Convergence of the Aging Rates for Healthy and Unhealthy Elderly at Advanced Ages*

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Abstract

We analyze the ability of a cumulative index of age-associated health and quality-of-life disorders, called a "frailty index" (FI), to characterize individual rates of biological aging in the elderly and, consequently, population heterogeneity in mortality models, using National Long Term Care Survey (NLTCS) data. We show that the FI in the NLTCS exhibits an accelerated increase with age resembling mortality-curve behavior. Such patterns suggest that FI may be a better indicator of aging than chronological age. Deficits accumulate faster in non-disabled elderly who, at younger ages, had a lower mean FI than in disabled individuals, who showed a higher FI at younger ages. We interpret this as a cross-sectional manifestation of compression of morbidity. Age-patterns for disabled and non-disabled males and females tend to converge at advanced ages. This suggests the existence of biological age limits associated with given levels of health-maintenance in the society.

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1. Introduction

Despite broad interest in the factors and mechanisms responsible for human aging and numerous efforts to identify the aging-associated processes underlying biological senescence and longevity, these mechanisms are still not well understood. At the population level, aging is viewed as being manifested in an exponential increase in mortality rates with age up to age 85+ – with a deceleration after that age (Akushevich, Manton, & Kulminski, 2005; Lew & Garfinkel, 1984; Vaupel et al., 1998). This deceleration has been observed not only for humans but also for insects, worms, and yeast (Vaupel et al., 1998 and references therein). Such a mortality age-pattern is likely the result of a dynamic interplay of a variety of aging-associated processes in an organism (Ukraintseva & Yashin, 2001; 2003) influenced by individual differences in survival chances (Yashin, Ukraintseva, Boiko, & Arbeev, 2002; Vaupel et al. 1998).

Survival curves are probably the most reliable indicators of the effects of anti-aging interventions in various species. However, in practice, they are more convenient for short-lived experimental animals (e.g., nematodes, flies and rodents). For long-lived animals, (e.g., primates, including humans), the use of biomarkers that reflect aging processes may be more useful due to faster feedback. The validity of indices to describe biological aging (BA) is justified by the fact that any intervention successful in slowing senescence and postponing the manifestation of age will, by definition, result in asynchrony between BA and chronological age (Butler & Sprott, 2000). Valid aging biomarkers would also be helpful in estimating variability (heterogeneity) in rates of individual aging. Though there have been many attempts to find reliable and universal biomarkers of somatic aging, most of them have failed. Why? Another question is that, although no one disagrees that individuals differ in their manifestation of aging and rates of aging, why has it then so difficult to find valid biomarkers of aging?

One reason may be that the overall rate of somatic aging is the product of a combination of rates of different biological processes with distinct age dynamics. For instance, age-related changes in physiological indices suggested as bio-markers of aging can accelerate, decelerate, have linear dynamics, or even oscillate (Ukraintseva & Yashin, 2001; Arbeev, Ukraintseva, Arbeeva, & Yashin, 2005; Nakamura, Lane, Roth, & Ingram, 1998). Thus, the rate of individual aging (the rate of age-related changes in a given index) may increase, decrease, be constant, or change non-monotonically, with age. In consequence, in the same organism, and at the same time, the rate of aging can be characterized by increasing, decreasing, constant or non-monotonic functions, depending on the chosen bio-marker (Ukraintseva & Yashin, 2001; Arbeev et al., 2005). This is probably a reason why attempts to find a "universe" biomarker to measure the BA rate with homogeneous dynamics have failed (Butler & Sprott, 2000).

The relative contribution of different aging-associated processes to the age phenotype may differ among individuals, creating variability in aging manifestations among age-peers. To capture variability in aging rates, which is not easily measured, Beard (1971) introduced the concept of a *longevity factor*. Vaupel, Manton, & Stallard (1979) conceptualized the variability of unobserved factors in the notion of *individual frailty*, which describes differences in susceptibility to disease and death among individuals in a population with respect to the proportional hazard of death. In most frailty models used in demographic and epidemiological applications, individual susceptibility is assumed fixed for life. Moreover, researchers seldom had either the ability, or intent, to identify a variable frailty for each individual because of the scarcity of data.

When more detailed longitudinal data on age-associated processes became available, concepts of frailty were refined. To address questions about the contribution of age trajectories

of biological and physiological indices to aging and mortality, more sophisticated (dynamic) models of *changing frailty* were developed (Woodbury & Manton, 1977; Yashin, Manton, & Vaupel, 1985; Manton & Yashin, 2000). Such models were applied to data from several longitudinal studies (see Yashin & Manton, 1997 and references therein). A consequence of this generalization is that frailty parameter in such models can be associated with measures of processes describing the health deterioration of human organisms with age.

The frailty concept acquired more physiological meaning in studies of the factors and processes associated with BA. These studies define *frailty as a specific physiological state* that is not necessarily associated with chronic conditions or disability and that typically arises at elderly ages. This meaning became convenient for epidemiologists and clinicians, although there is still no universally recognized definition of frailty that is valid across settings. Currently, frailty is most often viewed as a physiological state of individuals with increased vulnerability to stressors that results from decreased physiological reserves, and even deregulation of multiple physiologic systems (Fried, Ferrucci, Darer, Williamson, & Anderson, 2004). This decreased reserve results in difficulty maintaining homeostasis in response to "normal" environmental perturbations that might not create such problems at younger ages. Nevertheless, operational definitions of frailty remain controversial (Bortz, 2002). A problem is also how to identify frailty in population-based studies.

Rockwood and Mitnitski and colleagues (Mitnitski, Song, & Rockwood, 2004; Rockwood, Mogilner, & Mitnitski, 2004) argue that health and quality-of-life deficits (i.e., signs, symptoms, impairments, etc.) accumulated by individuals during their life course can be considered as indicators of physiological frailty. Then, the frailty state can be described by a composite measure of such deficits. They propose a *frailty index* (FI) calculated as the proportion of the deficits in an individual. The FI as the mean accumulation of deficits predicts death and describes health risks (Mitnitski et al., 2004). Frailty appears to not be characterized by the substance of the individual deficits used to define the FI but by their aggregate ability to characterize the decline in physiological performance, loss of redundancy, or complexity, in the interaction of various subsystems of an organism and, thus, to characterize its overall function. In this sense, the FI can be viewed as a measure of individual functional complexity of an organism which is necessary to successfully respond to a dynamic environment. The loss of such complexity suggests that the individual loses functional degrees of freedom in responding to dynamic environment. Loosing such functions the organism's ability to adapt to environmental changes is degraded and mortality increases. Such an approach, determining a FI using self-reports on various health deficits seems promising to assess frailty using survey and clinical data and, consequently, to practically capture heterogeneity in aging rates (Mitnitski et al., 2004).

In this paper, we apply the Rockwood-Mitnitski approach to constructing a FI using the National Long Term Care Survey (NLTCS). The NLTCS is a nationally-representative, longitudinal survey that assesses the health and functioning of U.S. elderly (65+) individuals over 18 years (1982 to 1999) (Manton & Gu, 2001). To define a FI, we use the same, or similar, health deficits as assessed in the Canadian Study of Health and Aging (CSHA) (Mitnitski et al., 2004). We will, thus, validate prior findings using a new population-based database and will focus on the connections of FI and age. We examine the FI age-patterns found in five NLTCS waves. We evaluate the potential of the FI to characterize the rate of individual BA. We show that the FI is a robust measure that provides an opportunity to introduce a physiological background for frailty parameters in sophisticated dynamic mortality models to adjust for variation in mortality. We also investigate health and sex differences in the FI's ability to

describe heterogeneity in individual aging rates. We show that FI age pattern for "healthy" elderly (i.e., those who, at younger ages, had a low mean FI) converges with that for disabled ("unhealthy") individuals (i.e., those who, at younger ages, had a high mean FI) at advanced ages. We interpret such convergence as a cross-sectional manifestation of compression of morbidity. Convergence of the FI age patterns indicates presence of BA age limits, which rather characterize given level of health maintenance in the society than a limit beyond which longevity cannot be extended.

2. Data

Waves of interviews of the NLTCS were conducted in 1982, 1984, 1989, 1994, and 1999. The NLTCS uses a sample of individuals drawn from national Medicare enrollment files. The survey instruments used in all five NLTCS waves asked the same disability, functional and medical condition questions in the same way to minimize bias in estimates. The likelihood of bias is also reduced by the high (95%) response rates in all NLTCS waves. The NLTCS samples contain longitudinal and cross-sectional nationally representative components.

A two-stage process was used to select NLTCS participants. First, a screening interview that assesses chronic disability is given to all members of the sample (roughly, 20,000 for each wave) – except persons who received a community or institutional detailed interview in a prior NLTCS who then are interviewed at each subsequent survey until death. Individual who reported in the screening interview at least one impairment in an (Instrumental) Activity of Daily Living, (I)ADL, that had lasted, or was expected to last, 90+ days were then given a detailed community or institutional interview.

To replace deceased individuals, and ensure that the screened sample is representative of the U.S. elderly, a new sample supplement (N~5,000) is drawn for each survey of persons who reached age 65 since the last NLTCS. All NLTCS records are linked to Medicare (to the end of 2001) and Medicare Vital Statistics (to August 6, 2003) files. The 1982 to 1999 NLTCS screener questionnaires represent about 42,000 different individuals. Detailed information on health and functioning of the community-survey participants is assessed from about 26,700 interviews in all five NLTCS waves.

In the 1994 NLTCS, an additional sub-sample of 1,762 "non-disabled" persons (the "healthy" supplement [HS]) was selected. These persons were designated to receive a detailed interview even if screened initially as non-institutional and unimpaired. The HS was selected from the entire sample, excluding only the 95+ supplement and persons who screened-in automatically because they had detailed interviews in 1989. The 1999 HS includes survivors from the 1994 HS (1,262), persons newly selected from the replacement (aged-in) component of the 1999 sample (219), and persons newly selected from the longitudinal component of the 1999 sample who were screened out in 1989 and not selected for the 1994 wave (64), producing a total of 1,545 persons.

3. Methods

The Frailty Index (FI). The NLTCS contains a wide set of self-reported measures on health and functioning. Consistent with the view of the FI as a measure of functional complexity, Rockwood et al. (2004) argue that only the proportion of deficits constituting the FI is important in its relation to aging and mortality – not their specific substance. This provides flexibility in choosing deficits to construct the FI. Nevertheless, to be able to validate prior findings, we restrict ourselves to deficits similar to those assessed in the CSHA (Mitnitski et al., 2004).

Specifically, we selected 32 questions, presented in all waves, and grouped them according to missing rate: (i) difficulty with eating, dressing, walk around, getting in/out bed, getting bath, toileting, using telephone, going out, shopping, cooking, light house work, taking medicine, managing money; (ii) arthritis, Parkinson's disease, glaucoma, diabetes, stomach problem, history of heart attack, hypertension, history of stroke, flu, broken hip, broken bones; (iii) vision problem; self-rated health; and (iv) trouble with bladder or bowels, dementia, hearing problem, visit of hearing therapist, dentist, and foot doctor. Following Mitnitski-Rockwood's approach, we define the FI as an unweighted count of such deficits divided by the total number of all deficits considered for a person. For instance, if an individual has been administered 30 questions and responded positively (indicating that there is a deficit) to 5 and negatively (no deficit) to 24 then his/her FI is $5/29 \approx 0.172$.

Missing Data. Complete information was gathered in the NLTCS on questions covering disability, part of which is represented in first group (13 measures). The second group (11 measures) represents answers with very low percentage of missing data ranging from 0.07% to 1.3%. In the third group (2 measures), the variability of the proportion of missing answers across the five NLTCSs is slightly larger (0.6% to 3.7%). The fourth group (6 measures) represents questions with low proportions of missing data (about 0.5%), but which were not administered to all NLTCS participants. Since, for most questions, the proportion of missing data is small, the maximum number of available responses (i.e., from questions administered to NLTCS participants) is 30 for all waves. We constructed two FIs: one covering all 32 deficits and the other only the first three groups (i.e., 26 deficits).

4. Results

We first evaluate the FI age-patterns for each NLTCS wave. Despite the relatively large samples, estimates for single years of age are not sufficiently precise at the advanced ages (90+) where there are less than 100 cases per year. To improve statistical precision, and to smooth estimates, we used two-year age groupings in our analyses. Figure 1 shows the two-year age-patterns of the full (32 deficits) FI for five waves. The 26-deficit FI shows a similar age-pattern and thus is not depicted.

Figure 1 is about here.

Visual inspection of the age-patterns in Figure 1 reveals a nonlinear (accelerated) increase of the FI with age. Sex-differences of the 2-year FI age-patterns were not statically significant. Averaging the FI over 5 years of age (Table 1) shows that statistically significant differences between FIs for males and females are seen only for the 90-94 age group of the 1982 NLTCS and for 3 age groups (70-74, 80-84, 90-94) of the 1994 NLTCS. For the entire sample (65+) mean FIs for males and females are statistically different for each NLTCS, being lower for males than for females.

Table 1 is about here.

To find the best description of the age-patterns in Figure 1, we estimated several functions: linear, log-linear (or exponential), power, and quadratic. In all five NLTCS waves, and for FIs with both 32- and 26-deficits, the best fit is obtained by the quadratic function, $FI = U + B_1 \times Age + B_2 \times Age^2$, as determined by comparisons of coefficients of determination

(R^2). T-test shows statistical significance for all coefficients except for B_1 and U for the 1994 wave. Because the quadratic function has three parameters, the standard errors of its coefficients are larger than for the log-linear ($\ln(FI) = U + B_1 \times Age$) function (Table 2). For comparison, Table 2 also shows R^2 for linear functions in parenthesis. Two-year averaging significantly improved these estimates increasing the percentage of the total variation in dependent variables explained by nonlinear relations between age and FI by up to 50%. The use of five-year age categories did not noticeably improve fits. Thus, a quadratic function accurately describes the FI age-patterns in NLTCS data (Figure 1). The best fit was obtained for 1989 ($R^2 = 98\%$).

Table 2 is about here.

Despite the qualitative (shape) similarity of the age-patterns, there are quantitative differences among the waves. The largest mean age-specific FIs are for the 1982 NLTCS (Figure 1). Their difference from those of the other NLTCS waves is likely due to over-sampling of disabled individuals in the 1982 community questionnaire (91.2% in 1982 vs. 83.5% in 1984). Deviations of the 1982 NLTCS FI estimates from the exponential pattern of the age specific FI in the CSHA is also the largest – that is also reflected in the regression coefficients (Table 2).

In 1994, the NLTCS design was changed by adding the HS. The community questionnaire was completed by 1,303 persons (of 1,762 in the HS) in 1994 and by 1,196 (of 1,545 in the HS) in the 1999 wave. Since individuals in the HS were designated before the survey to receive a detailed interview, the proportion of non-disabled individuals in these groups is significantly lower than in the remaining ("disabled") group (DG) of individuals (selected for a community interview by the screener), being closer to the proportion in the U.S. elderly. Specifically, according to the age-adjusted estimates by Manton & Gu (2001), the prevalence of non-disabled elderly individuals in the national U.S. population in the 1999 was 80.3% and in 1994 was 77.5%. The prevalence of non-disabled respondents to the community questionnaire in the HS of the NLTCS without age standardization is 65.5% in 1999 and 80.5% in 1994. The over-sampling of "healthy" individuals reduces the mean FI for the 1994 and 1999 waves – especially at "younger" ages providing good agreement with results from CSHA.

The presence of the HS in the 1994 and 1999 waves provides an opportunity to estimate the difference between survey and community samples distinguishing the age-patterns of the DG and HS. Figure 2 shows that the age-pattern for the DG shifts up becoming closer to the 1982-1989 patterns. Meanwhile, age-patterns for the HS shift down exhibiting smaller mean FIs than those assessed from CSHA for all age groups. Again, better fits are obtained for the quadratic function except for the 1999 HS, for which the log-linear (exponential) fit is better (Table 3).

Figure 2 is about here.
Table 3 is about here.

Figure 2 suggests that individuals in the HS (small FI at young ages) accumulate deficits faster than those in the DG (large FI). To increase statistical power, we pooled data for 1994 and 1999 waves and averaged the FI over 5 years of age. Figure 3 exhibits the 1994&1999 FI age-patterns for the entire sample (left panel) and for both sexes (right panel) along with their nonlinear fits (Table 4). Figure 3 clearly shows that individuals from the HS accumulate deficits faster than those from DG. The rate of deficit accumulation varies by sex. This is also seen considering each wave separately and averaging FI over larger age intervals (Table 5).

Specifically, males in the HS have smaller FI at younger ages than females. However, males accumulate deficits faster than females resulting in convergence of their FI age-patterns and crossing at advanced ages (~85).

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5. Discussion and Conclusions

Our analyses show that the mean FI increases with age, and that this increase is nonlinear (with acceleration), i.e., older people accumulate more deficits per year than younger. In most cases, the age-pattern is best described as a quadratic function. This means that the rate of increase also increases with age (in a linear fashion) stressing the nonlinear nature of deficit accumulation. The best fits, when quadratic fits are insignificant, were exponential. Correlation of the FI with age and similarity between the FI and mortality age patterns suggest that the FI could be used as an adequate indicator of BA (Mitnitski, Mogilner, & Rockwood, 2001). Although, usually, BA indicators are expected to have linear relation with chronological age (Karasik, Demissie, Cupples, & Kiel, 2005), it can be argued that the relation should be nonlinear. One argument for that is the high plasticity and age-dependence of mortality rate *variation* in experiments with anti-aging interventions aimed to increase longevity (Vaupel, Carey, & Christensen, 2003). Valid biomarkers of aging must capture these properties, i.e., they must have a nonlinear relation with chronological age.

The FI appears to have the potential to differentiate aging processes at individual level. Consequently, FI becomes useful characteristic describing population heterogeneity in various models of aging and mortality, which can be implemented using, for instance, microsimulation procedures designed to assess the impact of individual states (Akushevich, Kulminski, & Manton, 2005).

Our results reveal large differences between the FI age-patterns for the 1982, 1984, and 1989 NLTCS waves as compared to the 1994 and 1999 waves which appear due to the presence of a "healthy" sample in the two later waves. Only the patterns for the last two waves resemble those from the CSHA. The CSHA sample is representative of elderly (65+) Canadians who are being screened according to cognitive function (Mitnitski et al., 2001).

Our results show that survey design is a serious issue in constructing FIs even using similar community-based samples. This occurs because intentional, or unintentional, screening can result in over-representation of individuals with certain health/quality-of-life deficits. The NLTCS community sample is an example of an intentional selection of disabled individuals by screening and sample selection procedures. We dealt with that feature of the NLTCS sampling by stratifying on the HS versus the non-HS (DG). The CSHA focuses on selection of cognitively impaired elderly which, as a consequence of their mental abilities, have larger proportions of health deficits and poorer quality of life measures than those with intact cognitive functions (Heinik, 2004). Therefore, even if a survey does not directly focus on specific aspects of the individuals' health which constitute large part of the deficits included in the FI definition, such individuals can be over-sampled in the survey (i.e., the survey sample can approximate a non-community setting) thereby increasing the mean FI. Consequently, it is reasonable to expect that mean FIs for survey participants can be larger than for community-dwelling individuals provided that such deficits are part of the FI definition.

The presence of the HS in the NLTCS allowed us to directly confirm this fact. Individuals for the DG were selected following standard NLTCS procedures (i.e., screening in disabled individuals), while for the HS they were selected irrespective of disability. Since the screener NLTCS participants were primarily selected from the U.S. Medicare eligible persons to provide nationally representative sample according to demographic factors, the likelihood of systematic bias resulting in selection of individuals for HS with specific health problems is low.[1] Our analysis shows that the FIs for the general population of community-dwelling elderly should be lower than those estimated using particular surveys.

NLTCS data provide evidence on complex (nonlinear) relationships between the FI, sex, and age. To understand this complexity, we make four observations. First, the mean FI for males is smaller than for females for each NLTCS wave. This agrees with other findings (Mitnitski et al., 2004). However, this difference is not large. Second, there is not, generally, a statistically significant sex difference between age-specific FIs. Third, there is no overall tendency that the FI for males is less than for females. Fourth, analysis of the sex-specific FIs for different age groups shows two opposite tendencies in the sex-sensitivity of the FI behaviors with age (Table 1). Specifically, at younger ages in the early waves, FIs are nearly identical but have tendency to diverge with age. For the two latest waves, there is a tendency towards convergence of these indices at the extreme ages. Since the two later waves have a smaller proportion of disabled individuals due to the presence of the HS, it is reasonable to assume that the latter fact is responsible for such a change. Indeed, when considering the DG and HS separately (Figure 3), the qualitative change of the FI with age becomes more pronounced. Males and females in the DG have essentially similar FIs at younger ages — the opposite fact is seen for the HS. This is a clear nonlinear effect when the relation between FIs for males and females is FI- and age-dependent. A consequence is that in different settings (e.g., institutional vs. hospital vs. community) the relation between FI for males and females can be qualitatively different.

The intriguing finding of our study is that FIs for HS and DG converge at the oldest-old ages, i.e., the rate of deficit accumulation for individuals in the HS is larger than in the DG. We interpret this finding as a cross-sectional manifestation of compression of morbidity when "healthy" people accumulate health deficits faster than "unhealthy". This finding suggests that aging process itself rather than particular pathologies plays pivotal role in the risk of death at extreme ages. Such behavior becomes even more pronounced in male and female sub-groups. The rate of deficit accumulation for females is larger than for males for the DG. For the HS, we see the opposite situation. As a consequence, the difference in the rates results in divergence of FI age-patterns for males and females in DG and in their convergence in the oldest-old ages for the HS. In other words, for large FI at younger ages the FI age-patterns appear flatter than those for small FI at younger ages. Figure 3 (right panel) also suggests that sensitivity to the quantity of the accumulated deficits is higher for males than for females. This follows from the fact that males and females accumulate deficits with age at different rates and differently in the DG and the HS. Changes in rates between DG and HS are larger for males than for females.

These findings provide further support for considering the FI as a measure of BA. Since humans have limited life spans (i.e., no individuals live an unlimited time, although, the life-span-limits might change with improvements in economic standard of living, social conditions,

[1] This fact has been also verified by comparing the FI age-patterns for the HS and for the U.S. community-dwelling elderly. The latter sample was obtained from respective NLTCS wave (1994 or 1999) using weights developed by the U.S. Bureau of the Census and the Center for Demographic Studies (Duke University) to produce national estimates. Both (weighted and HS) estimates show excellent agreement, especially at younger ages.

and medical progress [Riley, 2001]), the FI – as a BA indicator – should be able to characterize BA limits associated with given level of health-maintenance in the society (Fogel, 1997). Specifically, in a community setting (approximated by the HS in our analysis), males and females have smaller mean FIs, especially at younger ages, than age-peers in non-community groups (e.g., the DG). However, the FI increases with age faster in the HS than in the DG. This may be due to the presence of a BA limit. Our data provide an opportunity to determine a BA limit from the extrapolated fits. For the HS and DG samples, this occurs at age 104.5 years at $FI = 0.435$. Individuals with both elevated (DG) and normal (HS) FI level at younger ages can reach this BA limit. However, individuals from the HS would have to age faster to reach the same limit.

Our data suggest that males and females have different BA limits. Interpolation of the female-specific fits for the HS and DG to extreme ages provides a reasonable estimate for the females' BA limit of approximately 109.4 years ($FI = 0.456$). For males, we obtain a lower limit of 92.5 years. This estimate, however, was imprecise due to the small sample of males at those ages. The difference in the BA age limits for males and females may be the reason why there are opposite tendencies in the sex-specific FI age-patterns in the DG and HS. Indeed, since males have a lower BA limit, those who are in the HS accumulate deficits with age faster than females. For the same reason, males in the DG accumulate deficits with age slower than females.

The presence of BA limit does not mean that longevity cannot be extended beyond certain age. It rather exhibits systemic feature of the aging process and indicates the need of development of adequate systemic methods of copying with this phenomena. Such methods focusing on slowing down the rate of deficit accumulation will result in extension of both life span and active live life span, even if the BA limit will remain unchanged. Consequently, health-care providers should focus their efforts not only on individuals with serious health problems, but also on "healthy" individuals (i.e., with mild health problems) at younger ages to reduce the likelihood of fast nonlinear accumulation of heath deficits at advanced ages. At the same time the progress in medical technology may affect the BA limit as well. How all such transformations will affect the quality of life at late ages deserves separate study.

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Table 1. Sex-specific FIs and the number of males (M) and females (F) for each NLTCS. An asterisk (plus) denotes $0.01 < p \le 0.05$ ($p \le 0.01$). Other sex differences are insignificant.

NLTCS	Sex	N	65+	Age groups						
				65-69	70-74	75-79	80-84	85-89	90-94	95+
1982	M	2166	.266 (.003)$^{+}$	.244 (.007)	.262 (.006)	.259 (.007)	.289 (.009)	.289 (.010)	.311 (.018)*	.337 (.027)
	F	3921	.277 (.002)$^{+}$	.243 (.005)	.252 (.005)	.274 (.005)	.282 (.005)	.311 (.006)	.352 (.010)*	.374 (.021)
1984	M	2038	.250 (.003)*	.229 (.007)	.238 (.007)	.244 (.007)	.264 (.008)	.283 (.011)	.321 (.018)	.355 (.034)
	F	3891	.259 (.002)*	.226 (.005)	.231 (.005)	.244 (.005)	.269 (.005)	.297 (.006)	.349 (.010)	.344 (.018)
1989	M	1470	.241 (.004)$^{+}$	.235 (.010)	.214 (.007)	.234 (.008)	.259 (.010)	.274 (.013)	.294 (.021)	.341 (.055)
	F	2992	.258 (.003)$^{+}$	.230 (.007)	.226 (.006)	.247 (.005)	.261 (.006)	.283 (.007)	.337 (.010)	.376 (.020)
1994	M	1736	.191 (.004)$^{+}$	.150 (.008)	.163 (.008)*	.187 (.006)	.206 (.009)$^{+}$	.255 (.013)	.255 (.023)*	.335 (.031)
	F	3336	.221 (.003)$^{+}$	.159 (.007)	.184 (.006)*	.195 (.004)	.238 (.006)$^{+}$	.272 (.007)	.317 (.012)*	.339 (.012)
1999	M	1805	.196 (.004)$^{+}$	.162 (.008)	.148 (.008)	.190 (.008)	.212 (.007)	.247 (.011)	.256 (.020)	.353 (.023)
	F	3341	.220 (.003)$^{+}$	.167 (.007)	.165 (.006)	.202 (.005)	.224 (.005)	.266 (.008)	.290 (.012)	.344 (.013)

Table 2. Coefficients for the log-linear (Ln) and quadratic (Q) functions along with coefficients of determination (R^2) for each NLTCS wave. R^2 in parentheses is given for linear functions for the sake of comparison. Estimated coefficients are significant at the 0.05 level or better. Superscript "#" denotes insignificant estimates.

NLTCS	Fit	B_1 $(SE)\times 10^2$	B_2 $(SE)\times 10^4$	U (SE)	R^2, %
1982	Ln	1.37 (.071)		-2.346 (.058)	96.1 (93.6)
	Q	-1.10 (.313)	0.93 (.193)	0.563 (.125)	97.7
1984	Ln	1.63 (.139)		-2.615 (.113)	90.2 (87.7)
	Q	-1.90 (.597)	1.46 (.368)	0.846 (.239)	94.2
1989	Ln	1.66 (.157)		-2.655 (.128)	88.2 (86.0)
	Q	-2.81 (.360)	2.03 (.222)	1.202 (.144)	98.0
1994	Ln	2.72 (.116)		-3.689 (.095)	97.3 (96.3)
	Q	-0.58 (.517)$^{\#}$	0.75 (.318)	0.210 (.207)$^{\#}$	97.4
1999	Ln	2.67 (.150)		-3.667 (.122)	95.5 (93.8)
	Q	-1.63 (.514)	1.39 (.317)	0.627 (.206)	97.4

Table 3. Coefficients for the quadratic and log-linear (denoted by "#") functions fitting data in Figure 2 for the HS and DG in 1994 and 1999 NLTCS waves. R^2 is also given for linear (Lin) function. For all estimates $p \le 0.05$.

NLTCS	Group	B_1 $(SE)\times 10^2$	B_2 $(SE)\times 10^4$	U (SE)	R^2, %		
					Q	Ln	Lin
1994	HS	-3.66 (.95)	2.73 (.61)	1.30 (.37)	95.1	92.3	86.2
	DG	-1.54 (.55)	1.27 (.34)	0.65 (.22)	95.8	92.9	91.6
1999	HS#	4.83 (.46)		-5.79 (.36)	89.0	90.2	87.3
	DG	-1.54 (.45)	1.23 (.28)	0.69 (.18)	96.2	92.5	91.0

Table 4. Coefficients of the best statistically significant ($p<0.05$) fits corresponding to the curves in Figure 3.

Group	Sex	Fit	B_1 $(SE)\times10^2$	B_2 $(SE)\times10^4$	U (SE)	R^2, %
HS	M&F	Ln	4.42 (.27)		-5.45 (.22)	98.1
	M	Q	-5.05 (.96)	3.84 (.62)	1.73 (.37)	99.3
	F	Ln	4.02 (.31)		-5.08 (.25)	97.1
DG	M&F	Q	-1.51 (.23)	1.24 (.15)	0.66 (.09)	99.7
	M	Ln	1.57 (.34)		-2.68 (.27)	81.0
	F	Q	-1.07 (.37)	0.98 (.23)	0.48 (.14)	99.4

Table 5. Mean FIs for males and females for age-specific age groups for HS and DG of 1994 and 1999 NLTCS waves.

Sex	Group	Age	1994		1999	
			Mean FI	SE	Mean FI	SE
Male	HS	65-74	.065	.005	.084	.007
		75-84	.097	.007	.129	.007
		85+	.237	.041	.217	.026
	DG	65-74	.218	.007	.189	.007
		75-84	.227	.006	.229	.006
		85+	.270	.011	.267	.010
Female	HS	65-74	.093	.006	.107	.007
		75-84	.125	.006	.138	.006
		85-94	.187	.019	.216	.016
		95+	.222	.074	.385	.054
	DG	65-74	.217	.006	.192	.006
		75-84	.239	.004	.236	.004
		85-94	.299	.007	.284	.007
		95+	.343	.012	.343	.013

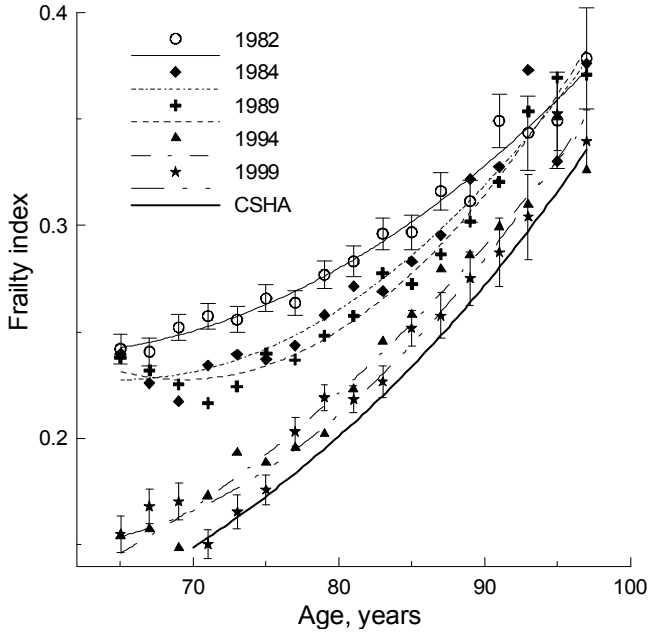

Figure 1. The two-year FI age-patterns for each NLTCS along with model estimate of the FI age distribution for the CSHA (thick line *FI=exp(0.029Age-4.05)*, Mitnitski et al. [2004]). The standard errors ($\pm SE$) of means are shown by bars for the 1982 and 1999 waves.

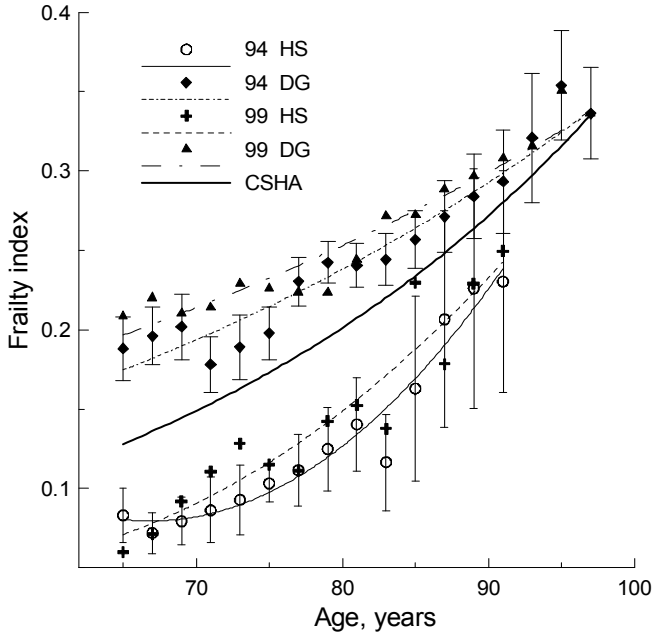

Figure 2. The two-year FI age-patterns for the HS and DG for the 1994 and 1999 NLTCS waves. The 95% confidence intervals (CI) of means are shown by bars for 1994 HS and 1994 DG.

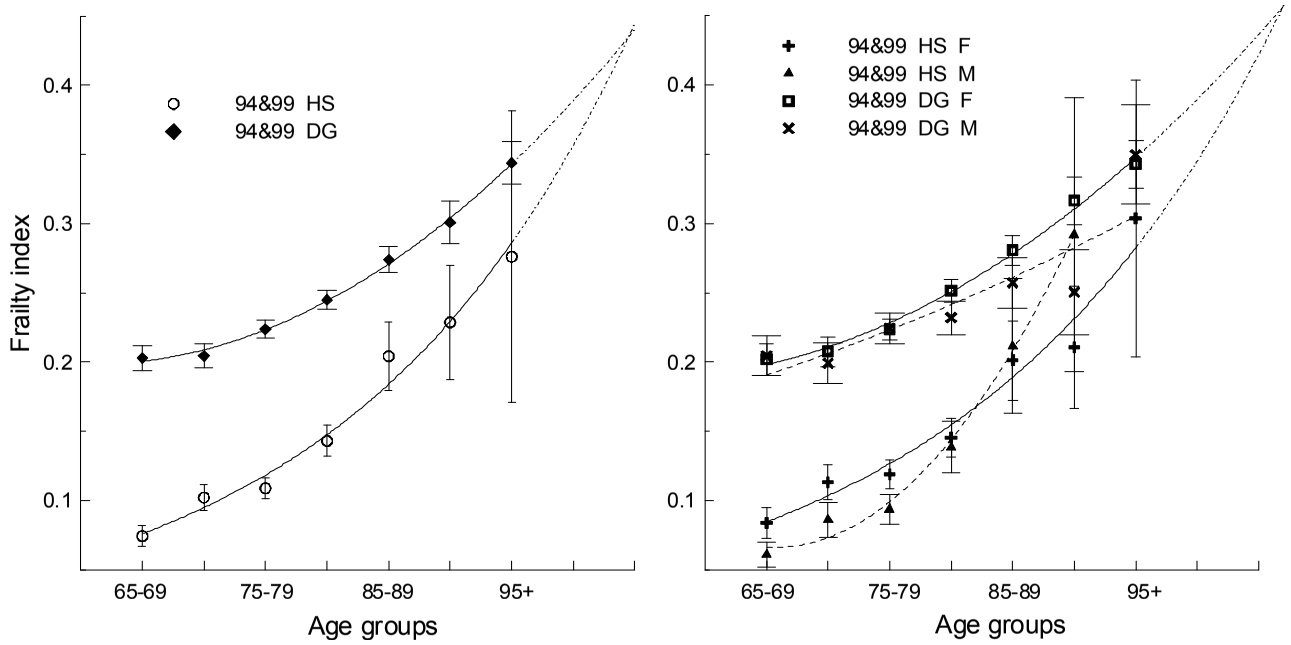

Figure 3. The five-year FI age-patterns for the HS and DG for pooled 1994&1999 data for entire sample (left panel) and for both sexes (right panel). Dashed-dotted line denotes extrapolation of the respective fitted curves. Bars show 95% CI. Dashed (continuous) line on the right panel denotes fits for males (females).