

Efficacy of The Metacognitive Training on Brain Injured Subjects

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Abstract

Metacognitive and Theory of Mind difficulties are among the most debilitating and long-lasting consequences of acquired brain injury. Nevertheless, a few studies have investigated the impact of cognitive rehabilitation on their recovery, especially after brain damage. An interesting approach in this field can be found in the Metacognitive Training by Moritz and colleagues, which was originally structured for psychotic subjects. The main aim of this study was to assess the effectiveness of such a training in improving metacognitive functions and social competences in a group of subject with acquired brain injury and frontal lesions. The Metacognitive Training was adapted to neurological patients and was performed twice a week for three months. Each group session lasted one hour. The outcomes highlighted a significant enhancement of metacognitive abilities, while an absence of significant improvements was found in the Social Competence Inventory, probably due to the reduced duration of treatment or to the topics treated during each session, not specifically focused on social cognition. In spite of that, the Metacognitive Treatment demonstrated its effectiveness also in the case of neurological damage.

Keywords: *Acquired Brain Injury; Metacognitive Abilities; Theory of Mind; Metacognitive Training; Frontal Lesions*

Abbreviations

ABI: Acquired Brain Injury; TBI: Traumatic Brain Injury; Tom: Theory of Mind; OCD: Obsessive-Compulsive Disorders; DLPFC: Dorsolateral Prefrontal Cortex; DMN: Default Mode Network

Introduction

Acquired brain injury (ABI) is one of the main causes of cerebral damage among adults, which may have serious consequences and long-term repercussions on patient's life [1]. In addition to cognitive disorders, involving attention, memory and executive functions [2-5] it may cause behavioural and emotional problems, often characterized by apathy, listlessness and indifference or, on the contrary, emotional dysregulation, impulsivity or uncontrolled anger [6].

One of the possible reasons for these difficulties lies in a defective metacognitive ability. The term "metacognition" refers to the capacity everyone has to reflect and track his/her own cognitive and emotional processes, in order to self-regulate in social contexts [7-10]. Related to metacognition is the concept of Theory of Mind, defined by Premack e Woodruff [11] as the ability to think about other people's mental states, such as thoughts, beliefs, emotions or motivations [12] and to use this knowledge to predict and understand their behaviour.

Many studies (e.g.: [13,14]) have highlighted the strong interconnection between these two constructs, which seem to be processed by a similar network of areas, involving both the frontal and temporal lobes [15-18]. Functional neuroimaging research has identified brain structures that seem to be part of this network in healthy individuals, including the temporoparietal junction, superior temporal sulcus,

precuneus, and medial prefrontal cortex [19,20]. In particular, Saxe [21] has proposed that right temporoparietal junction is engaged in achieving a representation of the others' mental states, while medial prefrontal cortex aids with the simultaneous management of different mental representations and seems to be activated in any task requiring the participants to think about themselves and/or about others who are similar to them [22].

Impaired performance in ToM and metacognitive tasks has been reported in patients with acquired brain damage due to various aetiologies, including stroke [23-28], frontotemporal dementia [29,30], neurosurgical lesions [31-33] and tumours [23,27,34]. Overall, metacognitive difficulties have been mostly described in subjects with lesions in the frontal lobes [32], in spite of their aetiology.

The central role of frontal lobes for metacognitive processes and ToM is widely confirmed in the literature. In a fMRI study Schelbel [35] demonstrated that the neural activations of a group of adolescents with TBI and metacognitive deficits, who were asked to perform a social attribution task, were similar to those of healthy controls, with the exception of prefrontal areas, which did not activate. Furthermore, in a meta-analysis on this topic Martin-Rodriguez, et al. [36] showed that one of the variables with a major impact on ToM tasks' effect size was the presence of frontal lobe lesions. Likewise, Carrington and Bailey [19], after revising the findings from functional neuroimaging research, concluded that the medial prefrontal area is the most consistently activated region in studies performed by using ToM tasks.

The frontal lobe lesions may also account for other more general (non-mental) cognitive deficits, which seem to be strongly related to both metacognitive and ToM disturbances: a generalized weakness in inferential skills, combined with working memory limitations, lack of flexibility and the tendency to persevere on invalidated feedbacks may explain the failures on tasks requiring the comprehension of one's own and others' mental states [35-37].

On the whole, these studies suggest a complex "panorama" underlying higher-order mental states, which is further complicated by the assumption that these deficits are long-lasting and, even if they may decrease with time [34], they are still present even in the chronic phase of injury, thus significantly interfering with the subject's functional recovery [38].

Therefore, rehabilitative programs aimed at training these abilities may be useful for adult subjects with ABI [34]: the incapacity to mentally represent one's own and the others' mental states may negatively impact on their social cognition [35,39,40], i.e. the ability to interpret others' behaviour in terms of mental states, to conceptualize relationships between oneself and others and to use these concepts to guide one's own behaviour or to anticipate that of the others [41].

An interesting approach in this field comes from Moritz and colleagues [42], who introduced the Metacognitive Training, a group intervention aimed at enhancing and improving metacognitive abilities in psychotic subjects [43-45]. This program is mainly focused on bringing patients' attention on the dysfunctional way in which they integrate social (e.g. expression and gaze direction) or environmental cues in order to evaluate how to behave or to interpret the actions of others. Through a series of drill and practice activities, they are invited to critically reflect about their cognitive biases and to transfer this knowledge in their daily life. Furthermore, the treatment is meant to provide corrective experiences to participants, with the hope that these will facilitate reduction of maladaptive behaviors and social reintegration [43,45].

Even if the Metacognitive Training has been used with clinical populations other than psychotic patients (e.g. [44]), we are not aware of any study on brain injured subjects.

This is certainly curious, since both schizophrenic and ABI subjects may present with frontal lobe lesions that may account for their metacognitive difficulties [32].

In the light of these data, the main aim of this article is to assess the efficacy of Metacognitive Training in a group of 13 brain injured subjects with metacognitive difficulties and frontal lobe lesions. Our hypothesis is that Metacognitive treatment would lead to a significant improvement of Theory of Mind, metacognitive abilities and social competences in all participants, resulting in an improved quality of life.

Materials and Methods

Participants

The study sample was composed of thirteen adult subjects (six females and seven males) with severe acquired brain injury. Their age ranged from 22 to 55 years ($M = 40,5$; $SD = 9,7$), while their level of education from 8 to 18 years of schooling ($M = 13,4$; $SD = 3,8$). All the participants entered the chronic phase of injury: onset occurred from 2 to 18 years before the beginning of the study ($M = 10,8$, $SD = 7,3$). The injury aetiology was different among subjects: seven presented with severe traumatic brain injury, four with vascular accident, one had anoxic damage and one had cerebral neoplasia. However, despite the heterogeneous lesions, damage occurred in the frontal lobe, as identified by MRI examination and patient's clinical documentation (Table 1). In order to be included in the training, participants had to present with metacognitive and ToM difficulties, as reported by clinicians and caregivers, and as resulted from their previous cognitive assessments. Further inclusion criteria comprised: (1) being at least 18 years old; (2) being at least at the 12th month after the brain injury, in order to have a stable cognitive profile; (3) being Italian-native speakers; (4) possessing adequate cognitive and comprehension skills, as assessed through the Mini Mental State Examination (MMSE) (cut-off score of 24/30; [46]) and the RBANS (global score over the 5th percentile; [47]). Exclusion criteria included: (1) prior history of brain damage or other neurological disease; (2) neuropsychiatric illness; (3) pre-morbid alcohol or drug addiction as reported in the patient's clinical history. A written informed consent was signed by each participants/legal tutor prior to the beginning of the research. The study approval was obtained from the local ethics committee.

Participant	Gender	Age	Educational level	Etiology	Imaging findings	Date of lesion
S1	M	42	13	Aneurysm	Left fronto-parietal lesion	2014
S2	F	48	18	Brain hemorrhage	Bilateral frontal lesion	2009
S3	M	29	18	Severe TBI	Cortico-subcortical left frontal lesion	2014
S4	M	22	8	Severe TBI	Bilateral frontal lesions	2009
S5	F	41	13	Anoxic damage	Diffuse brain damage	2013
S6	M	41	13	Severe TBI	Bilateral frontal lesion	1998
S7	F	49	8	Severe TBI	Bilateral frontal lesion	2006
S8	M	21	8	Brain hemorrhage	Left fronto-parietal lesion	2011
S9	F	44	16	Severe TBI	Right frontal and bilateral fronto-temporal lesions	2005
S10	M	46	8	Severe TBI	Bilateral frontal and left temporal lesions	2003
S11	M	55	13	Aneurysm	Bilateral fronto-parietal lesions	2012
S12	F	39	18	Severe TBI	Bilateral frontal cortex	2001
S13	F	38	18	Brain tumour	Left fronto-parietal lesions	1998

Table 1: Participants' clinical and demographic data ("The study approval was obtained from the local ethics committee").

Experimental design

This study was carried out over a three-month period and comprised three main phases.

- Pre-training assessment. The first assessment, preceding the beginning of the training, included two tests focusing on metacognitive functions and social skills: The Social Competence Inventory (SCI; [48]) and the Theory of Mind Assessment Scale (ThOMAS; [49]). The participants' answers were recorded and then transcribed verbatim by one of the authors (V.G.). After transcription, they were analyzed. The scoring procedures were performed independently by two raters and then compared among them. Acceptable inter-rater reliability was defined as $k \geq 0.80$. The residual discrepancies were resolved through discussion.

- Training. The treatment, based on the metacognitive training by Moritz., *et al.* [42,43,45] had a duration of three months and was performed twice a week. Every session lasted one hour.
- Post-Training. Immediately after the end of the training program, the ThoMAS and the SCI tests were re-administered to participants, in order to examine the efficacy of the treatment.

Materials

Metacognitive abilities were assessed by means of the Theory of Mind Assessment Scale [49], a semi-structured interview (including several open-answer questions) asking the subject to reflect both on his/her and the others' mental states, feelings or emotions and to report some examples to contextualize the answers given.

The assessment of social skills was performed through the Social Competence Inventory (SCI; [48]), a self-administered scale consisting of 48 items. Questions were divided into seven subscales, investigating the main components of social skills, such as empathy; social sensitivity (i.e. the sensitivity everyone has towards the other people he/she is interacting with); coping abilities; self-esteem; social expressivity (i.e. the ability to be successfully engaged in conversations); emotional sensitivity (i.e. the capacity of properly recognising and interpreting other's emotional states) and self-monitoring abilities.

The Metacognitive training

The intervention was structured according to the Metacognitive Training by Moritz., *et al* [42,43,45]. This treatment is divided into eight modules, targeting cognitive errors and problem solving biases which can cause misleading interpretations of the others' mental states and feelings and, therefore, metacognitive difficulties. The problematic thinking styles, which have been recognized to potentially foster the development of delusions, comprise an increased self-serving bias (module 1), jumping-to-conclusions behaviour (module 2 and 7), a bias against disconfirmatory evidence (module 3), deficits in Theory of Mind (module 4 and 6), over-confidence in memory errors (module 5), and depressive cognitive patterns (module 8). Every session aims at making participants aware of such problems by helping them to critically reflect and gradually modify their current repertoire of problem solving skills. This process is possible through two fundamental steps. The first deals with knowledge translation: current research findings on cognitive biases are explained to patients and illustrated by multiple examples. The second is the demonstration of the negative consequences of cognitive biases. Exercises targeting each bias are used to show the fallibility of human cognition, inviting subjects to critically reflect about that. Furthermore, patients are offered alternative strategies allowing them to arrive at more appropriate inferences, thus avoiding 'cognitive traps' [43].

The original structure of the training was partially modified by two clinical psychologists, expert in the field of neuropsychological rehabilitation, in order to be adapted to brain injured subjects.

Firstly, all the expressions referred to "psychotic disturb", "schizophrenia" or "hallucinations" were replaced with "acquired brain lesion" and "dysfunctional thought(s)". Then, the final part of each module became more focused on the daily consequences of metacognitive disturbances, by asking participants to report their own experiences and discuss them with the other members of the group.

The following is a description of the main changes made to each module:

Module I: "Attributional style"

The main changes made to this module concern the part focused on "hearing voices", which was replaced with a section inviting patients, during the causal attribution process, not to trust the "common thinking" - and then the first impression - but to take into account all the elements available, trying to be flexible and "ready to change their mind". Patients were then asked to think about alternative explanations to everyday situations, by taking into account attributional styles focused on themselves (it's my fault), on the others (it is the others' fault) or on the case. Each style was analyzed in detail, considering the advantages and disadvantages that characterized it, with particular attention to the practical aspects in the everyday life.

Module II: “Object identification”

In line with Moritz’ training, the first section of this module highlighted the tendency of patients with brain injury and frontal lesions to exclusively focus on an idea, without being able to change it according to the contextual information. Then, the implications of such a lack of flexibility were brought to light. In practical exercises the participants were invited to reflect on the fact that not always the apparently more likely response is the correct one and that, to accomplish a task in an appropriate manner, it is necessary to take into account as much information as possible.

Module III: “Corrigibility”

In the first part of this module the relationship between Theory of Mind and cognitive flexibility was analyzed, with particular reference to the ability to change one’s beliefs on the basis of the references coming from the environment. Then, images in reverse order were shown to patients, who were asked to choose the most feasible alternative, but remaining ready to “change their mind” according to the new information provided.

Module IV: “To empathize...I”

Empathic difficulties were attributed to a problem related to the Theory of Mind [50]. In the first (more theoretical) section, the characteristic difficulty found in subjects with frontal lesions to interpret and welcome the others’ emotional experiences was described. Subsequently, emotions recognition tasks -both from faces and gestures - were administered. The stimuli had increasing difficulties: at the beginning, they represented exclusively the person; then, the exercises required to integrate more elements in order to understand the emotion felt by the subject.

Module V: “Memory”

In the introductory section the main memory deficits that can follow a brain injury were described. It was then explained that often, in response to mnemonic deficits, false memories could emerge, through which people try to make sense of a reality in part unknown. The factors that contribute to the formation of these often fallacious memories and the modes to recognize them were also shown.

Module VI: “To empathize...II”

This appears to be a deepening of the fourth module, whose work was focused on the Theory of Mind. In the theoretical section, it was further clarified the importance of the signals sent by the interlocutor in order to empathize with him/her, and understand his/her communicative intentions and moods. The social implications of such a faculty were also shown, asking patients to provide examples from everyday situations and showing how the lack of empathy can lead to misunderstandings, especially at a interpersonal level. Then, participants were asked to understand other people’s intentions on the basis of their behaviour, by inhibiting their point of view in order to put themselves in other people’s shoes.

Module VII: “Picture interpretation”

This theoretical section brought back the concepts of the second module in a more detailed way, while the part related to the practical exercises requested the analysis of a series of ambiguous stimuli. In order to find a solution to the exercise, participants were invited to talk and discuss. In this way, the focus was also put on their ability to communicate with each other, listening to the other’s opinion and replying appropriately, while respecting the speaking turns.

Module VIII: “Self-esteem and mood”

The last module, purely focused on drop in mood often caused by the sudden onset of an illness, was addressed less thoroughly. In this case the depression characteristics and their role in changing the perception that individuals have of themselves and of their surroundings were described. It was then reported the frequency of depressive episodes as a result of events that had drastically altered an individual’s life, by stressing the importance of sharing these moments with others, rather than facing them alone.

Results and Discussion

Statistical analysis

The statistical analysis was carried out with the SPSS software, Version 22. All dependent variables were normally distributed and were therefore analyzed with parametric tests. A paired sample T-test was performed in order to compare the scores obtained by participants with ThOMAS and SCI tests in the pre and post-training evaluations. Significance p level was set at 0.05.

Results

As results pointed out, the ThOMAS mean score significantly increased in the second evaluation ($t = -7.303$; $p < 0.01$; Figure 1). On the other hand, no significant improvement was found in the SCI between the first and the second assessment ($t = -0.693$; $p = 0.503$; Figure 2).

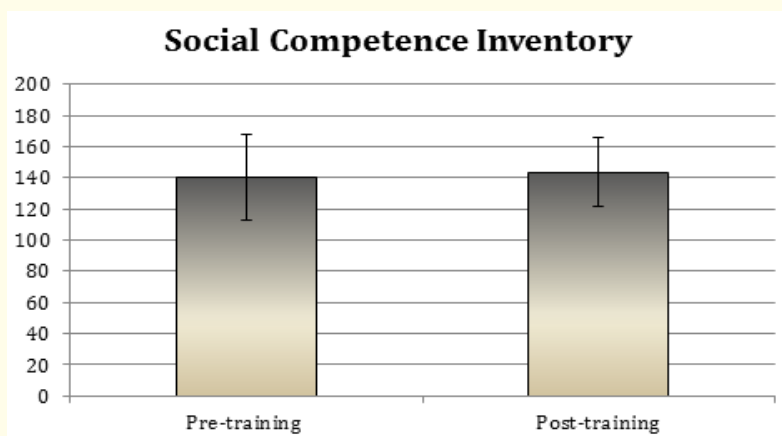


Figure 1: pre-and post-treatment results of the Social Competence Inventory. Asterisk indicates when the difference between groups is statistically significant. (between the first and the second assessment ($t = -0.693$; $p = 0.503$)).

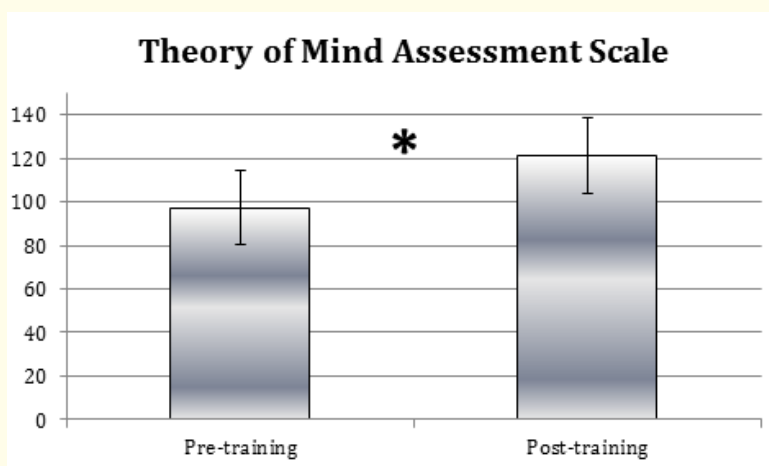


Figure 2: pre-and post-treatment results of the Theory of Mind Assessment Scale. Asterisk indicates when the difference between groups is statistically significant. (between the first and the second assessment ($t = -0.693$; $p = 0.503$)).

Discussion

The main purpose of this study was to evaluate the efficacy of the Metacognitive Training [42] in a group of subjects with acquired brain injury and frontal lesions. It was hypothesized that this treatment would have significantly improved the patients' metacognitive functions, thus positively impacting on their social competences.

Our initial assumptions were only partly confirmed: even if participants' performance at the ThOMAS significantly increased, their social competences, assessed through the SCI, did not improve after treatment.

Such a result may be due to different reasons: first of all, this construct may not have been sufficiently treated in the training. Social cognition is a very multifaceted concept, associated with a wide range of cognitive and affective aspects, such as social knowledge/competence, emotion detection (e.g. faces, prosody, and irony; [51]) and social reasoning, which need to be accurately treated to induce significant changes in the subject's behaviour. Instead, the Metacognitive Training mainly focused on participants' cognitive biases in interpreting the signals coming from the context or from their interlocutors, while less attention was paid to the social consequences of this dysfunctional way of thinking.

Another possible explanation may lie in the reduced duration of the treatment which was performed twice a week for three months. This frequency might not have been sufficient to induce noteworthy changes in participants' social competences.

However, in spite of the lack of significant improvements at the SCI, this treatment has shown to increase participants' metacognitive and ToM-related abilities. More specifically, in the post-treatment evaluation, they became more capable to name and contextualize their own mental states, feelings and emotions and to give specific examples coming from their everyday life. Furthermore, they could better differentiate between their own and the others' thoughts, sensations and desires: for instance, while in the first assessment many patients could not understand the difference between the questions "When you feel good, does something change in you?" and "When the other people realize that you are feeling good, does something change in them?", after the training an increased understanding was found and they could give more appropriate examples.

This improvement might also be due to the structure of this intervention, in which every module was well organised and the topics were treated in a linear way, so that participants' working memory and executive functions were not excessively charged.

These data seem to suggest that, although the Metacognitive Treatment was originally structured for psychotic subjects [42,43,45] and its effectiveness was only tested on obsessive-compulsive individuals (OCD; [44]), it might be useful also for neurological patients. In our opinion, a common aspect among these clinical populations is the presence of frontal lobe dysfunctions, which may account for their difficulty in representing their and others' thoughts, feelings, and actions across time [52].

Converging neuroimaging evidence indicates the noticeable involvement of frontal regions in both schizophrenia and obsessive-compulsive disorders [53,54]. The first data on this topic ascribed the neuropsychological and affective abnormalities observed in psychotic patients to a defective functioning of the circuits connecting the prefrontal cortex (DLPFC) with basal ganglia [55]. Likewise, Frith [56] proposed that schizophrenia was associated with anomalies in connections among medial prefrontal cortex, dorsolateral prefrontal cortex, and anterior cingulate cortex. Similar results were found in OCD patients, where a first model proposed by Wise and Rapoport [57] suggested a dysfunction of the circuits connecting the basal ganglia with the orbito-frontal cortex.

These data have been further confirmed by recent brain imaging experiments, more focused on the dysfunctional connectivity associated with psychiatric disorders. According to Buckner, *et al.* [58] schizophrenia might be caused by an over-activity of the Default Mode Network (DMN), which includes the posterior cingulate cortex, the retrosplenial cortex and parts of the ventromedial prefrontal cortex [59], supporting a series of mentalizing activities, both internally focused [60,61] and assuming the others' perspective.

These conclusions were further confirmed by Menon [62], who proposed that most major psychiatric disorders could be explained by an aberrant intrinsic interconnectivity between the networks involved in the control and monitoring of higher-order cognitive functions (such as the salience network, the central executive network and the DMN; [54]).

Similar findings were also reported with regard to ABI: a study carried out in 2011 by Sharp and colleagues [63] found that traumatic brain injury was associated with abnormal regional brain activation and dysfunctional connectivity among brain networks, especially fronto-temporal ones, supporting high-level cognitive functions.

Such maladaptive connectivity could be significantly improved by adequate clinical interventions (e.g. [64-67]), able to gradually re-integrate more advantageous neural connections, by activating, in a targeted manner, the network underlying the processing of a specific function.

This is the case of the training here described, where, in line with Robertson and Murre's suggestions [68] the intervention on meta-cognitive functions included both bottom up and top down approaches, in order to increase more functional and adaptive plasticity. In the first approach, simple cognitive and perceptive tasks (see Module II, III or V) were used to facilitate patients' understanding of the misleading way they had to analyze and integrate contextual information.

In the second approach, metacognitive functions were considered from a more global and generic perspective, asking participants to critically discuss about them, by comparing and integrating their perspectives into the others', even if different. This was also possible thanks to the use of a group intervention, where participants could experience, in a rehabilitative and protect setting, their way of interacting, in order to gradually modify it and make it more functional and efficient.

Despite the interesting and innovative data, a number of limitations must be considered in evaluating the results if this preliminary study, which needs to be further developed. The first is related to the lack of a control group, that would have been useful to rule out any spontaneous recovery, not specifically due to the treatment. However, the sample of our study included only subjects in the chronic phase of injury, so that a spontaneous improvement might be reasonably ruled out, especially in such a complex construct as the metacognitive abilities. Another limit of our study is the small sample size (13 TBI subjects). This aspect might have significantly influenced the significance of our results. However, in the clinical practice working with broader groups of subjects could be confounding and ineffective. Furthermore, we used self-reported measures to assess both metacognitive and ToM-related abilities, as well as social competences. These tests might be influenced by a number of variables, including the lack of self-awareness and biased perception [69,70]. However, as the average length of time post-injury was 10.8 years, and difficulties are likely to improve over time [71], the current findings shall be considered valid.

Nevertheless, this study has been the first to adapt the Metacognitive Training to a group of neurological patients. Furthermore, it highlighted the importance of including interventions on metacognitive and affective functions in clinical practice, especially in the presence of severe brain lesions involving core-areas as the frontal lobes [72].

Future studies should increase the size of the sample and include a control group, in order to definitively exclude spontaneous recovery, not expressly due to specific rehabilitative interventions. Moreover, it would be interesting to extend the treatment duration, in order to devote more time to every module and to its emerging contents.

Conclusions

This study investigated the efficacy of the Metacognitive Training [42] in enhancing metacognitive and ToM-related abilities in a group of subjects with acquired brain injury and frontal lesions. Although improvements were just limited to the ThOMAS scale (participants' performance at the SCI did not show significant increase), these data seem to demonstrate the effectiveness of this treatment also in case of neurological patients.

The inclusion of an intervention specifically focused on higher-order mental functions, such as the metacognitive ones, is consistent with the holistic rehabilitation proposal by Ben-Yishay, *et al.* [73], who often stressed on the usefulness of differentiating among cognitive, emotional, social or functional consequences of brain damage, as all these aspects may markedly interfere with the patient's recovery and should therefore be involved in the rehabilitative program.

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Conflict of interest statements

The Authors declare that there is no conflict of interest.

Bibliography

1. Holleman M., *et al.* "Effects of intensive neuropsychological rehabilitation for acquired brain injury". *Neuropsychological Rehabilitation* (2016): 1-14.
2. Park NW and Ingles JL. "Effectiveness of attention rehabilitation after an acquired brain injury: A meta-analysis". *Neuropsychology* 15.2 (2001): 199-210.
3. Dockree PM., *et al.* "Behavioural and physiological impairments of sustained attention after traumatic brain injury". *Cognitive Brain Research* 20.3 (2004): 403-414.
4. Ciaramelli E., *et al.* "Central executive system impairment in traumatic brain injury". *Brain Cognition* 60.2 (2006): 198-199.
5. Lundqvist A., *et al.* "Computerized training of working memory in a group of patients suffering from acquired brain injury". *Brain Injury* 24.10 (2010): 1173-1183.
6. Dams-O'Connor K., *et al.* "The impact of previous traumatic brain injury on health and functioning: A TRACK-TBI study". *Journal of Neurotrauma* 30.24 (2013): 2014-2020.
7. Flavell JH. "Metacognition and cognitive monitoring: A new area of cognitive-developmental inquiry". *American Psychologist* 34.10 (1979): 906-911.
8. Cicerone KD and Tupper DE. "Cognitive assessment in the neuropsychological rehabilitation of head-injured adults". *Clinical neuropsychology of intervention* (1986): 59-83.
9. Ownsworth T., *et al.* "A metacognitive contextual intervention to enhance error awareness and functional outcome following traumatic brain injury: a single-case experimental design". *Journal of the International Neuropsychological Society* 12.1 (2006): 54-63.
10. Chiou KS., *et al.* "Metacognitive monitoring in moderate and severe traumatic brain injury". *Journal of the International Neuropsychological Society* 17.4 (2011): 720-731.
11. Premack D and Woodruff G. "Does the chimpanzee have a theory of mind?" *Behavioral and Brain Sciences* 1.4 (1978): 515-526.
12. John S., *et al.* "Theory of Mind in Mild Traumatic Brain Injury". *Journal of the Indian Academy of Applied Psychology* 39.2 (2013): 254-259.

13. Northoff G., *et al.* "Self-referential processing in our brain: a meta-analysis of imaging studies on the self". *Neuroimage* 31.1 (2006): 440-457.
14. Mitchell JP, *et al.* "The link between social cognition and self-referential thought in the medial prefrontal cortex". *Journal of Cognitive Neuroscience* 17.8 (2005): 1306-1315.
15. Stone V. "The role of the frontal lobes and the amygdala in theory of mind". *Understanding Other Minds: Perspectives from Autism*, 2nd ed. (Eds S. Baron-Cohen, H. Tager-Flushberg, D. Cohen), Oxford University Press, Oxford, (2000): 253-273.
16. Gallagher HL and Frith CD. "Functional imaging of "theory of mind". *Trends in Cognitive Sciences* 7.2 (2003): 77-83.
17. Amodio DM and Frith CD. "Meeting of minds: the medial frontal cortex and social cognition". *Nature Reviews Neuroscience* 7.4 (2006): 268-277.
18. Abu-Akel A and Shamay-Tsoory S. "Neuroanatomical and neurochemical bases of theory of mind". *Neuropsychologia* 49.11 (2011): 2971-2984.
19. Carrington SJ and Bailey AJ. "Are there theory of mind regions in the brain? A review of the neuroimaging literature". *Human Brain Mapping* 30.8 (2009): 2313-2335.
20. Dodell-Feder D., *et al.* "fMRI item analysis in a theory of mind task". *Neuroimage* 55.2 (2011): 705-712.
21. Saxe R. "Uniquely human social cognition". *Current Opinion in Neurobiology* 16.2 (2006): 235-239.
22. McDonald S., *et al.* "Cognitive factors underpinning poor expressive communication skills after traumatic brain injury: Theory of mind or executive function?" *Neuropsychology* 28.5 (2014): 801-811.
23. Channon S and Crawford S. "The effects of anterior lesions on performance on a story comprehension test: Left anterior impairment on a theory of mind-type task". *Neuropsychologia* 38.7 (2000): 1006-1017.
24. Happé F., *et al.* "Acquired theory of mind impairments following stroke". *Cognition* 70.3 (1999): 211-240.
25. Siegal M., *et al.* "Theory of mind and pragmatic understanding following right hemisphere damage". *Brain and Language* 53.1 (1996): 40-50.
26. Stone VE., *et al.* "Frontal lobe contributions to theory of mind". *Journal of Cognitive Neuroscience* 10.5 (1998): 640-656.
27. Stuss DT, *et al.* "The frontal lobes are necessary for theory of mind". *Brain* 124.2 (2001): 279-286.
28. Surian L and Siegal M. "Sources of performance on theory of mind tasks in right hemisphere-damaged patients". *Brain and Language* 78.2 (2001): 224-232.
29. Gregory C., *et al.* "Theory of mind in patients with frontal variant frontotemporal dementia and Alzheimer's disease: theoretical and practical implications". *Brain* 125.4 (2002): 752-764.
30. Snowden J., *et al.* "Social cognition in frontotemporal dementia and Huntington's disease". *Neuropsychologia* 41.6 (2003): 688-701.
31. Happé F., *et al.* "Acquired mind-blindness following frontal lobe surgery? A single case study of impaired "theory of mind" in a patient treated with stereotactic anterior capsulotomy". *Neuropsychologia* 39.1 (2001): 83-90.

32. Rowe AD, *et al.* "Theory of mind impairments and their relationship to executive functioning following frontal lobe excisions". *Brain* 124.3 (2001): 600-616.
33. Stone VE, *et al.* "Acquired theory of mind impairments in individuals with bilateral amygdala lesions". *Neuropsychologia* 41.2 (2003): 209-220.
34. Milders M, *et al.* "Impairments in theory of mind shortly after traumatic brain injury and at 1-year follow-up". *Neuropsychology* 20.4 (2006): 400-408.
35. Scheibel RS, *et al.* "Brain activation during a social attribution task in adolescents with moderate to severe traumatic brain injury". *Social Neuroscience* 6.5-6 (2011): 582-598.
36. Martín-Rodríguez JF and León-Carrión J. "Theory of mind deficits in patients with acquired brain injury: a quantitative review". *Neuropsychologia* 48.5 (2010): 1181-1191.
37. Geraci A, *et al.* "Theory of Mind in patients with ventromedial or dorsolateral prefrontal lesions following traumatic brain injury". *Brain Injury* 24.7-8 (2010): 978-987.
38. Bibby H and McDonald S. "Theory of mind after traumatic brain injury". *Neuropsychologia* 43.1 (2005): 99-114.
39. Schmidt AT, *et al.* "Emotion recognition following pediatric traumatic brain injury: longitudinal analysis of emotional prosody and facial emotion recognition". *Neuropsychologia* 48.10 (2010): 2869-2877.
40. Babbage DR, *et al.* "Meta-analysis of facial affect recognition difficulties after traumatic brain injury". *Neuropsychology* 25.3 (2011): 277-285.
41. Baron-Cohen S. "The evolution of a theory of mind" (1999).
42. Moritz S, *et al.* "Metacognitive skill training for patients with schizophrenia (MCT)". Manual. Hamburg: *VanHam Campus Verlag* (2005).
43. Moritz S and Woodward TS. "Metacognitive training in schizophrenia: from basic research to knowledge translation and intervention". *Current Opinion in Psychiatry* 20.6 (2007): 619-625.
44. Moritz S, *et al.* "How to treat the untreated: effectiveness of a self-help metacognitive training program (myMCT) for obsessive-compulsive disorder". *Dialogues in Clinical Neuroscience* 12.2 (2010): 209-20.
45. Moritz S, *et al.* "Sowing the seeds of doubt: a narrative review on metacognitive training in schizophrenia". *Clinical Psychology Review* 34.4 (2014): 358-366.
46. Measso G, *et al.* "The mini-mental state examination: normative study of an Italian random sample". *Developmental Neuropsychology* 9.2 (1993): 77-85.
47. Randolph C, *et al.* "The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS): preliminary clinical validity". *Journal of Clinical and Experimental Neuropsychology* 20.3 (1998): 310-319.
48. Rydell AM, *et al.* "Measurement of two social competence aspects in middle childhood". *Developmental Psychology* 33.5 (1997): 824-833.

49. Bosco FM., *et al.* "Th.o.m.a.s.: An exploratory assessment of Theory of Mind in schizophrenic subjects". *Consciousness and Cognition* 18.1 (2009): 306-319.
50. De Sousa A., *et al.* "Why don't you feel how I feel? Insight into the absence of empathy after severe traumatic brain injury". *Neuropsychologia* 48.12 (2010): 3585-3595.
51. Spikman JM., *et al.* "Deficits in facial emotion recognition indicate behavioral changes and impaired self-awareness after moderate to severe traumatic brain injury". *PloS one* 8.6 (2013): p. e65581.
52. Baird A., *et al.* "Cognitive functioning after medial frontal lobe damage including the anterior cingulate cortex: a preliminary investigation". *Brain and Cognition* 60.2 (2006): 166-175.
53. Abbruzzese M., *et al.* "Frontal-lobe dysfunction in schizophrenia and obsessive-compulsive disorder-A neuropsychological study". *Brain and Cognition* 27.2 (1995): 202-212.
54. Penner J., *et al.* "Medial prefrontal and anterior insular connectivity in early schizophrenia and major depressive disorder: A resting functional MRI evaluation of large-scale brain network models". *Frontiers in Human Neuroscience* 10 (2015): 132.
55. Weinberger DR., *et al.* "Physiologic dysfunction of dorsolateral prefrontal cortex in schizophrenia: I. Regional cerebral blood flow evidence". *Archives of General Psychiatry* 43.2 (1986): 114-124.
56. Frith C., *et al.* "PET imaging and cognition in schizophrenia". *Journal of the Royal Society of Medicine* 85.4 (1992): 222-224.
57. Wise S and Rapoport J. "OCD: is it basal ganglia dysfunction in OCD in children and adolescents". *Obsessive Compulsive Disorder in Children and Adolescents*. Washington, DC: American Psychiatric Press (1989).
58. Buckner, *et al.* "The brain's default network". *Annals of the New York Academy of Sciences* 1124.1 (2008): 1-38.
59. Sharp DJ., *et al.* "Network dysfunction after traumatic brain injury". *Nature Reviews Neurology* 10 (2014): 156-166.
60. Gusnard DA., *et al.* "Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function". *Proceedings of the National Academy of Sciences* 98.7 (2001): 4259-4264.
61. Mason MF., *et al.* "Wandering minds: the default network and stimulus-independent thought". *Science* 315.5810 (2007): 393-395.
62. Menon V. "Large-scale brain networks and psychopathology: a unifying triple network model". *Trends in Cognitive Sciences* 15.10 (2011): 483-506.
63. Sharp DJ., *et al.* "Default mode network functional and structural connectivity after traumatic brain injury". *Brain* 134.8 (2011): 2233-2247.
64. Laatsch L., *et al.* "Investigating the neurobiological basis of cognitive rehabilitation therapy with fMRI". *Brain Injury* 18.10 (2004): 957-974.
65. Chen AJ and D'Esposito M. "Traumatic brain injury: from bench to bedside to society". *Neuron* 66.1 (2010): 11-14.
66. Chiaravalloti ND., *et al.* "Examining the Efficacy of the Modified Story Memory Technique (mSMT) in Persons With TBI Using Functional Magnetic Resonance Imaging (fMRI): The TBI-MEM Trial". *The Journal of Head Trauma Rehabilitation* 30.4 (2015): 261-269.

67. Sacco K, *et al.* "Concomitant use of transcranial Direct Current Stimulation and computer-assisted training for the rehabilitation of attention in traumatic brain injured patients: behavioral and neuroimaging results". *Frontiers in Behavioral Neuroscience* 10 (2016): 57.
68. Robertson IH and Murre JM. "Rehabilitation of brain damage: brain plasticity and principles of guided recovery". *Psychological Bulletin* 125.5 (1999): 544-575.
69. McDonald S and Saunders JC. "Differential impairment in recognition of emotion across different media in people with severe traumatic brain injury". *Journal of the International Neuropsychological Society* 11.04 (2005): 392-399.
70. Williams C and Wood RL. "Alexithymia and emotional empathy following traumatic brain injury". *Journal of clinical and experimental neuropsychology* 32.3 (2010): 259-267.
71. Long CJ and Ross LK. "Handbook of head trauma: Acute care to recovery". *Springer Science & Business Media* (1992).
72. Yuan, P and Raz, N. "Prefrontal cortex and executive functions in healthy adults: a meta-analysis of structural neuroimaging studies". *Neuroscience & Biobehavioral Reviews* 42 (2014): 180-192.
73. Ben-Yishay Y and Daniels-Zide E. "Examined lives: Outcomes after holistic rehabilitation". *Rehabilitation Psychology* 45.2 (2000): 112-129.

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