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THE EFFECTS OF MINDFULNESS-BASED COGNITIVE THERAPY ON DEPRESSIVE GAIT PATTERNS

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Abstract

According to embodiment theories, the experience of emotional states affects somatovisceral and motoric systems, whereas the experience of bodily states affects methods by which emotional information is processed. In the light of the embodiment framework, we proposed that formerly depressed individuals with a high risk of depressive relapse would display deviations in the way they walk, which might then play a role in the escalating process of depressive relapse. Moreover, we proposed that training in mindful body awareness during mindfulness-based cognitive therapy (MBCT) might have a normalizing effect on gait patterns. Gait patterns of 23 formerly depressed outpatients were compared to those of 29 never-depressed control participants. Also, gait patterns of formerly depressed patients were measured before and after MBCT to assess changes in patterns. A Fourier-based description of walking data served as the basis for the analysis of gait parameters. Before MBCT, gaits of formerly depressed patients were characterized by reduced walking speed and reduced vertical movements of the upper body. After MBCT, walking speed and lateral swaying movements of the upper body were normalized, and a trend towards normalization of vertical head movements was observed. It was concluded that MBCT has a normalizing effect on gait patterns, thus displaying not only cognitive, but also "embodied" effects.

***Keywords**:* embodiment, gait, major depression, mindfulness, relapse prevention

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> …when the practitioner walks, he is aware, "I am walking." When he is standing, he is aware, "I am standing." When he is sitting, he is aware, "I am sitting." When he is lying down, he is aware, "I am lying down." In whatever position his body happens to be, he is aware of the position of his body. (Buddha, trans. 1974, Hanh)

Introduction[1]

Mindfulness-based cognitive therapy (MBCT; Segal, Williams & Teasdale, 2002) was developed as an intervention for relapse prevention in depression, and its effectiveness has been demonstrated in three randomized controlled trials. Two of these trials (Teasdale, Segal, Williams, Ridgeway, Soulsby, & Lau, 2000; Ma & Teasdale, 2004) compared rates of relapse/recurrence in major depressive disorder (MDD) following treatment as usual (TAU) versus TAU plus MBCT. TAU consisted of help from family doctors or other sources normally contacted by patients when encountering symptomatic deterioration or other difficulties. In both studies, considerable reductions of rates of relapse/recurrence in MDD were reported for patients with three or more previous episodes of MDD (reduction of 44% and 54 % respectively). Moreover, a recent study (Kuyken et al., 2008) found MBCT to be at least as effective in reducing relapse/recurrence in MDD as maintenance pharmacological treatment with antidepressant medication, which forms the 'gold standard' of evidence-based relapse prevention in MDD (NICE, 2004).

The group-based MBCT-program (Segal et al., 2002) consists of eight weekly sessions of approximately 2.5 hours in duration. MBCT combines intensive training in mindfulness with elements of cognitive behavioral therapy. Mindfulness is rooted in eastern meditation traditions and can be characterized as paying attention in a certain way: on purpose, in the present moment, and non-judgmentally (Kabat-Zinn, 1990). Training in mindfulness is thought to enable patients to recognize and disengage from mind states characterized by self-perpetuating patterns of ruminative, negative thought, which might increase risk of relapse. By learning to recognize that thoughts and feelings are *events in the mind* as opposed to self-evident truths or aspects of the self, patients may be able to prevent the depressive thought-affect cycle from escalating (Teasdale, Moore, Hayhurst, Pope, Williams, & Segal, 2002).

In addition to changing one's relationship to thoughts, another fundamental characteristic of MBCT (and other mindfulness-based approaches) is its focus on increasing awareness of information the body provides. The quotation at the beginning of this article illustrates this characteristic in a classic Buddhist text on developing awareness of one's body. Indeed, the main part of MBCT

[1] Parts of this work were published by Michalak, Heidenreich and Troje (2010) as a letter to the editor in the *Journal of Psychosomatic Research*.

sessions and homework assignments consists of practicing formal mindfulness exercises that intensively train bodily awareness. For example, in the *body scan* – the first formal mindfulness practice in MBCT - participants learn to sequentially attend to each section of the body. They begin with the toes of the left foot and move systematically through each body part, ending with the top of the head. Thus, the entire body is scanned with a mindful and non-judgmental curiosity. The second formal mindfulness practice in MBCT is *sitting meditation*, in which participants mindfully follow the bodily experience of the breath. These practices are designed not only to help patients redirect and anchor their attention when they notice it has shifted away into thinking or daydreaming, but also to assist patients in developing a heightened awareness of their body-states.

But what could be the benefits of developing a heightened awareness of the body? MBCT proposes that the self-perpetuating patterns of ruminative, negative modes of mind that often lead to relapse are not solely cognitive in nature. Instead they are characterized by complex configurations or patterns of negative mood, thoughts and *body sensations* (Segal et al, 2002, p. 67). This notion is supported by recent research emphasizing the close and reciprocal relationships between bodily, cognitive, and emotional processes. This research converges in the idea of the *embodied* nature of emotion (e.g., Damasio, 1994; Niedenthal, 2007; Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005). According to these embodiment theories, emotional states affect somatovisceral and motoric systems, whereas bodily states affects methods by which emotional information is processed. For example, the "Interacting Cognitive Subsystems" (ICS) approach (Teasdale, 1999, Teasdale & Barnard, 1993) proposes that sensory input (e.g., input from the body) makes a direct and important contribution to emotional states, which distinguishes these states from more intellectual and rational processes. It is only when cognitive information (e.g., thematic semantic context of a situation) is interlocked with sensory (e.g., proprioceptive) information that a person *feels* sad or hopeless instead of just thinking about sadness or hopelessness. In the so called "depressive interlock configuration" (Teasdale, 1999), established bodily and cognitive feedback loops can "lock" subsystems into a self-perpetuating configuration that can maintain depression or lead to depressive relapse.

In our present research, we proposed that the way people walk would convey proprioceptive information that might serve as bodily feedback in emotional processing and depression. A number of studies have investigated gait patterns of currently depressed patients, and results have provided empirical evidence for a close relationship between gait and depression/depressed mood. (Bader, Bühler, Endras, Klippstein, & Hell, 1999; Lemke, Wendorff, Mieth, Buhl, & Linnemann, 2000; Paleacu, Shutzman, Giladi, Herman, Simon, & Hausdorff, 2007; Sloman, Berridge, Homatidis, Hunter, & Duck, 1982; Sloman, Pierynowski, Berridge, Tupling, & Flowers, 1987). For example, in a comprehensive three-dimensional analysis of gait characteristics in patients

suffering from a current episode of MDD, five features most strongly differentiated gait of currently depressed from never-depressed participants (Michalak, Troje, Fischer, Vollmar, Heidenreich, & Schulte, in press). Depressed patients showed reduced walking speed, smaller arm-swing amplitudes, smaller amplitude of vertical movements of the upper body, larger amplitudes of lateral body sway, and a slumped, forward-leaning posture. Moreover, a parallel gait pattern emerged in a non-clinical sample when sad mood was induced via music mood induction procedure (an animation which visualizes the differences observed from the two data sets can be viewed at http://biomotionlab.ca/Demos/BMLdepression.html).

Taken together, studies on gait patterns of depressed individuals indicate that depression is embodied in the way people walk. They suggest a complex interaction between bodily and emotional states. These findings also support the notion that established bodily and cognitive feedback loops might "lock" subsystems into a self-perpetuating configuration that can maintain depression and lead to depressive relapse. However, the exact nature and causal mechanisms within these feedback loops have yet to be established.

The major aims of our study were twofold. First, we investigated whether individuals with a history of depressive episodes but no current depression would show gait patterns that resemble those of currently depressed patients. We assumed that deviant gait patterns might be a persistent marker of depression and thus would be apparent in both currently and formerly depressed individuals. In the light of the aforementioned embodiment framework, we proposed that even when formerly depressed individuals are not currently depressed, proprioceptive feedback of the motor system might be part of an escalating process of depressive relapse. Thus, feedback from deviant gait patterns might be one route that increases risk to relapse/recurrence in those with a history of MDD (Burcusa & Iacono, 2007).

The second aim of our study was to investigate whether MBCT changes gait patterns in formerly depressed patients. By means of mindfulness practice during MBCT, patients may increase the awareness of body states and the complex interaction of body states with cognitive and emotional processes. This heightened awareness might not only assist patients in recognizing and disengaging from negative cognitive states (as cognitive theories of mechanisms in MBCT postulate) but also in recognizing and disengaging from negative movement patterns which might play a role in the escalating process of depressive relapse. For example, when patients mindfully realize that a certain kind of gait pattern has deleterious effects on their mood and well-being, they might be able to change their gait to a pattern that is more beneficial. Thus, we expected that MBCT would normalize gait patterns of formerly depressed patients. Additionally, we investigated whether changes in gait characteristics during MBCT were associated with changes in levels of residual depressive symptoms and self-reports of mindfulness.

Method

Participants

Twenty-three formerly depressed patients (5 male and 18 female) participated in the study. They were recruited by media announcement. The average age of the patients was 47.1 years (*SD* = 10.3). Inclusion and exclusion criteria were as follows: a) 18 to 65 years of age; b) meeting *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV; American Psychiatric Association, 1994) criteria for a history of recurrent major depression (history of three or more previous episodes of major depression with no history of mania or hypomania; in addition, at least two episodes of major depression must have occurred within the past five years, with at least one episode within the past two years; [diagnostic criteria were derived using the German version of the Structured Clinical Interview for DSM-IV; SCID; Wittchen et al., 1997]); c) in recovery/remission at the time of baseline assessment and for at least the preceding 12 weeks; and d) a 17-item Hamilton Rating Scale for Depression (Hamilton, 1960) score of less than 10 at baseline assessment. Exclusion criteria were a) history of schizophrenia or schizoaffective disorder; b) current substance abuse, eating disorder, or obsessive-compulsive disorder; c) organic mental disorder, pervasive developmental delay, or borderline personality disorder; d) dysthymia before age 20; e) current psychotherapy or counseling more frequent than once per month; and f) current practice of meditation more than once per week or yoga more than twice per week. Ten out of 23 formerly depressed patients had comorbid diagnoses (most of them anxiety disorders).

Inclusion and exclusion criteria largely corresponded to those of the study by Teasdale et al. (2000). However, in contrast to the study of Teadale et al. (2000) where patients with two or more episodes were included we restricted our analyses to patients with three or more previous episodes of major depression[2] because the previous trials have shown that MBCT is especially effective for this subgroup of patients. Moreover, in contrast to Teasdale et al. (2000), we included patients taking antidepressant medication because continued pharmacotherapy is common for this highly vulnerable group of patients. Ten patients were taking antidepressant medication at baseline assessment. Of these, six were taking selective serotonin reuptake inhibitors (mainly citaprolam, N = 4), two were taking serotonin norardrinalin reuptake inhibitors, one was taking St-John's wort and one was taking lithium.

Patients meeting the inclusion criteria and willing to participate in the study received written study information and provided written informed consent. They were also informed that they could terminate the study any time.

[2] The sample of the present study was a subsample of the study of Michalak, Heidenreich, Meibert and Schulte (2008).

Additionally, 29 never-depressed participants (7 male and 22 female) were recruited through announcements and public notices. All control participants were screened using the SCID interview and reported no prior or current history of depression. Their average age was 46.3 years (*SD* = 7.4).

Motion data acquisition

Motion data were collected with an optical motion capture system (Vicon 512, Oxford Metrics, UK) equipped with nine CCD video cameras (Pulnix TM 6710) that made it possible to track three-dimensional trajectories of 41 small, reflective markers attached to the participants' body with a spatial accuracy in the range of 1 mm and a temporal resolution of 120 Hz (for more details see Troje, 2002). The motion-capture system was set up in a 90 m^2 room with black walls and ceiling and enough space for recording human motion. Participants walked on a 7 x 0.70 meter red rug, which was placed within the elliptic capture volume. They could choose their walking speed freely.

Motion analysis was based on a framework developed by Troje (2002, 2008). In brief, the data were subjected to a discrete Fourier transform which was then used to determine the amplitudes of a number of different body parts. Prior research on gait patterns of currently depressed individuals (Michalak et al., in press) has identified five features that most strongly differentiate depressed and healthy gait. The five features were: 1) walking speed, 2) arm swing amplitude (i.e. difference between extreme horizontal positions of the wrist, averaged across right and left side), 3) amplitude of lateral body sway (i.e., difference between maximum right and left deflection of the shoulder), 4) posture of the upper body (i.e., angle between the line connecting clavicle and head marker and the vertical axis, with a positive angle indicating that the head is in front of the clavicle marker), and 5) vertical up and down movements of the upper body (measured in terms of the vertical amplitude of the head marker).

In our present research, we investigated whether formerly depressed participants show deviations in these five features that resemble deviations of currently depressed individuals. Moreover, we investigated whether MBCT has a normalizing effect on these deviant features.

Treatment

The treatment protocol followed the MBCT manual, which was developed by Segal and colleagues (2002) and based on an integration of aspects of CBT for depression (Beck et al., 1979) with components of Mindfulness-Based Stress Reduction (Kabat-Zinn, 1990). The treatment protocol consisted of weekly 2.5-hour group-sessions over 8 consecutive weeks. Session content included guided mindfulness practices (body scan, sitting meditation, yoga), inquiry into patients' experience of these practices, review of weekly homework (40 minutes of mindfulness practice per day and generalization of material taught in sessions),

and teaching/discussion of cognitive-behavioural skills. Adequate dose of treatment was defined as attending at least 5 out of 8 sessions of MBCT.

Measures

Hamilton Rating Scale for Depression (HRSD). The baseline assessment interview included the 17-item HRSD (Hamilton, 1960; German version CIPS, 2004), a widely used interview-based measure of the severity of depressive symptomatology. The HRSD covers a range of affective, behavioral, and biological symptoms. It has good psychometric properties (Rabkin and Klein, 1987) with high interrater-reliabilitiy (r = 0.97; Baca-Garcia et al., 2001); it was administered by trained raters.

Beck Depression Inventory (BDI). The BDI (Beck and Steer, 1987; German version by Hautzinger et al., 1995) is a widely used, 21-item self-report measure that was utilized to assess the severity of affective, cognitive, motivational, behavioral and biological symptoms of depression. The BDI has good psychometric properties (Rabkin and Klein, 1987; Hautzinger et al., 1995). It was administered at baseline and after the termination of the eight-week MBCT course.

Mindful Attention and Awareness Scale (MAAS). Mindfulness was assessed with the German translation of the MAAS (Brown and Ryan, 2003; German translation by Michalak, Heidenreich, Ströhle and Nachtigall; 2008). The MAAS is a 15-item self-report instrument measuring the tendency to be attentive to and aware of present-moment experiences in daily life (e.g., "I find it difficult to stay focused on what's happening in the present"). It consists of a single factor which explains 95% of variance and thus yields a single total score. The German version of the MAAS has a single factor structure and good psychometric properties. Previous analysis has shown that MAAS-scores increase significantly during the MBCT program (Michalak, Heidenreich, Meibert, & Schulte, 2008).

Statistical Analyses

Statistical Analyses were conducted using SPSS 15.0. For between-group comparison, independent-samples t-tests were used; for within-subjects comparisons, dependent-samples t-tests were computed. Correlations were computed using the formula of Bravais-Pearson. Because our hypotheses were directional, we performed one-tailed tests unless otherwise stated.

Results

Of the 23 patients assessed at baseline, one dropped out of treatment, and two did not attend post-treatment assessment. Thus, pre-post analyses were conducted with the remaining 20 patients. All of the patients received an adequate dosage (at least five sessions) of treatment. Seven of these patients were taking

antidepressant medication; one discontinued medication during the course of MBCT.

Differences between formerly depressed and never-depressed participants

The formerly depressed patients and never-depressed control group did not differ significantly in age, gender distribution, or weight (two-tailed tests). However, an independent-samples t-test (unequal variances) revealed that formerly depressed participants were significantly more depressed than the never-depressed controls (BDI; formerly depressed: M = 12.21, SD = 8.61; never-depressed participants: M = 2.62, SD = 3.59, t[28.07] = 5.01, $p < .001$).

Means and standard errors of gait characteristics of formerly depressed and never-depressed individuals are presented in Figure 1. Significant differences were found between formerly depressed and never-depressed participants in speed and vertical up-and-down movements of head. Formerly depressed individuals walked more slowly (formerly depressed [FD]: M = 1.16 m/s, SD = 0.12; never-depressed participants [ND]: M = 1.23 m/s, SD = 0.14, t[50] = -2.05, $p < .05$) and showed reduced vertical movements as measured in terms of the vertical amplitude of the head (FD: M = 35.28 mm, SD = 8.80; ND: M = 40.08 mm, SD = 7.23, t[50] = -2.16, $p < .05$). In contrast, differences in arm swing (FD: M = 375.92 mm, SD = 86.10; ND: M = 370.20 mm, SD = 91.53, t[50] = 0.23, *ns*), posture (FD: M = -1.40 deg, SD = 1.06; ND: M = -1.61 deg, SD = 1.11, t[50] = 0.71, *ns*) and lateral body sway (FD: M = 36.53 deg, SD = 9.56; ND: M = 34.13 deg, SD = 10.58, t[50] = -2.16, *ns*) were small and non-significant.

Effect sizes (Cohen's d) of group difference in gait characteristics were d = 0.54 for speed and d = .60 for vertical head movements. Small effect sizes emerged for lateral body sway (d = 0.28), posture (d = 0.20), and arm swing (d = 0.06).

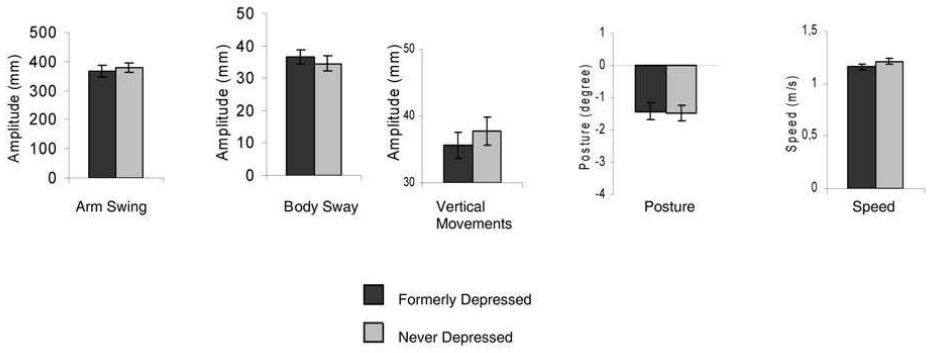

Figure 1. Gait parameters of formerly depressed (N = 23) and never-depressed participants (N = 29). Error bars represent one standard error.

Pre-post changes following MBCT

We conducted a dependent t-test to examine whether residual depressive symptoms decreased during MBCT. Compared to baseline levels, formerly depressed patients had significantly lower levels of residual symptoms after the eight week MBCT course (BDI; baseline: $M = 12.45$, $SD = 9.11$; post-treatment: $M = 7.60$, $SD = 7.54$, $t[19] = 3.19$, $p < .01$).

Dependent t-test revealed that post-treatment patients had normalized their walking speed (baseline: $M = 1.16$, $SD = 0.12$; post-treatment: $M = 1.21$, $SD = 0.13$, $t[19] = 2.64$, $p < .01$) and showed reduced lateral body sway (baseline: $M = 36.55$, $SD = 10.26$; post-treatment: $M = 34.46$, $SD = 10.37$, $t[19] = 3.39$, $p < .01$). The increase in vertical movements of the upper body showed a marginal significant trend (baseline: $M = 35.60$, $SD = 8.95$; post-treatment: $M = 37.72$, $SD = 9.20$, $t[19] = 1.52$, $p < .08$). Changes in arm swing (baseline: $M = 367.89$, $SD = 86.67$; post-treatment: $M = 377.74$, $SD = 74.16$, $t[19] = -0.90$, *ns*) and posture (baseline: $M = -1.42$, $SD = 1.14$; post-treatment: $M = -1.46$, $SD = 1.06$, $t[19] = 0.21$, *ns*) were small and non-significant. Figure 2 depicts pooled pre-post effect sizes of changes in gait parameters.

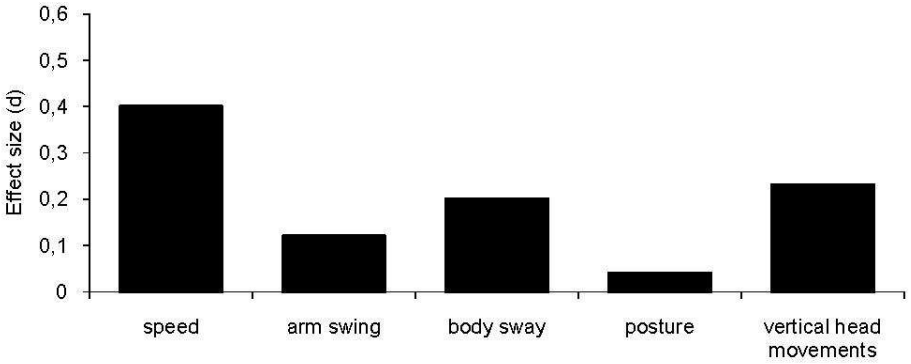

Figure 2. Effect sizes (d) for pre-post-changes of gait parameters during MBCT. Positive effect sizes indicate a change towards normalization.

To examine whether changes in gait characteristics were associated with changes in depression and mindfulness, we computed Pearson correlations between BDI and MAAS change scores and changes in gait characteristics. As can be seen in Table 1, most of the correlations were small in magnitude, and none were statistically significant.

Table 1. Correlations between changes in depression and mindfulness and changes in gait characteristics

Change in gait characteristics	Changes in BDI	Changes in MAAS
speed	.00	.30
arm swing	.10	.21
body sway	.03	.02
posture	.26	.27
vertical movements	.16	.13

Note. $N = 20$. BDI = Beck Depression Inventory, MAAS = Mindful Attention and Awareness Scale.
* $p < .05$.

Discussion and Conclusion

Our current study had two principal aims. First, we investigated whether patients with a history of depressive episodes but no current depression would show gait patterns that resembled those of currently depressed patients. Second, we investigated whether MBCT has a normalizing effect on deviant gait patterns of formerly depressed patients. Our results revealed that, compared to never-depressed individuals, patients with three-or-more previous episodes of depression walked more slowly and with reduced vertical movements of the upper body. Effect sizes for these differences were in the medium range (d's > 0.50 for speed and vertical head movements). Thus, formerly depressed individuals continued to show deviations in two of the five characteristics most strongly differentiating gait of currently and never-depressed individuals (Michalak at al., in press, Study 1). In other words, formerly depressed individuals show not only residual affective and cognitive symptoms (Burcusa, & Iacono, 2007), but also show residual psychomotoric symptoms, even after the end of an acute episode of MDD.

However, the exact nature of the interplay of the different types of residual symptoms has yet to be established. Previous research has shown that the induction of sad mood in normal individuals results in a gait pattern that parallels patterns found in currently depressed patients (Michalak et al., in press, Study 2). Whether or not the reverse directed mechanism is also operating (i.e., depressive gait pattern leads to depressive mood or cognition) is a topic that should be investigated in future research. Correspondingly, our notion that deviations in gait patterns might be a route that increases risk of relapse in those with a history of depression remains largely speculative. However, embodiment research in non-clinical samples has already shown that changes in simple movements and posture can indeed affect emotional processes (for a review see Niedenthal, 2007).

Moreover, future research should investigate whether the complex interaction between gait and depressive mood/cognition that is suggested by embodiment theories is relevant for relapse. Our present data only allows us to conclude that the gaits of formerly depressed patients show deviations. Larger samples and longitudinal designs are needed to draw stronger conclusions about the role the interaction between gait and mood/cognition might play in the process of depressive relapse.

An important question is whether or not changes in gait patterns can be explained fully by improved mood. One way to assess this would be to use an analysis of covariance (ANCOVA) to control depression levels when comparing gait patterns of formerly versus never-depressed individuals. However, when groups differ on a potential covariate, ANCOVA removes too much variance from the independent variable, so that the grouping variable is substantively altered in a way that is not meaningful or conceptual (Miller & Chapman, 2001). Although ANCOVA can be legitimately used as a technique to reduce noise (error) when groups do not differ in the covariate, attempts to statistically "control" substantial group differences are an invalid use of ANCOVA. Future research should analyze gait patterns of formerly depressed individuals free from residual affective symptoms to determine whether deviant gait patterns are present in individuals whose depressive symptoms have completely remitted.

Another question is what possible role antidepressant medication may have played in our results. As some of the formerly depressed patients were taking antidepressant medication, we cannot rule out the possibility that these medications might have affected our results. However, it should be noted that previous studies on antidepressant medication have found either no effects on gait parameters (Lemke et al, 2000) or small changes that were in the direction of the never-depressed gait pattern reported in our study (i.e., increased gait speed; Paleacu et al., 2007). Thus, it seems rather unlikely that the group differences found in our study are attributable to medication effects.

Besides analyzing gait patterns of formerly depressed individuals, the second focus of our study was to investigate whether MBCT has a normalizing effect on deviant gait patterns. Our results revealed that walking speed and lateral body sway showed significant pre-post-changes. While walking speed increased, lateral body sway was reduced. In addition, vertical head movements showed a marginally significant increase. All changes were in the direction of the healthy gait pattern. Even though the sizes of these changes were small, formerly depressed patients approximately halved the discrepancy between their performance and that of normal controls with regard to speed and vertical head movements. Moreover, it should be noted that our measure was nonreactive, thus making large effect sizes rather unlikely (Smith, Glass, & Miller, 1980).

The result for lateral body sway initially appears somewhat puzzling. Although only relatively small (d = .23) and non-significant differences were found between formerly and never-depressed participants in lateral body sway,

this parameter changed significantly during MBCT. However, one should be aware that the comparison between formerly and never-depressed individuals was based on between-subject data; there is substantial variance in body sway within normal populations. In contrast, the pre-post analysis was based on within-subject data with different handling of the variance. Thus, the seemingly conflicting results might be attributable to methodological factors. Moreover, in the study by Michalak and colleagues (in press), currently depressed patients showed pronounced lateral body sway ($d > .80$). Thus, a reduction in body sway seems to indicate a normalization of gait pattern.

The specific interrelationship between the different gait characteristics is not yet apparent. In the simplest case, parameters like body sway or reduced vertical movements might be caused by reduced walking speed. However, research on speed-dependent changes in gait parameters in non-clinical participants have shown that while amplitudes of vertical movements of the trunk increase when velocity increases (parallel to our comparison of formerly and never-depressed individuals), lateral swaying movements of the upper body increase when walking speed increases (Thorstensson, Nilsson , Carlson, & Zomlefer, 1984). The latter finding is in contrast to our finding that increases in speed were associated with decreases in lateral body sway.

A further notable result of our study was that changes in gait patterns were neither associated with changes in residual depressive symptoms nor with changes in self-reported mindfulness. The low correlations between changes in BDI-scores and changes in gait characteristics might indicate that changes in psychomotoric features and emotional/cognitive symptoms might have different trajectories. This would be comparable to asynchronous changes of different symptom clusters in other disorders (Cohen, 1978). The low correlations between changes in gait characteristics and changes in self-reported mindfulness were surprising for us. On one hand, this low association might indicate that our theory that changes in gait parameters are caused by increased levels of mindfulness is incorrect. On the other hand, self-report measures might not be optimally suited to assess mindfulness (Grossman, 2008). Of note is that the MAAS items do not tap *body awareness*, the facet of mindfulness that seems most important to our theoretical framework.

Further limitations of our study should be noted. First, the small sample size might have reduced the power to detect more subtle changes in gait characteristics. Moreover, because of the uncontrolled nature of our design, changes in gait patterns cannot be attributed unambiguously to the effect of MBCT. Finally, some ambiguities might be caused by the nature of the mindfulness exercises that are part of the MBCT course. Mindfulness exercises often involve a conscious 'slowing down' of movements to help participants bring mind and body in aware contact (e.g., eating a raisin slowly in the "raisin exercise" or walking slowly during walking meditation); thus, one might speculate that MBCT leads to a slower walking speed. However, as our data

show, participants walk *faster* after the MBCT course. Since the walking speed of patients after MBCT is nearly equivalent to the speed of never-depressed subjects, we consider it unlikely that MBCT artificially slowed the speed of patients.

Although keeping these methodological limitations in mind, we conclude that our study provides evidence that MBCT has some normalizing effects on gait patterns of formerly depressed individuals. This normalization might be part of the causal chain that helps patients to de-escalate mood/body vicious cycles that lead to depressive relapse. Future research will have to reveal the exact nature of such embodied effects of MBCT. We believe that analyzing these embodiment dimensions might foster a deeper understanding of mindfulness training.

REFERENCES

Association, A. P. (1994). *Diagnostic and statistical manual of mental disorders*. Washington: Author.

Baca-Garcia, E., Diaz-Satre, C., Basurte, E., Prieto, R., Ceverino, A., de Leon, J., Saiz-Ruiz, J. (2001). A paradox in studies of impulsivity in suicide attempts . *Journal of Clinical Psychiatry , 62*, 560-564.

Bader, J. P., Bühler, J., Endrass, J., Klipstein, A., Hell, D. (1999). Muskelkraft und Gangcharakteristika depressiver Menschen [Muscle power and gait characteristics of depressed individuals]. *Nervenarzt, 70*, 613-619.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, A. T., Steer, R. A. (1987). *Beck Depression Inventory - Manual.* San Antonio: The Psychological Corporation.

Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness and its role in psychological well-being. *Journal of Personality and Social Psychology, 84*, 822-848.

Burcusa, S. L., & Iacono, W. G. (2007). Risk for recurrence in depression. *Clinical Psychological Review, 27*, 959-985..

Damasio, A. R. (1994). *Decartes' error. Emotion, reason, and the human brain.* New York: Avon Books.

Grossman, P. (2008). On measuring mindfulness in psychosomatic and psychological research. *Journal of Psychosomatic Research, 64*, 405-408.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry, 23*, 56-62.

Hautzinger, M., Bailer, M., Worall, H., Keller, F., 1995. *Beck-Depressions-Inventar*. Bern: Hans Huber Verlag..

James, W. (1984). What is emotion? *Mind,19*, 188-205.

Kabat-Zinn, J. (1990). *Full catastrophe living: The program of the Stress Reduction Clinic at the University of Massachusetts Medical Center*. New York: Delta.

Kuyken, W., Byford, S., Taylor, R. S., Watkins, E., Holden, E., White, K., et al. (2008). Mindfulness-based Cognitive Therapy to Prevent Relapse in Recurrent epression. *Journal of Consulting and Clinical Psychology, 76*, 966-78.

Lemke, M. R., Wendorff, T., Mieth, B., Buhl, K., Linnemann, M. (2000). Spatiotemporal gait patterns during over ground locomotion in major depression compared with never depressed controls. *Journal of psychiatric research, 34*, 277-283.

Ma, S. H., Teasdale, J. D. (2004). Mindfulness-based cognitive therapy for depression: Replication and exploration of differential relapse prevention effects. *Journal of consulting and clinical psychology, 72*, 31-40.

Michalak, J., Heidenreich, T., Meibert, P., & Schulte, D. (2008). Mindfulness predicts relapse/recurrence in major depressive disorder following Mindfulness-based Cognitive Therapy. *Journal of Nervous and Mental Disease, 196*, 630-633.

Michalak, J., Heidenreich, T., Ströhle, G., & Nachtigall, C. (2008). Die deutsche Version der Mindful Attention and Awareness Scale (MAAS): Psychometrische Befunde zu einem Achtsamkeitsfragebogen. *Zeitschrift für Klinische Psychologie und Psychotherapie, 37*, 200-208.

Michalak, J. Troje, N., Fischer, J., Vollmar, P, Heidenreich, T. & Schulte, D. (2009). The embodiment of sadness and depression - gait patterns associated with dysphoric mood. *Psychosomatic Medicine.71*, 580–587.

Michalak, J., Troje, N. & Heidenreich, T. (2010). Embodied effects of mindfulness-based cognitive therapy. *Journal of Psychosomatic Research, 68*, 312-313.

Miller, G. A., Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of abnormal psychology, 110*, 40-48.

Hanh, N. T. (1974). *The Miracle of Mindfulness.* Boston: Beacon Press.

NICE (2004). *Management of depression in primary and secondary care*. Zugriff am 23.01.2009. Verfügbar unter: http://www.nice.org.uk/CG023NICEguideline

Niedenthal, P. M., Barsalou, L. W., Winkielman, P., Krauth-Gruber, S., Ric, R. (2005). Embodiment in attitudes, social perception, and emotion. *Personality and social psychology review : an official journal of the Society for Personality and Social Psychology, 9*, 184-211.

Niedenthal, P. M. (2007). Embodying Emotion. *Science, 316*, 1002-1005.

Paleacu, D., Shutzman, A., Giladi, N., Herman, T., Simon, E. S., Hausdorff, J. M. (2007). Effects of pharmacological therapy on gait and cognitive function in depressed patients. *Clinical neuropharmacology, 30*, 63-71.

Rabkin, J. G., Klein, D. F. (1987). The clinical measurement of depressive disorders. In: A. Marsella, R. Hirschfeld and M. Katz (Eds.), *The measurement of depression*. New York: Guilford Press, pp. 30-83.

Segal, Z. V., Williams, J. M. G., Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: a new approach to preventing relapse.* New York: Guilford Press.

Sloman, L., Berridge, M., Homatidis, S., Hunter, D., Duck, T. (1982). Gait patterns of depressed patients and normal subjects. *The American journal of psychiatry, 139*, 94-97.

Sloman, L., Pierrynowski, M., Berridge, M., Tupling, S., Flowers, J. (1987). Mood, depressive illness and gait patterns. *Canadian journal of psychiatry, 32*, 190-193.

Smith, M. L., Glass, G. V., Miller, T. I. (1980). *The benefits of psychotherapy.* Baltimore: John Hopkins University Press.

Teasdale, J. D., Barnard, P. J. (1993). *Affect, cognition and change: Remodelling depressive thought.* Hove: Lawrence Erlbaum Associates.

Teasdale, J. D. (1999). Multi-level theories of cognition-emotion relations. In: T. Dalgleish and M. Power (Eds.), *Handbook of cognition and emotion.* Chichester: Wiley, pp. 665-681.

Teasdale, J. D., Segal, Z. V., Williams, J. M. G., Ridgeway, V. A., Soulsby, J., Lau, M. A. (2000). Prevention of relapse/ recurrence in major depression by mindfulness-based cognitive therapy. *Journal of consulting and clinical psychology, 68,* 615-623.

Teasdale, J. D., Moore, R. G., Hayhurst, H., Pope, M., Williams, S., Segal, Z. V. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical evidence. *Journal of consulting and clinical psychology, 80,* 275-287.

Thorstensson, A., Nilsson, J., Carlson, H., Zomlefer, M. R. (1984). Trunk movements in human locomotion. *Acta physiologica Scandinavica, 121,* 9-22.

Troje, N. F. (2002). Decomposing biological motion: A framework for analysis and synthesis of human gait patterns. *Journal of vision, 2,* 371-387.

Troje, N. F. (2008). Retrieving information from human movement patterns. In: Shipley, T. F. and Zacks, J. M. (Eds.), *Understanding Events: How Humans See, Represent, and Act on Events.* New Yoirk: Oxford University Press, pp. 308-334.

Wittchen, H.-U., Wunderlich, U., Gruschwitz, S., Zaudig, M. (1997). *SKID I. Strukturiertes klinisches Interview für DSM-IV.* Göttingen: Hogrefe.