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To cite this article: A. Lewin, M. Jöbges & K. Werheid (2013) The influence of self-efficacy, pre-stroke depression and perceived social support on self-reported depressive symptoms during stroke rehabilitation, Neuropsychological Rehabilitation, 23:4, 546-562, DOI: 10.1080/09602011.2013.794742

To link to this article: http://dx.doi.org/10.1080/09602011.2013.794742

Published online: 08 May 2013.

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The influence of self-efficacy, pre-stroke depression and perceived social support on self-reported depressive symptoms during stroke rehabilitation

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Post-stroke depression (PSD) is the most common mental disorder following stroke; however, little is known about its pathogenesis. We investigated the predictive value and mutual relationship of psychological factors such as self-efficacy and social support and known risk factors such as pre-stroke depression, activities of daily living (ADL), cognitive functioning, and age for the emergence of depressive symptoms in the acute phase after stroke. Ninety-six ischaemic stroke inpatients residing at a rehabilitation centre completed an interview about 6.5 weeks post-stroke. The interview included demographic data, psychiatric anamnesis, the Barthel Index, Mini-Mental State Examination, Social Support Questionnaire, Generalized Self-Efficacy Scale, Stroke Self-Efficacy Questionnaire, and the Geriatric Depression Scale. A multiple regression analysis was performed to ascertain the predictive value of the factors on depressive symptoms. High self-efficacy, no history of pre-stroke depression, and high levels of perceived social support were the strongest protective factors for depressive symptoms. The influence of cognitive functioning on depressive symptoms was fully mediated by general self-efficacy, and general self-efficacy was a stronger predictor than stroke-specific self-efficacy. Neither ADL nor age significantly predicted depressive symptoms. Our
findings suggest that consideration of self-efficacy and perceived social support in the inpatient rehabilitation setting may help prevent PSD.

Keywords: Stroke; Depression; Self-efficacy; Social support; Predictors.

INTRODUCTION

Depression is the most common mental disorder following stroke, affecting about one third of all stroke patients (Hackett, Yapa, Parag, & Anderson, 2005). Post-stroke depression (PSD) has been associated with impaired recovery in activities of daily living (ADL; Kotila, Waltimo, Niemi, Laaksonen, & Lempinen, 1984), decreased quality of life (QOL; Aström, Adolfsson, & Asplund, 1993; Sturm et al., 2004) and increased mortality (Ellis, Zhao, & Egede, 2010). Longitudinal research has revealed that increased depressive symptoms in the first weeks after stroke persist over one year (Huff, Steckel, & Sitzer, 2003; Robinson, Bolduc, & Price, 1987) and represent an increased risk for chronic depressive disorders (Aström et al., 1993; Robinson et al., 1987).

Research over the past 25 years has revealed several factors associated with higher levels of PSD. Some, such as gender, are controversial (Eriksson et al., 2004; Shepers et al., 2009). Converging evidence suggests that impaired ADL, impaired cognitive functioning, younger age, pre-stroke depression, low levels of social support, and personal traits (e.g., self-efficacy) are promising predictors for depressive symptoms after stroke, although these factors have been measured using different assessment techniques (e.g., self-report, therapist scale) and patient samples (Berg, Palomaki, Lehtihalmes, Lönnqvist, & Kaste, 2001; for a review see Huff et al., 2003; Jones & Riazi, 2010). In the following, previous findings on these predictors will be described in an order according to their descending theoretical importance, based on Huff et al.’s (2003) findings.

Impaired ADL in stroke patients was frequently shown to be related to increased levels of PSD by several studies three months after stroke (Aström et al., 1993; Eriksson et al., 2004; Kauhanen et al., 1999).

Impaired cognitive functioning was often reported to be associated with PSD (Hackett & Anderson, 2005; Kauhanen et al., 1999); however, the follow-up intervals varied greatly. Some studies reported a negative correlation between cognitive functioning and depressive symptoms measured up to five years after stroke (Hosking, Marsh, & Friedman, 2000; Sharpe et al., 1994). To our knowledge, the only study of the relationship between cognitive functioning and depressive symptoms as soon as one month after stroke was conducted by Andersen, Vestergaard, Ingemann-Nielsen, and
Lauritzen (1995). Thus, little information is available concerning the predictive value of cognitive functioning for depressive symptoms in stroke patients.

Younger age has repeatedly been shown to be associated with increased levels of depressive symptoms (Eriksson et al., 2004; Robinson, Starr, Kubos, & Price, 1983). Being under 65 years of age has been identified as an important predictor for PSD, possibly as a function of occupational status. Compared to retired patients, younger patients who are not yet retired may experience stroke as a more dramatic turning point in their lives (Robinson et al., 1983).

History of pre-stroke depression was reported to be linked to PSD by several studies (Andersen et al., 1995; Pohjasvaara et al., 1998; Wade & Hewer, 1987). According to these findings, patients with pre-stroke depression are at a higher risk for developing depressive symptoms after a stroke.

Despite accumulating evidence suggesting the predictive value of stroke-related and demographic factors, the precise pathogenetic mechanisms underlying PSD are not well understood. It is not known how the predictive and demographic factors interact. Moreover, these factors cannot explain why PSD does not emerge in some patients who have common risk factors such as young age or impaired ADL or cognition. Individual psychological factors such as self-efficacy and perceived social support may play an important role in the emergence of PSD symptoms. The fact that psychotherapy, a treatment focusing on psychological rather than stroke-related factors, has been reported to have a significant preventive effect on PSD (Hackett, Anderson, House, & Haltleh, 2008; Hackett, Anderson, House, & Xia, 2008) provides support for this hypothesis. Thus, increased understanding of specific psychological risk factors may contribute to the development of individually tailored psychological treatments. Several psychological factors may play a role in the emergence of PSD.

Social support is a well-documented protective factor for the development of depressive symptoms in the acute phase after stroke (Eriksson et al., 2004). Social support has generally been assessed using demographic or socioeconomic variables such as marital status and living alone versus living with others (Carod-Artal, Egido, Gonzalez, & de Seijas, 2000; Eriksson et al., 2004), or as part of general QOL questionnaires (Wilz & Barkova, 2007). However, accumulating evidence suggests that perceived support, a recipient’s subjective judgement that providers such as family members or friends will offer aid during times of need (Gurung, 2006), is more consistently linked to mental health status than is received support and social integration (Uchino, 2004). To our knowledge, no studies have assessed the effect of perceived social support on depressive symptoms after stroke.
Self-efficacy may also play an important role in the emergence of PSD; however, few studies have investigated this psychological factor. Two approaches have been used to investigate self-efficacy in the clinical setting: general and disease-specific self-efficacy.

General self-efficacy (Bandura, 1995) refers to a global confidence in one’s ability to cope with life challenges. It is a personal trait that individuals continually refer to when struggling with life events. The ability to cope with challenges, such as stroke, is high when the person feels in control during threatening situations. In people without somatic diseases, low levels of general self-efficacy are associated with high levels of depression (Schwarzer, Luszczynska, & Gutierrez-Dona, 2005). A mediating effect of general self-efficacy for disability factors and depression has been reported by Arnstein, Caudill, Mandle, Norris, and Beasley (1999) in patients with chronic pain. To our knowledge no studies have investigated the role of general self-efficacy in stroke patients.

The concept of disease-specific self-efficacy was introduced to examine beliefs used to cope with common functional impairments caused by disease rather than relying on general attitudes (Schwarzer et al., 2005). Disease-specific self-efficacy (e.g., “balance self-efficacy”) has been reported to be negatively correlated with post-stroke depressive symptoms (for a review see Jones & Riazi, 2010). Jones, Partridge, and Reed (2008) developed a questionnaire to measure stroke-specific self-efficacy in terms of self-reported confidence in functional performance after stroke. The questionnaire contained questions in various domains in which stroke patients would be working towards a particular goal such as personal care or mobility.

The present study examined several potential risk factors frequently associated with PSD, which have been investigated individually in previous studies. Our study focused on the first weeks after stroke, at which time risk and protective factors for depressive symptoms can be used as a basis for initiating secondary prevention. Our primary goals were to investigate whether these predictive factors were related to depressive symptoms shortly after stroke onset, and examine their relationship to each other. On the basis of previous research, we hypothesised that low levels of ADL and cognitive functioning, younger age, and history of pre-stroke depression would be associated with higher levels of depressive symptoms.

Furthermore, we investigated the early influence of psychological risk factors such as perceived social support and self-efficacy. We were specifically interested in whether general self-efficacy had an impact on the development of depressive symptoms independent of stroke self-efficacy, and whether it mediated the relationship between stroke-related risk factors and depressive symptoms, as suggested by earlier research in other clinical populations.
METHODS

Participants

Consecutive patients admitted to a neurological rehabilitation centre (Brandenburg Klinik, Bernau, Germany) over a 6-month period were recruited for the study. They had undergone on average 6.5 weeks of a multidisciplinary neurological rehabilitation programme and were assessed shortly before discharge. Only patients with ischaemic stroke were examined to exclude other neurological causes of negative affective outcome (e.g., bleeding or surgery following haemorrhagic stroke). Inclusion criteria were (1) at least 4 weeks after stroke onset, (2) sufficient verbal comprehension (fluent in German, no aphasia, Token test score ≥12), (3) no severe comorbidities (e.g., diabetes), and (4) at least 8 years of education. Criteria referring to age were not included, to allow for unrestricted variance of this variable in regression analyses. Eligible patients were invited to participate in the study by their neurologist or neuropsychologist. They received more detailed information by a member of the research team not involved in rehabilitation. Consenting patients were asked to complete a standardised assessment form in the presence of study personnel. Patients with motor impairments received help when needed while filling out the questionnaire. The study was approved by the ethics committee at the Department of Psychology, Humboldt Universitaet zu Berlin, Reg.-No 2010-13. Of 400 eligible patients from the rehabilitation centre, 150 who could be reached by the interviewer were informed about the study and asked to participate. A total of 105 participants provided informed consent. Of those, nine patients provided incomplete data and were excluded from the analysis. Thus, 96 complete data sets were analysed in the present study. The sex and levels of the investigated factors of the nine drop-outs did not differ from those of the patients who completed the study. Drop-outs were younger ($p = .03$), less educated ($p = .00$), and reported fewer depressive symptoms ($p = .05$). Treatment duration was associated with age ($t = −2.87, p = .01$) and sex ($t = −2.15, p = .04$) such that older patients and females were in treatment longer than 6.5 weeks post-stroke.

Measures and assessment procedure

After an initial interview to obtain demographic information and psychiatric anamnesis, depressive symptoms were assessed using the 15-item short form of the Geriatric Depression Scale (GDS; Yesavage et al., 1983), an economical, yet psychometrically sound, questionnaire for depressive symptoms (Appelros & Viitanen, 2004). The German version of the GDS has been evaluated in patients who are 70.1 ± 8.1 years of age. We chose the GDS because it is easy for patients to fill out and thus minimises cognitive load during the interview. Moreover, this self-report measure focuses exclusively on depressive symptoms and
does not contain somatic symptoms, which are unreliable in PSD patients. Higher GDS scores indicated higher levels of depressive symptoms. Depression was defined as ≥ 5 points according to Almeida and Almeida (1999).

ADL was assessed using the Barthel Index (BI; Mahoney & Barthel, 1965), a standard stroke-specific instrument that evaluates functional abilities in everyday tasks. The BI consists of 10 items with a total score ranging from 0 to 100, where 100 represents functional competence in all 10 items and 0 represents total dependence. It is easy to use and has good internal consistency (α = .96) and construct and criterion validity (Gresham, Phillips, & Labi, 1980; Wade & Hewer, 1987).

The Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) has scores ranging from 0 to 30 and was used to assess cognitive functioning. According to Bour, Rasquin, Boreas, Limburg, and Verhey (2010) the MMSE is a sensitive measure of mild to severe cognitive impairment in stroke patients. Participants who scored <26 on the MMSE were considered to have cognitive impairment.

Perceived social support was measured using the 22-item short form of the Social Support Questionnaire (F-SozU; Sommer & Fydrich, 1991). The scores range from 0 to 110, with higher scores representing higher levels of perceived social support. The questionnaire has been shown to be a reliable (internal consistency α = .91) and valid instrument (Sommer & Fydrich, 1991). Participants were asked to rate items (e.g., “There are people I can talk to when I feel upset.”) on a 5-point scale ranging from 1 (strongly agree) to 5 (strongly disagree).

General self-efficacy was assessed using the German Generalized Self-Efficacy Scale (GSES; Schwarzer & Jerusalem, 1995) with scores ranging from 0 to 40 where 40 represents the maximum level of self-efficacy. Participants rated items (e.g., “I can always manage to solve difficult problems if I try hard enough.”) on a 4-point scale ranging from 1 (not at all true) to 4 (exactly true). Schwarzer and Born (1997) reported that in a variety of samples, Cronbach’s alpha ranged from .76 to .90, with the majority of values in the high .80s, thus proving its criterion validity.

The Stroke Self-Efficacy Questionnaire (SSEQ; Jones et al., 2008) was administered to assess participants’ confidence in their functional performance after stroke. The score ranges from 0 to 195, where 195 indicates maximum self-efficacy following a stroke. The participants were asked to rate their confidence on a 10-point scale for each item (e.g., “Do your own exercise program every day”) where 0 = not at all confident and 10 = very confident. The authors of the SSEQ reported high internal consistency and validity (Jones et al., 2008).

Statistical analysis

The sum of the GDS scores was selected as the dependent variable. We controlled for the effect of demographic variables other than age on depressive
symptoms. For this purpose, we first determined the correlation between sex and marital status and the dependent variable. Factors that were significantly related to depressive symptoms were used as covariates in the regression analysis.

A hierarchical multiple regression analysis was performed to ascertain the predictive value of the investigated factors on depressive symptoms. Factors predicting a cross-sectional outcome were identified as predictors according to Dodge (2003). The assumptions for regression analysis were checked separately. Independent variables were entered stepwise into the model according to their descending theoretical significance based on previous research: (1) ADL, (2) cognitive functioning, (3) age, (4) pre-stroke depression, (5) perceived social support, (6) stroke self-efficacy, and (7) general self-efficacy. General self-efficacy was entered in the last step to test whether it was an independent factor that added to the effect of stroke self-efficacy on post-stroke depressive symptoms. To ensure a sufficient participant-to-variable ratio, only variables that were significantly related to the dependent variable were included as predictors in the regression analysis. Therefore, we first conducted correlation analyses between all independent variables and the criterion. The Bonferroni correction for multiple testing was used in the multiple regression analysis.

A mediation analysis was performed if a significant effect disappeared when further factors were added to the model. A mediator is an intervening variable that influences the relationship between the predictor and the criterion. According to Frazier, Tix, and Barron (2004), mediation occurs when the following requirements are fulfilled: (1) the predictor must show a significant relationship to the proposed mediator and the criterion, (2) the mediator must be related to the criterion, and (3) the relationship between the predictor and the criterion must be reduced when the mediator is entered into the equation. We tested mediation criteria by means of correlation and regression analyses. All statistical analyses were performed using the Statistical Package for Social Sciences version 19.0 (SPSS Inc., Chicago, IL, USA). A two-tailed $p$-value of .05 was deemed to be statistically significant.

**RESULTS**

Descriptive information

All patient descriptive information is shown in Table 1. Of the 96 patients, one-third (35.28%) scored an average of five points on the GDS and were considered depressed. A total of 15 patients scored below 26 on the MMSE, indicating cognitive impairment, and 13 of the 96 patients reported a history of pre-stroke depression.
Analysis of potential confounders

The correlation analysis revealed that sex ($r = .12, p = .23$) and marital status ($r = .04, p = .72$) were not significantly related to depressive symptoms and thus were not considered as covariates in the regression analysis.

Effect of depressive symptom predictors

Graphic inspection of the data revealed that the assumptions of homoscedasticity and normally distributed residuals were fulfilled. Furthermore, the Durbin-Watson Test showed no autocorrelation of residuals. All independent variables used in the multiple regression analyses were screened for multicollinearity, and the correlation coefficients are shown in Table 2.

Most of the correlations between potential predictors of depressive symptoms were less than .05 and thus acceptable according to Tabachnick and Fidell (1989). However, there was a large amount of shared variance between stroke self-efficacy and the Bartel Index, and some of the variables were significantly correlated with each other. Thus, we tested all tolerance levels for multicollinearity, including the variance inflation factor, which was found to be acceptable for all variables.

We conducted correlation analyses between all independent variables and the criterion. Age was not significantly related to GDS ($r = -.02, p = .82$), and thus age was not included as a predictor in the regression analysis.

In Step 1 ADL explained 6% and in Step 2 MMSE explained 9% of additional unique variance in depressive symptoms. In Step 3 pre-stroke

### TABLE 1

<table>
<thead>
<tr>
<th>Pre-stroke depression</th>
<th>Yes: 13.5%</th>
<th>No: 86.5%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>M: 52%</td>
<td>F: 48%</td>
</tr>
<tr>
<td>Age</td>
<td>67.08</td>
<td>10.55</td>
</tr>
<tr>
<td>Years of education</td>
<td>11.11</td>
<td>3.30</td>
</tr>
<tr>
<td>Weeks since stroke</td>
<td>6.64</td>
<td>4.42</td>
</tr>
<tr>
<td>GDS</td>
<td>4.39</td>
<td>3.59</td>
</tr>
<tr>
<td>BI</td>
<td>80.99</td>
<td>22.15</td>
</tr>
<tr>
<td>MMSE</td>
<td>28.10</td>
<td>2.41</td>
</tr>
<tr>
<td>F-SozU</td>
<td>87.62</td>
<td>16.96</td>
</tr>
<tr>
<td>SSEQ</td>
<td>156.41</td>
<td>38.25</td>
</tr>
<tr>
<td>GSES</td>
<td>30.66</td>
<td>6.5</td>
</tr>
</tbody>
</table>

GDS: Geriatric Depression Scale; BI: Barthel Index; MMSE: Mini-Mental Status Examination; F-SozU: Social Support Questionnaire; SSEQ: Stroke Self-Efficacy Questionnaire; GSES: General Self-Efficacy Scale.
depression significantly improved the model by explaining 16% of additional unique variance in depressive symptoms. When perceived social support was entered into the model, 14% of additional unique variance was explained, and in Step 5 stroke self-efficacy explained 5% of additional unique variance in the dependent variable. When general self-efficacy was entered again, 10% of additional unique variance was explained. In the full model all six variables explained 60% of the total variance in depressive symptoms.

The sequential entries of MMSE in Step 2, $F(2, 93) = 9.85, p = .00$, pre-stroke depression in Step 3, $F(3, 92) = 21.71, p = .00$, social support in Step 4, $F(4, 91) = 22.13, p = .00$, stroke self-efficacy in Step 5, $F(5, 90) = 8.20, p = .01$, and general self-efficacy in Step 6, $F(6, 89) = 21.00, p = .00$, significantly improved the model.

Pre-stroke depression, perceived social support, stroke self-efficacy, and general self-efficacy had significant effects on the outcome in the full model (Table 3). Although patients’ cognitive functioning was significantly related to their depressive symptoms at Steps 2, 3, 4, and 5, this relationship was not significant in Step 6 indicating that the subsequent entry of general self-efficacy included, in part, information contained in the cognitive functioning variable. ADL was a significant predictor of depressive symptoms at Steps 1 and 3 only. This relationship was not significant in any other steps.

### Mediating effect of general self-efficacy

We tested the hypothesis that general self-efficacy mediated the relationship between cognitive functioning and depressive symptoms.

To test for significant relationships between predictor, mediator, and criterion, correlations between the variables were estimated (requirements 1 and 2). All correlations were significant: high levels of cognitive functioning were associated with high levels of general self-efficacy ($r = .39, p = .00$)
### TABLE 3
Stepwise regression analyses for variables predicting depressive symptoms

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Step 1</th>
<th></th>
<th>Step 2</th>
<th></th>
<th>Step 3</th>
<th></th>
<th>Step 4</th>
<th></th>
<th>Step 5</th>
<th></th>
<th>Step 6</th>
<th></th>
<th>R² total</th>
<th></th>
<th>R² adjusted</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>BI</td>
<td>( \beta = .24 )</td>
<td>( p = .02 )</td>
<td>( \beta = .14 )</td>
<td>( p = .17 )</td>
<td>( \beta = .21 )</td>
<td>( p = .02 )</td>
<td>( \beta = .15 )</td>
<td>( p = .08 )</td>
<td>( \beta = .01 )</td>
<td>( p = .89 )</td>
<td>( \beta = .03 )</td>
<td>( p = .70 )</td>
<td>( R^2 = .06 )</td>
<td>( R^2 = .05 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMSE</td>
<td>( \beta = -.32 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.34 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.23 )</td>
<td>( p = .01 )</td>
<td>( \beta = -.18 )</td>
<td>( p = .03 )</td>
<td>( \beta = -.08 )</td>
<td>( p = .33 )</td>
<td>( \beta = .08 )</td>
<td>( p = .15 )</td>
<td>( R^2 = .13 )</td>
<td>( R^2 = .05 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-stroke depression</td>
<td>( \beta = -.41 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.33 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.33 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.26 )</td>
<td>( p = .00 )</td>
<td>( \beta = .31 )</td>
<td>( p = .29 )</td>
<td>( R^2 = .29 )</td>
<td>( R^2 = .29 )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-SozU</td>
<td>( \beta = -.40 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.34 )</td>
<td>( p = .00 )</td>
<td>( \beta = -.22 )</td>
<td>( p = .01 )</td>
<td>( \beta = .45 )</td>
<td>( p = .42 )</td>
<td>( R^2 = .42 )</td>
<td>( R^2 = .42 )</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SSEQ</td>
<td>( \beta = -.28 )</td>
<td>( p = .01 )</td>
<td>( \beta = -.22 )</td>
<td>( p = .01 )</td>
<td>( \beta = .49 )</td>
<td>( p = .46 )</td>
<td>( R^2 = .46 )</td>
<td>( R^2 = .46 )</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSES</td>
<td>( \beta = -.38 )</td>
<td>( p = .00 )</td>
<td>( \beta = .59 )</td>
<td>( p = .56 )</td>
<td>( R^2 = .56 )</td>
<td>( R^2 = .56 )</td>
<td></td>
<td></td>
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</tbody>
</table>

\( \beta = \) standardised multiple regression coefficient, \( p = \) level of significance. BI: Barthel Index; MMSE: Mini-Mental Status Examination Social Support Questionnaire; SSEQ: Stroke Self-Efficacy Questionnaire; GSES: General Self-Efficacy Scale.
and low levels of depressive symptoms ($r = -.36, p = .00$). Low levels of depressive symptoms were associated with high levels of general self-efficacy ($r = -.64, p = .00$). An additional regression analysis was performed to determine the effect of general self-efficacy on the predictor and criterion (requirement 3). General self-efficacy was included as a mediator after cognitive function was entered in the analysis to predict depressive symptoms.

The analysis revealed that the relation between cognitive functioning and depressive symptoms disappeared ($\beta = -.13, p = .13$) when general self-efficacy was entered into the equation ($\beta = -.59, p = .00$) indicating that general self-efficacy was a valid mediator of the relationship between cognitive functioning and depressive symptoms.

**DISCUSSION**

The present study investigated the predictive value and mutual relationship among risk factors for depressive symptoms in inpatients at a stroke rehabilitation centre with a particular focus on psychological factors such as perceived social support and self-efficacy.

Our primary finding was that low general self-efficacy was the strongest predictor of depressive symptoms in the first weeks following a stroke. This finding is consistent with earlier reports of a protective effect of high self-efficacy in depressed patients with no comorbid somatic disorders (Schwarzer et al., 2005). Furthermore, our finding that low stroke self-efficacy was associated with depressive symptoms is also consistent with that of previous research on the relationship between stroke self-efficacy and depressive symptoms (Robinson-Smith, Johnston, & Allen, 2000).

General self-efficacy and stroke self-efficacy are overlapping concepts; thus, it is not surprising that both factors were found to be important predictors of depressive symptoms. The fact that general self-efficacy was a stronger predictor than stroke self-efficacy of depressive symptoms may reflect the scope of the measurement instruments we used. The SSEQ questionnaire focused on specific competences (e.g., use both hands for eating food), some of which may not represent relevant aspects of the adaption process after stroke. Coping with stroke and its physical and psychosocial consequences may require complex and varied adaptation skills rather than the specific behaviour assessed by the SSEQ. Furthermore, the items on the SSEQ may not have the same degree of applicability for all stroke patients. Questions about self-perceived competences in walking and lying down may have lowered the scores for hemiplegic, but not non-hemiplegic, patients.

An alternative explanation for the difference in predictive value between general and stroke-self-efficacy is that general self-efficacy is based on life
experience, and as such, may include more aspects of the premorbid self-concept. A few weeks after stroke onset, patients may call upon this familiar premorbid self-concept when asked to evaluate coping with stroke-related problems rather than the new and constantly changing stroke-related functional competencies.

Perceived social support was found to be a protective factor for depressive symptoms after stroke. In contrast, marital status was not related to depressive symptoms during rehabilitation after stroke. These findings extend previous reports on the relevance of social environment (Carod-Artal et al., 2000; Eriksson et al., 2004; Mackenzie & Chang, 2002; Wilz & Barskova, 2007), suggesting that the key feature of the protective value of perceived social support is the presence of a social relationship which is perceived as helpful by the patient.

Turning to the analysis of the previously known predictors and their mutual interactions, our study confirmed the influence of pre-stroke depression, which was the second strongest predictor for depressive symptoms in our sample. This result agrees with previous research (Andersen et al., 1995; Hackett & Anderson, 2005), indicating that a history of pre-stroke depression has a significant influence on the development of depressive symptoms shortly after stroke onset. Pohjasvaara et al. (1998) demonstrated that pre-stroke depression predicted depressive symptoms in patients three months post-stroke. The present study found that pre-stroke depression is associated with early depressive symptoms after stroke, confirming and extending earlier findings by Wade and Hewer (1987).

Global cognitive status initially appeared to be a significant predictor, but the mediation analysis revealed that it was fully mediated by general self-efficacy. The positive correlation between global cognitive status and general self-efficacy is consistent with Bandura’s (1995) hypothesis that people with cognitive deficits tend to have less cognitive capacity to process positive and helpful feedback from their environment, which facilitates optimistic self-beliefs. Similarly, stroke survivors with limited cognitive capacity may be less able to use positive experiences or verbal feedback to build up self-efficacy which prevents depressive symptoms. Stroke patients with cognitive limitations, such as a memory deficit, may have difficulty in maintaining general self-efficacy as this process requires, for example, the ability to successfully recall managed situations. Our finding should, however, be interpreted with caution as the MMSE is a brief and unspecific measure of cognition. This may also explain the discrepancy between our findings and those of previous studies (Hackett & Anderson, 2005; Kauhanen et al., 1999). Furthermore, the relatively high cognitive level of our sample may indicate limited variance in the MMSE data and further limit the interpretation of our results. The difficulty in choosing appropriate cognitive measures for the heterogeneous deficit patterns observed in stroke patients is
well known (Hackett and Anderson, 2005), and future studies should use more complex, stroke-specific measures to examine the interactions between cognitive functioning, self-efficacy and depressive symptoms.

Impaired ADL was associated with depressive symptoms in the first and third steps of the regression analysis. Our finding that ADL impairment was not associated with depressive symptoms in the final model is consistent with findings by Aström et al. (1993) who reported that a relationship between ADL impairment and depressive symptoms was not observed during the acute phase after stroke, but emerged three months after stroke onset. This finding is plausible because in many patients, the level of ADL impairment may constantly change over the first weeks after stroke and stabilise later on.

We did not find an association between age and depressive symptoms in contrast to previous reports (Eriksson et al., 2004; Robinson et al., 1983). To explain the relationship between young age and depressive symptoms, Robinson et al. (1983) suggested that younger stroke patients might experience greater disruption to their lives than older patients who had already retired and would not face as much social restructuring. However, in the early post-stroke phase investigated in the present study, patients may not have been fully aware of the consequences on their future professional and social lives. Furthermore, our results may be related to the fact that our sample selection was based on specific inclusion criteria. We included only patients with ischaemic stroke and excluded haemorrhagic cases, which are often associated with surgery or other sources of negative affective outcomes. Moreover, the nine drop-outs that did not complete all parts of the assessment were significantly younger than the total sample. However, the fact that they were less depressed than the final sample contradicts the argument that non-significant age effects were caused by age-biased drop-out.

The age distribution in our sample was a further limitation of our study. We used the GDS to assess depressive symptoms. Although the GDS is psychometrically sound, it was developed for older patients. However, the mean age range of our sample was slightly below that of the population norm for the German version of the GDS (67.1 vs. 70.7 years, respectively); 54% of our patients were younger than 70 years of age. Nevertheless, the GDS has several features that make it appropriate for assessing depression in patients with organic diseases. The self-report scale’s “yes/no” response format is suited for patients with limited cognitive capacity, and it contains few insensitive items regarding somatic symptoms or inferring guilt. Nevertheless, further research to examine the self-report measures that are best suited for patients with post-stroke depressive symptoms is needed.

The advantage of the present study was that the patients’ personal perspectives were taken into consideration by assessing self-efficacy and perceived social support. Examination of the relationship between self-efficacy and perceived social support and previously known stroke-related and demographic
predictors of depressive symptoms by means of hierarchical multiple regression analysis revealed some aspects specific to the acute post-stroke phase. The findings of our cross-sectional study should be followed up in a longitudinal study. Our study leaves open the questions of whether general or stroke self-efficacy would undergo major changes in the later phases of stroke recovery, for example, when permanent cognitive losses or incomplete functional recovery is realised, and how this would affect the level of depressive symptoms.

A better understanding of the role self-efficacy plays during rehabilitation may help medical staff develop effective interventions for stroke patients. Such interventions may, for example, involve workbooks providing stories about other patients coping with stroke-related impairments (Jones, Mandy, & Partridge, 2009). Moreover, our finding that perceived social support had a protective effect against depressive symptoms should be followed up in a longitudinal study and may serve as a basis for interventions that involve patients and their social networks. For example, treatment focusing on helpful communication between the patient and family members or friends (Huijbregts, Myers, Streiner, & Teasell, 2008) could help the patient meet the challenges of dramatic stroke-induced changes in their everyday lives (Carnwath & Johnson, 1987).

Finally, our finding that patients with a history of pre-stroke depression are at higher risk for developing early post-stroke depressive symptoms suggests that patients with pre-stroke depression should be followed up after discharge and be screened regularly for depression.

**CONCLUSION**

This study demonstrated the impact of self-reported self-efficacy, perceived social support, and pre-stroke depression on depressive symptoms in the acute post-stroke phase. Our results suggest that considering psychological factors such as self-efficacy and perceived social support and assessing previous episodes of depressive disorders early after stroke onset may be valuable for prevention of PSD.

**REFERENCES**


Manuscript received September 2012
Revised manuscript received April 2013
First published online May 2013