

# Applying the Russian Doll Model of Empathy from Animal Studies to Better Understand Social Cognition across Neuropsychiatric Disorders

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## Abstract

Several disorders, ranging from neurodevelopmental, to psychiatric and neurodegenerative disorders, are associated with impairments in social cognition. Applying the Russian Doll model for social cognition that is based on extensive research in animals will aid in organizing findings across social cognitive tests and across disorders. This multi-layer construct will increase our understanding and fuel possible opportunities for interventions to improve social cognition in patients suffering from various neuropsychiatric disorders. Future research in neurodegenerative disorders may shed light on the proposed evolutionary hierarchy of social cognition, when higher hierarchy functions will be lost in the early stages as a “recapitulate in reverse”. Clinical research on social cognition should not be limited to one social cognitive test in one specific disorder.

**Keywords:** Social Cognition, Transdiagnostic, Primates, Empathy, Autism, Schizophrenia, Borderline Personality Disorder, Bipolar Disorder, Frontotemporal Dementia

## Key points

**Question:** How can we better understand social cognition across neuropsychiatric disorder?

**Findings:** A multi-layer construct, based on extensive research in animals, can increase our understanding of social cognition and fuel possible opportunities for interventions to improve social cognition in patients suffering from various neuropsychiatric disorders.

**Importance:** Social cognition is so essential for survival and quality of life that disturbances in social cognitive functioning have a major impact. Research in patients with social cognitive disorders may aid our understanding of “normal” social cognition. In modern society many non-social cognitive tasks can be supported by technology, making social cognition a more prominent and essential quality.

**Next Steps:** Research using a transdiagnostic approach will yield results that are more specific on social cognition than on a specific disorder. A team of psychiatrists, psychologists, neurologists and fundamental researchers will be best suited for this task.

## Social cognition

Social cognition, defined as all cognitive processes that play in our social interactions, is of great evolutionary importance. It is essential for all species that provide extensive parental care, as tuning in to the needs of their offspring increases their survival rates, and work cooperative towards common goals, as this increases their own survival [1]. In humans several disorders are associated with impairments in social cognition, ranging from neurodevelopmental (autism), to psychiatric (personality disorders, mood and psychotic disorders) and neurodegenerative disorders (frontotemporal dementia).

To date, the majority of studies examining social cognition focus on performance on one or two social cognitive tests in a specific disorder using specific terminology and synonyms. Although each study provides a piece of the puzzle towards understanding social cognition, there are obvious shortcomings. One test will only represent a specific aspect of social cognition, failing to predict real world functioning that is dependent on social interaction [2]. Moreover, social cognition has many aspects and impairment in one domain may or may not be compensated by other (social) cognitive abilities. Next, comparing performance on a social cognitive task in a specific disorder with healthy controls may only reveal hampered global functioning that is non-specific. Therefore, a transdiagnostic model for social cognition integrating its different aspects is needed to increase our understanding of social cognition.

In this article we will discuss the applicability of a model for social cognition based on extensive research in animals [3] and organize findings on social cognition in several human neuropsychiatric disorders accordingly. Studying social cognition across disorders within an evolutionary model will increase our understanding and shed light on possible opportunities for interventions to improve social cognition in patients suffering from these social cognitive disorders.

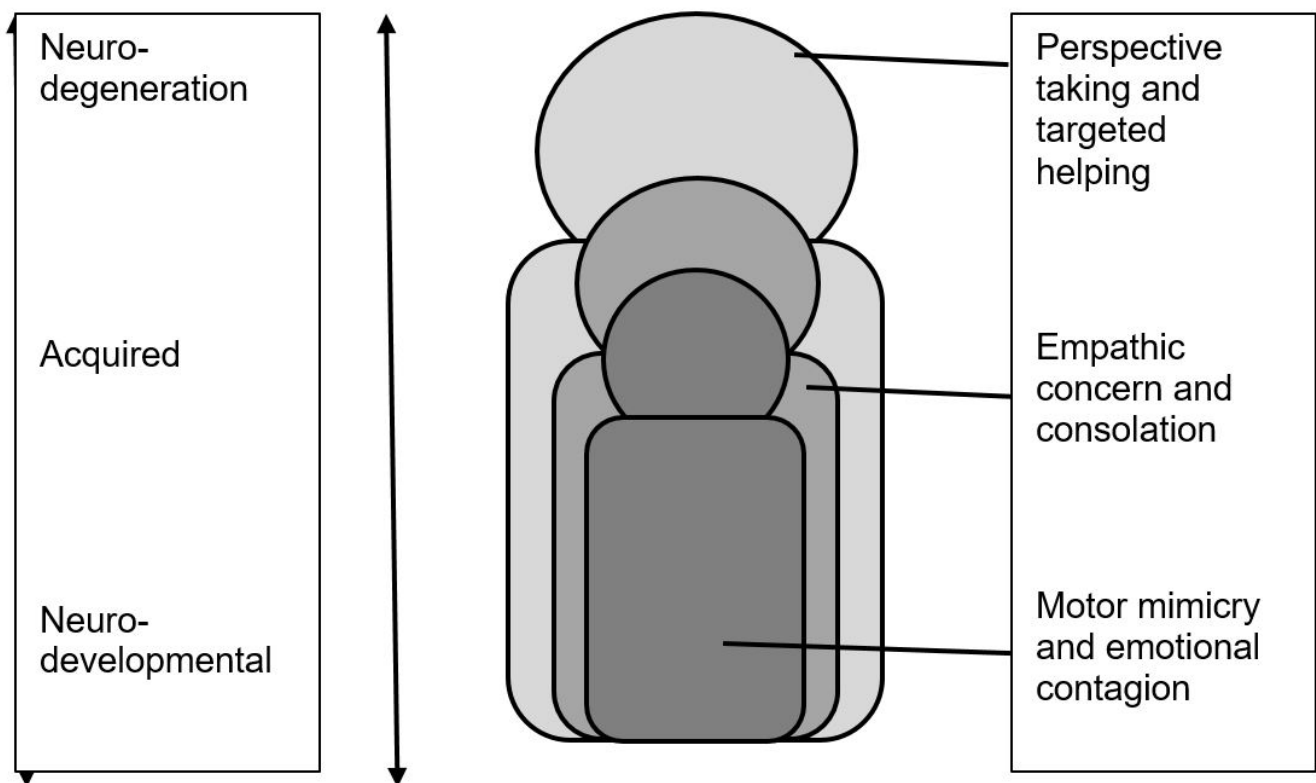
## Research in Animals

Extensive research in animals, both observations in their natural habitat as experiments in laboratory settings have confirmed that mammals are social with various social cognitive abilities. Animals living in groups need to coordinate travel, communicate about danger and assist group mates in need in a fast and efficient manner [4]. Bodily synchronization and sensitivity to the emotional states of others are utilized for rapid spreading of alarm through an entire group or returning to a whimpering youngster. A more complex social cognitive ability such as empathic behavior that requires one to take the perspective of the other into account has been demonstrated to be present in animals as well. For example, dolphins may lift an incapacitated companion to the surface so they can breathe and chimpanzees may bring down fruits from a tree to an elderly female who has lost her climbing abilities [3].

Following the Darwinian assumption that if related species show similar responses under similar circumstances, the underlying proximate are probably homologous (derived from a common ancestor) rather than analogous (independently evolved). From an evolutionary perspective, social cognitive skills will yield long-term benefits with increased survival rates for the individual as well as their offspring. Empathic responses are higher towards similarity, familiarity and social closeness, further underlining the evolutionary models of social cognition [1].

### Hierarchy of social cognition and its interrelational functioning

Empathic behavior has been demonstrated in several primates (chimpanzees, capuchins) and non-primates (dolphins and wolves). Based on these observations the group of Frans de Waal developed the Russian Doll model (Figure 1) [4]. The inner layer core consists of the perception-action mechanism that underlies motor mimicry and emotional contagion. Built around this hard-wired social affective basis, the doll's middle layer includes empathic concern, that requires self-regulation on top of emotional contagion, as one can't be overwhelmed by one's own emotions while comforting a distressed party. In the most outer layer there is targeted helping, defined as assistance based on an appreciation of the other's specific needs. The complexity of social cognition grows with increasing perspective-taking capacities, which depend on prefrontal neural functioning, yet remain fundamentally connected to the basis. A few large-brained species show all of the doll's layers, but most show only the inner ones.



**Figure 1:** The Russian-doll model of the evolution of social cognition. The model, based on research in vertebrated animals, in which various components of social cognitive response have been added layer upon layer during evolution yet functionally remain integrated. Impaired or altered social cognitive responses may occur in neurodevelopmental, acquired (psychiatric) and neurodegenerative disorders. In neurodegeneration the doll's outer layers may be affected first, where in neurodevelopmental disorders the more basic and rudimentary functions may be altered most prominent. As all layers are functionally connected, social cognitive reserve may occur and compensation of one layer by another layer is to be expected

The model presumes that social cognition is a multilayer process that starts with automatic state-matching based on motor mimicry and shared neural representations, enabling sympathetic concern that requires some self-regulation or even targeted helping that is more goal-driven.

In humans the different social cognitive domains, as defined in the Russian Doll model, have been shown to be interrelated. People who can accurately mimic others will be more empathic in daily life [5]. Next to motor mimicry, there is emotional contagion shown by the matching of a hormonal stress response between self and other, that correlates with empathetic concern and perspective-taking in daily life [6]. Once the emotion of the other is mimicked and contagiously felt, some degree of emotional self-regulation is needed for effective altruistic helping. Emotion self-regulation is compromised in neglected orphans, shown in bonobos [7] and humans [8]. This indicates that part of social behavior is vulnerable to external factors in early life.

### Trans diagnostic findings in humans

	Synonyms	Neuro-developmental Acquired Neuro-degeneration					
		Autism	Schizophrenia	Borderline Personality Disorder	PTSD	Bipolar Disorder	Fronto temporal dementia
Motor mimicry and emotional contagion	Motor resonance Bodily synchronization Affect sharing Sensitivity to emotional states of others	Increased susceptibility for emotions of others	Normal to enhanced affect sharing (overly sensitive and reactive to feelings of others)	Reduced facial activity More susceptible to (negative) emotions of others	Reduced contagion	Subtle abnormalities in emotion reactivity	Impaired
Empathic concern and consolation	Emotion processing Social perception	Impaired emotion perception and processing	Poor recognition of facial expressions	Reduced due to inferring emotions and thoughts	Altered or slowed perception of emotions	Difficulties in facial emotion recognition	Difficulties in facial emotion recognition
Perspective taking and targeted helping	(Cognitive/Affective) Theory of mind (ToM) Mentalizing Social behavior	Large impairment in ToM	Inefficient mentalizing (over-attribution or slowly inferring of other mental state)	Impaired e.g. due to idea that others are malevolent	Intact cognitive ToM, disturbed affective ToM	Pronounced risk taking and more hesitation in symptomatic patients	Lack of sympathy/empathy and apathy are core criteria

\*Overview is not exhausting, descriptions of the deficits are cited from the reviews, thereby illustrating the specific terminology and synonyms common in the specific fields of research.

**Table 1:** Overview of synonyms and deficits in various neuropsychiatric disorders according to Russian Doll Model\*

Hampered social cognition is not a pathognomic feature in humans. In the past few years, numerous systematic reviews have been published on social cognitive deficits in various psychiatric disorders such as autism [9], personality disorders [10], post-traumatic stress disorder (PTSD) [11], schizophrenia [12] and bipolar disorder [13] (Table 1). In the behavioral variant of frontotemporal dementia (bvFTD) loss of sympathy/empathy is one of the clinical core criteria [14]. The growing interest in disturbances in social cognition is further illustrated by the numerous remediation and pharmacological interventions aimed at social cognition deficits, mostly in patients with schizophrenia and autism [15, 16]. Across disorders impairments in social cognition appear to have a negative impact on quality of life and functioning [2, 17], explaining the clinical interest.

Although the phenotype of social cognitive deficits may appear similar, neurobiological underpinnings may vary. The most accepted paradigm for the origin of neuropsychiatric disorders is that there is some degree of genetic vulnerability (in some disorders more prominently proven than others) with environmental factors, such as childhood trauma, life-events, exposure to substances, on top of that. For social cognition, as a cross-disorder symptom, one could hypothesize that this may be due to neurodevelopmental altered brain structure (e.g. Von Economy Neurons), neurodevelopmental altered brain function (e.g. early life trauma or neglect), intermittent dysfunction of brain networks (e.g. due to hypometabolism during psychosis or depression) or neurodegeneration of the frontal lobes (e.g. frontotemporal dementia). Depending on the underlying mechanism primarily responsible for the disturbances of social cognition different aspects will be affected.

In order to facilitate our understanding of social cognition and its neurobiological underpinnings in various neuropsychiatric disorders a novel approach that integrates the different aspects of social cognition in a transdiagnostic model is needed. To that aim, we have organized the social cognitive deficits of several major neuropsychiatric disorders according to the Russian Doll model in Table 1. The proposed most rudimental and inner layer of the Russian Doll has varying disturbances, with increased emotional contagion in autism, borderline personality disorder and schizophrenia and decreased emotional contagiousness in PTSD, bipolar disorder and bvFTD. Most striking is the finding that the recognition of facial expression (mostly measured with the Ekman faces test) is impaired to some degree in all disorders, however obviously not in all patients. The most outer layer representing perspective taking and targeted helping is affected and reflecting social functional impairments across disorders. Depending on the diagnosis of the patient group these impairments in social cognition and functioning are labeled accordingly. For example, in bipolar disorders, impaired social cognition is linked to mood resulting in more hesitation or more risk taking and in borderline personality disorder inefficient mentalizing is accounted responsible for impairment in theory of mind. Explaining impaired social cognition of a patient with a specific disorder as a result of other symptoms that are even more characteristic for that disorder is circular reasoning and not moving in the direction of understanding the etiology of social cognitive impairment across disorders. Transdiagnostic research will aid in identifying which social cognitive impairments are disorder-specific, multi-level social cognitive research will aid in identifying which specific social cognitive domains are impaired.

## Applying the Russian Doll model in Neuropsychiatric Disorders

Given the extent of research on social cognition in neuropsychiatric disorders, we have made a selection to illustrate the applicability of the Russian Doll in the following disorders: autism, schizophrenia, borderline personality disorder, bipolar disorder and frontotemporal dementia.

### Autism

A well-known example of a psychiatric disorder with social cognitive disturbances is autism. Autism is defined by persistent impairment in social communication and interaction that manifests with deficits in reciprocity, non-verbal communication and social interactions (DSM 5). Organizing these deficits with the Russian Doll model, one can see that the inner layer has impairments, with impaired motor mimicry (rigid moving and non-reactive facial expressions) and increased contagion (susceptibility for emotions of others). With the absence of intuitive understanding of others and the failure to generate some degree of representation of the other's emotional state in patients with autism, the emotional perception and processing within the middle layer is impaired as is the the-

ory of mind of the outer layer [9]. Interventions targeting social cognition in autism show that some degree of emotion recognition and cognitive theory of mind can be learnt [18], and this aids patients with autism to move around in social situations with more confidence and be a sympathetic person. Despite these trainings, patients with autism may still suffer from stigma as their relative lack of motor mimicry has an impact on the other in every social interaction they have. Future strategies to improve social cognitive functioning in autism could include facial mimicking, in addition to emotion recognition and understanding perspectives of others.

## **Borderline Personality Disorder**

People with a borderline personality disorder (BPD) are characterized by a pervasive pattern of instability in interpersonal relationships, self-image, and affect, as well as markedly impulsive behavior (DSM 5). Their social interactions are fueled with their hypersensitivity for social threat and excessive activation of negative affect. From a psychodynamic viewpoint the key feature, a lack of integration of the concept of self, would be caused by the lack of integration of self-representations and of object-representations under contradictory loving and hateful affect states. Translating this to the Russian Doll model, one could say that for accurate motor mimicry and emotional contagion clear distinction between self and other is warranted, and this is clearly hampered in BPD. There is reduced facial activity, as seen in depressed patients, and increased emotional contagion, especially for negative emotions. A logical consequence of failure to separate one's own state from other's is impaired empathy. In the outer layer affective Theory of Mind (ToM) is hampered, whereas cognitive ToM tested in settings without emotions or affective involvement may be intact [10]. Of note, BPD is often accompanied by comorbid psychiatric disorders such as depression and post-traumatic stress disorder (PTSD), that have a more pronounced negative impact on social cognition in these patients [19], further underlining the notion to study social cognition across disorders.

## **Schizophrenia**

Schizophrenia is defined by the positive symptoms, such as delusions and hallucinations, in combination with negative symptoms, such as apathy (DSM 5). Neurocognitive and social cognitive impairments are not part of the diagnostic criteria but are clearly relevant for prognosis, outcome and global functioning. Findings indicate that patients with schizophrenia are overly sensitive to feelings of others, thereby more susceptible to emotional contagion (inner layer of the Russian Doll) [12]. Next, their interpretation of facial expression is hampered (middle layer). The overwhelming feelings of others followed by inability to indicate the nature and intent of these feelings is hypothesized to lead to hypermentalizing (outer layer) and has been linked to paranoid symptoms [12]. Organizing social cognitive impairments in patients with schizophrenia according to the Russian Doll model may open treatment options that are more aimed at arming towards or limiting emotional contagion and recognizing emotions of others.

## **Bipolar disorder**

In bipolar disorder the most consistent finding on social cognitive impairment is difficulties in facial emotion recognition [13], representing impairments in the middle layer. In addition, subtle abnormalities were shown in emotional reactivity, representing the most inner layer. Bipolar disorder is characterized by mood fluctuations ranging from depression to mania, with apathetic to inhibitory behavior accordingly (DSM 5). Aspects of social functioning such as reward processing and affective decision-making abnormalities were shown to be mood state related [13]. This may implicate that social cognitive impairment in the inner and middle layer, i.e. emotional reactivity, recognition and regulation may be subtle, and compensated resulting in empathic and altruistic behavior, except during mood episodes. To date, perspective taking and targeted helping in patients with bipolar disorder is studied sparsely, the various gambling and risk-taking paradigms are not a true representation of social cognition, but more an indication of disinhibition tendencies. Moreover, bipolar disorder is often associated with childhood neglect and/or trauma. To understand the full extent of social cognition in bipolar disorder future studies should include the relation with social functioning and possible risk factors.

## Front temporal dementia

Frontotemporal dementia (FTD) is caused by progressive nerve cell loss in the frontal and/or temporal lobes with impaired social functioning as one of the earliest symptoms. Deficits in social cognitive abilities have been repeatedly demonstrated in all three layers of the Russian Doll [20]. It can be hypothesized that with neurodegeneration the higher hierarchy functions (perspective taking and targeted helping in the outer layer) will be lost in the early stages of FTD as a “recapitula in reverse”. This could explain the changes in personality and loss of empathy as first symptoms and dependency on the environment for all stimuli (action and emotions) as a sign of motor mimicry and emotional contagion at the later stages. Studying individuals with a genetic mutation for bvFTD while they develop their first prodromal symptoms may increase our understanding of the evolutionary hierarchy of social cognition.

## Concluding Remarks and Future Perspectives

Social cognition is so essential for survival and quality of life that disturbances in social cognitive functioning have a major impact. Research in patients with social cognitive disorders may aid our understanding of “normal” social cognition. In modern society many non-social cognitive tasks can be supported by technology, making social cognition a more prominent and essential quality. Research using a transdiagnostic approach will yield results that are more specific on social cognition than on a specific disorder. In addition, using a model based on social cognition in animals will fuel understanding social cognition from an evolutionary perspective and aid in finding its neurobiological underpinning. A team of psychiatrists, psychologists, neurologists and fundamental researchers will be best suited for this task.

## Declaration of Interest

None of the authors have any disclosures.

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