Modeling Late-Life Adaptation in Affective Well-Being Under a Severe Chronic Health Condition: The Case of Age-Related Macular Degeneration

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Age-related macular degeneration (AMD) was used as a case model to longitudinally study adaptation in affective well-being under a prevalent chronic health condition. Measures of positive and negative affect, obtained at 5 subsequent measurement occasions with 3-month intervals in between, were analyzed in 90 older adults diagnosed with AMD. The authors proposed a pattern of adaptation that shows initial decline in affective well-being after disease outbreak, followed by a turnaround into a restorative phase of increase, implying nonlinear intrapersonal trajectories, with changes substantially related to disease duration. Analysis was conducted by means of a nonlinear mixed models approach. Results confirmed the hypothesized adaptation pattern for positive affect but not for negative affect, which was found more stable across measurement occasions.

Keywords: adaptation, positive and negative affect, subjective well-being, age-related macular degeneration, nonlinear mixed models

Adaptation of subjective well-being (SWB) in later life has become an important topic in gerontological research in the past decade, prompted by the absence of age-related decline in SWB as reported by numerous empirical studies and literature reviews (e.g., Diener & Suh, 1997; Herzog & Rodgers, 1981; Horley & Lavery, 1995; Smith, Fleeson, Geiselmair, Stettersten, & Kunzmann, 1999). To explain this finding, theoretical discussions have focused on the aging person’s ability to adapt to worsening conditions, maintaining or regaining SWB in view of multiple psychosocial losses and increased health risks (Baltes & Carstensen, 1996; Brandstätter & Greve, 1994; Staudinger, 2000). However, this line of reasoning is hampered by substantial pitfalls, preventing conclusive statements on adaptability of SWB in old age.

First, the evidence for “stability despite loss” of SWB as cited above has been largely derived from cross-sectional studies and for measures addressing life satisfaction. Given the characteristic weakness of cross-sectional designs, that is, the confounding of age with cohort effects, such studies are not able to definitively indicate the absence of age-related change in SWB. Regarding longitudinal evidence, recent studies that address changes in life satisfaction challenge the paradox by evidencing nonlinear age-related decline over the old age period, with substantial losses in life satisfaction noted particularly and increasingly with the “old-old” age period (Mroczek & Spiro, 2005; Schilling, 2005). However, these findings concern life satisfaction, which in well-being research is seen as the cognitive component of SWB, meaning the evaluation of one’s own life or living circumstances in terms of subjective judgments, which must be distinguished from the person’s emotional experiences (Diener, Suh, Lucas, & Smith, 1999). With respect to the affective component of SWB, typically operationalized as positive affect (PA) and negative affect (NA), reviews of the literature on age-related changes in PA and NA have revealed mixed findings (Charles, Reynolds, & Gatz, 2001; Kunzmann, Little, & Smith, 2000). At best, tentative stability or only a slight decrease in NA across the later years can be derived from the longitudinal evidence generated so far (Charles et al., 2001; Costa, McCrae, & Zonderman, 1987; Kunzmann et al., 2000; Stacey & Gatz, 1991). Analyzing the age-related development of depression, which should be closely related with PA and NA, Rothermund and Brandstätter (2003) found depression to be stable up to about age 70, with increases afterward. Therefore, given the evidence for stability despite loss of SWB in old age, additional longitudinal research is clearly needed to better understand affective well-being trajectories as people age.

Second, interpreting the absence of age-related decline in terms of adaptation bears on using age as proxy for losses associated with old age. In particular, health problems become more frequent, such that mean level stability of SWB, contrasting age-related decline of average levels of physical health, might be taken as support for the adaptation assumption. But looking only at the average change in levels of health and SWB ignores between-subjects variation of changes in SWB and health conditions and might thus obscure the relation between these changes. Therefore, the study of adaptation profits from the consideration of intrapersonal trajectories of SWB under severe chronic health and functional loss conditions. We use in this work vision loss caused by age-related macular degeneration.
by age-related macular degeneration as an example for such a chronic health condition.

**Rethinking the Concept of Adaptation Under a Chronic Health Loss Condition**

In the gerontological literature, the term *adaptation* has been used as a descriptor for psychosocial outcomes as well as for the process of adapting to changing conditions (George, 2003; Whitbourne & Cassidy, 1996). It is also a common practice to use adaptation as a synonym for related concepts, such as coping or other self-regulatory processes. However, Diener et al. (1999) warned that adaptation and coping should be distinguished, because only the latter concept puts emphasis on the individual’s investment into a scope of strategies aimed to regulate SWB. Coping theories (e.g., Folkman & Moskowitz, 2004) focus on people’s active role in managing stressors such as chronic disease, whereas the meaning of adaptation refers more globally to people’s reactions to changing conditions and is not restricted to active self-regulation. However, in the face of divergent and imprecise definitions of adaptation, more clarity continues to be needed.

In its broadest sense, the term *adaptation* was used to denote every facet of psychological functioning that can be understood in terms of adjustment to the individual’s living conditions, such that “little that goes on in mind and behavior is not in some way relevant to adaptation” (Lazarus, 1991, p.7). However, resting on a broad definition of adaptation, this statement has only minor empirical content, which could be enriched only by defining adaptation more distinctively. Basic concepts of psychological adaptation, as reviewed by Bevan (1965), comprised avoidance behavior, reduced responsivity to repeated or continued stimulation, and so-called normative adaptation, as conceptualized most prominently by Nelson’s (1948) adaptation level theory. Considering psychological adaptation of SWB to chronic health losses, it seems clear that this cannot mean avoidance behavior, as the focus is on the reaction to the loss experience and not on avoiding it. Notably, principles of reduced responsivity and of adaptation level theory have been adopted to theories on SWB, for example, Michalos’s (1985) multiple discrepancies theory, Lawton’s (e.g., Lawton & Nahemow, 1973) ecology of aging theory, and the set-point model of happiness proposed by several authors (e.g., Lucas, Clark, Georgellis, & Diener, 2003). Basically, all of these approaches have in common the implication that adaptation should be understood as a within-subject process, triggered by some outlasting change in the individual’s living conditions, leading toward reduction or redemption of the initial psychological consequence of that alteration.

With respect to people adapting their SWB to a chronic disease, this means that over the course of the illness, people may undergo a period of worsened SWB (which is an initial psychological consequence to the adverse condition), but over time this reaction may be reduced, meaning that the SWB improves again. In the long run, SWB may reach again or at least come close to its initial, preadaptational level. Thus, adaptation does not simply implicate “stability despite loss” in SWB but consists of changes following an adaptational pattern of temporary intraindividual variation in SWB, namely a worsening coming with the initial confrontation with the adversity, which is then dampened in the course of continuation of this condition to a point of turnaround into a restorative phase of positive change toward the initial level of SWB. Adaptation ends with the fadeout of this restorative increase, that is, SWB should level off or plateau at some point, which can be understood as the end of the adaptation period.

In a longitudinal study with affect measures observed across more than one subsequent interval, carried out in a sample of people with the same progressive chronic condition but differing in the time of continuation of this condition, such a pattern of adaptational change should appear with particular properties. Individuals early in adaptation at first measurement may start with worsening affect (PA decreases or NA increases) over the first measurement interval, and turn into enhancements of affective well-being in the subsequent intervals. Others in their restorative phase of adaptation may start with an enhancement in the first interval, slowing down to no further change in later intervals. In addition, at first measurement there may be participants with long-lasting exposure to the chronic health stressor who completed the phase of adaptation at all. For these, changes in affect may occur but no longer fit the suggested pattern. Altogether, change in affect measures observed over subsequent intervals may reveal a nonlinear pattern, such that a substantial proportion of trajectories show at least one general change in the trend of the curvature, with the initial rates of changes before the converse or slowdown observed over the first measurement intervals related to the duration of the adverse condition in the nonlinear fashion, following the pattern of early decline, turnaround into restorative increase, and fadeout in the long run of disease duration.

Thus, adaptation of SWB to age-related loss provides a fundamental methodological challenge with respect to the nonlinearity of adaptational changes provoked by an adverse condition. This should result in nonlinear intra-individual trajectories as well as in a nonlinear relation of the rates of change with the duration of the adversity. It seems that such nonlinearity has not yet been considered in empirical research on adaptation of SWB, and it must be understood that statistical methods implying “conventional” linearity assumptions might obscure rather than reveal adaptational change. With the present article, we would like to suggest a methodological approach to model the nonlinearities evolving from the concept of adaptation as explicated above.

**Adaptation of Affective Well-Being Under the Condition of Age-Related Macular Degeneration**

Age-related macular degeneration (AMD) qualifies as a chronic condition of particular relevance for adaptation in SWB in that it is an adverse condition, is highly frequent in old age, and provides continued and irreversible loss in a crucial domain of physical function. AMD is the leading cause of severe visual impairment in old age and affects nearly every fifth older person between 65 and 74 years of age and nearly every third person beyond the age of 75 (Fine, Berger, Maguire, & Ho, 2000). The typical consequence of AMD, in terms of vision function, is deterioration of the central visual field (scotoma), provoking severe reading difficulties as well as problems in conducting activities of daily living (Burmedi, Becker, Heyl, Wahl, & Himmelsbach, 2002; Rovner & Casten, 2002; Travis, Boerner, Reinhardt, & Horowitz, 2004; Wahl, Becker, Burmedi, & Schilling, 2004). In addition, there are very few medical treatment options that can halt the progression of the eye disease (Holz, Pauleikhoff, Spaid, & Bird, 2003). Most no-
tably, for AMD patients, high rates of depressive symptoms have been found. Rates are significantly higher for this group in comparison with the general population of community-based older adults but are similar to those found in outpatients with life-threatening diseases (Brody et al., 2001; Horowitz, Reinhardt, & Kennedy, 2005; Rovner & Casten, 2001). Thus, AMD seems to strongly impact the person’s affective experience. If older adults’ affective well-being is adaptive to age-related loss, the AMD condition should provoke adaptational changes in measures of affect and hence provide a particularly good case to observe adaptation.

Notably, it seems that the duration aspect of AMD, key to our understanding of adaptation, has not yet found far-reaching consideration in the psychosocial literature as it pertains to this chronic condition. Altogether, we found only two studies on visually impaired older adults that presented results on the cross-sectional relation of duration of the impairment with depressive symptoms: Evans (1983) found depression greatly increased among those with 30 to 59 months of duration of blindness but was on the same level for those with shorter or longer duration, which means a nonlinear relation in line with our considerations on adaptation. Hersen et al. (1995) found duration not correlated with depression, that is, no such relation under a linearity constraint.

In the study of AMD as a chronic health condition able to provoke adaptation of affective well-being, additional considerations deserve discussion. First, the finding of increased depression levels among AMD patients must not be mistaken as evidence that affective experiences are not adaptive to this condition at all. High incidence rates of depression are not contradictory to adaptation. According to our understanding, adaptation comprises decreases as well as restorative increases in affective well-being. Hence, an overall reduction in mean level affective well-being can be expected for AMD in samples compared cross-sectionally with older adults not affected by serious chronic conditions. It should also be noted that the depression rates observed in the AMD samples were about 30%, leaving a majority of persons with AMD as not clinically depressed.

Second, one must consider what depression means in terms of PA and NA. Following Clark and Watson’s (1991b) influential tripartite model of anxiety and depression, both syndromes share a component of high NA experience. Yet these syndromes are distinguished by their specific components, one of which is, for depression, the absence of PA. Despite some evidence that reduced PA may be less specific for depression in old age than in the general population (Shapiro, Roberts, & Beck, 1999; Wetherell, Gatz, & Pedersen, 2001), the crucial point is that also in old age depression comes with reduced PA (see also de Beurs et al., 2005; Pruchno & Meeks, 2004). Thus, given high rates of depressive symptoms among older adults with AMD, it can be concluded that PA may be highly reactive to this chronic condition, particularly with regard to the ongoing vision and concomitant functional loss associated with AMD (Rovner & Casten, 2002; Wahl, Schilling, & Becker, 2006). PA may hence undergo adaptational changes throughout the course of the disease.

However, a large body of empirical evidence suggests that NA has a strong dispositional component and is thus less reactive to changing situational influences (Baker, Cesa, Gatz, & Mellins, 1992; Clark & Watson, 1991a). This finding could explain the strong association between depression and neuroticism, as has been found by Rovner and Casten (2001) in a sample of AMD patients. On the other hand, evidence from diary studies reveals substantial within-subject variation of NA in rather short time intervals (Mroczek & Almeida, 2004), which seems at first glance contradictory to traitlike stability of NA. However, in the study of change in personality-related variables such as NA, short-term fluctuation should be distinguished from long-term change or stability (e.g., Mroczek, Spiro, & Griffin, 2006). Fleeson and colleagues suggested an analytical framework to integrate both perspectives by viewing personality-related behavior over a period of time in terms of a within-subject density distribution (e.g., Fleeson & Leicht, 2006). On the basis of this model, the intrapersonal level over a period of time (say, a few weeks) reflects the aspect of long-term stability, whereas intrapersonal variance reflects short-term fluctuation. Thus short-term variability is not contradictory to traitlike stability. Regarding our study, the focus of interest is long-term stability or change in affect resulting from adaptation to the AMD condition. Therefore, the findings on overall traitlike stability in NA mentioned above lead us to the assumption that we might find NA less reactive to AMD in terms of longer term adaptational changes.

Aim of the Study and Hypotheses

Driven by our conceptual and methodological considerations, we explore in the present article adaptation of PA and NA under the health condition of AMD with a research design based on a 1-year observation period with five measurement points conducted in 3-month intervals. Two hypotheses are tested. First, we expect that measures of PA will show adaptation to the AMD condition; that is, analysis of PA trajectories will reveal the following properties. Because the sample of AMD individuals comprised participants at different phases of an ongoing adaptation process with adaptational changes in different directions, no significant mean level change, but substantial interindividual variation in intrapersonal change, is observable. Also, the PA trajectories provide support for a nonlinear curvature with a tendency to slow down or reverse initial change. Furthermore, variation in intrapersonal PA change is nonlinearly related to the duration of the disease, with PA decline being largest in early stages of the disease, slowing down with temporal distance to the outbreak, followed by a phase of some restoration. With longer lasting AMD, PA changes are no longer related to the disease duration. Second, we expect that measures for NA will show adaptation to the AMD condition only tentatively or even not at all.

Method

Design and Sample

According to the design of the study, a sample of older adults with AMD was followed across 1 year. After first measurement occasion t0, four additional occasions were taken in 3-month intervals, ending with t4. The study sample at t0 consisted 90 older community-residing adults (26 men, 64 women) with a mean age of 79.5 years (SD = 6.6). Participants mostly lived in single households (57.8%) and had an average educational level of 10.7 years.

Participants were diagnosed by ophthalmologists with various forms of AMD. They all met the inclusion criterion of a far visual acuity of equal or worse than 20/70 in the better eye, which is generally regarded as a good
indication of low vision (e.g., Orr, 1992). This diagnostic assessment was provided in a clinical setting by ophthalmologists involved in this study.

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 AMD, approximately four additional diseases were reported based on a list adapted from the Multilevel Assessment Instrument (Lawton, Moss, Fulcomer, & Kleban, 1982). The most frequently mentioned were cardiovascular and arthritis-related diseases. Study participants did not show moderate or severe cognitive deficits based on a brief screener suggested by Klein et al. (1985).

Visually impaired older adults were recruited at the University Eye Hospitals in Heidelberg and Mannheim, Germany, and additionally from nine private ophthalmologists in the Heidelberg–Mannheim area. Patients fulfilling the inclusion criteria were asked by the ophthalmologists if they would like to participate. PA and NA were assessed at each of the five measurement occasions. It should be noted, however, that only the assessments at t0 and t4 were done in a face-to-face manner, whereas t1 to t3 assessments were conducted over the phone.

To reach the sample size of 90 at t0, ophthalmologists had to contact a total of 123 patients, leading to a participation rate of 73%. In measurement occasions t1 to t4, the number of study participants was 83, 71, 68, and 70, respectively, and 61 persons took part on all five measurement occasions. Comparison of this latter group with those with incomplete data across the five measurement occasions with regard to sociostructural and health-related variables (age, gender, household composition, education, visual acuity, number of self-reported illnesses) at t0 revealed no significant differences, which were, in terms of the effect size indicators proposed by Cohen (1988), small for age and gender and less than small for all other comparisons. Regarding PA, NA, and duration of AMD, these two groups also did not differ significantly, with effect sizes close to zero for PA and NA differences and “small” for duration (average duration was a bit longer for those who participated at all five occasions). Regarding sample drop-out, it should be noted that the main method of analysis described below does not imply listwise deletion of cases with missing values, that is, all available data from the 90 respondents were included in analyses.

**Measures**

**Affect.** PA and NA were assessed by the German version of the Positive and Negative Affect Schedules (PANAS; Watson, Clark, & Tellegen, 1988). Both PA and NA subscales consist of 10 adjectives each, connoting positive and negative emotions. Notably, the PANAS items have been classified as high-arousal emotions (Watson & Tellegen, 1999). Both PA and NA subscales consist of 10 adjectives each, connoting positive and negative emotions. Notably, the PANAS items have been classified as high-arousal emotions (Watson & Tellegen, 1999). Participants were asked to indicate on a 5-point scale, ranging from 1 (not at all) to 5 (very often), how frequently they had experienced each emotion during the last week. Total scores were divided by the number of items. For the “last week” time frame version of PANAS, Cronbach’s alphas reported by Watson and Clark (1994) ranged between .86 and .90 for PA and between .79 and .90 for NA; in the present study, these values were .79 and .78, respectively, at t0.

**Duration of disease.** This variable was based on information provided by the study participants and based on the question of when they received the diagnosis of AMD for the first time by their ophthalmologists. It is typical for such medical information that questions regarding accuracy are not easy to solve. In our study, interviewers were specifically trained to receive as precise as possible information on the date of diagnosis. This was done by back-and-forth questioning of the circumstances around the occurrence of being diagnosed with AMD; the aim was to make this time in the life of interviewees as vivid as possible during the assessment situation. It should be noted that nearly all participants were able to report the time of diagnosis on a year and month basis, except two outliers in AMD duration reporting outbreak of AMD approximately more than two decades ago, without being able to remember the exact time of AMD diagnosis.

**Statistical Modeling**

We conducted longitudinal analyses by using multilevel mixed models (e.g., Hox, 2002; Snijders & Bosker, 1999). The key concept of longitudinal multilevel models, often referred to as growth curve models, is to treat longitudinal data as hierarchically structured: The measurements at different points in time (first level) are nested within individuals (second level). This constitutes a hierarchical regression model that decomposes the dependent variable’s overall variance into a within-subject component (intraindividual variation over each person’s repeated measurements) and a between-subjects component (interindividual variation over the persons). Doing so, the models comprise a within-subject growth function, specifying intraindividual variation as dependent on the time of measurement, and between-subjects functions, modeling interindividual variation in some or all parameters of the growth function. Thus, intraindividual changes in PA or NA can be modeled by within-subject growth functions, whereas the basic properties of adaptation as considered above (i.e., interindividual variance of intraindividual change and relation of intraindividual change with duration of AMD) can be modeled by between-subjects functions.

To deal with the nonlinearity of individual trajectories as hypothesized, we specified models with linear, quadratic, and cubic within-subject growth functions:

\[ Y_{it} = \sum_{j=0}^{m} \beta_{j} t^j + R_{it}, \]  

with \( Y_{it} \) denoting the value of dependent variable (for individual \( i \) at measurement occasion \( t \)), \( t \) denoting the measurement occasion (\( t = 0, \ldots, 4 \)), \( \beta \) denoting a Level 1 regression coefficient, and \( R_{it} \) denoting the Level 1 residual value. Thus, \( m = 1 \), \( m = 2 \), and \( m = 3 \) define the linear, quadratic, and cubic growth functions, respectively.

Additionally, piecewise linear growth functions were fitted. Piecewise linear growth functions are composed of a linear growth component across all measurement occasions, to which a second linear component after some “node” within the range of measurement occasions is added, shaping the growth function as a kinked line:

\[ Y_{it} = \beta_0 + \beta_1 t + \beta_2 t^* + R_{it}, \]  

with \( t^* = t - a \) if \( t > a \) and \( t^* = 0 \) if \( t \leq a \). With five subsequent measurement occasions, the second, third, or fourth are capable as nodes, and the three resulting piecewise linear growth models were analyzed (i.e., \( a = 1 \), \( a = 2 \), \( a = 3 \)). The piecewise linear growth function has been chosen for the benefit of easier interpretability of its components compared with the quadratic function, but both models are useful to analyze trajectories with a change in the general trend of the slope. The piecewise linear function, though modeling this more coarsely, gives a more intuitive description, as its first component directly describes the initial trend of the slope, and the sum of both components is the trend after the node.

In a first step of analysis, growth functions 1 and 2 were combined with “random-only” between-subjects functions, allowing for intraindividual variation in regression coefficients \( \beta_j \):  

\[ \beta_j = \gamma_{j0} + U_{j0}, \]  

with \( \gamma_{j0} \) denoting a Level 2 regression intercept (the so-called fixed effect of \( \beta_j \)) and \( U_{j0} \) a Level 2 residual value. Tests of these models’ fixed and random effects serve to check for absent mean-level change and interindividual variation in intraindividual changes, which was hypothesized as a fundamental property of an adaptational pattern of change.

In a second step of analysis, the within-subject growth functions 1 and 2 were combined with between-subjects functions including duration of disease as predictor. Adaptation to AMD should show up in a curvilinear relation of duration with the slopes of the growth functions, which was
hypothesized to come to an end after some point of duration, with further changes in the dependent variable not related to duration. Therefore, we specified a piecewise function, implying a quadratic function of the duration impact up to some node within the duration range but no further duration impact after this node:

$$\beta_j = \sum_{i=0}^{2} y_{ij} d_i^2 + U_{ij}$$  \(4\)

with \(d_i\) denoting duration of disease in months, \(d_i^2 = d_i\) if \(d_i \leq b\), and \(d_i^2 = b\) if \(d_i > b\).

In a set of computations, the node was systematically altered in 3-month steps; that is, this model was specified and computed with the node after 3, 6, 9, and so forth months of disease duration (i.e., \(b = 3, 6, 9, \ldots\)). Variation of the node alters the prediction of intercept and slope components and hence the random variances for these components. Because our hypothesis puts emphasis on the prediction of the slopes, among the piecewise quadratic models differing in the placement of the node, the one producing minimal random-slope variance was chosen. It might be noted that this second step means cross-sectional analysis in a way, as it targets the relationship between interindividual differences in intraindividual change in affect measures and duration, which would result if the persons undergo a within-person process of adaptation as assumed hypothetically.

Examination of the distribution of the duration values revealed two outliers with duration values of 240 months, which might unduly affect the coefficients of a polynomial between-subjects function extended over the whole range of duration values. Also, as noted above, these persons did not remember the exact time of AMD diagnosis. Therefore, these outliers were excluded from the analyses involving duration as Level 2 predictor, hence reducing the maximum of included duration values to 148 months. Note that placing node at this maximum duration value implies a simple quadratic model of duration impact over the whole duration range.

All of the models were computed by use of the procedure MIXED (SAS 8.2 software package; SAS Institute, 1999). The restricted maximum likelihood method was chosen to fit the data.

Results

Data Description

Table 1 gives descriptive statistics for PA and NA values used in the statistical analyses. Across the five measurement occasions, mean levels of PA and NA did not change significantly. Note that the statistical tests shown in Table 1 are based on listwise deletion of cases with missing values, which will not be true for the results of the multilevel analyses reported below. Regarding disease duration prior to the first measurement, the participants had been diagnosed with AMD for 45.5 months (\(SD = 37.9\)) on average (two outliers excluded). Notably, the age of the respondents at \(t0\) did not correlate with duration (\(r = -0.10, ns\)), and age at AMD outbreak ranged between 58 and 92 years (average age of AMD outbreak was 75.3 years).

Positive Affect

To demonstrate PA changes in relation to the duration of the disease as observed in the sample, Figure 1A shows the individual PA trajectories over a time axis starting with the outbreak of the disease. As can be seen, severe fluctuations with abrupt turn-arounds cause many overlaps in the curves. To get a clearer picture, in Figure 1B only the endpoints of each individual trajectory, that is, the first and last observed PA value, have been connected, depicting the overall change across the observation period. Despite some remaining “fuzz,” a tendency echoing an adaptation pattern seems to emerge for the period from outbreak of the disease up to about 7 years of disease duration: Within the first 2 years, most of the linear trajectories show decline, but from about the 3rd to the 5th year, more lines increase, whereas most lines in the 5- to 7-year range do not show steeper decline or increase. Furthermore, the lines at the extreme right end of the figure, that is, for those participants with long-lasting AMD, do not show stability but some increases as well as some declines, with the latter appearing somewhat more pronounced.

Overall change in PA. For PA, the intraclass correlation was .61, which indicates that about 40% of the total PA variance is due to intraindividual changes (for computation, see Snijders & Bosker, 1999). Results from running the random-only growth curve models (Functions 1 and 2 combined with Function 3) on the affect measures are shown in Table 2, which provides information for both PA and NA (with the latter addressed below). With respect to PA, the fixed-slope effects are not statistically significant for all models, whereas owing to the single-parameter Wald tests (see Hox, 2002), the random-slope variances are. This means that there is no relevant change in the sample’s mean-level PA across the five measurement occasions, but rather substantial intraindividual variation in the trajectories, as has been predicted for PA in our first hypothesis. However, with the trustworthiness of Wald tests for random components added to the model by specifying between-subjects slope variation (i.e., the random-slope variances and the covariances involving the random-slope components) is not significant for the linear growth model but for the quadratic and piecewise linear model with node 3 (i.e., trajectory “kinked” at fourth measurement occasion).

Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>t0 (n = 90)</th>
<th>t1 (n = 83)</th>
<th>t2 (n = 71)</th>
<th>t3 (n = 68)</th>
<th>t4 (n = 70)</th>
<th>Test*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive affect</td>
<td>3.1</td>
<td>3.3</td>
<td>3.2</td>
<td>3.1</td>
<td>3.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Negative afford</td>
<td>1.9</td>
<td>2.0</td>
<td>1.9</td>
<td>1.9</td>
<td>1.9</td>
<td>0.6</td>
</tr>
</tbody>
</table>

Note. Values refer to means and standard deviations. * = measurement occasion.

*Test for significance of overall change in means (Wilks’s lambda).
With respect to the total PA variance of 0.544, the Level 1 residual variances shown in Table 2 correspond to 35.6%, 26.7%, and 27.4% “unexplained” variance for the linear, quadratic, and piecewise linear growth model, respectively. Thus, the piecewise linear model with node at $t_3$, chosen among the piecewise linear models due to minimal residual variance, fits the data nearly as well as the quadratic model. Notably, the piecewise linear growth model with node at $t_2$ produced 29.1% unexplained residual variance and would hence have been a good choice too, with random-slope variances significant again. Moreover, the cubic growth model fitted only slightly better than the quadratic (25.3% unexplained variance). Therefore, and as the modeling of cubic growth curves over five measurement occasions does not seem to provide any further benefits for the current research question, results of the cubic models are not reported in detail. Thus, a common growth function, which allows for a single change in the general trends of the slopes (quadratic and piecewise linear function), seems adequate to best describe trajectories in PA.

On a more detailed level, there is a statistically significant negative covariance between the piecewise linear model’s first and second slope component that comes to a correlation coefficient of −.69, showing a tendency to slow down or reverse rather than to accelerate the PA changes that occurred initially within the observed time period. Hence, the component of the first hypothesis proposing this tendency is confirmed for PA.

Impact of duration of AMD.

Table 3 shows results for the duration impact models (i.e., between-subjects Function 4 combined with Functions 1 and 2). We left out the models involving the quadratic and cubic within-subject function, because its slope components are more difficult to interpret than those of the piecewise linear growth function, which fitted about equally well. With respect to the first hypothesis, the most relevant statistics shown in Table 3 are the Fixed Slope × Duration interaction effects, which are the coefficients for predicting the slopes from the duration variables. Regarding the piecewise linear growth function, the initial rate of change in PA was hypothesized to be nonlinearly related with duration, hence the interaction of duration with the first linear slope component is particularly crucial.

First, it should be mentioned that modeling the impact of duration in terms of a simple linear between-subjects function (not particularly formulated in the Method section) did not reveal any substantial effects. Regarding “conventional” linear association only would give the impression of total independence of changes in PA from duration of the disease. Turning over to the nonlinear

\[ \text{Figure 1. Observed trajectories of positive affect across duration of age-related macular degeneration (AMD).} \]

A: All five measurement occasions. B: Only measurement occasions $t_0$ and $t_4$. 

With respect to the total PA variance of 0.544, the Level 1 residual variances shown in Table 2 correspond to 35.6%, 26.7%, and 27.4% “unexplained” variance for the linear, quadratic, and piecewise linear growth model, respectively. Thus, the piecewise linear model with node at $t_3$, chosen among the piecewise linear models due to minimal residual variance, fits the data nearly as well as the quadratic model. Notably, the piecewise linear growth model with node at $t_2$ produced 29.1% unexplained residual variance and would hence have been a good choice too, with random-slope variances significant again. Moreover, the cubic growth model fitted only slightly better than the quadratic (25.3% unexplained variance). Therefore, and as the modeling of cubic growth curves over five measurement occasions does not seem to provide any further benefits for the current research question, results of the cubic models are not reported in detail. Thus, a common growth function, which allows for a single change in the general trends of the slopes (quadratic and piecewise linear function), seems adequate to best describe trajectories in PA.

On a more detailed level, there is a statistically significant negative covariance between the piecewise linear model’s first and second slope component that comes to a correlation coefficient of −.69, showing a tendency to slow down or reverse rather than to accelerate the PA changes that occurred initially within the observed time period. Hence, the component of the first hypothesis proposing this tendency is confirmed for PA.

Impact of duration of AMD. Table 3 shows results for the duration impact models (i.e., between-subjects Function 4 combined with Functions 1 and 2). We left out the models involving the quadratic and cubic within-subject function, because its slope components are more difficult to interpret than those of the piecewise linear growth function, which fitted about equally well. With respect to the first hypothesis, the most relevant statistics shown in Table 3 are the Fixed Slope × Duration interaction effects, which are the coefficients for predicting the slopes from the duration variables. Regarding the piecewise linear growth function, the initial rate of change in PA was hypothesized to be nonlinearly related with duration, hence the interaction of duration with the first linear slope component is particularly crucial.

First, it should be mentioned that modeling the impact of duration in terms of a simple linear between-subjects function (not particularly formulated in the Method section) did not reveal any substantial effects. Regarding “conventional” linear association only would give the impression of total independence of changes in PA from duration of the disease. Turning over to the nonlinear
Table 2

Positive and Negative Affect: Results of Random Growth Curve Models

<table>
<thead>
<tr>
<th>Variable</th>
<th>Positive affect</th>
<th>Negative affect</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Linear</td>
<td>Quadratic</td>
<td>Piecewise linear</td>
<td>Linear</td>
<td>Quadratic</td>
</tr>
<tr>
<td>Fixed effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \gamma_{00} ) intercept</td>
<td>3.218***</td>
<td>3.179***</td>
<td>3.213***</td>
<td>1.946***</td>
<td>1.945***</td>
</tr>
<tr>
<td>( \gamma_{10} ) Slope 1*</td>
<td>-0.031</td>
<td>0.066</td>
<td>-0.022</td>
<td>0.001</td>
<td>0.004</td>
</tr>
<tr>
<td>( \gamma_{20} ) Slope 2*</td>
<td>-0.025</td>
<td>-0.027</td>
<td></td>
<td>0.000</td>
<td>-0.030</td>
</tr>
</tbody>
</table>

Random variances and covariances

\( \sigma^2_{\text{intercept}} \) intercept | 0.378***        | 0.442***        | 0.424***    | 0.312*** | 0.324*** | 0.320***    |
\( \sigma^2_{\text{Slope 1}} \) Slope 1 | 0.009*          | 0.165*          | 0.024**     | 0.006    | 0.056   | 0.018*      |
\( \sigma^2_{\text{Slope 2}} \) Slope 2 | 0.011**         | 0.394**         |            | 0.002    | 0.095   |            |
\( \sigma_{\text{Slope 1–Slope 2}} \) intercept–Slope 1 | -0.019          | -0.103          | -0.043      | -0.020   | -0.041  | -0.029      |
\( \sigma_{\text{Slope 1–Slope 2}} \) Slope 2–Slope 1 | 0.019           | 0.090           |            | 0.004    | 0.017   |            |
\( \sigma^2_{\text{Level 1 residual}} \) Level 1 residual | 0.193***        | 0.145***        | 0.149***    | 0.138*** | 0.130*** | 0.130***    |
Random slope \( \chi^2 \) | 3.8             | 16.0**          | 15.7***     | 4.4      | 8.1     | 9.4        |

Note. The random growth curve models use random-only between-subjects function combined with linear, quadratic, or piecewise linear (node \( a = 3 \)) within-subjects function.

* Linear slope component.  
\( \text{b Quadratic or second linear slope component for quadratic or piecewise linear models, respectively.} \)  
\( \text{c Likelihood ratio test for random slope components (SAS Institute, 1999).} \)

* \( p < .05 \).  
\( ** p < .01 \).  
\( *** p < .001 \).

Table 3

Positive Affect: Results of Duration Impact Growth Curve Models

<table>
<thead>
<tr>
<th>Variable</th>
<th>Linear growth</th>
<th>Piecewise linear growth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \gamma_{00} ) intercept</td>
<td>3.637***</td>
<td>3.691***</td>
</tr>
<tr>
<td>( \gamma_{01} ) Duration 1 linear</td>
<td>-0.029**</td>
<td>-0.038**</td>
</tr>
<tr>
<td>( \gamma_{02} ) Duration 2 quadratic</td>
<td>0.0003**</td>
<td>0.0004**</td>
</tr>
<tr>
<td>( \gamma_{10} ) Slope 1</td>
<td>-0.140**</td>
<td>-0.127</td>
</tr>
<tr>
<td>( \gamma_{20} ) Slope 2</td>
<td>-0.103</td>
<td></td>
</tr>
<tr>
<td>( \gamma_{11} ) Slope 1 ( \times ) Duration 1</td>
<td>0.008**</td>
<td>0.009*</td>
</tr>
<tr>
<td>( \gamma_{12} ) Slope 1 ( \times ) Duration 2</td>
<td>-0.0001**</td>
<td>-0.0001*</td>
</tr>
<tr>
<td>( \gamma_{21} ) Slope 2 ( \times ) Duration 1</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>( \gamma_{22} ) Slope 2 ( \times ) Duration 2</td>
<td>0.0000</td>
<td></td>
</tr>
</tbody>
</table>

Random variances and covariances

\( \sigma^2_{\text{intercept}} \) intercept | 0.325***        | 0.374***    |
\( \sigma^2_{\text{Slope 1}} \) Slope 1 | 0.006          | 0.019*      |
\( \sigma^2_{\text{Slope 2}} \) Slope 2 | 0.404**        |            |
\( \sigma_{\text{Slope 1–Slope 2}} \) intercept–Slope 1 | -0.005        | -0.032      |
\( \sigma_{\text{Slope 1–Slope 2}} \) Slope 2–Slope 1 | 0.108         |            |
\( \sigma^2_{\text{Level 1 residual}} \) Level 1 residual | 0.196***      | 0.153***    |
\( \text{d piecewise quadratic node} \) | 90             | 75           |

Note. Duration impact growth curve models use piecewise quadratic duration impact between-subjects function combined with linear and piecewise linear within-subjects function.

* \( p < .05 \).  
\( ** p < .01 \).  
\( *** p < .001 \).
relationships as hypothesized completely alters this impression: The estimates for interaction coefficients $\gamma_{11}$ and $\gamma_{12}$ are statistically significant for the models involving the piecewise quadratic between-subjects function, matching the expectation established with the first hypothesis. This provides support for a quadratic relation of disease duration with the PA slopes (first slope in case of the piecewise linear growth model) over some period after the outbreak of the disease, but no further relation beyond this period. Because of the criterion of maximum reduction in the slope variances explained in the Method section, the end of this period is estimated with about 90 months for the linear growth function and with about 75 months for the piecewise linear growth function.

To illustrate the meaning of these interaction coefficients, Figure 2 shows the curves of the PA slopes predicted from duration according to the results of the piecewise quadratic between-subjects functions. That is, for a given value of AMD duration, the curves show the rate of change in PA, which results from the fixed-effects estimates as shown in Table 3 (i.e., $\gamma_{10} + \gamma_{11}d + \gamma_{12}d^2$, where $d$ denotes the duration variable). Up to AMD duration of about 5 to 6 years, all curves show the hypothesized adaptational pattern: The initial phase of the chronic condition (minimum duration) starts with highest negative rates of change in PA. But with longer duration, PA declines get smaller, reaching the zero point at about 2 years of AMD duration. Moreover, within the array of about 2 to 6 years of duration the curves run above zero; that is, increases in PA are predicted, which fade out with the curve again reaching zero at about 5 to 6 years of duration. However, beyond 6 years of duration, the curves go down again, predicting ongoing PA declines, which were not expected hypothetically.

Considering the size of duration effects in terms of shares of explained variance is quite complicated (see discussion provided by Snijders & Bosker, 1999, pp. 108–109). To get some information in this regard, we proceeded as follows: Making use of the formulae proposed by Snijders and Bosker to compute the contribution of the random slopes to the total PA variance, we compared the random-slope variance contributions for the models with and without duration as Level 2 predictor. This reveals the reduction of random-slope variance achieved by predicting the slopes from duration. Following this strategy, the reductions of random-slope variance are 6.6% for the piecewise linear growth model and 37.0% for the linear growth model. For the piecewise linear growth model, the percentages of random-slope variance reduction might be lower because in this model duration predicts only the first of two slopes, leaving the variation in the second slope as vastly unpredicted contribution to the model’s random-slope variance. Thus, the lower percentage of reduction could paradoxically be seen as the price paid for greater accuracy in predicting only the initial rates of change in the trajectories, instead of the average overall intraindividual change rates.

It should also be regarded that the estimated fixed effects of duration (parameters $\gamma_{01}$ and $\gamma_{02}$) are significant for all the models shown in Table 3. These effects reflect the impact of duration on the level of PA at first measurement occasion $t_0$. With respect to these effects, it must be understood that adaptation also implies a nonlinear relationship of duration with the $t_0$ level of PA due to the adaptational changes that should have occurred before the first measurement. The estimated values for $\gamma_{01}$ and $\gamma_{02}$ are in line with this implication, predicting early decline, turning around into subsequent increase of the PA levels, in relation to the duration of AMD.

Taken altogether, the results of the multilevel models confirm the assumed pattern of adaptational changes for PA as stated in our first hypothesis. Results suggest an adaptation process with initial decline and subsequent restoration in PA lasting over about 6 years after outbreak of AMD. It should also be noted that in view of the broad range of age at AMD outbreak and the nonexistent correlation of duration with chronological age, the nonlinear relation of slopes with disease duration does not simply reflect some age-related process of PA changes. This was also confirmed by controlling for age in an additional analysis: Inclusion of age at $t_0$ as linear Level 2 predictor in the models reported in Table 3 did not change the pattern of significances.

**Negative Affect**

*Overall change in NA.* The intraclass correlation for NA was .63, showing again a substantial share of variance due to intraindividual variation. However, we hypothesized to find only tenta-

![Figure 2](image-url)  
*Figure 2.* Rates of change in positive affect predicted from duration of age-related macular degeneration (AMD) according to the results of piecewise quadratic duration impact growth curve models.
tive adaptational changes in NA, if any. Results for the randomly growth curve models (Functions 1 and 2 combined with Function 3) are also shown in Table 2. Again, as is the case for PA, the fixed-slope effects are not statistically significant for all models. However, unlike PA, the random-slope variances also were not significant, with the exception of the single parameter Wald test regarding the between-subjects variance of the first linear slope of the piecewise linear model (for which again node \( t_3 \) yielded minimum Level 1 residual variance). Also, the likelihood ratio tests for the random-slope components are not significant. The Level 1 residual variances shown in Table 2 add up to 33.5%, 31.5%, and 31.6% unexplained variance for the linear, quadratic, and piecewise linear growth models, respectively. Thus, the nonlinear growth functions fit the NA trajectories only slightly better than the linear function (which is also true with respect to the cubic function, which revealed 30.8% residual variance).

In sum, the participants of our sample have not been found to differ largely in the amount of changes in NA observed across the four subsequent measurement intervals. Therefore, between-subjects variability in within-subject change, proposed as a fundamental property of a pattern of adaptation, cannot be confirmed for the NA scores. Altogether, with insignificant fixed- and random-slope effects, the growth models provide support for our second hypothesis.

Impact of duration of AMD. Strictly speaking, without significant slope variance, computation of the models including any Level 2 predictor is useless, because there is no random-slope variance to be explained any further. However, to complete the analysis of a tentative pattern of adaptational change, the same analyses for PA were run for NA. The results can be summarized as follows: No significant interaction of the duration terms with the slope components was detected in any of the models computed, and reductions in the slope variances obtained from predicting the slopes from duration were close to zero. Altogether, in the analyses of the impact of disease duration on NA scores, the assumed nonlinear pattern of relation with duration reflecting adaptability of NA could not even be observed tentatively.

Discussion

The goal of this study was to empirically test a hypothesized longitudinal pattern of change in affective well-being reflecting a process of adaptation to the experience of AMD. Regarding PA, the results are largely in line with the expectations stated in our first hypothesis. First, we found evidence for a between-subjects variation in the average rates of PA change across the four subsequent measurement intervals and, in particular, strong evidence for substantial between-subjects variation in change rates if these are allowed to alter intraindividually over the observation period. Moreover, the suitability of the nonlinear piecewise linear growth functions, disclosing a tendency to reverse or slow down the initial change rates in PA within our observation period, is in line with the assumed pattern of adaptational changes. Second, we found evidence that the PA change rates were substantially related with the duration of disease in a nonlinear fashion, with rapid PA declines in the initial phase of the disease, followed by a period of recovery with PA increasing again, ending at some point of duration with no further duration-related change in PA.

Notably, our results on the relation of PA with AMD duration are similar to findings presented by Evans (1983), which supported a nonlinear relation of depression with AMD duration. Our findings also suggest that the phase of adaptational changes for PA may last, on average, for the first 6 years after the outbreak of AMD. However, regarding the postadaptational phase of AMD duration, our mixed models estimates suggest that duration impact on the PA slopes ends up with a constant negative value after this 6-year period (as depicted in Figure 2). This means ongoing decline of PA in later phases of disease duration, which might be interpreted rather pessimistically as some kind of failure of adaptation in the long run, in the sense that most people go through a period of PA “recovery,” but finally the continuous burden of ongoing disease progression, for example in terms of increasing levels of functional disability, deletes all adaptive gains. It is also notable that a cumulative effect probably occurs, in that comorbidity increases substantially as people age. This interpretation would be in accord with the literature on limits of psychological resilience in those particularly burdened by multimorbidity over many years (Staudinger, Freund, Linden, & Maas, 1999; Wahl, Schilling, Oswald, & Heyl, 1999). A less pessimistic interpretation might be that the constant negative rate of change in PA predicted for later phases of disease duration reflects normal age-related decline that would have been observed as well in a group of older adults without any chronic condition. That is, the equilibrium finally restored with the adaptation might consist in a moderate, but continuous PA decline. Exploring this latter possibility would be an interesting research perspective extending what we have done in the present study.

Concerning NA, we did not expect a pattern of adaptational changes, because prior research and theory indicates that the frequency of experiencing negative emotions might be mainly dispositional (Baker et al., 1992; Clark & Watson, 1991a). The consequence of this assumption would be that NA might rather be intraindividually stable, regardless of what happens in a person’s life, than adaptive in the sense of a process of reduction of emotional reactions to outlasting adverse conditions. In view of our results, it must be concluded that for NA the pattern of adaptational change was not even tentatively found. However, it might be questioned if this finding appeared due to dispositional stability of NA, as the intraclass correlation indicates a substantial amount of intraindividual NA variation, which is only slightly lower compared with the PA intraclass correlation. Yet, NA varied less than PA in terms of absolute variance. Thus, there is some intraindividual variation in NA, which is lower than in PA and comes with no trend of mean-level change. This might be interpreted in terms of fluctuation of NA values around individual NA levels, which are kept stable over time. This fluctuation, however, deserves further scientific interest, as it might also reflect short-term adaptation to changing conditions, too fast to be uncovered by the 3-month intervals of our study design. Thus, in view of our results, the conclusion of NA stability can be made with caution only.

Of course, the results of the present study come with limitations, which we highlight in some depth because they also echo general research challenges of future research on adaptation. First, the total sample size of 90 participants with AMD may be regarded as too small to draw definite conclusions on the existence and time span of a process of adaptation. This is particularly evident in Figure 2,
which shows these 90 participants distributed over a broad range of disease duration with only few participants within episodes of the disease deserving special consideration in an adaptation-driven theoretical approach (such as, e.g., the initial phase up to about 2 years after outbreak). With many more participants in each segment of the duration axis, the finding of the adaptation pattern as presented above would thus clearly rest on stronger evidence. Also, it might be argued that five measures of PA and NA per participant covering a 1-year observational period are too few to analyze the process of adaptation. Eid and Diener (1999) emphasized substantial within-subject variation of affect in very short terms (e.g., within a day). Regarding such fluctuation, it might be argued that within-subject variation in our measures reflects this short-term “noise” of affect values rather than a trend over the time units of our study. However, the PANAS instruction used a 1-week time frame, measuring affect aggregated over 1 week and not the fluctuant mood of the moment. This may mitigate the objection that short-term mood fluctuation may have been the major contribution to intrindividual variance observed in our affect measures.

With respect to our aim of presenting a conceptual and methodological approach to the study of adaptation, a limitation has to be made regarding the adequate timing of the longitudinal design. In particular, without prior knowledge about the speed of the hypothesized adaptation process, the measurement intervals may be too short or too large to uncover the adaptational changes. In our study, we could not be sure that the 3-month measurement intervals over the 1-year period chosen for our longitudinal design were optimal. However, taking into account the typical time frame of AMD progression, with visual losses occurring in small increments over a period of years (e.g., Holz et al., 2003), it could be assumed that adaptation after the first emergence of AMD symptoms may not have been completed for most participants in our sample before our first measurement occasion.

Another argument regarding the timing aspect could be made with respect to the speed of the functional ability losses caused by AMD and their particular impact on NA. It may take years of disease progression until a critical level of functional disability is reached, capable to cause increases in NA. In our sample there appeared no tendency of NA increase, even for those with 10 or more years of AMD duration; the results would suggest that this point of critical functional loss comes, on average, very late in AMD progression, such that only few adults facing AMD outbreak in their 60+ ages survive up to this point. However, given the low sample size, this statement can only be made tentatively.

It might also be questioned if duration measured as time since AMD was diagnosed is the optimal choice to study adaptation. Adaptation may have begun before this point, as the participants might have experienced symptoms of the disease before the diagnosis of AMD was made. Others might have been diagnosed with AMD in routine ophthalmologic screenings before they perceived substantial visual losses. Therefore, the first subjective perception of symptoms may be a more adequate point in time to define duration of the chronic condition. However, exact information on this starting point might not be easily obtained by retrospective self-reports, in particular if provided by respondents with long-lasting AMD, whereas prospective longitudinal studies on AMD development, starting with random samples of respondents without any visual problems and large enough to reveal a substantial number of AMD patients across the follow-up, would be quite expensive. Thus, disease duration in terms of time since diagnosis should be regarded at best as a proxy variable, useful to overcome practical problems in defining the duration of the chronic condition. It should also be considered that the diagnosis itself, confronting a person with the verdict of unavoidable and irreversible vision loss of unpredictable severity, may cause an emotional burden as such, provoking adaptational processes.

Concerning the role of duration for the concept of adaptation on which we based our analyses, a limitation has to be made with regard to possible modulations in the speed of visual decline caused by AMD. Periods with rapid aggravation of visual impairment may alternate with periods of only slight or no aggravation at all (Holz et al., 2003). Acute episodes of visual aggravation might produce emotional discomfort in every phase of disease continuation, and rather than working as a long-term process of initial worsening and subsequent restoration, starting from outbreak of the disease, adaptation of affective well-being might work in the short term, reactive to such acute episodes of visual loss. However, despite fluctuation of rapid and slow progression episodes across intra-individual AMD developments, AMD is marked by some vision loss in the long run for all participants affected by the disease. Therefore, we could expect a pattern of adaptational changes with respect to the general trends in intra-individual developments of affect measures, apart from oscillation in the trajectories due to variations in the speed of disease progression. Nevertheless, analysis of adaptational change could profit from more in-depth analyses of time series of affect measures related to detailed measurement of changes in subjective vision function.

In general, more in-depth analyses concerning the physical and psychosocial drivers and the psychological mechanisms of adaptational changes would be desirable to gain better insight into the psychological consequences of AMD. A number of such studies have revealed evidence on specific factors that might impact on affective outcomes, particularly on depression (e.g., Boerner, 2004; Horowitz, Reinhardt, & Boerner, 2005; Horowitz, Reinhardt, Boerner, & Travis, 2003; Reinhardt, 2001; Tolman, Hill, Kleinschmidt, & Gregg, 2005). It seems promising to connect this line of research with the analysis of adaptational change in PA and NA as proposed in the present article, modeling the impact of specific affect-relevant predictors on change in PA and NA. However, such detailed analysis was beyond the scope of the present work. Given longitudinal data containing possible covariates of the AMD adaptation process (e.g., coping strategies, comorbidity information), the statistical modeling as presented above could be expanded to comprise particular predictors of adaptational change. Moreover, we regard our analyses more as a bottom-line methodological approach and capable of dealing with additional particularities of adaptational changes, such as considering nonlinearities of intra-individual trajectories in more than one variable. This would also require larger sample sizes than what we had at our disposal in the present research.

Altogether, study designs with larger sample sizes and more measurement occasions over shorter measurement intervals would be needed, which could only be realized with high costs and might unduly burden the respondents with AMD. However, finding substantial effects as predicted from our first hypothesis for PA despite a number of major limitations is promising, because these limitations could be expected to obscure rather than unfold the observed effects.
Finally, we would like to emphasize again that we intended with our work to present a conceptual and methodological approach that might be useful for further research on adaptation not only to AMD but also to other chronic conditions frequent in old age. For example, dealing with strongly age-related chronic conditions such as arthritis or cancer may be ideal candidates for such future research. In any case, the suggestion is to test for a longitudinal pattern reflecting adaptation to a chronic condition, focusing on variability of intrapersonal trajectories due to the dynamics of an adaptive process of initial decline and subsequent restoration. This also implies nonlinearities in individual trajectories as well as in the “change in change,” that is, in the development of individual change rates due to the duration of the adaptation process. Regarding statistical methodology for such empirical analysis, this conceptual pattern of adaptation can be translated into multilevel regression models by modeling nonlinear functional relationships. In conclusion, the conceptual pattern of adaptation, as well as the statistical modeling presented above, may serve as a general and still underused strategy to study adaptation that is capable of providing further insight into the existence, magnitude, and temporal extension of adaptive changes under chronic health conditions.

References


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