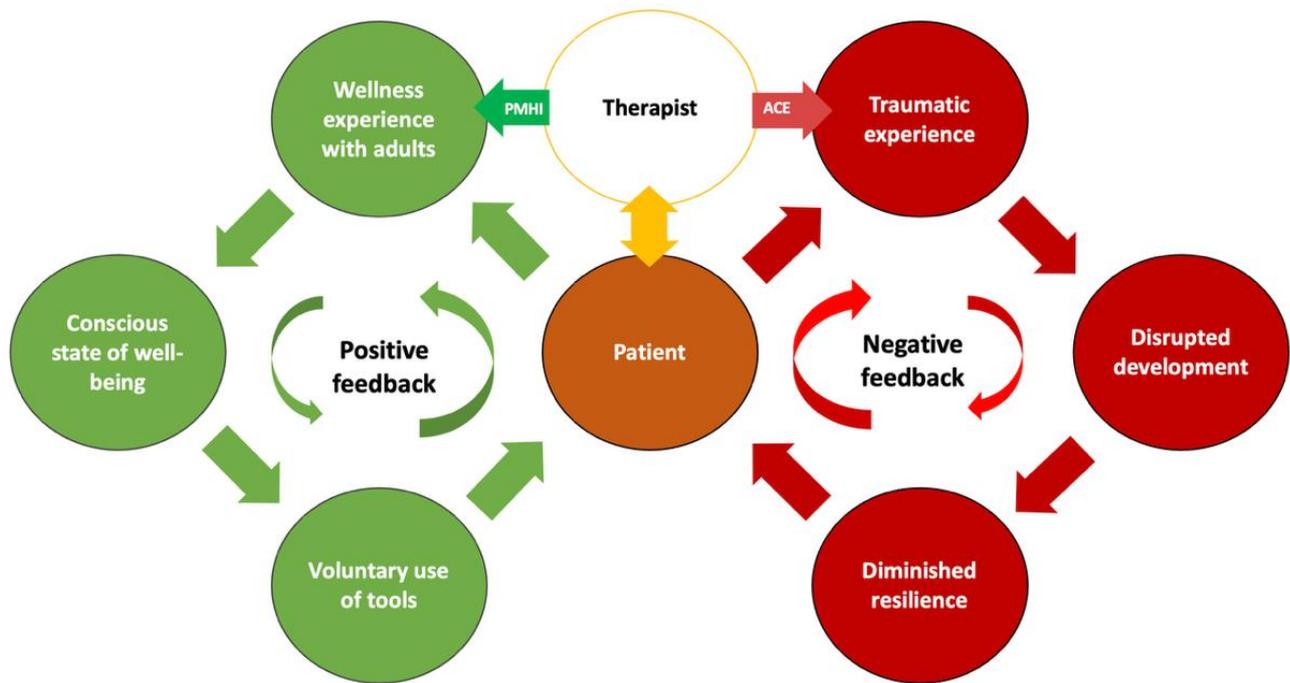


Figure 1: The quality of the therapeutic alliance can influence treatment outcomes by either reactivating and intensifying trauma through ‘negative feedback’ (ACE interventions) or by fostering well-being within the therapeutic relationship via ‘positive feedback’ (PMH interventions). These processes may contribute to alleviating distress or enhancing autonomy (adapted from Monzée 2025¹²⁵).

For individuals with an ADHD profile, the quality of the therapeutic alliance serves as an important biological regulator. By establishing a foundation of emotional security, clinicians promote activation of the social nervous system, which can moderate amygdala responses and facilitate access to prefrontal cortex functions. This attachment-based regulation may, in many cases, enhance attentional capacity in a manner that is both sustainable and considerate, compared to interventions relying solely on pharmacological treatment.

Conclusion

In urgent clinical cases, psychotropic medication may be needed to stabilize a patient before considering other treatments. However, advances in medicine support preventive strategies that lessen symptom severity through cognitive and lifestyle interventions as part of integrative care. This approach considers factors like early experiences, mentalizing abilities, lifestyle, and protective traits to guide individualized treatment plans. Furthermore, environmental and climatic factors, as well as hormonal homeostasis, will also need to be considered. Psychotropic drugs should not be the sole solution. Ethical, comprehensive care is essential. Collaboration with healthcare professionals and family supports strengthens

therapeutic alliances, encouraging behavioral change and symptom relief. Although integrative practices may require more time initially, they can improve autonomy and possibly reverse psychopathology.

In summary, an integrative approach to mental health extends beyond the accumulation of interventions. It signifies a substantive paradigm shift in which psychiatric illnesses are viewed as an entry point to a global understanding of the individual. The initial step involves expanding the conceptualization of mental disorders to encompass not only cognitive processes regulating attention and inhibitory control, but also emotional components and cerebellar mechanisms that influence behavioural responses. The next phase focuses on harmonising ‘Top-Down’ and ‘Bottom-Up’ regulatory processes. By centring the therapeutic alliance within neurobiological regulation, clinicians can transcend basic behavioural management strategies. Adopting these practices reflects a commitment to clinical ethics aimed at restoring physiological balance and enhancing patient autonomy, rather than solely normalising behaviour. While this model may demand a greater initial investment of time, it presents a promising approach for fostering sustainable resilience and achieving meaningful improvement in mental health outcomes.

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