Commentary

Zad's 4-Dimensional Model of the Self: An Integrative Conceptualisation

Mohamed Abdel-Maboud[1,2]

1. Al-Hussein University Hospital, Al-Azhar University, Egypt; 2. REMEDI Research Assembly, Egypt

The psyche is an emergent, multilayered system. Neurons, hormones, and genes set the stage; experiences and relationships script the play; thoughts, feelings, and drives write the dialogue; self-reflection and culture direct the performance. Yet despite the wealth of theoretical frameworks, psychoanalytic, cognitive-behavioral, humanistic, biopsychosocial, and neuroscientific, psychiatric education often remains siloed, with each model illuminating only a facet of the human story. This article propose a 4-Dimensional Model of the Self, which unites four cross-domain dimensions (Thoughts, Feelings, Behaviors, and Biology) with a novel metaconscious evaluative layer. The framework honors prior theories while offering an integrated lens for psychiatric reasoning and clinical application, anchoring DSM-5 categories into intuitive, phenomenologically grounded domains, offering a practical heuristic for educators and clinicians to scaffold case conceptualization, and demonstrating its applicability through clinical vignettes in psychosis, depression, and addiction.

Corresponding Author: Mohamed Abdel-Maboud, MohamedAbdel-Maboud.6.206@azhar.edu.eg

Contemporary psychiatric practice often fragments human experience into a collection of checklists, obscuring the rich interplays that underlie mental distress. This article proposes a novel conceptual framework for understanding the human psyche, designed to enhance both clinical reasoning and educational clarity, especially for junior practitioners who may feel fragmented when overly relying on symptom-based reasoning. By organizing mental phenomena into four interrelated dimensions (Thoughts, Feelings, Behaviors, and Biology) with an overlaying Metaconscious evaluative layer, clinicians can approach diagnosis and treatment with greater coherence and compassion. This shift empowers us to understand that psychiatric disorders are not merely collections of symptoms; they are expressions of distress that unfold over time, alter emotion, distort perception, invade the body, or erupt in behavior. The

 doi.org/10.32388/RJWU9J.2

framework requests a developmental and phenomenological lens for understanding the suffering person rather than merely labeling the syndrome.

1. Theoretical Anchoring

1. **Thoughts** evolve from psychoanalytic and cognitive traditions, integrating Freudian unconscious drives[1] and Beck's schemas[2] but extending into metaconscious self-evaluation.
2. **Feelings** draw on affective neuroscience[3] and humanistic emphasis on authentic emotion[4], while addressing regulation gaps in earlier models.
3. **Behaviors** build on behaviorism[5] and attachment theory[6], linking ritual/action patterns to social scripting and personality structure.
4. **Biology** synthesizes modern neurocircuitry and epigenetic research[7], bridging reductionist and systems approaches.

By juxtaposing these alongside the DSM-5's categories, our model transcends silos and illuminates cross-domain interactions.

2. Zad's 4D Model

2.1. Thoughts

1. *Conscious cognition*: real-time perception & awareness (e.g., attention networks)
2. *Subconscious schemas*: memory & mental frameworks (e.g., cognitive distortions)
3. *Unconscious drives*: impulses & defense mechanisms (e.g., Freudian id dynamics)
4. *Metaconscious evaluative self*: self-esteem, narrative identity, coherence (the "observer" that critiques inner dialogue)[8]

2.2. Feelings

1. *Needs-driven*: biological (hunger, sleep) & social motives (affiliation, power)[9]
2. *Experience-driven*: basic emotions (joy, fear, anger, sadness), attachment-related affect (like longing, shame, or contentment) and regulation (Bowlby's attachment emotions)[10]

2.3. Behaviors

1. *Personal tendencies*: approach/avoidance/ritualized actions (Skinnerian operants)[11]
2. *Interpersonal*: attachment patterns & social scripts (Ainsworth's styles)[12]

2.4. Biology

1. *Circuits*: reward (mesolimbic), fear (amygdala), stress (HPA axis)[13]
2. *Neurochemicals*: dopamine, cortisol, serotonin
3. *Plasticity*: epigenetic modulation & neuroadaptation[14]

3. Expanded Metaconsciousness

The metaconscious layer ("evaluative self") mediates between cognition and identity. It is shaped by culture, trauma, and reflective practice:

1. **Culture** imparts narratives that shape life scripts and self-worth[15]
2. **Trauma** can fragment coherence, leading to dissociative defenses[16]
3. **Reflective practice** (e.g., mindfulness) enhances metacognitive control and emotional resilience[17]

3.1. Thoughts

Cultural Schemas

Cognitive frameworks do not develop in isolation; they are shaped by the cultural narratives, values, and belief systems that individuals absorb from an early age. For instance, in honor-based societies, thought patterns often prioritize reputation and social standing, whereas in more individualistic contexts, internal goals and personal achievement may dominate mental schemas. These cultural schemas influence attention biases, memory encoding, and interpretive styles—literature in cultural neuroscience demonstrates that neural networks for self-referential processing (such as the medial prefrontal cortex) respond differently depending on culturally ingrained self-construals[18][19].

Cross-Cultural Cognitive Distortions

While core cognitive distortions—such as overgeneralization or catastrophizing—occur across populations, their content and triggers vary by culture. A clinician using our model would explore how

culturally specific beliefs (for example, fatalism in some communities or mindfulness in Eastern traditions) shape automatic thoughts and deeper schematic structures.

Metaconscious Evaluation

At the metaconscious level, cultural narratives inform the "inner critic" and overarching life story. Cross-cultural research shows that narrative identity differs in collectivist versus individualist societies, affecting self-esteem and coherence [20]. Our framework invites assessment of how these culturally rooted narratives support or undermine reflective self-appraisal.

3.2. Feelings (Expanded)

Needs-Driven Affects

Biological imperatives (hunger, sleep) and social motives (belonging, status) arise from hardwired drives. Culture shapes the needs that are foregrounded and how they are expressed. For example, some societies valorize stoicism while others encourage emotional disclosure.

Experience-Driven Emotions

Basic emotions (joy, fear, anger, sadness) interact with attachment-related affects (longing, shame, contentment). Affective neuroscience identifies the prefrontal–amygdala circuitry that underlies regulation; our model integrates these findings with humanistic ideals of authentic emotion, thereby filling gaps in prior frameworks that omitted neuronal pathways.

Regulation Gaps

Earlier models often left out the neural mechanisms of emotion control. We address this by linking dysregulation, such as rumination or affective inertia, to specific circuits (for instance, reduced prefrontal inhibition of limbic structures).

Cultural Modulation

Cultural norms guide how feelings are labeled and managed. For example, collectivist contexts may discourage overt anger, leading to somatic presentations; our model encourages clinicians to inquire about culturally sanctioned emotion rules.

3.3. Behaviors

Culturally Scripted Rituals

Behaviors often follow scripts learned through cultural practices—rituals that mark transitions, express belonging, or manage distress. For example, communal rites of passage may channel adolescent risk-taking into socially sanctioned activities, whereas in other settings, individuals may adopt solitary coping behaviors. Anthropological and social-psychological studies highlight how ritual enactment engages operant learning systems and symbolic meaning [21][22].

Social Roles and Norms

Behavioral tendencies emerge from roles prescribed by family, religion, or profession. In collectivist cultures, deference and interdependence drive behavioral inhibition; assertiveness and self-expression are rewarded in more egalitarian contexts. Our model prompts clinicians to map patient actions onto these social scripts and consider how departures from expected roles may contribute to distress.

Habitual and Impulsive Patterns

Beyond formal rituals, culture shapes everyday habits—dietary customs, sleep-wake cycles, leisure activities—and even impulsive behaviors, such as substance use patterns that vary by community norms. Behavioral neuroscience links these patterns to reinforcement circuits, demonstrating that culturally patterned rewards modulate the striatum's activity [23].

3.4. Biology

Gene–Culture Coevolution

Biological processes are not solely determined by genetic endowment; they interact with cultural practices in shaping epigenetic marks and neural development. Studies of gene-culture coevolution reveal, for example, that cultural diets can influence microbiome-gut-brain signaling, affecting neurotransmitter synthesis [24].

Epigenetic Modulation

Exposure to cultural stressors—war, migration, discrimination—can leave epigenetic signatures on stress-regulation genes (such as FKBP5), altering HPA-axis responsiveness across generations [25]. Our

model encourages attention to these epigenetic factors as part of the biological dimension.

Cultural Neuroscience Foundations

Neuroimaging research demonstrates that culturally shaped behaviors and beliefs leave discernible traces in brain structure and function. For instance, cultural meditation practices correlate with increased cortical thickness in attention networks [26]. Incorporating such findings, the 4D Model situates biology within an interplay of genetic predispositions and culturally mediated environmental inputs.

4. Visual Framework

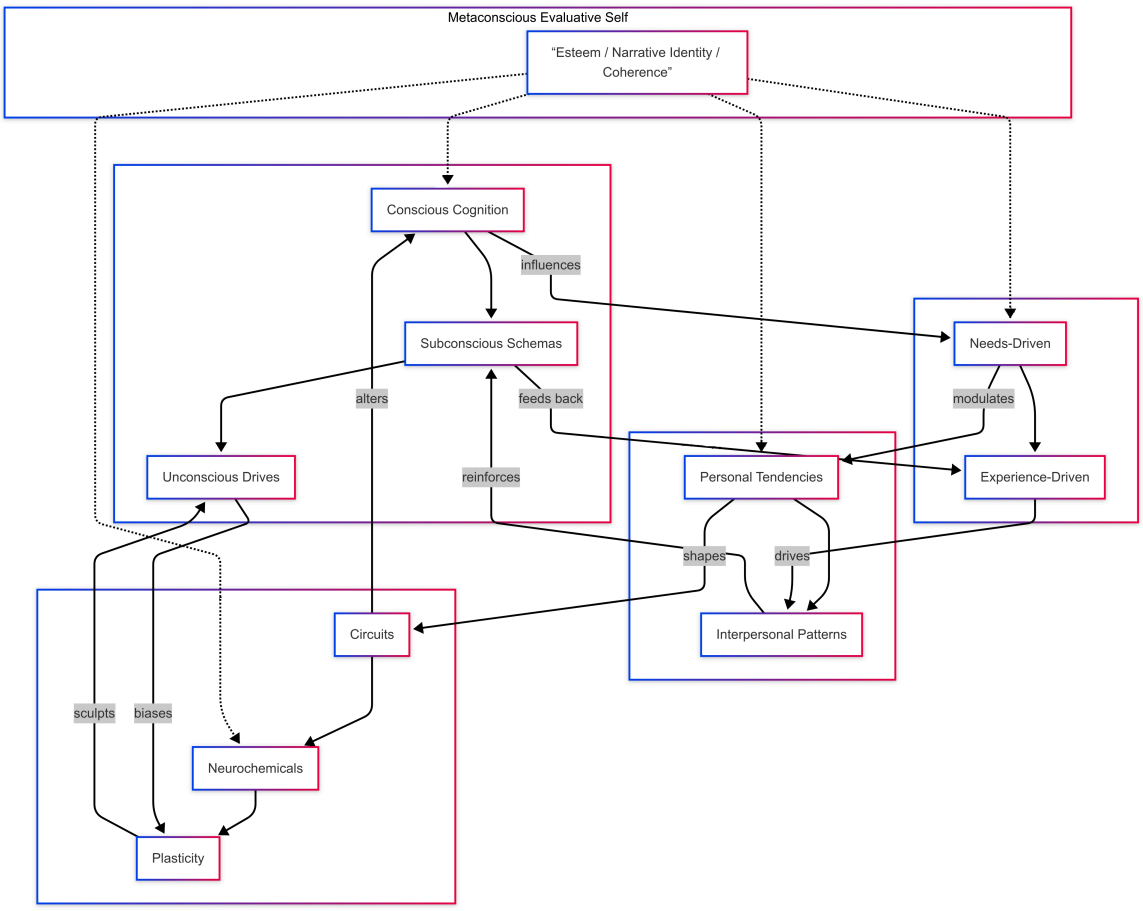

Figure 1. Schematic of Zad's 4D Model with bidirectional inter-domain arrows and the metaconscious evaluative overlay.

Depicting the self as a four-string instrument, if one string is off-tune, the whole melody changes. Biology can influence Feelings, which in turn shape Behaviors; Behaviors reinforce Thoughts; Thoughts modulate Behaviors;

doi.org/10.32388/RJWU9J.2

Behaviors drive Emotions; and Emotions impact Biology.

5. Clinical Vignettes

5.1. Applying the 4D Model in Assessment and Treatment

To translate theory into practice, we propose a four-step clinical flow:

1. Mapping Symptoms to Domains

Clinicians begin by categorizing presenting issues, such as insomnia or self-criticism, under Thoughts, Feelings, Behaviors, or Biology.

2. Identifying Domain Interactions

Next, practitioners trace how disturbances in one area (for example, thought distortions) amplify others (such as sleep-avoidant behaviors or HPA-axis activation).

3. Formulating a Multi-Modal Plan

Interventions are selected for each domain: cognitive restructuring for maladaptive beliefs, emotion-regulation skills for affective instability, habit reversal or behavioral activation for dysfunctional actions, and pharmacotherapy or neuromodulation for biological dysregulation.

4. Monitoring and Refinement

Progress is tracked across all domains, using standardized measures or brief checklists. Adjustments are made when one domain lags, for example, adding mindfulness exercises to bolster metaconscious regulation if narrative coherence remains poor.

Vignette A: Psychosis (Schizophrenia)

A 24-year-old presents with auditory hallucinations and disorganized thought. Our model maps these to a breakdown in:

1. **Thoughts** disrupted: auditory hallucinations, persecutory delusions
2. **Feelings** blunted: affective flattening
3. **Behaviors** disorganized: social withdrawal, disordered speech
4. **Biology** dysregulated: mesolimbic dopamine hyperactivity

5. **Metaconscious** collapse: fragmented narrative identity → cognitive remediation + antipsychotics [27]

Vignette B: Depression (Major Depressive Disorder)

A 45-year-old reports persistent sadness, anhedonia, and fatigue. Mapping onto:

1. **Thoughts**: negative cognitive triad (self-blame, hopelessness)
2. **Feelings**: pervasive sadness, anhedonia
3. **Behaviors**: social withdrawal, psychomotor slowing
4. **Biology**: HPA axis hyperactivity, monoamine deficits
5. **Metaconscious**: self-criticism, cyclical hopeless narrative → SSRI + mindfulness [28]

Vignette C: Addiction (Opioid Use Disorder)

A 30-year-old with opioid misuse shows compulsive rituals (Behaviors) and reward-circuit hijacking. The model guides DBT-informed behavior modification and neurobiological adjuncts

1. **Thoughts**: craving schemas (“just one more dose”)
2. **Feelings**: dysphoria between use episodes
3. **Behaviors**: compulsive drug seeking, ritualized consumption
4. **Biology**: reward-circuit sensitization (ventral tegmental dopamine surge)
5. **Metaconscious**: conflict between self-ideal and actions → DBT + MAT [29]

Vignette D: Borderline Personality Disorder

A 28-year-old presents with chronic feelings of emptiness, intense interpersonal conflicts, and recurrent self-harm urges. Applying the 4D Model:

1. **Thoughts**: "Others will abandon me," rigid splitting schemas
2. **Feelings**: Rapid-onset anger and despair, difficulty soothing intense affect
3. **Behaviors**: Impulsive spending, self-injurious actions during crises
4. **Biology**: Heightened stress-axis reactivity (elevated cortisol), dysregulated serotonergic tone
5. **Metaconscious**: Fragmented self-narrative, alternating idealization and devaluation of self
    - **Intervention Plan**: Dialectical behavior therapy modules for emotion regulation (Feelings), schema-focused work to modify splitting (Thoughts), distress-tolerance skills to curb impulsivity (Behaviors), and SSRIs to address biological hyperarousal.

6. Discussion

6.1.

Zad's 4D Model offers an integrative architecture that both synthesizes and transcends existing psychological schools. By explicitly mapping Freudian drives, Beckian schemas, affective neuroscience, behaviorist operants, attachment patterns, neurocircuitry, and epigenetics into four cohesive domains, anchored by a metaconscious evaluative layer, our framework provides a unified language for describing human experience. This model addresses a key limitation of siloed theories: the lack of a shared ontology that accommodates dynamic, cross-domain interactions. In practice, clinicians can use the model to trace presenting symptoms through multiple levels of analysis, from neurotransmitter imbalances to narrative identity disruptions, thereby avoiding reductionism or over-reliance on any single paradigm.

Clinically, the 4D Model serves as a heuristic for case conceptualization, treatment planning, and interprofessional communication. For example, in depression, pharmacotherapy may target HPA-axis dysregulation (Biology) while cognitive interventions modify negative schemas (Thoughts), mindfulness builds metacognitive awareness (Metaconscious), and behavioral activation addresses withdrawal patterns (Behaviors). Training programs can incorporate the model to help trainees recognize how a single symptom, such as insomnia, may arise from intertwined dysfunctions across domains (e.g., stress-circuit hyperactivity, ruminative thought loops, affective dysregulation, maladaptive sleep habits). By doing so, it cultivates comprehensive, personalized care rather than one-size-fits-all protocols.

From a research perspective, the 4D Model invites quantitative and qualitative studies that measure domain-specific disturbances and their interactions. Factor-analytic work could validate whether questionnaire and neuroimaging metrics cluster into the four proposed domains plus a metaconscious factor. Longitudinal designs could test how shifts in one domain (e.g., improved emotion regulation through therapy) precipitate changes in others (e.g., reduced reward-circuit sensitization or modified self-narrative coherence). Moreover, the model's explicit inclusion of culture and trauma as modulators of metaconsciousness opens avenues for cross-cultural and lifespan research.

6.2. Improvements over DSM-5 and ICD-11

1. Integrated Ontology

Rather than separate symptom lists, our model offers a unified structure that links diagnostic categories to underlying domains.

2. Cross-Domain Interaction

DSM-5 and ICD-11 treat symptoms in isolation. By mapping how Thoughts, Feelings, Behaviors, and Biology influence one another, the 4D Model accounts for dynamic processes, such as how chronic rumination (Thoughts) sustains HPA-axis activation (Biology).

3. Developmental and Cultural Context

Standard nosologies pay limited attention to cultural variation or developmental trajectories. Our framework embeds cultural scripts within each domain and emphasizes how early attachment experiences shape emotion regulation and narrative self.

4. Treatment Sequencing

Current classifications do not guide intervention order. Based on domain severity and interaction patterns, the 4D Model explicitly suggests when to prioritize biological treatments over cognitive or behavioral strategies. By addressing these four gaps, the 4D model enhances diagnostic clarity and informs personalized, multi-modal treatment planning.

Several limitations warrant acknowledgement. First, the 4D Model remains a conceptual framework and requires empirical validation; the boundaries between domains may blur in practice, and certain phenomena (e.g., somatic symptom disorders) straddle multiple domains. Second, integrating such a broad model into routine clinical workflows demands practical tools, such as domain-specific assessment checklists or visual mapping software, that we have yet to develop. Finally, the relative weighting of each domain in different disorders (or even within subtypes of a single disorder) needs systematic study to avoid nominal “domain overload.”

Future directions include (1) operationalizing domain constructs into standardized assessment batteries; (2) designing training modules that teach clinicians to apply 4D mapping in real time; (3) piloting digital tools that allow patients to self-report domain-specific experiences, thereby enhancing shared decision-

making; and (4) exploring how metaconscious interventions (e.g., narrative therapy, mindfulness-based cognitive therapy) specifically bolster coherence and identity integration. Ultimately, by providing a shared, multidimensional framework, Zad's 4D Model aspires to bridge theory and practice, enriching both psychiatric education and patient-centered care.

Footnote 1 (Etymology of "Zad")

The name "Zad" comes from the Arabic root زاد, which refers to provisions or sustenance carried on a journey, whether physical supplies, knowledge, deeds, or essential resources for growth and survival. Metaphorically, we invoke "Zad" to signify the inner reserves and developmental nourishment, across thought, feeling, behavior, and biology, that support the self's ongoing growth and resilience.

Statements and Declarations

Conflicts of Interest

None declared.

Funding

No external funding was received.

Human Subjects Ethical Considerations

Not Applicable.

Author Contributions

Conceptualization: MA; Writing, original draft: MA; Writing, review & editing: MA.

Acknowledgments

We thank collaborators in psychiatric education for insightful feedback during model refinement. Figure 1 was created with the help of ChatGPT and Mermaid.js.org

References

1. ^*Freud S (1923). "The Ego and the Id." SE. **19**:12–66.*

2. ^ *Beck AT (1976). Cognitive Therapy and the Emotional Disorders. New York: Intl. Universities Press.*
3. ^ *Panksepp J (1998). Affective Neuroscience. Oxford: Oxford Univ. Press.*
4. ^ *Rogers CR (1961). On Becoming a Person. Boston: Houghton Mifflin.*
5. ^ *Skinner BF (1953). Science and Human Behavior. New York: Macmillan.*
6. ^ *Ainsworth MD, et al. (1978). Patterns of Attachment. Hillsdale (NJ): Erlbaum.*
7. ^ *Meaney MJ (2010). "Epigenetics and the Biological Definition of Gene–Environment Interactions." Child De v. **81**(1):41–52.*
8. ^ *Flavell JH (1979). "The Developmental Psychology of Metacognition." Monogr Soc Res Child Dev. **44**(1):i–11 2.*
9. ^ *Deci EL, Ryan RM (2000). "Self-Determination Theory." Psychol Inq. **11**(4):227–68.*
10. ^ *Bowlby J (1988). A Secure Base. New York: Basic Books.*
11. ^ *Bijou SW, et al. (1968). "Child Development: Reinforcement and Imitation." J Child Psychol Psychiatry. **9**(2):6 9–90.*
12. ^ *Main M, Solomon J (1990). "Disorganized Attachment Classification Procedures." In: Greenberg MT, et al, ed s. Attachment in the Preschool Years. Chicago: Univ. Chicago Press.*
13. ^ *Arnsten AFT (2009). "Stress Signalling Pathways That Impair Prefrontal Cortex Structure and Function." N at Rev Neurosci. **10**(6):410–22.*
14. ^ *Zoghbi HY (2003). "Role of MeCP2 in Neurological Diseases." J Neurosci. **23**(29):9020–31.*
15. ^ *Markus HR, Kitayama S (1991). "Culture and the Self." Psychol Rev. **98**(2):224–53.*
16. ^ *van der Kolk BA (2014). The Body Keeps the Score. New York: Viking.*
17. ^ *Kabat-Zinn J (1990). Full Catastrophe Living. New York: Delta.*
18. ^ *Markus HR, Kitayama S (1991). "Culture and the Self: Implications for Cognition, Emotion, and Motivation." Psychol Rev. **98**(2):224–253.*
19. ^ *Chiao JY, Harada T, Komeda H, Li Z, Mano Y, Saito DN, et al. (2010). "Neural Basis of Individualistic and Colle ctivistic Perspectives on the Self." Hum Brain Mapp. **31**(1):67–74.*
20. ^ *Wang Q (2006). "Culture and the Development of Self-Knowledge." Curr Dir Psychol Sci. **15**(4):182–187.*
21. ^ *Turner V (1969). The Ritual Process: Structure and Anti-Structure. Aldine Publishing.*
22. ^ *Cohn MA, Fredrickson BL, Brown SL, Mikels JA, Conway AM (2009). "Happiness Unpacked: Positive Emotio ns Increase Life Satisfaction by Building Resilience." Emotion. **9**(3):361–368.*
23. ^ *Schultz W. "Behavioral Theories and the Neurophysiology of Reward." Annu Rev Psychol.*

24. ^ *Kim Sterelny, Explaining Culture: A Naturalistic Approach. Dan Sperber, Mind, Volume 110, Issue 439, July 2001, Pages 845–854*

25. ^ *McGowan PO, Sasaki A, D'Alessio AC, Dymov S, Labonté B, Szyf M, et al. (2009). "Epigenetic Regulation of the Glucocorticoid Receptor in Human Brain Associates with Childhood Abuse." Nat Neurosci. **12**(3):342–348.*

26. ^ *Tang YY, Holzel BK, Posner MI (2015). "The Neuroscience of Mindfulness Meditation." Nat Rev Neurosci. **16**(4):213–225.*

27. ^ *Morrison AP (2013). "Cognitive Therapy for Psychosis." Behav Cogn Psychother. **41**(3):299–33.*

28. ^ *Farb NAS, Anderson AK, Segal ZV (2012). "The Mindful Brain." Can J Psychiatry. **57**(2):70–9.*

29. ^ *Marlatt GA, Gordon JR (1985). Relapse Prevention. New York: Guilford Press.*

Declarations

Funding: No specific funding was received for this work.

Potential competing interests: No potential competing interests to declare.