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The Effect of Lifetime Cumulative Adversity and Depressive Symptoms on Functional Status

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Objectives. The study aimed to examine whether lifetime cumulative adversity (LCA) and depressive symptoms moderate time-related trajectories of functional status.

Method. A total of 15,073 older adults (mean age = 63.91 at Wave 1) who participated in the first four waves of the Survey of Health, Ageing and Retirement in Europe reported on exposure to negative life events, depressive symptoms and three measures of functional status—difficulty in performing daily and instrumental activities, and functional limitation.

Results. Growth–curve models showed that time-related increase in disability and functional limitation was steeper among those exposed to higher levels of lifetime adversity. Moreover, a three-way interaction between time, lifetime adversity, and depressive symptoms emerged across measures of functional status, so that when exposure to lifetime adversity was accompanied by high level of depressive symptoms, the time-related increase in disability and functional limitation was the steepest.

Discussion. LCA is associated with a hastening of the disablement process, especially under conditions of high distress. Although the overall modest effects imply that resilience to lifetime adversity is widespread among older adults, prevention and intervention programs should consider that distressed older adults previously exposed to high levels of lifetime adversity are at risk for more rapid impairment in functional status.

Key Words: Difficult life events—Functional impairment—Mental health—SHARE.

THE notion of lifetime cumulative adversity (LCA) or lifetime adversity, as it will be referred to in this paper, captures a life-span perspective on accumulated exposure to a wide spectrum of potentially traumatic events (Turner & Lloyd, 1995). The construct draws upon cumulative advantage/disadvantage theory (CAD), which maintains that limiting conditions that are experienced throughout life differentially deflect individuals from optimal life-course trajectories, resulting in health and other inequalities (Dannefer, 2003; O'Rand & Henretta, 1999). Although cumulative adversity can stem from the persistent effects of a single hardship, adverse events generally tend to co-occur, leading to a successive replacement of one hardship by another, or to a layering of hardships one upon another (Hatch, 2005).

Previous research has shown that lifetime adversity can have a detrimental effect on mental health in older age (Kessler, 1997; Seery et al., 2010; Shmotkin & Litwin, 2009; Shrira, 2012; Shrira, Shmotkin, & Litwin, 2012). Whether or not the effect of lifetime adversity on mental health also influences the physical health of older adults is a less studied topic (for exceptions, see Krause, Shaw, & Cairney, 2004; Schafer & Ferraro, 2012).

Recent works show that lifetime adversity is related to trajectories of cognitive and physical health over time (Brown, 2010; Comijs, van den Kommer, Minnaar, Penninx, & Deeg, 2011; Petkus, Wetherell, Stein, Liu, & Barrett-Connor, 2012; Schafer & Ferraro, 2012). This is of major importance to gerontology, insofar as aging processes are best captured across time. It is relevant, therefore, to assess how LCA affects within-person changes over time. Despite the importance of the topic, however, the relationship between adversity over the life span and time-related trajectories of physical health remains largely unexplored.

The aim of the current study, therefore, is to examine whether lifetime adversity predicts time-related trajectories of functional status. A related aim is to investigate what happens when exposure to potentially traumatic events is combined with depressive symptoms—a major marker of mental distress. In order to address these two related aims, the study uses data from the Survey of Health, Ageing and Retirement in Europe (SHARE) (Börsch-Supan et al., 2008), a large longitudinal study of older Europeans. The present analysis utilizes measures from the four available waves of SHARE.

LCA and Physical Health in Late Life

A recent expansion of the CAD, termed cumulative inequality theory, seeks to explicate the mechanisms through

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which inequality develops among people during the life course (Ferraro & Shippee, 2009). The cumulative inequality theory holds that personal exposure to risk adds to the effect of social systems (stratifying society according to class, race, income, etc.) in generating inequality, which accumulates across the life span. Cumulative inequality interacts with one's ability to mobilize social, economic, and psychological resources, together with human agency (i.e., the ability to change one's environment), in shaping the individual's mode and level of functioning in old age. The theory proposes, moreover, that accumulated stress may lead to premature mortality, and discusses how cumulative inequality present in early periods in life produces biological changes that hasten aging and senescence. This suggests that lifetime adversity may be related not only to a low level of functioning at a specific point in time, but also to increasingly deteriorating functional impairment.

A large body of evidence supporting some of the above assumptions suggests that exposure to negative life events produces, among other effects, neuroendocrinological and immunological dysregulation (McEwen, 1998). When stress-related bodily dysregulation extends over long periods of time, it contributes to the creation of disease and frailty in late life (Miller, Chen, & Parker, 2011). Health behaviors, post-traumatic distress, and depressive symptoms may intervene in this process (Lapp, Agbokou, & Ferreri, 2011), but there is ample evidence that exposure can also lead to physical impairment after controlling for a myriad of possible intervening variables (Schafer & Ferraro, 2012; Wilmoth, London, & Parker, 2010). For example, Lin, Epel, and Blackburn (2012) review evidence that childhood adversity is associated with later life shortened telomeres—DNA–protein complexes that serve as an important marker of cellular aging—even in the absence of a clinical level of mental distress.

More direct support for the accelerated decline hypothesis that stems from the cumulative inequality theory comes from studies examining trajectories of physical health. These studies mostly focused on older veterans. One of them found a positive relationship between exposure to combat and other traumas, on the one hand, and physical dysfunction, on the other hand. However, it did not find those with combat trauma alone, or those with combat trauma combined with another trauma, to differ in their age-related trajectory of physical symptoms from those with no such exposure (Schnurr, Spiro, Aldwin, & Stukel, 1998).

Other studies have shown differences in age-related trajectories of health. For example, Elder, Shanahan, and Clipp (1997) found that combat exposure in World War II predicted a pattern of stronger physical health decline that was observed 15 years after the war. In a more recent inquiry using data from the Health and Retirement Study, war veterans and especially those who served in World War II and Korea showed a more rapid decline in physical health over time than did nonveterans (Wilmoth et al., 2010). Schafer and Ferraro (2012) used data from the Midlife Development in the United States Study to show that with each additional adversity in childhood, the odds of remaining disease free across the two waves of the study decreased by 12%.

The Importance of Functional Status

Especially pertinent in the context of the relationship between lifetime adversity and physical health is the need to focus on functional status. First, functional status in late life has direct implications for the quality of longer life among older adults (Crimmins, Saito, & Ingegneri, 1997). Second, the ability of societies to continue to meet the needs of their oldest members may be increasingly challenged by an increase in the rates of late-life functional impairment (Chernew, Goldman, Pan, & Shang, 2005).

Functional status encompasses both disability and functional limitation (Guralnik & Ferrucci, 2003). Disability refers mainly to difficulties in vital self-maintenance capabilities. Functional limitation refers primarily to difficulties in the adaptive use of physical capabilities such as movement and vigor. It is important to assess both disability and functional limitation because the two are related components in the disablement process, and because measures of functional limitation may identify functional decrements that are not observable from the reporting of more severe outcomes, such as disability in activities of daily living (ADL). Moreover, disability and functional limitation may show different age-related trajectories (Parker & Thorslund, 2007), and self-reports of disability and functional limitation may be affected by different variables on both the individual and the societal level (Litwin, Shrira, & Shmotkin, 2012).

Previous studies have found a positive relationship between lifetime adversity and disability among older adults (Krause et al., 2004; Shrira, Palgi, Ben-Ezra, & Shmotkin, 2011), but none of them examined disability over time at the within-person level. In view of the previously noted gap in the literature in this area, the current study assesses the effect of lifetime adversity on within-person trajectories of functional status using two markers of disability: difficulties in performing ADL and difficulties in performing instrumental ADL (IADL), and one marker of functional limitation that focuses on difficulties in performing physical activities involving the limbs.

The Moderating Role of Mental Distress

The effect of lifetime adversity on various outcome measures has been generally found to be moderate (Kraaij, Arensman, & Spinhoven, 2002; Krause et al., 2004; Shrira et al., 2011). This suggests that the majority of older adults successfully cope with negative life events and maintain resilience, even in the face of cumulative stress (Ferraro & Shippee, 2009). Nevertheless, some people may be more susceptible than others to lifetime adversity and it is

important to know who is at higher risk for its detrimental effects.

There is evidence that among people suffering from high levels of mental distress, the accumulation of negative events has a strong impact on functioning. For example, the effects of lifetime adversity on mental health were exacerbated among people high in neuroticism (Kendler, Kuhn, & Prescott, 2004) and among those with a previous history of depression (Kessler & Magee, 1994; Maciejewski, Prigerson, & Mazure, 2000).

The moderating role of mental health on the relationship between lifetime adversity and physical health has not been widely explored in the literature. A related finding was reported by Elder and colleagues (1997) who found a positive effect of combat exposure on physical dysfunction among veterans who reported low self-worth, but not among those who reported high self-worth. Expanding upon this finding, the current study assumes that high mental distress, and more specifically high levels of depressive symptoms, moderates the relationship between lifetime adversity and time-related trajectories of functional status.

The Study Hypotheses

In sum, it is evident that the potential effect of lifetime adversity on time-related trajectories of functional status has not yet been sufficiently examined. The need to study the relationship between these two factors emanates from two main reasons. One is that lifetime adversity covers the entire life span and it most probably affects a large proportion of older adults (Krause et al., 2004; Shmotkin & Litwin, 2009). The second is that functional status is a major concomitant of both successful aging (Depp & Jeste, 2006) and survival (Ben-Ezra & Shmotkin, 2006).

Therefore, the current study tested two main hypotheses. Hypothesis 1 maintains that lifetime adversity moderates time-related trajectories of functional status. More specifically, we hypothesize that with time, the increase in disability and functional limitation will be greater among those exposed to higher levels of lifetime adversity. This hypothesis was derived from the axiom embedded in the cumulative inequality theory (Ferraro & Shippee, 2009) stating that cumulative inequality may lead to premature mortality, and from the above-mentioned evidence supporting that axiom. Hypothesis 2 holds that there will be a three-way interactive effect of time, lifetime adversity, and depressive symptoms on functional status. That is, when exposure to lifetime adversity is accompanied by a high level of depressive symptoms, the time-related increase in disability and functional limitation will be the greatest. This hypothesis follows from another axiom of the cumulative inequality theory (Ferraro & Shippee, 2009), which contends that the detrimental effect of cumulative adversity can be moderated by one's psychological resources and agency (or the lack thereof, as, e.g., in cases of depression).

Method

Participants

Data were drawn from the first four waves of the SHARE (Börsch-Supan et al., 2008). The SHARE survey queries persons aged 50 years and older from 19 countries and their spouses of any age. Based on probability samples of households in each participating country, the SHARE database presents a representative picture of the community-dwelling older population. The data were collected by means of a comprehensive computer-assisted personal interview, and a supplementary paper Drop-Off questionnaire (used primarily in W1). Informed consent was obtained from all respondents prior to the interview.

Wave 1 (W1, 2004), Wave 2 (W2, 2006), and Wave 4 (W4, 2010) of SHARE included queries about the physical, mental, and social functioning of participants. Wave 3 (W3, 2008–2009), known as SHARELIFE, constituted a retrospective inquiry into the life histories of the participants and included questions on several potentially stressful life events.

As noted earlier, the current study examined whether and how lifetime adversity moderates time-related change in functional status across time. Therefore, the target sample in the analysis included only those respondents who had completed both the retrospective lifetime adversity items in W3 and the earlier baseline measurements in W1 (N = 15,073). Of these respondents, 13,192 and 8,160 respectively, also participated in W2 and W4. These respondents came from the 11 countries that comprised the first wave of SHARE (Austria, Belgium, Denmark, France, Greece, Italy, Netherlands, Germany, Spain, Sweden, and Switzerland). The countries considered in the current study were those that participated consistently in all four waves of SHARE (thus, countries added to the SHARE project after W1, for example, Poland and the Czech Republic, were not included in this analysis). The participants' retrospective lifetime history and their data from waves 1, 2, and 4, enabled us to assess the moderating role of lifetime adversity reported to have occurred before W1 on the time-related changes in functional status across waves 1, 2, and 4.

Attrition analyses compared those of the 15,073 respondents who completed SHARELIFE and who participated only in W1 (n = 996) with those who participated in more than one wave. These analyses showed that those who participated in W1 only reported more difficulties in ADL and IADL, but the size of these differences was relatively small (Cohen's d = 0.04 and 0.06, respectively). The groups also differed in gender composition, with a somewhat higher percentage of women among those who participated in more than one wave (55.3% vs. 51.9%), as well as in verbal fluency (higher among those who participated in more than one wave), and in medical conditions and alcohol consumption (lower among those who participated in more than one wave; Cohen's d ranged 0.08–0.18). On the other hand, the

groups did not differ in age, education level, household income, depressive symptoms, functional limitation, smoking, weight, verbal learning, and arithmetic.

Table 1 presents the descriptive statistics for the study participants across the relevant waves. As can be seen, the mean age at W1 was 63.91 (*SD* = 9.37), 55.1% were women, the mean education level was 2.52 (*SD* = 1.55), corresponding to less than an upper secondary education, and the mean household income in Euro was 42,815.75 (*SD* = 53,350.90). The age of respondents naturally increased across waves, whereas the education level and gender composition did not change.

Measures

As mentioned, the third wave of SHARE—SHARELIFE—was devoted to retrospective examination of the respondents' lives. Participants were presented with a CAPI-based Event History Calendar which marked each year of their lives with their corresponding age. Key personal events (e.g., marriage, having children, moving, getting jobs) were marked in the calendar so that they could be used as reference points. General country-specific events were also used as reference points. Respondents were asked to indicate whether each of the events had ever happened to them. After confirming an event, respondents were asked to specify the year when it had taken place (for more details, see Schröder, 2011).

The measure of lifetime adversity employed in the current analysis was derived from a list of difficult life events queried in SHARELIFE. Events were included in our lifetime adversity measure if they fulfilled the criteria for a traumatic event or met the criteria for an event of an adjustment disorder in which the stressor can be of any severity, as described by the DSM-IV-TR (American Psychiatric Association, 2000). We also corroborated that all of the selected events had previously appeared in major studies on cumulative adversity (Green et al., 2010; Kraaij et al., 2002; Krause et al., 2004). The final list consisted of 15 difficult life events that reflected early familial adversity (e.g., parents/guardians drank heavily, being fostered, living in a children's home), persecution (e.g., being incarcerated in a concentration/labor/war camp), late familial adversity (e.g., relationship breakdown/divorce, death of a partner or a child), and other adversities (e.g., being in a prison, being homeless). We omitted physical health vulnerabilities (childhood or adult physical illness and physical injury) from the current analysis to avoid confounding with the functional status outcomes. Table 2 presents a complete list of the events in the lifetime adversity measure.

As respondents could cite the reoccurrence of some, but not all, of the events, each event was coded as not confirmed (0) or confirmed (1). In order to calculate overall lifetime adversity, the number of confirmed events was summed (possible range 0–15). In the present inquiry, difficult events reported to have occurred in or after the year of the W1 interview were omitted from the final count, given that the outcome measures were tapped in waves 1, 2, and 4. These particular events were nevertheless included in the analysis among the covariates. Event counts have been used in many studies as an index of cumulative adversity (Seery et al., 2010), as they avoid potential ambiguities and confounds that are characteristic of other methods of assessment that weight for subjective perception of severity or event duration (for a discussion, see Kessler, 1997).

Table 1. Descriptive Statistics for the Participants Across the Study Waves

Variable	W1: *n* = 15,073	W2: *n* = 13,192	W4: *n* = 8,160
Mean age (*SD*)	63.91 (9.37)	66.40 (9.28)	70.88 (8.91)
No. (%) women	8,303 (55.1)	7,276 (55.2)	4,537 (55.6)
Mean education (*SD*)[a]	2.52 (1.55)	2.52 (1.55)	2.56 (1.56)
Mean household income in Euro (*SD*)	42,815.75 (53,350.90)	—	—
Mean depressive symptoms (*SD*)	2.26 (2.17)	—	—
Mean ADL[b]	0.14 (0.61)	0.18 (0.70)	0.29 (0.96)
Mean IADL[b]	0.24 (0.80)	0.31 (0.94)	0.47 (1.27)
Mean functional limitation[b]	0.63 (1.04)	0.71 (1.11)	0.89 (1.27)
Mean medical conditions (*SD*)	1.22 (1.25)	1.26 (1.26)	1.49 (1.34)
No. (%) ever smoked	7,145 (47.5)	6,232 (47.3)	3,823 (46.9)
Mean alcohol consumption (*SD*)[c]	4.23 (2.26)	4.31 (2.26)	4.17 (2.32)
Mean body mass index (*SD*)	26.50 (4.27)	26.61 (4.41)	26.53 (4.53)
Mean verbal fluency (*SD*)	18.93 (6.76)	—	—
Mean word recall (*SD*)	3.44 (1.97)	—	—
Mean arithmetic (*SD*)	2.39 (1.08)	—	—

Notes. W1 data collection: 2004–2006; W2 data collection: 2006–2008; W4 data collection: 2010–2012. Only respondents who completed the cumulative adversity items in W3 were included. ADL = difficulty in activities of daily living; IADL = difficulty in instrumental activities of daily living.

[a]Recorded by one of seven education levels according to the International Standard Classification of Educational Degrees (ISCED-97; United Nations Educational, Scientific and Cultural Organisation (UNESCO), 1997): (0) preprimary education, (1) primary education, (2) lower secondary education, (3) upper secondary education, (4) postsecondary education, (5) first tertiary education, and (6) second stage tertiary education.

[b]In all three waves, the median score was 0 and the range was 0–6, 0–7, and 0–5, for ADL, IADL, and functional limitation, respectively.

[c]Referred to the past 6 months, scale ranged from 1 (not at all in the last 6 months) to 7 (almost every day).

Table 2. Occurrence of Difficult Life Events

	n	%
Early familial adversity		
Been fostered with another family	260	1.7
Lived in a children's home	242	1.6
Parents/guardians drank heavily	1,200	8.0
Persecution		
Concentration camp	22	0.1
Discrimination	610	4.0
Dispossession of property due to persecution	526	3.5
Evacuated/relocated during war	769	5.1
Labor camp	65	0.4
War camp	84	0.6
Late familial adversity		
Child dead	950	6.3
Partner dead	2,194	14.6
Relationship breakdown/divorce	2,320	15.4
Stillborn baby	488	3.2
Other adversities		
Been homeless (for 1 month or more)	60	0.4
Prison	50	0.3

Note. N = 15,073. Events which were reported to occur at or after W1 interview year were omitted from the final count.

Baseline depressive symptoms (at W1) were assessed by the European Depression scale (Euro-D; Prince et al., 1999). This scale contains 12 items that specify recent depressive symptoms (e.g., "In the last month, have you cried at all?"), scored as a sum of "no" (0) and "yes" (1, indicating presence of a symptom) encoded answers. Five items were phrased in positive terms (e.g., "do you keep up your interests?"). In the present analysis, a minimum of completion of 10 items was required for scoring a sum, with scores of 10–11 items being interpolated. Cronbach's alpha coefficient of internal consistency for the Euro-D in the current study was .70 at W1.

The outcome variable—functional status—was assessed by two self-reported disability measures and by a self-reported functional limitation instrument. In these measures, the scores were the sum of activities respondents had difficulties in performing; higher scores represented greater disability and limitation.

The two disability measures were: (1) difficulties in basic ADL (adapted from Katz, Downs, Cash, & Grotz, 1970), and (2) difficulties in IADL (adapted from Lawton & Brody, 1969). ADL included six functions: dressing, crossing a small room, bathing, getting in or out of bed, eating, and toileting. The IADL measure included seven functions: using a map, preparing meals, daily shopping, using the telephone, taking medications, doing housework, and handling personal finances. Difficulties in all of the disability functions were rated with a dichotomized answer (not having difficulties/having difficulties). For ADL, internal reliability measured by Kuder–Richardson's ρ was .75, .79, and .84 at W1, W2, and W4, respectively. The corresponding Kuder–Richardson's ρ for IADL was .75, .79, and .86.

The functional limitation measure (adapted from Nagi, 1976) included five physical activities, specifically stooping, kneeling, or crouching, reaching or extending arms above shoulder level, pulling or pushing heavy objects, lifting or carrying heavy weights, and picking up a small coin from a table. Each limitation was rated with a dichotomized answer (not having difficulties/having difficulties). Internal reliability (Kuder–Richardson's ρ) was .70, .73, and .76 at W1, W2, and W4, respectively.

Covariates included the background characteristics of gender, country, baseline education, and baseline household income. Baseline education was recorded as one of seven levels according to the International Standard Classification of Educational Degrees (ISCED-97; United Nations Educational, Scientific and Cultural Organisation (UNESCO), 1997). Baseline household income referred to the gross annual income (in Euro) adjusted for relative purchasing power parity within the participating SHARE countries and standardized by the household size square root to get the equivalent disposable income per standard person. Baseline income was used due to variation in its measurement over waves.

Markers of medical conditions and health behaviors that may affect functional status were also accounted for. Medical conditions were assessed by the number of illnesses, (0–14), reported to have been diagnosed by a physician (e.g., heart disease, diabetes, arthritis). Smoking behavior was assessed by the survey question that asked the participants if they had ever smoked. The item was scored as 0 (no) or 1 (yes). Alcohol consumption was assessed by asking the participants to rate how often they drank alcoholic beverages in the previous 6 months, on a scale ranging from 1 (not at all in the last 6 months) to 7 (almost every day). Respondents' weight was assessed by calculating the body mass index (BMI) and recoding the result to reflect three categories: below 18.5 (1 = underweight), 18.5–24.99 (2 = normal), and 25 and above (3 = overweight).

Finally, baseline cognitive functioning—another major concomitant of functional status—was measured by three tests: (1) verbal learning was assessed by number of words immediately recalled out of a 10-word list; (2) word fluency was assessed by number of correct animal names produced within 1 min; (3) arithmetic ability was assessed by number of correct answers to four arithmetic questions. These three tests have been shown to load on a single latent factor of cognitive functioning (Kavé et al., 2012). Baseline cognitive scores were used, as they were not assessed at W4.

Data Analyses

The outcome measures were standardized to a T metric (M = 50 and SD = 10 at W1), with the baseline sample (n = 15,073) serving as the reference. This transformation ensured a common metric across variables while maintaining the psychometric properties of the scores and the longitudinal changes in means and variances (e.g., Gerstorf, Ram, Lindenberger, & Smith, 2013). This method also facilitated interpretation of relative changes (Infurna,

Gerstorf, & Zarit, 2013). LCA and other continuous covariates were centered.

To test whether lifetime adversity moderated change in functional status, we fit three separate growth–curve models (Peugh & Enders, 2005) for each functional status measure (ADL, IADL, and functional limitation), with years since study entry as the time variable (Model 1). Following the recommendations by Mendes de Leon (2007), in addition to using years since study entry, we concurrently examined the effect of baseline age. The Level 1 (within-person level) model was parameterized as:

$$\text{Functional status}_{it} = \beta_{0i} + \beta_{1i}(\text{TIME}_{it}) + e_{it} \quad (1)$$

In this model, functional status for person i at time t, Functional status$_{it}$, is a function of (a) an intercept parameter for person i (across the t times for which the person provided data), β_{0i}; (b) a slope parameter that captures the rate of time-related change for person i, β_{1i} (TIME$_{it}$); and (c) the within-individual random error, e_{it}. The Level 2 (between-person level) model was parameterized as:

$$\begin{aligned}\beta_{0i} = {} & \gamma_{00} + \gamma_{01}(\text{LCA}_i) + \gamma_{02}(\text{AgeW1}_i) \\ & + \gamma_{03}(\text{LCA}_i \times \text{AgeW1}_i) + u_{0i}\end{aligned} \quad (2)$$

$$\beta_{1i} = \gamma_{10} + \gamma_{11}(\text{LCA}_i) + \gamma_{12}(\text{AgeW1}_i) + u_{1i} \quad (3)$$

In this model, γ_{00} represents the intercept of functional status for a person with average age and average lifetime adversity (i.e., age and lifetime adversity equal to the sample mean); γ_{01} represents the effect of lifetime adversity on the initial level of functional status for person i, γ_{02} represents the effect of baseline age on the initial level of functional status for person i, γ_{03} represents the effect of the interaction between lifetime adversity and baseline age on the initial level of functional status for person *i*, and u_{0i} represents the error of β_{0i}. In addition, γ_{10} represents the slope of time-related change in functional status for a person of average lifetime adversity, γ_{11} represents the effect of lifetime adversity on the covariance of time and functional status, γ_{12} represents the effect of baseline age on the covariance of time and functional status, and u_{1i} represents the error of β_{1i}.

Substituting Equations 2 and 3 back into Equation 1, we have the following:

$$\begin{aligned}\text{Functional status}_{it} = {} & \gamma_{00} + \gamma_{01}(\text{LCA}_i) + \gamma_{02}(\text{AgeW1}_i) \\ & + \gamma_{03}(\text{LCA}_i \times \text{AgeW1}_i) \\ & + \gamma_{10}(\text{TIME}_{it}) \\ & + \gamma_{11}(\text{LCA}_i \times \text{TIME}_{it}) \\ & + \gamma_{12}(\text{AgeW1}_i \times \text{TIME}_{it}) + u_{0i} \\ & + u_{1i} \times \text{TIME}_{it} + e_{it}\end{aligned} \quad (4)$$

The first seven terms are the fixed effects that capture the average model. The last three terms are the random effects that capture the variation between individual regression models and the average model, as well as the variation between individual observations and the regression model within each person.

To test whether lifetime adversity, depressive symptoms, and their interaction moderated change in functional status, we fit an additional three separate growth–curve models for each functional status measure, with years since study entry as the time variable (Model 2). In these analyses, depressive symptoms were dichotomized at the optimal Euro-D cutoff point for predicting a diagnosis of depression in semistructured clinical interviews, 0–3 symptoms and 4 symptoms or above (Prince et al., 1999). The Level 1 model was parameterized similarly to Equation 1, whereas Level 2 model was parameterized as:

$$\begin{aligned}\beta_{0i} = {} & \gamma_{00} + \gamma_{01}(\text{LCA}_i) + \gamma_{02}(\text{AgeW1}_i) \\ & + \gamma_{03}(\text{DEPRESS}_i) \\ & + \gamma_{04}(\text{LCA}_i \times \text{DEPRESS}_i) \\ & + \gamma_{05}(\text{LCA}_i \times \text{AgeW1}_i) \\ & + \gamma_{06}(\text{AgeW1}_i \times \text{DEPRESS}_i) \\ & + \gamma_{07}(\text{LCA}_i \times \text{DEPRESS}_i \times \text{AgeW1}_i) + u_{0i}\end{aligned} \quad (5)$$

$$\begin{aligned}\beta_{1i} = {} & \gamma_{10} + \gamma_{11}(\text{LCA}_i) + \gamma_{12}(\text{AgeW1}_i) \\ & + \gamma_{13}(\text{DEPRESS}_i) + \gamma_{14}(\text{LCA}_i \times \text{DEPRESS}_i) + u_{1i}\end{aligned} \quad (6)$$

Substituting Equations 5 and 6 back into Equation 1, we have the following:

$$\begin{aligned}\text{Functional status}_{it} = {} & \gamma_{00} + \gamma_{01}(\text{LCA}_i) + \gamma_{02}(\text{AgeW1}_i) \\ & + \gamma_{03}(\text{DEPRESS}_i) \\ & + \gamma_{04}(\text{LCA}_i * \text{DEPRESS}_i) \\ & + \gamma_{05}(\text{LCA}_i * \text{AgeW1}_i) \\ & + \gamma_{06}(\text{AgeW1}_i * \text{DEPRESS}_i) \\ & + \gamma_{07}(\text{LCA}_i * \text{DEPRESS}_i * \text{AgeW1}_i) \\ & + \gamma_{10}(\text{TIME}_{it}) + \gamma_{11}(\text{LCA}_i * \text{TIME}_{it}) \\ & + \gamma_{12}(\text{AgeW1}_i * \text{TIME}_{it}) \\ & + \gamma_{13}(\text{DEPRESS}_i * \text{TIME}_{it}) \\ & + \gamma_{14}(\text{LCA}_i * \text{DEPRESS}_i * \text{TIME}_{it}) \\ & + u_{0i} + u_{1i} * \text{TIME}_{it} + e_{it}\end{aligned} \quad (7)$$

Where the first 13 terms are the fixed effects that capture the average model. The last three terms are again the same random effects mentioned above. All models were also run with covariates including adversity occurring between W1 and W3, gender, country (divided into 10 dummy-coded variables), baseline education, baseline household income, medical conditions, the three health behavior markers, and the three indices of baseline cognitive functioning. Covariates were added to control for the intercept term.

The models were examined with SPSS 19. Incomplete data were treated as missing at random (Little & Rubin, 1987).

Results

Descriptive Statistics and Preliminary Analyses

Table 1 presents the descriptive statistics for the outcome measures across waves. All three functional status measures showed an increase across waves indicating that disability and functional limitation increased with time.

Table 2 presents the frequencies for each of the 15 difficult life events. Early familial adverse events and events of persecution were reported by relatively few respondents. Late familial adverse events were more frequent. The mean lifetime adversity score was 0.65 (SD = 0.87, median = 0, range = 0–7).

When examining the relationship between lifetime adversity and background characteristics in W1, lifetime adversity was moderately positively correlated with age (r = .19, p < .0001) and was higher among women (M women = 0.74 [SD = 0.90] vs. M men = 0.54 [SD = 0.80], t[15071] = −14.46, p < .0001, Cohen's d = 0.23). Lifetime adversity was not correlated with education (Spearman's ρ = −.007), and showed a weak correlation with household income (r = −.07, p < .0001). After controlling for age, gender, education, and household income, lifetime adversity significantly differed across countries, $F(10,14941) = 59.35$, $p < .0001$, $\eta^2_p = .038$. It was highest in Germany (adj. M = 0.88, SD = 1.01) and lowest in Greece (adj. M = 0.37, SD = 0.62).

We should note that the 15,073 respondents who completed the functional status measures in at least one of the waves (W1, W2, and W4) and the lifetime adversity items in W3 provided 37,717 observations across time. Of these, 34,927 (92.6%) and 23,826 (63.1%) were longitudinal observations across the first two waves and across all waves, respectively. The average number of observations per individual was 2.60 (SD = 0.54). Preliminary intraclass correlation (the proportion of the between-individual variance to the sum of the between- and within-individual variances) for ADL, IADL, and functional limitation was .57, .50, and .46, respectively, suggesting that there was between 43% and 54% within-person variance in functional status over time.

Growth–Curve Models

In a preliminary model predicting ADL using time alone (data not shown), the intercept was 50.090 (SE = 0.069, p < .0001), and the slope was 0.330 (SE = 0.018, p < .0001). The annual increase in ADL was equivalent to 0.33 T-score units across all respondents. In Model 1 with ADL as the dependent (Table 3), the estimate for time (γ_{10}) was 0.328, p < .0001. Thus, the increase in ADL was equivalent to 0.328 T-score units per year for respondents with an average age at study entry and an average level of lifetime adversity. The estimate for LCA (γ_{01}) was 0.481, p < .0001. The increase in initial level of ADL was equivalent to 0.481 T-score units per one lifetime adversity level. The estimate for the time × LCA (γ_{11}) was 0.068, p < .0001.

The time × LCA interaction was probed using Preacher, Curran, and Bauer's (2006) procedure. It revealed that those with no lifetime adversity had an increase of 0.328 T-score ADL units per year, whereas those with high levels of lifetime adversity (two events and more) had an increase of 0.464 T-score ADL units per year. That is, respondents who reported higher lifetime adversity showed a steeper time-related increase in ADL. These effects were observed after accounting for the significant effects of baseline age, its interaction with LCA and its interaction with time. The effects indicated that older respondents had an increased initial level of ADL, especially when they reported high level of lifetime adversity, and that older respondents had a steeper slope of increased ADL with time. The Δ pseudo-R^2 (Snijders & Bosker, 1999) showed that Model 1 explained 35.3% of the variance in ADL.

In Model 2, time, lifetime adversity, and depressive symptoms predicted ADL. The estimate for depressive symptoms (γ_{03}) was 3.477, p < .0001, indicating that the increase in initial level of ADL from low to high levels of depressive symptoms was equivalent to 3.477 T-score units. The estimate for the time × LCA × depressive symptoms interaction (γ_{14}) was 0.149, p < .0001.

The time × LCA × depressive symptoms interaction indicated that the time-related increase in ADL became much steeper among those with high lifetime adversity combined with a high level of depressive symptoms. That is, while the slope for time-related change in ADL was 0.269 among respondents with no lifetime adversity and a low level of depressive symptoms, it was 0.781 among those with lifetime adversity combined with a high level of depressive symptoms. These effects were observed after accounting for the effects of baseline age, its two-way interactions with LCA, time, depressive symptoms, and the three-way interaction between baseline age, LCA, and time. Model 2 explained 35.8% of the variance in ADL. Figure 1a presents the three-way interaction for ADL.

Turning to the IADL outcome, we note that in a preliminary model predicting IADL using time alone (data not shown), the intercept was 50.157 (SE = 0.072, p < .0001), and the slope was 0.408 (SE = 0.017, p < .0001). The annual increase in IADL was equivalent to 0.408 T-score units across all respondents. In Model 1 with IADL as the dependent (Table 3), the estimate for time (γ_{10}) was 0.402, p < .0001, the estimate for LCA (γ_{01}) was 0.536, p < .0001, and the estimate for the time × LCA (γ_{11}) was 0.064, p < .0001. The time × LCA interaction showed that respondents who reported higher lifetime adversity had a steeper time-related increase in IADL. More specifically, those with no lifetime adversity had an increase of 0.402 T-score IADL units per year, whereas those with high level lifetime adversity had an increase of 0.530 T

Table 3. Growth–Curve Models for Functional Status by Time, Lifetime Cumulative Adversity, Depressive Symptoms, and Baseline Age and Their Interactions

	Difficulties in activities of daily living					
	Model 1			Model 2		
Fixed effect estimates	Estimate	*SE*	*p* Value	Estimate	*SE*	*p* Value
Intercept	50.011	0.068	.000	49.030	0.075	.000
Time	0.328	0.018	.000	0.269	0.021	.000
LCA	0.481	0.069	.000	0.124	0.079	.118
Baseline age	0.223	0.007	.000	0.155	0.008	.000
Depressive symptoms				3.477	0.152	.000
LCA × depressive symptoms				0.607	0.141	.000
Baseline age × LCA	0.035	0.005	.000	0.022	0.006	.001
Baseline age × depressive symptoms				0.199	0.014	.000
Baseline age × LCA × depressive symptoms				0.004	0.011	.672
Time × LCA	0.068	0.018	.000	0.011	0.021	.589
Baseline age × time	0.038	0.002	.000	0.039	0.002	.000
Time × depressive symptoms				0.210	0.042	.000
Time × LCA × depressive symptoms				0.149	0.037	.000
AIC/BIC	261,587/261,680			257,256/257,401		
	Difficulties in instrumental activities of daily living					
Intercept	49.977	0.068	.000	48.881	0.074	.000
Time	0.402	0.016	.000	0.354	0.019	.000
LCA	0.536	0.069	.000	0.252	0.078	.001
Baseline age	0.312	0.007	.000	0.277	0.008	.000
Depressive symptoms				3.900	0.149	.000
LCA × depressive symptoms				0.366	0.139	.009
Baseline age × LCA	0.073	0.005	.000	0.054	0.006	.000
Baseline age × depressive symptoms				0.243	0.014	.000
Baseline age × LCA × depressive symptoms				0.002	0.011	.802
Time × LCA	0.064	0.016	.000	0.022	0.019	.258
Baseline age × time	0.050	0.001	.000	0.051	0.001	.000
Time × depressive symptoms				0.190	0.039	.000
Time × LCA × depressive symptoms				0.096	0.034	.005
AIC/BIC	257,937/258,030			252,956/253,100		
	Functional limitation					
Intercept	50.042	0.067	.000	48.611	0.074	.000
Time	0.357	0.015	.000	0.375	0.017	.000
LCA	0.840	0.068	.000	0.501	0.079	.000
Baseline age	0.307	0.007	.000	0.248	0.008	.000
Depressive symptoms				5.569	0.150	.000
LCA × depressive symptoms				0.190	0.141	.179
Baseline age × LCA	0.022	0.006	.000	0.019	0.007	.008
Baseline age × depressive symptoms				0.170	0.015	.000
Baseline age × LCA × depressive symptoms				0.021	0.012	.092
Time × LCA	0.043	0.015	.004	0.022	0.018	.214
Baseline age × time	0.025	0.001	.000	0.027	0.001	.000
Time × depressive symptoms				0.085	0.036	.018
Time × LCA × depressive symptoms				0.081	0.031	.011
AIC/BIC	259,816/259,910			255,965/256,109		

Notes. N = 15,073 respondents who provided 37,717 observations. Unstandardized estimates and standard errors are presented. AIC = Akaike's information criterion; BIC = Schwarz's Bayesian criterion; LCA = Lifetime cumulative adversity.

units. Again, these effects were observed after accounting for the significant effects of baseline age, its interaction with LCA and its interaction with time. The Δ pseudo-R^2 indicated that Model 1 explained 36.5% of the variance in IADL.

In Model 2 with IADL as the dependent, time, lifetime adversity, and depressive symptoms predicted IADL. The estimate for depressive symptoms (γ_{03}) was 3.900, $p <$ .0001, and the estimate for the time × LCA × depressive symptoms interaction (γ_{14}) was 0.366, p = .009. The time × LCA × depressive symptoms interaction showed that the time-related increase in IADL became quite steeper when examining those with high lifetime adversity combined with a high level of depressive symptoms. The

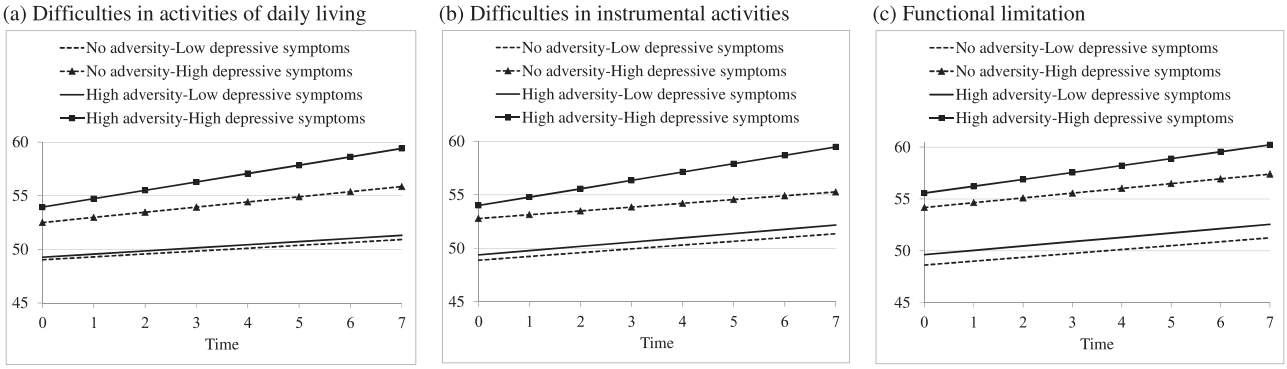

Figure 1. Time-related trajectories (in T-score units) for (a) difficulties in activities of daily living, (b) difficulties in instrumental activities, and (c) functional limitations as a function of lifetime cumulative adversity and depressive symptoms.

slope for time-related change in IADL was 0.354 among those with no lifetime adversity and a low level of depressive symptoms, compared with 0.780 among those with lifetime adversity combined with a high level of depressive symptoms. As in the ADL model, these effects were observed after accounting for the effects of baseline age, its two-way interactions with LCA, time, depressive symptoms, and the three-way interaction between baseline age, LCA, and time. Model 2 explained 37.5% of the variance in IADL. Figure 1b presents the three-way interaction for IADL.

Finally, the results for functional limitations revealed that when predicting this outcome using time alone, the intercept was 50.076 ($SE = 0.071$, $p < .0001$), and the slope was 0.361 ($SE = 0.015$, $p < .0001$). The annual increase in functional limitation was equivalent to 0.361 *T*-score units across all respondents. Table 3 shows that with functional limitations as the dependent (Model 1), the estimate for time (γ_{10}) was 0.357, $p < .0001$, the estimate for LCA (γ_{01}) was 0.840, $p < .0001$, and the estimate for the time × LCA (γ_{11}) was 0.043, $p = .004$. The time × LCA interaction indicated that respondents who reported higher lifetime adversity showed a slightly steeper time-related increase in functional limitations. That is, those with no lifetime adversity showed an increase of 0.357 *T*-score functional limitations units per year, whereas those with a high level of lifetime adversity showed an increase of 0.443 units. As in the case of the other indices, these effects were observed after accounting for the significant effects of baseline age, its interaction with LCA, and its interaction with time. Based upon its Δ pseudo-R^2, Model 1 explained 18.3% of the variance in functional limitations.

In Model 2, time, lifetime adversity, and depressive symptoms predicted functional limitations. The estimate for depressive symptoms (γ_{03}) was 5.569, $p < .0001$, and the estimate for the time × LCA × depressive symptoms interaction (γ_{14}) was 0.081, $p = .011$. The time × LCA × depressive symptoms interaction showed that the time-related increase in functional limitations became much steeper when examining those with high lifetime adversity combined with a high level of depressive symptoms. Comparatively, the slope for time-related change in functional limitations was 0.375 among those with no lifetime adversity and a low level of depressive symptoms, and 0.666 among those with high lifetime adversity and many depressive symptoms. Once again, these effects were observed after accounting for the effects of baseline age, its two-way interactions with LCA, time, depressive symptoms, and the three-way interaction between baseline age, LCA, and time. The Δ pseudo-R^2 showed that Model 2 explained 18.4% of the variance in functional limitations. Figure 1c presents the three-way interaction for functional limitations.

After adding the other covariates into Model 2, the time × LCA remained significant for all three functional status indices (for ADL: estimate = 0.051, $SE = 0.017$, $p = .003$, for IADL: estimate = 0.055, $SE = 0.015$, $p = .001$, and for functional limitation: estimate = 0.063, $SE = 0.015$, $p = .018$). The time × LCA × depressive symptoms interaction also remained significant for two of the indices (for ADL: estimate = 0.106, $SE = 0.036$, $p = .004$, for IADL: estimate = 0.085, $SE = 0.033$, $p = .012$), but was nonsignificant for functional limitation (estimate = 0.060, $SE = 0.032$, $p = .063$). Among the covariates, alcohol consumption, medical conditions, and the three indices of cognitive functioning were consistently related to functional status. Moreover, IADL and functional limitation were higher among women and those with lower BMI score. Among the countries, only functional limitation differed, showing lower levels in Denmark and Greece.

Supplementary Analyses

As late familial adverse events were most common, we examined whether more recent events most related to the aging process could explain the effects we found. Therefore, we performed additional analyses modeling an interaction between partner death and time, and analyses in which we modeled an interaction between the other events and time. These models also accounted for the effect of baseline age and its interaction with time and partner death/other events besides partner death.

The time × partner death interaction was significant for all three functional status indices (for ADL: estimate = 0.192, *SE* = 0.054, $p < .0001$, for IADL: estimate = 0.175, *SE* = 0.050, $p = .001$; for functional limitation: estimate = 0.114, *SE* = 0.045, $p = .013$). The time × other events besides partner death interaction was also significant for all three functional status indices (for ADL: estimate = 0.073, *SE* = 0.022, $p = .001$, for IADL: estimate = 0.069, *SE* = 0.020, $p = .001$; for functional limitation: estimate = 0.047, *SE* = 0.018, $p = .010$). Together, these findings indicate that although late-life events, such as partner death, increase functional disability status, other events, occurring at earlier periods in life, increase functional decline as well.

Finally, because the functional status indices are commonly skewed in large-scale samples of community-dwelling older adults, we reanalyzed our data using transformation of ADL, IADL, and functional limitation by square root. After adding the covariates into Model 2, the time × LCA remained significant for all three functional status indices (for ADL: estimate = 0.003, *SE* = 0.001, $p = .002$, for IADL: estimate = 0.003, *SE* = 0.0009, $p < .0001$, and for functional limitation: estimate = 0.002, *SE* = 0.001, $p = .029$). The time × LCA × depressive symptoms interaction also remained significant for two of the indices (for ADL: estimate = 0.006, *SE* = 0.002, $p = .005$, for IADL: estimate = 0.004, *SE* = 0.002, $p = .023$), but nonsignificant for functional limitation (estimate = 0.003, *SE* = 0.002, $p = .064$).

Discussion

This study examined the effect of LCA on time-related trajectories of functional status. We found that the time-related increase in disability and functional limitation was steeper among those exposed to higher levels of lifetime adversity. Moreover, a three-way interaction between time, lifetime adversity, and depressive symptoms emerged across measures of functional status, so that when exposure to lifetime adversity was accompanied by a high level of depressive symptoms, the time-related increase in disability and functional limitation was the steepest. These findings remained significant after controlling for background characteristics and markers of physical health and cognitive functioning.

Corroborating our first hypothesis, we found that those with high levels of lifetime adversity (exposed to two events or more) showed an increase in disability and functional limitation which was 47%–55% higher than those who did not report exposure to lifetime adversity. These findings join the increasing evidence on postadversity hastened decline that is manifested in other domains, such as cognition (Brown, 2010; Comijs et al., 2011; Petkus et al., 2012), physical illness (Elder et al., 1997; Schafer & Ferraro, 2012; Wilmoth et al., 2010), and cellular aging (Lin et al., 2012).

Supporting the second hypothesis, the data revealed that respondents who experienced a combination of exposure to lifetime adversity and a high level of depressive symptoms showed an increase in disability and functional limitation which was 1.7–2.9 times higher than among respondents who reported no such exposure. These findings are in line with those from previous studies that stress the moderating role of mental distress (Elder et al., 1997). The current results show that mental distress can also moderate within-person increase in disability and functional limitation across time. Persons with a combination of high lifetime adversity and high depressive symptoms should be considered a high-risk group, therefore, as they demonstrated a substantial increase in disability and functional limitation, equal to between one half and a full standard deviation per decade.

Viewed together, our findings correspond with the assumptions of the cumulative inequality perspective which expands upon CAD. According to this perspective, cumulative adversity shapes functioning in old age and may lead to a hastened aging process. Moreover, the fact that the negative effects of lifetime adversity were aggravated among those with high mental distress in our sample highlights the possible interaction that Ferraro and Shippee (2009) delineate between cumulative inequality and one's ability (or impaired ability) to mobilize resources and human agency.

It should be noted that depressive symptoms among respondents in the analytic sample were low in most cases, and that lifetime adversity had only a modest effect on the outcome measures when depressive symptoms were low. This complements the assumption that resilience to the accumulation of difficult life events is common (Seery et al., 2010). Blazer and Hybels (2005) suggest several explanations for such resilience to lifetime adversity in older adults. First, they maintain that many difficult events are predictable so that older adults prepare themselves to cope with such events. There is also a tendency among older people to de-emphasize negative experiences and to selectively optimize positive experiences. Moreover, if forced to face negative events, older adults are nevertheless equipped with experience and wisdom that were acquired throughout life.

The current findings should be assessed in light of the study limitations and strengths. First, the study did not examine the reciprocal influences that lifetime adversity and functional status may have. Lifetime adversity may indeed increase the risk for functional deterioration, but disability and limitation can elicit or exacerbate certain stressful events and difficulties. Moreover, certain predispositions, such as early social circumstances, genetic liability, aspects of personality or access to social support, may render people prone to both lifetime adversity and functional impairment. Therefore, future studies should delineate the possible causal paths between lifetime adversity and functional status.

Second, depressive symptoms may also exacerbate lifetime adversity or increase the tendency to remember and report difficult life events (Kessler, 1997). Still, it should be noted that aside from other possible causal paths, the one leading from cumulative adversity to greater mental distress has already been established (Moos, Schutte, Brennan, & Moos, 2005). Third, the present study did not address posttraumatic distress, which is often considered a relevant outcome in studies of lifetime adversity. There is an advantage in using depressive symptoms in research because of their applicability to large community populations as well as their sensitivity to both clinical and subclinical conditions (Blazer & Hybels, 2005). Nevertheless, evidence should still be gathered regarding the role of posttraumatic distress in the LCA–functional status relationship. Fourth, a few countries were underrepresented in our specific sample, and although we controlled for country in our models, future studies should examine whether the effect of LCA varies across countries and cultures. Finally, SHARE currently has four waves of data available for analysis. Future studies using additional data points would further substantiate our findings.

The current study has also several strengths of note. First, it longitudinally examined a large heterogeneous sample drawn in a multinational survey. Secondly, recall bias in retrospective reports of lifetime adversity was partially mitigated in the current design. Fairly objective and major events, such as those assessed in this analysis, are better remembered (Hardt & Rutter, 2004). SHARELIFE also included a significant memory aid procedure—the Event History Calendar—that was shown to reduce errors in retrospective reports of life events (Schröder, 2011). Finally, reports on lifetime adversity were made at a different time point than self-evaluations of the outcome measures.

In conclusion, this study adds to the current literature by showing that older adults exposed to lifetime adversity are at greater risk of showing a time-related increase in disability and functional limitation over time. An especially high-risk group includes those older people who have both exposure to lifetime adversity and a high level of depressive symptoms. A practical implication of these findings is that prevention and intervention programs should focus on targeting older adults in this high-risk group, especially given their greater risk of experiencing a hastened process of disablement.

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