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Theory of Mind deficits in childhood mental and neurodevelopmental disorders

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Summary

Theory of mind (ToM) is a complex cognitive mechanism which refers to our ability to understand that other people have beliefs, plans, desires, hopes, intentions, and knowledge that may differ from our own mental and emotional states. Theory of mind is critical for social and interpersonal functioning and allows people to make sense of other's behavior. The initial aim of theory of mind research was to record normative development in preschool age children. Almost 30 years ago, when researchers discovered that theory of mind is altered in individuals with autism spectrum disorder, they also explored impairments in ToM in different clinical disorders. Research results indicate the presence of ToM deficits in childhood mental and neurodevelopmental disorders, such as: autism spectrum disorders, attention deficit hyperactivity disorder, oppositional defiant disorder, Gilles de la Tourette Syndrome, fetus alcohol syndrome, mood disorders, eating disorders, or obsessive-compulsive disorder. This article reviews significant studies of theory of mind impairments in individual childhood disorders and selected mental disorders.

Key words: theory of mind, childhood mental disorders, ToM deficits

Introduction

Mindreading, or theory of mind (ToM) [1, 2], constitutes a complex cognitive process whose function is: to predict other people's states of mind; to attribute psychological features to oneself and others and to predict and understand behaviors. It conditions a correct understanding of: mutually exclusive emotions, intentionality of behavior, manipulation, deceit as well as detecting sarcasm and irony. Currently, ToM is not considered to be a homogenous process. On the contrary, we delineate cognitive and emotional (or perceptual) types of ToM. It is still unclear whether ToM is an autonomous cognitive ability (i.e., independent of executive functions, memory

and abstract thinking) (domain specific processes), or an ability to create metarepresentations, dependent on other cognitive processes (domain general processes) [3, 4]. The latter approach stresses interactions between basic cognitive processes, useful in processing social information, such as: joint attention, emotion recognition, facial emotional expression recognition, recognition of emotional prosody cues, as well as understanding of intentions.

Research shows that developmental pathways of ToM are culturally independent and that, despite some environmental effects, ToM related abilities are acquired by neurotypical children at the age of 3–4 years [4]. Recognition of intentions, characteristic of so-called implicit ToM, develops as early as 3–4 months [5] and, according to many researchers, is an indispensable milestone on the pathway to explicit ToM [6]. Development of ToM has a significant influence on communication and social skills, such as: conversation, negotiation, play, making friends. Deficits of ToM are observed in many childhood mental disorders, such as: autism spectrum disorders, intellectual disability, attention deficit hyperactivity disorder, and obsessive-compulsive disorder. This paper aims to review the current body of research related to ToM in selected mental disorders.

Autism spectrum disorder (ASD)

ASD is characterized by deficits in: social relations, communication, behavior, and cognitive functions [7, 8]. Those are observed in many areas, however, due to high idiosyncrasy, a general clinical description of a prototypical ASD patient remains impossible. It is believed that ASD symptoms are related to deficits of ToM [1, 3, 9, 10], a claim which is supported by over 30 years of research. ToM enables the processing of information of a social nature and therefore is crucial for social skills development [6, 10] and for communication sensitive to social context [10, 11]. Impairment of ToM potentially explains problems with social communication and imagination in this group of patients [6, 10, 12].

Children with ASD show specific difficulties, such as inadequate emotional reactions, social withdrawal, troubles initiating contact with peers. Other troublesome areas include: team working, reciprocity, making friends, empathy, and providing emotional support. They perform much worse on tests requiring ToM (such as identifying false beliefs [12, 13], interpreting complex emotional stimuli, understanding intentions, beliefs, metaphors, pragmatics, and humor [3, 6, 9, 10, 12]) compared to their neurotypical peers. Interestingly, not all ASD patients have impaired ToM [12, 14]. Good results in terms of performed tasks seem to be related to good language skills. However, such persons may still fail to apply their ToM abilities in everyday functioning [14, 15].

Attention deficit hyperactivity disorder (ADHD)

Over 60% of patients with ADHD show symptoms of ASD [16]. It has been demonstrated that people suffering from ADHD perform poorer on tests measuring emotional perception and empathy. Links have been made between symptoms of ADHD, especially attention and executive functioning deficits, and ToM [17]. It seems reasonable to link executive functions with ToM, as predicting other people's states of mind requires inhibition of own perspective and fluent switching between different ways of thinking. Moreover, one needs to integrate information from many sources into a single, coherent view.

A negative correlation between ToM test results and attention deficits has been reported. Additionally, executive functioning seems to be impaired especially in children suffering from the so-called inattentive subtype of ADHD. This kind of deficit may deteriorate social skills acquisition, acting as a predictor of social and emotional problems later in life. Social problems may result from inappropriate social perception and delays in the development of appropriate knowledge and concepts. Research shows that children with ADHD perform worse, compared to healthy controls, on emotion recognition tasks in emotionally laden situations. No differences were found for emotionally neutral situations. This suggests a moderating role of emotional state as tasks were similar in terms of cognitive load [17].

People with ADHD have troubles identifying emotions, especially fear and anger [18], regardless of visual stimuli presentation (static, dynamic, picture story) or context information [17, 19]. They do not perform worse on tasks using simple ToM skills, but the difference becomes statistically significant when ToM needs to be engaged in tasks imitating real life situations [20]. Results on attribution of neutral and emotional states are ambiguous. Social problem solving, reasoning and interpretation of social stimuli as well as predicting other people's behavior can be problematic for children with ADHD [21], especially those with comorbid oppositional defiant disorder. Those results seem to corroborate findings on understanding false beliefs – some reports point to serious problems with attribution of mental states of other people [17], while other found no differences against controls.

Oppositional defiant disorder (ODD) and conduct disorder (CD)

Researchers contend that children with ODD and CD perform worse at interpreting social information [22, 23] and that aggression is linked to inappropriate mental states attribution in such a way that aggressive children seem to perceive others as hostile, they react to the literal aspect of communication and external situational manifestations as well as take offence more often [22]. Research on problem solving based on social cues demonstrates that it is much more difficult for children with ODD or CD to code

this type of cues, making adequate response more problematic. Boys with ODD tend to respond aggressively to difficult situations [22–24] and are more confident about winning. A similar tendency to make overoptimistic internal attributions predicts more CD symptoms later in life [25]. Children with ODD or CD with comorbid ADHD perform worse on social cognition tasks than their peers with pure ADHD [26].

There is scant research on social cognition in children with pure ODD or CD, including just two on deficits of ToM. Studies of preschoolers [27] provide valuable guidance. Excellent ToM skills, like taking another person's perspective that enables children to anticipate the behavior of others in response to their behavior, are a protective factor when ODD symptoms develop or become more severe. These findings corroborate those where a negative correlation was obtained between ToM skills (reasoning based on understanding the perspective of others, especially in situations related to exclusion and breaking the rules) and severity of ODD symptoms.

de la Osa et al. [28] found that in preschoolers the severity of ODD symptomatology related to irritability (anger and temper tantrums) correlated with slower response to social and emotional prompts. Impaired ability to code, understand and make conclusions from social cues was linked to worse functioning – children with ODD showed lower flexibility and responsiveness in predicting other people's reactions and responding to them. Moreover, rule breaking, defying adults and blaming others was correlated with better ToM affective tasks scores and better reaction time. These results, albeit counterintuitive, corroborate other findings showing increased neuronal activity in response to affective stimuli in children without antisocial behaviors [29].

Social skills are directly associated with mutual aggression, whereas empathy is associated with it indirectly [22]. The youth with CD present lower empathy levels and perform worse at identifying interpersonal and relational cues. Nonetheless, a meta-analysis by Eisenberg [30] showed moderate relationship between empathy and prosocial behavior. As in the case of children with ADHD, research underlines the importance of environmental factors in developing social skills.

Gilles de la Tourette syndrome (GTS)

ASD symptoms in children with ticks, and with GTS in particular, are an interesting, although not thoroughly researched, subject. The same goes for social cognition in children with GTS. Up to this date, research focused on understanding nonverbal language, inappropriate social interactions, recognition of emotional states based on facial expression, understanding humor and false beliefs, and understanding conflicting emotions. First conclusions were drawn from research on ASD children where children with GTS acted as controls [9]. The latter group presented difficulties with understanding facial emotional expression and understanding nonliteral contents of speech.

Further work evaluated problem solving in social situations. Patients with GTS were impaired on real-life-type decision making and scored much lower, compared to controls, in number of generated solutions and quality of chosen solution. Patients with GTS overly exaggerated the degree of awkwardness of the situation. No differences were found in relation to the level of satisfaction from choice of solution. Interestingly, evaluation of other people's solutions was not problematic for GTS patients. We link this performance to problems with mentalization, as no relationship was found between the quality of problem solving skills and inhibition or executive functions [31, 32]. Moreover, ToM deficits in GTS patients are independent from executive functioning, especially with regard to inhibition and set-shifting.

Problems were also found in: use of sarcasm and metaphors, understanding *faux pas*, attribution of beliefs, emotion reading, understanding of competitive emotions, understanding others' intentions, working under conflict and cooperation [31, 32].

So far, no one addressed how GTS patients regulate social behavior. The problems in understanding *faux pas* may result from the fact that these tasks require attribution of intention, therefore from not being able to put the ToM into practice, rather than from ToM deficits per se. Patients with GTS may understand other people's beliefs but use ToM differently, according to their emotional reactivity, especially in conflict situations [31]. On a neurobiological level, disfunctions in the functioning of the neural networks involving the regions of frontal lobes and basal ganglia, especially the ventro-medial frontal cortex and amygdala – linked to etiology of GTS – are associated with such problems [32, 33].

Eating disorders (ED)

Affect regulation in patients with anorexia nervosa (AN) was a subject of ample research where food restrictiveness was conceptualized as a non-adaptive compensatory strategy against deficits in emotion regulation [34]. It was demonstrated that AN patient got worse scores on emotional mental states attribution measures [35] and substantially worse scores on *faux pas* recognition. They showed limited capacities for social, emotional and cognitive reasoning in both experimental and control conditions, and for reasoning based on text-context relationships within a story [36]. Moreover, fMRI studies revealed lower activation of social cognition neural networks, a result which makes AN patients similar to those with ASD [37]. The assessment of the relationships between the presence of ToM deficits in eating disorders and the severity of co-occurring depressive symptoms presents ambiguous conclusions. Some researchers show a lack of such relationships, concluding that ToM deficits are solely associated with eating disorders, while others suggest a possible mediating role for anxiety and depressive symptoms [37, 38].

On the other hand, the results of studies on patients with bulimia nervosa are inconclusive. Some studies report no ToM deficits [36, 38], while other show specific difficulties within the emotional component of ToM, especially concerning recognition of positive emotions and emotionally neutral states [38].

Fetus alcohol syndrome (FAS)

Rasmussen et al. [39] demonstrated that younger children afflicted with FAS presented problems with understanding false beliefs. This was attributed to a general mental development retardation, as this skill was present in older children [40, 41]. Lindinger et al. [41] assessed 9–11-year-old children with FAS and showed that they had troubles with interpreting emotional expression based on pictures of the eyes, with no significant differences between the study and control group in terms of first – and second-order false beliefs as well as understanding and explaining *faux pas*. After controlling for frequency of fetal exposure to alcohol (regular or occasional), children regularly exposed to alcohol performed much worse than controls in four areas: explaining *faux pas*, explaining high-order false beliefs, interpreting emotional expressions based on pictures of the eyes, and understanding the relationship between emotions and appropriate affect in various social situations.

Further analysis showed that these difficulties should be related to cognitive and social deficits that are specific for prenatal alcohol exposure, rather than executive function deficits. Children who were regularly exposed to alcohol in utero experience problems in the basic aspects of ToM, such as: attribution of mental states necessary for understanding higher-order false beliefs and understanding ambiguous social situations. The type of exposure has not been shown to affect understanding of: *faux pas*, lies, white lies, humor, pretence, double bluff, persuasion, misunderstanding, metaphors, irony, and competitive emotions. Comorbid ADHD correlated with poorer results in recognition of emotions based on eye expressions but did not affect performance on false beliefs tests or other ToM measures. A strong mediating effect of the type of prenatal alcohol exposure was obtained, regardless of ADHD symptomatology [41].

Obsessive-compulsive disorder (OCD)

Research on ToM deficits in OCD patients is scarce. Potential explanations of how ToM deficits influence social reasoning of OCD patients are currently under debate. One line of reasoning is that they are part of the disorder itself, another links them with comorbid mental disorders. Lastly, ToM deficits are conceptualized as consequences of specific neurocognitive deficits or possible associations of selected ToM components with neurocognitive deficits [42–45].

Research results indicate a negative relationship between the severity of OCD symptoms and performance on ToM tasks [43]. Sayin et al.[42] claim that patients with OCD do not differ from healthy controls on simple ToM tasks (such as false beliefs) but are outperformed on more complex ToM tests, such as: understanding metaphors, pragmatic use of language (e.g., hinting tasks, especially understanding double bluff). Moreover, OCD patients are characterized by difficulty reasoning based on ambiguous social cues and they achieve poorer results on tasks requiring taking the perspective of the other person [43]. Additionally, understanding disgust seems to be problematic for them – a finding which corroborates a hypothesis linking OCD to impaired processing and experiencing of this emotion in reaction to perceived 'contamination' or 'pollution'. It should be added that the basal ganglia, traditionally associated with OCD etiology, play an important role in processing emotional stimuli related to disgust. Impaired perception of disgust seems also to be related to the severity of depressive symptoms in OCD patients. It seems that the coexistence of depressive disorders is a mediator rather than a moderator of this effect [46].

The above-mentioned studies did not take into account the differences between the selected ToM components. On the other hand, a work by Lui et al. [44] included three ToM research conditions – cognitive, affective and physical (neutral) one (the so-called Yoni test). Their findings, unlike previous ones, showed no impairment on simple ToM tasks but significant impairment of reasoning and of attribution of complex mental states. These differences may be explained by the fact that the tests used in the previous research took into account only the cognitive aspect of ToM (common false beliefs tests, with tasks based on linguistic processing), unlike the tasks of the Yoni test, which use verbal signals, the line of sight and perception of facial expression..

Liu et al. [44] not only refuted a connection between ToM deficits and the severity of OCD symptoms as well as comorbid depressive or anxiety disorders. However, they did find a link between understanding emotional and mental states and insight into illness. According to Tulaci et al. [45], patients with OCD performed significantly poorer than controls on complex ToM tasks (double bluff) but also on tests of false beliefs, *faux pas* and hinting tasks, with low insight patients performing significantly poorer than their high insight counterparts [47].

Mood disorders

Presence of ToM deficits in adult patients diagnosed with a depressive episode is well evidenced [48–52]. Troubles seem to affect not only the mental and emotional aspects but also seem to result from disturbed processes related to the decoding of information and reasoning processes [49, 52]. The severity of ToM deficits is related to the severity, duration and relapse of depressive episodes [49, 50]. Moreover, fMRI

studies revealed that brain areas related to ToM are hampered in depressive patients [51]. Additionally, depressive patients interpret positive information as neutral and neutral information as negative.

It is unclear whether depressive patients have troubles with perception of emotional facial expressions in general or whether they are limited to positive affect [50] It is also unclear whether these deficits influence the reasoning about mental states of others, but it is known that difficulties in this area persist into the time of remission [50]. Even patients in remission performed poorer than healthy controls on recognizing joy and on self-reported recognition of joy, anger and fear. Perhaps depressed individuals attribute their own emotional states (sadness) to any presentation of negative affect by other people.

People with depressive episode show no difficulties in understanding and reasoning about other people's intentions based on nonverbal cues (body language) and prosody, in the case of minor discrepancies, but have serious difficulties in recognizing sarcasm. They can take another person's intentions more literally [53]. Depressive patients are oversensitive to negative stimuli: their identification of negative affect is much more precise, but no differences were reported in the case of neutral and positive states [53, 54].

Interesting results were obtained by Koelkebeck et al. [55]. They reported significant main effects of gender and attachment style, regardless of clinical symptomatology, on ToM deficits. Female patients with depression performed significantly poorer than controls and their scores were predicted by their attachment style (high scores on dependency and anxiety subscales), empathy and neurocognitive functions. It is worth noting that worse ToM functioning was a predictor of relapse in more than half of the patients in the euthymic phase [54].

A meta-analysis of data from 1990–2012 [56] revealed that patients with bipolar disorder showed impaired ToM not only during active episodes but also during remission. All patients made mistakes in recognizing first-, second – and third-order false beliefs. Additionally, patients with active mania did not understand humor well. Subclinical patients and those in remission still experienced difficulties in understanding false beliefs that are characteristic of active phases. Research on understanding *faux pas* yields conflicting results with some studies reporting difficulties in this area [53, 56], and others reporting intragroup differences (between higher and lower functioning patients) [57]. Bipolar patients have problems with understanding intentions, especially when ambiguous stimuli are involved, as measured by tests of illusion and strange stories. The ability to recognize fear, disgust and joy seems to be related to the phase of the illness (manic, depressive, euthymic) [56].

Recapitulation

The works cited above prove the presence of ToM deficits among children and adolescents with various mental disorders. The severity of deficits and their consequences for functioning vary depending on the type of disorder. Because of the complexity of ToM, a single cause of its deficits in many disorders cannot be outlined. Research points to a complex etiology and highlights a potential link between ToM deficits and language skills, cognitive and executive functioning [10]. Further research concentrates not only on establishing how those factors and their development affect ToM, but also which aspects of ToM they are associated with. In fact, future studies should be focusing on developing characteristic of resources, competences and mechanisms related to ToM obtained during ontogenetic development (e.g., work on the functional profile of the neural networks responsible for ToM in younger children), which would complement the current knowledge of typical development, and thus enable a better understanding of the impact of potential abnormalities in development on the formation and functioning of ToM..

In conclusion, there is a need for more research including advanced neuroimaging techniques and neuropsychological assessment in order to establish neural and cognitive correlates of ToM. This is crucial for finding out which ToM aspect would be associated with functioning difficulties in childhood mental and neurodevelopmental disorders. The combination of those researches and currently fast improving ToM studies, based on advanced technology, online and robot platforms [58], would be a start of new therapy methods, rehabilitation programs designed to meet individual needs of each patient, introduced at the earliest stage of development.

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