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A MOTOR IMAGERY TRAINING FOR IMPROVING ACTION COGNITION RESULTS IN THE REDUCTION OF RESIDUAL SYMPTOMS AFTER MAJOR DEPRESSIVE DISORDER: A SINGLE-CASE STUDY

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Abstract

The high rate of recurrence and residual symptoms of depression continuously motivate the search for new treatments addressing this challenging condition. In this single-case study, we reported the application of a cognitive rehabilitation skills training based on remote kinematics as a treatment for residual depression by targeting deficits of mental simulations of action. We found that the administration of Kinect-based rehabilitation training resulted in important improvements showed by significant mean baseline reductions (MBLR) of difficulty in imagining positive actions (MBLR= 54 %), negative emotions (MBLR = 36.3 %), cognitive flexibility (MBLR= 69.7 %), depressive symptoms (MBLR= 80%), and physical retardation (MBLR= 50 %). Similarly, improvements in positive affect (MBLR = 107 %) and vividness of motor imagery for positive actions were registered (MBLR= 100 %). We also found unique effects of our intervention such as reports of involuntary action simulations to distant stimuli or extended affordances. The training was well-accepted and the patient considered it was an entertaining way to do physical exercises and to get in physical and mental shape. We concluded that further scientific research of remote Kinematic interventions in depression may be warranted.

Keywords: embodied cognition; action simulation; Kinect; depression; rehabilitation.

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Theoretical and Research Basis for Treatment

Major depressive disorder (MDD) is one of the leading causes of disability worldwide with high recurrence rates and a chronic course (World Health Organisation; WHO, 2017). Although treatments for acute episodes of major depression are moderately effective in short-term (Undurraga & Baldessarini, 2012), the high rates of relapse (up to 75%, Hollon et al., 2005), recurrence and residual symptoms have always been a challenge for the treatment of depression. It was estimated that after treatment, 30–50 % of recovered patients are still troubled by residual symptoms (Hollon et al., 2005). Moreover, residual symptoms result in impaired functioning and increased relapse of depression (Beshai et al., 2011; Simet al., 2016). Currently, the National Institute for Health and Care Excellence (NICE) guidelines recommendation for people with residual depression is to continue either with antidepressant treatment or with psychological therapy focused on residual symptoms that act as maintenance factors (avoidance, rumination and interpersonal difficulties, NICE, 2019).

One way to improve the efficacy of psychological treatment for residual depression is to adapt the treatment to specifically address core residual symptoms (Hvenegaard et al., 2015). Previously, addressing rumination as a residual symptom has been shown as a promising strategy for treatment of residual symptoms (e.g., Watkins et al., 2007, 2011). Yet, rumination was not the only residual symptom of depression. Recent reports suggested treating other types of residuals, such as cognitive deficits associated with psychomotor retardation, as a viable target for developing future treatments for residual depression (Bortolato et al., 2016). One such cognitive deficit linked to psychomotor retardation was alteration of motor imagery (Bennabi et al., 2014). Motor imagery has been defined as mental simulations of action or mental rehearsal of actions/movements without executing the movements involved (Decety, 1996; Jeannerod, 2001). Action simulations have been proposed as key cognitive markers involved both in higher thinking (self-efficacy, Bandura, 1997) and control of conscious actions (Land et al., 2013) which are affected in depressive disorder. Thus, targeting deficits of action simulations may improve several thought processes and action related manifestations as well, making the motor imagery deficits appealing for the treatment of residual depression.

Building on the existing classifications of general mental imagery in clinical disorders (Holmes et al., 2016; Pearson et al., 2013, several processes and types of motor imagery were distinguished. Based on a computational model of mental imagery, Kosslyn et al. (2006) proposed (1) generation, (2) maintenance, (3) inspection, and (4) transformation processes of action simulations (Pearson et al., 2013). According to the mechanism of generation of action simulations can be (1) voluntary (mental action transformation, such as mental rotation tasks) and (2) involuntary action simulations (automatic mental rehearsal). Furthermore, according to whether the individuals have access to the content of simulations, action

simulations were identified as conscious (e.g., mental rehearsal or transformation) or as implicit (e.g., action identification, hand laterality judgments). Specifically, motor imagery was classified as (1) visuomotor imagery (imagining doing an action from the first perspective) and (2) kinesthetic imagery (imagining the feelings of movement during the action).

Previous research has suggested that depressed individuals are slower than non-depressed individuals in the mental generation (Bennabi et al., 2014) and transformation of actions (Chen et al., 2013). They imagine actions less vividly (Holmes et al., 2016), more general (Dickson & Moberly, 2013) and using an external perspective (Lemogne et al., 2006) than non-depressed individuals. This research highlighted a deficit of motor imagery in depressed individuals. Moreover, motor imagery deficits and, more generally, psychomotor retardation were proposed to play an important role in (residual) depression because they: (a) represent a common residual symptom and they remain significant after remission from depression (Bennabi et al., 2014; Bortolato et al., 2016; Gorwood et al., 2014); (b) are associated with reduced responsiveness to both common antidepressant (SSRIs) and cognitive-behavioural interventions (Dunkin et al., 2000); (c) prospectively predict the severity and the recurrence of depression (e.g., Bennabi et al., 2014; Chen et al., 2013; Hasselbalchet al., 2010; Wagner et al., 2015), and d) are proposed to underlie the gender differences in depression (Oshiyama et al., 2018).

We hypothesized that motor imagery deficits are potential targets for treatment because manipulation of different types of motor imagery such as action simulations in thinking of depressed individuals (1) resulted in improvements in emotional reactivity (Watkins, Moberly, & Moulds, 2008) and problem-solving (Watkins & Baracaia, 2002; Watkins & Moulds, 2005), (2) mediated the effect of well recognized cognitive mechanisms on depression manifestations (rumination, Moberly & Watkins, 2006), (3) moderated the effects of mindfulness cognitive-behavioural treatments (Kuyken & Moulds, 2009), and (4) promoted behavioural activation (Renner et al., 2017). Previously, it has been suggested that improving motor imagery deficits have potential therapeutic benefits for the treatment of depression. However, to our knowledge, there were no studies that investigated interventions targeting the rehabilitation of motor imagery in depression.

In this case study we investigated the effects of rehabilitation of motor imagery in the thinking of a patient with a previous history of MDD. We reported the application of a cognitive rehabilitation intervention based on remote kinematics (Kinect) as a treatment for residual depression by targeting an intermediate cognitive endophenotype: deficits of motor imagery (mental simulations of action). We expected that following motor rehabilitation training, the patient will experience improvement in cognitive processes affected by psychomotor retardation such as action simulations (frequency and easiness), subjective reports of kinaesthetic sensation and speed at psychomotor tasks. We also expected that the patient will show improvement in higher action cognition such as hopelessness.

Mental Simulation of Action in Depression

Deficits of motivation and action are core features of MDD. Increasing evidence across multiple research paradigms on motor imagery showed that depressed individuals are also characterized by deficient representational dimensions of action systems. For instance, deficits of motor simulation of action in thinking of depressed people have been evidenced in both explicit (i.e., motor imagery, Bennabi et al., 2014; Chen et al., 2013; Rogers et al., 2002) and implicit (i.e., action identification, Watkins et al., 2011; external visual orientation, Lemogne et al., 2006; Kuyken & Howell, 2006; overgeneralisation of goal representations, Dickson & Moberly, 2013) cognitive tasks across multiple research perspectives such as motor imagery, action identification, visual orientation, and goal representations. Furthermore, emerging research indicated that these deficits play the role of maintenance mechanism; they moderated treatment effects and improved with antidepressant treatment. Thus, the deficits in motor imagery were promising targets for treatment of depression. Of importance, studies showed that (1) deficits in thinking-related action simulations in depressed people moderated the effect of well recognized cognitive mechanisms on depression manifestations (rumination, Moberly & Watkins, 2006), predicted depression recurrence (Chen et al., 2013), and moderated the effects of mindfulness cognitive-behavioural treatments (Kuyken & Moulds, 2009), and (2) promoting simulations of action in thinking of depressed individuals resulted in improvements in both emotional reactivity (Moberly & Watkins, 2006; Watkins et al., 2008) and problem-solving (Watkins & Baracaia, 2002; Watkins & Moulds, 2005).

There was a growing interest in depression research for evidencing alterations of explicit mental simulations (mental imagery) and imagery-based treatment strategies in depression (Holmes et al., 2016; Renner et al., 2017). Yet, the main focus was on positive and negative explicit visual imagery rather than whether rehabilitation and promotion of motor simulations in thinking would be effective in depressed individuals with motor imagery deficits or recurrent depression. This paper presented a motor imagery rehabilitation training programme developed to enhance and promote motor simulations in thinking aimed to reduce residual motor imagery deficits in a formerly depressed patient.

Embodied Distorted Cognition: Integrating Motor Simulations in Distorted Cognition

We based our intervention on an embodied model of distorted cognition (Tiba, 2010, 2018; Tiba et al., 2012; Tiba & Manea, 2018) applied for deficits in action cognition. According to the embodied model of distorted cognition (in certain contexts) cognition has the ability to re-use the experience-related systems (affective and motor brain resources) referenced by cognition. Furthermore, it is proposed that this ability is responsible for the emotional and behavioural effects of cognition. When these experience-related systems are altered, the alterations are incorporated

into cognition that carries them into our mental states and maintains them in higher cognitive processing or in the top-down determination of behaviour. This model explains the application of cognitive rehabilitative methods beyond the basic cognitive processes into action cognition and thinking. According to the application of the model to action cognition, the quality (and the effect on emotion and behaviour) of higher motor cognition are largely dependent on the quality of sensorimotor resources that were simulated in thinking (Glenberg, 2010). Thus, higher motor cognition such as planning, imagery, action, linguistic sentences, and perceived ability to carry out actions can be rooted in sensorimotor resources. Since depressed individuals are deficient in the use of sensorimotor resources, these deficiencies result in deficits in higher action cognition (thinking about action, self-efficacy, and thoughts with reduced ability to influence action etc.) and negative cognitive adaptations (i.e*.,* hopelessness). Furthermore, this model hypothesized that there are several control mechanisms of sensorimotor simulations (e.g., language, episodic memories, schemas, body gestures and postures, environmental contexts and body states) that can be altered and may result in distorted activation of embodied simulations in thinking (Tiba & Manea, 2018).

This model is distinct from the metaphoric model of hopelessness (Lindeman & Abramson, 2008). Opposed to the metaphoric model of hopelessness that focuses on simulations of motor incapacity we have focused on deficits in recruiting sensorimotor resources in thinking. Yet we have not excluded the important role of metaphors of motor incapacity in depression (Lindeman & Abramson, 2008) but we included them as verbal or visual image control mechanisms for recruiting sensorimotor simulations in thinking. To improve higher action cognition, our training aimed both to rehabilitate motor and sensorial resources of action systems (by Kinect practice) and to develop habits for the use of the mechanisms that promote sensorimotor resources in thinking (such as action memory, action language, and gestures that act as supports for motor simulations). Furthermore, our training not only improved the embodiment of action cognition in depressed individuals, but also its grounding in the environment by developing extended (training by Kinect to act on out of reach objects), augmented (by mimicking gestures and fluent postures) and prospective (imagining future contexts for action) affordances (action possibilities afforded by the physical characteristics of the stimuli). Similarly, interventions targeting sensorial mechanisms in depression by using light therapy (recommended for seasonal mood disorders) have recently received supportive evidence as both a stand-alone or add-on (with fluoxetine) treatment for depression (Lam et al., 2016). The promising role of motor imagery training as an effective intervention for depression was also suggested by evidence showing that motor imagery training induces changes in brain networks (default mode network) that are impaired in depression (Chen et al., 2015). Additionally, interventions based on increasing motor activity such as physical exercises (Cooney et al., 2013) or behavioural activation (Cuijpers et al., 2007) were effective in treating depression.

The main objective of this case study was to illustrate and explore the effects of a novel intervention targeting rehabilitation of action simulation in action cognition in a previously depressed individual. We hypothesised that the intervention improves the mental simulations of action, action-related cognition and it reduces psychomotor retardation and depressive symptomatology in a previously depressed individual. Because of the novelty of the intervention we selected a single case study design that allowed the ongoing calibration of the intervention based on direct feedback from the participant. Moreover an idiographic approach allows us to observe in depth fluctuations in the functioning of the participant.

Case Presentation

The Patient

Maria (identifying details, including the client's name, have been modified to protect the anonymity of the client) was a 37-year-old woman who lives alone. She expressed her interest in undergoing an after-depression consolidation treatment. She signed an informed consent form to participate in this programme and she received no incentive for her participation. At the commencement of treatment, she had a full-time job in a marketing department. She had divorced 1 year prior to the beginning of the program. The divorce ended a 10 years troubled marriage. At the assessment interview, Maria reported that she was still experiencing some negative mood and fatigue but not meeting the threshold for a depression episode diagnosis. She also reported reduced interest in doing things. According to the structured clinical interview for DSM-5 criteria, no current depressive episode was identified. She reported no rumination or negative thinking apart from worries and some concerns about problems at work. She reported being stressed about her job. She was countering these thoughts by adaptive self-talk.

History

Maria was the single child of a mixt Romanian-Hungarian family. She remembered being loved by both parents in her early childhood, yet she characterized her mother a**s** being overprotective and critical. She reported that during adolescence her father faced financial difficulties and started drinking and became abusive. She tried to help her father overcome his drinking problem and rescue her family. During that time she learned she was worthy only by doing things perfectly, saving others and being approved by significant others. Many of her symptoms were tracked back in her late adolescence. She left her home town at 19 years old enrolling in university studies. She had two long-term relationships. Soon after graduating from her university studies she got married. SCID 5 interview identified two episodes of depression in the past. The patient's history of depressive

disorder had begun 8 years previously when she was 29. At that time she had experienced panic attacks for several months. Consequently, she developed a depressive episode. The first episode of depression lasted about 9 months and was marked by dysphoric mood and loss of hope about the future. The main trigger of the episode was the death of one of her best friends. She reported that she saw a psychiatrist only 6 months after the beginning of panic attacks and depression when both the panic disorder and depressed mood were severe. The psychiatrist prescribed her medication (escitalopram (Cipralex) and clonazepam (Rivotril) she took for 2 years. Following treatment, the panic attacks stopped and the depressed mood was ameliorated.

The second episode started 2 years later when her husband started to abuse alcohol. Lack of affection, loneliness, verbal abuse, and constant accusations of guilt about his drinking problem were the main triggers of this second episode. The second episode was more severe and began with psychomotor retardation and negative thoughts about self and future. Yet, she remembered the second episode as lasting significantly less than the first one (about 3 months). She also had social anxiety and avoided social contacts due to fear of others being critical regarding her husband's use of alcohol. Her presentation was marked by severe dysphoric mood and social isolation. During this depressive episode she refused to go to work. She received the same medication (Cipralex and Rivotril) that she discontinued after the first episode. Medication improved her functioning and enabled her to go to work but she had no other activities or social contacts. She met no additional diagnosis of anxiety disorders or other personality disorder yet she had anxiety in social situations linked to expectations that she would be negatively evaluated by others because her husband was an alcoholic.

The first depressive episode she had has not been treated with medication for several months. The depression subsided after medication was started. Yet Maria reported that she continued to feel significantly depressed for a long period afterwards. During her second depressive episode (2 years later) she started cognitive behavioural treatment (CBT). CBT treatment lasted for 2 years up to 2016. During the treatment, she learned about the CBT model of depression and how to challenge depressive thoughts. A significant part of the treatment focused on behavioural activation and social problem-solving. She also learned to control perfectionism and the feelings of loneliness. She had a recurrence of depressed mood when she divorced her husband due to his alcohol problems. She had two boost sessions of CBT 6 months before the application of the MIKE treatment.

Assessment

During the evaluation sessions, Maria presented as engaged and interested. She was well oriented to time and place. Maria showed adequate insight into her

symptoms and past manifestation of depression. She denied alcohol or drug use. She also denied suicidal ideation and plans to complete a suicide. At the time of assessment, she displayed a good range of affect reactivity. There was no evidence of hallucinations or psychotic phenomena. Maria appeared to lack energy, and sat in a slumped position. She complained of feeling slowed down but only mild signs of psychomotor retardation were observed. Her speech was low in rate and pitch but not markedly slowed-down. Maria's assessment consisted of a face-to-face assessment in which SCID 5 interview was administered along with clinical scales. Clinical and cognitive scales were administered again one week after the end of the treatment.

Two weekly assessments for main variables were completed before intervention (for the 2 weeks before the training), one mid-treatment (at the end of the week 2), and one week after the end of the intervention. The mid-treatment assessment consisted of 4 daily assessments (the patient did not complete the form for the fifth day) that were aggregated in one score. Given the weekly assessment of data, data analysis included visual inspection of data, minimal important clinical difference (Norman et al., 2003) and mean baseline reduction (MBLR; Campbell, 2003). Mean baseline reduction measures the average reduction of behaviour from baseline calculated by subtracting the mean of posttreatment scores from the mean of the baseline scores, dividing by the baseline score and multiplying by 100 (Campbell, 2003). Based on suggestions of Bell et al. (2009) MBLR values were interpreted as being small (.20), medium (.50) or high (.80).

Posttreatment and follow-up assessments were averaged to provide a post treatment score. According to minimal important difference it was considered that an improvement in more than one-half of standard deviation shows a minimal clinically important difference (Norman et al., 2003). Normative clinical and non-clinical data were used to compare the scores of the patient to establish the significance of change.

Clinical Measures

The structured clinical interview for DSM-5 (SCID5 CV; First et al., 2015)

The SCID 5 was administered by an experienced clinician (A.T.) for both past and current diagnoses. The interview was used pre and post-treatment to ensure that Maria met the study criteria and to examine whether diagnostic status changed across the course of treatment. At pre-treatment, no current diagnosis of a major depressive episode was identified. Two major depressive episodes were identified in the past along with a past diagnosis of panic disorder. Post-treatment the clinical interview revealed no depressive episode or other clinical syndromes.

16 items Quick Inventory of Depressive Symptomatology (QIDS-16 SR; Rush et al., 2003)

It is a 16-item questionnaire measuring the severity of major depressive disorder symptoms. Ratings are made on a four-point scale ranging from 0 to 3.anchored at all points by a description. For example, Question 11, *view of myself* is anchored at 0= "I see myself as equally worthwhile and deserving as other people", 1= "I am more self-blaming than usual", 2="I largely believe that I cause problems for others", and 3="I think almost constantly about major and minor defects in myself" (Rush et al., 2003).

Generalized Anxiety Scale 7 (GAD-7; Spitzer et al., 2006)

It is a self-report scale measuring the severity of generalized anxiety disorder (GAD). GAD-7 contains 7 items related to DSM criteria for GAD.

The Ruminative response styles questionnaire (RRS; Nolen-Hoeksema & Jackson, 2001)

We used the short form of RSS scale. The scale contains 10 items that assess ruminative responses to sad and depressed mood. Participants rate the frequency of ruminative strategies used. Higher scores reflect higher levels of rumination. RRS assesses the less helpful style of rumination, with a number of items focusing on abstract evaluations of the self (e.g., Think "Why do I always react this way?''), with elevated scores on the RRS predicting worse outcomes. RRS was administered pre-treatment and post-treatment.

Kuopio Ischemic Heart Disease Hopelessness Scale (KIHD-HS; Everson et al., 1996)

It is a short scale comprising two items: (1) "I feel it is impossible for me to reach the goals that I would like to strive for" and, (2) "The future seems hopeless to me and I can't believe that things are changing for the better" (Everson et al., 1996). Maria had to indicate whether agree strongly, agree somewhat, disagree somewhat and disagree strongly for each question.

Trial Making Test (TMT; Partington & Leiter, 1949)

It is a commonly-used neuropsychological drawing test that can measure psychomotor retardation (Buyukdura et al., 2011). The TMT consists of two parts: TMT-A requires the drawing of lines sequentially to connect 25 encircled numbers distributed on a sheet of paper in ascending order. Task requirements are similar for TMT-B, except that the subject must alternate between numbers and letters (1, A, 2, B, 3, C, and so on). The score for each part represents the amount of time required to complete the task. It was originally designed to test processing speed (TMT-A) or cognitive flexibility (TMT-B). We used the classic form of the test at pre-treatment (Partington & Leiter, 1949) and an alternative form at post-treatment (Cranston & Blanton, 2016).

The Behavioral Activation for Depression Scale (BADS-short form; Manos et al., 2011)

It measures avoidance and behaviours targeted for treatment by behavioural activation. The BADS-sort form consists of 9 items on a 7-point Likert scale ranging from 0 (*not at all*) to 6 (*completely*)

Repeated measures

Maria completed a form containing items referring to several variables. The form was completed each time in the 19.00-20.00 time intervals.

Mood

We used ten adjectives describing emotion (e.g., sad, depressed, happy) that assessed the level of negative and positive emotions. Items were previously used in a validated version of Profile of Affective Distress (PDA; Opris & Macavei, 2007). Additional items describing fatigue, psychomotor retardation, activation, hopelessness were included. Maria endorsed how much she experienced the emotion described by each item on a Likert type scale from 1 (*not at all*) to 5 (*very much*) for the last week for the pre-treatment and post-treatment assessments. For mid-treatment assessment, Maria completed the same form (at the end of the day) endorsing the daily level for each item.

Psychomotor Retardation

Three items from Mood Spectrum Self-Report (MOODS-SR; Dell'Osso et al., 2002) were selected to continuously assess the behavioural aspects of psychomotor retardation: (1) fatigued, weak, or tired for the smallest task; (2) physically "slowed down"; (3) speech or thinking seems slowed down. Two additional items were added. It felt easy to do my daily tasks (reversed scoring), and I felt difficulty in doing my usual activities. Maria responded on 5-point Likert scale ranging from 1 (*not at all*) to 5 (*very much*). Her response indicated how much each statement was true for her in the last week (day for mid-treatment).

Action Simulation

Two types of action simulations were measured: (1) spontaneous action simulations and (2) object-related action simulations. For spontaneous action simulations, Maria had to rate 3 items on two dimensions: how frequently she experienced that simulation and how much she had kinaesthetic sensations associated with each item. The items were: (1) I had images of me doing things I have to do next days; (2) When I thought of what I will do, I had feelings of movement as I would have if I was doing that action; (3) I had images in my mind of how I would do an action right before doing that action. Two items measured simulations in response to objects: (1) When I saw an object/clothes/shoe, I also saw

in my mind how I use them; and (2) When I saw an object/tools/clothes/shoe, I felt in my body as I would have used it. Maria also responded to additional items regarding the mood in response to thoughts and coping (I have felt my mood changing just thinking of how I would do some activities; When something was bad, I could see in my mind "the movie" of how I can change the things to be better). The last item measured the use of internal or external perspective.

Vividness and Difficulty of Action Simulation

Maria had to imagine three actions in neutral, positive, and negative situations. Then she had to rate for each image, the easiness of simulation and the vividness of kinaesthetic sensations. For vividness, the scale was a 7-point Likert scale ranging from 1 (*not at all*) to 7 (*very vivid*). For difficulty, the scale was a 7-point Likert scale ranging from 1 (*not at all*) to 7 (*very difficult*).

Case Conceptualisation

According to the embodied model of distorted action cognition there are several mechanisms that may maintain the deficits in action cognition in depressed/formerly depressed patients. First, Maria demonstrated difficulty in mental simulation of actions. Maria reported that she did not simulate actions before doing them and she did not have the feelings of movement when she imagines actions (difficulties to represent kinaesthetically action simulations). She also found it very difficult to imagine new actions and felt almost no kinaesthetic sensations when she imagined actions in negative and positive situations. Efficient mental simulations of actions are thought to help us build a sense of efficacy about what we can do and what will follow in everyday situations. For instance, when seeing a cup full of tea before reaching the cup to drink, our mind automatically runs simulations of what would be to reach the cup, drink and taste the tea. When seeing an object such as an opened window, our mind unconsciously simulates possible behaviours for closing the window. When we evaluate the probability of achieving something important we rely on feelings about the easiness of simulating performing that action. Furthermore, the easiness (fluency) of action simulations is also involved in affective evaluations. People judge as more positive the objects and the situations when they can easily act on those objects (Hayes et al., 2008).

Because Maria was deficient in recruiting mental simulations of action in thinking, she probably also developed a sense of low efficacy (she had a low level of self-efficacy), losing the trust in her ability to solve problems and control the external world. She also developed a reduced level of action. Furthermore, she perceived as less positive activities and interactions in daily situations (she reported that she experienced little pleasure in what she was doing). In turn, these changes

might further result in high anhedonia and low activity levels maintaining vulnerability to depression.

An essential condition for proper use of motor simulations is good functioning of our body, of the motor and sensorimotor brain areas and attention.

There are several pathways through which Maria may have developed a deficit of action simulations. Probably, Maria's previous depressive episodes and associated neuro-hormonal changes (in the dopaminergic pathways, depending on her biological vulnerability) affected the proper functioning of the motor and related executive systems providing the structural base for action simulations. This may have resulted in deficiencies in recruiting motor-related areas in cognitive processing. Due to deficiencies in recruiting motor-related areas, she probably developed difficulties of (automatically and voluntarily) simulating possible movements and actions in response to situations or in the content of her thinking, especially in difficult situations.

These deficiencies in recruiting action simulations in thinking are probably to result longitudinally in several cognitive accommodations that may further maintain the deficit. For instance, due to difficulties in recruiting motor resources in thinking, Maria may have developed a view of self as weak and incapacitated with little chance to obtain what is important for her. She may also accommodate, by using predominantly visual representations to imagine actions, over-general thinking, slumped postures, habits of quick disengagement from difficult situations, ruminating about problems and so on. Furthermore, deficient action simulations may result also in low experiences of positive affect related to situations and actions (anhedonia). In turn, these accommodations may further maintain the deficit in action simulation (in a vicious cycle).

A second pathway to the deficit in action simulations was derived from current negative cognitive and mood states. Maria's negative self-focused thinking in response to problem situations ("it is something wrong with me when I cannot find a parking place") may have exacerbated the deficit in action imagery by focusing on her lack of worth instead of engaging action simulations in thinking about overcoming obstacles. Similarly, bouts of dysphoric mood, fatigue, not doing the preparatory parts of activities, and associated slumped posture acted as non-fluent action conditions that blocked the action simulations. Also, Maria described her thinking as lacking specific details which constitutes another pathway through which the patient acquired deficits in action cognition. Not being able to think of specific details of a situation accentuates the deficit of action simulation (Williams et al., 1996), specific simulations of actions requiring specific details in our mind so as to know what action to simulate.

MIKE training aimed to rehabilitate and compensate for action simulation deficits. Through Kinect, we aimed to help Maria boost the activity in the motor and related brain regions so motor representations can be recruited more easily in action-related thinking. We helped Maria (through Kinect) to associate action simulations to out-of-reach objects generating extended affordances. By inducing repeated

practice and memories of actions we helped Maria make action simulations more readily available in memory. By prompting thinking through and including details of future states we supported the simulation of specific actions and fostered prospective affordances. Furthermore, we taught Maria to use movement congruent postures to optimize the current affordances (enhanced affordances). Also, we trained Maria to build habits of using action simulations in recognition of objects (gerundival perceptions; Lambie & Marcel, 2002) thinking and action preparation. Furthermore, we compensated for the action simulation deficit by developing habits of using „online" supports for action simulations such as action language, action memories, and partial imitation to more efficiently recruit action simulations in thinking and action preparation.

Description of the Program

In session 1 (90 minutes) the MIKE program and the session agenda were introduced. We explained to the patient the rationale of the programme, emphasizing the development of the habit of using supports (language, gestures, posture, specific thinking) in order to balance back the action in our thinking. Then, Maria began Kinect practice for 20 minutes. We used a Microsoft Xbox Kinect 360, and a TV output Samsung 100 cm, 40MU6102, 4K Ultra HD screen for the psychologist-assisted sessions. Maria chose bowling (Sports Kinect game) as a practice game. During Kinect exercise, Maria was prompted to rate the level of the kinaesthetic sensations (from 0 to 10) before overtly simulating the movements. After finishing the Kinect practice, Maria was advised that in three sessions she will learn to use Action Simulation Balancing (ASB) Skill in three situations: to perceptions, to obstacles and to disengagement from difficult situations/useless actions. Further, she was shown how to practice the skill: the applied practice, spontaneous practice and the sustained practice exercise.

A skill training format (Miltenberger, 2004) was used to introduce the ASB skill. The psychologist described the ASB skill and demonstrated it using the example of an open window. Then Maria was asked to apply the skill to the open door situation. Feedback was given. The importance of practice was then discussed and applied, and generalization practices of the ASB skill were introduced. The applied practice was explained and demonstrated. The patient was asked to list several situations for applied practice and to illustrate how she would use the applied practice for three objects in the room. In the Applied Training, Maria was asked to practice each action balancing training to 3-5 objects from 3 scenes daily. The spontaneous practice was further explained ("Imagine you are in a real-life Kinect and you practice ASB for an object you see"). Maria was advised to use spontaneous practice whenever she wanted/opportunity arose. Next, the focused exercise was introduced in order to maintain the practice of the ASB skill. Maria was asked to

exercise daily after Kinect. We explained that the exercise aims to strengthen the ASB through practicing dynamic simulation. Then, we presented and demonstrated the steps of the exercise. The steps were (1) Choose a situation and 3-5 objects for practice (2) Count, look at the object, name the action by which to recognize the object (recognize-by-action), get into the stance and use dynamic simulation. Go through each object, give all your attention to the object and simulate the action, (3) Count, go through each object, recognize by action, use a dynamic simulation and repeat two times covert simulation for each object. (4) Count, go rapidly through each object, give all your attention, recognize by action and do a quick covert action simulation. In the second part of the session, we explained to the patient the Intention ASB skill. We explained that the deficit in action simulation following a depressive episode may also extend to our intentions and the exercise aims to address this ("Depression leaves scars on our mind. One such scar is, as in the case of perceptions, that our intentions are no longer balanced with action simulations. Usually, before performing an action, we automatically simulate the action we are about to do. We balance intentions with action simulations. After depression, this balancing is affected. In the following exercise, we relearn to balance our intentions with actions. We will simulate actions before doing them, memorize them and then simulate them to remember next time how we would do those actions"). Then, we demonstrated the exercise, choosing an "Action of the day" simulated before, memorizing while doing the action and re-simulating it for the future. Then the Action of the day log was introduced. Maria was instructed to describe the action of the day in the log and to imagine the action in a neutral and a positive situation. For the next 5 days, Maria practiced daily at home (from Monday to Friday) 15 minutes of Kinect training and then the ASB exercises. The Kinect device was installed at home.

Session 2 began with reviewing the mood and the forms from the previous week. Then the agenda was introduced: (1) learning corrective ASB and (2) review the Kinect practice. The aim of session 2 was to practice the ASB in response to obstacles as a corrective action simulation. The negative and self-focused thinking was introduced as a blocking factor to action simulation in thinking of obstacles. [We explained to the patient that "Most of the success of our actions depends on the adjustment of movements to the feedback from the environment or to its consequences; in other words, our actions require fine tuning to succeed. After depression, when the action does not result in desired consequences we give up on tuning action/or generate corrective action and we evaluate our worth instead. This depressive habit is blocking the action simulation in negative situations. The action balancing skill breaks this depressive habit – in each difficult situation we will balance action to attune it and block the focus on self. It is important to focus on what and how we can do, the consequences of our corrective action and to let go of negative thinking. If negative thoughts pop in just observe them, let them go and bring your attention to action simulations. Stress and negative mood feed on lack of action simulation from our perceptions to grow into depression and hopelessness. By this skill, we stop future stress and negative mood from growing into depression and

thus we become more resilient to further depression." Then, the steps of corrective ASB skill were described [For example, by looking at the mug from an out of reach position, we simulate the correction of the position, the new position and then the action (grab the mug) and its consequences. Steps: recognize as a to-be-adjusted/corrected situation, name the corrective action, simulate the new position of the object/body, get in the stance and then simulate the action and its consequences. It is important to focus on the action and let negative thinking go. If negative thoughts pop in, just observe them, let them go and bring your attention to action simulations]. Maria was asked to practice the scenario of one forgetting to lock the door and then feedback was provided. At the end of the session, the patient was asked to make a list of common obstacles during the day (daily obstacles) when she can use the corrective ASB, utilize the corrective ASB in applied training and in generalization training. She was also prompted to use the corrective ASB in Action of the day – i.e., to imagine the action after overcoming an obstacle without engaging in negative thoughts. She was prompted to use the corrective ASB in the Kinect practice. For the next 5 days, Maria continued the daily practice of Kinect at home. This week Maria had to practice the focused exercise using pragmatic ASB skills. Additionally, she practiced the previously applied ASB exercises.

Similarly to the previous session, Session 3 began by reviewing the mood and the forms. Then the agenda was introduced: (1) learning the pragmatic action simulation balancing skills (pragmatic ASB) and (2) reviewing the Kinect practice. The aim of session 3 was to practice balancing action simulations in response to situations that cannot be changed as a simulation of an alternative useful action. We explained to the patient that ["Depression diminishes our ability to generate simulations of useful actions (and adaptive problem solving and disengagement) in negative situations and prompts the tendency to react with self-criticism instead. By pragmatic ASB skill, we develop the habit of simulating useful action in response to negative mood/situations instead of criticism"]. Then, we explained to the patient the pragmatic ASB skill. Pragmatic ASB involves balancing appraisals of negative situations we cannot change/negative mood ("useless actions") with simulations of actions that are useful for us in that situation [for other desires], more specifically reacting to negative situations with pragmatic action instead of negative thinking. Basically, it involves imagining what else that is useful to me I can do instead of something I perceive as unachievable – i.e., pragmatic action simulation balancing. For instance, if I spill my coffee, I am imagining what I can do in this situation that is useful. The steps are: naming the negative situation, recognising it as a "to disengage" situation, identifying something useful to do, getting into stance and simulating the useful action and its consequences, then engaging in the useful action. In response to negative mood, this skill answers the question: what can I do usefully despite the fact I feel bad/did not do what I wanted in the first place and how can I do it. This is a very useful skill for becoming more resilient after depression. It is important to focus on a useful action and its consequences and to let go of negative

thinking. If negative thoughts recur just observe them, let them go and bring your attention to action simulations.

Similarly to the previous session, Maria was asked to practice the skill to not be able to lock the door (she was asked to imagine she was not able to open the door) and then feedback was provided. At the end of the session, the patient was prompted to make a list of common situations to disengage from during the day (daily obstacles) when she can use pragmatic ASB, insert the pragmatic ASB in Applied Training and in Generalisation Training. Also, she was prompted to insert in Action of the Day imagining the action after negative mood.

In Session 4, Maria undertook the assessment and an interview focused on the acceptability of the treatment. The patient engaged well with the treatment and was able to achieve most of her daily exercises.

Results

Clinical scores were described in Table 1. We hypothesized that Mike training will result in reduction of psychomotor retardation. As expected, after MIKE training, Maria experienced important improvements in her speed of processing (TMT-A) and cognitive flexibility (TMT-B) compared to pre-treatment. Maria showed a reduction in the processing time at TMT-A from 30 seconds to 21 seconds. Pre-treatment, Maria completed the TMT-A in 30 seconds. This result situated Maria (below the scores obtained by depressed patients at 6-8 weeks after antidepressant treatment (M= 40.3, SD = 36.8, Gorwood et al., 2014) more closely to the scores obtained by healthy people in the same age category (M = 28.5, SD =10, Tombaugh, 2004). Although she showed virtually no deficits in processing speed at pre-treatment, after Kinect, Maria needed less time to complete the task. Since the change is more than ½ SD (9 > 5) this change may be considered a minimally important difference. Maria showed an important reduction in the processing time at TMT-B from 149 seconds to 45 seconds. At pre-treatment, Maria completed the TMT-B in 149 seconds (2 mistakes) which indicates a severe deficit above that of formerly depressed (M = 66.3, SD = 58.7) and even above the score of currently depressed individuals (M = 96.8, SD = 77.3, 2.16 mistakes). Post-treatment, Maria completed TMT-B in 45 seconds (no mistake). This score situates Maria in the lowest end of the performance obtained by same age healthy individuals (45< 58.4, SD = 16.4; Tombaugh, 2004) the change being a minimally important difference and indicating a large effect size of the intervention (effect size MBLR = 69.7%).

We hypothesized that MIKE training will reduce depression. No meaningful difference between pre and post-treatment was seen in depression. Although (one week after the end of the treatment) the depression level was reduced after treatment compared to pre-treatment, both the pre (score 5) and post-treatment (score 1) scores situated Maria's level of depression in the no depression interval. Yet the reduction

in depression level corresponded to a large effect size (MBLR = 80%). Also, a small improvement was observed in scores obtained at activation subscale of BADS-SF. Both the pre-treatment and posttreatment scores at BADS (Table 1) situated the level of behavioural activation of Maria in an interval comparable with scores obtained by healthy individuals (activation $M = 13$, $SD = 5.9$; avoidance $M = 7.5$, $SD = 4$; Shudo & Yamamoto, 2017). No meaningful difference between pre and post-treatment was seen in rumination (Table 1). Both pre and post-treatment levels of hopelessness (low level), anxiety (mild anxiety), and rumination (low level) were in the normal non-clinical range (Table 1). Pre-treatment, Maria scored 10 at the brooding scale of RSS. A score of 10 placed Maria`s rumination in the lower interval of negative rumination, people diagnosed with depressive disorder scoring on average around values of 13.2 ($SD = 3.6$; Parola et al., 2017). Maria scored 5 (4 without the last item) at reflection scale. A score of 4 placed the rumination level in the lower interval of reflection, people diagnosed with depressive disorder scoring on average around values of 10.7 ($SD = 2.7$) for reflection (Parola et al., 2017).

Table 1. Scores on Clinical Scales at Pre and Post-Treatment

Time	TMT A	TMT B	QUIDS	GAD 7	BADS Activ	BADS Avoid	HS	RSS-B	RSS-R
Pre	30	149	5	7	12	10	2	10	5
Post	21	45	1	8	15	11	1.5	10	7

Note. Scores for clinical scales at pre and post treatment. TMT-A= Trail Making Test A. TMT-B= Trail Making Test B.QUIDS=16 items Quick Inventory of Depressive Symptomatology. GAD-7= Generalized Anxiety Disorder Scale. BADS Activ = the activation scale of the Behavioural Activation for Depression Scale. BADS Av= the avoidance subscale of the Behavioural Activation for Depression Scale. HS= mean score of the Kuopio Ischemic Heart Disease Hopelessness Scale. RSS-B=The brooding subscale of The Ruminative Response Styles Questionnaire RSS-R = the Reflection subscale of the Ruminative Response Styles Questionnaire.

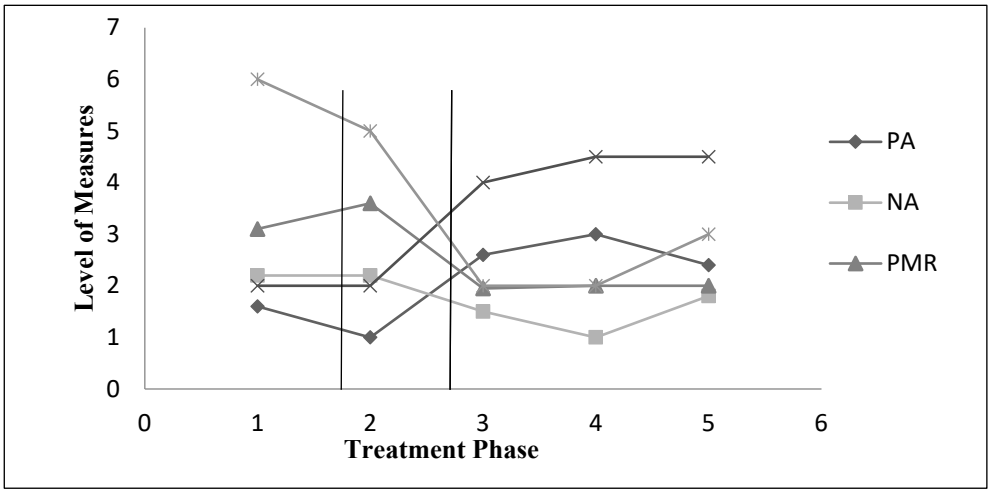
In Figure 1, we described the results from 2 weekly pre-treatment assessments, a mid-treatment assessment (that was an aggregate score composed by the mean of daily assessment of each variable-last week of treatment), one post-treatment (evaluation of the last week after the end of the MIKE) and one follow up (at 1 month after treatment). Maria endorsed the level for each variable on a rating scale where 1 means not at all, 2 little, 3 medium, 4 much and 5 very much (except for the kinaesthetic sensation for which the scale was from 1(no sensation) to 7 (as real sensations). No important differences were observed between the two pre-treatment assessments. Similarly, no important differences were observed between mid-treatment, post-treatment and follow up (see Figure 1). When analysing the pre-treatment with post-treatment/follow up there were several notable differences (Figure 1).

We hypothesized that after MIKE training Maria will have less psychomotor retardation on repeated measures. As expected, the results indicated that after MIKE treatment, Maria experienced a reduction in the psychomotor retardation at

behavioural level (decreasing from a medium level to a little level of psychomotor retardation). Consistently, when asked to rate how slow and slow-down she was, she rated that she was average at pre-treatment and little at mid and post-treatment, change corresponding to a medium effect size (effect size MBLR = 50 %).

We also hypothesized that Mike training results in improvements in action simulations. As expected, Maria improved her level of action simulations (except in automatic simulations to objects and imagery perspective). Although Maria improved in all dimensions of motor imagery, an important reduction was in the difficulty with which she imagined doing actions (from very difficult to little, medium effect size, MBLR = 54 %). Similarly, effects were observed regarding the vividness of kinaesthetic sensations when she was asked to imagine actions in positive situations (from 2 at pre-treatment to 4 at post-treatment, large effect size, MBLR = 100 %).

Additional analyses showed that Maria endorsed experiencing significantly more positive affect (rising from not at all-little positive affect at pre-treatment to average level at post-treatment) (large effect size, MBLR = 107 %). Consistently, when she was asked "*How much she feels pleasure in what she does*'' she responded with little at pre-test and average at mid and post-treatment. Maria also experienced less negative affect at post-treatment compared to pre-treatment (decreasing from a little to not at all) (small effect size, MBLR = 36.3 %).



Note. PMR-psychomotor retardation; PA-positive affect; NA-negative affect; KI= kinaesthetic imagery. The scores for the main variables on a Likert Scale from 1 (not at all), 2 (little), 3 (medium), 4 (much), to 5 (very much). For the vividness and the difficulty of the imagery the rating scale was from 1 (not at all) to 7 (very vivid/difficult). At the horizontal axis, 1=Pre-Treatment 1; 2=Pre-Treatment 2; 3=Mid-Treatment; 4=Post-Treatment; 5=Follow-up.

Figure 1. Changes in the Main Variables as a Function of the Treatment Phase

Complicating Factors

The primary complicating factor was fatigue. Maria had to work 10 hr each day and had many problems at work. Secondary complicating factors were the high working load and the relationship strain. During the treatment, Maria's ex-husband tried to come back and they had some arguments. On that day she did not practice the exercises. However, treatment was robust and positive outcomes remained. Maria was contacted via telephone by A.T for a welfare check. Support was offered but Maria handled the situation well and did not want to interrupt the training. Thirdly, Maria left the country for 4 days after week 2 of Kinect and she did not practice the exercises during that period. Although Maria interrupted the training she continued on return and the results were maintained.

Discussion and Treatment Implications

In this case study, we have illustrated the application of a new cognitive rehabilitation intervention for residual symptoms (motor imagery deficits) in a formerly depressed patient. As opposed to computerized cognitive training (targeting executive function or memory) and physical exercise approaches (Morimoto et al., 2014; Porter et al., 2013; Sun et al., 2017), we targeted deficits in motor imagery by promoting the use of motor resources in daily thinking of a formerly depressed patient. This intervention was new in using remote kinematic technology (Kinect) and skills training to rehabilitate motor cognition. We reported several main findings after administration of Kinect-based rehabilitation training in a formerly depressed patient: (1) an improvement in motor imagery (Maria endorsed higher feelings of vividness of kinaesthetic sensations during action simulations and she found it easier to imagine actions), (2) an improvement in cognitive (TMT-B) and behavioural aspects of psychomotor retardation, (3) an improvement in positive affect, and (4) reports of involuntary action simulations to distant stimuli (extended affordances). Furthermore, the training was well-accepted. Maria thought that the training was an entertaining way to do physical exercises and that the training helped her to get in physical and mental shape.

Firstly, we found that after MIKE Maria found it easier to imagine actions and felt more strongly the kinaesthetic sensations (when she imagined actions) than she did before the MIKE training. One possible explanation for this result was that MIKE training had improved her motor imagery. Thus, Maria imagined kinaesthetic sensations during actions more easily/vividly because MIKE training increased the accessibility of motor representational resources. This result was in line with studies that found that Kinect sessions improved motor imagery in healthy (Wriessnegger et al., 2014) and neurologically disordered (i.e., stroke, Parkinson disorder, cerebral

palsy, and multiple sclerosis) individuals (Da Gama et al., 2015; Mousavi-Hondori & Khademi, 2014; Webster & Celik, 2014). Here, we saw a similar improvement in kinaesthetic imagery after Kinect training in a formerly depressed individual. This result was also consistent with studies which found that repeatedly imagining positive events results in an increase in the vividness of visual imagery (Holmes et al., 2016). If this finding is extrapolated to other persons by subsequent research, Kinect-based training may function as a treatment for residual deficits in motor cognition after MDD. Secondly, Maria's scores at TMT test (form A and B) suggest improvements in the cognitive aspects of psychomotor retardation (processing speed and task switching). The improvement in processing speed may be explained by the rehabilitation effects of Kinect exercises but also by the training in speeding the simulation of actions in response to objects (the focused exercise has a speeding of simulation component). In the third week of the training, Maria also practiced simulation of disengagement from useless actions and engagement in useful actions. This skill might explain the improvement in TMT-B time which involves a set-switching executive process (Arbuthnott & Frank, 2000).

Cognitive deficits (executive control, processing speed) have been consistently linked to depression (McDermott & Ebmeier, 2009; Rock et al., 2014), persisting after treatment as residual symptoms in recurrent depressive disorder (Scult et al., 2017). Furthermore, the antidepressant treatment had limited effect on cognitive deficits improving mainly executive functioning (Austin maze task that involves visuospatial memory) and cognitive flexibility (Stroop task) but no other cognitive deficits (attention, response inhibition, verbal memory, decision speed, and information processing; Shilyansky et al., 2016). Previous, studies found an improvement in cognitive control (executive attention) after physical activity in MDD (Vasques et al., 2011). Yet recent meta-analyses found no effect of physical exercise on cognitive control functions (Sun et al., 2017). Instead, small effects were found for physical exercise interventions with added cognitive activities (Brondino et al., 2017). Thus, our finding was in line with studies that employed physical activity plus cognitive activity (Brondino et al., 2017). Previous findings suggested that when they do, physical exercises improve cognitive control due to changes in the plasticity of prefrontal cortex and the involvements of task-specific processes (Voelcker-Rehageet al., 2011). Thus, one explanation for this improvement in cognitive functioning is that Kinect may involve a similar plasticity mechanism. Research studies may investigate whether this finding can be generalized to other patients and whether Kinect training alone has the same effect. If so, Kinect may be an appealing way to provide physical activity in depressed patients. Since Kinect is a friendly, safe and socially-prone (can be done together with other persons) way to provide physical activity to depressed individuals, this observation warrants further research.

The improvement in cognitive control may also be explained by the reduction in physical aspects of motor retardation. Maria experienced a reduction in behavioural aspects of psychomotor retardation (from medium to little) after Kinect-

based training. Cognitive deficits have been previously linked to motor retardation. Moreover, studies linked cognitive slowing in motor imagery to sensory or motor deficits (Zarrinpar et al., 2006). Thus, it was possible that this reduction in the motor component of psychomotor retardation explained why we also observed an improvement in cognitive control. Yet, Maria showed increased levels of fatigue both pre and post treatment which may question this explanation. It was possible that Maria's fatigue was normal in the context of the prolonged time she spent at work and the increase in job demands (Maria linked her fatigue to increased demands at the job). Since fatigue is a common residual symptom in depression, which is present even in patients who responded to antidepressant treatment (Nierenberg et al., 1999), Maria was monitored to check whether her fatigue was linked to high job demands or persisted even with low work demands.

Nonetheless, this finding, if replicated by research studies, has significant treatment implications. Rehabilitative cognitive treatments targeting cognitive deficits, mostly in computerized format, have been developed for depression (Morimoto et al., 2014; Porter et al., 2013). However, their effects are mixed, small and limited in generalizability to other cognitive tasks and day-to-day functioning. These limits impose constraints on their applicability (Motter et al., 2016). Targeting rehabilitative efforts to intermediate cognitive markers (i.e., motor imagery, the speed of motor simulations) that support higher action cognition and adaptive behaviour (along with prompting generalization to day-to-day functioning) may overcome these limits. Thus, Mike training may be a skill-based alternative of cognitive rehabilitation treatment for intermediate cognitive endo-phenotypes. Maria endorsed experiencing more positive affect in the week after MIKE training. Although we did not expect this result, this finding was consistent with studies that showed that increasing motor fluency (the ease of mentally simulating acting upon an object) towards objects through imagery increased positive emotions to those objects (de la Fuente et al., 2017; Dennehy et al., 2012). According to this idea, targeting motor fluency would recover positive affect. Maria reported an increase in "feelings of fluency" during imagined actions (reduced difficulty of imagining) and the experience of kinaesthetic sensations after Kinect-based training. Some preliminary studies suggested that this motor-related affect is constrained by using kinaesthetic (not visuomotor) imagery (Hayes et al., 2013), individuals who imagine more vividly the kinaesthetic sensations rating more positively the objects they acted on (motor-related affect, Alhashil, 2016). Furthermore, the effect of fluency on affect has been observed only when people reactivate motor information in memory and not for people who have a low ability to do so (Vrana & van den Bergh, 1995).Thus, it was possible that our training involved an imagery-based increase in motor fluency as a mechanism of increasing positive affect in a formerly depressed individual. We also asked Maria to imagine an 'Action of the day' each evening in a positive and another specific (neutral, challenging or disengaging) situation. As such, it was possible that the increase in positive affect resulted also from imagining actions in positive situations.

Notably, after Kinect training, Maria reported involuntary simulations of actions in response to natural stimuli (seeing a door triggered involuntary partial movements of opening doors). Since Maria reported she never had before these manifestations, one possibility was that these simulations were a result of Kinect-based training. This effect may be explained by the fact that by repeatedly reinforcing overt simulations with perceptual consequences (in Kinect) we also extended the affordances of objects. As a consequence, simulations of actions were triggered by remote perceptual stimuli. This finding points to the possibility of a new mechanism that may be used in behavioural interventions. If this mechanism is replicated in research studies it would be a mechanism that would benefit behavioural activation. Thus, clinicians could research whether there are benefits by adding Kinect-augmentation short sessions to behavioural activation in the treatment of depression or to build augmented reality-driven behavioural activation interventions.

Furthermore, the rehabilitation of motor imagery may foster higher action cognition besides the increase in behavioural initiation. These findings are in line with the embodied model of higher cognition (Glenberg, 2010). Accordingly, because the training increased the accessibility of motor resources for thinking and/or the feelings of fluency for actions (Chambon & Haggard, 2012; Wenke et al., 2010) Maria estimated more control and being more efficient in solving problems. This finding raises the intriguing possibility that by increasing the accessibility of motor simulation, patients perceive themselves as more efficacious and in control. Thus, Kinect training may serve as a pre-treatment "booster" for cognitive restructuring and problems solving interventions.

Perhaps the most important implication of this case study was that it provides clinicians with an illustration of a skill-based cognitive rehabilitation treatment targeting an intermediate cognitive endophenotype (deficits in motor imagery) for a patient with residual symptoms after antidepressant treatment. The protocol utilized in this case stretches the traditional cognitive rehabilitation methods in depression. Instead of using a computerized exercise format, we used a skill training format that focused on the generalization of the trained cognitive skills into daily functioning of the patient. The treatment format used in this case is also promising for the (1) use as an adjunct treatment to CBT and antidepressant medication in patient with psychomotor retardation; (2) use as maintenance therapy to prevent recurrences of depression; (3) use as an endophenotype-targeted component treatment for deficit in action cognition in patients who show this deficit in various disorders not limited to MDD, and (4) use as a "pre-treatment boost" for cognitive restructuring, problem-solving or behavioural activation. Remote kinetics is an appealing intervention for individuals with friendly attitudes to technology yet people who are not open to technology will most likely refuse this method. Should the method be applied, attention is required to (1) problems with kinetic apparatus and good preparation, (2) positive interpretation and sustaining adaptive responses to failure (e.g., missing a strike in a bowling match) during the games, and (3) physical limitations of the patients and their level of fatigue.

Several limitations should be considered. Being a single-case study, these findings should be interpreted as illustrative and may not be generalizable to other patients. Another limit was related to controlling the effects of the previous interventions: observed effects may be attributable to after effects of CBT procedures such as behavioural activation. Yet, pre-and post-measures of behavioural activation level show no significant changes in the activation level. Unfortunately having no behavioural activation control group no conclusion about this result may be drawn. Furthermore, the concurrent effect of the intervention on negative cognition may result in the observed benefits of the intervention. An important concern was regarding replicability of the results. The intervention included Kinect exposure along motor imagery rehearsal. Thus, we cannot separate the effects of the training from the effects of the Kinect sessions. It may be possible that the effects were a consequence of the Kinect exercises alone. Further research should address these shortcomings. The next step would be to use multiple baselines across participants' design, feasibility studies and should the intervention prove reliable and feasible then pilot randomized trials may be designed. Experiments using healthy and at risk participants should be carried out to investigate the effect of Kinect intervention alone to dissociate the effects of Kinect exposure and motor training on psychological functioning.

Nevertheless, these findings are valuable in that they provide new ideas for research in the treatment of residual symptoms after MDD.

Final Conclusions

In a single case-study we have illustrated an intervention based on an embodied conceptualisation of cognition in depression: motor rehabilitation of action cognition. We described methods that can change the action-related ingredients of cognition and showed that increasing action simulation results in benefits for improving residual manifestations of depression. Further research using more reliable experimental design is needed.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interests with respect to their authorship or the publication of this article.

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