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ORIGINAL RESEARCH

Visuomotor Adaptation and Savings Are Preserved in Individuals With Depression

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Depression is a mental disorder associated primarily with mood and cognitive symptoms. Several studies show that depression is additionally accompanied by decrements in gross and fine motor functioning. The present study examined to what extent depression affects the ability to adapt sensorimotor behavior. A total of 20 participants with depression and 22 controls performed a manual adaptation task in which they had to adapt joystick movements to a visual perturbation. We assessed the rate of adaptation following the introduction of the visual perturbation (both for early and later stages of adaptation) and the rate of deadaptation following its removal. In addition, questionnaires were used to measure severity of depressive symptoms and quality of life. Participants completed these assessments twice (i.e., at baseline and after an approximately 15-week interval), allowing us to also assess savings of adaptation as well as the link between changes in mood and sensorimotor performance. We found that across all participants' reliable adaptation patterns and savings of adaptation were observed. Results of Bayesian models showed moderate evidence indicating that depression was not associated with poorer adaptability or savings. Furthermore, adaptation rates were not associated with the severity of depressive symptoms. These findings further refine the psychomotor profile of depression and suggest that it does not affect sensorimotor adaptation or savings of adaptation.

Keywords: motor adaptation, sensorimotor behavior, depressive disorder, sensorimotor adaptability

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Key Points

- Several motor functions, including gross and fine motor skills, are affected in those with depression.
- The present study indicates that the ability to adapt sensorimotor behavior is preserved in depression.
- Future studies should explore different adaptation paradigms and use multimethod approaches to further elucidate effects of depression on sensorimotor adaptation.

While depression is mostly known for its characteristic emotional and cognitive symptoms (e.g., persistent feelings of sadness, loss of pleasure, and decreased interest in activities), a growing body of evidence additionally points toward the presence and clinical importance of motor symptoms in this disorder (Schrijvers et al., 2008; van Diermen et al., 2017, 2019). Two well-known motor symptoms that can present in depression are psychomotor retardation and agitation. Psychomotor retardation refers to a significant deceleration of physical movements (e.g., slowed speech, slumped posture; Bennabi et al., 2013), whereas psychomotor agitation refers to a state of physical and mental restlessness, resulting in increased motor activity (e.g., fidgeting, trembling, constant walking; Vieta et al., 2017). Previous research demonstrated that other aspects of motor function are also affected in depression (e.g., Elkjær et al., 2022; Lohr et al., 2013; Paquet et al., 2022). For instance, individuals with depression exhibit changes in muscle function (e.g., elevated muscle tone, reduced force steadiness), gross motor skills (e.g., slowed gait, reduced postural stability), and fine motor skills (e.g., slower tapping, less precise hand movements). Various underlying mechanisms have been proposed to explain such motor difficulties, including depression-related neurobiological alterations (e.g., Northoff et al., 2021), effects of antidepressants (e.g., Schrijvers et al., 2008), cognitive problems, including executive dysfunction (e.g., Bennabi et al., 2013), and decreased motivation and energy levels that can manifest as motor disturbances. It is likely that a combination of these factors underlies the motor disturbances observed in depression.

While impairments in various types of motor performance thus have been consistently reported in individuals with depression, it remains unclear whether the ability to *adapt* motor behavior is also affected. Sensorimotor adaptation refers to the ability to adjust motor commands and representations in response to changing environmental or internal demands, allowing maintenance of appropriate, goal-directed motor performance (e.g., Krakauer & Mazzoni, 2011). Previous studies have shown that changes in motor representations due to sensorimotor adaptation can outlast the training session—such savings of adaptation have been reported 1 day after initial learning (e.g., Bedard & Sanes, 2011; Malone et al., 2011), several weeks or months later (Ruitenberg, De Dios, et al., 2018, Ruitenberg, Koppelmans, et al., 2018; Shadmehr & Brashers-Krug, 1997), and even as much as 1 year after initial learning (e.g., Landi et al., 2011). In terms of neural correlates, sensorimotor adaptation and savings of adaptation are thought to rely on corticostriatal and cerebellar-thalamocortical networks involving a variety of prefrontal, sensorimotor, cingulate, and parietal cortical areas (e.g., Ruitenberg, Koppelmans, et al., 2018; Seidler, 2010; Wolpe et al., 2020). Given observations that these

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structures and networks are altered in individuals with depression (e.g., Peng et al., 2011; Walther et al., 2012; Wüthrich et al., 2024), it is conceivable that depression may negatively affect sensorimotor adaptability.

Another open question is to what extent sensorimotor adaptability is associated with the severity of depressive symptoms. Prior studies involving relatively coarse measures of motor function observed that more severe depression was associated with slower average walking speed (Knapen et al., 2012), fewer gross body movement (Sandmeir et al., 2021), and reduced overall motor activity (Finazzi et al., 2010; but see Razavi et al., 2011). However, it remains to be studied specifically in depression populations to what extent the severity of depressive symptoms impacts the ability to perform motor adaptations. In addition, exploring the association between changes in adaptation and changes in depression severity over time could provide insight into the extent to which potential sensorimotor adaptation deficits in depression represent states rather than traits. That is, changes in sensorimotor adaptability that are related to decreases in depression symptomatology would suggest that deficits are changeable (i.e., a "state"), whereas sensorimotor dysfunctions that do not resolve with improvement in symptoms would be indicative of more structural differences in those with depression (i.e., a "trait").

The present study employed a visuomotor adaptation paradigm in which participants used a joystick to hit targets presented on a screen. Initially, they performed the task with normal visual feedback, but then had to adapt to 45° rotated feedback. The first aim of this study was to compare sensorimotor adaptability between individuals with and without depression. We hypothesized that participants with depression would exhibit poorer adaptation compared with control participants. Participants performed the same task for a second time approximately 15 weeks after having first performed the task. This allowed us to examine potential effects of depression on savings of adaptation. Finally, we evaluated whether the severity of depressive symptoms was associated with the extent of adaptation. We hypothesized that more severe depressive symptomology would be associated with poorer sensorimotor adaptation.

Methods

Participants

A total of 42 individuals (15 males/27 females) were enrolled in the study, ranging in age from 19 to 58 years (mean age = 31 ± 10 years). The sample comprised two groups: individuals with depression and healthy controls (HC). Their demographic and clinical characteristics are presented in Table 1; an overview of medication use in the group of individuals with depression is presented in Supplementary Figure S1 (available online). According to scores on the Dutch Handedness Questionnaire (Van Strien, 1992), 41 participants were right-handed and one was left-handed. Participants with depression were recruited from the University of Utah and through referrals and flyers, while control subjects were recruited via the university's study locator website, Center on Aging, researchmatch.org, referrals, and flyers.

Inclusion criteria required all participants to be over 18 years of age and be proficient in English. Individuals with depression were required to be experiencing

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Table 1 Demographic Data and Questionnaire Scores (Mean ± *SD*) for DEP and HC

	DEP ($n = 20$)	HC ($n = 22$)	Group difference
Age	30.6 ± 11.1	31.8 ± 10.2	$F(1, 40) = 0.137$, $p = .713$
Sex ratio (M/F)[a]	8/12	7/15	$\chi^2(1) = 3.429$, $p = .064$
Years of education	15.8 ± 3.23	17.1 ± 1.75	$F(1, 40) = 3.07$, $p = .087$
HAM-D-24 score[b]	24.1 ± 6.66	N/A	N/A
HAM-D-17 score[b]	24.1 ± 4.94	N/A	N/A
GAD score	11.5 ± 5.08	2.6 ± 3.73	$F(1, 40) = 42.94$, $p < .001$
PHQ-9 score	15.8 ± 5.87	2.5 ± 2.28	$F(1, 40) = 96.93$, $p < .001$
WHOQOL domains			
Physical health	21.4 ± 4.31	30.5 ± 3.29	$F(1, 40) = 59.8$, $p < .001$
Psychological health	14.4 ± 3.56	23.2 ± 3.19	$F(1, 40) = 70.34$, $p < .001$
Social relationships	8.8 ± 2.28	11.3 ± 2.33	$F(1, 40) = 12.0$, $p = .001$
Environment	28.4 ± 4.31	32.6 ± 3.29	$F(1, 40) = 8.88$, $p = .005$

Note. GAD-7 = Generalized Anxiety Disorder-7; PHQ-9 = Patient Health Questionnaire; WHO-QOL = World Health Organization Quality of Life questionnaire; HAM-D = Hamilton Depression Rating Scale; DEP = participants with depression; HC = healthy control participants.
[a]Ratio as opposed to $M \pm SD$ is provided. [b]Scores on the HAM-D-24 and HAM-D-17 are based on data from $n = 17$ participants with depression.

a mild, moderate, or severe depressive episode (with mild defined as a score of 10–13, moderate as a score of 14–17, and severe as a score of >18 on the 17-item Hamilton Depression Rating Scale [HAM-D]). Exclusion criteria included: poorly controlled medical conditions, current pregnancy, schizophrenia or schizoaffective disorder (lifetime), traumatic brain injury within the past year, history of neurological conditions within the past year, substance use disorder within the past 3 months, neurocognitive disorders within the past year, autism spectrum disorder or intellectual disabilities, and any contraindications for undergoing magnetic resonance imaging. Additionally, individuals were excluded from the HC group if they had a history of depressive episodes. bipolar disorder, posttraumatic stress disorder, obsessive compulsive disorder, eating disorder, or daily use of psychotropic medication. Accurate group classification was corroborated with the administration of the Mini Internal Neuropsychiatric Interview and the HAM-D.

Sensorimotor Adaptation Task

The manual visuomotor adaptation task used in the current study has been used extensively to study sensorimotor adaptation by us and others (e.g., Anguera et al., 2010, 2011; Lametti et al., 2020; Ruitenberg, De Dios, et al., 2018, Ruitenberg, Koppelmans, et al., 2018, Ruitenberg et al., 2022, 2023; Seidler, 2005, 2006; Seidler et al., 2006). Participants used their preferred hand to move a dual-axis joystick to hit targets presented on a screen. A cursor (red circle) on the screen provided real-time feedback on the joystick location. Each trial started with the

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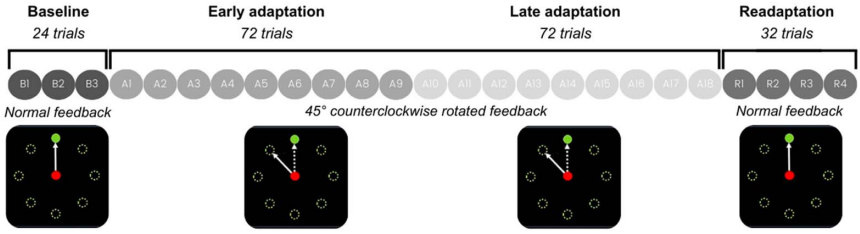

Figure 1 — Overview of the visuomotor adaptation task structure and visual feedback across blocks.

joystick and cursor being at the central position. Then, a target (green circle) was displayed for 1,000 ms at one of eight possible locations 4.6 cm from the center of the screen (see Figure 1). Participants were instructed to move the cursor toward the target as fast and accurately as possible, and to hold the cursor on target until it disappeared. At this point, they could release the joystick to allow its return to the starting position.

As illustrated in Figure 1, the task comprised 25 blocks of eight trials each. Baseline blocks (B1–B3; 24 trials) were performed under normal visual feedback (i.e., cursor location would correspond with the location of the joystick). Adaptation blocks (A1–A18; 144 trials) involved 45° counterclockwise rotated feedback. Last, in the readaptation blocks (R1–R4; 32 trials) the rotation was removed, and participants performed the remaining trials under normal visual feedback which allowed us to measure after-effects of adaptation. Participants could take a self-paced break (1-min max) after Blocks 8 and 16. Stimulus presentation, timing, and data recording during the task were controlled by PsychoPy software (version 1.84.1; Peirce et al., 2019).

Questionnaires

Severity of depressive symptoms was assessed with the Hamilton Depression Rating Scale (HAM-D; 17- and 24-item versions; Hamilton, 1960) and the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001). For both instruments, higher total scores reflect more severe depressive symptoms. In addition, the abbreviated version of the World Health Organization Quality of Life questionnaire (WHO-QOL-BREF; The WHOQOL Group, 1998) was used to assess quality of life in four domains: physical health, psychological health, social relationships, and environment. Higher scores on this questionnaire indicate better perceived quality of life in the respective health domain.

Procedure

After undergoing screening and the Mini Internal Neuropsychiatric Interview interview via phone, eligible participants provided digital informed consent through Research Electronic Data Capture (Harris et al., 2009, 2019). Participants with depression completed the HAM-D with one of the study psychiatrists or psychiatry

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residents (Douka, Weischedel, and Mickey). A week prior to their study visit, all participants were sent the Generalized Anxiety Disorder-7 (GAD-7), PHQ-9, and WHOQOL questionnaires to be filled out in their own time. During the first in-person site visit, participants completed the sensorimotor adaptation task in addition to other motor behavioral assessments, cognitive tests, and an magnetic resonance imaging scan (these data will be presented elsewhere). During a return visit the same in-person motor and cognitive tests were repeated, on a separate day the online questionnaires and HAM-D interview were completed again as well. The median interval between the first and the second test session was 14.3 (range = 10–23.6) weeks for the control group and 16.78 (range = 12.3–36.6) weeks for the depression group. From the first to the second session, three participants with depression and three controls were lost to attrition.

Sensorimotor Adaptation Data Processing

Performance on each trial of the motor adaptation task was assessed using the direction error (DE), an approach widely used in sensorimotor adaptation research (Lametti et al., 2020; Ruitenberg et al., 2022; Shadmehr & Mussa-Ivaldi, 1994; Seidler, 2005, 2006). DE is defined as the angle between the line from the starting position to the target and the line from the starting position to the cursor's location at peak velocity. Trials with DEs exceeding 2.5 *SD* from the mean DE per phase per participant were excluded, resulting in the removal of 1.56% of the trials. Participants with missing data on Session 2, due to technical issues (DEP = 4, HC = 3) or attrition (DEP = 3, HC = 3), were excluded only from analyses involving Session 2.

For each participant, we then calculated the mean DE per block. In addition, we determined the rate of adaptation by calculating the decay constant across adaptation trials and used this as the primary outcome measure for studying adaptation (cf. Ruitenberg, Koppelmans, et al., 2018, Ruitenberg et al., 2022, 2023). An exponential decay function was fit to DEs using the Nonlinear Least Squares function from the R stats package which is part of R core (mean $R^2 = .23 \pm .16$), following previous work demonstrating that sensorimotor adaptation data can be characterized well by exponential decay functions (Burge et al., 2008; Krakauer et al., 2000; Morehead et al., 2015; Zarahn et al., 2008). This was done for all adaptation trials of each participant to obtain an estimate of the adaptation rate constant, as well as separately for the first and second half (i.e., 72 trials each) of all adaptation trials to differentiate between early and late phases of adaptation. We also determined the decay constant across all 32 readaptation trials. More positive adaptation and readaptation rates indicate a steeper decay across the trials, and thus reflect faster improvement. Visual inspection of each individual's adaptation plots in combination with Grubb's test identified one HC participant as an outlier; their adaptation rate data were therefore excluded from the analyses. Last, to examine savings of adaptation, we determined individual savings scores by subtracting the mean DE in the first adaptation block of Session 1 from the mean DE in the first adaptation block of Session 2 for each participant (cf. Ruitenberg, De Dios, et al., 2018, Ruitenberg, Koppelmans, et al., 2018). Savings scores were obtained for 29 participants (DEP = 13 and HC = 16).

Finally, we determined movement onset (reaction time; RT) and movement duration (movement time: MT) via a method described in Haith et al. (2015) and Lametti et al. (2020). In brief, position data were smoothed using a second-order

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Savitzky–Golay filter with a frame length of 11 samples. The data were differentiated and tangential velocity was calculated. Movement onset was identified as the time after trial start at which tangential velocity first exceeded 2.5 cm/s (less than 4% of peak velocity). Movement duration was calculated as the time during each trial for which tangential hand velocity exceeded 2.5 cm/s.

All statistical analyses were conducted using R (version 4.3.1, R Core Team, 2021), employing both frequentist and Bayesian models. For the latter, Bayes factors ($BF_{inclusion}$ for analyses of covariance [ANCOVAs] and BF_{10} for *t* test and regression analyses) were determined using the BayesFactor package (version 0.9.12.4.7; Morey et al., 2024). An advantage of Bayesian models is that they allow for determining the presence or absence of an effect particularly in small sample sizes (e.g., Keysers et al., 2020; Van Doorn et al., 2021). A $BF > 3$ is regarded as moderate evidence for the alternative hypothesis (with $BF > 10$ indicating strong evidence), and a BF between one and three as merely anecdotal evidence. In contrast, a $BF < 0.33$ is regarded as moderate evidence for the null hypothesis (with $BF < 0.1$ indicating strong evidence), and a BF between 1 and 0.33 as anecdotal evidence (Dienes, 2011; Keysers et al., 2020).

Results

General Adaptation Pattern

To determine if our data were in line with the general adaptation pattern typically observed in sensorimotor adaptation studies, we performed an ANCOVA on DEs with group (2; depression vs. control) as between-subjects factor, block (25; see Figure 1) as within-subjects factor, and age as a covariate. The latter was included as prior studies have consistently shown age effects on sensorimotor adaptation (e.g., Seidler, 2006; Wolpe et al., 2020). Results showed an effect of block, $F(24, 38) = 44.83$, $p < .001$, $\eta^2 = .68$, $BF_{inclusion} > 100$, and age, $F(1, 38) = 14.535$, $p = .001$, $\eta^2 = .01$, $BF_{inclusion} = 0.80$, but no group effect, $F(1, 38) = 2.40$, $p = .121$, $\eta^2 < .001$, $BF_{inclusion} = 0.07$, nor interactions. Post hoc analyses using Tukey's method confirmed the general adaptation pattern (see Figure 2) characterized by a significant increase in DE with the introduction of the rotated feedback, followed by a gradual decrease in DE. Finally, when the rotated feedback was removed, DE increased in the opposite direction as participants readapted to the normal feedback condition. Results of a post hoc regression analysis showed that older age was associated with larger DE, $\beta = 0.131$, $SE = 0.060$, $t = 2.18$, $p = .029$, $BF_{10} = 0.716$.

To evaluate potential movement speed differences between groups, we performed separate ANCOVAs on RT and MT with group (2; depression vs. control) as a between-subjects factor, phase (3; baseline, adaptation, readaptation) as within-subjects factor, and age as a covariate. Results showed no group differences in RT, $F(1, 38) = 0.21$, $p = .650$, $\eta^2 = .001$, $BF_{inclusion} = 0.093$, or in MT, $F(1, 38) = 1.37$, $p = .244$, $\eta^2 = .01$, $BF_{inclusion} = 0.278$. For RT, results showed an effect of age, $F(1, 38) = 4.37$, $p = .039$, $\eta^2 = .04$, $BF_{inclusion} = 0.124$. Results of a post hoc regression analysis showed that older age was associated with longer RTs, $\beta = 0.00027$, $SE = 0.00011$, $t = 2.40$, $p = .017$, $R^2 = .0063$. There were no other significant effects for RT or MT ($Fs < 1$, $ps > 0.39$).

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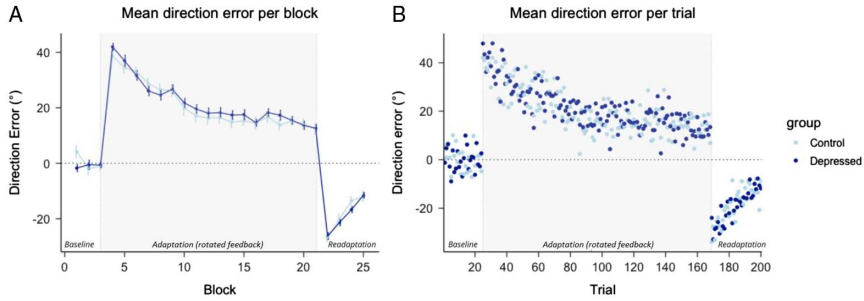

Figure 2 — Mean direction error per block (panel A) and per trial (panel B) across the task. Data from the group with depression are shown in dark blue, and data from the HC group are shown in light blue. Shaded areas represent blocks and trials with rotated feedback. Error bars represent *SE*s. See online article for color version of the figure.

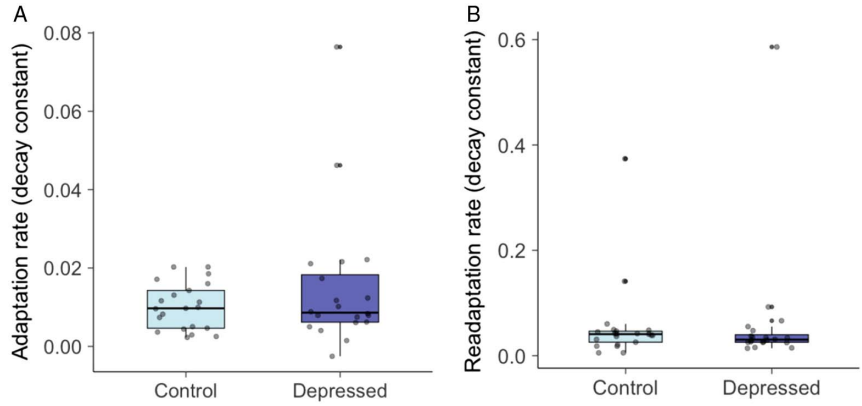

Figure 3 — Box plots illustrating mean adaptation rates across the adaptation phase (left panel) and readaptation phase (right panel) of participants in the depression and control groups. More positive rates indicate a steeper decay over the trials, that is, faster improvement. Note that the *y*-axes have different scales.

Adaptation Rates

Group differences in sensorimotor adaptation were assessed separately for each phase using ANCOVAs on adaptation rates, with group (2) as a between-subjects factor and age as a covariate. For the adaptation phase (Figure 3, left panel), results showed no group differences in overall adaptation rates, $F(1, 38) = 1.123$, $p = .296$, $\eta^2 = .04$, $BF_{inclusion} = 0.50$. However, in line with prior studies, we did observe a main effect of age, $F(1, 38) = 4.146$, $p = .049$, $\eta^2 = .10$, $BF_{inclusion} = 1.71$. A post hoc regression analysis showed an association of older age with slower adaptation, $\beta = -0.0004$, $p = .039$, $R^2 = .10$, $BF_{10} = 1.765$. When separately examining the early and late adaptation phases, results showed no group differences in early

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adaptation rates, $F(1, 38) = 0.452$, $p = .505$, $\eta^2 = .02$, $BF_{inclusion} = 0.38$, or late adaptation rates, $F(1, 38) = 0.019$, $p = .892$, $\eta^2 < .001$, $BF_{inclusion} = 0.31$. Also, no effects of age were observed for the early, $F(1, 38) = 2.578$, $p = .116$, $\eta^2 = .06$, $BF_{inclusion} = 0.911$, and late adaptation phases, $F(1, 38) = 0.955$, $p = .334$, $\eta^2 = .02$, $BF_{inclusion} = 0.448$. Last, results for the readaptation phase (Figure 3, right panel) also showed no group difference, $F(1, 38) = 0.014$, $p = .907$, $\eta^2 < .001$, $BF_{inclusion} = 0.31$, nor an effect of age, $F(1, 38) = 1.306$ $p = .260$, $\eta^2 = .03$, $BF_{inclusion} = 0.92$.

Motor Adaptation Savings

Savings scores were obtained for 28 participants (DEP = 13 and HC = 15). A one sample *t* test across all participants determined that motor adaptation savings scores were significantly different from zero, $t(27) = 2.09$, $p = .045$, $BF_{10} = 1.31$. Next, to determine group differences in savings an ANCOVA was performed on savings scores with group as between-subjects factor and age as covariate. As illustrated in Figure 4, results indicated no significant effects of group, $F(1, 25) = 0.579$, $p = .454$, $\eta^2 = .052$, $BF_{inclusion} = 0.43$, or age, $F(1, 26) = 0.069$, $p = .794$, $\eta^2 < .001$, $BF_{inclusion} = 0.39$.

Associations Between Adaptation and Depression Severity

To determine if adaptation rates were associated with the severity of depressive symptoms of individuals within the depressed group, we conducted separate linear regressions on the decay constants per phase (adaptation, early adaptation, late adaptation, and readaptation), and the HAM-D-24, HAM-D-17, PHQ-9, and WHOQOL questionnaire scores. Regression analyses showed no significant associations ($ps > .055$, $\beta s < 0.153$, $BF_{10} = 0.267–1.174$) between adaptation rates and severity of symptoms. The associations between the (re)adaptation rates and depression severity questionnaires are illustrated in Figure 5.

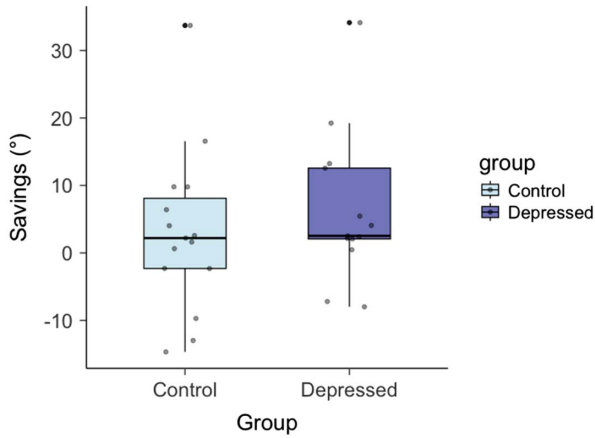

Figure 4 — Box plot illustrating savings in the depression and control groups.

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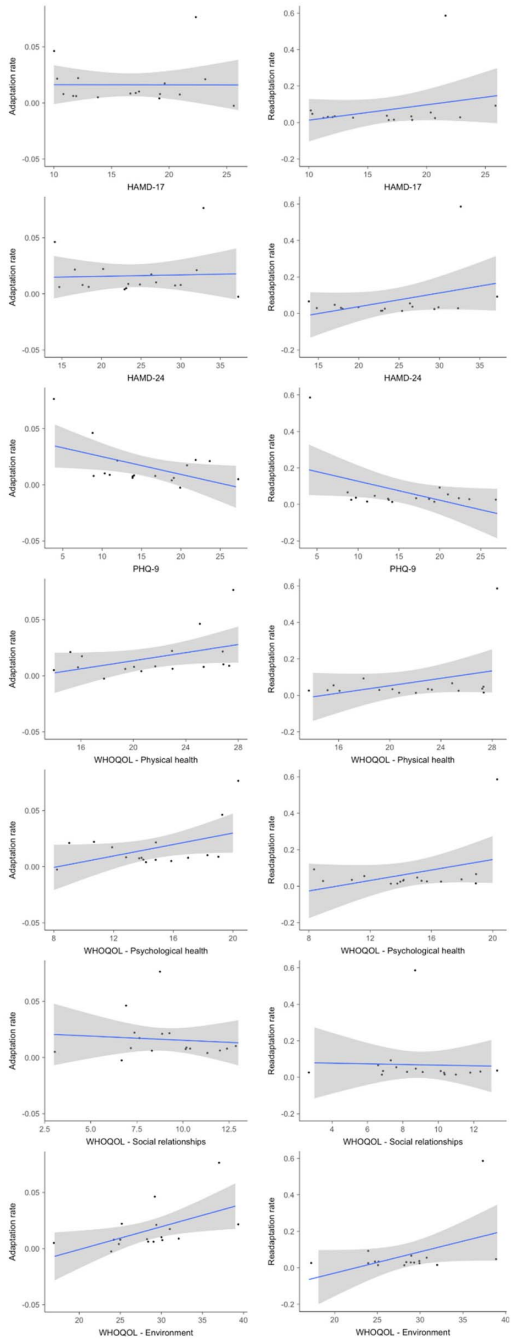

Figure 5 — Scatter plots showing the associations between depression severity (as reflected in questionnaire scores) and adaptation rates for the adaptation and readaptation phases of participants in the depression group. Note that the *y*-axes have different scales; shaded areas denote the 95% confidence interval. PHQ-9 = Patient Health Questionnaire; WHOQOL = World Health Organization Quality of Life questionnaire; HAM-D = Hamilton Depression Rating Scale.

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Table 2 Associations Between Changes in Adaptation Rates and Changes in Depression Severity Between the Two Test Sessions for Individuals in the Depression Group

	Change in adaptation				Change in readaptation			
Change in severity	***n***	**β**	***p***	**BF_{10}**	***n***	**β**	***P***	**BF_{10}**
HAM-D-24	6	0.0005	.436	0.525	5	0.0001	.886	0.478
HAM-D-17	7	0.0007	.436	0.498	6	0.0004	.959	0.391
PHQ-9	12	–0.0006	.559	0.446	11	0.0067	.239	0.340
WHOQOL domains								
Physical health	12	0.0014	.669	0.810	11	0.0071	.281	0.312
Psychological health	12	–0.0003	.874	0.342	11	–0.024	.051	0.832
Social relationships	12	0.0077	.087	0.491	11	–0.013	.516	0.239
Environment	12	–0.0015	.157	0.670	11	–0.004	.533	0.236

Note. PHQ-9 = Patient Health Questionnaire; WHOQOL = World Health Organization Quality of Life questionnaire; HAM-D = Hamilton Depression Rating Scale; BF = Bayes factor.

Linear regressions were used to analyze the associations between changes in adaptation rates and changes in depression severity. We first calculated the change in adaptation and readaptation performance, as well as depression severity by determining the differences between the first and second test session in adaptation, and readaptation rates, as well as change in the HAM-D-24, HAM-D-17, PHQ-9, and WHOQOL questionnaire scores. Separate regression analyses were then performed on changes in adaptation and readaptation change rates for each of the questionnaires, with age included as a covariate. As shown in Table 2, results of these regressions indicated no significant associations between changes in depression severity and changes in rates of adaptation or readaptation.

Discussion

The present study used a manual visuomotor adaptation task to examine whether sensorimotor adaptation is affected in individuals with depression compared to individuals without a history of depression. Across our sample, we observed a typical adaptation pattern. Furthermore, participants were less perturbed by the rotated feedback in the second session as compared to the initial test session that took place several weeks earlier, indicating that savings of adaptation occurred. While these general patterns observed across both groups align with the well-established patterns seen in previous sensorimotor adaptation studies, results showed no indications that they were affected by depression. Specifically, Bayesian models showed moderate evidence for an absence of group differences, suggesting that motor adaptability and savings are preserved in depression. As this absence of group differences was observed in the adaptation and readaptation phases, this suggests that both the formation of sensorimotor representations to accommodate performance in the new, rotated context (i.e., adaptation) and the strength of the formed sensorimotor representations during adaptation

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(i.e., readaptation) are preserved. Somewhat unexpectedly, it was also observed that movement speed in the present task did not differ between groups. This is in contrast with previous findings of slower motor performance in individuals with depression (e.g., Elkjær et al., 2022; Lohr et al., 2013; Paquet et al., 2022). Since our participants were all community-dwelling patients and not hospitalized (which for example is in contrast to those in Paquet et al., 2022), their depression may not have been severe enough for motor impairments to become apparent. Overall, the present findings suggest that the ability to adapt movements may be preserved in individuals with depression, though replication in another, more severely depressed sample that displays reduced movement speed is desirable.

Despite these novel findings that depression does not affect sensorimotor adaptation, an open question remains as to what extent this generalizes to both explicit, strategic processes versus implicit processes of adaptation. It is thought that strategic, cognitive processes contribute to the relatively early phase of sensorimotor adaptation, characterized by fast improvements, whereas more implicit processes and increasing reliance on automaticity contribute to later phases, characterized by relatively slow improvements (e.g., Huberdeau et al., 2015; McDougle et al., 2016). Given that, in particular, the early phase is thought to rely on cognitive processes such as spatial working memory and inhibitory control, that are known to also be affected in depression (e.g., Galkin et al., 2020), it is conceivable that depression may be linked to impairments in early but not late adaptation. Within the field of sensorimotor adaptation, the categorization of the early and late adaptation phases is often made rather arbitrarily—most studies on sensorimotor adaptation define the early and late phase within the context of their design, depending, for example, on the duration of the experiment and the total number of adaptation trials. In the present study, the latter method was used to define early and late to reflect the relatively explicit and implicit phases. We acknowledge that perhaps this arbitrary method may have obscured more subtle depression-related differences between early and late adaptation that arise even earlier (or later, for that matter) during learning. Future studies should evaluate whether depression differentially affects the implicit and explicit processes contributing to sensorimotor adaptation by using other adaptation paradigms (e.g., gradual rotation and clamped feedback) that can dissociate explicit and implicit processes, lending a more comprehensive understanding of the impact of depression on motor functioning.

Regarding potential associations between the severity of depressive symptoms (as measured by various standardized questionnaires, i.e., HAM-D-24, HAM-D-17, PHQ-9, and WHOQOL) and sensorimotor adaptability, the current results did not give indications for the presence of such associations. This suggests that, within our sample, the severity of depression does not appear to influence an individual's ability to adapt to the rotated feedback. In addition, longitudinal analyses assessing the relationship between changes in depression severity and changes in sensorimotor adaptation yielded no significant findings. Taken together, these findings suggest that sensorimotor adaptability is relatively independent of symptom severity in depression.

While the present study did not aim to examine effects of age on sensorimotor adaptation, it is noteworthy that older age was associated with slower improvements in performance (i.e., poorer adaptability) in our sample. This corroborates

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prior studies that specifically set out to investigate age effects on adaptation in adults and that have reported similar findings, although those samples included individuals up to 75–89 years of age (e.g., Seidler, 2006; Wolpe et al., 2020). Furthermore, studies using a lifespan approach demonstrated that the relationship between age and sensorimotor adaptation follows an inverted u-shape in a sample spanning roughly 8–70 years (e.g., Ruitenberg et al., 2022, 2023). The present findings extend prior observations by showing that age also affects sensorimotor adaptability for middle-aged individuals that are considered neither developmentally young nor old.

The use of Bayesian models that allowed us to interpret nonsignificant results following traditional null hypothesis statistical testing models is a strength to the study. However, we also acknowledge several limitations. The first pertains to the relatively small sample size that did not allow for differentiation between potential subtypes of depression (e.g., Tozzi et al., 2024). Future studies should distinguish between patients with versus without psychomotor disturbances (i.e., agitation or retardation) for a more comprehensive understanding of how depression affects sensorimotor adaptation. Furthermore, our sample comprised individuals with a currently active episode of depression who were taking medications. An open question therefore remains whether our findings generalize to other medication or depression states (e.g., remitted). Another limitation of the present work is that behavioral measures may not directly reflect brain mechanisms therefore the absence of an effect of depression on behavioral motor adaptation outcomes does not refute the possibility of effects at the neural level. For example, there may be changes in brain recruitment during the task that allow individuals with depression to maintain intact performance (i.e., compensatory mechanism), which could obscure group differences in behavior. Indeed, a prior functional magnetic resonance imaging (fMRI) study revealed functional differences in individuals with depression despite similar behavioral performance on a finger tapping task, indicative of compensation (Sarkheil et al., 2020). Future studies should therefore combine behavioral tasks with neuroimaging methods to further understand the effects of depression on motor adaptation. Furthermore, as our study only encompassed one sensorimotor adaptation task that required adaptation of hand movements to rotated visual feedback, the generalizability of findings to other types of adaptation remains unclear. Future studies should examine how depression affects manual adaptation to other types of perturbations (e.g., force-field reaching; prism adaptation) and other modalities of adaptation (e.g., locomotor adaptation, which requires bilateral gross motor control), as these may recruit different cognitive processes and/or brain areas and thus be differently affected by depression. Moreover, future studies could also examine the effect of depression on motor sequence learning, which is another type of movement skill.

Overall, results of the present study show evidence that depression does not affect motor adaptability or savings, indicating that some aspects of motor functioning are relatively spared in depression. This suggests that for individuals in rehabilitation settings (e.g., those recovering from injuries), clinicians may focus on sensorimotor training and rehabilitation techniques without needing to modify interventions based on depressive symptoms that can typically co-occur in these individuals. Future studies should explore different adaptation paradigms and use

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multimethod approaches to further elucidate the effect of depression on sensorimotor adaptation at both the behavioral and neural level.

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