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From Social Brain Modules to Dynamic Network Dysfunction: An Integrative Neuropsychological Framework for Social Cognition

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Review article

From Social Brain Modules to Dynamic Network Dysfunction: An Integrative Neuropsychological Framework for Social Cognition

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Abstract

Social cognition is commonly partitioned into emotion recognition, empathy, theory of mind, and social decision-making, yet these labels do not map clearly onto discrete neural modules. Recent taxonomic, meta-analytic, network-neuroscience, and clinical evidence instead indicates that adaptive social behavior depends on context-sensitive coordination of partially specialized and domain-general systems. We conducted a structured, search-informed integrative review of PubMed/MEDLINE-indexed literature updated on 6 September 2026, supplemented by citation chaining and targeted 2024–2026 searches. We synthesize evidence across social perception and action observation, interoception and affective salience, mentalizing and social-semantic knowledge, valuation and executive control, and large-scale network dynamics. The incremental contribution of this review is not to introduce node dysfunction, disconnection, or network dynamics as isolated concepts, but to integrate them into a cross-diagnostic neuropsychological framework that links task demands and component computations to distinct mechanisms of clinical dysfunction. The framework distinguishes two comparatively evidence-grounded mechanisms namely, (1) node dysfunction, affecting a computationally important region or subsystem, and (2) disconnection, impairing integration among otherwise viable components from (3) hypothesis-generating network-reconfiguration dysfunction, concerning context-appropriate transitions among network states. Frontotemporal dementia provides convergent evidence for node and disconnection mechanisms; Alzheimer disease and Parkinson disease illustrate the importance of general cognitive support and network state; acquired brain injury demonstrates the consequences of focal injury and long-range disconnection; and schizophrenia and autism emphasize mechanistic heterogeneity. Recent psychometric and ecological-validity syntheses further show that conventional task performance maps only incompletely onto everyday social behavior. The framework generates falsifiable predictions for dynamic imaging, electrophysiology, perturbation, lesion-network methods, and naturalistic assessment, with the aim of linking neuropsychological profiles more directly to mechanisms and real-world function.

Keywords: empathy, frontotemporal dementia, network dynamics, neuropsychology, social cognition, theory of mind

Introduction

Social cognition encompasses the processes that permit people to detect socially relevant information, represent the mental and affective states of others, retrieve social knowledge, assign value to interpersonal outcomes, and regulate behavior in context. These capacities are central to communication, relationships, caregiving, employment, and independent living. Their disruption can be clinically profound even when conventional measures of memory, language, visuospatial function, or general intelligence are only mildly affected[1,2].

The field nevertheless faces a construct problem. Terms such as theory of mind (ToM), mentalizing, cognitive empathy, affective empathy, emotion recognition, social perception, and social knowledge are used inconsistently, and many commonly used tasks recruit several of these processes simultaneously. The updated Hierarchical Interdependent Taxonomy of Social cognition (HITS) explicitly highlights poor construct validity in parts of the assessment literature and argues that definitions should be constrained by empirical and theoretical evidence rather than expert terminology alone[3]. This matters for neuropsychology: a poor score on a faux-pas task, for example, may reflect impaired mental-state inference, degraded semantic or pragmatic knowledge, memory dysfunction, language comprehension difficulty, or executive inability to suppress a literal response.

The field also faces an anatomical problem. Classical work identified reproducible contributions from the amygdala, superior temporal sulcus, temporoparietal junction (TPJ), medial prefrontal cortex (mPFC), anterior insula, anterior cingulate cortex (ACC), orbitofrontal/ventromedial prefrontal cortex (OFC/vmPFC), anterior temporal lobes (ATL), and inferior frontal-parietal action-observation systems. These discoveries were indispensable, but one-to-one mappings between a named social function and a region are difficult to sustain.

The same regions participate in non-social computations, and meta-analyses increasingly reveal overlapping, hierarchical, and task-dependent architectures. A 2024 meta-analysis of 344 functional neuroimaging studies, for example, found substantial overlap between ToM and semantic cognition, particularly in ATL and TPJ, while retaining relative ToM specialization in right TPJ, anterior mPFC, and precuneus[4]. A complementary 2024 ALE meta-analysis again demonstrated that the mentalizing network varies with task and social-scene characteristics rather than behaving as a fixed unit[5].

A third problem is temporal. Everyday interaction unfolds continuously: gaze and prosody change, intentions remain uncertain, prior knowledge is retrieved, and one person's response alters the next action of the other. Static task-fMRI maps obscure this sequence. Dynamic functional connectivity during live social exchange can distinguish cooperative from competitive interaction states, demonstrating that the configuration of canonical networks changes systematically with interpersonal demands[6]. Naturalistic and second-person approaches similarly show that the neural organization of reciprocal interaction cannot be inferred fully from passive observation paradigms[7,8].

These conceptual and mechanistic gaps are clinically consequential. Two 2026 papers in Neuropsychology Review sharply define what remains unresolved. Pittet et al. synthesized psychometric evidence from 55 validation studies and showed substantial condition- and instrument-specific heterogeneity, including limited ecological validation[9]. Gelmers et al. examined 21 studies linking performance-based social cognition to proxy-rated social behavior in neurodegenerative disease and found mostly negligible or weak associations, with only modest relationships for selected domains. Thus, laboratory impairment cannot be assumed to explain everyday social behavior directly[10].

The present review is intentionally complementary to those recent syntheses. It does not propose another taxonomy, rank psychometric instruments, or re-meta-analyze disease-group effect sizes. Instead, it asks a mechanistic neuropsychological question: how are social-perceptual, affective-salience, mentalizing and social-semantic, valuation, and executive processes coordinated over time, and how can disruption of that coordination generate different clinical phenotypes? Its incremental novelty lies in linking four levels that are usually treated separately- task demands, component computations, mechanisms of clinical dysfunction, and context-sensitive network dynamics- within one cross-diagnostic framework.

We advance a three-mode dysfunction framework (Table 1). Node dysfunction refers to dysfunction of a region or subsystem that contributes a relatively indispensable computation. Disconnection refers to impaired structural or functional integration among components that may be locally viable. These first two tiers are grounded in established lesion, neurodegenerative, and connectivity evidence. Hypothesis-generating network-reconfiguration dysfunction is deliberately treated differently: it is the novel prospective tier, referring to impaired entry into, maintenance of, or transition between configurations required by changing social context. Throughout this review, references to reconfiguration dysfunction denote a candidate mechanism requiring time-resolved prospective validation rather than an established clinical category. The three mechanisms are not mutually exclusive and should not be equated with diagnoses. Rather, they provide testable hypotheses for explaining why similar task errors can arise from different diseases, why a single disease can produce heterogeneous social phenotypes, and why explicit task performance may diverge from behavior in complex environments.

Insert Table 1

The review has four aims: (1) to separate social-cognitive constructs from the component operations required by their tasks; (2) to integrate region-level neuropsychology with contemporary network and dynamic-systems evidence; (3) to use neurological and psychiatric syndromes as mechanistic tests of node, disconnection, and reconfiguration hypotheses; and (4) to derive predictions that can be falsified using lesion methods, time-resolved functional imaging, EEG/MEG, perturbation, and ecologically valid interaction paradigms.

Review Methodology

This article is a structured, search-informed integrative narrative review rather than a systematic review or meta-analysis. The design was chosen because the primary objective is theory building across heterogeneous evidence classes- focal lesions, neurodegenerative syndromes, psychiatric and neurodevelopmental cohorts, task and resting-state imaging, structural and functional connectivity, electrophysiology, perturbation, psychometrics, and computational/network models- rather than estimation of a common pooled effect.

On 6 September 2026, PubMed/MEDLINE-indexed literature was updated using 11 prespecified concept blocks: (1) social-cognition taxonomies and frameworks; (2) ToM/empathy meta-analyses; (3) default-mode, salience, and executive/control networks; (4) dynamic connectivity and network reconfiguration; (5) social-semantic/anterior temporal systems; (6) mirror/action-observation controversies; (7) frontotemporal dementia, Alzheimer disease, and Parkinson disease; (8) stroke, traumatic brain injury, and acquired brain injury; (9) schizophrenia and autism; (10) ecological validity, naturalistic paradigms, and second-person neuroscience; and (11) a focused 2024–2026 update. Searches were supplemented by backward and forward citation tracking from meta-analyses, systematic reviews, concept-defining papers, and recent articles in Neuropsychology Review. The search was designed for structured updating and evidential balance rather than exhaustive study identification. Authenticated PsycINFO, Scopus, and Web of Science interfaces were not available during this revision, so no database-specific results or hit counts from those sources are claimed. To facilitate reproducible completion before submission, PsycINFO-ready keyword translations of all 11 concept blocks have been added to Online Resource 1 alongside the existing Scopus and Web of Science translations. Exact PubMed search strings, execution status, and selection principles are provided in Online Resource 1.

Evidence was weighted preferentially toward systematic reviews and meta-analyses, convergent findings across methods, lesion or neurodegenerative data with stronger anatomical or causal leverage, and studies linking neural measures to clinically meaningful behavior. Individual task-fMRI studies were used selectively to address mechanistic gaps. Functional activation was not interpreted as proof of necessity or social specificity; functional connectivity was treated as statistical dependence rather than direct information transfer; and diffusion tractography was treated as evidence for candidate structural pathways rather than dedicated monosynaptic social-cognitive routes.

Studies were synthesized around processing demands and dysfunction mechanisms rather than diagnosis alone. Clinical syndromes were treated as natural experiments to ask whether a phenotype is better explained by degradation of social-perceptual representations, impaired affective salience, dysfunction of mental-state/social-semantic modeling, altered valuation or control, disconnection among these systems, or impaired dynamic reconfiguration. Contradictory findings were retained when they defined a substantive boundary condition rather than resolved by selective citation.

Because the review is integrative and does not claim exhaustive retrieval, no PRISMA flow diagram, database-complete record count, duplicate independent screening procedure, or formal study-level risk-of-bias synthesis is presented. The structured search functions as a reproducible updating scaffold rather than as an exhaustive evidence-identification process. The absence of an executed PsycINFO search is therefore treated as a limitation rather than obscured; psychology-focused evidence was additionally checked through citation chaining from major social-cognition taxonomies, psychometric syntheses, meta-analyses, and naturalistic/second-person reviews. This distinction is important because the review integrates heterogeneous causal, clinical, neuroimaging, psychometric, and theoretical evidence that cannot be reduced to a single effect-estimation question.

Use of generative AI and AI-assisted technologies: During manuscript development, ChatGPT (OpenAI) was used as an assistive tool for literature-search query formulation, manuscript structuring, and rephrasing selected passages. All AI-assisted outputs were critically reviewed and substantially edited by the authors. The cited literature and factual claims were independently verified, and all scientific interpretations, conclusions, and final editorial decisions were made by the authors, who take full responsibility for the manuscript. No generative-AI images are included in the submitted figures.

Defining Social Cognition: From Constructs to Processing Demands

A mechanistic model should begin with the operations needed to solve a task, not with the task's conventional label. The Reading the Mind in the Eyes Test, a false-belief vignette, facial-emotion labeling, moral judgment, and reciprocal conversation can all be described as measures of social cognition, yet they differ in perceptual input, semantic and language demands, working memory, explicitness of mental-state reasoning, value computation, and response selection. Performance therefore cannot be interpreted mechanistically without decomposing these demands.

Taxonomic work provides a useful starting point. The HITS framework emphasizes hierarchical and interdependent social-cognitive constructs[3], whereas experimental dissociation with the EmpaToM paradigm and subsequent neuroimaging meta-analysis support partially separable but interacting empathy and ToM processes, with more affective, more cognitive, and mixed tasks recruiting overlapping but distinguishable neural configurations[11,12]. These perspectives converge on two principles: social cognition is fractionable, but its components are not independent.

Domain-general support is equally important. Balgova et al. demonstrated neural overlap between ToM and semantic cognition, challenging accounts in which mentalizing is implemented entirely by social-specific machinery[4]. ATL and temporoparietal regions can support retrieval and integration of conceptual knowledge that becomes 'social' when the represented content concerns persons, traits, relationships, actions, or norms[13]. Executive control, language, autobiographical memory, and general valuation likewise contribute to many social tasks without being intrinsically social[14].

We therefore organize the review around six recurrent operations (Table 2). First, social-perceptual systems extract structure from faces, gaze, voice, bodies, and biological motion. Second, action-observation and embodied systems map observed bodily states or actions onto sensorimotor and interoceptive representations. Third, affective-salience systems determine biological and motivational relevance. Fourth, mentalizing/default-mode and social-semantic systems construct latent representations of beliefs, intentions, traits, relationships, and contextual meaning. Fifth, valuation and executive systems arbitrate among goals, norms, reward, harm, and response alternatives. Sixth, interactive control continuously updates these representations as another person's behavior changes.

These operations are recurrent rather than serial. A facial movement may alter inferred intention; the new interpretation changes salience; salience reallocates attention; social-semantic context changes the meaning of the same movement; and executive goals alter the response. The model therefore predicts that identical stimuli can produce different social judgments depending on prior knowledge, uncertainty, motivation, and the current network configuration.

The processing architecture and its cross-cutting dysfunction modes are summarized schematically in Figure 1.

Insert Table 2 here

Insert Figure 1 here

Functional Architecture of Social Cognition

Social perception, action observation, and embodied representations

Social cognition begins with socially informative perception but is not reducible to it. Posterior superior temporal sulcus (pSTS) is especially responsive to dynamic facial, gaze, and biological-motion cues, whereas ventral occipitotemporal regions support detailed visual representations of faces and bodies [15, 16, 17]. These

systems provide evidence about what another agent is doing and attending to; they do not by themselves establish why the action is occurring or what the agent believes.

Action-observation research identified overlapping responses during action execution and observation in inferior frontal/premotor and inferior parietal regions. Meta-analysis supports reproducible 'mirror-like' properties in these regions[18], but their functional interpretation requires restraint. A meta-analysis comparing action-observation and mentalizing paradigms found the systems to be largely complementary: action-observation regions are preferentially engaged by visible biological actions, whereas TPJ, mPFC, and precuneus become more prominent when goals or beliefs must be inferred from abstract contextual information[19].

The relation between mirror-system activity and empathy is similarly graded rather than categorical. A Neuropsychology Review systematic review and meta-analysis of 52 studies found moderate associations between putative mirror-system activity and emotional/cognitive empathy, but results varied by measurement technique and empathy component[20]. The findings support interaction, not identity, between sensorimotor resonance and empathy. In autism, the historically influential 'broken mirror' hypothesis is particularly problematic: Hamilton's systematic review found mixed evidence and no general support for a globally dysfunctional mirror system[21].

Recent work broadens embodiment beyond motor mirroring. Canino et al., synthesizing 21 studies and 3,466 participants, found that interoceptive sensibility and accuracy were positively related to social cognition, with stronger associations for empathy than ToM[22]. This suggests that representations of one's own bodily state can contribute to affective understanding without implying that bodily simulation is necessary or sufficient for all social inference.

Accordingly, the present framework treats perceptual and embodied systems as evidence-generating components. They constrain later inference by specifying dynamic cues, actions, and internal bodily relevance. Their contribution should be strongest when social meaning is carried by movement, gaze, gesture, prosody, or rapidly changing bodily contingencies, whereas abstract belief reasoning should increasingly recruit mentalizing, semantic, and control systems.

> Dysfunction-mode implication: Node dysfunction in occipitotemporal or pSTS systems should selectively degrade the quality of social-perceptual evidence; node dysfunction in action-observation or interoceptive systems should preferentially affect imitation, biological-action understanding, or selected forms of affect sharing. Disconnection should produce disproportionate difficulty when perceptual evidence must be integrated with semantic, mentalizing, or valuation systems. Hypothesis-generating network-reconfiguration dysfunction predicts that a patient can decode an explicit cue in isolation but fails to shift from rapid perceptual/embodied processing to higher-order inference as ambiguity or context increases.

Affective salience and interoception

The amygdala, anterior insula, and cingulate cortices are recurrently implicated in the detection and evaluation of biologically relevant events. The amygdala is better conceptualized as a relevance- and value-sensitive structure than as a dedicated 'fear center'; it contributes to orienting, learning under uncertainty, and evaluation of facial and interpersonal signals[23]. This broader account explains why amygdala damage can disturb emotion recognition, gaze allocation, trustworthiness judgments, and social learning without requiring these functions to be identical.

The anterior insula provides an interface between external events and internal bodily state, while cingulate regions contribute to motivational control, conflict monitoring, and value. Empathy paradigms frequently recruit anterior insula and cingulate cortex, but these regions also respond to one's own pain, effort, uncertainty, and salient non-social events[24, 25]. Their role is therefore better described in terms of affective/interoceptive weighting than as an empathy module.

Neurodegenerative evidence strengthens this interpretation. Pasquini et al. linked TDP-43 pathology in right fronto-insular von Economo neurons and fork cells to salience-network atrophy and loss of emotional empathy across the bvFTD/ALS spectrum, providing an unusually direct chain from cell type to network degeneration to behavior[26]. A 2025 neuroimaging meta-analysis of 170 studies and 7,032 participants further

found convergence of interoception, emotion, and social-cognition abnormalities across an allostatic-interoceptive network involving insula, amygdala, OFC, ACC, striatum, thalamus, and hippocampus, with particularly strong convergence in bvFTD[27].

The salience system is also a plausible coordinator of transitions. A socially unexpected cue should increase the probability that internally oriented contextual models or externally oriented control systems are recruited. This proposed switching function is consistent with broader triple-network accounts[28, 29], although direct causal evidence for a specifically social switching mechanism remains limited.

Dysfunction-mode implication. Node dysfunction in anterior insula, amygdala, or cingulate regions is expected to alter the motivational or interoceptive weighting of social information. Disconnection from mentalizing, semantic, or valuation systems should leave cues perceptually available but behaviorally underweighted or misinterpreted. Hypothesis-generating network-reconfiguration dysfunction predicts deficient recruitment or disengagement of mentalizing/control states following salient social change—a prediction that requires time-resolved testing rather than static activation maps.

Mentalizing, default-mode, and social-semantic systems

Mentalizing—the representation of another person's beliefs, intentions, knowledge, or perspective—is among the most reproducible social-cognitive constructs in neuroimaging. Meta-analytic work consistently implicates bilateral TPJ, mPFC, posterior cingulate/precuneus, and temporal association cortex[15, 30, 31]. Yet this 'mentalizing network' is internally heterogeneous. Gan et al. showed that ToM-related activation varies with social-scene and age-related task characteristics, underscoring the dependence of network expression on representational demands[5].

These regions overlap extensively with the default mode network (DMN). Modern accounts treat the DMN not as a unitary task-negative system but as interwoven association networks supporting internally constructed representations across episodic memory, semantics, language, self-reference, future simulation, narrative comprehension, and social inference[32, 33]. Social mentalizing can therefore be understood as one use of a broader capacity to integrate information across long timescales and construct latent models that are not directly specified by immediate sensory input.

Social-semantic knowledge is central to this construction. ATL degeneration can impair knowledge about people, traits, social concepts, and contextual norms despite relatively preserved elementary perception[12]. Balgova et al. provided large-scale meta-analytic evidence that ToM and semantic cognition overlap substantially in ATL and TPJ, while retaining relative ToM specialization in right TPJ, anterior mPFC, and right precuneus. The important conclusion is not that ToM is 'just semantics,' but that mental-state inference depends on a mixture of social-tuned and domain-general conceptual operations[4].

Hierarchical meta-analysis likewise rejects a simple separation between 'ToM network' and 'empathy network.' Schurz et al. found graded organization across cognitive, affective, and mixed social tasks. This is compatible with a processing-demand model in which the relative contribution of semantic/DMN, salience/interoceptive, and executive systems changes with the problem being solved[12].

Clinically, this distinction is crucial in temporal-lobe disease. A patient may fail to infer another person's intention because the mentalizing operation is damaged, because knowledge about people and social concepts has degraded, or because contextual information can no longer be integrated over time. Similar behavioral errors therefore do not imply the same neural mechanism.

Dysfunction-mode implication: Node dysfunction in TPJ/mPFC should most directly compromise mental-state modeling, whereas ATL node dysfunction should disproportionately affect person and social-semantic knowledge. Disconnection within or between DMN, ATL, salience, and control systems should impair integration of beliefs with context. Hypothesis-generating network-reconfiguration dysfunction predicts that a patient can state social rules or infer beliefs under explicit prompting but fails to update a latent model when new evidence contradicts an initial interpretation.

Valuation and executive-control systems

Understanding another person does not determine how one behaves. Social behavior requires valuation of outcomes and selection among competing goals. vmPFC/OFC, cingulate regions, striatum, and lateral frontoparietal systems contribute to reward and value integration, conflict monitoring, working memory, rule use, and response control. These computations are not exclusively social, but they become socially consequential when the valued variables include reputation, harm, fairness, trust, affiliation, or another person's welfare[34].

Lesion evidence provides causal leverage. Damage to vmPFC can produce marked changes in judgment, affective decision-making, personality, and social conduct despite relatively preserved conventional intelligence[35]. Young et al. showed that vmPFC lesions altered moral evaluation of attempted harms by reducing the weight assigned to harmful intent. Such findings demonstrate that mental-state inference and valuation are separable stages: a person may represent another's intention yet fail to assign it the expected behavioral significance[36].

The anterior cingulate gyrus contributes to representing the motivation and value of outcomes for other individuals, whereas lateral prefrontal-parietal control systems become important when social decisions are ambiguous, novel, or conflictual[37]. Thus, deficits described as 'moral cognition' or 'social reasoning' can emerge from abnormal valuation with intact ToM, executive inability to implement an appropriate response, or impaired integration of inferred intentions with outcome value.

This distinction also explains clinically familiar dissociations. A patient may accurately label another person's anger yet continue an offensive behavior because the cue fails to alter value or response policy. Another patient may show strong affect sharing but mis-infer beliefs. A distributed processing account predicts such profiles; a unitary empathy or social-intelligence construct does not.

> Dysfunction-mode implication: Node dysfunction in vmPFC/OFC/ACCg can selectively distort social value or motivation, while lateral frontoparietal injury can reduce rule-guided control. Disconnection from mentalizing or semantic systems should decouple knowledge of another person's state from the value assigned to it. Hypothesis-generating network-reconfiguration dysfunction is predicted to be most evident in rapidly changing social conflicts that require switching between empathic, norm-based, self-protective, and long-term goals.

Structural connectivity and the disconnection principle

A distributed architecture depends on communication among systems, but association-fiber labels should not be treated as dedicated social-cognitive pathways. The uncinate fasciculus, cingulum, superior longitudinal system, inferior longitudinal system, and callosal fibers provide candidate routes linking temporal, parietal, limbic, cingulate, and frontal regions. Diffusion tractography cannot by itself establish directionality, monosynaptic connection, or the computation transmitted by a tract.

The stronger neuropsychological principle is disconnection: locally viable representations can fail behaviorally when they cannot be integrated. Acquired brain injury provides direct support. Cavallo et al. reviewed 43 stroke/TBI studies and found alterations across emotion recognition, ToM, perspective taking, and empathy, with recurrent involvement of insula, cingulate cortex, middle frontal regions, and corpus callosum[38]. The pattern argues against a single social-cognition locus and highlights interhemispheric and cross-system integration.

Disconnection can also be functional rather than purely structural. In neurodegeneration, synaptic dysfunction and network topology can deteriorate before or beyond focal atrophy. A 2026 FTD study found increased path length, reduced nodal strength, and widespread connectivity reductions in affective and cognitive ToM networks, particularly in bvFTD and nonfluent/agrammatic PPA, while social-cognitive impairment extended across FTD variants[39]. Such data make disconnection a mechanistic construct rather than merely a metaphor.

Insert Table 2 here

From Static Maps to Dynamic Network Reconfiguration

A distributed account becomes mechanistic only if it explains coordination over time. During a brief interaction, the dominant computational problem can shift from face and gaze processing to intention inference, then to conflict monitoring, memory-based context, and response inhibition. A static average of activation over an entire trial can obscure both the order and the conditional dependence of these operations.

The salience, default-mode, and executive/control networks provide a useful—although simplified—large-scale scaffold. Intrinsic imaging established separable salience and executive-control systems[28], and Menon's triple-network model proposed that abnormal engagement, disengagement, or switching among salience, DMN, and executive systems can contribute to psychopathology[29]. Applied to social cognition, the central variable is not whether a network is globally 'on' or 'off,' but whether the system adopts a configuration suited to the current inferential demand.

The DMN is particularly suited to integration across long timescales because it supports autobiographical, semantic, language, and internally generated representations[33, 40]. A fleeting expression can therefore be interpreted against relationship history, cultural knowledge, and expectations about another person's goals. Executive-control systems become increasingly important when evidence is ambiguous, a habitual interpretation must be inhibited, or explicit rules conflict with affective preference.

Dynamic functional connectivity provides an initial empirical test of this idea. During real interpersonal exchanges, Shaw et al. identified six connectivity states with distinct integration/segregation patterns across canonical networks; those states differed systematically between cooperation and competition and generalized to classification in an independent sample[7]. The study does not establish a universal 'social state' architecture, but it demonstrates that interaction type is encoded partly in temporally evolving cross-network configurations rather than only in regional activation magnitude.

Naturalistic and second-person paradigms are essential complements. Movies and narratives preserve temporal context, while reciprocal interaction introduces contingency: another agent changes behavior in response to the participant. Redcay and Schilbach argued that mechanisms of direct interaction can differ from those engaged by passive observation, and Redcay and Moraczewski showed how naturalistic imaging can interrogate social cognition over longer timescales[7, 8]. These designs are particularly relevant to patients who perform adequately on explicit tests yet fail during fast, uncertain social exchange.

Predictive-processing accounts provide a possible computational vocabulary. A social agent can be modeled as maintaining hypotheses about another person's latent state, weighting prior knowledge and current evidence, and updating when behavior violates expectation. This formulation naturally connects semantics, uncertainty, salience, and learning. However, it remains a hypothesis rather than an all-purpose explanation: many apparent 'prediction-error' phenomena can also be described by attention, reinforcement learning, working memory, or semantic updating. The dynamic-network framework does not require a specific predictive-coding implementation.

We use network reconfiguration operationally to mean time-varying changes in the relative engagement and interaction of perceptual, salience, mentalizing/semantic, valuation, and control systems as task demands change. Candidate measures include state-transition probability, dwell time, network flexibility, time-resolved effective or functional connectivity, temporal ordering with EEG/MEG, and perturbational responses to TMS. A strong test of the framework requires showing that such dynamic variables explain clinically meaningful behavior beyond static activation, static resting connectivity, disease severity, and general cognitive performance.

Node dysfunction, disconnection, and reconfiguration dysfunction form a hierarchy of explanations rather than mutually exclusive categories. Focal tissue damage can cause secondary disconnection; disconnection can constrain which network states are reachable; and persistent network-state abnormalities can amplify the behavioral impact of relatively modest local pathology. The model is therefore explicitly multiscale.

Figure 2 illustrates the related hypothesis that relative engagement of component systems changes continuously across an evolving social interaction; the depicted stages overlap rather than forming a fixed serial sequence.

Insert Figure 2 here

Neuromodulatory Control: State Setting Rather Than Social Modules

Neuromodulators can influence social cognition indirectly by altering arousal, gain, reward sensitivity, learning, and large-scale integration rather than by implementing discrete social faculties. Dopamine, serotonin, oxytocin, and vasopressin have all been linked to social behavior, but their effects are context-, task-, and person-dependent; for example, intranasal oxytocin can alter trust and attention to social cues, while serotonergic manipulation can modify responses to unfairness without implying dedicated 'social' neurotransmitter functions[41, 42, 43].

At the systems level, neuromodulators are plausible state-setting influences on transitions between segregated and integrated network configurations. Shine et al. linked dynamic cognitive integration to neuromodulatory receptor architecture, providing a possible bridge between molecular state and network dynamics[44]. However, direct evidence that a specific neuromodulator causes clinically meaningful social-network transitions remains insufficient. Neuromodulation is therefore treated here as a secondary modifier of the framework, not as an independent social-cognitive mechanism or evidence for hypothesis-generating network-reconfiguration dysfunction.

Clinical Neuropsychology as Mechanistic Evidence

Frontotemporal dementia spectrum

Frontotemporal dementia (FTD) offers the strongest clinical test of the framework because social-emotional change can be early, severe, and anatomically patterned. In bvFTD, loss of empathy, disinhibition, altered social norms, reduced emotion recognition, and impaired ToM may occur before severe global cognitive decline. Meta-analysis demonstrates large ToM impairment in bvFTD relative to healthy controls and substantially greater impairment than in Alzheimer disease even when general cognition is matched[45]. Facial-emotion recognition is likewise markedly impaired relative to both controls and Alzheimer disease [46].

These effects cannot be reduced to 'frontal dysfunction.' bvFTD preferentially affects anterior insula, ACC, OFC/vmPFC, and related salience/valuation systems, while temporal variants can disrupt person knowledge, emotion concepts, and social-semantic representations. Pasquini et al. provide node-level evidence: pathology in fronto-insular von Economo neurons and fork cells was associated with salience-network atrophy, which mediated loss of emotional empathy[26]. Hazelton et al. broaden this observation by showing cross-domain convergence of interoception, emotion, and social cognition within an allostatic-interoceptive network, particularly in bvFTD[27] (Table 3).

The 2026 Tripodi et al. study adds a crucial disconnection result[39]. Across 67 patients with bvFTD, nonfluent/agrammatic PPA, semantic-variant PPA, and right-temporal FTD, affective and cognitive ToM impairment was present across variants, while network analysis showed increased path length, reduced nodal strength, and widespread connectivity loss in ToM networks, most marked in bvFTD and nonfluent/agrammatic PPA. Salience-network connectivity also related to behavioral dysfunction[39]. The observation that similar ToM impairment can coexist with different connectivity patterns argues against a one-region/one-syndrome explanation.

FTD therefore provides strong evidence for node dysfunction and disconnection, while hypothesis-generating network-reconfiguration dysfunction remains a plausible additional mechanism. It may be relevant when

behavior becomes rigid, context-insensitive, or unable to recruit an alternative strategy, but direct time-resolved clinical testing is still required.

A mechanistic neuropsychological assessment should accordingly distinguish at least emotion/cue processing, social-semantic knowledge, affective and cognitive ToM, and behavioral regulation. A single social-cognition composite risks obscuring whether the dominant problem lies in affective salience, semantic representation, mental-state inference, or control.

Alzheimer disease, mild cognitive impairment, and Lewy body dementia

Social cognition in Alzheimer disease (AD) is impaired but generally less selectively than in bvFTD. Earlier meta-analysis showed progressive deterioration of emotion recognition and ToM from healthy aging through MCI and AD[47]. A 2025 meta-analysis of 28 studies and 2,409 participants found that individuals with MCI outperformed those with AD dementia in emotion recognition ($d = 0.69$) and ToM ($d = 0.70$); deficits were substantially larger in FTD, while emotional empathy appeared comparatively preserved in AD across the included literature[48].

A 2026 meta-analysis specifically addressing emotion recognition in AD identified 24 studies and a large pooled impairment (Hedges' $g = -1.06$). Global cognitive status moderated the deficit, but did not fully explain it[49]. These data are important because they reject the simplistic view that social cognition is preserved in AD, while also warning that memory, semantic, language, attentional, and executive decline can contaminate ostensibly social tasks.

Network interpretation is therefore different from bvFTD. AD strongly affects DMN, episodic-memory, and semantic-support systems that contribute to contextual mental-state modeling. Resting-state work has shown divergent group-level patterns in bvFTD and AD, with reduced salience-network connectivity in bvFTD and reduced DMN connectivity in AD[50], although disease progression produces overlap and such findings should not be converted into a one-network-per-disease rule.

From the three-mode dysfunction perspective, AD often illustrates distributed node degradation plus disconnection within memory, semantic, and DMN systems rather than early dysfunction of a social-specific node. Hypothesis-generating network-reconfiguration dysfunction is a candidate explanation when fluctuating task demands exceed compensatory capacity, but direct evidence remains substantially weaker than for static network abnormalities. A false-belief or faux-pas error in AD should therefore be interpreted only after accounting for memory of the scenario, language, semantic knowledge, and executive demands.

Data in dementia with Lewy bodies (DLB) remain more limited. Heitz et al. found early impairment of both cognitive and affective ToM in DLB, with associations involving prefrontal, TPJ, precuneus, fusiform, and insular regions[51]. Fluctuating attention, visuoperceptual dysfunction, executive instability, and limbic/fronto-striatal disease can all contribute. DLB is therefore a useful candidate for future reconfiguration studies, but current evidence is insufficient to assign a distinctive dynamic-network signature.

Insert Table 3 here

Parkinson disease

Parkinson disease (PD) is an instructive example of why social-cognitive impairment should not be assumed to arise directly from a disease label. Earlier meta-analysis found moderate-to-large deficits in cognitive ToM and cognitive-empathy/affective-ToM tasks, with relative preservation of affective empathy[52]. However, heterogeneity in disease stage, cognition, mood, medication state, and task demands has produced inconsistent findings.

A 2026 etiological systematic review specifically addressed whether ToM impairment is independent of general cognition. Across 14 stronger-causal-design studies, ToM performance was robustly dependent on cognitive ability: cognitively impaired patients consistently performed worse, whereas comparisons between

cognitively preserved PD and healthy controls were mixed[53]. A larger 2026 systematic review integrating 191 behavioral and neuroimaging studies likewise concluded that negative-emotion recognition deficits are relatively consistent, whereas empathy and ToM findings are heterogeneous and become more prominent with advanced disease or cognitive impairment; social problem-solving remains understudied[54].

These findings favor a distributed dysfunction account. Fronto-striatal/executive dysfunction can impair maintenance and manipulation of mental-state information; dopaminergic state can alter valuation and learning; autonomic/interoceptive changes may modify affective salience; and later cortical disease can produce additional node and disconnection effects. The strongest current evidence therefore supports cognitive-support and network-level mechanisms rather than a dedicated PD social-cognition lesion.

In framework terms, PD often exposes the interaction between disconnection/reconfiguration and general cognitive reserve. A patient with preserved cognition may compensate for subtle network inefficiency under explicit testing, whereas time pressure, ambiguity, or multitask social interaction may reveal a deficit. This is a testable prediction rather than an established clinical rule.

Stroke, traumatic brain injury, and acquired brain injury

Acquired brain injury offers stronger causal leverage because lesion timing is known and focal or diffuse injury can be related to specific neuropsychological changes. A meta-analysis of 28 TBI studies involving 1,031 adults with TBI and 865 controls found a large ToM deficit (Hedges' g = -1.13), confirming that mental-state reasoning is frequently affected after injury[55].

Cavallo et al. integrated 43 stroke and TBI studies and found abnormalities across emotion recognition, ToM, perspective taking, and empathy[37]. Recurrent neural correlates included insula, cingulate cortex, middle frontal regions, and corpus callosum. The callosal result is especially important for the present framework because it illustrates how disruption of integration can contribute to social impairment even when no single cortical 'social node' accounts for the phenotype.

A 2026 Neuropsychology Review meta-analysis of empathy after acquired brain injury found moderate deficits in self-reported cognitive empathy (g = -0.68), smaller deficits in affective empathy (g = -0.43), and small-to-moderate reduction in empathic concern (g = -0.38) across 29 studies[56]. The dissociation again argues against a single empathy faculty and highlights measurement dependence.

Acquired brain injury (ABI) therefore provides the clearest separation between node and disconnection mechanisms. Focal vmPFC, amygdala, TPJ, or insular lesions can produce component-specific impairment, whereas diffuse axonal injury, callosal damage, or multifocal lesions can degrade integration across systems. Hypothesis-generating network-reconfiguration dysfunction should be considered when explicit component tests are relatively preserved but behavior deteriorates under rapid, reciprocal, or multitask conditions.

Clinical interpretation must nevertheless control for visual and auditory impairment, aphasia, processing speed, memory, executive dysfunction, reduced self-awareness, fatigue, and mood. Informant data and naturalistic observation are particularly important because laboratory performance and everyday behavior may diverge.

Schizophrenia spectrum disorders

Schizophrenia is characterized by clinically important but heterogeneous impairments in emotion processing, social perception, mentalizing, attribution, and pragmatic communication. Neuroimaging meta-analysis has implicated medial prefrontal, temporal, insular, and cingulate systems, but no single abnormality accounts for the spectrum[57].

Recent work reinforces heterogeneity rather than a unitary 'social brain' lesion. Fu et al. meta-analyzed 38 task-fMRI studies across clinical high-risk, genetic high-risk, and first-episode groups and found limited shared activation abnormalities; a shared right ACC alteration emerged only for selected group comparisons, with age-related insular effects in clinical high risk. A 2026 task-fMRI meta-analysis directly comparing schizophrenia and autism likewise found no corrected spatial conjunction across socio-emotional tasks, instead identifying diagnosis- and paradigm-specific abnormalities[58].

Upstream perceptual contributions also matter. Ganse-Dumrath et al. synthesized evidence linking early visual processing, social cognition, and functional outcomes in schizophrenia-spectrum disorders, supporting models in which degraded sensory evidence can propagate into higher-order social judgments[59]. Pragmatic language provides a second bridge: Frau et al. found that pragmatic impairment was associated with both social cognition and executive function, with a stronger association for social cognition and a mediating role for social cognition between executive function and pragmatics[60].

Social cognition is related to functioning, but not deterministically. Across 166 studies and 12,868 participants, Halverson et al. found that social cognition explained more unique variance in functional outcome than neurocognition and partially mediated the neurocognition-function relationship; nevertheless, most functional variance remained unexplained[61]. This gap is exactly what a component-plus-dynamics model would predict if motivation, symptoms, environment, opportunity, and real-time interaction impose additional constraints.

Schizophrenia therefore resists simple assignment to one dysfunction mode. Node-level abnormalities are inconsistent across stages, while distributed sensory, salience, mentalizing, and control abnormalities support disconnection. Hypothesis-generating network-reconfiguration dysfunction is a candidate mechanism for impaired context updating or state regulation, but it should be tested directly rather than inferred from static fMRI. Ecological-validity work is particularly relevant: a 2025 systematic review found that only a minority of schizophrenia studies explicitly defined ecological validity, and most focused on task resemblance to real life (verisimilitude) rather than prediction of real-world behavior (veridicality)[62].

Autism

Autism provides perhaps the strongest warning against collapsing heterogeneous social functioning into a single mechanism. Social communication and interaction differences can involve social attention, sensory processing, action prediction, language/pragmatics, mentalizing, motivation, learning, and uncertainty, with substantial variation across individuals and development.

The mirror-neuron literature illustrates the risk of over-localization. Although some studies reported reduced inferior frontal or parietal responses, Hamilton's systematic review found no general support for a globally 'broken' mirror system[20]. Bekkali et al. further showed that associations between putative mirror-system activity and empathy are moderate and method-dependent even in nonclinical samples[19]. Action-observation dysfunction can therefore contribute to specific phenotypes without explaining autism as a whole.

Recent large-scale evidence favors multicomponent organization. Li et al. synthesized 2,622 behavioral studies encompassing 94,114 autistic and 172,847 non-autistic participants across 32 countries[63]. Twenty-two social components clustered into five domains, with substantial overall group differences but marked heterogeneity, developmental change, cross-domain interdependence, and uneven cultural representation. This scale of heterogeneity is difficult to reconcile with a universal single-node account.

Connectivity evidence is likewise distributed. A 2025 meta-analysis of 26 seed-based resting-state studies found robust abnormalities involving medial frontal DMN and ventral-attention-network regions, but also emphasized inconsistency across seeds and studies[64]. The 2026 schizophrenia-autism task-fMRI meta-analysis found distinct rather than shared corrected patterns across diagnoses[58], reinforcing the need to avoid a generic transdiagnostic social-network explanation.

Within the present framework, autism is best represented as mechanistic heterogeneity across cue weighting, perceptual/embodied evidence, contextual integration, mental-state modeling, learning, and network flexibility. Node dysfunction is unlikely to be a universal explanation; disconnection and reconfiguration hypotheses may fit selected subgroups but require individual-level validation. Neurodiversity-informed interpretation also requires distinguishing clinically disabling mismatch or distress from mere difference in interaction style and avoiding the assumption that neurotypical social behavior is the only valid reference state.

The Ecological Gap: From Test Scores to Social Behavior

The clinically meaningful endpoint of social cognition is adaptive behavior in relationships and real-world roles, not performance on a decontextualized test. Yet recent evidence shows that these levels are only partially coupled. Pittet et al. synthesized psychometric data from 55 studies (3,845 patients and 3,807 controls) and found robust known-group validity for some populations and instruments but substantial heterogeneity across conditions and limited ecological validation[8].

Gelmers et al. addressed the more direct question of whether performance-based social cognition predicts proxy-rated social behavior in neurodegenerative disease[9]. Across 21 studies, most associations were negligible or weak. Better emotion recognition was only weakly associated with fewer overall behavioral symptoms (r approximately -0.23) and less apathy (r approximately -0.24), and affective ToM showed somewhat more consistent associations than cognitive ToM[9]. The result does not invalidate social-cognitive tests; it shows that test performance is not interchangeable with behavior.

Dynamic and multimodal assessment has developed partly in response to this gap. Msika et al. screened 3,884 studies and identified 149 reports representing 65 dynamic and/or multimodal tools[65]. The review found promising paradigms but a shortage of standardized, well-validated instruments, especially for multidimensional and first-person interaction. Thus, increasing ecological realism does not automatically solve psychometric problems.

Ecological validity itself has two separable meanings. Verisimilitude refers to how closely a task resembles everyday activity, whereas veridicality refers to whether the measure predicts real-world behavior. In schizophrenia research, Cavieres et al. found that most studies seeking ecological validity emphasized verisimilitude while relatively few directly tested veridicality[65]. A cinematic or virtual task can therefore look realistic without necessarily predicting social outcome.

The three-mode dysfunction framework offers a specific explanation for the test-behavior gap. Conventional tests often reduce ambiguity, explicitly tell the participant that mental-state information is relevant, constrain response options, and remove reciprocal consequences. These features reduce the need for salience-gated selection, cross-system integration, and rapid network reconfiguration. A patient may therefore show acceptable component skills under scaffolded conditions while failing when the environment demands self-initiated cue selection, temporal integration, uncertainty management, and response updating.

Clinical assessment should consequently be layered. Component tasks remain useful for specifying perceptual, mentalizing, semantic, empathic, or regulatory weaknesses, but they should be interpreted alongside general cognition, informant behavior, pragmatic communication, and naturalistic observation. Discrepancy between apparently intact social-cognitive testing and severe everyday dysfunction should be treated as data-not dismissed as inconsistency-and should prompt consideration of motivation, insight, context sensitivity, and dynamic coordination.

How This Framework Differs From Existing Models

The proposed framework is intended to complement rather than replace existing taxonomies and network models. Its distinctive contribution is to link four levels that are usually treated separately: task-level processing demands, component computations, mechanisms of clinical dysfunction, and context-sensitive network dynamics. Existing models typically emphasize one or two of these levels. The present framework therefore asks not only which construct or network is engaged, but also how a similar social-cognitive phenotype can arise through local computational disruption, impaired interregional integration, or dysfunction of context-appropriate network-state reconfiguration.

This distinction is especially important clinically. Taxonomies such as HITS improve construct definition; hierarchical empathy/ToM models clarify partial functional fractionation; triple-network accounts emphasize large-scale coordination; social-semantic models explain the contribution of conceptual knowledge; and second-person/naturalistic approaches restore reciprocity and ecological context. None of these, however, was designed primarily as a cross-diagnostic neuropsychological framework that explicitly separates evidence-

grounded node and disconnection mechanisms from a testable reconfiguration hypothesis and then uses those mechanisms to explain dissociations between laboratory performance and everyday social behavior (Table 4).

Insert Table 4 here

A Unified Three-Mode Dysfunction Framework

The proposed model has two levels. The processing level describes what information must be represented: social-perceptual/embodied evidence, affective salience, mental-state and social-semantic models, value, and control. The dysfunction-mode level describes how those computations can break down: local node dysfunction, disconnection, or network-reconfiguration dysfunction.

Node dysfunction is the most familiar neuropsychological mechanism. It is strongest when focal lesions, selective degeneration, or perturbation demonstrate a relatively specific and reproducible deficit. Examples include vmPFC contributions to the weighting of harmful intent and fronto-insular degeneration associated with loss of emotional empathy. Node models remain useful but should be framed at the level of computation rather than as claims that a region contains an entire social faculty.

Disconnection becomes necessary when a task requires integration across domains. Social behavior depends on linking observed action to semantic context, inferred intention to value, affective salience to control, and self-related knowledge to another person's perspective. Corpus-callosal injury after ABI and distributed connectivity alterations in FTD provide concrete examples. The prediction is that disconnection should disproportionately impair tasks that require cross-domain binding compared with tasks that isolate component processes.

Hypothesis-generating network-reconfiguration dysfunction is the novel prospective tier of the framework and is less established than node dysfunction or disconnection. It predicts that the accessibility and timing of network states matter independently of average regional integrity. A patient may possess adequate component representations but fail to recruit them at the correct moment, sustain them under uncertainty, or disengage from an obsolete interpretation. Dynamic interaction, fluctuating attention, rigid social behavior, and the laboratory-to-life discrepancy are candidate phenomena, but direct causal evidence remains limited. Accordingly, reconfiguration dysfunction should be treated as a falsifiable mechanistic hypothesis rather than as an established explanatory category.

The three mechanisms can coexist. Node injury can generate downstream diaschisis and disconnection; chronic disconnection can limit the repertoire of reachable states; neuromodulatory or executive abnormalities can alter transition probabilities without producing major focal atrophy. Conversely, an abnormal dynamic-connectivity metric is not automatically evidence of causal reconfiguration dysfunction; it must predict behavior beyond static pathology and ideally be manipulated or temporally linked to performance.

This framework is complementary to, rather than competitive with, taxonomies such as HITS. A taxonomy specifies which social-cognitive constructs should be distinguished; the present model asks how component processes are implemented and how their coordination fails. It is also deliberately different from the psychometric synthesis of Pittet et al.[8] and the cognition-behavior synthesis of Gelmers et al.[9]- those studies establish measurement constraints that a mechanistic theory must explain.

A qualitative clinical mapping of the three dysfunction modes is provided in Figure 3.

Insert Figure 3 here

Clinical inference using the framework

1. *Establish the task demand***.** Identify perceptual, language, semantic, memory, executive, affective, and value components before interpreting a 'social' score.

2. *Look for a component-specific deficit***.** A reproducible impairment across low-context tasks may support node-level dysfunction, especially when anatomically convergent.

3. *Test integration***.** Compare isolated component performance with tasks requiring combination of cues, context, mental states, and value. Disproportionate decline supports a disconnection hypothesis.

4. *Increase temporal and contextual complexity***.** Compare static/explicit tasks with narratives, dynamic scenes, or reciprocal interaction. Selective deterioration supports a reconfiguration hypothesis.

5. *Triangulate with behavior and anatomy***.** Use informant report, pragmatic behavior, lesion/atrophy pattern, and connectivity while controlling for general cognitive status.

6. *Preserve uncertainty*. The framework generates candidate mechanisms; it does not permit diagnosis of a dysfunction mode from a single test or imaging metric.

Testable Predictions

Prediction 1: ambiguity-dependent recruitment. As ambiguity increases, performance should depend increasingly on coupling of perceptual/action-observation systems with mentalizing/social-semantic and executive-control systems. Time-resolved cross-network measures should predict trial-level accuracy beyond activation in any single node.

Prediction 2: salience-gated switching. Unexpected socially relevant cues should be followed by measurable reconfiguration of mentalizing and control systems. Frontotemporal patients with frontoinsular/salience-network injury should show reduced or delayed transition despite partially preserved explicit mental-state knowledge.

Prediction 3: integration load will expose disconnection. Patients with structural or functional disconnection should show a larger performance decrement when tasks require simultaneous integration of emotion, context, intention, and value than when these components are tested separately.

Prediction 4: dynamic metrics will add ecological validity. State-transition probability, dwell time, flexibility, or temporally directed coupling should explain additional variance in real-world social behavior after accounting for conventional social-cognitive scores, global cognition, symptom severity, and static connectivity (Table 5).

Prediction 5: FTD phenotypes will fractionate by mechanism. Loss of emotional empathy should relate more strongly to frontoinsular/salience pathology; social-semantic degradation to ATL disease; and disinhibited value-based choice to OFC/vmPFC-control dysfunction. Similar global social-cognition scores should therefore conceal distinct mechanistic profiles.

Prediction 6: cognitive support will moderate PD social cognition. The difference between cognitively preserved PD and controls should be smaller and less consistent than the difference between cognitively impaired PD and controls, especially on executive- and memory-demanding ToM tasks, consistent with Liu et al.[53]

Prediction 7: action-observation abnormalities will show domain limits. Perturbation or injury affecting inferior frontal/parietal action-observation systems should better predict imitation or action-understanding deficits than explicit false-belief impairment after controlling for language and executive function.

Prediction 8: ecological complexity will magnify reconfiguration deficits. Patients with near-normal static tasks but impaired social functioning should deteriorate disproportionately as assessment becomes dynamic, ambiguous, reciprocal, and self-initiated. The effect should be stronger for measures of veridicality than for superficial verisimilitude.

Prediction 9: diagnostic similarity will not imply network identity. Autism and schizophrenia can show superficially overlapping socio-communicative difficulties but should not necessarily show the same regional or network abnormalities; recent meta-analysis already suggests substantial diagnostic divergence[58].

Insert Table 5 here

Translational and Clinical Implications

The framework favors multidomain profile analysis over a single social-cognition composite. A practical clinical battery should sample social perception/emotion recognition, mental-state inference, social-semantic or pragmatic interpretation, and regulation/decision components, with background assessment of language, semantic memory, attention, executive function, and perceptual ability. Mechanistic attribution should be avoided when these non-social supports are not measured.

Assessment should also distinguish capacity from spontaneous deployment. Explicit tasks estimate whether a component can be used when the examiner defines the social problem. Informant measures and naturalistic tasks estimate whether the patient detects the need for that process and deploys it in real time. The weak test-behavior associations identified by Gelmers et al. make this distinction clinically necessary[9].

For biomarkers, the model predicts limited value for static group-average activation alone. Candidate markers include lesion-network measures, task-dependent connectivity, dynamic state transitions, temporally resolved electrophysiology, and multimodal models linking network variables to specific neuropsychological profiles. However, any proposed biomarker should demonstrate incremental validity over disease severity and conventional cognitive assessment.

Intervention should likewise specify mechanism. Training may target perceptual cue discrimination, explicit mentalizing strategies, social-semantic/pragmatic interpretation, or response regulation. A 2026 systematic review of nonpharmacological social-cognition interventions in neurological patients identified 17 eligible reports and suggested potential benefit across several diagnoses, but the evidence base remains small and heterogeneous[66]. Future trials should therefore include mechanistic outcomes: did treatment improve a component process, cross-system integration, or generalization to dynamic real-world behavior?

Neuromodulation and pharmacology require the same discipline. Stimulation of a cortical node should not be assumed to 'enhance empathy' without demonstrating the predicted component and network effects. Similarly, a drug that changes social behavior may act through arousal, reward, anxiety, executive control, or learning. Mechanistic readouts can distinguish these possibilities.

Cultural and linguistic context must be considered part of the mechanism rather than a nuisance covariate. Social-semantic knowledge, pragmatic inference, eye-gaze norms, emotional display rules, and moral conventions vary across communities. Tests validated in one language or culture cannot be assumed to retain construct validity elsewhere. The dynamic framework predicts that culturally learned priors alter both interpretation of cues and the network configurations recruited to resolve ambiguity.

Limitations and Competing Explanations

This review is integrative rather than systematic. Although the literature was updated using prespecified PubMed/MEDLINE concept blocks and citation chaining, it does not claim exhaustive retrieval, duplicate independent screening, or formal risk-of-bias assessment of every primary study. Narrower empirical questions should be addressed by preregistered systematic reviews and meta-analyses.

The evidence base is uneven. Node-level conclusions are often supported by focal-lesion, perturbational, or neurodegenerative data, and disconnection is supported by structural and functional connectivity evidence across several conditions. By contrast, reconfiguration claims rely more heavily on correlational dynamic imaging, electrophysiology, and naturalistic paradigms. The three dysfunction modes should therefore not be treated as equally established: node dysfunction and disconnection are evidence-grounded tiers, whereas

network-network-reconfiguration dysfunction is explicitly hypothesis-generating and requires prospective validation.

Network labels can create a false sense of anatomical precision. DMN, salience, and frontoparietal/executive networks contain subsystems whose boundaries vary across parcellations, tasks, individuals, and analytic methods. The framework uses these labels as coarse organizational tools, not immutable anatomical entities.

Reverse inference remains a central threat. Activation of TPJ, anterior insula, mPFC, or vmPFC cannot establish that a participant is mentalizing, empathizing, or performing a social-specific computation. Likewise, connectivity does not demonstrate causal information flow. Strong mechanistic claims require convergence from lesions, perturbation, longitudinal degeneration, neurophysiology, or explicitly causal models.

Clinical diagnoses are heterogeneous. bvFTD, AD, PD, TBI, schizophrenia, and autism vary in pathology, stage, comorbidity, medication exposure, cognitive reserve, development, and social environment. Group-average mechanisms are probabilistic and should not be projected onto an individual patient without convergent evidence.

The ecological-validity problem is unresolved. Increasing realism can reduce experimental control, increase cultural and language dependence, and introduce new psychometric uncertainty. Dynamic or virtual tasks should therefore demonstrate reliability and veridicality rather than being assumed superior because they look more natural.

Finally, simpler explanations must remain viable. Apparent reconfiguration deficits may reflect general task difficulty, arousal, attention, processing speed, working memory, or semantic load. The dynamic model earns explanatory value only if its measures predict behavior beyond these alternatives and if manipulating the proposed transition changes the predicted social-cognitive outcome.

Conclusion

Social cognition is neither a set of isolated social modules nor an undifferentiated whole-brain faculty. Current neuropsychological evidence supports a middle position in which partially specialized social-perceptual, embodied, affective-salience, mentalizing and social-semantic, valuation, and executive processes interact within distributed networks whose configuration changes with context. The incremental contribution of the proposed three-mode framework is to connect task-level processing demands with two comparatively evidence-grounded mechanisms, namely node dysfunction and disconnection, and a third, explicitly hypothesis-generating mechanism, network-reconfiguration dysfunction. FTD provides convergent examples of node pathology and functional disconnection; AD and PD demonstrate the importance of domain-general cognitive support; acquired brain injury highlights focal and long-range disconnection; and schizophrenia and autism emphasize mechanistic heterogeneity. Recent psychometric and ecological-validity evidence shows why laboratory task impairment cannot be equated directly with everyday social behavior. The decisive next step is empirical: time-resolved and perturbational measures must show that network transitions add explanatory and predictive value beyond static anatomy, static connectivity, general cognition, and diagnosis. If that criterion is met, the framework would provide a clinically testable bridge from component neuropsychology to the dynamic assembly of adaptive interpersonal behavior.

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Statements and Declarations

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Table 1. The three proposed dysfunction models

Dysfunction mode	**Operational definition**	**Typical evidence**	**Predicted neuropsychological signature**	**Key limitation**
Node dysfunction	Damage or dysfunction of a region/subsystem contributing a relatively indispensable computation	Focal lesion; selective atrophy; perturbation; cell-to-network pathology	Relatively stable deficit tied to the computation even under simplified tasks	Few regions are functionally pure; diaschisis can mimic local effects
Disconnection	Impaired structural or functional integration among components that may remain locally viable	White-matter injury; corpus-callosal damage; network topology/FC; lesion-network evidence	Disproportionate dysfunction when tasks require cross-domain integration	Functional connectivity is not causal communication
Network-reconfiguration dysfunction	Impaired entry into, maintenance of, or transition between network configurations appropriate to changing context	Time-resolved fMRI; EEG/MEG; hidden-state models; dynamic interaction paradigms; perturbation	Performance worsens with ambiguity, reciprocity, temporal updating, or context change despite adequate component skills	Most current evidence is correlational; metrics depend on analytic choices

Table 2. Processing demands in social cognition and major interpretive confounds

Processing demand	**Representative paradigms**	**Principal systems**	**What the evidence supports**	**Major confounds**
Social-perceptual cue extraction	Face/emotion recognition; gaze; prosody; biological motion	Occipitotemporal cortex; fusiform regions; pSTS; auditory temporal cortex	Reliable contribution to extraction of socially informative sensory structure	Vision/hearing; attention; naming; cultural familiarity
Action observation/embodied mapping	Action observation; imitation; body-state/interoception tasks	Inferior frontal/premotor; inferior parietal; pSTS; insula/interoceptive systems	Action matching and selected associations with empathy/interoception; not sufficient for ToM	Motor ability; arousal; stimulus familiarity; construct overlap
Affective salience	Emotion relevance; empathy for pain; social threat/reward	Amygdala; anterior insula; ACC; subcortical salience systems	Relevance, arousal, interoceptive and motivational weighting	General salience; anxiety; autonomic state
Mentalizing/social semantics	False belief; faux pas; trait/intention inference; person/social knowledge	TPJ; mPFC; PCC/precuneus; ATL; DMN subsystems	Mental-state modeling depends partly on semantic/contextual representation	Language; semantic memory; autobiographical memory; task explicitness
Valuation/executive regulation	Moral judgment; norms; social choice; conflict; inhibition	vmPFC/OFC; ACCg; striatum; lateral frontoparietal control systems	Value integration and rule/goal control shape social response selection	Risk/reward sensitivity; working memory; inhibition
Interactive updating	Conversation; joint attention; cooperation/competition; naturalistic narratives	Time-varying coupling among salience, DMN, control, sensorimotor systems	Context-dependent network states can distinguish forms of interaction	Partner behavior; language; culture; reduced experimental control

Table 3. Clinical syndromes mapped onto the three-mode dysfunction framework

Condition	**Representative phenotype**	Node dysfunction	**Disconnection / reconfiguration**	**Interpretive caution**
FTD spectrum	Early empathy, emotion recognition, ToM, social-semantic and behavioral-control deficits	Strong evidence for frontoinsular/salience, vmPFC/OFC, ATL contributions	Strong evidence for network-topology/FC disruption; reconfiguration plausible but under-tested	Variants differ; similar ToM errors can arise through different mechanisms
AD/MCI	Progressive ER and ToM deficits; affective empathy may be relatively preserved	Distributed degeneration rather than a single social node	DMN/memory-semantic disconnection; dynamic dysfunction plausible	General cognition, memory, language and disease severity strongly affect tasks
DLB	Early cognitive/affective ToM impairment reported	Prefrontal, TPJ, fusiform, insular associations	Fluctuation and attentional state make reconfiguration particularly plausible	Evidence base remains small

Condition	Representative phenotype	Node dysfunction	Disconnection / reconfiguration	Interpretive caution
PD	Negative-emotion recognition relatively consistent; ToM/empathy heterogeneous	No single established social node	Frontostriatal/executive and dopaminergic-state mechanisms; stronger deficits with cognitive impairment	Medication, mood, disease stage, motor/visual demands
Stroke/TBI	ER, ToM, empathy and perspective-taking deficits	Focal lesion evidence for component nodes	Corpus-callosal/diffuse injury strongly supports disconnection; dynamic deficits likely	Sensory/language/executive deficits and awareness can confound tests
Schizophrenia	Broad social-cognitive/pragmatic impairment with functional relevance	Stage- and task-specific regional findings	Sensory-to-social propagation, salience/mentalizing/control dysconnectivity; reconfiguration hypothesis	Symptoms, medication, chronicity and environment; no universal neural signature
Autism	Heterogeneous social attention, inference, pragmatics and interaction differences	Universal node-dysfunction model poorly supported	Distributed connectivity and flexibility hypotheses may apply to subgroups	Development, task design, culture and neurodiversity; avoid 'broken mirror' simplification

Table 4: Comparison between present framework with existing frameworks

Existing framework	Primary contribution	Principal limitation for the present question	Increment added by the present framework
Modular / social-brain accounts	Identify reproducible regional specialization	Risk one-region/one-function interpretation; limited account of distributed compensation	Recasts regional effects as node-level computational contributions within distributed systems
HITS taxonomy	Clarifies and organizes social-cognitive constructs	Taxonomic rather than a mechanistic network-dysfunction model	Links constructs to component processes and clinically testable dysfunction mechanisms
Hierarchical empathy–ToM models	Demonstrate graded and partially separable neural organization	Primarily task/neuroimaging organization; limited cross-diagnostic dysfunction analysis	Adds node, disconnection, and dynamic reconfiguration levels
Triple-network model	Explains large-scale salience/DMN/control interactions	Not specific to social cognition or neuropsychological phenotype	Specifies social processing demands and disease-relevant predictions
Social-semantic / ATL accounts	Explain person and social-concept knowledge	Do not by themselves explain full-system temporal coordination	Integrates semantic representations with salience, mentalizing, valuation, and control
Second-person / naturalistic approaches	Restore reciprocity, temporal context, and ecological realism	Primarily paradigm-level perspective; limited clinical mechanism taxonomy	Provides a reconfiguration hypothesis linking dynamic demands to clinical dysfunction
Present framework	Integrates component processing with node dysfunction, disconnection, and dynamic reconfiguration	Requires prospective validation, especially for reconfiguration	Cross-level, clinically oriented, falsifiable synthesis

The comparison is conceptual and is not intended as a formal superiority ranking of existing models.

Table 5. Experimental tests of the dynamic network framework

Hypothesis	Preferred paradigm	Primary readout	Finding that would weaken the model
Ambiguity shifts processing toward mentalizing/control	Parametric ambiguity in action/intention inference	EEG/MEG timing; dFC/effective connectivity	No demand-dependent change in cross-network engagement
Salience gates switching	Unexpected cue embedded in an ongoing narrative or interaction	Anterior-insula/ACC timing; state-transition probability	Switching preserved despite focal salience-system dysfunction
Disconnection impairs integration more than components	Matched isolated-component vs integrated social tasks	Behavioral interaction term; structural/functional connectivity	Component and integration tasks decline proportionally

Hypothesis	Preferred paradigm	Primary readout	Finding that would weaken the model
Dynamic metrics predict everyday function	Clinical cohort + naturalistic task + informant/ecological outcome	Flexibility/dwell time/state transitions in multivariable model	Static regional measures fully account for behavior
FTD phenotypes map to different mechanisms	Multidomain social battery + MRI/connectomics	Salience, ATL, vmPFC/control integrity	All phenotypes relate equally to one global atrophy score
Ecological complexity unmasks reconfiguration dysfunction	Static image -> dynamic narrative -> reciprocal interaction	Performance slope and temporal coordination	No selective worsening with increasing contextual/temporal demand

Figures

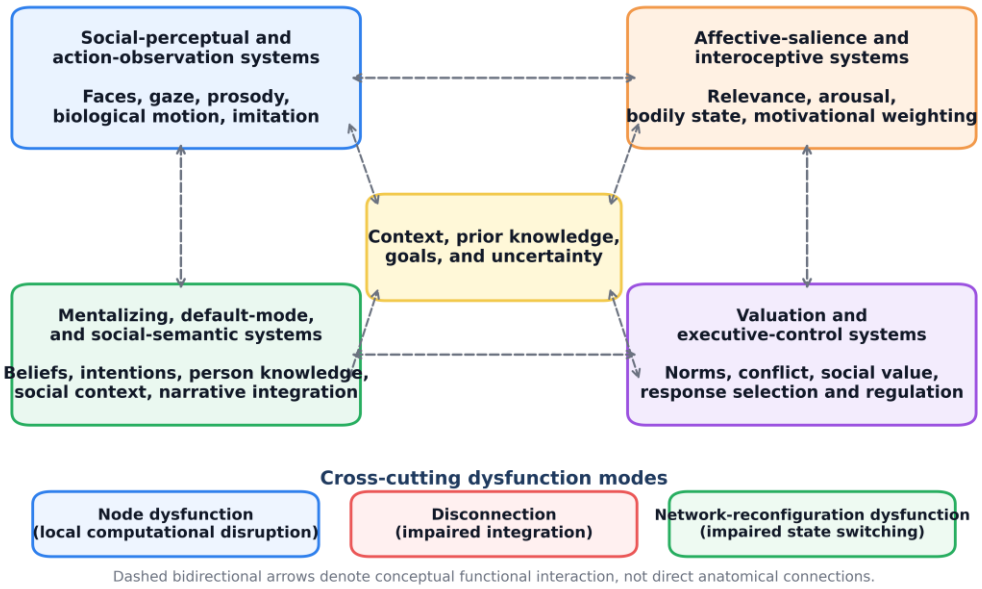


Figure 1. Multilevel processing architecture of social cognition. Adaptive social behavior is conceptualized as emerging from recurrent functional interactions among social-perceptual/action-observation, affective-salience/interoceptive, mentalizing/default-mode/social-semantic, and valuation/executive-control systems. Node dysfunction, disconnection, and hypothesis-generating network-reconfiguration dysfunction are shown as cross-cutting mechanisms rather than diagnosis-specific categories. Dashed bidirectional arrows indicate conceptual functional interaction and should not be interpreted as direct anatomical connections.

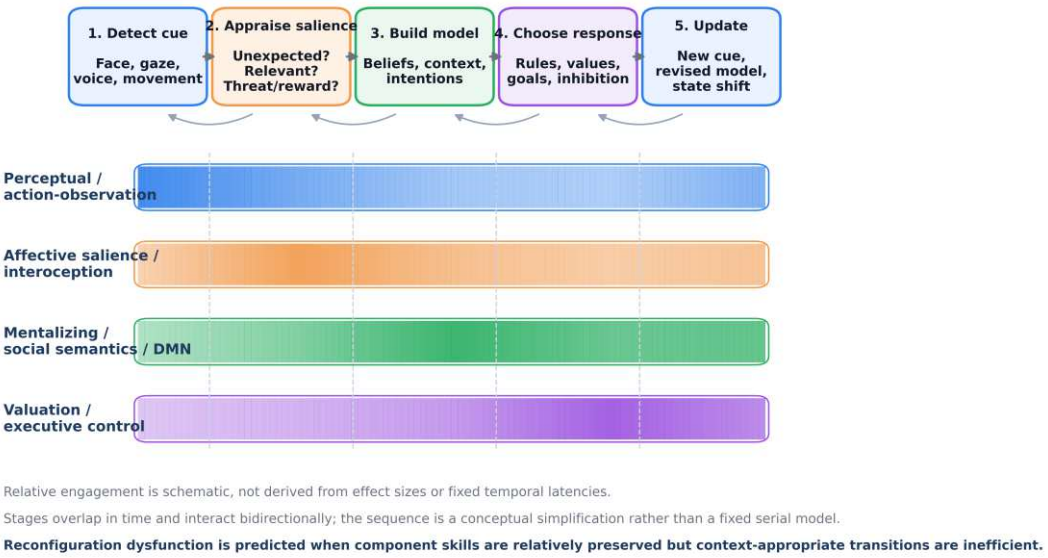


Figure 2. Conceptual dynamic network reconfiguration during social interaction. The relative contribution of perceptual/action-observation, affective-salience/interoceptive, mentalizing/social-semantic/default-mode, and valuation/executive-control systems is illustrated across an evolving social episode. Shading represents schematic relative engagement only and is not derived from quantitative effect sizes or fixed temporal latencies. The depicted stages overlap and interact bidirectionally; the sequence is not intended as a fixed serial physiological model. The reconfiguration component is hypothesis-generating rather than an established clinical mechanism.

Condition	Node dysfunction	Disconnection	Reconfiguration dysfunction	Interpretive status
FTD spectrum	Strongly supported	Strongly supported	Emerging	Node + disconnection best supported
AD / MCI	Distributed / moderate	Supported	Emerging	Predominantly distributed degeneration + disconnection
DLB	Moderate	Moderate	Plausible	Dynamic-state account remains provisional
Parkinson disease	Limited as primary mechanism	Moderate	Hypothesis-generating	Strongly moderated by general cognition
Stroke / TBI / ABI	Strongly supported	Strongly supported	Emerging	Focal and disconnection mechanisms often coexist
Schizophrenia	Heterogeneous	Moderate-strong	Emerging	Distributed and stage/task dependent
Autism	Heterogeneous	Heterogeneous	Subgroup-dependent / emerging	No unitary lesion or network account

Interpretive status is descriptive, not a formal certainty-of-evidence grade.
Reconfiguration dysfunction is presented as an emerging or hypothesis-generating mechanism unless direct time-resolved evidence exists.
Diagnoses can express multiple mechanisms and substantial within-diagnosis heterogeneity.

Figure 3. Clinical mapping of dysfunction modes and interpretive status. Selected neurological, neurodegenerative, neurodevelopmental, and psychiatric conditions are mapped qualitatively onto node dysfunction, disconnection, and hypothesis-generating network-reconfiguration dysfunction. The final column provides a descriptive interpretive status rather than a formal certainty-of-evidence grade. Reconfiguration dysfunction is labelled as emerging, plausible, subgroup-dependent, or hypothesis-generating where direct time-resolved clinical evidence is limited. Individual disorders and patients may express more than one mechanism.