Review

Biological Psychiatry

Facilitating Thought Progression: A Neurocognitive Framework Linking Thought Dynamics and Mood Disorders

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ABSTRACT

Mood and thought are tightly coupled, but the mechanisms that link them are not understood. This link is particularly important when considering mood disorders such as depression. We propose the facilitating thought progression (FTP) framework, which characterizes depression as a disorder of thought dynamics, encompassing both the temporal evolution and semantic expanse of mental activity. Five parameters jointly determine the fluency of thought progression: breadth, speed, flexibility, novelty, and scope. Diminished progression, reflected by repetitive, slow, and narrowed thinking, is associated with excessive inhibition, reduced plasticity, blunted reward signaling, and overly deep attractor dynamics. Conversely, interventions that enhance associative expansion, cognitive flexibility, and exploratory behavior restore adaptive brain dynamics. FTP reframes depression as a disorder of cognitive flow rather than content and provides a mechanistic, testable model for translational research.

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Major depressive disorder and related mood disorders are among the most disabling conditions worldwide, affecting more than 280 million individuals and contributing substantially to the global disease burden (1). Mood shapes nearly every domain of psychological functioning; it influences perception (2), memory (3,4), attention (5), motivation (6), decision making (7), physical health (8,9), and vulnerability to mental disorders (10). However, despite advances in pharmacological and psychotherapeutic treatments, the neurobiological mechanisms that give rise to core depressive symptoms such as psychomotor slowing, cognitive rigidity, and anhedonia remain incompletely understood.

A central but underexamined feature of depression is that it alters the way thoughts unfold over time. Thinking is inherently dynamic; the mind progresses from one concept to another through a semantic network of memory representations (11). Thinking can advance quickly, traverse broad associations, and flexibly shift perspectives. Alternatively, thinking can be slow or remain confined to a narrow thematic region (12–14). Depression is consistently marked by ruminative, repetitive, and sluggish thinking (15). Although extensive research has focused on the content of those negative thoughts (14,16–19), far less attention has been allocated to the possibility that the dynamics of thinking play a role in mood.

Growing evidence supports this view. Neural studies have shown that depression is associated with reduced variability and altered transitions in large-scale networks, including the default mode, salience, and frontoparietal networks (15,20). Recent dynamic modeling of spontaneous thought further suggests separable clusters, including stuck patterns versus more flexible mind wandering, consistent with an emphasis on trajectories rather than content alone (21).

Here, we propose the facilitating thought progression (FTP) framework, which is a mechanistic model that links mental flow and mood through convergent neurocognitive paths. FTP identifies 5 dimensions that jointly determine how fluidly the mind traverses the representational landscape. Each dimension reflects a neurocognitive process that may be altered in depression. Below, we describe each dimension and discuss how their modulation may restore adaptive mood dynamics.

DIMENSIONS OF THOUGHT PROGRESSION AND THEIR RELATIONSHIPS TO MOOD

Breadth: Associative Expansion and Network Connectivity

Breadth refers to the semantic distance between successive thoughts. Positive mood reliably increases associative breadth, enabling remote associations and recruitment of broader semantic fields (22–25). Behaviorally, broadened states display more unusual word associations. In contrast, negative mood constrains associative spread, producing short-range, repetitive transitions that characterize ruminative thought (26,27). Individuals with depression often cycle through a narrow cluster of representations with minimal semantic distance, which is a hallmark of maladaptive spontaneous thought (28). Longitudinal evidence indicates that this narrowing of associative trajectories predicts later depressive symptoms (29–31), suggesting a mechanistic role in mood deterioration. The link between breadth of thought and mood

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is bidirectional because experimental manipulations that increase associative breadth causally improve mood (32–34).

Semantic priming provides a complementary assay of associative dynamics by indexing spreading activation. In depression, priming effects are often altered, although they vary with task parameters (e.g., automatic vs. controlled retrieval), consistent with state-dependent changes in the efficiency of associative propagation rather than content-specific effects (35). Neuroimaging work further indicates that depression alters the neural implementation of priming (36). Importantly, priming can shift with clinical improvement, suggesting partial normalization of associative dynamics with recovery (37). Although relatively few studies have tracked semantic priming across antidepressant treatment, antidepressants reliably modify emotional information processing, suggesting that pharmacotherapy could alter priming-related indices of associative propagation (38,39).

At the neural level, reduced breadth corresponds to lower variability and diminished functional integration within the default mode network (DMN), along with weakened coupling with frontoparietal systems (20). Such reduced network flexibility limits the brain's ability to traverse distant semantic spaces, reinforcing the behavioral narrowing that is seen in depression.

Clinical interventions, including psychedelic-assisted therapy, have been shown to enhance large-scale network integration and reduce depressive symptoms, consistent with the broadening of associative trajectories (40).

Speed: Temporal Dynamics of Neural Processing

Speed reflects the temporal rate at which thoughts evolve. Behaviorally, individuals with depression exhibit decreased thought speed (41–44) and broader deficits in processing speed (45,46). These cognitive signatures parallel psychomotor retardation (47), fatigue (48), and diminished motivation (49), highlighting speed as a cross-cutting dimension of depressive impairment.

Depression is associated with slowed cortical dynamics and reduced metastability, which is an index of how rapidly the brain shifts between transient functional configurations (15). Mechanistically, slowed thinking has been linked to an imbalance in cortical excitation-inhibition dynamics and alterations in thalamocortical oscillatory activity, both of which constrain neural transition rates (15,50).

Clinically, accelerating cognitive tempo through rapid reading or thought-acceleration paradigms reliably elevates mood (51–53). Interventions that enhance fast-frequency neural activity, such as gamma-range neuromodulation, likewise increase mood and cognitive fluency by restoring the rate of neural transitions.

Flexibility: Cognitive Switching and Neural Reconfiguration

Flexibility refers to the ability to shift between mental sets, perspectives, or strategies and depends on the capacity of large-scale neural networks to reconfigure efficiently. Depression is marked by reduced cognitive flexibility, which is evident in impaired task switching (54), lower divergent and convergent creative output (23,32,55–57), and difficulty disengaging from habitual thought patterns (58,59). Importantly, cognitive inflexibility prospectively predicts the onset of depressive episodes, highlighting flexibility as a vulnerability factor (59,60).

At the neural level, flexibility is supported by functional diversity within the frontoparietal control network (61,62) and by the reconfiguration of prefrontal-hippocampal circuits (63,64). Depression is associated with reduced network diversity and diminished synaptic adaptability, limiting the system's ability to shift between cognitive modes (65,66). These constraints map directly onto behavioral inflexibility; when networks cannot disengage from a current state, thought becomes repetitive and rigid.

Pharmacological interventions that enhance plasticity provide evidence for a causal link between neural reconfiguration and cognitive flexibility. Psilocybin increases both neural and cognitive flexibility in patients with depression through serotonergic and network-level mechanisms (67), whereas ketamine promotes rapid synaptogenesis and dendritic growth, enabling new cognitive trajectories (65). These findings converge with behavioral work showing that inducing creative thinking (32,68) or increasing task-switching demands improves mood by facilitating exits from maladaptive cognitive loops.

Novelty: Reward-Driven Exploration and Cognitive Updating

Novelty reflects mentally visiting less-frequented cognitive paths, and it engages neural systems that promote exploration. Patients with depression exhibit compromised processing of novel stimuli and a tendency to return to familiar cognitive themes (69–71). Novel information elicits phasic dopaminergic responses in the mesolimbic system, particularly the ventral tegmental area and nucleus accumbens, linking exploratory cognition to intrinsic reward (72,73). These signals facilitate shifting away from overlearned representations and support the updating of internal models, making novelty a key driver of cognitive progression. In depression, this novelty-reward linkage is impaired. Blunted dopaminergic signaling and reduced reward sensitivity diminish the motivational value of novel information (74,75). Consequently, thought patterns fail to activate reward circuitry, thereby depriving cognition of the intrinsic reinforcement that normally encourages exploratory transitions. Consistent with this view, real-world indices of exploration (e.g., variability in daily movement patterns) have been linked to affect and social connectivity, supporting associations between exploratory behavior and well-being (76).

Interventions that re-engage novelty processing may restore exploratory thought. However, chronic engagement in ruminative, repetitive thought has been associated with structural alterations in cortical volume (77), suggesting that reversing entrenched cognitive patterns may require sustained effort and extended training.

Scope: Local-Global Balance in Cognitive Integration

Scope refers to whether attention and thought are locally or globally focused. Depression is characterized by a local

attentional scope (78) and the attentional scope model of rumination proposes that such narrowing reduces the likelihood that alternative or positive representations enter awareness (79). In contrast, positive mood broadens attentional and conceptual scope, increasing the probability that new information will be incorporated. This broadening effect has been demonstrated across multiple domains, including attentional selection (80,81), perceptual processing (16), associative breadth (82), abstract construal (83), and behavioral action tendencies (84).

At the neural level, narrowed scope reflects reduced top-down modulation from frontoparietal and fronto-cingulate control regions, which impairs global integration and promotes a local, self-focused processing style (85–87). This diminished control allows DMN dominance and restricts transitions to attentionally broader, contextually informed representations.

Clinically, these mechanisms align with therapeutic strategies that explicitly aim to expand perspective and broaden cognitive scope, including metacognitive therapies, reappraisal-based approaches, and attentional-broadening interventions (88,89). Biological interventions reinforce this principle; psilocybin treatment has been associated with enhanced large-scale network integration and increased coherence across distributed cortical systems (40), which are changes that likely facilitate global information integration.

Interaction and Dissociability of FTP Dimensions

The 5 dimensions that define the multiscale landscape of thought progression are distinct yet somewhat interdependent; for example, reduced speed can constrain associative breadth, and impaired flexibility may amplify the effects of excessive inhibition on scope. At the same time, interdependence does not imply redundancy. In an integrated cognitive system, the dimensions remain conceptually and computationally distinguishable and can, in principle, vary independently even if they often covary clinically. Because they interact, empirical progress requires definitions that permit dissociation. Breadth indexes how far successive thoughts travel in semantic space; speed indexes the rate of transitions; flexibility indexes the probability of switching away from an active attractor, goal, or cognitive set; novelty indexes the selection of low-frequency, high-information-gain states; and scope indexes the integration window or level of abstraction, which can vary even when semantic distance does not (Figure 1). Furthermore, FTP does not assume that more is always better; each dimension likely relates nonmonotonically to adaptive functioning, with an optimal balance between exploration and stability. Candidate behavioral assays, computational metrics, neural readouts, and measurement guidance are summarized in Figure S1.

MECHANISMS LINKING THOUGHT DYNAMICS AND MOOD

Cortical Inhibition and the Constriction of Thought Flow

Depression is associated with dysregulated prefrontal control, often manifesting as inefficient or reduced recruitment of the dorsolateral prefrontal cortex (dlPFC) during demanding cognitive control tasks (90,91). In other contexts, particularly

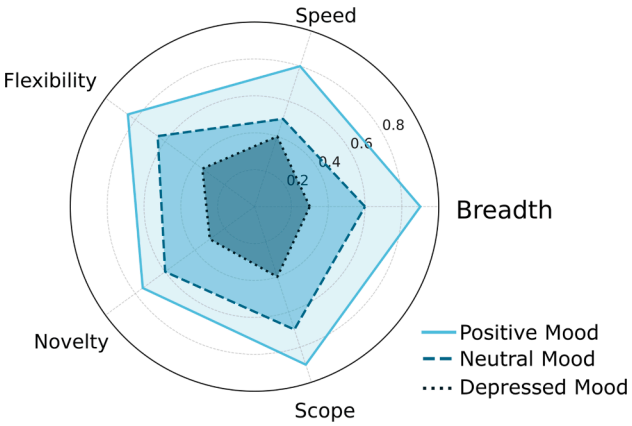

Figure 1. Relating the dimensions of facilitating thought progression and mood: A schematic radar-plot illustration of the proposed dimensions of thought progression, showing potential individual profiles associated with positive mood (light blue), neutral mood (blue), and negative mood (dark blue). Different combinations of these thought dimensions are expected to yield different affective states.

those involving negative self-referential processing, compensatory control, or rumination, depression can instead be associated with increased prefrontal engagement (92). These context-dependent alterations can yield excessive top-down inhibition on associative and mnemonic systems [including hippocampus and medial temporal cortex (50,93,94)], thereby narrowing the progression of thought and increasing the tendency to remain within familiar self-referential basins (94).

In computational terms, heightened inhibition reduces the transition probability between thoughts, leading to cognitive inertia and perseverative loops (95). Conversely, interventions that regulate cortical inhibition, such as serotonergic psychedelics, transcranial magnetic stimulation (TMS), or GABAergic (gamma-aminobutyric acidergic) modulation, can restore thought progression by increasing neural entropy and enhancing connectivity between large-scale networks (40,96). For example, psilocybin has recently been found to reduce rumination and thought suppression in patients with depression (97). Along these lines, evidence from creativity research suggests that reduced inhibition in combination with broader associative activation enables more flexible and creative thought (70,98,99). This highlights the role of inhibitory mechanisms in regulating the breadth and fluidity of thought, such that excessive inhibition constrains associative movement while moderate inhibition supports exploratory cognitive trajectories.

Note that within the FTP model, a moderate level of inhibition should be beneficial for stabilizing thought while allowing flexible transitions. Both extremes, hyperinhibition (ruminative stagnation) and hypo-inhibition (disorganized or psychotic thought), represent departures from optimal thought progression (100,101).

Cognitive Load and Resource Bottlenecks

Although excessive inhibition constrains thought through increased top-down suppression, cognitive load restricts thought progression through resource depletion. These mechanisms can co-occur but represent distinct pathways to

cognitive stagnation. Rumination imposes an endogenous cognitive load that draws heavily on working-memory and executive-control systems, limiting the capacity for representational updating (102,103). Neuroimaging work demonstrates that depression is associated with dlPFC-parietal resource depletion and reduced prefrontal efficiency during demanding tasks (2,104), consistent with impaired gating of working-memory representations as described in computational models of control. Along similar lines, extended durations of depleted resources induce mental passivity (105). High cognitive load narrows associative breadth and reduces the spread of activation (106,107), thereby constraining thought progression even when inhibition is not elevated. In this context, negative thoughts repeat not because of their content but because the system lacks the resources to progress beyond them.

Sleep disturbance is likely to exacerbate this resource-bottleneck pathway. In depression, insomnia and fragmented sleep are associated with poorer cognitive performance, including executive function and processing speed (108,109). Experimental acute sleep loss and fatigue studies further indicate similar reductions in control efficiency and increased perceived effort (110,111), thereby lowering the capacity to sustain adaptive transitions in thought. Within FTP, sleep-related depletion is expected to manifest as longer dwell times, reduced transition rates, and narrower semantic trajectories.

Attractor Dynamics and Reduced State Transitions

A complementary computational account of thought stagnation comes from attractor-network models of cognition. When inhibition is high or network flexibility is reduced, thought tends to settle into habitual attractor states characterized by repetitive patterns (112). This is consistent with the constructed theory of emotion, which proposes that the depressed mind fails to effectively update internal models in response to new information (113), as well as recent computational accounts suggesting that mood disorders are marked by low transition probabilities between top-down and bottom-up processing states (95). Empirically, negative mood has been shown to hinder learning from new experiences (114), and the states of mind framework similarly posits that healthy cognition depends on fluid transitions between internal states that are disrupted in depression (115). Crucially, attractor dynamics represent an emergent systems-level property of recurrent network interactions and therefore cannot be reduced to excessive inhibition or cognitive load; rather, they reflect a distinct mechanism through which depression deepens basin stability, increases dwell time in maladaptive states, and restricts transitions through thought space. Figure 2 schematizes this contrast by illustrating a more traversable landscape in which thought moves more broadly across states versus a deeper, dominant attractor in which thought becomes stuck for longer periods.

Reward Deprivation and the Cost of Stagnation

Depression is marked by profound disruptions in reward processing, which are reflected in reduced dopaminergic transmission within the mesolimbic system, including the ventral tegmental area, nucleus accumbens, and orbitofrontal cortex (116). FTP proposes that mental progression itself is intrinsically rewarding; traversing novel or richly connected associative paths engages dopaminergic and opioidergic systems, thereby generating hedonic value (117–119). This view is consistent with evidence that information seeking, curiosity, and epistemic surprise are rewarding even without external reinforcement (120–122). Importantly, the hedonic value of richly connected information declines under cognitive load (123,124), and repeated information is frequently experienced as aversive (125,126), suggesting that novelty and associative richness are central drivers of intrinsic reward.

Opioidergic mechanisms may also contribute. Mu opioid receptor density increases along the visual cortical hierarchy, implying that deeper and more elaborated perceptual processing triggers greater endorphin release (127). Therefore, dysregulated opioid activity in depression (128) may diminish the hedonic impact of cognitive exploration and contribute to reduced novelty seeking (107,129).

Critically, FTP frames reward deprivation, reduced exploration, and thought rigidity as a mutually reinforcing feedback loop; blunted reward signals reduce exploratory transitions, yielding more predictable, low-information trajectories that further diminish intrinsic reward. The system can thus become self-maintaining even when the initial driver is not reward-related (e.g., cognitive load or inhibitory constraint) because reduced exploration progressively erodes intrinsic reinforcement.

Restoring the reward-exploration cycle requires interventions that enhance dopaminergic sensitivity and reintroduce cognitive novelty seeking. Behavioral activation can increase contact with reinforcing experiences and environmental novelty (130), and cognitive behavioral therapy can introduce and train new cognitive strategies that promote exploratory engagement (131,132). Pharmacological and neuromodulatory approaches that enhance reward sensitivity or reduce excessive constraint may likewise increase the hedonic value of cognitive progression, thereby counteracting anhedonia. We further hypothesize that when thought becomes persistently repetitive and low in informational gain, reduced intrinsic reward may contribute to anhedonia. Repetition itself is not inherently maladaptive; rather, FTP emphasizes trajectories that are low in informational gain and fail to update internal models. In this context, reduced discovery of novel associations may limit intrinsic reinforcement, sustain negative affect, and perpetuate rumination (116,133,134). This account generates testable predictions; individuals with greater thought rigidity should show reduced exploratory choice and blunted reward reactivity.

Plasticity and Cognitive Flexibility

At a cellular level, depression is characterized by reduced synaptic and dendritic plasticity across prefrontal and hippocampal regions (65,66). Several accounts propose that these plasticity deficits may precede and amplify depressive symptoms, limiting the brain's capacity to update internal models or incorporate new emotional and cognitive information (65,135). When neural circuits cannot readily modify synaptic weights or generate new dendritic connections, the formation of novel associative links is constrained, reducing

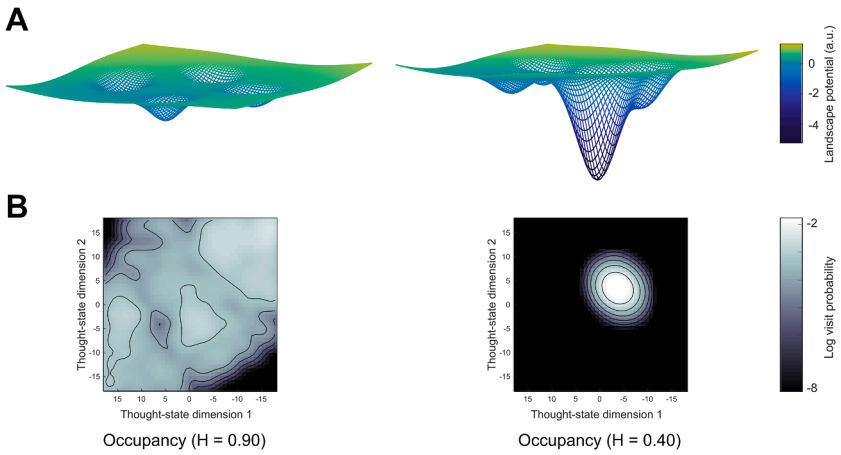

Figure 2. Schematic attractor landscapes and state visitation in thought progression vs. thought stagnation: **(A)** Conceptual thought landscapes in a 2-dimensional state space. The left panel shows a more traversable landscape with shallower attractors, whereas the right panel shows a depression-like landscape with a deeper dominant attractor, reflecting greater trapping of thought. **(B)** Corresponding trajectory-based state visitation. Shading indicates log-visit probability, and H indicates normalized Shannon entropy, with lower values reflecting more concentrated time in fewer states and higher values reflecting broader exploration. *Occupancy maps were derived from simulated noisy gradient-based trajectories, followed by spatial binning, smoothing, normalization, and log transformation. Axes represent abstract thought-state dimensions.

the system's ability to sustain flexible cognitive trajectories. Reduced plasticity therefore contributes to affective inertia, cognitive rigidity, and potentially the onset of depressive states themselves (136).

Pharmacological treatments that enhance plasticity provide causal support for this mechanism. Ketamine and serotonergic psychedelics induce rapid synaptogenesis and dendritic remodeling, opening short-lived "windows of plasticity" in which new cognitive and emotional associations can form (67). Similarly, selective serotonin reuptake inhibitors have been shown to improve neuronal plasticity over longer timescales (65). Within the FTP framework, these interventions expand the brain's representational landscape, enabling more diverse and fluid thought trajectories.

These physiological observations converge with computational psychiatry models that describe depression as a low-variability regime in which brain activity becomes overly predictable and transitions between neural states are infrequent (137). By enhancing plasticity, FTP-based interventions may shift the system back toward a flexible, metastable regime, restoring adaptive variability in both thought and mood.

THE FTP FRAMEWORK: MECHANISTIC AND TRANSLATIONAL IMPLICATIONS

FTP as a Mechanistic Model for Mood Disorders

FTP functions as a translational model bridging basic neuroscience, computational psychiatry, and intervention science. It shifts emphasis from the content of thought to the transitions between mental representations, reframing depression as a disorder of state-space mobility. The core dimensions of cognitive progression map onto identifiable neural mechanisms: associative breadth is related to large-scale network connectivity; thought speed reflects the temporal properties of cortical dynamics and excitation-inhibition balance; cognitive flexibility depends on prefrontal-hippocampal connectivity dynamics and network reconfiguration; novelty processing engages dopaminergic mechanisms that support exploratory cognition; and scope is governed by frontoparietal control over global versus local integration.

When dysregulated, these processes collectively produce the cognitive rigidity and reward deficits that are characteristic of depression. According to this view, rumination is not merely a symptom but a mechanistic contributor that pushes cognition into a low-entropy, low-variability regime, restricting transitions across representational space.

Lastly, although FTP emphasizes thought dynamics over thought content, the two likely interact. Threat-related content may increase avoidance, narrow attention, and raise the perceived cost of exploration, thereby deepening attractor basins and reducing transition probabilities. In such cases, constrained dynamics may be partially driven by negative content that biases cognition toward safe, familiar trajectories.

Importantly, constrained dynamics can remain maladaptive even when content is not overtly negative if the system persistently fails to update models or integrate broader context. Even initially positive content may become problematic when cognition remains low in informational gain over time; novelty and learning signals decline, intrinsic reward diminishes, and repetitive loops become more likely. Over time, this reward depletion may shift affective tone and motivation, linking seemingly benign content to depressive persistence through the same reward-exploration-rigidity feedback loop. Therefore, FTP treats content as a context-setting influence on trajectories, whereas dynamic parameters determine whether cognition can flexibly move out of those content-defined basins.

Translational Implications and Therapeutic Targets

Because FTP specifies the cognitive and neural mechanisms that support healthy thought flow, it highlights multiple intervention targets for restoring adaptive dynamics in depression. One therapeutic pathway involves reducing excessive inhibition and prefrontal hypercontrol. Treatments such as psilocybin, TMS, or GABAergic modulation can transiently relax top-down constraints, increase neural entropy, and enable broader associative exploration (40,96). A second avenue centers on enhancing reward responsiveness and novelty engagement. Pharmacological agents that boost dopaminergic or opioidergic signaling, as well as behavioral strategies

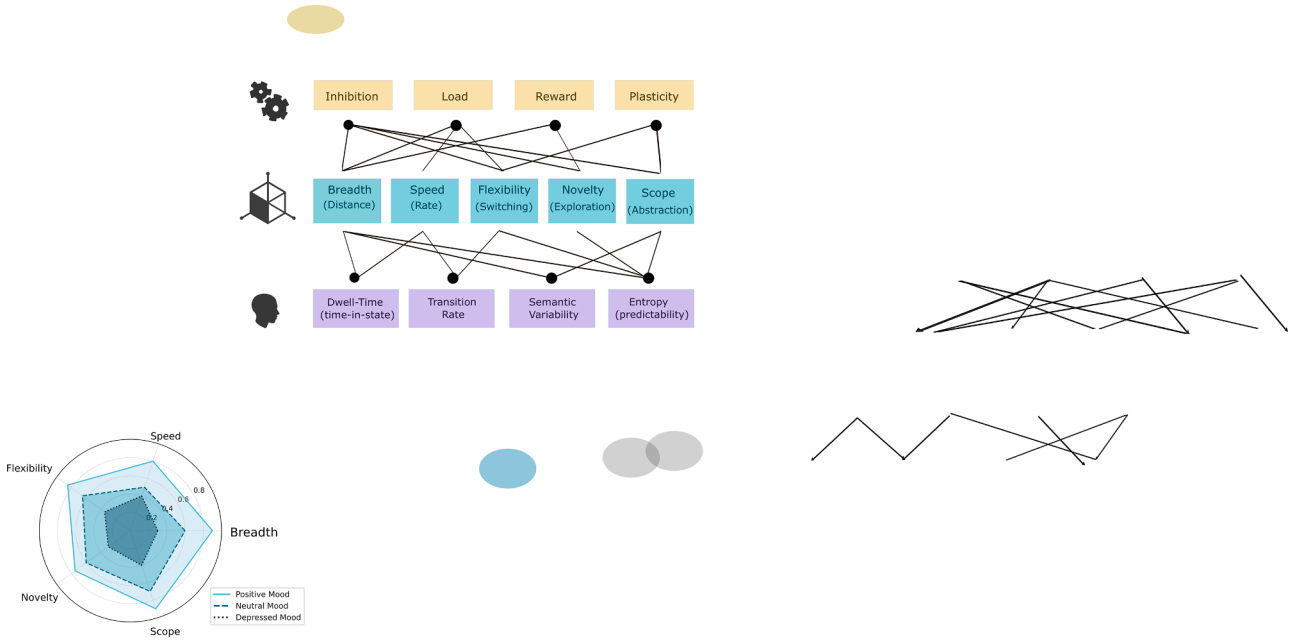

Figure 3. Facilitating thought progression (FTP) is a mechanistic bridge from neurocognitive levers to measurable thought dynamics. (Top) Candidate mechanisms that constrain thought progression. (Middle) FTP dimensions of thought. (Bottom) Observable behavioral signatures predicted by FTP. Lines illustrate the main hypothesized mappings between mechanisms, FTP dimensions, and measurement signatures.

that increase exposure to novelty, may re-engage the reward circuitry that reinforces exploratory thought (120,121,128,129). Interventions that increase cortical plasticity offer another promising target. Glutamatergic agents such as ketamine and serotonergic psychedelics can rapidly enhance synaptogenesis and dendritic remodeling, potentially opening "windows of plasticity" during which new emotional and cognitive associations become easier to form (65,67). Such increases in plasticity expand the brain's representational landscape, supporting transitions out of rigid attractor states. Finally, exercises that increase associative breadth, semantic variability, or cognitive switching may restore more flexible trajectories through conceptual space. Recent clinical findings show that cognitive interventions that directly train thought dynamics and explicitly enhance thought progression can significantly reduce depressive symptoms (138). A more detailed comparison of FTP with existing models of depression and cognition is provided in the Supplement (see Supplemental S2). By decomposing cognitive rigidity or mental inertia into separable parameters of thought progression, FTP specifies which "lever" is altered and what behavioral and neural signature should follow (Figure 3), enabling clearer cross-treatment comparison and mechanism-matched outcome selection.

Computational Psychiatry and Thought Dynamics

Recent advances in computational neuroscience (139,140) and computational psychiatry (141–143) offer tools for formalizing the constructs that are central to FTP. Network control theory quantifies the brain's ability to transition between states and can reveal whether depression corresponds to greater control effort (144,145). Complementing this systems-level view, models of spontaneous thought treat cognition as a dynamic process unfolding over an attractor landscape, enabling simulations of how excessive inhibition or reduced plasticity deepen attractor basins and stabilize ruminative loops (137). Reinforcement learning models further suggest that blunted reward sensitivity can bias cognition away from exploration, shifting thought trajectories toward exploitative, repetitive patterns that are consistent with reduced novelty-driven transitions (137,141,146).

In parallel, recent empirical work has begun to quantify thought dynamics directly. Natural language processing-based semantic-trajectory analyses provide behavioral markers of cognitive flow, such as semantic distance or variability derived from moment-to-moment thought reports (147,148). These approaches can be combined with paradigms that sample ongoing experience (e.g., think-aloud sampling) and with dynamic neuroimaging analyses that identify latent brain states and their transition structure (26,149,150). Together, these methods enable cross-level mapping between observable properties of thought progression and neural state-transition signatures, providing a concrete empirical route for testing FTP's predictions (see Supplemental S3 for an extended testable predictions section).

CONCLUSIONS

Depression can be understood as a disorder of impaired thought progression across 5 dimensions: breadth, speed, flexibility, novelty, and scope. FTP integrates evidence from cognitive science, computational psychiatry, and systems neuroscience to show how alterations in inhibition, reward

processing, attractor stability, and neural plasticity bidirectionally shape cognitive flow and mood. In this view, rumination and negative mood reflect reduced cognitive fluidity, but the link is not one-way; promoting thought progression may itself help shift mood in an adaptive direction. Therefore, FTP offers a translational framework in which interventions that broaden, accelerate, or increase the flexibility of thought, whether by reducing excessive inhibition, enhancing reward responsiveness, or increasing plasticity, may alleviate depressive symptoms. Future neuroimaging, computational, and longitudinal studies will be crucial for testing FTP and identifying its most informative markers.

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