

Temporal and Reciprocal Relationship Between IADL/ADL Disability and Depressive Symptoms in Late Life

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A strong association between functional disability and depressive symptoms in older people has frequently been reported. Some studies attribute this association to the disabling effects of depression, others to the depressogenic effects of physical health-related disability. The authors examined the reciprocal effects between depressive symptoms and functional disability and their temporal character in a community-based cohort of 753 older people with physical limitations who were assessed at yearly intervals. They compared structural equation models that differed in terms of direction and speed of effects between patient-reported disability in instrumental and basic activities of daily living (IADL/ADLs) and depressive symptoms. The association between disability and depression could be separated into three components: (a) a strong contemporaneous effect of change in disability on depressive symptoms, (b) a weaker 1-year lagged effect of change in depressive symptoms on disability (probably indirect through physical health), and (c) a weak correlation between the trait (or stable) components of depression and disability. IADL/ADL disability and depressive symptoms are thus mutually reinforcing over time. Compensatory forces like effective treatment and age-related adaptation may protect elders against this potential downward trend. To improve quality of life in elderly adults, treatment should target disability when it is new and depression when it is persistent.

CONSIDERABLE research efforts have been given to understanding the association of disability with major depression and subthreshold depressive disorder (referred to below as depression) since the introduction of the International Classification of Impairments, Disabilities and Handicaps in 1980 and the publication of landmark studies in the 1980s that found a strong association between disability and depression (Aneshensel, Frerichs, & Huba, 1984; Berkman et al., 1986; Wells et al., 1989). The depression–disability association appears universal. It is not limited to North American and European countries with their highly developed economics and social welfare systems, but is found in Asian, South American, and African populations as well (Ormel et al., 1994).

However, most studies to date have not examined reciprocal effects between depression and disability or the temporal character of the effects, but instead have focused on unidirectional relations. A notable exception is Aneshensel and colleagues' (1984) longitudinal study in a largely nonelderly population sample, showing that the association between disability and depression might have complex origins, including a variety of quick and delayed reciprocal effects.

Understanding the nature of the association between depression and disability is particularly important for the causal interpretation of the association between depression and functional disability in populations with a relatively high prevalence of physical disease, such as elderly adults.

In such populations disability may result from depression as well as from physical illness, and disability due to physical illness may increase risk of depression.

The aim of this study is to clarify the temporal and directional character of the relationship between depression and functional disability in later life using prospective data. Various mechanisms could account for the association between depression and disability. First, the association could be due to the disabling effects of depression (VonKorff, 1999). Depression is a demonstrated risk factor for onset of disability. There are now at least five prospective studies reporting that preexisting depression is a risk factor for onset of disability (Armenian, Pratt, Gallo, & Eaton, 1998; Bruce, Seeman, Merrill, & Blazer, 1994; Ormel et al., 1999; Ormel & VonKorff, 2000; Penninx et al., 1998). Moreover, effective treatment of depression improves functional outcomes (Coulehan, Schulberg, Block, Madonia, & Rodriguez, 1997; Mintz, Mintz, Arruda, & Hwang, 1992; Mynors-Wallis, Gath, Lloyd-Thomas, & Tomlinson, 1995; Tiemens et al., 1999).

Depression may cause disability by a different mechanism than physical illness does. Physical illness may produce disability because it impairs physical capacities such as mobility, vision, aerobic capacity, strength, manual dexterity, and continence. Depressive illness may produce disability because it impairs cognitive and motivational capacities, affect regulation, and social perception and increases a tendency to amplify physical symptoms (e.g.,

fatigue, pain; Ormel et al., 1994; VonKorff, 1999). In addition to this direct effect of depressive illness on functioning, depressive illness may cause disability indirectly, through a variety of behaviorally mediated pathways and psychobiological mechanisms (Penninx et al., 1998). These range from poor health behavior and compliance with medical treatment to psychoneuroendocrine pathways. Thus, it makes sense to distinguish a priori between a direct effect of depression on disability and a more indirect effect, through physical health.

Second, the association between depression and disability could be due to the depressogenic effects of disability produced by chronic physical illness. There is ample evidence that the disability associated with chronic medical conditions predict the onset and chronicity of depressive symptoms (Kennedy, Klerman, & Thomas, 1990; Phifer, 1986; Prince, Harwood, Thomas, & Mann, 1998; Turner & Noh, 1988; Zeiss, Lewinsohn, Rohde, & Seeley, 1996). Much late-life depression appears attributable to functional limitations caused by physical disease, in particular if these limitations reduce the ability to engage in usual social functions and contacts (Prince et al., 1998; Zeiss et al., 1996). Increasing and decreasing disability levels often have consequences for independence, self-esteem, valued activities, and social contacts (Brilman & Ormel, 2001).

Finally, the association between depression and disability might be due to common causes. Individual differences in generic liability to both physical and emotional ill health could be such a common cause. Specific physical diseases, in particular vascular diseases like arteriosclerosis, heart disease, and stroke, could be others. Substantial evidence has accumulated that implicates vascular disease as a risk factor for depression in later life, resulting in the diagnostic category of vascular depression (Alexopoulos et al., 1997; Baldwin & Tomenson, 1995; Krishnan & Gadge, 1996).

We report analyses of three-wave data on depressive symptoms and limitations in instrumental and basic activities of daily living (IADL/ADLs) in an older population with physical limitations. More specifically, we compared the fit of a series of models that differ in terms of direction and timing of effects, using structural equation modeling. Figure 1 shows the contemporaneous and 1-year lagged cross-variable effects in which the competing models differ. The two most extreme models are a model without any connection between depressive symptoms and IADL/ADL disability (the null model) versus a model that includes both contemporaneous (Paths d and e) and 1-year lagged (Paths a and b) cross-variable effects.

In summary, our aim was to examine the temporal character of the reciprocal effects between IADL/ADL disability and depressive symptoms. This way we strove to obtain insight in the processes that underlie the disability–depression association and help to reconcile apparently inconsistent evidence. This is the first published effort to prospectively examine the direction and timing of effects in an older population. We hypothesized that disability and depression are mutually reinforcing over time, setting off a potential downward spiral.

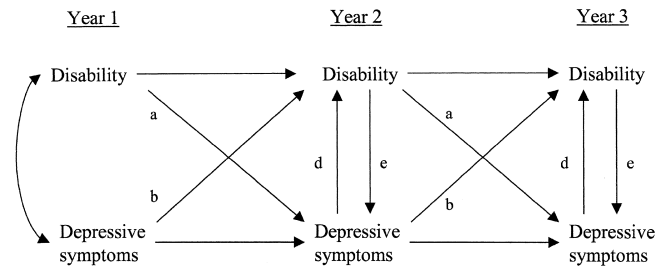


Figure 1. Possible contemporaneous and lagged cross-variable effects between disability and depressive symptoms.

METHODS

Participants

The present article reports on a cohort of 753 persons from the Groningen Ageing Study (GLAS) who were eligible—as a result of their physical limitations—for follow-up during 2 years with three waves of measurement. The cohort was selected from the source population of the 5,279 participants of GLAS, a cross-sectional population-based study of health-related quality of life in noninstitutionalized late-middle-aged and older people. This study was carried out in 1994 in the north of The Netherlands (56% women; 35% aged 57–64; 39% aged 65–74; 22% aged 75–84; 4% aged 85 and older; Kempen, Ormel, Brilman, & Relyveld, 1997; Ormel et al., 1998). The source population consisted of all persons aged 57 or older who were on the patient panels of the 27 general practitioners, most of whom participate in the Morbidity Registration Network Groningen. In The Netherlands nearly 100% of the noninstitutionalized population is on the panel of a general practitioner. They were interviewed face-to-face in their homes. (See for details regarding cross-sectional study Kempen et al., 1997; Ormel et al., 1998.)

The cohort of 753 participants included from the source population only those who had four or more physical limitations according to the Physical Functioning subscale of the Medical Outcome Study Short-Form General Health Survey (MOS-SF20; Kempen, Steverink, Ormel, & Deeg, 1996; Stewart, Hays, & Ware, 1988). The underlying assumption for this selection of physically limited elderly people was that they might experience more change in health status in a 2-year period than a random sample would. The cohort in the cross-sectional study reported four (35.9%), five (45.7%), or six (18.5%) limitations on the MOS-SF20 Physical Functioning scale. Examples of the questions that signal physical limitations in the MOS-SF20 are “Has your health limited you in strenuous activities, like running or lifting heavy objects?” “. . . in walking uphill or climbing a few flights of stairs?” “. . . in bending, lifting or stooping?” and “. . . in walking one block?” The response categories were no or yes.

Five hundred seventy-five persons (76.4%) of the cohort completed all three interviews. The first interview took place a few weeks after the cross-sectional study (Time 1); the other two 1 (Time 2) and 2 years (Time 3) later. Attrition was due to mortality ($n = 58$; 7.7%), very poor health ($n = 66$; 8.8%), and refusal ($n = 54$; 7.2%). The cohort con-

sisted, at Time 1, of 544 women (M age = 73, SD = 7.6 at Time 1) and 209 men (M age = 71, SD = 8.7). Four hundred forty-four participants were younger than 75 years of age (but at least 57), and 311 were older than 75.

Measures

IADL/ADL disability.—This was assessed at each wave with the Groningen Activity Restriction Scale (GARS), a well-established reliable and valid measure of IADL/ADL disability, with a clear one-dimensional structure that implies that the ADLs and IADLs included in the measure lie on the same underlying continuous dimension (Kempen, Steverink, et al., 1996; Kempen, Miedema, Ormel, & Molenaar, 1996). The GARS comprises 18 (instrumental) ADL items. Examples of GARS items are “Can you, fully independently, dress yourself?” “. . . stand up from a chair?” “. . . go up and down the stairs?” “. . . prepare dinner?” “. . . get in and out of a car?” “. . . do the grocery shopping?” and “. . . take a bath or shower?” Each item has four response categories (1 = yes, I can do that easily and without help; 2 = yes, I can do that without help but it takes some effort; 3 = yes, I can do that without help but it takes a lot of effort; 4 = no, I can not do that without help). The GARS scores range from 18 (no disability) to 72 (maximum disability). Internal consistency (Cronbach’s α) ranged from .90 at Time 1 to .92 and .93 at Times 2 and 3, respectively. The observed GARS scores were logarithmically transformed ($5 \cdot \ln[x]$) to adjust for non-normal distributions. The resulting range for the transformed GARS scores was 14.45 to 21.38.

Depressive symptoms.—Depressive symptoms were measured at each wave with the Depression subscale of the Hospital Anxiety and Depression Scale (HADS; Spinhoven et al., 1997; Zigmond & Snaith, 1983). The seven items target the affective and cognitive aspects of depression. The HADS does not contain explicit somatic items, and consequently the HADS is less sensitive to confounding by physical disease. Examples are “I have lost interest in my appearance,” “I look forward with enjoyment to things,” and “I feel as if I am slowed down.” Each item has four response categories. Scale values range from 0 to 21; higher scores indicate more symptoms. Internal consistency, as indicated by Cronbach’s alpha, ranged from .71 at Time 1 to .80 and .81 at Times 2 and 3, respectively. Validity of the HADS in elderly and in nonelderly but physically sick people is well established (Silverstone, 1991; Spinhoven et al., 1997; Zigmond & Snaith, 1983).

Dichotomous versions.—The continuous disability and depression measures were used for all analyses except the description of transitions (Table 1) where the variables were dichotomized. For depressive symptoms we used the conventional cut-off of 8 (coded: 0 is 7 or less, the nondepressed group; 1 is 8 or more, the depressed group). In most population studies 15% to 20% of middle-aged and older people have scores of 8 or more (Ormel et al., 1997; Spinhoven et al., 1997). For IADL/ADL disability, the GARS, no conventional cut-off exists. Therefore we choose the cut-off of 29 on the nontransformed GARS scale, as this cut-off

Table 1. Intraindividual Change and Stability in Depression and Disability Status (Dichotomized Variables) per Pair of Waves for Men and Women Separately

Course Patterns (Transition Status)	Depression T1–T2		Depression T2–T3		Depression T1–T3	
	Men	Women	Men	Women	Men	Women
Depression						
00–Persistently non-depressed	49	50	54	50	49	50
01–Onset of depression	17	17	11	16	17	19
10–Remission of depression	12	13	6	13	12	11
11–Persistently depressed	22	20	28	22	22	20
Disability						
00–Persistently non-disabled	31	33	36	35	32	32
01–Onset of disability	8	11	10	8	12	14
10–Remission of disability	9	7	4	8	8	9
11–Persistently disabled	51	49	50	49	48	45

Note: T1 = Wave 1; T2 = Wave 2.

score corresponds with the 85th percentile of the GARS in a large random population sample of older people (Kempen, Steverink, et al., 1996; 0 is less than 29, the non-disabled group; 1 is 29 or more, the disabled group).

Description of the Full Model

The model depicted in Figure 2 consists of three parts: two identical trait and state (T&S) models (Duncan-Jones, Fergusson, Ormel, & Horwood, 1990; Eid, Notz, Steyer, & Schwenkmezger, 1994; Kenny & Campbell, 1989; Ormel & Schaufeli, 1991) for three time points, one addressing disability (top of figure) and one depressive symptoms (bottom of figure), and two correlations (Paths c and f) and four regression effects (Paths a, b, d, and e) linking the two T&S models. The T&S disability model assumes that the participant’s disability level at each time point is the function of two latent (unobserved) variables: a trait component (common factor) and a state component. The state component represents the variance that is not accounted for by the trait factor and hence reflects change within-subject over the 2-year study period, in part as a result of measurement error. The T&S depression model makes the same assumptions.

The across-time structure of the latent state disability variable (State 1, State 2, State 3) in the T&S model was modeled as a first-order auto-regressive model. State 2 and 3 variances thus consist of variance transmitted from an earlier time point (Paths p and q) and innovation (or new) variance (Paths t2 and t3) resulting from the effects of unobserved change agents to which the person has been exposed during the interval. The across-time structure of the latent state depression variable was modeled in the same way (Paths r and s indicating transmitted variance and u2 and u3 innovation variance).

By linking the T&S models for depression and disability, a combined model is obtained (depicted in Figure 2) in which the latent state variables of depression can act as a change agent of disability and, vice versa, the cross-variable effects. These effects can be rather instantaneous (Paths d and e) and/or more lagged (Paths a and b). In addition, the model allows correlation between the two trait factors (Path f)

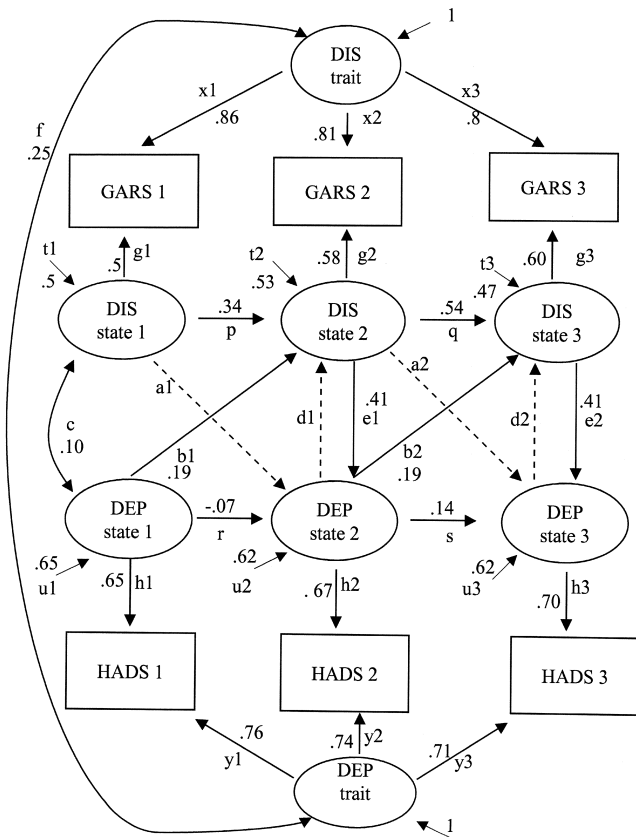


Figure 2. Path diagram of the full model of disability (DIS) and depressive symptoms (DEP), each modelled as a Trait-State model, and contemporaneous (d,e) and 1-year lagged (a,b) cross-variable effects linking their state components (state) and correlation between the trait factors (f) and between the state components at time 1 (c). Included are also the standardized estimates of the best fitting model (#10). Note that the following equality constraints were applied to identify model equations: $x1 = x2 = x3; y1 = y2 = y3; a1 = a2; b1 = b2; d1 = d2; e1 = e2$. Note that the paths without a standardized estimate (broken lines) could be fixed to zero (e.g., a1, a2, d1, and d2). GARS = Groningen Activity Restriction Scale; HADS = Hospital Anxiety and Depression Scale.

and between the first state component of disability and depression (Path c).

Model Specification and Identification

To solve the structural equations of the full model, we found it was necessary to make the following assumptions for both the depression and disability T&S parts of the model: (a) The regressions of the observed depression and disability scores on their respective latent trait factor are equal over time (equality constraints: $x1 = x2 = x3; y1 = y2 = y3$) and (b) the contemporaneous and lagged cross-variable effects at Time t equal those at time T + 1 ($a1 = a2, b1 = b2, e1 = e2, d1 = d2$). These are reasonable assumptions (Duncan-Jones et al., 1990; Ormel & Schaufeli, 1991).

We tested whether the auto-regression effect (transmitted variance) for the 1st year (T1–T2) could be set equal to the one for the 2nd year (T2–T3) but they could not; $p = q$:

$\Delta\chi^2(1, N = 753) = 13.5, p < .001$, and $r = s: \Delta\chi^2(1, N = 753) = 7.3, p < .05$, thus they were not constrained.

The full model, depicted in Figure 2, requires the estimation of 18 parameters (variance of the six latent state variables was fixed at unity). Hence the full model has 3 degrees of freedom left. The full model is identified. Very different starting values gave the same solution. To allow readers to interpret the model more easily, we provide standardized estimates (the unstandardized estimates can be obtained on request). Standardized estimates, or path coefficients, have a theoretical range from zero (no effect) to ± 1.0 (maximum positive or negative effect). Their squared value indicates the proportion of variance they account for.

Statistics and Model Fitting

Descriptive statistics as well as model fitting were accomplished using the structural equation modeling program Mx (Neale, 1995). A more detailed account can be obtained on request. Participants who did not take the T2 or T3 follow-up interview were included in the analyses. In the saturated model the expected covariance matrix of the six observed variables is estimated with the maximum number of parameters (six variances and 15 covariances). The degrees of freedom for the chi-square statistic is equal to the difference in degrees of freedom of the two models. Because the series of competing models is nested (i.e., all of one model’s free parameters are a subset of the other model’s free parameters), chi-square-difference tests can be performed to compare the fit of competing models.

Information about the precision of parameter estimates (and their explained variance) in Mx were obtained by likelihood-based confidence intervals (CIs) rather than standard errors. In this method a parameter is progressively moved away from its maximum likelihood estimate in either direction (while the other model parameters are optimized) until the difference in fit, distributed as chi-square with one degree of freedom, is significant. For 95% CI the .05 level of significance is ≈ 3.84 in each direction.

We adopted two model fitting strategies, *forward* and *backward* fitting. The backward strategy, the most accurate of the two, started with the full model (depicted in Figure 2) and then proceeded by dropping, one by one, the paths linking disability and depression that did not differ significantly from zero ($p > .05$). The forward strategy started with the null model (no. 2 in the final table), which did not include any link between the depression and disability state variables. In a systematic way paths were allowed (see the final table). Because the models are nested, they could be compared in terms of fit ($\Delta\chi^2$ statistic and Δdf) mutually as well as against the saturated model (no. 1 in the final table).

RESULTS

Intraindividual Transitions in Disability and Depression Status

Averaged across waves, approximately 37% of the men and 36% of the women were classified as depressed. This is about twice the prevalence of 17% found in the source population. With the cut-off of 29 on the GARS, approximately 60% of the men and 57% of the women were classified as

IADL/ADL disabled. The dichotomized variables were used to construct four course patterns for each of the following periods: T1 to T2, T2 to T3, and T1 to T3. The four course (or transition) patterns were labeled *persistently nondepressed* (disabled), *onset*, *remission*, and *persistently depressed* (disabled). Table 1 presents the results.

Depression status changed slightly more often than disability status. Both onset and remission occurred, albeit onset was more frequent than remission. The most prevalent course patterns were persistently nondepressed and persistently disabled. Because normative data on change and stability are lacking, it is unclear whether the observed change in our cohort differs from what would have been found in a random population sample of older people.

We also examined transition patterns across all three occasions simultaneously. The results for women regarding depression were 43% persistently non-depressed, 13% persistently depressed, 17% onset, 13% remission, and 15% who changed depression status twice. Regarding disability, 30% were persistently non-disabled, 40% were persistently disabled, 12% were classified as onset, 8% as remission, and 10% changed disability status twice. The results for men were very similar.

Change Over Time in Mean Disability and Depression Scores

Mean disability levels did differ between waves, and so did mean depression levels (see Table 2, *Ms* column). There was a significant increase in mean disability: $\chi^2(1, N = 753) = 40.6, p < .001$, and in mean depression, $\chi^2(1, N = 753) = 18.1, p < .001$, during the 2-year interval in the interviewed cohort.

Gender, Age, and Aging Differences

If the longitudinal correlations depend on gender and age, the invariance of the model has to be examined. Therefore, we examined gender and age effects. There were no significant gender differences in means and variances of depression, $\Delta\chi^2(6, N = 753) = 3.10, p = .79$, and disability, $\Delta\chi^2(6, N = 753) = 7.37, p = .29$, across the three waves, but there were gender differences in the longitudinal correlations. Half of the across-variable, across-time correlations were slightly stronger among men.

Two age groups were constructed: the young old (age <75

at Time 1, $n = 442, M$ age = 67.68, $SD = 4.9$) and the old old (age $\geq 75, n = 311, M$ age = 80.85, $SD = 4.12$). Age was not correlated with depression scores at any time point but, as expected, was associated with disability. Disability levels in the old old group at any wave were significantly higher than in the young old group. The (co)variance matrices of the two age groups did not differ, variances: $\Delta\chi^2(6, N = 753) = 9.03, p = .17$; covariances: $\Delta\chi^2(15, N = 753) = 17.26, p = .30$.

Tests for Selective Attrition

We examined whether sample attrition had introduced response bias. Compared with completers, dropouts at Time 2 were older (72.29 vs. 74.31), $\Delta\chi^2(1, N = 753) = 7.1, p < .01$, more disabled (17.02 vs. 17.74), $\Delta\chi^2(1, N = 753) = 22.3, p < .001$, and more depressed (6.02 vs. 7.20), $\Delta\chi^2(1, N = 753) = 8.92, p < .001$. Compared with completers, dropouts at Time 3 (including dropouts at Time 2) were older (72.29 vs. 75.29), $\Delta\chi^2(1, N = 753) = 17.3, p < .001$, more disabled (17.02 vs. 17.89), $\Delta\chi^2(1, N = 753) = 45.57, p < .001$, and more depressed (6.02 vs. 7.06), $\Delta\chi^2(1, N = 753) = 8.68, p < .001$. Variances and covariance of Wave 1 depression and disability scores, however, did not differ significantly between completers and dropouts at Time 2, $\Delta\chi^2(3, N = 753) = 1.86, p < .64$, and Time 3, $\Delta\chi^2(3, N = 753) = 4.9, p < .18$.

Estimated Variances and Correlations

Table 2 shows the estimates of the variance and correlation (below diagonal) with their 95% CIs of the disability and depression scores as obtained with the saturated model (covariances can be obtained on request). The longitudinal correlations suggest considerable stability of individual differences over time in disability and depressive symptoms, and hence match the results presented in Table 1 on within-subject change in depression and disability status. The across-variable correlations between disability and depressive symptoms are moderately strong and tend to become smaller the longer the interval measurements.

Model Fitting

Table 3 presents the standardized parameter estimates of the full model with which the backward-fitting strategy

Table 2. Maximum Likelihood Estimates of the Variances and Correlations of Depression and Disability Scores

Wave	Depression			Disability			<i>M</i>	<i>n</i>
	Wave 1	Wave 2	Wave 3	Wave 1	Wave 2	Wave 3		
Depression								
Wave 1	14.861						6.16	753
Wave 2	.570 (.52-.62)	15.735					6.68	629
Wave 3	.558 (.50-.61)	.667 (.62-.71)	17.041				6.82	574
Disability ^a								
Wave 1	.194 (.12-.26)	.213 (.14-.29)	.191 (.11-.27)	2.236			17.21	753
Wave 2	.241 (.17-.31)	.320 (.25-.39)	.276 (.20-.35)	.807 (.78-.83)	2.535		17.37	631
Wave 3	.183 (.11-.26)	.306 (.23-.38)	.325 (.25-.40)	.757 (.72-.79)	.864 (.84-.88)	2.632	17.50	575

Notes: Maximum likelihood estimates appear on the diagonal, correlations appear below the diagonal, and 95% confidence intervals appear in parentheses.

^aTransformed scores.

Table 3. Standardized Parameter Estimates of the Full Model

Path	β
Trait Dis to GARS1 (x1)	.865
Trait Dis to GARS2 (x2)	.813
Trait Dis to GARS3 (x3)	.799
Trait Dep to HADS1 (y1)	.756
Trait Dep to HADS2 (y2)	.738
Trait Dep to HADS3 (y3)	.705
State Dep 1 (u1) to HADS1 (h1)	.656
State Dep 2 (u2) to HADS2 (h2)	.67
State Dep 3 (u3) to HADS3 (h3)	.71
State Dis 1 (t1) to GARS1 (g1)	.5
State Dis 2 (t2) to GARS2 (g2)	.58
State Dis 3 (t3) to GARS3 (g3)	.6
State Dis 1 to State Dis 2 (p)	.355
State Dis 2 to State Dis 3 (q)	.581
State Dep 1 to State Dep 2 (r)	-.113
State Dep 2 to State Dep 3 (s)	.089
State Dis 1(2) to State Dep 2(3) (a1/a2)	-.066
State Dep 1(2) to State Dis 2(3) (b1/b2)	.215
State Dep 2(3) to State Dis 2(3) (d1/d2)	-.172
State Dis 2(3) to State Dep 2(3) (e1/e2)	.599
Correlation between State Dis 1 and State Dep 1 (c)	.11
Inn State Dep 1	.656
Inn State Dep 2	.68
Inn State Dep 3	.71

Notes: $\Delta\chi^2(3, N = 753) = 1.01, p = .798$. DIS = disability; GARS = Groningen Activity Restriction Scale; Dep = depression; HADS = Hospital Anxiety and Depression Scale; Inn = innovation variance.

started. Four cross-variable parameters in the full model did not differ significantly from 0 (a1, a2, d1, d2). They could be dropped without a significant loss of fit, $\Delta\chi^2(2, N = 753) = 0.18$. The results of the forward-fitting strategy are presented in Table 4. Although four models (10, 13, 14, and 15)

Table 4. Model Fitting Results of the Forward Fitting Procedure

Model	$\Delta\chi^2$	Δdf	p
1. Saturated model*	—		
2. Parameters a, b, d, e, and c fixed to zero. Estimation of all other parameters.	68.35	8	<.001
3. Parameters a, b, d, and e fixed to zero. Estimation of all other parameters	65.99	7	<.001
4. As model 3 but parameter a estimated	63.09	6	<.001
5. As model 3 but parameter b estimated	65.19	6	<.001
6. As model 3 but parameter d estimated	19.09	6	.004
7. As model 3 but c parameter e estimated	21.19	6	.002
8. As model 3 but a and e estimated	20.91	5	<.001
9. As model 3 but a and d estimated	15.91	5	.007
10. As model 3 but b and e estimated	1.19	5	.95
11. As model 3 but b and d estimated	12.4	5	.03
12. As model 3 but e and d estimated	18.48	5	.002
13. As model 3 but e, d, and a estimated	6.28	4	.18
14. As model 3 but e, d, and b estimated	.80	4	.94
15. As model 3 but e, d, and a, b estimated	1.01	3	.798

Notes: The χ^2 values and df refer to the difference with the χ^2 value and df of the saturated model; $-2LL = -2$ the log = likelihood of the observed data, a = paths State Dis 1(2) to State Dep 2(3); b = paths State Dep 1(2) to State Dis 2(3); d = paths State Dep 2(3) to State Dis 2(3); e = paths State Dis 2(3) to State Dep 2(3); c = correlation between State Dis 1 and State Dep 1; * $-2LL = 16028.51, df = 3888$.

cannot be rejected (p values associated with their χ^2 are $>.05$), Model 10 (with a1, a2, d1, and d2 fixed at zero), $\Delta\chi^2(5, N = 753) = 1.19, p = .95$, fits significantly better than the other three, as it is more parsimonious (5 vs. 4 or 3 df). The trait correlation can not be omitted from Model 10, $\Delta\chi^2(1, N = 753) = 8.67, p = .001$. Thus, forward and backward fitting strategies yielded the same best fitting model, Model 10, presented in Figure 2.

Invariance of Best Fitting Model Across Gender and Age

Model 10 also fit well in the subgroups of young old and old old and in men and women; young old: $\Delta\chi^2(5, N = 753) = 2.47, p = .78$; old old: $\Delta\chi^2(5, N = 753) = 2.82, p = .73$; women: $\Delta\chi^2(5, N = 753) = 4.3, p = .51$; men: $\Delta\chi^2(5, N = 753) = 9.2, p = .10$. Two-group model analyses did not reveal significant differences in the parameters linking the state depression and state disability variables between men and women, $\Delta\chi^2(6, N = 753) = 9.83, p = .13$, and between the two age groups, $\Delta\chi^2(6, N = 753) = 11.2, p = .08$. Thus, Model 10 is invariant across gender and age in this cohort of physically limited elderly people.

Best Fitting Model: Standardized Estimates and Interpretation

The standardized parameter estimates of Model 10 in Figure 2 represent path coefficients and may be interpreted as follows:

1. The estimated trait variance in the disability scores ranged from 75% (.86²) at Time 1 to 66% and 64% (.8²) at Times 2 and 3. This suggests that, in initially physically limited elderly adults, two thirds to three quarters of the between-subject differences in IADL/ADL disability are stable across a 2-year period. Correspondingly, the amount of state variance in disability scores (the proportion that is unique to each occasion) ranged from 25% at Time 1 to 34% and 36% at Times 2 and 3, respectively. However, the state variance contains both true and measurement error variance. Adjusting for measurement error variance in the observed GARS disability scores (estimated at 7%–10% on the basis of the Cronbach’s α , which ranged from .90 to .93 for the GARS, and which provides an estimate of the proportion of true variance in the GARS measure), approximately 15% to 29% of the observed variance in disability scores reflects true change over the study period.
2. Fifty-eight percent of the variance in the depression scores at Time 1 (.76²) and 55% and 50% at Times 2 and 3, respectively, could be accounted for by the trait depression factor. Thus, half to three fifths of the between-subject differences in depression scores was stable across a 2-year period. Correspondingly, 42% of the variance at Time 1 to 45% and 50% at Times 2 and 3, respectively, reflects state variance. Because the measurement error variance of the depression scores ranged from 29% at Time 1 to 19% at Time 3 according to Cronbach’s alpha, the amount of true change variance in depression scores ranges from 13% to 31%. Hence, individual differences in depressive symptoms are slightly less stable than are those in disability.

3. The standardized estimates of the cross-variable effects show that these were small to moderate. Change in state disability had a moderately strong contemporaneous effect of .41 on depressive symptoms, but no 1-year lagged effect. Changes in disability appear to quickly affect depressive symptoms. On the other hand, change in depressive symptom level did not have a significant immediate contemporaneous influence on disability, but it had weak 1-year lagged effect of .19. This suggests that, with some delay, an increase or decrease in depressive symptoms will produce a parallel effect on IADL/ADL disability.
4. To put these cross-variable effects into perspective, we need to adjust them for measurement error. Unadjusted 16% (.409²) of the change variance in depressive symptoms is accounted for by change in disability, but adjusted 26% to 50%, that is, a quarter to half of the true change in depressive symptom level, is due to change in disability. For the lagged effect of depressive symptoms on disability, the corresponding percentages are 3% (unadjusted) and 5% (adjusted).
5. Trait depression and trait disability are weakly correlated ($r = .25$).

DISCUSSION

This study is the first to suggest that the association between disability in IADL/ADLs and depressive symptoms in noninstitutionalized older people with significant initial physical limitations can be attributed to at least three processes: first, a contemporaneous effect of change in disability on depressive symptoms; second, a lagged effect of change in depression on disability; and third, a correlation between the trait components of depressive symptoms and disability. Although a substantial proportion of the true change variance in depressive symptoms could be accounted for by change in disability, only a minority of the true change variance in disability was due to a change in depressive symptoms. This positive feedback cycle was invariant across gender and between the young old and old old. Our results are consistent with Aneshensel and colleagues' (1984) four-wave study of a largely young and middle-aged population. They found that physical health (number of illnesses and disability days) produced effects rather quickly and strongly on depressive symptoms. Depressive symptoms had a delayed and weaker effect on physical health.

Because other studies have rarely examined reciprocal effects between depression and disability, but focused instead on one of the two as outcome, two separate bodies of knowledge have accumulated, one documenting effects of depression on disability and one showing effects of disability on depression. Our results connect the evidence, supporting both positions and integrating them in terms of a feedback cycle.

Limitations

The most obvious limitation is that our longitudinal study was limited to older people with initial physical limitations. Although the source population from which they were recruited was representative of the noninstitutionalized population who were aged 57 and older and living in the north of The Netherlands, the longitudinal cohort was selective for those with some disability. The underlying assumption for

this selection of physically limited elderly adults was that they might experience more change in health status in a 2-year period than physically healthy persons do. The selective nature of our cohort limits the generalizability of the findings and stresses the need for replication in other samples. Our study might not apply to a first lifetime onset of either IADL/ADL disability or depression.

The importance of this limitation may be limited for the following reasons. First, there is the remarkable similarity between the findings of Aneshensel and colleagues (1984) in a younger but unselected sample and our results. Second, individual levels of depressive symptoms and IADL/ADL disability are not fixed but dynamic. As we have shown, there was substantial intraindividual change across the two 1-year intervals. Third, it is reasonable to assume that, prior to any study enrollment, most people will have experienced earlier temporary episodes of physical disability and/or depressive symptoms because of the self-limiting nature of most acute physical diseases, accidents, and depressogenic events. So it will be hard to find people who never experienced an episode of disability. Fourth, it is very difficult to study potential reciprocal effects in a healthy population, because onset of persistent disability will be rare and require long-term and frequent monitoring to detect. Furthermore it is uncommon that an onset of depression in physically healthy persons triggers, within a short period of time, the positive feedback process by causing an onset of physical illness and associated disability. As stated at the beginning of this article, we think (a) that the physically vulnerable—that is, people with one or more chronic medical conditions—will be most sensitive to the development of reciprocal effects and (b) that in physically healthy persons the indirect effects of depression on disability (through onset of physical health problems) will require persistent depressive symptoms and have a long brought forward time. Hence, the optimal population to study reciprocal effects could well be a “high-risk” population of people with chronic physical health problems and/or physical limitations.

A second limitation of our study is sample attrition. During the 2-year period we had a 23.6% attrition rate, largely as a result of mortality and very poor health. Attrition was associated with age, disability, and depressive symptoms. However, Wave 1 variances and covariances did not differ between completers and dropouts at Waves 2 and 3. We think, as argued below, that the selective attrition may have deflated the estimates of the reciprocal effects by effectively removing from the study cohort those with the fastest downward spiral.

Another limitation is that potential confounders other than gender and age were not examined. In particular the lack of a measure of biomedical severity of physical ill health is unfortunate, as this would have allowed us to examine its role as a potential mediator of the delayed effect of depression on disability.

Gender Differences

Unexpectedly, mean depression level and prevalence of probably clinically significant depression were not significantly higher in women compared with men, whereas they were in the source population from which our cohort was

recruited. It should be noted, however, that the gender difference in the source population was small and even reversed in the oldest age group (men: age 57–65 = 12%, 66–75 = 15%, 76–85 = 22%, 86 and older = 36%; women: age 57–65 = 14%, 66–75 = 20%, 76–85 = 24%, 86 and older = 25%).

We think that the difference in gender ratio between cohort and source population is, in a complex way, due to selection effects, described here.

1. Physical limitations and depressive symptoms were correlated in the source population (Pearson $r = 0.36$). Consequently, the selection on physical limitations also selected for higher levels of depressive symptoms. This accounts for the higher prevalence of depressed people in the cohort (35%) compared with the source population (17%).
2. The prevalence of persons with physical limitations in the source population is higher among women than among men. Because we applied the same threshold on physical limitations for both genders, relatively more women (19%) than men (15%) were enrolled from the source population into the cohort. This accounts for the difference in male–female ratio between the source population, 56% women versus 72% in the cohort.
3. Combined, these factors may have resulted in the disappearance of the gender difference in depression level from source population to cohort. This selection effect should not have biased model fitting and estimation of the across-variable and across-time relationships.

Whereas some of the across-variable, across-time correlations were slightly larger among men, gender-specific model fitting did not reveal statistically significant gender differences in the across-variable, across-time effects. Apparently, the gender differences in correlations were too small to affect the gender-specific model estimates in a significant way. The slightly larger correlations in men, however, seem consistent with literature on the greater impact of loss of important aspects of life routine of older men as compared with older women.

The Lack of a Contemporaneous Effect of Depressive Symptoms on IADL/ADL Disability

Change in depressive symptoms had a delayed but not a contemporaneous effect on disability. A delayed effect is consistent with the hypothesis that a change in depressive symptoms has an indirect effect on disability through a time-taking influence on physical health status. In addition, we had expected that change in depressive symptoms would have a direct effect because of the disabling effects of depressive symptoms on cognitive and motivational capacities and affect regulation. Could this be due to measurement issues or sample characteristics? We measured depressive symptoms, not the clinical syndrome of major depression. To avoid confounding with physical disease, a serious problem in the elderly population, we choose the HADS, which does not include physical symptoms of depression such as weight change and fatigue. It is possible that a contemporaneous effect would have been found if major depression had been measured instead of HADS depressive symptoms. Re-

garding disability, we used the GARS, which measures the extent to which people can do specific IADL/ADLs, not overall physical or social functioning. It is possible that change in depression status does have a quick influence on self-reported, overall physical and/or social functioning, but not on IADL/ADLs like bathing, getting in and out of bed, getting in and out of a chair, going up and down the stairs, cooking, shopping, cleaning the house, and so forth. The higher prevalence of depressive symptoms and physical limitations in the cohort as compared with the source population should not have obscured a contemporaneous effect of depression on disability.

Stability and Change in Depressive Symptoms and IADL/ADL Disability

We modeled the longitudinal associations of depressive symptoms and those of IADL/ADL disability according to the state–trait model, which combines a common factor with a first-order auto-regression component. Such a model has several advantages compared with a pure auto-regression model (Kenny & Campbell, 1989; Ormel & Schaufeli, 1991) as used by Aneshensel and colleagues (1984). The most important advantage is that modeling stability of individual differences as resulting exclusively from auto-regression is difficult to interpret. It is much more plausible to assume—as the trait–state model does—that differential stability reflects ongoing influences from (relatively) stable person and/or environmental characteristics as well as carry-over effects due to a certain “inertia” of depression and disability. The state–trait model makes the modeling of unrealistic higher-order auto-regression effects unnecessary. The fitted model showed that individual differences in depressive symptoms and IADL/ADL disability are to a large extent stable across a 2-year period in an already physically limited sample of older people.

Depression Is More Prevalent Among the Disabled but Still Not a Common Result

Thirty-five percent of this cohort of older people with significant disabilities had an initial total depression score of 8 or above, a cut-off score often used to indicate probable clinically significant depression. Whereas this doubling of the 17% rate observed in the source population indicates the relevance of disability to depression, it clearly shows that depressive illness is not an inevitable result of disability.

If a Positive Feedback Cycle Exists, Why Do Mean Levels Not Increase More Rapidly With Aging?

A positive feedback cycle between IADL/ADL disability and depressive symptoms may propel an upward spiral of increasing levels of disability and depressive symptoms with the passage of time. Although we found an increase in mean disability and depressive symptoms in the surviving cohort during the 2-year study period, the increase was modest (less than 0.10 *SD* unit per year for disability). For various reasons a positive feedback cycle might not raise mean levels too quickly in an elderly cohort during a few years. First, death and nonresponse due to being very sick effectively remove those with high levels of poor physical and/or mental health. Major depression is associated with

increased and premature mortality (e.g., Penninx et al., 1999). Second, those who experience a significant deterioration in health are more likely to obtain medical care and rehabilitation than are those with stable health, which may in part neutralize the deterioration. In addition, developmental processes in elderly adults may make them less prone to depression when disability has become an expected part of aging (Sullivan, 1997).

Policy Significance

The existence of this feedback cycle suggests mutual reinforcement over time of depression and disability for disabled elderly adults. Our findings suggest that the most immediate and strongest benefit to this population will be obtained by reducing IADL/ADL disability, because the effect of IADL/ADL disability on depression is faster and stronger than the 1-year lagged effect of depression on IADL/ADL status. Efforts to minimize depressive symptoms and improve mental health are also likely to be cost-effective compared with other interventions, because depression may be more reversible than the disability associated with chronic and degenerative disease. As reported by Tinetti, Inouye, Gill, & Doucette (1995), reduction of risk factors that are shared by a variety of geriatric syndromes, among which is depression, may help to restore functional independence. Interventions to reduce disability and depression in elders may be among the most cost-effective means to increase the quality of life in the aging population. What appears particularly important is to watch for depression when disability is new, and to watch for disability when depression is persistent.

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REFERENCES

- Alexopoulos, G. S., Meyers, B. S., Young, R. C., Kakuma, T., Silbersweig, D., & Charlson, M. (1997). Clinically defined vascular depression. *American Journal of Psychiatry*, *154*, 562–565.
- Aneshensel, C. S., Frerichs, R. R., & Huba, G. J. (1984). Depression and physical illness: A multiwave, nonrecursive causal model. *Journal of Health and Social Behavior*, *25*, 350–371.
- Armenian, H. K., Pratt, L. A., Gallo, J., & Eaton, W. W. (1998). Psychopathology as a predictor of disability: A population-based follow-up study in Baltimore, Maryland. *American Journal of Epidemiology*, *148*, 269–275.
- Baldwin, R. C., & Tomenson, B. (1995). Depression in later life. A comparison of symptoms and risk factors in early and late onset cases. *British Journal of Psychiatry*, *167*, 649–652.
- Berkman, L. F., Berkman, C. S., Kasl, S., Freeman, D. H., Leo, L., Ostfeld, A. M., et al. (1986). Depressive symptoms in relation to physical health and functioning in the elderly. *American Journal of Epidemiology*, *124*, 372–388.
- Brilman, E. I., & Ormel, J. (2001). Life events, difficulties, and onset of depressive episodes in later life. *Psychological Medicine*, *31*, 859–869.
- Bruce, M. L., Seeman, T. E., Merrill, S. S., & Blazer, D. G. (1994). The impact of depressive symptomatology on physical disability: MacArthur Studies of Successful Aging. *American Journal of Public Health*, *84*, 1796–1799.
- Coulehan, J. L., Schulberg, H. C., Block, M. R., Madonia, M. J., & Rodriguez, E. (1997). Treating depressed primary care patients improves their physical, mental, and social functioning. *Archives of Internal Medicine*, *157*, 1113–1120.
- Duncan-Jones, P., Fergusson, D., Ormel, J., & Horwood, L. (1990). A model of stability and change in minor psychiatric symptoms: Results from three longitudinal studies. *Psychological Medicine* (Monograph Suppl. No. 18). Cambridge, England: Cambridge University Press.
- Eid, M., Notz, P., Steyer, R., & Schwenkmezger, P. (1994). Validating scales for the assessment of mood level and variability by latent state-trait analyses. *Personal and Individual Differences*, *16*, 63–76.
- Kempen, G. I. J. M., Miedema, I., Ormel, J., & Molenaar, W. (1996). The assessment of disability with the Groningen Activity Restriction Scale: Conceptual framework and psychometric properties. *Social Science and Medicine*, *43*, 1601–1610.
- Kempen, G. I. J. M., Ormel, J., Brilman, E. I., & Relyveld, J. (1997). Adaptive responses among Dutch elderly: The impact of eight chronic conditions on health-related quality of life. *American Journal of Public Health*, *87*, 38–44.
- Kempen, G. I. J. M., Steverink, N., Ormel, J., & Deeg, D. J. H. (1996). The assessment of ADL among frail elderly in an interview survey: Self-report versus performance-based tests and determinants of discrepancies. *Journal of Gerontology: Psychological Sciences*, *51B*, P254–P260.
- Kennedy, G. L., Klerman, H. R., & Thomas, C. (1990). The emergence of depressive symptoms in late life: The importance of declining health and increasing disability. *Journal of Community Health*, *15*, 93–104.
- Kenny, D. A., & Campbell, D. T. (1989). On the measurement of stability in over-time data. *Journal of Personality*, *57*, 445–481.
- Krishnan, K. R., & Gadde, K. M. (1996). The pathophysiologic basis for later-life depression: Imaging studies of the aging brain. *American Journal of Geriatric Psychiatry*, *4*, S22–S33.
- Mintz, J., Mintz, L. I., Arruda, M. J., & Hwang, S. S. (1992). Treatments of depression and the functional capacity to work. *Archives of General Psychiatry*, *49*, 761–768.
- Mynors-Wallis, L., Gath, D. H., Lloyd-Thomas, A. R., & Tomlinson, D. (1995). Randomised controlled trial comparing problem solving treatment with amitriptyline and placebo for major depression in primary care. *British Medical Journal*, *310*, 441–445.
- Neale, M. C. (1995). *Mx: Statistical modeling*. Available from M. C. Neale, Box 710, Department of Psychiatry, MCV, Richmond, VA 23298.
- Ormel, J., Kempen, G. I. J. M., Deeg, D. J. H., Brilman, E. I., van Sonderen, E., & Relyveld, J. (1998). Functioning, well-being, and health perception in late middle-aged and older people: Comparing the effects of depressive symptoms and chronic medical conditions. *Journal of the American Geriatrics Society*, *46*, 39–48.
- Ormel, J., Kempen, G. I. J. M., Penninx, B. W. J. H., Brilman, E. I., Beekman, A. T. F., & van Sonderen, E. (1997). Chronic medical conditions and mental health in older people: Disability and psychosocial resources mediate specific mental health effects. *Psychological Medicine*, *27*, 1065–1077.
- Ormel, J., & Schaufeli, W. (1991). Stability and change of psychological distress and their relationship with self-esteem and locus of control. *Journal of Personality and Social Psychology*, *60*, 288–299.
- Ormel, J., & VonKorff, M. (2000). Synchrony of change in depression and disability. What next? *Archives of General Psychiatry*, *57*, 381–382.
- Ormel, J., VonKorff, M., Oldehinkel, A. J., Simon, G., Tiemens, B. G., & Üstün, T. B. (1999). Onset of disability in depressed and non-depressed primary care patients. *Psychological Medicine*, *29*, 847–853.
- Ormel, J., VonKorff, M., Üstün, T. B., Pini, S., Korten, A., & Oldehinkel, A. J. (1994). Common mental disorders and disability across cultures: Results from the WHO collaborative primary care study. *Journal of American Medical Association*, *272*, 1741–1748.
- Penninx, B. W. J. H., Geerlings, S. W., Deeg, D. J., van Eijk, J. T. M., van Tilburg, W., & Beekman, A. T. F. (1999). Do minor and major depres-

- sion increase the risk of dying in older persons? *Archives of General Psychiatry*, 56, 889–895.
- Penninx, B. W. J. H., Guralnik, J. M., Ferrucci, L., Simonsick, E. M., Deeg, D. J. H., & Wallace, R. B. (1998). Depressive symptoms and physical decline in community-dwelling older persons. *Journal of American Medical Association*, 297, 1720–1726.
- Phifer, J. F. (1986). Etiologic factors in the onset of depressive symptoms in older adults. *Journal of Abnormal Psychology*, 95, 282–291.
- Prince, M. J., Harwood, R. H., Thomas, A., & Mann, A. H. (1998). A prospective population-based cohort study of the effects of disablement and social milieu on the onset and maintenance of late-life depression. The Gospel Oak Project VII. *Psychological Medicine*, 28, 337–350.
- Silverstone, P. H. (1991). Is anhedonia a good measure of depression? *Acta Psychiatrica Scandinavica*, 83, 249–250.
- Spinhoven, P., Ormel, J., Sloekers, P. P. A., Kempen, G. I. J. M., Speckens, A. E. M., & VanHemert, A. M. (1997). A validation study of the Hospital Anxiety and Depression Scale (HADS) in different groups of Dutch subjects. *Psychological Medicine*, 27, 363–370.
- Stewart, A. L., Hays, R. D., & Ware, J. E., Jr. (1988). The MOS Short-Form general health survey. *Medical Care*, 26, 724–732.
- Sullivan, M. D. (1997). Maintaining good morale in old age. *Western Journal of Medicine*, 167, 276–284.
- Tiemens, B. G., Ormel, J., Jenner, J. A., van der Meer, K., van Os, T. W. D. P., van den Brink, R. H. S., et al. (1999). Training primary-care physicians to recognize, diagnose and manage depression: Does it improve patient outcomes? *Psychological Medicine*, 29, 833–845.
- Tinetti, M. E., Inouye, S. K., Gill, T. M., & Doucette, J. T. (1995). Shared risk factors for falls, incontinence, and functional dependence: Unifying the approach to geriatric syndromes. *Journal of American Medical Association*, 273, 1348–1353.
- Turner, R. J., & Noh, S. (1988). Physical disability and depression: A longitudinal analysis. *Journal of Health and Social Behavior*, 29, 23–37.
- VonKorff, M. (1999). Disability and psychological illness in primary care. In M. Tansella & G. Thornicroft (Eds.), *Common mental disorders in primary care* (pp. 52–65). London: Routledge.
- Wells, K. B., Stewart, A., Hays, R. D., Burnam, M. A., Rogers, W., Daniels, M., et al. (1989). The functioning and well-being of depressed patients: Results from the Medical Outcome Study. *Journal of American Medical Association*, 262, 914–919.
- Zeiss, A. M., Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1996). Relationship of physical disease and functional impairment to depression in older people. *Psychology & Aging*, 11, 572–581.
- Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica of Scandinavia*, 67, 361–370.

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