

The Ecological Imperative: Defining Neurodivergence, Treatment, and Diagnosis through the Lens of Lived Experience and Affirming Practice

I. Introduction: Establishing the Neurodiversity Paradigm and Core Definitions

The concept of neurodivergence, which includes conditions such as Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD), necessitates a comprehensive diagnostic and therapeutic approach that integrates traditional clinical measurement with the subjective reality of the individual's lived experience.¹ Both ASD and ADHD are recognized as neurodevelopmental disorders that fundamentally affect brain function and development, often leading to challenges in attention, focus, impulsivity, and executive functioning.² The frequent co-occurrence of these conditions, sometimes termed AuDHD, represents a complex neurological profile requiring nuanced clinical understanding.

The framework employed in this report is the Neurodiversity Affirming Practice (NDAP) lens.³ This model challenges the historical pathology paradigm, moving away from viewing neurodivergence as an illness or "bad difference" to instead conceptualize it as a "mere-difference".⁴ This perspective posits that observed difficulties frequently arise not solely from internal deficits but from the challenging interaction between a non-standard individual and an unaccommodating environment.⁴ Consequently, the goals of expert assessment and support must pivot toward celebrating inherent strengths, upholding personal autonomy, and fostering a positive self-identity.¹

A. Defining Neurodivergence: A Strengths-Based, Contextual Definition

Neurodivergence refers to the natural variation in human neurological functioning.¹ This includes individuals diagnosed with neurodevelopmental disorders such as ASD and ADHD, characterized by similarities in affecting attention, focus, social communication, and executive functioning (EF).² EF is a critical set of mental skills, encompassing working memory, flexible thinking, and self-control, deficits of which present challenges in decision-making, emotional regulation, and prioritization.²

A particularly crucial complex neurotype is AuDHD, the co-occurrence of ASD and ADHD. Research, including meta-analyses, indicates that among individuals diagnosed with autism, approximately 39% also exhibit ADHD traits.² This substantial overlap highlights shared neurobiological pathways, particularly concerning attentional control and executive functioning.⁶ However, the behavioral manifestation of AuDHD involves internal opposition: individuals with ASD often seek routine and sameness and struggle with transitions, while those with ADHD are characterized by seeking constant stimulation and novelty.²

The critical shift introduced by the NDAP framework is that the ultimate definition of neurodivergence should reflect this interaction with the environment.⁴ If the goal is no longer "recovery" from autism or ADHD but rather facilitating an authentic and meaningful life¹, the focus moves to identifying systemic and environmental accommodations necessary for the neurodivergent individual to thrive.³

B. Defining Treatment and Medication Options

Treatment options for neurodivergent individuals, particularly those with ADHD or AuDHD, fall into pharmacological, behavioral, and psycho-educational categories, all requiring careful management by specialized clinicians.⁷

Definition of Medication

Medication in this context primarily refers to psychoactive compounds designed to modulate neurotransmitter systems to support executive functions.

- **Stimulants:** This class includes methylphenidates (e.g., Concerta, Ritalin) and amphetamines (e.g., Adderall, Vyvanse).⁸ These medications primarily function by increasing the levels of dopamine and norepinephrine in the brain, two chemicals

recognized for their influence on executive functioning.⁸ Stimulants are generally considered the most effective pharmacological support for executive functioning deficits associated with ADHD.⁸

- **Non-Stimulants:** These alternatives are also effective and may be used as a first-line treatment or in combination with stimulants.⁷ Atomoxetine (Strattera) is a common non-stimulant that works similarly to SSRIs by elevating norepinephrine levels, which can mitigate core symptoms such as hyperactivity, inattention, and impulsivity.⁹

Definition of Treatment Options

Treatment encompasses a range of evidence-based interventions designed to address skills and environmental fit.¹⁰

- **Behavioral Interventions:** Current clinical guidelines recommend including non-pharmacological interventions in treatment planning.¹⁰ For children under six years of age, parent behavior training (PBT) is recommended as the first-line intervention.¹⁰ When discussing behavioral programs, language must be family-centered to avoid suggesting parents are at fault.¹¹ Other evidence-based strategies include organizational skills training.¹⁰
- **Affirming Therapies:** These interventions are trauma-informed and client-centered.¹² Examples include:
 - **Executive Function Coaching:** This involves specialized coaching that targets executive functions (task initiation, prioritization, time management) and emotional regulation, areas often compromised in ADHD.¹³ Coaching uses questions and modeling to encourage planning and decision-making, helping individuals develop internalized organizational habits.¹⁴
 - **Dialectical Behavior Therapy (DBT) and Radically Open DBT (RO-DBT):** RO-DBT has shown promise for autistic individuals, especially those with co-occurring anxiety and depression.¹⁵ It focuses on emotion regulation, distress tolerance, and interpersonal effectiveness (setting boundaries), facilitating self-acceptance of their neurodivergent identity and improving social interactions without imposing rigid, neurotypical standards.¹⁵

Crucially, all therapeutic interventions must uphold the principle of client autonomy.⁵ Expert practice rejects goals intended to "normalize" the individual at the expense of their physical, emotional, or psychological well-being.¹²

C. The Challenge of Dual Intervention: Biology vs. Ecology

While pharmacological intervention provides essential functional gain by modulating the biological substrate (dopamine/norepinephrine deficits)⁶, the therapeutic process is incomplete if it fails to address the ecological trauma caused by enforced conformity. Stimulants are highly effective in managing core ADHD symptoms, with response rates approaching 70% in autistic children for hyperactivity and inattention.¹⁶ However, if the individual operates in an environment that continuously demands high-effort masking—the suppression of authentic autistic or ADHD traits¹⁷—the associated mental health consequences, such as severe anxiety and burnout¹⁸, persist.

Therefore, effective treatment requires a comprehensive, integrated approach. Medication supports the intrinsic biological needs, providing the necessary executive function resources; however, the neurodiversity-affirming ecological approach must simultaneously work to adjust societal expectations and environments.³ Without this dual focus, the individual uses the functional support gained from medication merely to maintain an unsustainable state of cognitive suppression, delaying, rather than preventing, eventual psychological collapse.

II. Definitive Diagnostic Methods: A Top-Down, Bottom-Up Analysis

The current clinical practice in diagnosing neurodivergence is shifting from solely relying on standardized, top-down diagnostic checklists to incorporating bottom-up, context-driven assessments that prioritize the lived experience of the individual.

A. Traditional Diagnosis (Top-Down): The Deficit Model and Systemic Bias

Traditional diagnostic frameworks, such as the DSM-5, are inherently deficit-focused and have historically struggled to recognize the subtle or camouflaged presentations of neurodivergence, particularly in adults and females.²⁰

Limitations of Standard Criteria

The diagnostic criteria were largely established based on presentations observed predominantly in males, often resulting in a systemic bias. For example, girls with ASD often display better verbal communication and less obvious social difficulties than boys.²⁰ Similarly, girls with ADHD often present with inattentive type symptoms and less disruptive, externalized hyperactivity, leading them to be overlooked by parents, teachers, and clinicians.²⁰

This diagnostic gap is primarily driven by **camouflaging** or **masking**.²¹ Masking involves deliberate efforts to hide or suppress autistic traits to appear neurotypical and includes strategies such as using learned social scripts, imitating behaviors (Compensation), or modifying natural behaviors to blend in (Assimilation).²¹ These survival mechanisms, developed in response to social rejection and the pressure of neuronormativity¹⁷, severely impede accurate clinical identification.

Consequences of Delayed Diagnosis

The difficulty in diagnosis, particularly for those who camouflage, results in profound, long-term psychological distress.¹⁸ Undiagnosed neurodivergent individuals often experience a pervasive feeling of alienation, described as "being an alien" or "being different".²² They frequently struggle with peer relationships and report difficulties with teachers and meeting family expectations, contributing to an external locus of control.²³ This chronic struggle to adapt to an unsuitable environment contributes significantly to co-occurring mental health issues, including anxiety, depression, and sleep or eating disorders, which accumulate over a lifetime of unrecognized effort.¹⁸

B. Side-by-Side Neurocognitive Comparison: Differential Diagnosis of AuDHD

The co-occurrence of ASD and ADHD (AuDHD) presents a complex neurocognitive profile. Both conditions affect the brain's executive functioning, yet they display distinct patterns of impairment that require careful differentiation for effective intervention.²⁴

Overlapping and Distinctive Neurocognitive Profiles

Research indicates that ASD and ADHD share deficits across several neurocognitive domains, including attention processing, performance monitoring, face processing, and sensory processing.²⁵ Critically, deficits in working memory (WM) and general attention are not typically strong discriminators between individuals with ASD alone, ADHD alone, or the AuDHD group.²⁴

However, the AuDHD profile often represents an **additive comorbidity** of specific EF deficits, suggesting that the individual carries the burden of impairments typical of both conditions simultaneously.²⁴

1. **ASD-Associated Deficits:** Impairments in **cognitive flexibility** and **planning/sequencing** are shared between the ASD group and the AuDHD group.²⁴ This aligns with the known difficulties in transitioning between activities and rigid thinking patterns characteristic of autism.²
2. **ADHD-Associated Deficits:** Deficits in **response inhibition** (the ability to stop a prepotent response) are shared between the ADHD group and the AuDHD group.²⁴ This reflects the core challenges of impulsivity and disinhibition central to ADHD.
3. **Sensory Processing:** Sensory differences are highly common in both neurotypes, reaching a prevalence of up to 90% in ASD.²⁷ These differences—which can manifest as sensory seeking or sensory avoidance, often fluctuating by domain—are strongly linked to attentional challenges and behavioral dysregulation.⁶

The co-occurring AuDHD profile is further complicated by the internal conflict arising from the opposing behavioral traits—the autistic preference for sameness clashing with the ADHD drive for novelty.² This continuous, high-effort internal negotiation requires immense cognitive resources for regulation, depleting executive function reserves and accelerating the path toward masking and burnout.¹⁹ Consequently, traditional EF deficits are compounded by this intrinsic internal battle.

| Cognitive Domain | ASD Dominance | ADHD Dominance | AuDHD Profile (Additive Comorbidity) |
|-----------------------|--------------------------|------------------|---|
| Response Inhibition | Variable/Less Pronounced | High Deficit | Shares high deficit with ADHD group ²⁴ |
| Cognitive Flexibility | Significant Deficit | Variable Deficit | Shares significant |

| | | | |
|--------------------------|---|--|--|
| | | | deficit with ASD group ²⁶ |
| Planning and Sequencing | Significant Deficit | Variable Deficit | Shares significant deficit with ASD group ²⁴ |
| Attention/Working Memory | Not Distinctive | Not Distinctive | Not distinctive in discriminating groups ²⁴ |
| Sensory Processing | Extreme Avoidance/Seeking (High Prevalence) ²⁷ | Common, often related to seeking/hypo-sensitivity ⁶ | Highly prevalent; linked to attentional failure and dysregulation ⁶ |

C. Neurodiversity-Affirming Assessment (Bottom-Up): Focus on Ecology and Function

To counteract the limitations of the traditional deficit model, a bottom-up, neurodiversity-affirming approach is required. This methodology integrates standardized psychometrics with a qualitative assessment of real-world adaptive behavior and ecological fit.²⁸

Assessment Method

Formal diagnosis typically involves specialized professionals, such as child and adolescent psychiatrists, psychologists, and developmental-behavioral pediatricians.⁷ For adults and those who camouflage, screening instruments designed to capture nuanced traits are often utilized as an initial step.²⁹ These include self-report questionnaires like the Camouflaging Autistic Traits Questionnaire (CAT-Q), the Ritvo Autism Asperger Diagnostic Scale – Revised (RAADS-R), and the Adult ADHD Self-Report Screening Scale (ASRS).²⁹ These psychometric tests offer comparison scores against neurotypical populations but are explicitly noted as not being substitutes for a formal, comprehensive diagnostic evaluation.²⁹

The Contextual Imperative

The core tenet of ND-affirming assessment is the recognition that function is context-dependent. Real-world adaptive behavior often shows significant variance across different settings (e.g., home vs. school).³¹ Therefore, the assessment must shift the inquiry from "What are the challenges?" to "When and where do these challenges arise?" and "What patterns emerge when environments are adjusted to better fit the individual's neurotype?".²⁸

This approach necessitates prioritizing the individual's internal experience, using measures of safety and regulation as clinical indicators. Clinicians must actively seek to understand the environmental factors that cause dysregulation and distress.¹⁷ This includes assessing for:

- **Sensory Triggers:** What events, activities, or situations in the environment cause the individual to become dysregulated or feel unsafe?¹⁷
- **Burnout and Depletion:** Assessing persistent, deep physical exhaustion and the observable decline in executive functioning abilities, which are hallmarks of autistic burnout resulting from sustained environmental stress.¹⁹
- **Motivation and Interests:** Tapping into core interests to understand intrinsic motivation, rather than demanding compliance.¹⁷

A comprehensive diagnosis, therefore, mandates the synthesis of formal neurocognitive data (top-down) with context-driven adaptive behavior ratings (bottom-up) to accurately determine where an individual's internal challenges meet environmental failure. The fact that functional performance varies across settings implies that relying solely on a single-setting assessment is insufficient, highlighting the clinical necessity of capturing adaptive behavior across ecological contexts.³¹

III. Treatment Options: Comparative Analysis of Interventions

Treatment for neurodivergence must be multimodal, ethical, and tailored to support the specific additive cognitive profile observed in conditions like AuDHD, while strictly adhering to neurodiversity-affirming principles.

A. Defining Pharmacological Interventions

Pharmacotherapy is often a highly effective component of treatment, particularly for managing ADHD traits such as hyperactivity, impulsivity, and inattention.¹⁶

Efficacy and Limitations of Stimulants

Stimulants, such as methylphenidate (the most frequently used psychostimulant)¹⁶, increase dopamine and norepinephrine, resulting in significant improvements in executive function.⁸ In studies involving autistic children, stimulants were associated with favorable responses in 69.4% of treatment episodes related to hyperactivity, impulsivity, and inattention.¹⁶

However, the use of stimulants in AuDHD requires stringent clinical management.⁷ Stimulants appear less effective for individuals with high support needs or those whose profile is dominated by autistic traits, such as a strong preference for routine.⁸ Furthermore, a large percentage of children (66%) experience at least one side effect when treated with stimulants, necessitating a cautious titration strategy.¹⁶ It is clinically recommended to initiate treatment with immediate-release formulations at a very low dose, slowly increasing until ADHD symptoms improve without exacerbating ASD symptoms.⁷ Ethical considerations emphasize the importance of dignity, respect, and client-centeredness in managing pharmacological recommendations, particularly across different racial and ethnic backgrounds.³²

Non-Stimulant Options

Non-stimulant medications provide a valuable alternative or complement. Atomoxetine (Strattera) is utilized as a first-line non-stimulant, working by raising norepinephrine levels to target hyperactivity and inattention.⁷ Non-stimulants are recommended if a child cannot tolerate stimulants, does not respond to them, or may be used adjunctively to improve overall effectiveness and mitigate side effects.⁷

B. Defining Non-Pharmacological Treatment Options

Non-pharmacological interventions focus on skill support, psychoeducation, and environmental modification.⁷ The Neurodiversity Affirming Practice (NDAP) framework critically re-evaluates the goals of these therapies.

Evidence-Based Skill Supports

For younger children (under six), Parent Behavior Training (PBT) is the recommended first-line intervention.¹⁰ Organizational skills training is recognized as an evidence-based intervention for older children and adolescents.¹⁰

Affirming Therapeutic Models

- **Executive Function Coaching:** This client-led approach focuses on internalizing strategies for time management, organization, and task initiation.¹³ By scaffolding problem-solving and guiding reflection, coaching helps individuals develop intrinsic systems rather than relying on external demands.¹⁴ It also specifically emphasizes emotional dysregulation, an area often overlooked by conventional life coaching but central to the ADHD/AuDHD experience.¹³
- **RO-DBT:** Radically Open Dialectical Behavior Therapy is particularly effective for managing co-occurring challenges in ASD, such as anxiety and depression.¹⁵ RO-DBT facilitates emotional regulation and improved social interactions by focusing on self-acceptance, helping individuals identify personal values and goals, and thereby empowering them to embrace their authentic neurotype.¹⁵

The Ethical Imperative: Rejecting Normalization

The NDAP perspective fundamentally critiques therapeutic practices, such as traditional Applied Behavioral Analysis (ABA), that target normalization.³³ ND-affirming providers actively reject approaches that aim to suppress core neurodivergent traits, such as stimming, or that prioritize neurotypical social skills training.¹⁷

This rejection stems from the understanding that forcing observable change (e.g., increased

eye-contact or cessation of fidgeting) often equates to teaching high-cost **masking**—a trauma response that suppresses necessary self-regulation mechanisms.¹⁷ Masking is correlated with severe negative outcomes, including mental health problems, meltdowns, and autistic burnout.¹⁷ If an intervention results in a reduction of external symptoms but simultaneously increases the individual's internal distress and fatigue, it is considered ethically problematic.

When unquestionably harmful behaviors, such as self-injurious behavior, are present, intervention must remain function-based and behavior-analytic.⁴ The goal must be to identify and remedy environmental barriers contributing to distress, rather than pathologizing the individual's neurotype.⁴

Table 2

Comparison of Core Interventions for Neurodivergent Individuals

| Intervention Type | Mechanism of Action / Goal | Target Symptoms/Domains | Neurodiversity Paradigm Alignment |
|------------------------------------|--|--|--|
| Stimulant Medication | Increases Dopamine/Norepinephrine in Prefrontal Cortex ⁸ | Hyperactivity, Impulsivity, Inattention, Executive Dysfunction ¹⁶ | High functional utility; requires client autonomy and monitoring for side effect impact on ASD symptoms ⁵ |
| Atomoxetine (Non-Stimulant) | Norepinephrine Reuptake Inhibition ⁹ | Inattention, Hyperactivity, Impulsivity | Alternative for intolerance or adjunct to reduce side effects ⁷ |
| DBT/RO-DBT | Mindfulness, Emotion Regulation, Distress Tolerance, Self-Acceptance ¹⁵ | Emotional Dysregulation, Co-occurring Anxiety/Depression, Interpersonal Conflict | High alignment; promotes self-advocacy and emotional stability without normalization goals ¹⁵ |
| Executive | Scaffolding for | Task Initiation, | High alignment; |

| | | | |
|--------------------------|--|---|--|
| Function Coaching | planning, decision-making, developing internal systems ¹⁴ | Organization, Time Management, Emotional Regulation ¹³ | client-centered and focuses on genuine skill internalization and autonomy ¹ |
| Traditional ABA | Operant Conditioning focused on externally observable behaviors | Compliance, Skill Acquisition, Elimination of Stimming | Low/Zero Alignment; often enforces masking, causing trauma, and targeting neuronormative outcomes ¹⁷ |

C. Assessing the Internal Cost of Intervention

A critical evaluation of therapeutic efficacy necessitates moving beyond simple external performance metrics. The evidence suggests a concerning causal relationship between apparent behavioral success (e.g., stopping fidgeting or forcing eye contact) and increased internal psychological cost. An observed reduction in neurodivergent traits, if achieved through external pressure or traditional behavior correction, should be viewed as a demonstration of high-effort **masking**.¹⁷

This means that a clinical outcome review must quantify the individual's *internal cost*, including fatigue, anxiety, and distress, alongside any measured external functional gain. If an intervention helps an individual function better but simultaneously drives them toward autistic burnout, the intervention is deemed ecologically unsustainable.¹⁹ Therapeutic choices must ensure that the individual's comfort and reduced internal distress are prioritized over superficial compliance or adherence to neurotypical expectations.¹⁷

IV. Synthesis: The Neurodivergent Ecological Assessment Questionnaire (NEA-Q)

To address the demand for a diagnostic tool rooted in real-world neurodivergent experience, the Neurodivergent Ecological Assessment Questionnaire (NEA-Q) is proposed. This instrument shifts the clinical focus from quantifying internal deficits (the core of the

traditional approach) to quantifying the **ecological barriers and cognitive cost** associated with navigating an unaccommodating environment.

A. Rationale and Developmental Framework

The NEA-Q is designed to capture the nuanced experiences of camouflaging and burnout, which are frequently missed by standard psychometric screens focused only on core DSM criteria.¹⁹ The design synthesizes established psychometric domains—specifically Camouflaging Autistic Traits (CAT-Q)²¹—with emerging research on autistic burnout¹⁹ and the necessity of context-driven assessment.²⁸ By focusing on internal exhaustion and environmental fluctuation, the NEA-Q provides metrics that are directly applicable to determining the necessity for environmental accommodation, rather than individual correction.

B. The Neurodivergent Ecological Assessment Questionnaire (NEA-Q)

The NEA-Q uses a 5-point Likert scale (1 = Never/Rarely, 5 = Almost Always) across four critical domains of lived experience, providing a "bottom-up" metric of functional impairment rooted in environmental mismatch and internal suppression.

Table 3

The Neurodivergent Ecological Assessment Questionnaire (NEA-Q): Lived Experience Metrics

| Assessment Domain | Focus of Inquiry (Lived Experience) | Rationale / Clinical Application | Sample Questions (Abridged) |
|--|--|---|--|
| Domain 1: Cognitive Load & Camouflaging | Quantifying the effort required for social compensation and trait suppression (Masking). ²¹ | Measures the sustained psychological toll associated with late diagnosis and mental health co-occurrence. ²⁰ | 1. I spend significant mental energy analyzing interactions to choose the "correct" response (Compensation). ²¹ |

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| | | | <p>2. I hide my intense interests or knowledge because I fear social rejection (Masking).²¹ 3. I feel exhausted after minimal social interaction, regardless of whether I enjoyed the person (Social Draining).¹⁹</p> |
| <p>Domain 2: Autistic/ADHD Burnout</p> | <p>Assessing the state of functional collapse resulting from sustained stress and masking.¹⁹</p> | <p>A critical marker of systemic failure; quantifies physical and cognitive depletion not captured by standard symptom checklists.¹⁹</p> | <p>4. I feel a persistent, deep physical exhaustion that is not relieved by sleep.¹⁹ 5. My general ability to initiate or complete tasks (Executive Function) has noticeably declined recently.¹⁹ 6. I have withdrawn from interests or hobbies I once loved due to low energy (Depletion).</p> |
| <p>Domain 3: Contextual Executive Functioning</p> | <p>Assessing how EF abilities fluctuate based on environment, interest, and task structure.²⁸</p> | <p>Shifts focus from an inherent "deficit" to "environmental mismatch." Identifies accommodation needs over inherent impairment.³¹</p> | <p>7. My planning and organization skills are excellent for tasks I am interested in, but non-existent for routine tasks.²⁸ 8. I perform much better in focused, quiet environments than in open,</p> |

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| | | | stimulating ones. ²⁸ 9. I struggle with decisions and prioritizing due to the conflict between stability (ASD) and novelty (ADHD). ² |
| Domain 4: Sensory Needs and Safety | Identifying specific sensory triggers that cause dysregulation, protective behavior, or communication shutdown. ¹⁷ | Directly informs environmental adaptation; links behavioral dysregulation to physical discomfort and trauma. ²⁷ | 10. Certain sounds or visual stimuli frequently cause me physical pain or immediate intense anxiety. ¹⁷ 11. When distressed, I often lose access to my spoken language (selective mutism/shut down). ¹⁷ 12. I frequently use self-soothing movements (stimming) to regulate my emotions, but suppress them in public. ¹⁷ |

C. The Ecological Definition of Dysfunction

The structure and focus of the NEA-Q fundamentally redefine neurodivergent dysfunction. Dysfunction is conceptualized not as the failure to meet a neuronormative standard, but as the **unmanageable gap between the individual's specific sensory and cognitive needs and the resources or tolerance provided by their specific environment.**

High scores in domains such as Burnout (Domain 2) and Cognitive Load (Domain 1) serve as clinical proof that the individual's current ecological setting is fundamentally unsustainable for

their neurotype. This approach enables a radical departure from the traditional clinical model where high diagnostic scores trigger intervention focused on correcting the individual. Instead, high scores in the NEA-Q immediately direct intervention toward removing ecological barriers¹⁷ and adjusting the system to fit the individual.³ The purpose of this assessment framework is to transition clinical practice from individual correction toward necessary environmental adaptation.

V. Conclusion: The Ecological Diagnosis of the Neurodivergent Mind

The pursuit of an exhaustive definition, diagnosis, and treatment plan for the neurodivergent mind requires integrating the precision of neurocognitive science with the critical ethics of the neurodiversity paradigm.

Top-to-Bottom/Bottom-to-Top Synthesis

A truly expert diagnostic protocol must incorporate a **top-down** analysis, utilizing neurocognitive profiling to understand the additive nature of executive function deficits in co-occurring conditions like AuDHD (impairment in flexibility, planning, and inhibition).²⁴ Simultaneously, it requires a **bottom-up** assessment, prioritizing the lived, ecological experience captured by tools like the NEA-Q. The internal cost of masking and the presence of autistic burnout¹⁹ become the primary measures of pathological impairment, as they reflect the trauma caused by environmental demands rather than intrinsic deficits.

End Result: A Definition Redefined

The **Neurodivergent Mind** is defined by its specific, additive cognitive profile (particularly in executive function and sensory processing) and its intrinsic vulnerability to ecological mismatch. While pharmacological interventions offer valuable functional support to mitigate internal symptom severity (e.g., ADHD traits)⁸, long-term well-being depends on client-centered treatment that rejects normalization and prioritizes the adaptation of external

systems.

Effective, ethical practice mandates a shift in clinical goals: instead of focusing on conformity, expert practitioners must promote autonomy, nurture positive self-identity³, and help tailor environments to accommodate natural neurological variations.¹ The diagnostic process is therefore not a judgment of pathology, but the crucial first step in identifying the specific ecological modifications necessary for the neurodivergent individual to achieve sustainable functioning and authenticity.

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