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Comparing Changes in Late-life Depressive Symptoms across Aging, Disablement, and Mortality Processes

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Abstract

Developmental processes are inherently time-related, with various time metrics and transition points being used to proxy how change is organized with respect to the theoretically underlying mechanisms. Using data from four Swedish studies of individuals aged 70–100+ ($N = 453$) who were measured every two years for up to five waves, we test whether depressive symptoms (CES-D) are primarily driven by aging-, disablement-, or mortality-related processes, as operationally defined by time-from-birth, time-to/from-disability onset (first reported impairment in Activities of Daily Living), and time-to-death metrics. Using an approach based on Akaike weights, we tested whether developmental trajectories (for each time metric) of depressive symptoms in late life are more efficiently described as a single continuous process or as a two-phase process. Comparing fits of linear and multi-phase growth models, we found that two-phase models demonstrated better fit than single-phase models across all time metrics. Time-to-death and time-to/from-disability-onset models provided more efficient descriptions of changes in depressive symptoms than did time-from-birth models, with time-to-death models representing the best overall fit. Our findings support prior research that late-life changes in depressive symptoms are driven by disablement and, particularly, mortality processes, rather than advancing chronological age. From a practical standpoint, time-to/from-disability-onset and particularly, time-to-death metrics may provide better “base” models from which to examine changes in late-life depressive symptoms and determine modifiable risk and protective factors. Developmental researchers across content areas can compare age with other relevant time metrics to determine if chronological age or other processes drive the underlying developmental change in their construct of interest.

Keywords

Depressive symptoms; time-to-death; disablement; aging; mortality

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Gerontological studies often consider advanced age as a major risk factor for depressive symptoms (Blazer, 2003). With prevalence rates of 1–5% for major depression and 10–25% for “clinically significant” depressive symptoms among older adults (Zarit & Zarit, 2012), as well as underdiagnosis and undertreatment (VanItallie, 2005), further understanding of the progression of both clinical and subclinical depressive symptoms is needed so that we may more adequately support late-life well-being and quality of life. A key issue is that depressive symptoms are often simply attributed to *aging*. However, disablement and mortality processes may also play a substantial role in symptom progression (Mirowsky & Ross, 1992).

Aging is a complex process representing a myriad of overlapping sets of influences and changes that accrue over time (Birren & Cunningham, 1985). *Primary aging* consists of normative processes that accumulate with age. *Secondary aging* represents processes that are caused by disease and disability, and *tertiary aging* processes are driven by mortality (deteriorations arising shortly before death). Aging can be charted over time indices that proxy the underlying developmental processes (Hertzog & Nesselroade, 2003; Wohlwill, 1973). Age-, dementia-, dropout-, mortality-, or study-related changes can be articulated by examining how an outcome (e.g., depressive symptoms) changes in relation to time-since-birth, time-since-dementia-diagnosis, time-to-dropout, time-to-death, or time-since-baseline (see Nesselroade & Featherman, 1997; Sliwinski et al., 2003). For example, late life changes in cognitive performance and well-being have been modeled in relation to *age*, (*time-from-birth*), *disability* (*time-to/from-disability onset*; Fauth et al., 2012; Ram et al., 2010), and *mortality* (*time-to-death*; Gerstorf et al., 2008a,b, 2010; Johansson et al., 2004; Sliwinski et al., 2006; Thorvaldsson et al., 2006; Wilson et al., 2003, 2007). In this paper we utilize population-based samples and novel analytic approaches to determine whether change in depressive symptoms is better articulated as an age, disability, or mortality-related process and whether changes in depressive symptoms are better characterized as a single phase (incremental continuous change process) or as a transition between two phases (transformational change process) that might indicate qualitative shifts in underlying mechanisms (Ram & Gerstorf, 2009).

Changes in Depressive Symptoms: The Role of Age, Disablement, and Mortality

Age

Advancing chronological age is a commonly cited risk factor for depressive symptoms, with the oldest-old being at the highest risk (Stek et al., 2006). Past research has repeatedly documented that the number of depressive symptoms individuals report generally increases with age (George, 1999; Mirowsky & Ross, 1992). Despite the repeated associations found between increased age and increased depressive symptoms, the “aging effect” on depressive symptoms, may in fact disappear when controlling for “third variables”, such as gender, socioeconomic status, cognitive ability, and most notable for our purposes, physical functioning (Blazer, 2003; Blazer et al., 1991; Yang, 2007). While the relationship between age and increased depressive symptoms may reflect the influence of normative aging processes (proxied by a *time-from-birth* time metric), it may also be more heavily influenced

by other processes such as the accumulation of pathology and disability or impending death (which would be proxied by metrics other than *time from birth* or years of age).

Disability

Poor health, functional impairment, and disability are all associated with higher depressive symptoms (Bruce, 2001; for a recent meta-analysis, see Chang-Quan et al., 2010). For example, using data from the Established Populations for Epidemiologic Studies of the Elderly, Taylor and Lynch (2004) found that increases in depressive symptoms accompanied increases in disability (for examples with other datasets, see also Hoppmann et al., 2011).

Mortality

Evidence supports a similar role for tertiary aging processes – depressive symptoms relate to greater mortality hazards in both hospitalized (Sherwood et al., 2007) and community-based samples (Cuijpers & Smit, 2002). Mortality-related deteriorations before death (i.e. terminal decline) have repeatedly been reported for cognitive (Bäckman & MacDonald, 2006) and physical variables (Diehr et al., 2002), and recent evidence supports terminal decline processes also influence emotional changes (Gerstorf et al., 2010; Mroczek & Spiro, 2005; Palgi et al., 2010). In sum, mounting evidence supports that primary, secondary, and tertiary aging processes all influence late-life depressive symptoms (albeit differentially). To our knowledge, our study is the first direct comparative approach to examine which of these types of processes best describes late life change in depressive symptoms.

Continuous vs. Multi-phase Change

Developmental change (in any outcome) may manifest as a continuous process and/or as transitions through discrete phases (Ford & Lerner, 1992). For example, although chorological age accumulates incrementally (suggestive of a continuous, single-phase), gerontologists have distinguished between the “third age” (young-old, age 65–85) and the “fourth age” (old-old, age 85+; Baltes & Smith, 2003). Thus, primary aging might be a two-phase process, with a natural inflection/transition occurring at or around age 85. Similarly, disablement processes can unfold as a continuous trajectory, where changes in impairment accrue incrementally across time, or as a stage transition(s) between an no-impairment, independence phase and a functional impairment, dependence phase where the point of inflection or phase-shift may occur at a known event (e.g., stroke) or unknown event (in which case the inflection point is estimated post-hoc through analysis of observed changes; Fauth et al., 2012; Hall et al., 2003; Taylor, 2010). Mortality processes may also progress as a single continuous decline (e.g., incremental change with each year closer to death) or involve a discrete shift from a pre-terminal to terminal phase (Kleemeier, 1962). Recent evidence suggests that the terminal decline inflection point occurs at three to five years prior to death (Gerstorf et al., 2010). Building on prior work, the current study explores whether changes in depressive symptoms are best described as a single, continuous or a two-phase process across aging, disablement, and mortality processes, with prior support for both continuous and phased change.

The Present Study

Acknowledging that late-life changes in depressive symptoms are influenced by aging, disablement, and mortality processes our current objective is to directly compare the fit of single-phase and multi-phase change trajectories charted across age- (*time-from-birth*), disablement- (*time-to/from-disability-onset metric*), and mortality- (*time-to-death metric*) based metrics. We use a novel, comparative approach to corroborate that disablement and mortality-oriented stage transition models provide the best representation of late-life changes in depressive symptoms. By doing so, the current study identifies the best “base model” on which to measure change; future studies can build upon base models identified here to examine factors or interventions that moderate and/or modify the magnitude of change and the timing of transitions.

Method

Participants and Procedure

We pooled data ($N = 1,440$) from four multidisciplinary population-based studies of aging in Sweden: GENDER (Gold et al., 2002), OCTO (Johansson & Zarit, 1995), OCTO-TWIN (McClearn et al., 1997) and NONA (Fauth et al., 2007), each of which obtained at least two waves of data obtained at approximately two-year intervals (four years for GENDER). In combination, these studies provide 10 years of data from individuals aged 70 to 94 years at baseline. Across studies, more than 80% of individuals contacted agreed to participate, with participants living in both ordinary and institutional housing.

OCTO and NONA participants were selected randomly from the population registry for Jönköping municipality – a region including rural, suburban, and urban settings. OCTO recruited individuals aged 84, 86, 88, and 90 years and NONA, individuals aged 86, 90 and 94 years. OCTO-TWIN and GENDER participants were recruited from national population-based registries that list all multiple births in the country. Twin samples identified from this registry are similar in social, psychological and biomedical characteristics to samples of singletons of the same age (Simmons et al., 1997). Using the inherent opportunity for pseudo-replication we randomly selected only one twin from each OCTO-TWIN pair for the main analyses, and then replicated all analyses with the data from the other twins. Our sample included 453 participants (62% women; mean age at baseline 85.58, $SD = 4.48$) who (a) provided valid baseline data on depressive symptoms, (b) reported disability at some point during the study, and for whom (c) death dates have been obtained from population records (see Table 1). Relative to excluded participants, our sample was older ($M = 85.58$, $SD = 4.48$ vs. $M = 81.20$, $SD = 6.02$; $F [1, 1,755] = 201.52$, $p < .01$), received fewer years of education ($M = 6.89$, $SD = 2.15$ vs. $M = 7.18$, $SD = 2.22$; $F [1, 1,579] = 5.73$, $p < .05$), and reported more baseline depressive symptoms ($M = 0.73$, $SD = 0.52$ vs. $M = 0.51$, $SD = 0.44$; $F [1, 1,438] = 68.75$, $p < .01$), but did not differ by gender (62% vs. 63% women, $p > .10$). The small differences suggest this subsample can be generalized to the population of Swedish elders who live into old age, experience disability, and eventually die.

Measures

Depressive symptoms—Self-reported depressive symptoms were measured via the short 10-item (OCTO study, only) or full 20-item CES-D (Radloff, 1977). Participants rated frequencies of depressive symptoms over the last week (response range 0-*not at all* to 3-*most of the time*). For consistency across studies, depressive symptoms scores were calculated as the sum of the common 10-items. Scores were transformed to a T-metric ($M = 50$, $SD = 10$) using the pooled sample of all participants at baseline assessment as the reference ($N = 1,440$). Of note, 20-item and 10-item scores were highly correlated ($r = .97$), and post-hoc analyses using 20-items for all studies (prorated for OCTO) yielded substantively identical results to those reported here. In the original 20-item CES-D, a score of 16 represents the cut-point for high depressive symptoms (Radloff, 1977), although for older populations, a cut-point of 21 is suggested (Lyness et al., 1997). For descriptive (not diagnostic) purposes, 154 participants (or 34%) likely had experienced clinical depression at some point during the study (based on a comparable cut-point for the 10-item version of 10, T-score = 55).

Age and Time-to-Death—Time-from-birth or chronological *age* was recorded at each assessment as the number of years since an individual's birth. *Time-to-death* was calculated post-hoc for each assessment as the difference between assessment date and an individual's population registry death date.

Time-to/from-Disability—Disability was assessed via self-reported impairment in four *Personal Activities of Daily Living* (PADL; Katz et al., 1963), which included bathing, dressing, toileting, and feeding oneself. Following usual procedures, we defined disability onset as the first wave where at least one PADL difficulty was reported (Guralnik et al., 2002; Seeman et al., 1996). *Time-to/from-disability* was calculated for each wave as the time-to or time-since the onset of disability. Because individuals may have been disabled for months or even years (e.g., prior to entry to the study) before the onset of disability was recorded, the time-to/from-disability metric contains some error of measurement (for further detail, see Ram et al., 2010 and the discussion section).

Data Analysis—Our main task was to determine whether between-person differences in within-person change were better represented as age-related, disability-related, or mortality-related change (= 3 time metrics) that organized either as a developmentally continuous (single-phase linear) change pattern or as a transition between two phases of development (= 2 types of change functions). In total, six (3×2) sets of models were specified and fit to the data, without sociodemographic or other control variables. These initial efforts focus on finding “the best base model”. Future analyses can extend the base model obtained here to examine whether and how trajectories differ by sociodemographic and other background characteristics, as well as modifiable factors related to targeted prevention or intervention efforts.

The first class of models were specified as typical linear growth models,

$$depressive\ symptoms_{ti} = \beta_{0i} + \beta_{1i}(time_{ti}) + e_{ti}, \quad (1a)$$

where person i 's level of depressive symptoms at occasion t , $depressive\ symptoms_{ti}$, is a function of an individual-specific intercept parameter, β_{0i} , and individual-specific slope parameter, β_{1i} , that captures rate of linear change, and residual error, e_{ti} . Following standard multilevel and latent growth modeling procedures (McArdle & Nesselroade, 2003), individual-specific intercepts and slopes (β s from the Level 1 model given in Equation 1) were modeled as

$$\beta_{0i} = \gamma_{00} + u_{0i},$$

$$\beta_{1i} = \gamma_{10} + u_{1i}, \quad (1b)$$

where interindividual differences, u_{0i} and u_{1i} are assumed normally distributed, correlated with each other, and uncorrelated with the residual errors, e_{ti} . Within this class of models, three versions were fit to the longitudinal data (repeated measures nested within persons) with $time_i$ being replaced by age_{ti} , $time\text{-}to\text{/}from\text{/}disability_{ti}$, or $time\text{-}to\text{-}death_{ti}$ (centered at age 87, at disability onset, and at 2 years before death, respectively).

The second class of models was specified as multi-phase growth models (Cudeck & Klebe, 2002; Ram & Grimm, 2007). Here, the repeated measures were modeled as

$$depressive\ symptoms_{ti} = \beta_{0i} + \beta_{1i}(time_{ti} - k) + e_{ti}, \text{ when } time_{ti} < k, \text{ and}$$

$$depressive\ symptoms_{ti} = \beta_{0i} + \beta_{2i}(time_{ti} - k) + e_{ti}, \text{ when } time_{ti} \geq k, \quad (2a)$$

where person i 's level of depressive symptoms at time t is a function of an individual-specific slope parameter, β_{1i} , that capture rates of linear change during a "pre-transition" phase, an individual-specific intercept parameter, β_{0i} , that captures level of depressive symptoms at a transition point k that specifies the timing of the transition, a second individual-specific slope parameter, β_{2i} , that capture rates of linear change during a "post-transition" phase, and residual error, e_{ti} . Individual-specific intercepts and slopes (β s from the Level 1 model given in Equation 2a) were modeled as

$$\beta_{0i} = \gamma_{00} + u_{0i},$$

$$\beta_{1i} = \gamma_{10} + u_{1i},$$

$$\beta_{2i} = \gamma_{20} + u_{2i} \quad (2b)$$

where interindividual differences, u_{0i} , u_{1i} and u_{2i} are assumed to be normally distributed, correlated with each other, and uncorrelated with the residual errors, e_{ti} . For each of the three time variables (age_i , $time\text{-}to\text{/}from\text{/}disability_i$, $time\text{-}to\text{-}death_i$), we fit models where the value of k (i.e., the timing of the transition between developmental phases) was successively

changed in half-year increments. Specifically, k was varied between 80 and 90 for the age models; between 4 years prior to and 4 years after disability onset for the time-to/from-disability models, and between 10 and 1 years before death for the time-to-death models. Although models where k is estimated as a free parameter could be used, the structure of the data (453 participants provided between 1 and 5 repeated measures, $M = 2.21$, $SD = 1.23$, over eight years for a total of 980 observations) data required fitting a series of models with k fixed to specific values. Further, models with variance in all three growth factors (random effects on u_{0i} , u_{1i} , and u_{2i}) were not consistently estimable. Interpreting the convergence issues as an indicator of severe model misfit, we restricted our analysis of multi-phase growth to those models with interindividual differences in only two of the growth factors (random effects in u_{0i} and either u_{1i} or u_{2i} , whichever fit best).

Results

Fifty-eight models were evaluated with respect to fit and convergence. Two criteria were used to identify which time index (age, time-to/from-disability, time-to-death) and pattern of change (single-phase vs. two-phase) provided a “better” description of the data, relative model fit (AIC = Akaike Information Criterion, lower is better) and relative proportion of variance explained (pseudo- R^2 ; see Snijders & Bosker, 1999). Relative model fits (AIC) for single-phase and multi-phase age, time-to/from-disability, and time-to-death models are shown in Figure 1 (pseudo- R^2 comparisons followed a similar pattern). Two-phase models with different values of the transition point, k , are connected with a line. Weight of evidence in favor of the best model is derived using Akaike weights (see Burnham & Anderson, 2002). Specifically, we calculated differences between each model in the set and the best fitting model (relative likelihoods) and normalized these differences to obtain the Akaike weights. Normalized probability ratios indicate how much support there is for the conclusion that one model is preferred over another (see Wagenmakers & Farrell, 2004).

Within *age* models (upper left in Figure 1), many two-phase models fit better (lower AIC) than the single-phase model. Of the two-phase models, the point of transition between phases of $k = 84.5$ years of age provided the overall best fit. Among Akaike weights of age-based models, this model was 1.05 times more likely than the next best fitting model and 9.0 times more likely than the single phase model. *Time-to-from-disability* models (middle of Figure 1) also suggested that two-phase models generally fit better than the single-phase model, with the best-fitting model one where the transition point was located $k = 1.5$ years after disability onset (1.05 times more likely than the next best fitting model and 73.7 times more likely than the single-phase model). *Time-to-death* models (lower right in Figure 1) showed that the majority of two-phase models fit better than the single-phase model, and the best fitting model had the transition point at $k = 5$ years prior to death (1.05 times more likely than the next best fitting model, and 63.4 times more likely than the single phase model). Model parameters and the implied trajectory of depressive symptoms for the best fitting model for each time index are shown in Table 2 and Figure 2.

Among these best fitting models, the two-phase time-to-death model fit the data better (AIC = 7,091; pseudo- $R^2 = .093$) than the two-phase time-to/from-disability (AIC = 7,096; pseudo- $R^2 = .083$) or age (AIC = 7,109; pseudo- $R^2 = .085$) models. As seen in graph C of Figure 3,

the typical trajectory was characterized by relative stability ($\gamma_{10} = 0.10$; $p > 0.10$) in depressive symptoms during a “pre-terminal” phase, and a transition at 5 years prior to death to a “terminal decline” phase characterized by an upward shift in depressive symptoms ($\gamma_{20} = 0.27$; $p_{diff} = 0.10$) and substantial between-person differences around that trend. A confidence interval, of sorts, based on the Akaike weights of the entire set of 58 models indicates that this model fits the data better, with 95% certainty, than models with AIC 7097. The certainty of better fit than the time-to-death models with k between 1.5 and 8 years prior to death ranges between 51.2% and 94.7%, and certainty of better fit than the time-to-from-disability models with $k = 1.5$ and $k = 2$ was 94.0% and 94.3%, respectively.

To substantiate our results, we ran a series of post-hoc analyses. First, the other twin’s data from OCTO-TWIN, we replicated findings presented here. Second, we re-ran the analyses excluding individuals who recovered from disability ($n = 43$, 9%). Results did not differ from those presented here. Third, to examine the unique variance associated with each metric, we ran a separate time-in-study based model with individuals’ age, time-to/from-disability, and time-to-death at initial observation included as between-person predictors of level and change in depressive symptoms. Of interest, we found that between-person variance in age ($\sigma^2 = 15.80$) was 3.5 times larger than that for time-to death ($\sigma^2 = 4.53$) and more than 13 times larger than variance in time-to/from-disability ($\sigma^2 = 1.16$). This suggests that it takes some 30 years of age differences to describe a tad bit more about late-life change in depressive symptoms than what can be described by less than 10 years of time-to-death differences and less than 3 years of time-to/from-disability. Finally, we “mapped” the three change points (k) onto one another. As seen in Table 2, the change point k in the best time-to/from-disability model was located at 1.5 years after disability onset and at 5 years prior to death in the best time-to-death model. The sample-level average of participants’ age 1.5 years post their disability onset and 5 years prior to their death were then used to convert timing across metrics. The average age at change point k (which for time-from-birth models is 84.5 years) is 86 years for time-to-death models, and 88.3 years for time-to/from-disability models. The similarity of the mapped k for age and mortality models is apparent. The difference in how k maps across the age and disablement models is probably due, in part, to error of measurement of the exact date of disability onset imposed by the study design (whereas exact dates for birth and death were known).

Discussion

Gerontological research suggests that changes in depressive symptoms are likely influenced by advancing age (primary aging), illness and disablement (secondary aging), and mortality (tertiary aging) processes. Although evidence suggests that disability and mortality play a considerable role, researchers traditionally model changes in depressive symptoms over an age (i.e., time-from-birth) metric. Our major objective was to systematically compare depressive symptoms as they change in relation to these processes, proxied by three time metrics (time-from-birth, time-to/from-disability onset, and time-to-death) so as to obtain the best overall base model for describing changes in depressive symptoms. Our results support prior suggestions that late-life changes in depressive symptoms proceed not so much as a function of normative aging, but more as a function of where an individual is on his or her disablement process, and where an individual is in relation to death. The time-to-death

metric, proxying mortality-related processes, provided the best fit to our data and accounted for the most variance in interindividual differences in late-life change in depressive symptoms.

In addition, for each time metric, two-phase models offered more efficient characterizations of late-life changes in depressive symptoms as compared to single-phase models. Findings for our *time-since-birth* models are in line with distinctions between a third age, characterized by relative stability, and a fourth age, marked by steeper decline (Baltes & Smith, 2003), represented here by worsening of depressive symptoms in the later phase. *Time-to/from-disability* models indicate that average depressive symptoms increased significantly with the accumulation and onset of disability up until 1.5 years after disability onset. We speculate that individuals may be aware of increasing restrictions in functional abilities, and negative affect may increase concurrently with impending disability and loss of independence (disability onset). After disability onset, depressive symptoms, on average, no longer increased, suggesting that people may adapt to disability-related challenge, or at least not experience increasingly worse affect. These patterns align with theories of hedonic adaptation (Brickman & Campbell, 1971), whereby negative affect increases following a challenge (here, onset of disability and need for assistance with PADL), but eventually stabilizes or decreases when individuals adjust to their circumstances. Our *time-to-death* models, where depressive symptoms were relatively stable until five years prior to death and then shifted into a new phase, are consistent with other reports that well-being follows a two-phase pattern of terminal decline (e.g., Gerstorf et al., 2010). In sum, our findings suggest that changes over time in depressive symptoms correspond with aging, disablement, and mortality processes, but that terminal decline provides the most powerful underlying set of processes to describe these overall trajectories of late life depressive symptoms. On a rather speculative note, our finding that disability-based models do not provide better model fit than mortality-based models may indicate that the overlap between disability/pathology and ‘normal’ dying is large.

Limitations and Contributions

Although the current sample included individuals living at home and in institutional housing, we note that those with the highest physical or cognitive impairment were certainly selected out of the sample at baseline or along the way through refusal, inability, or death. Therefore, trajectories of depressive symptoms observed here might not generalize to the frailest subset of older adults, or those with significant dementia. We included individuals with disability onset prior to baseline, which is helpful in avoiding sample biases where frail individuals are excluded, but also complicates our ability to model a more precise point of inflection along the time-to/from-disability metric. By not “capturing” the transition into disability for all participants, and allowing some individuals to already be disabled at initial visit, our finding that the inflection point actually occurs at 1.5 years post-disability includes “noise”, and may not necessarily reflect changes among individuals who are first free of disability, then develop disability, and finally adjust to disability (see additional discussion in Ram et al., 2010).

Likewise, the current use of the time-to/from-disability-onset as a time metric is a simplified proxy for the disablement process, much in the way that time-to-death is a simplified proxy for mortality processes. We acknowledge that in “real life”, and in a small subset of our own sample, individuals may become disabled temporarily, and then recover into a non-disabled state (Hardy & Gill, 2004), and that for some individuals the onset of disability is acute (i.e., catastrophic disability), but for others it is a lengthy process not easily demarcated by a date of onset (i.e., progressive disability; Ferrucci et al., 1996). We suggest that future investigations track progression of and fluctuations in disability more often than every two-years so as to obtain a more detailed proxy of the underlying process. We also suggest that future studies first determine the best base model (i.e., appropriate time metric) and then target covariates associated with differential rates of change. For example, we recently found that participants with low social support reported steep increases in disability-related depressive symptoms (Fauth et al., 2012). It is well possible that effect sizes of known correlates differ considerably depending upon the specific time metric over which they act.

Our analytical procedures used data mining techniques to find an optimal model, or range of models, by comparing model fits using Akaike weights. While computationally intensive, this paper is the first, to our knowledge, to use such an approach to directly compare the extent to which late-life depressive symptoms change as a function of aging, disablement and mortality by examining the relative fit of these developmental processes in a single sample. Founded in theory related to primary, secondary, and tertiary aging, we empirically validate some of the assumptions made, but not necessarily tested, in the literature.

Uncovering the time-metric that most closely maps onto the developmental change has practical utility. Our approach suggests that researchers should not necessarily default to the metric of age (time-from-birth) as the “best” time metric for describing change. For those studying late life change, our modeling efforts confirm that more appropriate base models may one that uses time to death (or time-to/from-disability). We encourage applying the model comparison approach to other datasets and within clinical applications. Once the better-fitting base model is established, future studies should move forward, adding predictors into the most appropriate base model to determine factors that influence and moderate the trajectory of depressive symptoms over time. We also encourage applying the approach to other phenomena. Researchers with developmental interests in areas outside of depressive symptoms, and in other stages of the lifespan, should consider that alternative time metrics (besides chronological age) may map more closely onto the course of change within their construct of interest.

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Figure 1.

Relative model fits (AIC) for single-phase and multi-phase models, for each time metric (chronological age, time-to/from-disability onset, and time-to-death). Two-phase models with different values of the transition point, k , are connected with a line. Lower values of AIC are considered better fitting models.

Figure 2.

Average (thick line) and individual (thin lines) changes for depressive symptoms as modeled over three time metrics, reflecting three underlying processes. (A) aging processes (time from birth); (B) disablement processes (time-to/from-disability onset); and (C) mortality processes (time-to-death).

Table 1

Sample Characteristics across the Four Studies.

	Study			
	GENDER	OCTO-TWIN	OCTO	NONA
Start year	1995	1990	1987	1999
Years of follow-up	4	8	6	4
Occasions	2	5	4	3
<i>N</i> at T1 ^a	40	127	217	69
<i>n</i> (%) women	19 (48%)	79 (62%)	140 (65%)	44 (64%)
Years of education ^b	7.43 (2.37)	7.38 (2.43)	6.50 (1.83)	6.87 (2.18)
<i>n</i> (%) widowed	14 (35%)	68 (54%)	132 (61%)	47 (68%)
<i>n</i> (%) institutionalized	2 (5%)	20 (16%)	72 (33%)	28 (41%)
Depressive Symptoms T1	50.28 (9.06)	46.63 (9.92)	51.98 (9.85)	49.82 (9.67)
Age T1	75.61 (2.67)	84.04 (2.82)	86.99 (2.29)	89.78 (3.31)
Time-to-disability T1	− 1.65 (1.87)	− 1.79 (2.14)	− 0.40 (1.02)	− 0.70 (1.12)
Time-to-death T1	− 4.28 (2.11)	− 6.03 (3.53)	− 4.41 (3.55)	− 2.45 (1.76)

Note.

^a = random selection of one twin from each twin pair in the OCTO-TWIN study. Scores for depressive symptoms standardized to a *T* metric (*mean* = 50; *SD* = 10) using the pooled sample of all oldest old participants at baseline assessment T1 as the reference (*N* = 1,440).

^b =for Swedes in this cohort, individuals started formal schooling around age 7, and had compulsory schooling for 6–7 years. Thus years of formal education in our sample are representative of older Swedish adults.

Table 2

Best-Fitting Two-Phase Growth Models for Depressive Symptoms over Three Time Metrics.

Parameter	Depressive Symptoms					
	Chronological age ^c		Time-to/from-disability ^d		Time-to-death ^e	
	Est.	SE	Est.	SE	Est.	SE
Fixed effects						
Intercept ^a , γ_{00}	48.79*	(0.63)	51.20*	(1.14)	49.27*	0.63
Slope 1 ^b , γ_{10}	-0.14	(0.17)	0.41*	(0.17)	0.10	0.19
Slope 2 ^b , γ_{20}	0.29*	0.13	-0.59	(0.35)	0.27	0.16
Transition point, k	=87.0		=1.5		=5.0	
Random effects						
Variance intercept, σ^2_{u0}	59.72*	(8.25)	81.14*	(23.36)	72.00*	6.95
Variance slope 1, σ^2_{u1}	0.87*	(0.37)	2.57*	(1.07)	1.33*	0.49
Covariance, σ_{u1u0}	-1.46	(1.54)	-7.21	(4.65)	7.00*	1.71
Residual variance, σ^2_e	41.64*	(2.85)	41.93*	(2.70)	41.46*	2.67
% change in <i>Pseudo R</i> ²	8.45		8.29		9.33	
-2LL	7,095		7,082		7,077	
AIC	7,109		7,096		7,091	

Note. Unstandardized estimates and standard errors are presented. Unfortunately, models with variance in all three growth factors (random effects on u_{0i} , u_{1i} , and u_{2i}) were not consistently estimable. Interpreting the convergence issues as an indicator of severe model misfit, we restricted our analysis of multi-phase growth to those models with interindividual differences in only two of the growth factors (random effects in u_{0i} and either u_{1i} or u_{2i} , whichever fit best).

^a = Intercept is centered at the transition point k (e.g., 87 for the age-based model);

^b = Rate of change scaled in T-units per year.

^c = Best-fitting two-phase model for chronological age with the timing of the transition located at age 84.5 years.

^d = Best-fitting two-phase model for time-to/from-disability with the timing of the transition located at 1.5 years after the onset of disability.

^e = Best-fitting two-phase model for time-to-death with the timing of the transition located at 5 years before death.

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$N = 453$ who provided 980 observations. Scores for depressive symptoms standardized to a T metric ($mean = 50$; $SD = 10$) using the pooled sample of all participants at baseline assessment T1 as the reference ($N = 1,440$). $AIC = Akaike Information Criterion$; $-2LL = -2 \text{ Log Likelihood}$, relative model fit statistics. The change in Pseudo R^2 was obtained from comparing the residual within-person variance from the conditional model including the time variable (age, distance-to/from-disability, time to death) with that obtained from an unconditional or intercept-only model (for our data $= 45.72$; see Snijders and Bosker, 1999, pp. 99–105).

* $p < .05$.