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Age-Related Macular Degeneration and Change in Psychological Control: Role of Time Since Diagnosis and Functional Ability

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We apply the life-span theory of control proposed by Heckhausen and Schulz to study the change in use of control strategies related to age-related macular degeneration (AMD). A mixed-model approach considers nonlinear relations of rate of change in the use of control strategies with time since diagnosis and functional ability. Data stem from a sample of 90 individuals with AMD (age, *M* = 79.5 years at Time 1), of whom 71 were assessed two times over 1 year. Compensatory primary control strategies increased shortly after the diagnosis, whereas the increase in compensatory secondary control strategies was related to functional loss in instrumental daily activities. Findings provide support for the critical role of compensatory control strategies in the event that individuals with AMD are faced with anticipated or real functional loss.

AGE-RELATED macular degeneration (AMD) is the leading cause of visual impairment among elderly individuals; it affects about every fifth older person between 65 and 74 years of age and every third person beyond the age of 75 (Fine, Berger, Maguire, & Ho, 2000). Unfortunately, the medical treatment options related to AMD are still rather limited (Holz, Pauleikhoff, Spaide, & Bird, 2003; Schwartz, 2000). The typical long-term consequence of AMD is a dramatic decrease in central vision as a result of progressive degeneration of the macula, which is essential to reading, face recognition, and the conduction of activities of daily living (e.g., Burmedi, Becker, Heyl, Wahl, & Himmelsbach, 2002; Wahl, Schilling, Oswald, & Heyl, 1999).

AMD qualifies for the consideration of *change* in use of psychological control strategies in at least two regards: First, receiving the diagnosis of AMD presents a major threat to the person, because the anticipation of losing one's sight has been found to be among the most anxiety-provoking health stressors (Branch, Horowitz, & Carr, 1989). Second, AMD also brings long-term loss in functional ability and thus questions the exertion of agency in daily life (Burmedi et al., 2002; Rovner & Casten, 2002; Travis, Boerner, Reinhardt, & Horowitz, 2004; Wahl, Becker, Burmedi, & Schilling, 2004). We argue that the life-span theory of control proposed by Heckhausen and Schulz (1995) is particularly helpful for the understanding of intra-individual change in the use of control strategies related to AMD. According to Heckhausen and Schulz, human development is embedded in an infinite number of action possibilities; therefore, selectivity is a major challenge for the successful achievement of life projects and the concomitant goals across the life span. However, everyday life also regularly brings failure, so compensation for such failure and loss experience is an important need as people age. This leads to the distinction of four control strategies as follows: selective primary control strategies involve investing internal resources such as effort, time, and ability in order to attain important goals. Compensatory primary control strategies involve finding external resources in order to facilitate goal attainment. Selective secondary control strategies operate at the cognitive level and serve to increase the motivational commitment toward desired goals. Compensatory secondary control strategies rely on the replacement of no longer achievable goals, socially downward comparisons, self-serving attributions, or distancing from one's failure experiences.

The reasoning of Heckhausen and Schulz's (1995) control theory is important for the consideration of change in the use of control strategies related to AMD, because both selectivity and compensation can be expected to be highly salient when vision loss occurs. Being selective is increasingly critical in the experience of AMD, because investing in effort and time (selective primary control strategies) and focusing or strengthening one's motivational resources in order to achieve important life goals (selective secondary control strategies) must be increasingly evaluated and balanced in light of likely failure. In order to avoid failure or to minimize the negative consequences of experienced failure, a key prediction of the theory is a shift toward the increased use of compensatory secondary control (Heckhausen, 1997, 1999). On a more general level, goal disengagement from unattainable goals, a major aspect of secondary compensatory control, facilitates engagement with attainable goals and thus promotes primary control (Heckhausen & Schulz).

To apply this reasoning, one can state that, in the earlier phase of AMD, frequently around the time of receiving the diagnosis, there is often no threat to the attainment of personal goals and thus no need to adjust in terms of increasing compensatory secondary control strategies. This is due to the persisting high level of functional ability that is normal in the early stages of the disease. However, receiving the AMD diagnosis may evoke advice-seeking and searching for support from others. Such activities are typical expressions of compensatory primary control strategies in the Heckhausen and Schulz (1995) control framework (also see Heckhausen & Schulz, 1998). In the longer run, a kind of rollback may take

Table 1. Sample Description

Variable	Sample at T1		T1–T2 Sample		Dropout	
	%	*M* (*SD*)	%	*M* (*SD*)	%	*M* (*SD*)
Age (years)		79.5 (6.6)		78.9 (6.5)		81.7 (6.8)
Gender (% female)	71.1		69.0		79.0	
Living alone	57.8		57.8		57.9	
Years of education		10.7 (3.0)		10.7 (3.1)		10.8 (2.7)
Subjective health		3.4 (0.8)		3.4 (0.8)		3.2 (0.9)
No. of self-reported illnesses		3.9 (2.4)		4.0 (2.5)		3.7 (2.2)

Notes: T1 = Time 1; T2 = Time 2; *SD* = standard deviation. For the sample at T1, $N = 90$; for the sample followed from T1 to T2 (1-year observational interval) at T1, $n = 71$; for dropout, $n = 19$ (all differences of the T1–T2 sample compared with dropout were not significant, according to a *t* test for means and a chi-square test for frequencies). For subjective health, higher scores indicate higher impairment; theoretical range = 1–5. For number of self-reported illnesses besides vision loss, the theoretical range = 0–18.

place, in that individuals reduce such compensatory primary control strategies, because help and advice have already been generated. Thus, taking the viewpoint of treating intraindividual change as an interindividual differences variable to be explained (Mroczek, Spiro, & Griffin, 2006), we expect in our first hypothesis that change in compensatory primary control strategies depends on time since diagnosis, such that over the time continuum (starting from receiving the diagnosis) the intraindividual change in compensatory primary control strategies follows a *curvilinear* course, starting with positive change (increase) and turning into negative change (decrease), which further turns again toward zero change.

In contrast, we argue in our second hypothesis that the driving force behind the increase in the use of compensatory secondary control strategies is remaining functional ability. We assume that while individuals move from high levels of remaining functional ability to lower levels, intraindividual changes in the use of compensatory secondary control strategies will occur differently. As long as individuals are high in functional ability, they may not change or even reduce compensatory secondary control strategies, because other strategies such as selective primary and secondary control strategies may seemingly remain more efficient for dealing with AMD. However, this should be different when loss in functional ability reaches a critical threshold beyond which day-to-day life becomes substantially threatened. Within this threshold region of functional ability, we expect a turnover to increasing use of compensatory secondary control strategies, because the exertion of autonomy becomes increasingly difficult. Beyond the threshold, individuals may tend to continuously increase compensatory secondary control strategies, but there would no longer be a systematic relation between intraindividual change in the use of compensatory secondary control strategies with the level of functional ability. In sum, our second hypothesis states that interindividual differences in intraindividual change in the use of compensatory secondary control depend on the degree of loss in functional ability, yet again not in terms of a simple linear relationship but rather following a curvilinear pattern across the functional ability bandwidth.

With respect to selective control strategies, according to the theory of Heckhausen and Schulz (1995), we expect both primary and secondary selective control strategies to keep their importance in order to serve goal attainment. However, selectivity may become increasingly difficult to maintain in the highly constraining situation of experiencing an ongoing decrease in functional ability as a result of AMD progression. Therefore, we also explore in this study whether functional ability is related to the change in use of selective control strategies.

Furthermore, we assume that the change in use of these control strategies is specifically related to one component of functional ability, that is, instrumental activities of daily living (IADLs), and it is not related to the more basic everyday functioning (activities of daily living, or ADLs; see Lawton & Brody, 1969, for this classic distinction). As previously found, IADL loss occurs earlier and typically shows more rapid decline in visually impaired elders, including those with AMD (Wahl et al. 1999; Wahl, Schilling, & Becker, 2005). The interpretation of this finding is that IADLs, compared with ADLs, include more complex activities such as cooking, handling of medication, banking, shopping, or using public transport, all of which strongly depend on central vision (Burmedi et al., 2002; Wahl et al., 1999; Wahl, Heyl, & Schilling, 2002). Decline in IADLs is thus the early and undeniable sign that AMD progresses further, threatening one's autonomy and future attainment of important goals in life.

Finally, in all of our analyses we control for calendar age. First, a diagnosis such as AMD may play a different role, when appearing *off time* as in a young-old person, or *on time*, as in an old-old person (Montada, Filipp, & Lerner, 1992). Second, functional ability has been found to be substantially correlated with chronological age (e.g., Steinhagen-Thiessen & Borchelt, 1999).

Methods

Participants

We followed a sample of older adults with AMD across 1 year. We chose the 1-year interval because we assumed that this period would be long enough to see a substantial intraindividual change in control strategies. The study sample at the first measurement occasion, Time 1 (T1), consisted of $N = 90$ older community-residing adults (26 men, 64 women) with a mean age of 79.5 years and an age range at first measurement between 62 and 94 years (see Table 1).

We recruited participants from the University Eye Hospitals in Heidelberg and Mannheim, Germany, and from nine private ophthalmologists in the Heidelberg–Mannheim area. Our inclusion criteria were that participants be diagnosed by ophthalmologists as suffering from various forms of AMD and had a remaining far visual acuity of equal or worse than 20/70 in the better eye, which generally is regarded as a good indication of low vision (e.g., Orr, 1992). After the participants signed a consent form, trained project research assistants conducted interviews in individual face-to-face meetings at the study participants' homes.

Participants rated their overall health as being worse than the theoretical scale mean of 3 on a one-item measure from 1 (excellent) to 5 (poor). With respect to illnesses other than the eye disease, approximately four additional diseases were reported on the basis of a list adapted from the Multilevel

Assessment Instrument (MAI; Lawton, Moss, Fulcomer, & Kleban, 1982). Study participants did not show moderate or severe cognitive deficits according to a brief screener suggested by Klein and colleagues (1985).

With respect to selection effects, a total of 123 patients had to be contacted by the ophthalmologists in order to reach the sample size of $N = 90$ at (73% participation rate). The most frequently mentioned reasons for refusal were "feeling too sick" (35%), and "I don't want somebody to visit me at home" (27%). The number of participants dropped between T1 and T2 (Time 2) from 90 to 71; all of the participants still lived in private households. As one can see in Table 1, the T1 differences between the sample followed from T1 to T2 and those dropped between T1 and T2 were not statistically significant with respect to sociostructural and health-related variables.

Measures

Control strategies.—We assessed control strategies at T1 and T2 by using the German version of the Optimization in Primary and Secondary Control Scale by Heckhausen, Schulz, and Wrosch (1998). Because of our interest in control strategies, we did not assess the optimization part of the instrument, but only the four control strategies. Each dimension included eight items to be rated on a 5-point scale (0, "never true," to 4, "almost always true"), leading to a theoretical range from 0 to 32; higher scores indicated higher use of the control strategy in question. Typical items regarding the four control strategies are as follows. For selective primary control, "Once I decide on a goal I do whatever I can to achieve it" and "When something really matters to me, I invest as much time as I can on it." For compensatory primary control, "When I cannot solve a problem by myself I ask others for help" and "When difficulties become too great, I ask others for advice." For selective secondary control, "When I have decided on a goal, I always keep in mind its benefits" and "Once I decided on something, I am not easily distracted by other things." For compensatory secondary control, "When something becomes too difficult, I can put it out of my thoughts" and "When it turns out that I cannot attain a goal in any way, I let go of it." Cronbach's alphas for the control-related scales at T1 ($N = 90$) were $\alpha = 0.81$ (selective primary control), $\alpha = 0.68$ (compensatory primary control), $\alpha = 0.70$ (selective secondary control), and $\alpha = 0.59$ (compensatory secondary control).

Time since diagnosis.—We based the time since diagnosis (TSD) variable on information provided by the study participants. We specifically trained interviewers to receive as precise information as possible on the date of diagnosis. The did this by asking back and forth the circumstances near the time of the AMD diagnosis, with the aim of making this time of life as vivid as possible. In addition, in 25 cases we found it possible to compare the interviewers' information with existing medical files. We found agreement, that is, the discrepancies in the given times were less than 3 months, in 80% of the cases.

Functional ability.—We measured IADLs at T1 and T2 by using a slightly modified version of a scale taken from the MAI (Lawton et al., 1982), which follows the tradition of the widely used Lawton and Brody (1969) approach. The original scale has been expanded for research purposes with visually impaired elders with four activities that specifically address functional tasks that may be affected by vision loss (e.g., identifying coins and bills). The 11 items of this extended scale were assessed on a 4-point scale (0, "performs task with no difficulty," to 3, "can perform task only with help"; theoretical range = 0–33). Cronbach's alpha was $\alpha = 0.76$. We also applied an ADL scale derived from the MAI (7 items, theoretical range = 0–21), which amounted to a Cronbach's alpha of $\alpha = 0.90$. We finally reversed the scores so that higher scores indicate higher functioning.

Statistical Modeling

To analyze the relation of baseline TSD and functional ability, specifically IADL, with the intraindividual 1-year changes in control strategies, we applied a multilevel mixed-model approach (Snijders & Bosker, 1999; see also Hofer & Sliwinski, 2006). As the design covered only two measurement occasions for control strategies, we could not apply the "classical" growth models comprising random slopes; hence, we specified random intercept models. To deal with the possible confounding role of age, we controlled for age by including age at T1 as a predictor in the models. In these models, we specified piecewise quadratic functions of the predictors TSD or IADL to model the kind of nonlinear relationships of intraindividual change in control strategies with TSD and IADL that we already hypothesized, namely, curvilinear patterns of relation holding over only some parts of the predictors' full range. The piecewise quadratic functions imply a curvilinear relation up to or from some "node" within the TSD and IADL range, but no relation with TSD or IADL after or before this node. In formal terms, one can write this as follows:

$$Y_{ti} = \gamma_{00} + \gamma_{01}X_i^* + \gamma_{02}X_i^{*2} + \gamma_{03}A_i + \gamma_{10}t + \gamma_{11}X_i^*t + \gamma_{12}X_i^{*2}t + U_{0i} + R_{ti},$$

$$\text{with} \quad X_i^* = X_i \quad \text{if} \quad X_i \leq b \quad \text{and} \quad X_i^* = b \quad \text{if} \quad X_i > b \quad \text{(quadratic function up to node b)},$$

$$\text{or} \quad X_i^* = 0 \quad \text{if} \quad X_i < b \quad \text{and} \quad X_i^* = X_i - b \quad \text{if} \quad X_i \geq b \quad \text{(quadratic function starting at node b)},$$

and Y_{ti} is the control measure at measurement occasion t ($t = 0$ at T1 and $t = 1$ at T2), X_i is the TSD or IADL at T1, A_i is the age at T1, U_{0i} is the random intercept component, and R_{ti} is the residual component.

With respect to our hypotheses, the model's parameters of particular importance are the interactions of measurement occasion T2 with TSD or IADL, that is, the regression coefficients γ_{11} and γ_{12}. Whereas γ_{10} denotes the estimated mean intraindividual change in control strategies, given zero TSD (or IADL), γ_{11} is the increment to be added per additional month of TSD (or additional scale point in IADL) to this mean intraindividual change, and γ_{12} gives the increment to be added per square of each additional month of TSD (or scale point in IADL). Thus, the interaction coefficients γ_{11} and γ_{12} together comprise the impact of predictor X_i on intraindividual 1-year change in use of control strategies as modeled by the piecewise quadratic function. According to our first hypothesis, we expect at least one of the coefficients, γ_{11} and γ_{12}, to be statistically

significant in the model for compensatory primary control strategies as dependent variable Y_{ti}, including TSD as predictor X_i. According to our second hypothesis, we expect γ_{11} or γ_{12} to be statistically significant in the model for compensatory secondary control strategies as dependent variable Y_{ti}, including IADL as predictor X_i. Note that γ_{01} and γ_{02} are the linear and quadratic parts of predictor X_i's effects on the T1 level of Y_{ti}, whereas γ_{00} can simply be understood as a regression constant as in conventional regression analysis.

For the TSD models (i.e., X_i denoting TSD), we computed models for $b = 3, 6, \ldots, 144$ months of TSD. That is, we computed a series of models, systematically altering the "node" in the TSD continuum, up to or from which the quadratic relation of TSD with control strategies change holds, in 3-month steps. In these computations, we excluded two outlier cases with 216 and 240 months of duration from the analysis because they would unduly impact the coefficients of the quadratic slope functions. For the IADL models, we systematically altered the node, computing models of a quadratic IADL with control change relationship up to and from node $b = 1, 4, \ldots, 33$. Among the piecewise quadratic models differing in the placement of the node, we chose the one producing minimal residual variance. We computed the reduction of residual variance achieved by the piecewise quadratic functions of TSD or IADL with respect to the residual variance of the reference model:

$$Y_{ti} = \gamma_{00} + \gamma_{10}t + \gamma_{20}A_i + U_{0i} + R_{ti}.$$

We conducted mixed-model analyses by using SAS proc mixed software (SAS Institute Inc., 1999), applying the program's restricted maximum likelihood estimator. With respect to the treatment of dropouts, it should be noted that this procedure does not imply conventional listwise deletion of cases with missing values; it includes available T1 data of those dropped out into the data-fitting procedure. The method thus provides state-of-the-art missing data treatment, enabling an unbiased parameter estimation if the data dropout conforms to the "missing at random" condition (Schafer & Graham, 2002).

Results

Data Description

On average, the TSD of AMD amounted to 49.6 months, with a large standard deviation of $SD = 46.4$ prior to the first measurement. This supported our empirical approach related to the role of TSD, because high variability in TSD is a prerequisite for testing our first hypothesis. The age that the participants were when they received the diagnosis varied between 58 and 92 years, and the average age was 75.2 ($SD = 7.5$). Age did not correlate with TSD ($r = .04$, *ns*). TSD was also not related to the status of ADLs and IADLs at T1 ($r = .06$, *ns*; $r = .09$, *ns*, respectively); that is, the pace of development of functional loss varies considerably from person to person. Age was not related to ADLs ($r = -.12$, *ns*) and only weakly with IADLs ($r = -.22$, $p < .05$), which is probably due to the limited age distribution in our sample, with the majority of participants being in the "old-old" range.

Table 2. Results of Model Tests Regarding TSD

	Selective Control		Compensatory Control	
Model[a]	Primary	Secondary	Primary	Secondary
Intercept	32.666***	28.297***	21.334**	22.198**
Change	−0.492**	−0.616***	1.748*	−0.669
TSD linear	1.888	−0.071	0.846	−0.641
TSD quadratic	−0.062	0.003	−0.032	0.019
TSD Linear × Change	−0.848	0.047	−0.471**	0.193
TSD Quadratic × Change	0.032	−0.001	0.016**	−0.006
Age	−0.156	−0.119	−0.024	0.023
Level var.	28.992**	24.859***	16.864***	15.018***
Residual var.	18.106**	16.266***	11.922***	16.111***
Total R^2	0.635	0.629	0.589	0.489
Red. Ref. (%)[b]	0.1	0.0	11.3	0.0

Notes: TSD = time since diagnosis; PQ = piecewise quadratic; for selective primary and secondary control, PQ from 120 and from 84, respectively; for compensatory primary and secondary control, PQ up to 24 and up to 6, respectively.

[a]Model chosen; PQ from b = piecewise quadratic starting at age b, PQ up to b = piecewise quadratic up to age b (see the Results section for explanation).

[b]Percentage of reduction in residual variance in comparison with the reference model (see the Results section).

*$p < .05$; **$p < .01$; ***$p < .001$.

Test of Hypotheses

Results related to the role of TSD are depicted in Table 2. The Change × TSD interaction effects, comprising the TSD impact on intraindividual change in control strategies, were significant only with respect to change in compensatory primary control. The model chosen implies a piecewise quadratic relationship up to 24 months of TSD. To illustrate our findings, Figure 1 shows the intraindividual change in use of compensatory primary control strategies as predicted from TSD according to the estimates. That is, for a given TSD, change $= \gamma_{10} + \gamma_{11}\text{TSD} + \gamma_{12}\text{TSD}^2$ (e.g., for TSD = 6 months, the predicted change value is $1.748 - 0.471 \times 6 + 0.016 \times 36 = -0.502$). Consistent with our first hypothesis, the findings underscore an intraindividual increase in the use of compensatory primary control strategies rather early after a participant receives the diagnosis of AMD, followed by an intraindividual decrease rather soon thereafter. The choice of the quadratic model up to 24 months of TSD (due to minimal residual variance; see the Statistical Modeling section) also supports the assumption that for longer lasting periods after the participant receives the diagnosis, further changes in use of compensatory primary control do not occur in relation to TSD. Moreover, as the rate-of-change line beyond 24 months runs close to zero, no further changes are predicted at all, meaning that for a given level of TSD greater than 24 months, the changes in use of compensatory primary control strategies are predicted to sum to a mean of zero. Thus, the choice of the piecewise quadratic model up to 24 months of TSD also contributes to the specification of what "early" and "longer lasting" might mean in this context: The results predict an increase in the use of compensatory primary control strategies in about the first 6 months after the participant received the diagnosis; after approximately 2 years of TSD, no more systematic tendency for intraindividual change in the use of compensatory primary control strategies appeared. In addition, the fact that there are consistent noneffects with respect to the other three control strategies accords well with our theoretical prediction.

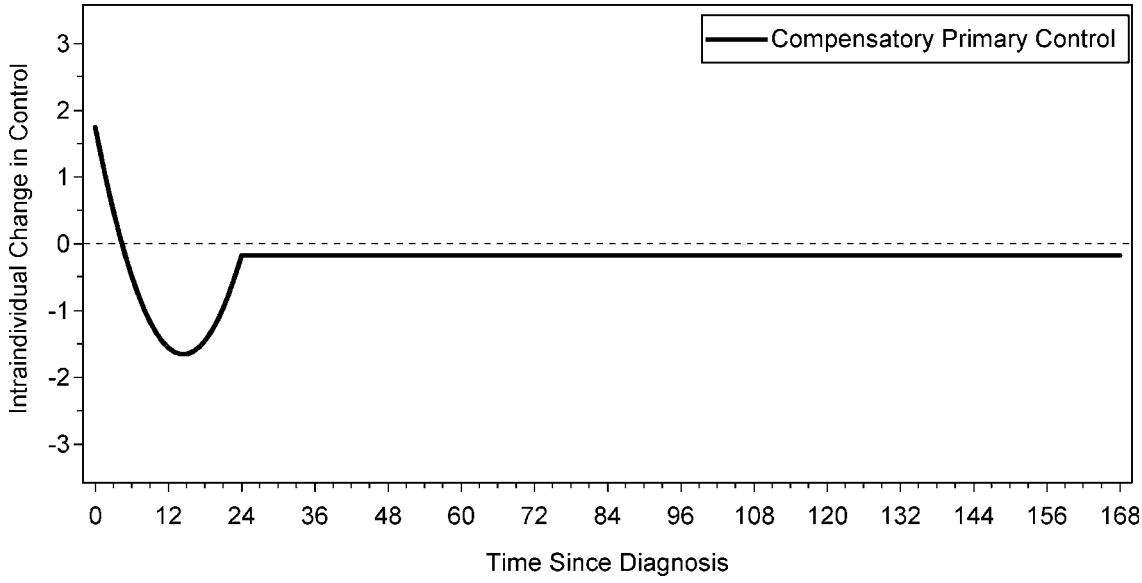

Figure 1. Rates of intraindividual change in use of compensatory primary control strategies as predicted from time since diagnosis (in months) with piecewise quadratic predictor functions (see text for further explanation): Early after participants received the diagnosis of age-related macular degeneration (<3 months), there is an increase in the use of compensatory primary control strategies, followed by a period of decrease. After this period (>24 months), no further intraindividual changes in use in relation to time since diagnosis appear.

The results of the model testing with respect to IADLs are given in Table 3. As we expected in our second hypothesis, we observed significant Change × IADL interaction effects with respect to compensatory secondary control strategies, where the "linear part" of the interaction is significant whereas the coefficient for the quadratic term is not. To illustrate the meaning of this result, Figure 2 shows the intraindividual change in compensatory secondary control strategies predicted from IADLs as a result of the piecewise quadratic coefficients shown in Table 3. (In other words, the depicted change values have been computed by change $= \gamma_{10} + \gamma_{11}\text{IADL} + \gamma_{12}\text{IADL}^2$).

Table 3. Results of Model Tests Regarding IADLs

	Selective Control		Compensatory Control	
Model[a]	Primary	Secondary	Primary	Secondary
Intercept	17.847	27.105**	24.365***	20.551**
Change	−1.345	−2.493**	−0.382*	0.914***
IADL linear	0.898	−0.603	2.998	0.741
IADL quadratic	−0.018	0.032	−0.848	−0.044
IADL Linear × Change	0.312	0.469**	1.577	−0. 307*
IADL Quadratic × Change	−0.015	−0.019**	−0.481	0.013
Age	−0.077	−0.094	−0.028	−0.012
Level var.	25.514***	25.746***	14.015***	17.697***
Residual var.	16.599***	14.333***	13.847***	13.608***
Total R^2	0.659	0.673	0.520	0.574
Red. Ref. (%)[b]	7.7	14.0	1.7	19.9

Notes: IADL = instrumental activity of daily living; PQ = piecewise quadratic; for selective primary and secondary control, PQ up to 19 and up to 21, respectively; for compensatory primary and secondary control, PQ from 29 and from 21, respectively.

[a]Model chosen; PQ from b = piecewise quadratic starting at age b, PQ up to b = piecewise quadratic up to age b (see the Methods section for explanation).

[b]Percentage of reduction in residual variance in comparison with the reference model (see the Methods section).

*$p < .05$; **$p < .01$; ***$p < .001$.

The model chosen implies a quadratic relation starting from a sum score of 21 within the IADL range of between 0 and 33. Consistent with the hypothesis, the findings underscore that in very high ranges of IADL functioning, even some tendency to decrease in the use of compensatory secondary control strategies exists, but with IADL levels lowered to 21 points, this tendency is transformed to a pattern of increase.

Note that the insignificance of γ_{12}, that is, the "quadratic part" of the Change × IADL interaction, corresponds with the rather smooth curvature after the node. Below a score of 21 points, intraindividual change in the use of compensatory secondary control no longer appears to be related to IADL level; that is, below 21 points of IADL level, the results indicate no more "change in change" in the use of compensatory secondary control strategies as a result of varying levels of IADL, but a constant increase of compensatory secondary control strategies over the lower functioning IADL range. Thus, the choice of the piecewise quadratic model starting from an IADL score of 21 also contributes to the specification of what a critical transition array in terms of IADL functioning and intraindividual change in use of compensatory secondary control strategies might be. Change in use of compensatory secondary control strategies from decrease to increase happened in the upper third of IADL functioning, as indicated by the scale used in this study. A tendency to constantly increasing use of compensatory secondary control strategies appears below the threshold of a score of 21, which is also echoed in the significant estimate for coefficient γ_{10} (see Table 3).

Beyond our hypothetical expectations, we also found significant Change × IADL interaction effects with respect to selective secondary control strategies (see Table 3). The model chosen implies a piecewise quadratic effect up to a sum score of 21 within the given IADL range of between 0 and 33. Figure 2 also shows intraindividual 1-year change in use of selective

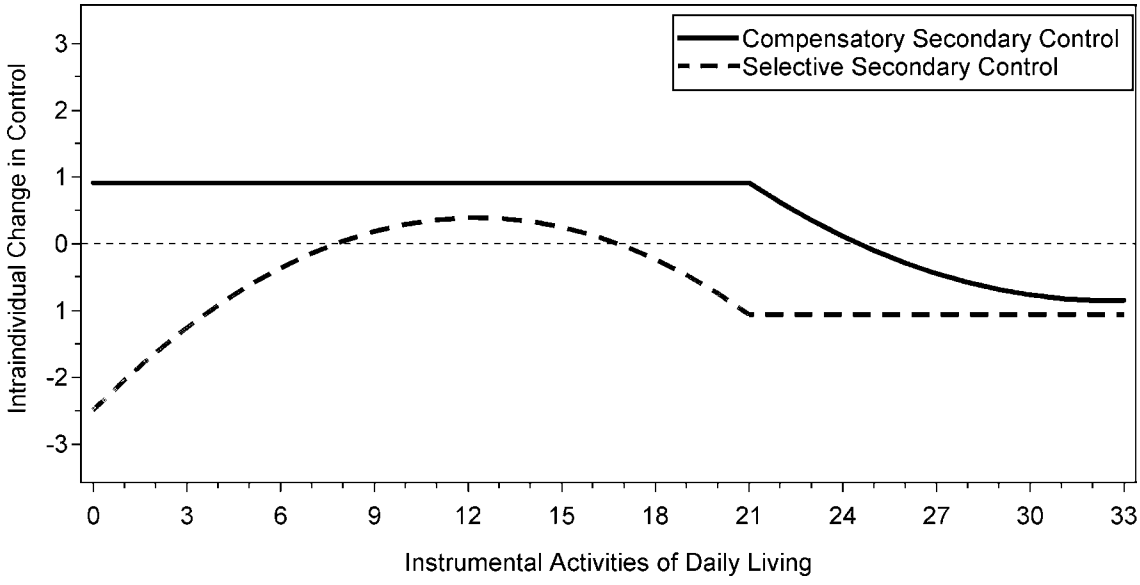

Figure 2. Rates of intraindividual change in use of selective secondary and compensatory secondary control strategies as predicted from instrumental activities of daily living (IADL) with piecewise quadratic predictor functions (see text for further explanation): for compensatory secondary control strategies, the decrease related to the highest level of IADL functioning switches to an increase as the level of IADL declines. For lower levels of functioning (IADL < 21), there is constant increase. For selective secondary control strategies, there is some constant decrease for those without larger functional losses (IADL > 21), switching to some increase slightly above the zero rate of change as level of functional ability declines, turning around to pronounced decrease for those lowest in IADL functioning.

secondary control strategies predicted from IADL score. As we noted, the use of selective secondary control strategies showed a pattern of pronounced intraindividual decrease, particularly in the very low range of IADL functioning, which in the IADL range from about 9 to 18 changes to increase slightly above the zero line of change, thereafter changing again into a decrease. For higher levels of IADL functioning, that is, beyond the score of 21, we observed some constant decrease in use of selective secondary control strategies, which were unrelated to IADL score.

Furthermore, we found no meaningful relation between IADL level and intraindividual change in use of compensatory primary control strategies and intraindividual change in use of selective primary control strategies. We also found no effect with respect to calendar age in any of the analyses. In addition, we ran all the analyses with the ADL component of functional ability, but, as expected, we did not find any statistically meaningful interaction term.

Another point deserving of mention is that the observed effects, though rather consistently supporting our hypotheses, were limited in effect size. For all models, we computed the percentages of reduction in residual variance achieved by the inclusion of TSD or IADL (e.g., Snijders & Bosker, 1999). We observed the relatively strongest finding for predicting compensatory secondary control, with 20% reduction of residual variance achieved by adding the piecewise quadratic effects of IADL (and the control variable of age) to the model.

Finally, there also appeared some significant levels for γ_{10}, the main effect of intraindividual 1-year change, in Tables 2 and 3. Simplifying a bit, we state that these are estimates of the average intraindividual change "outside" of the piecewise quadratic predictor (TSD or IADL) range, as, for example, the tendency of increase in compensatory secondary control below the IADL score of 21. In short, these significant levels indicate substantial proportions of intraindividual 1-year change in use of the control strategies that have not yet been explained by our TSD and IADL predictor variables.

Discussion

We used the Heckhausen and Schulz (1995) control theory to predict intraindividual 1-year change in the use of compensatory control strategies related to the experience of AMD. We found that the time that has passed since a person received the diagnosis of AMD plays a substantial role with regard to intraindividual change in the use of compensatory primary control strategies. The use of compensatory primary control increased in a rather early period after the participant received the diagnosis and then decreased again. About 24 months after the participant received the diagnosis, no meaningful relation between intraindividual change in use of compensatory primary control and TSD existed. We interpret this finding as being consistent with the control theory of Heckhausen and Schulz. It could be expected that a life-threatening situation, such as receiving a critical diagnosis (AMD), does not fundamentally question one's capability to exert selective control strategies. However, there is much psychological insecurity about what the diagnosis will mean for one's autonomy and the attainment of important life goals in the future. In a life situation of anticipated loss of control, an increase in strategies such as searching for help or advice-seeking from others, that is, compensatory primary control strategies, is more adaptive than an increase in compensatory secondary control strategies, for instance premature goal disengagement. Because this is an

adaptive response occurring particularly early after a participant received the diagnosis, it makes sense that we found a disconnection between TSD and change in use of compensatory primary control in the longer run.

Furthermore, IADL functioning appeared to be an important driving force for intraindividual change in the use of compensatory secondary control. According to our findings, it is a critical threshold rather high in the range of IADL functioning (the array above a score of 21 and the maximum of 33 points in our IADL assessment instrument), which brings the switch from decrease to increase in use of compensatory secondary control. To illustrate, consider an older person who has the goal of traveling around the world among his or her highest personal preferences. Even some loss in IADLs, such as problems with public transport, will probably lead to a substantial goal blockade. In such a life situation, the control theory of Heckhausen and Schulz (1995) would predict an increase in compensatory secondary control as an adaptive strategy; this seems, as we have found, to actually be the case in a substantial number of AMD individuals. Also note that, according to the life-span theory of control, disengagement from goals that are no longer attainable will facilitate the engagement with those that are still attainable, and by this means it will promote primary control (Heckhausen & Schulz, 1995).

We also found evidence for a curvilinear relation between IADL status and intraindividual change in use of selective secondary control strategies. A change pattern of decrease in the use of this strategy was observed particularly in the array of very low IADL functioning, which was roughly below a score of 9. In the higher range of IADL functioning, particularly between 21 and 33, there appeared some constant decrease; that is, the decrease was no longer associated with the level of IADL functioning. This finding mirrors what has been observed with respect to compensatory secondary control strategies: Particularly during processes of progressive disability (as in AMD), it is essential that individuals invest their resources into the pursuit of controllable goals and not waste them on futile pursuits. Therefore, increase in the use of compensatory secondary control is coupled throughout a wide band of losing IADLs with the use of selective secondary control, though there also is some decrease in the use of this control strategy. A pronounced increase in rate of decrease in selective secondary control only begins further down the road, when IADL losses are very significant.

In sum, given the important role of psychological control for outcomes such as well-being and depression (e.g., Chipperfield, Perry, & Menec, 1999; Heckhausen & Schulz, 1995; Lachman & Firth, 2004; Schulz & Decker, 1985; Schulz & Heckhausen, 1999; Wrosch, Schulz, & Heckhausen, 2002), our findings add to the better understanding of the dynamics of change in control strategies in a prevalent situation of chronic loss in old age, such as central vision loss due to AMD. It seems that both time since receiving the diagnosis of AMD as well as IADL functioning play an important role and that the associated effects follow a nonlinear change pattern, which is not detectable with statistical tools driven by linearity assumptions. Such insights are helpful for future control-related research in the field of vision loss in general and on the psychosocial consequences of AMD in particular, in which linearity is the dominant analytic paradigm (Kleinschmidt et al., 1995; Wahl et al., 2004). Further, we also hope that our data-analytic strategy will stimulate research with respect to other stressful chronic conditions in later life, such as hearing loss or loss in mobility, and their relation with change in control (see also Schilling & Wahl, 2006).

In terms of limitations, we observed a rather small sample of 71 individuals, and our observation period of 12 months was rather short. In addition, only two measurement occasions per individual were at our disposal and the findings were limited in their effect size. As a consequence, research targeting the role of chronic life stressors on change in psychological control based on larger samples, longer observation intervals, more measurement occasions, a scope of different chronic conditions, and the application of nonlinear modeling approaches for data analysis is needed to further this field of inquiry in the future.

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