Change in Psychological Control in Visually Impaired Older Adults Over 2 Years: Role of Functional Ability and Depressed Mood

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Objectives. The life-span theory of control is applied to study change in vision-specific control strategies in visually impaired older individuals, depending on performance in instrumental activities of daily living (IADL) and depressed mood.

Method. Longitudinal data from visually impaired individuals (at baseline: N = 364; mean age = 82.8 years; visual acuity less than 20/60) measured at three occasions with 1-year intervals in-between were analyzed. A newly established vision-specific control scale to assess selective primary control (SPC), selective secondary control (SSC), compensatory primary control (CPC), and compensatory secondary control (CSC) was used. Linear and nonlinear (quadratic and piece-wise) generalized mixed models with gamma response distribution to fit the skewed data were applied.

Results. CPC progressively increased as IADL capacity decreased up to a turning point, at which CPC plateaued, whereas all other strategies declined linearly with IADL decrease. Controlling for depressed mood did not change these relationships for CPC, SPC, and SSC but absorbed IADL-related decline of CSC. Higher depression was associated with less SPC, SSC, and CSC, but only slightly with less CPC.

Discussion. IADL plays an important role triggering a shift in adaptational strategies from selective control to CPC in visually impaired older adults and possibly other disabled populations.

Key Words: Age-related macular degeneration—Depressed mood—Generalized mixed models—Life-span theory of control—Vision impairment.

INTRODUCTION

This study examines changes in control strategy use in older adults with visual impairment due to age-related macular degeneration (AMD), framed within the lifespan theory of control (Heckhausen & Schulz, 1995; Heckhausen, Wrosch, & Schulz, 2010). AMD causes a decrease in central vision due to progressive degeneration of the macula and is the leading cause of visual impairment affecting about 20% of those between 65 and 74 years of age and one-third age of 75 and older (Fine, Berger, Maguire, & Ho, 2000). Unfortunately, the medical treatments for AMD are still rather limited (Holz, Pauleikhoff, Spaide, & Bird, 2003). Visual impairment results in a loss of central vision necessary for reading, face recognition, activities of daily living, and fall prevention (Burmedi, Becker, Heyl, Wahl, & Himmelsbach, 2002; Chen, Peronto, & Edwards, 2012; Wahl, Schilling, Oswald, & Heyl, 1999). Therefore, AMD provides an excellent opportunity to consider change in use of psychological control strategies in two areas. First, receiving the diagnosis of AMD presents a major threat to the person because no substantial cure is widely available and those affected have to adapt to this chronic stressor. Second, AMD brings long-term progressive loss in functional ability and thus questions the exertion of agency in daily life (Burmedi et al., 2002; Wahl, Becker, Burmedi, & Schilling, 2004; Travis, Boerner, Reinhardt, & Horowitz, 2004).

In this study, we examine the linkage of functional ability and control strategy use in a longitudinal sample of older adults with AMD. Also, we use a novel vision-specific control measure, which is particularly sensitive to capturing vision-specific control change dynamics over time.

Visual Impairment and Control Strategy Use

Heckhausen and Schulz (1995; see also Heckhausen et al., 2010), in their life-span theory of control, suggested four control strategies as follows: Selective primary control (SPC) strategies involve investing internal resources such as effort, time, and ability in order to attain important goals. Compensatory primary control (CPC) strategies involve finding external resources in order to facilitate goal attainment. Selective secondary control (SSC)
strategies operate at the cognitive level and serve to increase the motivational commitment toward desired goals. Compensatory secondary control (CSC) strategies rely on the replacement of no longer achievable goals, socially downward comparisons, self-serving attributions, or distancing from one’s failure experiences.

Translating the life-span theory of control to AMD, vision may be considered as a sensory resource crucial for an individual’s means of goal achievement, largely established when visual function was intact. Therefore, even intensifying goal pursuit “selectively” may not guarantee success any longer when this basic resource needed for selective strategies is compromised by AMD. In order to avoid or minimize the negative consequences of failure, a key prediction of the theory is a shift toward increased use of compensatory control modalities (see also Heckhausen [1997, 1999]). However, this rather general prediction does not answer the question of what specifically are the driving forces behind control changes in visually impaired older adults. Here, we assume the importance of two of these, namely functional ability in instrumental activities of daily living (IADL) and depressed mood.

IADL loss as a trigger for control strategy use.—We argue that functional ability is a crucial driver of changes in the use of control strategies. As long as individuals are high in functional ability, selective control strategies may seem most efficient for dealing with AMD. However, when loss in functional ability substantially threatens day-to-day life, a move to increasing use of compensatory control strategies may take place. Functional abilities could be understood as a prerequisite for the use of selective control strategies targeting goal achievement in everyday life. Thus, increasing functional impairment may not only impede the success of intensifying selective control to reach desired goals but may also represent a loss of means to exert selective control (i.e., ability of independent goal achievement). Hence, it may imply a reduced, rather than intensified, use of selective control strategies. In contrast, compensatory control strategies might become more important with increasing impediments in independent goal achievement. This may be particularly expected with respect to CPC. Older adults may not easily refrain from life habits by abandoning or changing goals but rather seek external resources to compensate for constraints in reaching goals on their own. Thus, increasing CPC strategy utilization could be expected as a major consequence of the functional losses provided by conditions such as AMD.

Furthermore, in line with our previous findings (Wahl, Schilling, & Becker, 2007), we assume that change in use of these control strategies is specifically related to one component of functional ability, that is, IADL, and not as much with more basic everyday functioning (ADL; see for this classic distinction Lawton & Brody [1969]). IADL loss generally occurs at an earlier age and shows more rapid decline in people with visual impairments, compared with those with no vision impairment (Wahl, Schilling, & Becker, 2005; Wahl et al., 1999). The interpretation of this finding is that IADL, compared with ADL, includes more complex activities such as cooking, handling of medication, shopping, or using public transportation, all of which strongly depend on one’s central vision capacity (Burmedi et al., 2002; Wahl et al., 1999; Wahl, Heyl, & Schilling, 2002). Decline in IADL may, therefore, be seen as early and undeniable sign of AMD progression, threatening one’s autonomy and future attainment of important life goals.

However, IADL losses may affect control strategy use in a more complex way than a simple pattern of “the more loss of IADL, the more or less use of a particular control strategy.” Thinking of the intrindividual course of IADL loss, starting typically from an unconstrained level of functional abilities, it may be that experiencing even the first declines of everyday function disrupts habitual ways of reaching everyday goals and triggers changes in control strategy use early in the loss of IADL. If so, changes in control strategy use may attenuate to some level of stability across the course of further IADL loss, for instance, if selective control has already been abandoned and cannot be reduced further in reaction to greater IADL decline. In contrast, it may be that such changes do not occur before IADL loss progresses substantially to a degree that cannot be handled by clinging to the habitual strategy use. This means that early IADL loss would not be related to control strategy use, whereas changes in strategies are likely to be triggered under high constraints of functional abilities. These potential dynamics of IADL and control may apply differentially to the four control strategies, but it would be mostly speculative to predict the exact pattern of these relationships for individual strategies at this point. Therefore, respective data analyses must be flexible to detect nonlinear changes in control strategy use depending on change in IADL loss such as considered above.

Depressed mood and control strategy use.—Experiencing a chronic condition such as AMD, including loss of functional capacity, as well as the emotional burden of losing vision, likely increases depressive mood (Horowitz & Reinhardt, 2000). Hence, depressive mood may precipitate a demotivating effect on peoples’ adaptive efforts to maintain their level of everyday life functioning, constraining investments in autonomy and goal-directed behaviors. As a consequence, depressed mood may obstruct the exertion of psychological control. In addition, depressed affect will make it more difficult to change goals flexibly or exert helpful social downward comparisons (“I am still better off than those sitting in a wheelchair.”), which are prototypical means of CSC strategies.

It may be, however, that depressed mood is less influential on CPC than on selective strategies or CSC. CPC is more directed toward activities to compensate for disability and is seemingly less “affective” than other strategies, which are more psychologically focused. The latter involve
intensification of internal resources or motivation, or psychological reappraisals of one’s goal achievement, aspects that are closely linked with affective experiences. Thus, depressed mood may interfere less “directly” with CPC strategies, though it may also constrain people’s motivation to engage in compensatory activities.

Overall, depressed mood may narrow selective control efforts and also interfere with the use of CSC strategies. Focusing on the relationship between IADL loss and control strategy use, it seems crucial to consider depressed mood as a confounder, which, if not taken into account, may cause changes in control strategy use to be erroneously attributed to loss of functional ability. Moreover, if IADL loss also increases depressed mood (Fauth, Gerstorf, Ram, & Malmberg, 2012), this could mediate to some degree the effect of IADL loss on control strategy use.

Research Goals and Expectations
First, by use of a vision-specific control strategy assessment applied to AMD individuals over 2 years, we aim to add empirical evidence of change to the life-span theory of control by providing a detailed description of the observed change dynamics in control.

Second, we aim to examine the impact of IADL loss on the four control strategies. In general, we expect a shift from selective to compensatory control strategies when people under AMD conditions lose functional ability. More particular, IADL loss is expected to be a major driver of increase in CPC. Considering that varying levels of IADL loss may be differentially effective in triggering control changes, we check for nonlinear relational patterns best suited to reveal the impacts of IADL loss on changes in control strategy use.

Third, we proceed from the latter analysis by adding depressed mood as predictor of control strategy use. In general, we assume that depressed mood demotivates use of control strategies. Thus, we consider depressed mood as potential confounder of IADL-related changes in control strategy use.

METHOD
Sample
Data from 364 older adults with AMD were collected by in-person interviews at baseline (T1; N = 364), one-year (T2; n = 231), and two-year (T3; n = 186) follow-up measurements. Participants were recruited from the pool of applicants at Lighthouse International, a vision rehabilitation agency in the Greater New York area. Eligibility criteria included age 65 or older, diagnosis of AMD, best-corrected acuity 20/60 or worse, first time applicant for vision rehabilitation services, and having received only low-vision clinical services (i.e., eye examination only, no additional rehabilitation services prior to the baseline interview). Potential participants meeting inclusion criteria were identified using the case records. The response rate was 50%. Participants were slightly younger than refusals (p < .05), but these two groups did not differ with respect to gender or visual acuity.

The average age at baseline was 83 years (range 65–98), including 63% women, 42% married, 93% white, 97% reporting adequate incomes, and 87% with at least a high school education. Visual acuity levels spread out fairly evenly, with a similar percentage of participants having scores of 20/60 to 20/100 (43%) versus 2/200 or worse (39%). A more detailed sample description can be found in the study by Boerner, Brennan, Horowitz, and Reinhardt (2010).

Participants who completed all interviews (n = 172; 47%) differed from those who missed at least one of the two follow-up interviews (n = 192; 53%) significantly only with respect to age (M = 82 vs. M = 84, respectively). Other sociodemographic characteristics (race/ethnicity, gender, living arrangements, marital status, and income adequacy) and health variables (self-rated health status, functional disability, subjective and objective vision status, and depressive symptoms) revealed no significant differences between these groups.

Measures
Control strategies.—To assess control strategy use in response to vision-related challenges in daily life, a vision-specific version of the Optimization in Primary and Secondary Control Scale (OPS; Heckhausen, Schulz, & Wrosch, 1998) was developed (VIS-OPS: Brennan-Ing, Boerner, Horowitz, & Reinhardt, 2012). The instrument consists of four subscales representing SPC (6 items), CPC (7 items), SSC (6 items), and CSC (4 items). Example items are “I do whatever I can to continue my everyday activities as I did before I had a vision problem” (SPC); “I often think how important it is to me to keep up my daily activities in spite of my vision problem” (SSC); “If there is something that I can no longer do because of my vision problem, I don’t hesitate to ask others for help” (CPC); “When my vision problem gets me down, I remind myself that I have coped with worse things in my life” (CSC). Respondents indicated on a 4-point Likert scale, ranging from not at all to most of the time, to what extent they felt that each statement applies to them. Mean scores (range 1–4) of SPC, CPC, SSC, and CPC were computed, with higher values indicating more control strategy use.

The VIS-OPS scale development (for details see Brennan-Ing et al. [2012]) started with a pool of 9 to 16 items for each of the four control dimensions (i.e., items from the general OPS rephrased to address vision-specific strategy use and additional items covering vision-specific control efforts). Problematic items (e.g., not well understood) were identified in a pretest with 40 visually impaired older adults. The latent factor structure was checked by means of confirmatory factor analyses (using T1 data from this
study) and fit the data well after removal of items with low factor loadings (e.g., RMSEA: .04–.06 and not significantly > .05 for single latent factor models, as well as the four factor model). Cronbach’s alpha was .62, .71, .72, and .70 for SPC, CPC, SSC, and CSC, respectively. SPC, SSC, and CSC, representing more psychologically focused subscales, were significantly related to aspects of positive psychosocial functioning (e.g., correlations with positive affect about .30), whereas the more activity focused CPC was not but showed strong correlations with functional disability measures (e.g., r = .31 with functional vision loss). For this study, we also analyzed factorial invariance at T1–T3, running repeated-measures confirmatory factor analyses (Meredith, 1993). For all subscales, constraints of strong factorial invariance (i.e., equality of factor loadings across repeated measurements) did not significantly reduce model fit, but strict factorial invariance (i.e., equality of factor loadings and error variances) was rejected.

Functional ability.—ADL/IADL competence was measured with a modified version of the OARS Multidimensional Functional Assessment Questionnaire (Center for the Study of Aging and Human Development, 1975). Six ADL and seven IADL items are assessed on a 4-point rating scale ranging from does task with no difficulty to needs help/cannot do task. The sum scores of ADL (range 6–24; α = .62) and IADL (range 7–28; α = .83) indicate loss of functional abilities.

Depressed mood.—The 20-item Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) was used to assess depressive symptoms experienced in the past week. The items employ a 4-point Likert-type scale that ranges from less than one day to 5–7 times a week. Higher values of the sum score (range 0–60, α = .88) indicate more depressed mood.

Statistical Modeling

Apart from descriptive analyses, longitudinal mixed models were analyzed (Verbeke & Molenberghs, 2000). To analyze the relations of IADL and depressed mood with control strategy, we ran Models 1–4 as specified in Table 1. Models 1 and 2 establish linear and curvilinear (quadratic) relationships of control with the predictor. To check for potential nonlinear relationships such as considered theoretically, we analyzed also “piecewise” linear-quadratic and quadratic-linear models. That is, running Model 3 with IADL as predictor means that the control outcome is related to IADL in a linear fashion up to some turning point IADL = τ, after which this relationship may accelerate or decelerate following a quadratic curve. In contrast, Model 4 employs a curvature linear under high values of IADL > τ, accelerating or decelerating from the turning point τ toward the lower end of the IADL range. Note that we specified these piecewise models “smooth” in the turning point (Cudeck & Klebe, 2002). All models were run with IADL or depressed mood as a “stand-alone” predictor. Next, we ran the IADL models controlling for depressed mood, which are also shown in Table 1. In addition, age and gender were included as predictors in all models to control for potential confounds of IADL or depression. For parsimony, random slopes were not contained in any models.

As the model equations relate the control outcome measured at occasion t to the concurrent measure of the predictor X_τ (i.e., IADL or CES-D), it should be noted that these imply as well models of change in control related with change in X_τ. These implications may be self-evident for the linear and quadratic models but should also be understood with regard to the piecewise models. For example, due to Model 3 the predicted change in a control strategy between two measurement occasions is as follows: Y_τ − Y_τ−1 = β_1 (X_τ − X_τ−1) + β_2 (max(0, X_τ−1 − τ)^2 − max(0, X_τ−1−1 − τ)^2)). Thus, the amount of change predicted by a 1-unit increase of IADL (i.e., X_τ−1 = X_τ−1−1) is ΔY_τ = β_1 (X_τ−1 − X_τ−1−1) + β_2 (max(0, X_τ−1 − τ)^2 − max(0, X_τ−1−1 − τ)^2)). The function implies that Y_τ changes by the constant rate β_1 per 1-unit increase of X_τ−1 below τ and a linearly increasing rate of change per 1-unit increase of X_τ−1 from a level above τ. In-between, if X_τ−1 increases 1 unit from below to above τ, the rate of change in Y_τ increases quadratically from β_1 (at X_τ−1 = τ) to β_1 + β_2 (at X_τ−1 = τ + 1), and the function is smooth in both turning points. Also, for Model 4, similar characteristics apply to the functional relationship between changes in X_τ−1 and Y_τ−1.

Table 1. Specification of Mixed Models of Control Strategies Regressed on Instrumental Activities of Daily Living (IADL) and Depressive Mood (CES-D)

<table>
<thead>
<tr>
<th>Model</th>
<th>Intercept and controls</th>
<th>IADL or CES-D^3</th>
<th>Control CES-D^3</th>
<th>Random</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (linear): Y_i =</td>
<td>β_0 + β_A + β_S_i + β_X_i</td>
<td>+</td>
<td>β_X_i</td>
<td>+</td>
</tr>
<tr>
<td>2 (quadratic): Y_i =</td>
<td>β_0 + β_A + β_S_i + β_X_i + β_X_i</td>
<td>+</td>
<td>β_X_i</td>
<td>+</td>
</tr>
<tr>
<td>3 (linear-quadratic): Y_i =</td>
<td>β_0 + β_A + β_S_i + β_X_i + β_X_i</td>
<td>+</td>
<td>β_X_i</td>
<td>+</td>
</tr>
<tr>
<td>4 (quadratic-linear): Y_i =</td>
<td>β_0 + β_A + β_S_i + β_X_i + β_X_i + min(0, X_i−τ)^2)</td>
<td>+</td>
<td>β_X_i</td>
<td>+</td>
</tr>
</tbody>
</table>

Notes. Y_i = control score (SPC, CPC, SSC, or CSC) measured on individual i at measurement occasion t; A = age at baseline; S = sex (dummy); β_0−β_i = fixed effects; v_i = random intercept component; ε_i = residual component.

^Models run: X_τ = IADL score; X_i = CES-D score; X_τ = IADL score with X_τ = CES-D score added.

^X_i = CES-D score, added to models run with X_i = IADL score.
SAS procedure GLIMMIX was used for all mixed model computations (SAS Institute Inc., 2009). GLIMMIX fits generalized linear mixed models for a wide range of response variable distributions, not restricted to normality. All four control strategies appeared skewed substantially, suggesting use of the gamma distribution to fit the data in mixed model analyses. We ran the Laplace integral approximation method, which enables the computation of likelihood-based fit statistics (for computational details, see the GLIMMIX manual; SAS Institute Inc., 2009, p. 2080–2430). \( R^2 \) statistics were computed as proposed by Liu, Zheng, and Shen (2008). For all Model 1–4 computations, IADL and CES-D were grand mean centered to avoid high multicollinearity of linear and quadratic predictor terms.

To estimate the turning point \( \tau \) in Models 3 and 4, we followed the piecewise growth curve procedure used by Hall, Lipton, Sliwinski, and Stewart (2000). That is, we ran the models with prespecified turning points incremented in steps of .5 ranging from the 10th to the 90th percentile of IADL or CES-D and chose the turning point, which received the best model fit in terms of the Bayes Information Criterion (BIC). BIC reveals the same selection of the optimal fitting turning point as the \(-2 \times \log \text{likelihood} \) (used by Hall et al. [2000]) and is suited for comparison between all models we ran. Thus, we used BIC to select the best fitting solution obtained from running Models 1–4 for each of the four control strategies.

**Results**

**Description of Study Variables**

Table 2 shows descriptive results for the four control strategies, IADL, and depressed mood. All analysis variables showed skewed distributions, indicating that most respondents tended to report high use of all four control strategies. Also the CES-D scores were skewed to the left, meaning that few respondents showed very high depression. Each variable’s distribution deviated significantly from normality (\( p < .001 \), Shapiro–Wilk test). These skewness characteristics were also found in the single measurement occasion distributions.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total sample ((T1 + T2 + T3)) (N = 781)</th>
<th>(T1n = 364)</th>
<th>(T2n = 231)</th>
<th>(T3n = 186)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SPC</td>
<td>Mean 3.58, SD 0.48, Skewness -1.35</td>
<td>Min 1.5, P25 3.33, P50 3.67</td>
<td>Max 4.00, P75 3.57</td>
<td>Max 3.54, P75 3.62</td>
</tr>
<tr>
<td>CPC</td>
<td>Mean 2.99, SD 0.69, Skewness -0.47</td>
<td>Min 1.0, P25 2.54, P50 3.14</td>
<td>Max 3.57, P75 4.00</td>
<td>Max 2.90, P75 3.07</td>
</tr>
<tr>
<td>SSC</td>
<td>Mean 3.20, SD 0.71, Skewness -0.88</td>
<td>Min 1.0, P25 2.83, P50 3.33</td>
<td>Max 3.83, P75 4.00</td>
<td>Max 3.16, P75 3.22</td>
</tr>
<tr>
<td>CSC</td>
<td>Mean 3.24, SD 0.81, Skewness -1.08</td>
<td>Min 1.0, P25 2.75, P50 3.50</td>
<td>Max 4.00, P75 4.00</td>
<td>Max 3.24, P75 3.22</td>
</tr>
<tr>
<td>IADL</td>
<td>Mean 14.08, SD 5.02, Skewness 1.10</td>
<td>Min 7.0, P25 10.00, P50 13.00</td>
<td>Max 17.00, P75 28.00</td>
<td>Max 13.79, P75 14.37</td>
</tr>
<tr>
<td>CES-D</td>
<td>Mean 11.29, SD 10.10, Skewness 1.10</td>
<td>Min 0.0, P25 3.00, P50 9.00</td>
<td>Max 17.00, P75 52.00</td>
<td>Max 11.66, P75 10.57</td>
</tr>
</tbody>
</table>

Notes. \( T1, T2, T3 \) = baseline, first, and second follow-up measurements, respectively; Min = sample minimum; P25, P50, P75 = 25-, 50-, 75-percentile, respectively; Max = sample maximum.

With respect to the sample means, there were only mild changes between measurement intervals in all control strategies. Some increase from \( T1 \) to \( T2 \) appears for SPC, CPC, and SSC but not so for CSC. Repeated-measures ANOVA on the control strategies revealed significant overall change only for CPC (\( p < .05 \)), where also the \( T1 \)–\( T2 \) contrast was significant (\( p < .01 \)). However, these ANOVAs are based on deletion of all cases missing at some of the follow-ups. To provide an overview of how control strategy use was related to study dropout, Table 3 shows the \( T1 \) and \( T2 \) means of those who dropped out at \( T2 \), at \( T3 \), or participated in all three measurements. Overall, there appears a tendency for those who dropped out at \( T2 \) or \( T3 \) to show lower control strategy use before dropout than those who did not drop out of the study.

On average, IADL loss increased significantly across the observation period (\( p < .001 \); repeated-measures ANOVA), but only the \( T2 \)–\( T3 \) contrast was significant—contrasting with the means shown in Table 2, which show only increase from \( T1 \) to \( T2 \). This inconsistency appears due to dropout dynamics, in that those who participated in all measurements had lower average IADL loss at \( T1 \) compared with those who dropped out in \( T2 \) or \( T3 \), and much lower IADL loss in \( T2 \) compared with \( T3 \) dropouts (see Table 3). In the depressive mood scores, neither significant mean level changes nor significant mean differences between dropout groups occurred.

**Intraindividual Change in Control Strategy Use**

As a measure of intraindividual changeability in general, we estimated for each control strategy the within-subject proportion of overall variance by computing 1 minus the total effects \( R^2 \) (Liu et al., 2008) of a random intercept only–mixed model (implying variance decomposition into stable interindivdual differences and intraindividual variation across the measurements). These proportions were .36, .33, .28, and .24 for SPC, CPC, SSC, and CSC, respectively. Thus, about one third of the primary and one quarter of the secondary control strategies’ variation was attributable to intraindividual changes of the values measured at the three occasions. It may be noted as well that IADL and...
depressed mood appeared with much lower proportions of intra-individual variation of .07 and .10, respectively.

In addition to the proportions of within-subject variance, which may reflect intra-individual trends and measurement occasion-specific fluctuations including measurement error, we analyzed general trends of intra-individual change by means of linear growth curve models (van der Leeden, 1998). The fixed linear slope effects, representing the average rates of intra-individual change per 1-year measurement interval, were close to zero and not significant for SPC, SSC, and CSC, and only the fixed slope of CPC was significant ($p < .001$), indicating average increase across the observation period. Further, the random linear slope variance appeared minor for SPC, CPC, and SSC but was significant for CSC ($p < .001$, likelihood-ratio test). Hence, the results suggest a tendency of “systematic” linear increase of CPC, and some inter-individual variation in linear rates of 1-year change in CSC, whereas changeability in the selective strategies unfolds more “fluctuant” between measurement occasions, not well approximated by linear trajectories.

To complete the description of intra-individual change in the four control strategies, we plotted the individual trajectories across the measurement occasions in Figure 1 (to visualize overlapping trajectories, these have been “jittered,” i.e., elevated by some tiny and varying value). Also, the sample means and the mean linear trajectories due to the growth curve model estimates are depicted in order to provide a sense of how these reflect the changes that occurred in our analysis sample.

### Change in Control Strategy Use Depending on IADL Loss and Depressed Mood

Running Models 1–4 with depressed mood as predictor revealed significant relationships of all four control strategies as shown in Table 4. In general, there appears a difference in IADL-related dynamics between SPC, SSC, and CSC compared with CPC. That is, for SPC, SSC, and CSC, the linear Model 1 was chosen due to the criterion of lowest BIC, meaning that the linear relationship with IADL is sufficient to fit the data (and also, the quadratic component was not significant when we run Models 2-4 on each of these strategies). For all of these strategies, the sign of the linear effect indicates a decline in control strategy use that goes along with increased loss of IADL. In contrast, for CPC, the quadratic-linear Model 4 was chosen, with turning point $\tau = .5$ on the grand mean centered IADL scale (which translates to IADL = 13 on the original IADL scale). The significant quadratic effect estimate indicates that declining IADL capacity promotes increase of CPC use, which however diminishes as IADL loss accumulates up to the level of the turning point. Notably, running these models without age and gender adjustment revealed no differences in terms of the IADL effects, with the estimated IADL coefficients only marginally higher for SPC and SSC. It may also be noted that although age had a unique and significant effect on SPC, CPC, and SSC, indicating less control strategy use with increasing age, there were no gender differences in control strategy use.

The “adjusted fixed effects only” $R^2$’s (Liu et al., 2008) printed in Table 4 indicate that the effects of IADL are in the “low” range (Cohen, 1988). However, accounting for about 4%–7% of variance (controlling for age and gender) in SPC, CPC, and SSC, the IADL effects appear substantial, noticing that these indicate the proportions “explained” only by the average linear or quadratic-linear trend, leaving aside all individual deviations from this trend as model residuals. In contrast, the effect on CSC appears rather small, accounting only for about 2% of CSC variation.

In generalized mixed models with non-normal response distributions, the fixed regression coefficients and random intercept variances do not directly refer to the scale of the data analyzed (see SAS Institute Inc., 2009, p. 2080–2430). Therefore, to provide a better sense of the meaning of the fixed effects, we depicted the regression lines of SPC, SSC, and CPC, and the piecewise regression curve of CPC in Figure 2.

Running the Models 1–4 with depressed mood as predictor (results not shown in Table 4), the linear model was chosen due to the lowest BIC criterion for all control strategies. The fixed effects of CES-D indicated significant ($p < .001$) depression-related decrease of SPC, SSC, and CSC, but notably no such significant effect on CPC. The $R^2$’s were .039, .000, .043, and .065 for SPC, CPC, SSC, and CSC, respectively.

Next, we ran the IADL models including depressed mood as predictor. Results are shown in Table 4, right hand columns. Controlling for the impact of depressed mood on control strategy use, again the simple linear Model 1 was chosen for SPC, SSC, and CSC and the quadratic-linear Model 4 with turning point $\tau = -.5$ for CPC. For all control strategies but CPC, depressed mood revealed a significant
effect, indicating decline in control strategy with increased depressed mood. For CPC, this negative depression effect was only tentatively significant (and was significant at $p < .05$ when not controlling for age and gender). However, the significant IADL effects found without depressed mood for SPC, CPC, and SSC were retained. Thus, controlling for the CES-D scores does not fundamentally change the pattern of the impact of IADL on the use of these control strategies. For CSC though, the IADL-related decrease became insignificant after controlling for CES-D scores.

Overall, depressed mood showed a minor effect on CPC use but some “low” negative effects on the other control strategies. These effects did not confound but rather worked additively to those of IADL on SPC, CPC, and SSC. However, a small effect of IADL loss on CSC use may appear only because IADL losses and increases in depressed mood tend to occur simultaneously, the latter triggering decline of CSC (IADL and CES-D were moderately correlated across individuals and measurement occasions, i.e., $r = .35$, $p < .001$.) Again, running these models without controlling for age and gender only marginally changed the size of the effects found for IADL and depressed mood.

Finally, it should be noted that running these mixed models with basic ADL instead of IADL as predictor, the results were rather similar with respect to SPC, SSC, and CSC. For CPC, however, no significant effect of ADL was found in any of the models. Thus, it is the loss of IADL in particular that triggers the increasing use of CPC.

**Discussion**

The life-span theory of control, originally proposed by Heckhausen and Schulz (1995), has found empirical
support in various study populations including older adults with severe chronic conditions (Heckhausen et al., 2010). In this paper, we examined control strategy change dynamics in a large group of visually impaired older individuals across 2 years and three measurement occasions. We also used a newly established vision-specific control measure in order to better grasp stability and change in control strategy use related to this specific chronic condition.

Table 4. Results of Generalized Linear Mixed Models of Gamma-Distributed Response Variables Run on Four Control Strategies Regressed on Instrumental Activities of Daily Living (Model 1) and Controlling for Depressive Mood (CES-D; Model 2)

<table>
<thead>
<tr>
<th></th>
<th>Model 1: IADL</th>
<th>Model 2: IADL and CES-D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate</td>
<td>CI 95%a</td>
</tr>
<tr>
<td>Fixed effects:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.258***</td>
<td>1.237–1.279</td>
</tr>
<tr>
<td>Age</td>
<td>−0.003**</td>
<td>−0.005 to −0.001</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>0.012</td>
<td>−0.015 to 0.038</td>
</tr>
<tr>
<td>IADL</td>
<td>−0.007***</td>
<td>−0.009 to −0.005</td>
</tr>
<tr>
<td>DEPMOOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random variances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.009***</td>
<td>0.007–0.013</td>
</tr>
<tr>
<td>Residual</td>
<td>91.9***</td>
<td>80.2–106.5</td>
</tr>
<tr>
<td>R^2(ΔR^2) fixed^d</td>
<td>0.079 (0.057)</td>
<td></td>
</tr>
<tr>
<td>Fixed effects:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.101***</td>
<td>1.062–1.141</td>
</tr>
<tr>
<td>Age</td>
<td>−0.007***</td>
<td>−0.010 to −0.003</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>−0.008</td>
<td>−0.053 to 0.037</td>
</tr>
<tr>
<td>IADL</td>
<td>0.004</td>
<td>−0.001 to 0.009</td>
</tr>
<tr>
<td>DEPMOOD</td>
<td>−0.003***</td>
<td>−0.005 to −0.001</td>
</tr>
<tr>
<td>Random variances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.027***</td>
<td>0.021–0.035</td>
</tr>
<tr>
<td>Residual</td>
<td>31.0***</td>
<td>27.1–35.7</td>
</tr>
<tr>
<td>R^2(ΔR^2) fixed^d</td>
<td>0.075 (0.066)</td>
<td></td>
</tr>
<tr>
<td>Fixed effects:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.141***</td>
<td>1.103–1.179</td>
</tr>
<tr>
<td>Age</td>
<td>−0.006**</td>
<td>−0.009 to −0.001</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>−0.006</td>
<td>−0.053 to 0.042</td>
</tr>
<tr>
<td>IADL</td>
<td>−0.008***</td>
<td>−0.012 to −0.004</td>
</tr>
<tr>
<td>DEPMOOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random variances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.034***</td>
<td>0.027–0.043</td>
</tr>
<tr>
<td>Residual</td>
<td>35.2***</td>
<td>30.8–40.8</td>
</tr>
<tr>
<td>R^2(ΔR^2) fixed^d</td>
<td>0.066 (0.042)</td>
<td></td>
</tr>
<tr>
<td>Fixed effects:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.136***</td>
<td>1.088–1.183</td>
</tr>
<tr>
<td>Age</td>
<td>−0.001</td>
<td>−0.005 to 0.004</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>0.019</td>
<td>−0.041 to 0.071</td>
</tr>
<tr>
<td>IADL</td>
<td>−0.008***</td>
<td>−0.012 to −0.003</td>
</tr>
<tr>
<td>DEPMOOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random variances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.058***</td>
<td>0.048–0.073</td>
</tr>
<tr>
<td>Residual</td>
<td>27.4***</td>
<td>24.0–31.6</td>
</tr>
<tr>
<td>R^2(ΔR^2) fixed^d</td>
<td>0.017 (0.017)</td>
<td></td>
</tr>
</tbody>
</table>

Notes. SPC = selective primary control; CPC = compensatory primary control; SSC = selective secondary control; CSC = compensatory secondary control; IADL = instrumental activities of daily living; DEPMOOD = depressed mood; CI = confidence interval.

*95% confidence interval.

Model chosen: Linear only relationship with IADL.

Model chosen: Quadratic-linear relationship with IADL with turning point τ = −0.5.

Fixed effects only adjusted R^2 (Liu et al., 2008), in brackets R^2-difference to baseline model including only age and gender.

n/a: Confidence interval too narrow for printout.

p < .10. *p < .05. **p < .01. ***p < .001.
Figure 2. Regression curves of SPC, CPC, SSC, and CSC regressed on IADL due to fixed effect estimates of Model 1 (SPC, SSC, CSC) and Model 3 (CPC).

Our analyses revealed substantial intraindividual changeability in all four control strategies, both in terms of the proportions of within-subject variation comprised in the control strategy variances, as by graphical plots of intraindividual trajectories, which showed that quite a few individuals changed their control strategy scores across the measurements. Notably, the changes of control strategy use varied within our sample in terms of overall rate of change and direction between individuals and measurement intervals, poorly represented by linear trajectory models and even more poorly by simple checks of mean level changes.

For an explorative statistical approach to “disentangle” such heterogeneity of changes, growth mixture modeling (GMM; Muthén & Muthén, 2000) may serve to identify potential subgroups of different prototypical trajectories of control strategy use and may also employ predictors of the probability of subgroup-membership, such as IADL and depressive symptoms. However, with respect to our focus on how changes in IADL (and/or depressive symptoms) are linked with changes in control strategy use, this modeling may be too coarsely meshed. Bearing in mind that GMM with three measurement occasions would be constrained to linear trajectories over 2 years, such analyses would also be limited to the theoretical assumption that a countable number of distinct linear trends is sufficient to represent the IADL-related changes across the 2-year range. Thus, nonlinear and shorter termed intraindividual dynamics of the control-IADL linkage may be better revealed by our approach modeling the intraindividual covariation without taking the “detour” over latent trajectory classes. In addition, concerns about the use of GMM for explanatory rather than explorative purposes with nonnormal data should be noted, as the trajectory classes found under nonnormality may not represent subgroups existent in the population (Bauer & Curran, 2003).

It might be argued that such heterogeneity of intraindividual changes is in a sense characteristic of “adaptive” psychological variables observed under disabling chronic conditions. Psychological outcomes may generally be reactive to the perceived aggravations of living circumstances caused by these conditions, hence changing rather differently due to varying patterns of occurrence and intensity of such aggravations. With respect to control strategy use under AMD, we considered IADL loss and depressive mood as such aggravating conditions crucial for changes.

We found that IADL and CPC showed a nonlinear change pattern, in that CPC use progressively increases with declines of IADL capacity, starting from fully intact functional ability, but as IADL declines further, this CPC increase attenuates until people have switched to a plateau of CPC use, which is not increased further by more loss of IADL. Notably, this finding does not indicate that the respondents plateau at the possible maximum use of CPC strategies. Bearing in mind that the regression curve shown in Figure 2, running clearly below the maximum scale value, shows predicted mean scores conditional on the value of IADL, this finding indicates that individuals seem to develop an individually adjusted elevated level of CPC strategy use when faced with mild to medium loss of IADL. In contrast, the relationship between IADL and the remaining three control strategies was linear such that decreasing IADL over time went along with decreasing control strategies. Although this was theoretically expected in the case of SPC and SSC, it seems somewhat surprising regarding CSC. However, as the negative IADL effect on CSC was rather small and disappeared when depressed mood was included in the model, it seems to be the depressed mood rather than IADL loss that promotes decrease in CSC strategies. Generally, these findings may be taken as confirmation of our basic expectation of a switch from selective strategies to CPC when functional abilities get increasingly constrained under AMD.

With respect to selective control strategies, depressed mood neither mediated nor confounded the IADL-related effects. More depressed mood was related to less selective control, as we expected considering depressed mood as demotivating such adaptive efforts, but the linkage of selective control strategies with IADL losses seems to work independent of depressive affect. CPC appeared overall less affected by depressed mood, as expected considering that CPC strategies tend to be less psychologically focused. An interesting alternative interpretation could consider that one component of CPC is seeking or receiving the help of others. AMD may signal some kind of vulnerability to the social network, such that members offer and provide help more readily. Thus, social resources may be more easily
accessible to the visually impaired such that little engagement is required to gain these resources, and even those with depressive mood and lower motivation can use support resources, one aspect of CPC. Also, greater accessibility of social resources for persons with impaired sensory health status could explain findings from our previous research showing that extroverted behavior is not as connected to positive outcomes for visually or hearing impaired compared with those with no impairment (Wahl, Heyl, & Schilling, 2012). It may be argued, however, that clinical depression in old age has been found linked with deficits in social support (Fiske, Wetherell, & Gatz, 2009), and that low perceived social support particularly increases the risk of depressive symptoms in the development of functional disability (Fauth et al., 2012). However, research has failed to find depression predictive for declines of supportive social relationships (Blazer, 1983; Murphy, 1985). Thus, although late-life depression may partially be promoted by less supportive social networks, the social support available may be facilitated under AMD, such that respective CPC strategies may not need high motivation (or activation). However, this reasoning is speculative, and this important question deserves further empirical investigation.

In conclusion, findings from this study indicate that older adults adapt to the adversities encountered from age-related visual impairments such as AMD by changing control strategy use. Loss of IADL abilities has to be regarded as one such adversity, which crucially triggers control strategy changes, in particular by shifting from selective strategies to CPC. The explanation may be that IADL capabilities denote basic resources needed for employing selective strategies, and intensifying the use of these resources may become less promising and exhausting when these deteriorate. It seems that this adaptive shift typically reaches a CPC plateau under average levels of IADL loss, such that worsening toward severe levels of functional disability is not related with further increase of CPC. However, this must not be confused with a limit of successful adaptation as it rather suggests a kind of change in the person’s adaptive style, which is driven by experiences of IADL loss and completed until a certain threshold of loss is reached. Whether this change toward CPC use will be successful in adapting to further increasing disability remains another question worthy to be researched. Finally, depressive mood is an important motivator of selective control and CSC investments but not so for CPC. Thus, it seems that depressed mood adds to, rather than confounds or mediates, the IADL-related dynamics of shifting to CPC. Thus, a depressed person may tend to reduce selective efforts but still be able to increase CPC use.

In this study, we considered IADL and depressed mood theory driven as triggers of control strategy change, but causality may also work in the reverse direction. In particular, life-span theory of control-related empirical evidence has typically viewed depressed mood as an outcome (Heckhausen et al., 2010, Wrosch, Schulz, & Heckhausen, 2004). Our data analysis, though sophisticated, cannot rule out such a possibility. That is, decrease of selective strategies may promote decline of functional ability and more depressed mood (Gerstorf, Röcke, & Lachman, 2010). However, we argue that it may be a relevant addition to this theory to also regard depressive affect as a motivational antecedent of control strategy use. This view is supported by evidence of trait-like characteristics of depressed mood (Davey, Halverson, Zonderman, & Costa, 2004), which also emerged in our study in terms of low intraindividual changeability. Also IADL showed low changeability. Thus, IADL and depressed mood changed much slower than control strategy use, such that intraindividual variability within two successive 1-year intervals was not extensive. This supports our view that these factors are drivers rather than outcomes of control strategy use. In principle, however, it may be taken as a study limitation that even our longitudinal approach involving relationships between concurrent changes in the predictor and response variables does not unambiguously provide evidence of the causal direction underlying these changes.

It is also important to note that our findings based on a vision-specific control assessment are not completely in line with our previous findings based on the general OPS (Wahl et al., 2007). In that earlier study, neither SPC nor CPC was related with the status of IADL loss at study baseline, whereas a relationship between baseline IADL and subsequent 1-year change in CSC was evidenced, as well as some more complex pattern of such relationship with SSC. Given the differences between the previous and current paper in terms of sample size, longitudinal design, and statistical modeling, any comparison seems difficult. However, the domain-specific VIS-OPS measures may also work differently compared with the highly generalized OPS. Notably, vision-specific SPC, SSC, and CSC were highly skewed, which was not the case for the OPS measures in our previous study. Also, the vision-specific version of CPC strategies, which included strategies related to both initiating and accepting help, as well as use of technological aids, may be much more sensitive to the challenges of vision impairment compared with the more generally articulated strategies in the OPS. Thus, it is another limitation of this study that we did not also apply the OPS, which would have allowed us to check if our findings differ by use of the VIS-OPS or the OPS measures.

In sum, we conclude that domain-specific control assessment may be a significant addition to apply and test the lifespan theory of control in older populations with chronic disabilities. Our focus on visually impaired older adults may be extended with other disabled subpopulations in the future. Going further, the simultaneous consideration of function (IADL) and a fundamental motivational constraining condition (depressed mood) seems important to better understand control change dynamics over time in such disabled subpopulations of older adults, but possibly also when it comes to age-related adaptation at large.
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