Affective Development in Advanced Old Age: Analyses of Terminal Change in Positive and Negative Affect

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Late-life development of affect may unfold terminal changes that are driven more by end-of-life processes and not so much by time since birth. This study aimed to explore time-to-death-related effects in measures of affect in a sample of the very old. We used longitudinal data (2 measurement occasions: 2002 and 2003) from 140 deceased participants, covering a period of up to 9 years to death, from a German oldest-old sample between 80 and 90 years old at baseline measurement (Projects ENABLE-AGE and LateLine). Nonlinear time-to-death and age effects on baseline level and intraindividual 1-year changes in positive affect (PA) and negative affect (NA) were analyzed with latent change score models. With respect to PA, no time-to-death-related effects were discovered, but linear age-related decline was found. For NA, time-to-death effects were found for both baseline level and 1-year change, indicating an increase of NA closer to death, which turns around to a rapid reduction of NA in the approach of the end of life. These effects suggest a twofold dynamic of terminal changes in NA. First, a general increase of NA across a larger period preceding death potentially mirrors basic processes of degradation of the human system. Second, approaching the end, these processes may exhaust negative activation, prompting a terminal drop of NA close to death.

Keywords: affect, advanced old age, terminal decline, time to death

In this study, we conducted analyses of terminal development of affect at the end of the human life span. We argue that in advanced old age, changes of affective experiences may—rather than being influenced by age-graded (i.e., distance-from-birth) processes—be driven by processes of degradation and loss that unfold in the approach of death. Thus, a distance-to-death perspective may contribute to a better understanding of affective development in late life. To do so, we analyzed change related with time to death (TTD) in repeated measures of positive affect (PA) and negative affect (NA) in a sample of the very old.

The Distance-to-Death Perspective in Late Life Development

A huge body of research has centered on the beginning of human development and the first years of the life span. In sharp contrast, the very last years of the life span and the end of human development have been given only limited consideration. Compared with questions concerning development at the first years of the human life span, the question “What is going on in the last years of the human life span?” is still much less answered. Two classic answers have been provided by developmental scientist Erik H. Erikson and by the advent of thanatology or, as this field of study has been labeled during the recent decades, the death and dying research. Erikson’s (1950) unique contribution was not only the explicit spelling-out of a full life span developmental sequence; even more groundbreaking was his assumption that human development may involve advancement until the end of life—a possibility Erikson coined ego integrity. The seminal contribution of the death and dying literature (Kastenbaum, 2000) to human development may be recognized by the argument that every developmental model remains incomplete as long as it does not involve research attention paid to the end of life as well as to any other developmental phase of the life span. Going further, while the Eriksonian approach starts from birth and acknowledges development as an age-driven phenomenon, thanatology starts from the end of life and asks for the impacts of the event of death on humans knowing about their finitude. Bringing both of these perspectives together, a central developmental research question comes to the fore: Is there a development corridor in the human life span, in which one developmental force, age, loses importance and another developmental force, death, is taking over? It has been the merit of Robert Kleemeier (1962) to translate this fundamental question to the empirical world of aging by inaugurating the concept of terminal decline, which implies a distance-to-death perspective of human development.

The basic idea underlying the distance-to-death perspective is that multiple degrading processes of biological systems at the end of life (e.g., cerebrovascular diseases, reduction of immune functioning) might also stimulate change in psychological functioning.
However, due to varying patterns of genetic and contextual impacts on the aging process, individuals differ in the ages at which biological loss accumulates to the degree of collapsing into terminal dynamics leading to death. Thus, psychological changes triggered by such terminal dynamics are less accounted for by the individuals’ calendar age than by their proximity to death, meaning time-to-death (TTD)-related change in the variables affected. Because TTD-related psychological change is expected as a consequence of biological degradation and physical loss, it seems coherent to consider it primarily as change for the worse, that is, as terminal decline or terminal drop in psychological functioning (for definition and differentiation of these constructs, see Bäckman & MacDonald, 2006). However, it should be noted that terminal change could refer to any kind of TTD-related changes in psychological aspects and must not be restricted theoretically to patterns of decline or dropping. It may also be considered that psychological adaptation to and coping with terminal degradation leads to protective changes of affective reactions, providing relief from rather than increase of emotional burden in the face of impending death. Such processes may reflect a key component of Erikson’s ego integrity, namely that “death loses its sting.” (1950, p. 269).

Research on Terminal Change in Psychological Domains

Across more than 40 years after its inception, the distance-to-death approach has nearly exclusively been followed in the cognitive aging research, providing a large body of studies dealing with terminal decline in cognitive function (Bäckman & MacDonald, 2006). However, more recently, noncognitive areas, such as subjective well-being, also became the target of empirical research with distance-to-death focus. Findings suggesting terminal decline in life satisfaction, the cognitive component of subjective well-being (Diener, Suh, Lucas, & Smith, 1999), have been presented by work of Gerstorf and colleagues (Gerstorf et al., 2010; Gerstorf, Ram, Estabrook, et al., 2008; Gerstorf, Ram, Röcke, Lindenberger, & Smith, 2008) and by other studies as well (Berg, Hassing, Thorvaldsson, & Johansson, 2011; Mroczek & Spiro, 2005; Palgi et al., 2010). Thus, converging evidence supports a pronounced decline dynamic of life satisfaction as individuals approach their death.

In contrast to cognitive components of well-being, affective development has so far rarely been targeted in terms of terminal change. To our knowledge, only two studies have applied the distance-to-death focus to the analysis of changes in affect in old age. Gerstorf et al. (2010) used data from nationally representative panel studies from Germany, the United Kingdom, and the United States to analyze terminal change in well-being measures, which were in the British and the U.S. samples indicators of affective well-being. Covering multiple measurement waves and large numbers of respondents from the full adult age range (United Kingdom: 15 annual waves, 2,030 respondents; United States: six biannual waves, 6,195 respondents), both samples revealed a phase of steep decline of affective well-being prior to death, starting at about 3 or 5 years before death, respectively. As the study focus was on terminal change in well-being in general, Gerstorf and colleagues analyzed only global indicators of affective well-being (United Kingdom: sum-score of 12 items from the General Health Questionnaire; Goldberg, 1978; United States: eight items from the Center for Epidemiological Studies–Depression Scale, or CES–D; Radloff, 1977). Thus, from the viewpoint of affect—rather than well-being—research, questions on terminal changes in affect remain to be researched, concerning specific end-of-life dynamics in positive affect (PA) and negative affect (NA), which are viewed as mutually independent dimensions of emotional experiences by many researches in the field of emotion (e.g., Watson, Vaidya, Vaidya, & Tellegen, 1999).

Palgi et al. (2010) analyzed TTD- and age-related change in several measures, including CES–D scores of PA and depressed affect (used as measure of NA) obtained from an Israeli aging study (involving three measurement waves and 1,369 respondents, ages 75–94 at first measurement). They found an effect of general TTD-related increase (but no significant age-related change) in NA, whereas neither TTD- nor age-related change in PA was significant (but TTD-related decline in PA became significant after controlling for several covariates, including age, gender, education, and marital status). For both affect outcomes, the TTD-related change model performed better than the age-related change model in terms of model fit and variance explained. Thus, these findings suggest some TTD-related worsening of affective well-being, mainly due to increase of NA (but not so much decline in PA).

However, their findings are limited due to the longitudinal study design, because modeling TTD-related linear trajectories over three measurements with large time intervals of about 3 and 8 years in between, as Palgi and colleagues did, may underestimate rapid terminal changes that unfold rather close to the end of life.

Overall, the findings presented by Gerstorf et al. (2010) and Palgi et al. (2010) suggest distance-to-death-related changes of affect close to death. However, with the data they could use, Gerstorf and colleagues could not analyze terminal changes differentiating PA and NA, whereas Palgi and colleagues could only analyze change over time-intervals that may have been too long to fit to the speed of terminal changes. Thus, much more research is needed to reach conclusive knowledge about terminal development of PA and NA. In the present study, we add to this research a TTD analysis of 1-year change of PA and NA in advanced old age.

Palgi et al.’s (2010) finding of larger TTD-related changeability of NA compared with PA contrasts with available data on “normative” age-related development of affect, showing the reversed pattern of more changeability of PA than NA. Cross-sectional as well as longitudinal findings suggest that PA declines slightly in advanced old age, whereas NA is kept relatively stable after the age 60 (Charles, Reynolds, & Gatz, 2001; Kunzmann, 2008; Kunzmann, Little, & Smith, 2000). Pinquart’s (2001) meta-analysis of age effects on PA and NA in old age, mainly from cross-sectional studies, revealed a general tendency of age-related decline of PA, but only minor increase of NA related to age; both PA and NA had a curvilinear trend of acceleration across the old age range, resulting in more pronounced worsening of affective well-being in very old age. However, minor age-related change of PA or NA may be found even in the presence of much more pronounced terminal changes, as individuals differ in their age at reaching the terminal phase, such that terminal change occurs only in few “young old” study subjects, but is observable among most studied at very old age. If so, curvilinear trends as evidenced by Pinquart (2001) may occur in age-related changes of affect. Overall, research on age-related changeability in old age provides only
little evidence with respect to terminal changes of the affect components, but in view of the weak evidence of age-graded changes in affect in old age, a distance-to-death perspective may contribute to a better understanding of affective development in late life.

**Theoretical Considerations on Terminal Change of Positive and Negative Affect**

When distance-to-death development of affect is the target of analysis, the theoretical status of PA and NA in terminal processes must be considered. From the viewpoint of well-being research (e.g., Diener, Suh, Lucas, & Smith, 1999), PA and NA, viewed as the affective components of subjective well-being, are typically treated as outcome measures reflecting more or less optimal adaptation to the individual’s living conditions. That is, developmental changes in affective well-being are considered as kind of a distal endpoint of causal pathways arising from basic processes, which may be regarded as driving forces of human development (such as degradations of biological function or changes in social or environmental conditions). This view also implies attention for “something in-between” the changing conditions and the affect outcome, mediating this causal relationship, which may be targeted in terms of affect regulation, coping, or adaptation (e.g., Schilling, Wahl, Horowitz, Reinhardt, & Boerner, 2011).

By contrast, personality and emotion psychology have provided views of human affect not simply as a distal outcome, but also as a more central process able to regulate adaptation. PA and NA have been considered in this theory tradition as part of distinct motivational systems (Fowles, 1987; Gable, Reis, & Elliot, 2003; Quilty & Oakman, 2004; Watson et al., 1999). That is, NA was considered as subjective component of a behavioral inhibition system, serving to enhance reactivity to (in order to avoid) negative stimuli. PA serves the operation of a behavioral activation system, which promotes individuals’ engagement in activities that yield pleasure and reward. Concluding from these theoretic approaches, fundamental differences between PA and NA should be considered as crucial for a distance-to-death perspective of PA and NA development.

First, PA is directed toward activation to gain positive experiences and positive stimulation, which is found in contact with the “outside world.” Thus, PA involves the individual’s alertness to the success of his or her behavior, and the generation of PA can be constrained by external conditions that reduce individual prohedonic activity. Kunzmann (2008) termed this an outward focus inherent in PA. If it is true that end-of-life processes degrade basic biological functions (such as brain function, cardiovascular functions, and so on), such degradation may interfere with the individual’s approach to the outside world, disabling prohedonic activities that produce a gain in positive stimulation. Thus, terminal processes may affect PA, in that they cause losses of health and physical function, which constrain the person’s everyday life conduct such that less PA can be produced.

Second, NA is directed toward behavioral inhibition and avoidance of negative stimuli. Thus, individual activity, requiring attention to external conditions that provide opportunities to gain positive experiences, is not a prerequisite of NA generation. Rather, NA is considered in terms of an expression of internal aversions toward stimuli, met passively in the outside world or in the internal “biological environment” (e.g., physical malfunctions). Kunzmann (2008) termed this an inward focus on internal perceptions (e.g., low self-esteem, health symptoms, and so forth) inherent in NA. If biological degradations occur in the end-of-life phase, NA could be expected to mirror these internal processes more directly than PA, which has to take a “detour” via experiences made in outside world contacts. Thus, the inward-focused NA component may be a highly sensitive and unmediated indicator of basic distance-to-death-related degradation. Notably, the findings reported by Palgi et al. (2010) provide tentative evidence of larger terminal change in NA, compared with PA.

To summarize, we assume that terminal decline in PA may occur as a more distal outcome of basic degradation processes close to life’s end, mediated by interferences with the person’s approach to the outside world. However, sensitivity of PA to terminal processes could be lowered to the extent to which individuals are able to adapt to functional declines even in the terminal phase of life, maintaining prohedonic activities. Also, if basic terminal processes do not constrain prohedonic activities, no respective terminal changes in PA would be expected. In contrast, NA is not considered a distal outcome of terminal processes and distance-to-death development; that is, NA is expected to represent a direct and unmediated implication of end-of-life degradations of biological functions. This leads to the overall prediction that a terminal increase of NA may occur in the approach of death.

**Terminal Decline and Affect Arousal: Does It Matter?**

In addition to the foregoing expectations of worsening emotions in terms of direct terminal increase of NA and a more indirect terminal decline of PA, overall affective arousal may become important. The two-dimensional circumplex models that by now dominate the field of affect (e.g., Russell & Carroll, 1999; Tellegen, Watson, & Clark, 1999) employ the dimensions of valence (positive vs. negative, pleasant vs. unpleasant) and arousal to describe the structure of affect. The arousal component denotes the degree of physical activation inherent in an emotional experience. It may be asked with respect to processes of biological degradation unfolding at the end of life if these do apply on affective arousal, apart from considerations with respect to the valence-oriented distinction of PA versus NA.

Terminal processes at the biological level may work in terms of a loss of vigor including affective arousal. Thus, highly activated affective experiences may decline with distance-to-death, and this effect would appear particularly in measures of high arousal affect. In this study, we used measures regarded as indicators of high-arousal PA and NA (see Methods section), such that in our data loss of affective arousal may add to losses of positive stimulation in promoting terminal decline in PA. However, this consideration adds complexity to the expectation of terminal change in NA as follows: Whereas the basic function of NA, signaling internal perceptions of negative states, may mirror terminal degradations by means of terminal increase in NA, loss of affective arousal could counteract this dynamic, providing terminal decline of high-arousal NA. Moreover, such decline of NA activation as death comes closer could be adaptive for individuals: to avoid excessive emotional burden when approaching the end of life. Overall, concerning terminal decline in affective arousal complicates hy-
Aims and Hypotheses

In this study, we sought to analyze terminal changes in PA and NA in very old age. We analyzed affect with distance-to-death relations, exceeding and not attributable to age-related change of affect. Overall, we expected that distance to death would outperform age in accounting for changes in affect in very old age; hence, stronger TTD effects on change in PA and NA should be found compared with respective age effects. Moreover, we expected stronger effects of TTD on NA, as compared with PA, because theoretical considerations of the psychological functions of PA and NA suggest that PA is a more distal outcome of such degradation processes. As outlined previously, we expected in particular that end-of-life degradation of biological functions would promote terminal decline in PA and terminal increase of NA.

However, these expectations may be counteracted by a loss of affective arousal due to terminal degradations. Therefore—and in view of rare empirical research on this topic conducted so far—we regarded these hypothetical expectations as tentative only and conducted this study as a more explorative analysis in order to further the empirical evidence of terminal changes in affect in very old individuals. Being able to use data from a sample of the very old provides a particular opportunity to study terminal processes, because samples including participants who died at rather young ages may contain larger numbers of death due to acute causes, such as fatal injuries and acute lethal diseases. Such acute causes of death bring on lethal effects within short periods of time, instead of longer lasting terminal degradation processes, which are considered the core of TTD-related change.

Method

Sample

We analyzed data from the German subsample of the European ENABLE-AGE Project, which was a random sample of community-dwelling elders living in the Heidelberg and Mannheim areas who were ages 81–90 at first-measurement point in 2002 (T1; N = 450, 21.6% male) and were surveyed again in 2003 (T2; for more details on the sample and the ENABLE-AGE Study, see Iwarsson et al., 2007). Since 2009, we have continued to follow up the German ENABLE-AGE subsample, that is, the T2 participants who consented to participate in future surveys (N = 322), including a study we called LateLine. Starting the LateLine Study (comprising measurement in 2009 and 1-year follow-ups in 2010 and 2011, ongoing with half-year follow-ups since), we conducted a survey of mortality after T2. For those who could not be contacted any more, we retrieved survival information from registration offices and relatives and obtained complete records of cases and dates of death. With continued monitoring of cases of death, we have recorded 140 (43.5%) participants who died between T2 measurement and January 2012; these constitute the sample analyzed in the present study.

Measures

PA and NA were measured with the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS comprises a 20-item questionnaire of 10 positive and 10 negative emotion adjectives; respondents are asked how often they have experienced these emotions over a prespecified time period, which in our study was the past year. Answers are provided on 5-point Likert-scales. For descriptive purposes, we report meanscores of PA and NA, that is, the individual mean value of the positive and negative affect items, respectively (higher values indicating more PA or NA). The PANAS, though well established as instrument to measure affect, implies a high activation component of affect experiences, in that it includes high-arousal emotion items only (Russell & Carroll, 1999; Tellegen et al., 1999).

TTD was recorded as distance in months between the T1 interview date and the date of the participant’s death. For the structural equation model analyses (reported later), TTD was divided by 12 to be scaled in years.

Statistical Modeling

We analyzed TTD relationship with the affect variables by means of structural equation modeling, testing a latent change scores model (e.g., McArdle, 2009), as shown in Figure 1, Model 1. Latent scores for T1 (LS1) and T2 (LS2) affect (i.e., PA or NA) were modeled by use of parcel indicators. The latent change score (LCS) represents the absolute T1–T2 change of latent affect. To generate three parcel indicators from 10 PANAS items for PA and NA, respectively, we proceeded for each dimension as follows: We first averaged the two items showing lowest correlation with total (of other items) and assigned the resulting nine items randomly to the parcels, each computed as three-item mean score. With respect to the modeling of the mean structure, we followed the approach as suggested by McArdle (2009, see their Figure 4b). Note that in Figure 1 the bended double arrow between LS1 and LCS is meant to symbolize covarying residuals. All models were computed under constraints of mild measurement invariance (of respective LS1 and LS2 factor loadings), which caused only slight and insignificant reduction of overall model fit.

In Model 1, TTD and TTD squared (both at T1) were used as predictors of LCS, implying a curvilinear relationship between TTD and 1-year change in the affect outcome. This means that T1–T2 change is enabled to increase or decrease across TTD not...
only by a constant linear rate, but that this increase or decrease may also accelerate in the approach of death. However, a quadratic curvature of LCS over TTD implies a cubic polynomial relationship between LS1 and TTD, as the change in affect depends on TTD due to the first derivate of the affect-on-TTD function. Thus, we also included TTD cubed as predictor of LS1. To distinguish TTD effects from age effects, we added age, age squared and age cubed (each at T1 measurement) as predictors. Overall, the Model 1 functional relationships of T1 level and T1–T2 change with TTD and age due to Model 1 are:

\[ \text{LS1} = b_{10} + b_{11} T + b_{12} T^2 + b_{13} A + b_{14} A^2 + b_{15} A^3 + D_1, \]

and

\[ \text{LCS} = b_{20} + b_{21} T + b_{22} T^2 + b_{23} A + b_{24} A^2 + D_2, \]

where \( T \) and \( A \) are TTD and age at T1, respectively, the \( bs \) denote the path coefficients, and \( D_1 \) and \( D_2 \) denote the residuals. However, the model of TTD- and age-related processes due to these equations indicates that the values of the LCS equation path coefficients relate to the values of the LS1 path coefficients as follows:\(^1\)

\[ b_{20} = b_{10} + b_{12} - b_{11}; b_{21} = b_{13} - 2b_{12}; b_{22} = -3b_{13}; b_{23} = 3b_{14} + 2b_{15}; \text{and } b_{24} = 3b_{16}. \]

We specified these implied relations by means of respective parameter constraints on the path coefficients. In addition, we tested for comparative reasons several simpler versions of Model 1, containing only TTD, TTD squared, and TTD cubed (Model 2) or only age, age squared, and age cubed (Model 3) as predictors.

Overall, the cubic curvature of the LS1 component in Models 1 and 2 provides large flexibility with respect to the shape of TTD-related affect trajectories, allowing not only for an acceleration (or slow down) of the intraindividual change in terms of a linear increase (or decrease) of the rates of 1-year change closer to death, but also for such an effect becoming more pronounced, “accelerating the acceleration” in terms of a curvilinear increase of the rate of 1-year change toward the end of life. Thus, the model could fit to acceleration as imposed by the terminal drop pattern (cf. Bäckman & MacDonald, 2006) and even stronger or more complex patterns of accelerating change.

With respect to the TTD-related curvature of the LS1 level component, however, the cubic model may impose some artificially S-shaped relationship, which may be counterintuitive to the pattern of terminal change expected theoretically. That is, the typical cubic slope involves a twofold bended curve, which would mean that there are two periods of more rapid change in the affect outcome across the TTD range, instead of only one such rapid change period toward the end of life. Therefore, we checked also for TTD-related curvatures, which are intuitively more consistent with the idea of a single rapid change period at the far end of the life span. First, we tested a model with an only quadratic TTD-related curvature of LS1, which implies an only linear TTD effect on LCS. Second, we modeled an exponential TTD function for LS1 and LCS, which implies a nonlinear curvature approximating a linear relation under a larger distance to death, from which it deviates exponentially getting close to death.\(^2\) We checked the results in particular for the \( R^2 \) of predicting LS1 and LCS. Overall, compared with the cubic models, all these models could not improve the prediction of LS1 and LCS and revealed lower values of \( R^2 \) for both, PA and NA, and even model fit indexes were worse; hence, we do not report these results.

For Model 1–3 computations, we mean-centered TTD and age to facilitate interpretation of the coefficient estimates. To avoid collinearity with the squared and cubed predictors, we residualized these on TTD or age (this implies some minor changes in the coefficient constraints denoted previously, these were modified accordingly). We performed all analyses using Mplus software and full information maximum likelihood (ML) estimation (Muthén & Muthén, 2006). To deal with potential nonnormality, we performed all analyses additionally with the Mplus robust ML estimation option, and we used bootstrapping, which enables nonparametric confidence interval estimation for all free model parameters, unbiased by skewed distribution. The results of significance tests based on the bootstrap confidence intervals did not differ from those based on the standard normal distribution of the parameter estimates, and the robust ML estimates did not differ in more than decimals from the “conventional” ML results; hence, we report the latter. With TTD data from only 140 deceased, estimation may be hampered by low power; hence, we applied the more liberal one-tailed 5% significance level to test the effects.

### Results

**Descriptive Findings**

Table 1 shows descriptive findings for PA and NA, TTD, and age. Overall, average T1–T2 changes were minor and statistically not significant for the PANAS mean scores of PA and NA. However, individual difference scores range from \(-1.6\) to \(2.5\) (PA) and from \(-1.3\) to \(1.3\) (NA); hence, intrapersonal increases and decreases in PA and NA occurred across the 1-year period. For both PA and NA, the initial value at T1 correlates negatively with the difference score. With regard to PA, this may partly be due to a ceiling effect, as the sample shows a tendency of high PA at T1, with mean and median (which was also 3.4) shifted a bit toward the theoretical maximum, such that some respondents already had high PA at T1 and could not improve further. However, the T1 distribution of PA is not substantially skewed to the right (skewness = \(-0.1\); only one person reached the maximum value of 5), such that for the most, PA increase would have been possible. With regard to NA, the situation appears the other way round, in that the negative correlation between initial and difference scores may be partly due to a bottom effect. Here, the T1 sample distribution is slightly skewed to the left (skewness = \(-0.2\), median 2.0; 0 indicates the “steepness” of TTD-related change toward the end of life). We would like to thank an anonymous reviewer for suggesting that we try out an exponential function.

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\(^1\) If affect at time \( t \) is \( L = b_{10} + b_{11} T + b_{12} T^2 + b_{13} T^3 + b_{14} A + b_{15} A^2 + b_{16} A^3 \) (\( T \) and \( A \) are TTD and age at time \( t \), respectively), then the subsequent 1-year change \( LCS = b_{20} + b_{21} (T - 1) + b_{22} (T - 1)^2 + b_{23} (T - 1)^3 + b_{24} A + b_{25} A^2 + b_{26} A^3 = (-b_{11} + b_{12} - b_{13} + b_{14} + b_{15} + b_{16}) + (-2b_{22} + 3b_{23})T - 3b_{24} T^2 + 2b_{25} T^3 + (2b_{26} + 3b_{16}) A + 3b_{15} A^2. \)

\(^2\) The exponential model was \( L = b_{10} + b_{11} T + b_{12} \exp(T) + b_{13} A + b_{14} A^2 + b_{15} A^3 + D_1 \) and \( LCS = b_{20} + b_{21} \exp(T) + b_{22} A + b_{23} A^2 + b_{24} A^3 + D_2 \). To meet a “linearized” specification within the structural equation modeling framework, we could not employ a free parameter \( c \) in the exponential term. Instead, we used an ad hoc procedure testing the model with varying prespecified negative values of \( c \), which we varied systematically to choose the best fitting solution (note that the value of \( c < 0 \) indicates the “steepness” of TTD-related change toward the end of life).
Table 1
Sample Scores of Positive and Negative Affect, Time to Death, and Age: Means, Standard Deviation, Minimum (Min), Maximum (Max), and Correlations

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sample descriptive</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Min</td>
</tr>
<tr>
<td>Positive affect T1 (PA1)</td>
<td>3.4 (0.7)</td>
<td>1.8</td>
</tr>
<tr>
<td>Positive affect, difference T2 – T1 (DPA)</td>
<td>-0.0 (0.6)</td>
<td>-1.6</td>
</tr>
<tr>
<td>Negative affect T1 (NA1)</td>
<td>2.1 (0.6)</td>
<td>1.1</td>
</tr>
<tr>
<td>Negative affect, difference T2 – T1 (DNA)</td>
<td>-0.1 (0.5)</td>
<td>-1.3</td>
</tr>
<tr>
<td>Months to death T1 (TTD)</td>
<td>62.9 (25.3)</td>
<td>14</td>
</tr>
<tr>
<td>Age T1 (years)</td>
<td>85.2 (3.2)</td>
<td>80</td>
</tr>
</tbody>
</table>

Note. Means scores for Positive and Negative Affect Schedule positive affect (PA) and negative affect (NA) mean; Positive/negative affect T1 = first measurement; positive/negative affect difference T2 – T1 = second minus first measurement.

† \( p < .1 \)  ‡ \( p < .01 \)  *** \( p < .001 \) (significance tests reported for correlations only).

Comparing the T1 mean scores and T1–T2 mean changes of the deceased with those who completed T2 measurement and are still alive, all values differed only slightly by the second decimal, with no significant effects in a group by time repeated-measures analysis of variance. The retest correlations were .52 and .67 for sum scores of PA and NA, respectively; this is evidence of both high rank-order consistency, as well as substantial intraindividual “changeability” across the 1-year interval.

Latent Change Score Model Results

Model 1 parameter estimates of the structural model part (i.e., leaving out the measurement model for the latent constructs LS1 and LS2) are summarized in Table 2. Model fit was fairly good for

Table 2
Latent Change Scores Model Results for Measures of Positive and Negative Affect

<table>
<thead>
<tr>
<th>Variable</th>
<th>Positive affect</th>
<th>Negative affect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate</td>
<td>SE</td>
</tr>
<tr>
<td>Path coefficients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TTD → LS1</td>
<td>0.049†</td>
<td>.027</td>
</tr>
<tr>
<td>TTD² → LS1</td>
<td>0.010</td>
<td>.011</td>
</tr>
<tr>
<td>TTD³ → LS1</td>
<td>0.000</td>
<td>.003</td>
</tr>
<tr>
<td>Age → LS1</td>
<td>-0.042**</td>
<td>.017</td>
</tr>
<tr>
<td>Age² → LS1</td>
<td>0.007</td>
<td>.005</td>
</tr>
<tr>
<td>Age³ → LS1</td>
<td>0.001</td>
<td>.001</td>
</tr>
<tr>
<td>TTD → LCS</td>
<td>-0.020</td>
<td>.018</td>
</tr>
<tr>
<td>TTD² → LCS</td>
<td>-0.000</td>
<td>.010</td>
</tr>
<tr>
<td>Age → LCS</td>
<td>0.012</td>
<td>.009</td>
</tr>
<tr>
<td>Age² → LCS</td>
<td>-0.002</td>
<td>.004</td>
</tr>
<tr>
<td>Residual (cov)variances</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Variance LS1</td>
<td>0.304**</td>
<td>.064</td>
</tr>
<tr>
<td>Variance LCS</td>
<td>0.189**</td>
<td>.073</td>
</tr>
<tr>
<td>Covariance LS1→LCS</td>
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<td>.051</td>
</tr>
<tr>
<td>Intercepts</td>
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<tr>
<td>LS1</td>
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<td>.052</td>
</tr>
<tr>
<td>LCS</td>
<td>-0.085†</td>
<td>.053</td>
</tr>
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</table>

Note. For path coefficients, unstandardized estimates, standard errors, and standardized coefficients are presented. TTD² and TTD³ residualized on TTD (TTD = time to death); Age² and Age³ residualized on age. LS1 = latent score Time 1 (first measurement); LCS = latent change score.

* Residual share of the latent variable variance = 1 – \( R^2 \).
† \( p < .10 \), one-tailed.  ‡ \( p < .05 \), one-tailed.  *** \( p < .01 \) one-tailed.

Testing Model 1 on PA revealed only some tentatively significant linear TTD effect and a significant linear age effect on latent T1 level of PA; none of the quadratic or cubic effects (neither of TTD nor age) on PA level at T1 and latent PA change were significant. Overall, the $R^2$ of the LS1 was .102; thus, the third-order polynomials of TTD and age account for about 10% of the T1 variance in PA, whereas the $R^2$ of the LCS was .018, meaning less than 2% of the 1-year change variance explained by TTD and age due to Model 1. In testing Model 2 (TTD effects only), the linear relation of TTD with the T1 level appeared significant ($p < .05$), and TTD accounted altogether for about 5% of latent level variance in PA. In both model versions, this linear TTD effect was positive in sign, indicating lower level of PA as death nears. Testing Model 3 (age effects only) revealed again significance of the linear age component ($p < .01$), and age accounted altogether for about 8% of latent T1-level variance. As in Model 1, no effects on T1–T2 were significant, and only 1% of variance change was explained by the age function. Thus, we revealed weak evidence of a linear TTD-related tendency in latent PA level but a stronger effect of linear age-related decline of PA; both however were too weak to predict 1-year change in PA. Altogether, it seems that the T1-level variance of PA explained by Model 1 is largely due to the linear age effect. This age effect, though, does not account effectively for interindividual differences in intraindividual 1-year change. This suggests that age-related development of PA unfolds rather slowly, accumulating only over longer time intervals PA losses large enough to account for differences at a “cross-sectional” observation.

Testing Model 1 on NA revealed a cubic pattern of TTD-related variation of the latent T1-level component, with significant effects of the quadratic and cubic components of the third-order polynomial. The cubic relation of TTD with the T1 level implies a quadratic relation with the 1-year change; hence, the quadratic TTD effect on LCS is significant too. It may be noted that if the effects on LS1 and LCS were estimated to be mutually independent (releasing the parameter constraints noted in the Statistical Modeling section), the same effects were significant, but the quadratic and cubic effects on the T1 level slightly increased and the quadratic effect on LCS slightly decreased. Thus, there is no evidence of some complexity in terms of different TTD- or age-related dynamics accounting for interindividual variation in T1 level and T1–T2-change, such as, for example, some unmodeled TTD- or age-related predictor that caused variation in the affect outcomes at first measurement occasion but was not effective at T2. The cubic functions of TTD and age accounted for about only 4% of the T1 variance in NA, whereas the $R^2$ of the LCS indicates about 11% of the 1-year change variance explained by TTD and age due to Model 1. Testing Model 2, the TTD effects were virtually the same, differing from those in Model 1 only by the third decimals, and the $R^2$s were .030 and .07 for LS1 and LCS, respectively. Models 1 and 3 revealed no age effects either in terms of statistical significance or in terms of effect size and variance explanation. Overall, the results indicate some kind of TTD-related dynamic in NA, which accounts for a rather small proportion of cross-sectional interindividual variation of NA, but for a more substantial proportion of the interindividual differences in 1-year change of NA among the very old participants.

To illustrate the meaning of the Model 1 coefficients of the TTD polynomial, the implied NA-with-TTD relation is visualized in Figure 2. It shows latent NA scores for T1–T2 trajectories as predicted across our sample’s TTD range by the Model 1 estimates (i.e., $LS1 = -.005 - .017 \times TTD + .023 \times TTD^2 + .011 \times TTD^3$; $LS2 = LS1 - .082 + .012 \times TTD - .043 \times TTD^2$, using the residualized values of TTD$^2$ and TTD$^3$; note that the linear coefficients of the curves, though insignificant, are included, as these are constitutive components of the whole polynomial function fitted to the data). That is, the predicted T1–T2 trajectories for the 140 deceased participants are shown on the right-hand plot of Figure 2. In contrast, we also depicted the individual T1–T2 trajectories on the left-hand plot, showing each of the deceased’s NA factor scores at T1 and T2, connected by a line. Figure 2 shows that the cubic TTD effect predicts a kind of S-shaped curve of NA across the end of life period, showing a terminal drop of NA, beginning about 2 to 3 years before death, but also a phase of NA increase, which precedes this terminal drop, beginning about 6 to 7 years before death. For TTD greater than 6 to 7 years, the curve shows again steeper decline of NA, suggesting some unexpected changes.
trend of far-to-death decline of NA more than 7 years before death. Focusing on evidence of terminal processes in NA closer to death, the findings suggest a twofold dynamic of NA in the approach of life’s end, namely a phase of terminal increase in NA, starting at about 6 years before death, which turns around to terminal drop when death comes rather close, that is, across the last 2 years of life.

Discussion

This study based on a sample of very old individuals revealed mixed evidence regarding terminal changes in PA and NA unfolding across a time span of 9 years prior to death. On the one hand, only a weak relationship between TTD and PA was found, namely a linear effect of TTD on the PA level observed at the baseline of our longitudinal study, indicating decline of PA as death comes closer. This effect appeared rather weak, in particular when controlled for age-related decline. In contrast, a linear effect of age-related decline appeared stronger, such that it can be concluded that PA may be largely unaffected by terminal processes, whereas we found a linear age effect that conforms with previous evidence of some age-related decline of PA in old age (Kunzmann, 2008; Pinquart, 2001). This age effect could account for interindividual differences of the T1 level of PA observed in our study, though, it appeared too slow to provide a significant average rate of intraintividual 1-year change in PA.

On the other hand, NA appeared substantially related with TTD. That is, we found a cubic (third-order polynomial) TTD function predicting T1 level of NA, which implies a curvilinear (quadratic) relationship between TTD and 1-year change of NA. Focusing on interindividual differences in NA at T1, this TTD effect could be rated as small, because it accounts for less than 4% of NA-level variation. With respect to the 1-year changes in NA, however, TTD appeared as a more substantial predictor, explaining more than 10% of the interindividual variation of the changes in NA that occurred in this sample of very old.

All in all, age performed worse than TTD in predicting NA, and NA compared with PA was found more sensitive to distance to death, which altogether supports our theoretical expectations. In particular, our findings confirm that late-life developmental trajectories of NA may be better understood in terms of a distance-to-death perspective than as an age-graded normative development. This terminal dynamics found for NA, but not for PA, goes hand in hand with the theoretical reasoning of an inward focus inherent in NA (Kunzmann, 2008). That is, degradations of biological systems that lead to the death of the individual may be mirrored more directly in NA than in PA. In contrast, if PA is understood in terms of an outward focus (Kunzmann, 2008), PA decline in old age may be more driven by external factors that interfere with the active involvement with the outward environment by most people in advanced old age, regardless of their physical status, such as age-related loss of social network resources and concomitant roles (Baltes & Smith, 1999).

Regarding the pattern of the TTD-with-NA relationship, Model 1 (and 2) analysis revealed a tendency of increasing NA as individuals approached death—that is, a terminal increase, as expected theoretically—conversely to a reduction of NA when they were closer to death. How could such at first glance inconsistent trends be explained? As we already considered theoretically, a twofold dynamic may shape NA development across the end-of-life period, namely a terminal increase due to NA sensitivity toward internal processes of degradation unfolding in the approach of death, but also a terminal decline of NA activation, due to a general loss of arousal as part of such degradation of biological functioning. The TTD-related pattern of NA revealed in this study may reflect the overlay of these dynamics at the end of life. First, across a larger range of time in the advance of death (starting at about 6 to 7 years prior to death), the progression of basic biological degradation processes may trigger and enhance NA. Second, if this degradation accumulates, it may dampen the individual’s affective arousal in general. Keeping in mind that the PANAS measures specific high-arousal emotions (Watson et al., 1999), a tendency of steeper 1-year decline in PANAS-measured NA could signal such loss of arousal close to death. That is, the increase of NA across the early end-of-life period may be counteracted by some “terminal deactivation” in the latest end-of-life phase. Such deactivation may be adaptive for individuals, preventing excessive emotional burden in the face of death. If so, though, it must be questioned why a terminal loss of affective arousal was not found for PA as well? To deal with that question, we take into account that we also found no TTD-related change of PA that parallels the terminal increase of NA preceding the drop close to the end. This “primary” terminal increase in NA, however, may have provided the basis for the subsequent terminal drop close to the end, in that the former leads to an elevated level of NA activation, which may then promote exhaustion and drop of arousal close to death.

Another explanation for terminal drop in NA close to death could be that people perceiving signals of impending death “calm down” in a sense and find sense in and reconciliation with the unavoidable end of life (Erikson, 1950). In particular dying at very old age, having received a good deal of life, may facilitate such processes and the acceptance of death. This could prevent negative high-arousal feelings, such as anger and fear, without enhancing positive affective arousal (feeling enthusiastic, excited, and so on).3

Apart of the twofold distance-to-death development of a long-run terminal increase followed by a short-term terminal drop close to the end of life, the TTD pattern revealed for NA also indicates a kind of far-to-death dynamic, namely a decline of NA more than 7 years before death. This was not expected theoretically, and it may be suspected that this far-to-death-decline came as an “artifact” of fitting the cubic polynomial to few cases observed that distant to death. However, as can be seen from the left-hand plot of Figure 2, several individuals (20%) were observed more than 7 years prior to their death, and the decline appears visible in most of their trajectories. Thus, this finding may reflect more than just a model artifact and suggests that people decline in NA before entering the terminal phase of affect development. Without theoretical considerations of TTD-related mechanisms effective on NA more than 7 years prior to death, this trend may indicate some kind of “normative” preterminal decline of NA in very old age, which attenuates when terminal processes take over as drivers of NA development. Though this conclusion appears contradictory to findings on normative age-related changes in affect (suggesting

3 We would like to thank an anonymous reviewer for pointing to this possible explanation.
that NA in general is kept relatively stable in old age, Kunzmann, 2008), it is not at odds with evidence of age-related decline of high-arousal feelings, both negative and positive (e.g., Pinquart, 2001). Thus, an explanation could be that the far-to-death decline of NA in our data points to some preterminal old-age-related loss of high-arousal feelings, as measured by the PANAS.

However, grounded on the data we analyzed in this study, explanations for the TTD-related trends revealed are highly speculative. One basic limitation of our study is the scarcity of our longitudinal data with respect to analyses of TTD-related change. Larger sample size and more measurement occasions with shorter time intervals preceding death would be needed to apply more complex statistical models suited to test for a twofold (or threefold) terminal dynamic (e.g., mult iface modeling, Cudeck & Klebe, 2002; see also Gerstorf, Ram, Estabrook, et al., 2008; Gerstorf, Ram, Röcke, et al., 2008, for two-phase TTD models of life satisfaction). Thus, our findings might be interpreted with some caution as tentative evidence suggesting such dynamics in late life NA development and calling for investigation of data better suited for the in-depth study of terminal changes in affect. However, as only a few longitudinal databases from samples of very old meeting these desirable properties have been generated so far, the presentation of a modeling approach to data limited to only two measurement occasions may be advocated as methodological advancement for the research on terminal change with existing databases.

Another study limitation is the lack of low-arousal measures of affect. We considered decline of affective arousal as a TTD-related dynamic of affect development in late life. To reach more clear-cut evidence of this effect, it would be highly desirable to compare TTD-related effects on measures of high versus low arousal affect, as the latter would not be impacted by loss of arousal. Thus, analyzing only high-arousal PA and NA, we could draw only speculative conclusions with regard to terminal changes in affective arousal.

As a further limitation of the results presented, the rather weak size of the TTD-effects found in this study deserves consideration. Accounting for less than 10% of variation in NA baseline levels and about 11% of variation of 1-year changes in NA, the previous conclusions should not be overstated. However, in general, we must consider human affect as driven by multiple conditions, including developmental processes such as terminal degradation, which vary largely among individuals at any time-point of the human life span. Therefore, variation of PA or NA cannot be expected to be largely accounted by any developmental trends, whether age- or TTD-related. Thus, the effects found in our analyses may as well not be underrated, as they show some substantial TTD-related variation in NA, despite large interindividual differences.

To conclude, acknowledged theoretical ideas of NA suggest that the human NA system may be highly sensitive to internal processes of terminal decline at the end of the human life span and that PA also may be affected by such “terminality.” Thus, more research on distance-to-death development in NA and PA seems promising to understand late life development of affect and affective well-being. The findings presented in this study suggest terminality of NA development in advanced old age. The terminal phase of the life span may come with increasing NA, but may also accompany some adaptive dampening of negative activation close to death. PA, however, seems not affected by terminal processes.

References


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