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Time-to-death patterns in markers of age and dependency

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Abstract

We aim to determine the extent to which variables commonly used to describe health, wellbeing, and disability in old-age vary primarily as a function of years lived (chronological age), years left (thanatological age), or as a function of both. We analyze data from the US Health and Retirement Study to estimate chronological age and time-to-death patterns in 78 such variables. We describe results from the birth cohort born 1915-1919 in the final 12 years of life. Our results show that most markers used to study well-being in old-age vary along both the age and time-to-death dimensions, but some markers are exclusively a function of either time to death or chronological age, and others display different patterns between the sexes.

Background

For an individual, age across the life course consists of two components: time since birth and time to death, the *chronological* and *thanatalogical* dimensions of age, respectively. In the aggregate, thanatological age is determined

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by the mortality rate schedule to which a birth cohort is subject until its extinction. Individuals do not know their thanatological age with certainty. To guess this quantity one projects an expectation of lifespan based on scenarios or extrapolations of how mortality rates might change over time. Data classified by chronological age, like census population counts, can be reclassified into thanatological age in this way.[1]

Prospectively, decreasing mortality has the effect of moving population into higher thanatological ages, thereby increasing remaining life expectancy (Sanderson and Scherbov 2005). In this case, the notion and measure of future remaining lifespan is elastic, subject to uncertainty. In retrospect (after the death of a cohort), the thanatological age structure of a population is a fixed characteristic. Furthermore, since a closed birth cohort is akin to a stationary population,[2] the chronological and thanatological age profiles are identical (Brouard 1989, Vaupel 2009, Rao and Carey 2014). Yet, even in the case of stationary populations, the age profiles of other demographic characteristics in the population are decidedly different when viewed chronologically versus thanatologically. Distinct patterns emerge in the aggregate due to an interaction between lifespan variation and the age profile(s) of demographic characteristics.[3] In the Results section, we provide an example of a deceptive pattern over chronological age that is due to such an interaction with lifespan.

Some life transitions, states, and changes in state intensities are almost exclusively a function of time to death. There are other instances where chronological age captures almost all pertinent variation. In cases where a characteristic strongly varies as a function of time to death, the common practice of aggregation over chronological age may misrepresent time trends and misguide analyses about change over time and expectations for the future. Measurement of the end-of-life trajectories of characteristics is useful in such cases as a way of separating mortality patterns from patterns in characteristics themselves. Characteristic measurements are taken while the respondent is alive, but thanatological age at each interview is unknown until

[1] A paper on this topic is currently under review (Riffe et al. 2014). Brouard (1986; 1989) had already done the same thing some 30 years earlier, and we understand that S. Scherbov also unwittingly produced the same result over a decade ago.

[2] The age structure of a birth cohort over time is proportional to the $l(x)$ column of the lifetable that describes its mortality, which is proportional to the stable age structure determined by the Lotka-Euler renewal model when the intrinsic growth rate is equal to zero.

[3] When we state that a characteristic is a function of either age perspective we do not imply that age causes the given characteristic to vary, but rather that a characteristic varies in some smooth, regular, or parsimonious way over age.

the date of death is known, and is therefore retrospectively assigned. This final analytical step lends clarity to the understanding of how characteristics vary within and between lifespans.

Incorporating a time-to-death perspective in demographic studies is especially important when assessing the impact of "population ageing". To the extent that the health, welfare, and social care demands of a population are functions of thanatological rather than chronological age structure, forecasts of the social and economic "costs" of ageing that are based only on chronological age profiles are prone to misinterpretation. In our concluding discussion we return to this point.

Research exploring time-to-death patterns has been done in other domains, and topics examined can be roughly categorized into two types: 1) things that are a function of apparent or perceived time to death (Hamermesh 1985, Hurd and McGarry 1995, Carstensen 2006, Gan et al. 2004, Bíró 2010, Salm 2010, Van Solinge and Henkens 2010, Cocco and Gomes 2012, Payne et al. 2013, Balia 2013), and 2) things that are a function of actual time to death (Miller 2001, Seshamani and Gray 2004, Werblow et al. 2007). The first kind are mostly studies on cognitive transitions and economic or health behaviors, while the second kind are mostly studies on health expenditure. Another branch of research relates perceived and actual remaining lifetime (Perozek 2008, Delavande and Rohwedder 2011, Post and Hanewald 2012, Kutlu-Koc and Kalwij 2013). In this paper we will expand the second group, focusing on a broad range of questions from ten waves of the US Health and Retirement Study (HRS 2013).

We aim to understand the end-of-life age patterns of various dimensions of morbidity, as measured by a set of 78 characteristics and indices. To do this, we score the degree to which these characteristics vary in terms of thanatological age, chronological age, or both. In all, we define four different age and lifespan axes, which we use to classify the end-of-life patterns of each characteristic tested. The axis along which a given characteristic varies ought to determine how we measure, understand, and respond to the characteristic. We show that often chronological age ought to be used in conjunction with thanatological age in order to classify patterns, but in many cases chronological age provides no information at all, and it even obfuscates true temporal patterns.

Our analytical approach is retrospective rather than prospective, meaning that no lifetable assumptions are made in the measurement of thanatological age, and no censoring adjustments are necessary. Although more data are available for adjacent cohorts, we report results only for the cohort born from 1915 to 1919, which contains the most extensive set of observa-

tions in the dataset used. In the following section we describe the methods in greater detail. We then demonstrate the four age axes by way of example, and summarize all characteristics tested in terms of these four axes. Finally, we discuss some implications and applications of this work.

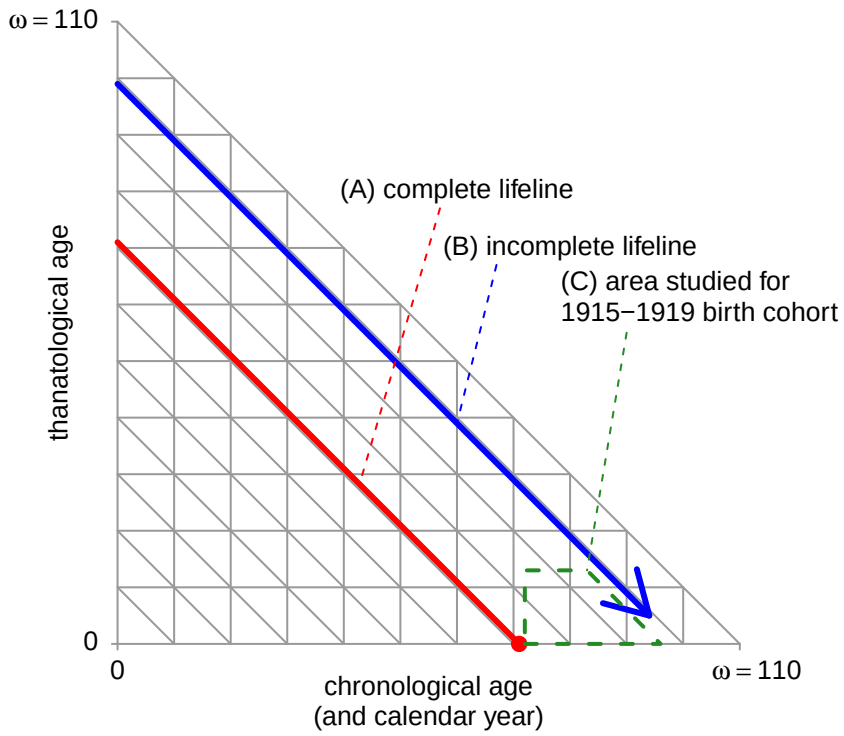
Data & Method

All findings reported in this paper are based on data from the US Health and Retirement Study (HRS). We use the Rand edition of the data, which is conveniently merged across all ten waves. This data is free to download, and all details of data processing and methods are made freely available in an open code repository.[4] We restrict the sample to only those individuals born between 1900 and 1930 that died between 1992 and 2011, which narrows the dataset down to 37051 observations of 9238 individuals. 8137 of these observations are from the 1919 individuals that died from the 1915-1919 cohort. Adjacent cohorts are kept for the sake of a smoother loess fit to the data, which we describe in the following paragraphs.

Underpinning this investigation are a series of demographic surfaces indicating the average intensity of a given marker along the chronological and thanatological time dimensions within a series of quinquennial birth cohorts, from which we focus only on the central 1915-1919 birth cohort. This visual tool is similar to but orthogonal to the familiar Lexis surface. Figure 1 orients the reader with the temporal coordinates we use. This diagram represents the various possible lifespans within a given birth cohort, with an arbitrary final age, ω, of 110. One's thanatological age at birth is equal to one's chronological age at death, such that both axes close out with ω. Members of the birth cohort are born on the left side of the diagram, at chronological age zero and with an unknown y coordinate (remaining lifetime) at the time of birth. Lifelines advance downward and to the right, where the downward direction indicates the approach to death, and the rightward direction represents both the progression of calendar years and chronological age. The blue arrow (B) indicates a hypothetical lifeline that will eventually expire at age 99, although this property is unknown until death. The present study contains only complete lifelines, such as that depicted in the color red (A) in Figure 1, which completes its lifespan at age 71. In this diagram, diagonal lines represent death cohorts, rather than the birth cohorts found in the standard Lexis diagram.

[4]This repository includes R code used to process data, as well as generate results and figures: `https://github.com/timriffe/ThanoEmpirical`

Figure 1: Years lived and years left over the lifespan of a birth cohort



We limit the current study to the 1915-1919 cohort due to the characteristics of the data source. Using the HRS, enough observations are available such that we can measure the patterns of within the area outlined in green (C) in Figure 1. The left bound of this area is chronological age 72, and the diagonal right bound belongs to the completed lifespan of 95. While there are some observations at thanatological ages greater than 12, there are too few to produce reliable estimates.[5] Future waves will expand the area applicable to all but the oldest birth cohorts that are already extinct in the data.

The 1915-1919 birth cohort was exposed to the 1918 Spanish influenza

[5]Since the HRS spans 20 calendar years (1992-2011), the theoretical upper bound of observation for thanatological age is 20.

epidemic as toddlers (1915-1917 cohorts), as infants (1917-1918) cohort and in-utero (1919 cohort). There is some evidence that this exposure manifested in various ways in late life (e.g., Almond 2006), and so the reader may rightly question whether the results presented here are in some way anomalous. While more precise methods may detect effects, the methods we expound here are not precise. Specifically, the smoothing procedure we apply borrows information from adjacent birth cohorts, which itself may erase whatever otherwise detectable health artifacts this cohort may have carried into late life. At these ages, we assume that other risk factors, some of them cumulative over the life course, senescence itself and other forces likely drive patterns to a much greater extent. Our justification is here speculative, but we report that the results for this cohort do not appear visually distinct from those present in other cohorts. More importantly, our goal here is not to describe the end-of-life experience of this birth cohort, but to add resolution to the measurement and description of ageing and morbidity indicators, and contribute to the practice of demography in general.

Age Thanatological age is calculated for each individual as the lag between interview and death dates expressed as decimal years. Chronological age is calculated as the lag between birth and interview date in decimal years. Each individual is therefore assigned a chronological and thanatological age at each interview, along with measures of our variables of interest. Since we are interested in viewing characteristics over both chronological age and thanatological age simultaneously, we require observations spread over a wide range of combinations of thanatological and chronological age.

The current HRS dataset runs from 1992 to 2011, which means that each birth cohort is observed over a different range of ages. For example, the 1925-1929 cohort enters observation in 1992 at age 62 (at the youngest) and acheives a maximum completed age of 85 by the end of 2011. On the other end, the 1905-1909 enters the HRS in 1992 at age 72 at the youngest and has a maximum completed lifespan of 105 by the last wave in 2011, albeit with few observations at the upper extreme. Results from these and other birth cohorts are also valid, but portions of these surfaces are based on fewer data points (lifespans > 100) or ages in which labor market exits appear to drive patterns at least as much as senescence (ages < 67, approximately). We selected the 1915-1919 cohort because its observation window is centered on the chronological ages in which most deaths occur and in which most recent mortality improvements in low-mortality countries have occurred ,[6]

[6]Own calculations based on UN data (United Nations, Department of Economic and

and because the HRS provides a good density and spread of data points over this window. The lower and upper age bounds may vary if questions were not available in the first, second or final waves.

Characteristics We aim for a broad overview of the age variation across different dimensions of old-age disability and wellbeing. For this reason we select a wide variety of questions from the HRS data. These include questions grouped roughly into the following categories:

1. Activities of Daily Living (ADL): six items, and two composite indices.
2. Instrumental Activities of Daily Living (IADL): seven items and two composite indices.
3. Health Behaviors: five items.
4. Functional Limitations: six items.
5. Chronic Conditions: eight items and one composite index.
6. Cognitive Function: 15 items and two composite indices.
7. Psychological Wellbeing: nine items and one composite index.
8. Healthcare Utilization: 14 items.

The the specific variables included in our survey are found in the appendix tables following the same numbering scheme as above. In all, we summarize results from 78 individual and composite items. We excluded variables that were not asked continuously from at least wave 3 through 9. Variables not available in the first or second wave have left age bounds at higher ages than 72, whereas items not asked in wave ten have upper lifespan bounds that are below 95.

Each survey question must be in a format suitable for numeric operations. This requires some compromises in data quality, since some coded responses are less directly quantifiable, and our translation of categorical or ordinal responses to numeric values was at times improvised. In most cases, responses are easy to imagine as "good" and "bad", in which case we assigned higher values to "bad" and lower values to "good" outcomes. For

Social Affairs, Population Division 2013). The modal ages at death for the 1915-1919 cohort are 80-81 for males and around 87 for females. These calculations are based on partially observed cohort mortality rates, $M(x)$ (Human Mortality Database).

example, respondents were asked if they felt depressed. We assigned 0 to answers of "no" and 1 to answers of "yes". Rather than divide all questions into binary responses, we assigned intermediate values on a case by case basis. For example, self-reported health had possible responses of "excellent", "very good", "good", "fair", and "poor", which we assigned values in equal intervals from 0 to 1, respectively. Some response sets for particular questionnaire items changed between waves. In these cases, we attempted to assign numerical codes that were consistent over the transition. These recodes are imprecise, but they are good enough to meet the goals of this study. In other words, the surfaces we present are not exact measurements, but are meant to provide *impressions* about how characteristics change over age.[7]

Variables with compact or bounded numeric responses were rescaled to range from 0 to 1. Variables with no clear bounds or very large upper bounds, such as medical expenditure, body mass index, or number of hospital visits were not rescaled. These rescalings are intended to simplify the visual interpretation of surfaces, and they do not alter the quantitative summary measures we use later.

Some questionnaire items in the HRS are only asked every second interview. In these cases, we impute within-individual trajectories assuming a linear trend. For example one item asks respondents whether they experience back pain. If in wave 3 an individual responded "no", wave 4 is skipped, and in wave 5 the respondent answered "yes", then we assign 0 to wave 3, 0.5 to wave 4 and 1 to wave 5 for this question. If a response is missing in a given wave, but available in the previous wave, we carry it over as a constant, but we do not impute backwards in time. We also do not impute questions that were not part of the questionnaire for a given wave. These within-individual procedures reduce missing cases for within valid interviews by 30-40%, which in some cases provides our statistical procedures with a better fit, but does not skew results.

Weighting The population universe of the HRS and this study is the resident population of the United States. Therefore person weights are needed in order to estimate population-level means. One difficulty with the HRS is that the institutionalized population is treated as a second target population. In all waves but 5 and 6, there are no person weights assigned to

[7]The pre-processing of variables is full of details that would clutter this paper. Rather than a lengthy and detailed appendix describing the case by case treatment of variables, we make the full code base used in the generation of results available in an open repository.

institutionalized individuals. We try to impute missing person-weights according to some simple assumptions. If the individual was assigned a weight in a previous wave, we carry this weight over as a constant, unless there was also a non-zero weight in a future interview, in which case we assign the weight according to the within-individual linear pattern. Individuals and interviews that still have missing person-weights after this procedure are discarded from our study. Person weights compensate for minor detectable attrition in the HRS (Kapteyn et al. 2006), which for our purposes may be considered unbiased [8].

Loess smoothing Direct tabulations of the weighted data are legible if all birth cohorts are combined, but doing this distorts results due to cohort composition bias. To overcome birth cohort heterogeneity within surfaces, we use birth cohorts as a third time dimension. Tabulations within this three dimensional space are noisy, and so we enhance surface legibility by using a non-parametric local smoother. We specify a loess model of the given characteristic over chronological age, thanatological age, and quinquennial birth cohorts, using all observations of since-deceased individuals from the 1900 through the 1934 birth cohorts. We fit the model using the `loess()` function in base `R` (Cleveland et al. 1992, R Core Team 2013)[9] to the weighted individual-level data for each sex separately, and then predict a surface for the 1915-1919 birth cohort within the study area outlined in green (C) in Figure 1. Weighting is therefore explicit by person-weights, and implicit by point density within the three temporal dimensions.[10]

[8] Small biases in the survey only appear with respect to baseline characteristics that we do not consider. Attrition due to health conditions, e.g., mental impairment, is mostly mitigated due to the use of proxy respondents in such situations (Weir et al. 2011).

[9] Using the fitted model, surfaces are produced using the related loess prediction function, `predict.loess()`. The smoothing parameter, `spar`, is set to 0.7 for the results we present in the paper. All results were also produced using smoothing parameters of .5, and .9, and we concluded that the specific choice of smoothness does not drive results. The three predictor dimensions are not normalized, in order to preserve year units.

[10] Note that smoothing over these three particular time dimensions is not an overidentification. Within a cohort, to smooth over thanatological age, chronological age and completed lifespan would be an overidentification, similar to the familiar APC problem. The full set of lifespan indices the demographer has to choose from are: birth cohort, death cohort, chronological age, thanatological age, complete lifespan, and period. Within this set of six lifespan dimensions, some combinations invoke overidentification and others do not. For instance, it would be possible to smooth over years lived, years left, and period in this case, but birth cohorts are the more meaningful category for this study.

Results

We first present examples of four surfaces that exemplify the major ways in which characteristics tend to vary temporally over the lifespan within a birth cohort. These four major patterns of variation provide a way to categorize and understand markers of ageing. We summarize the results of our set of 78 characteristics by calculating Pearson correlation coefficients for each of these four axes and display results graphically, as well as in an appendix table.

Four major surface axes In most situations it is obvious to the eye whether a variable operates over thanatological age or over chronological age, but there are many instances where both are at play, or where the relationship is complex. We first present surfaces representing psychological problems for males (Figure 3a) and back pain for females (Figure 3b). These two surfaces are examples of thanatological and chronological characteristics, respectively.

From the direction of the contours on the surface in Figure 3a, we conclude that the chances of ever having been diagnosed with psychological problems increases with the approach to death and not with the advancing of chronological age, at least in the window of observation studied here. However, since the risk of death itself also increases according to an exponential pattern in these same ages, aggregating individual results by chronological age produces an increasing pattern over age for this same characteristic (see Figure 2). In this case, the apparent chronological age pattern is due to an interaction between the thanatological pattern seen in Figure 3a and the age pattern of mortality itself. We argue that it is imprecise to consider chronological age a risk factor for characteristics that display such strong thanatological patterns, as an apparent chronological age pattern along said margin is a deceptive artifact. Instead, such characteristics appear to more closely operate as effects of the body shutting down or possibly as a signal on average that death is not far off, a demographic corroboration of substantive findings in the psychology literature (Carstensen 2006). Many characteristics studied here display patterns that are strongly thanatological.

Figure 3b tells just the opposite story about back pain for females. Back pain is a function of chronological age, at least at the population level until around chronological age 90. This is the dominant way of thinking about most aspects of the ageing process. In these ages, back problems provide no information about remaining years of life. Of the characteristics included in this study, only current smoking, arthritis, and self reports of current versus

former memory exhibit such clear chronological patterns (both for males and females).

Figure 2: Psychological problems (ever) by chronological age only. Males, 1915-1919 birth cohort. With 95% confidence bands from loess fit.

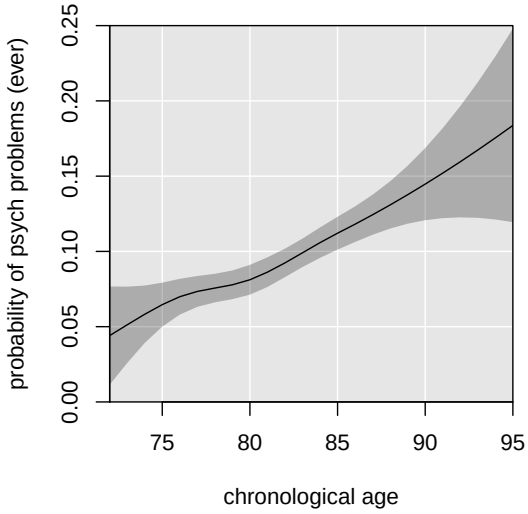
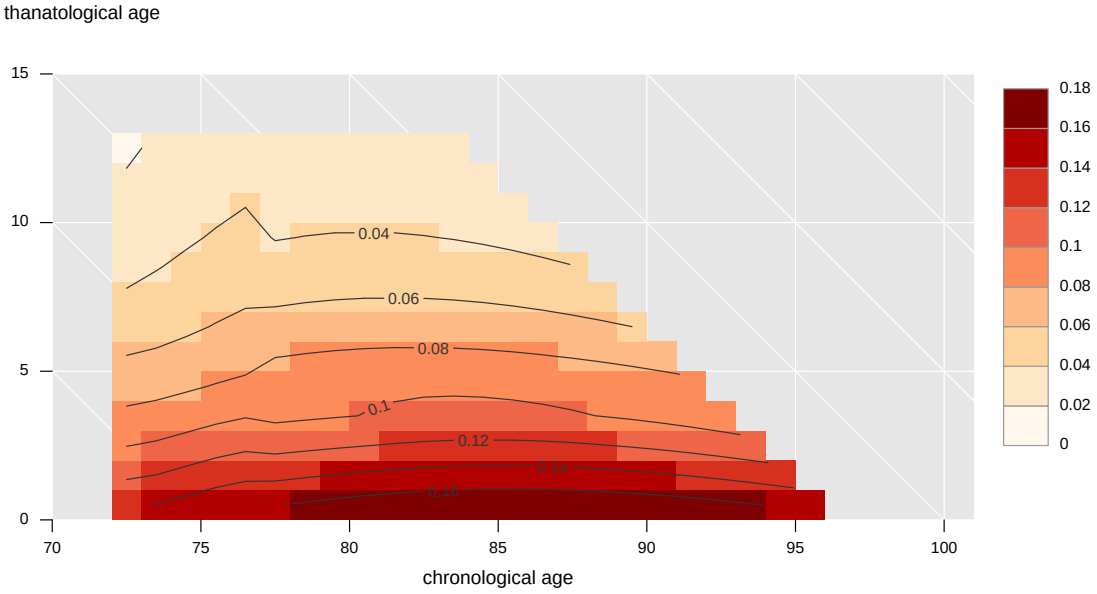
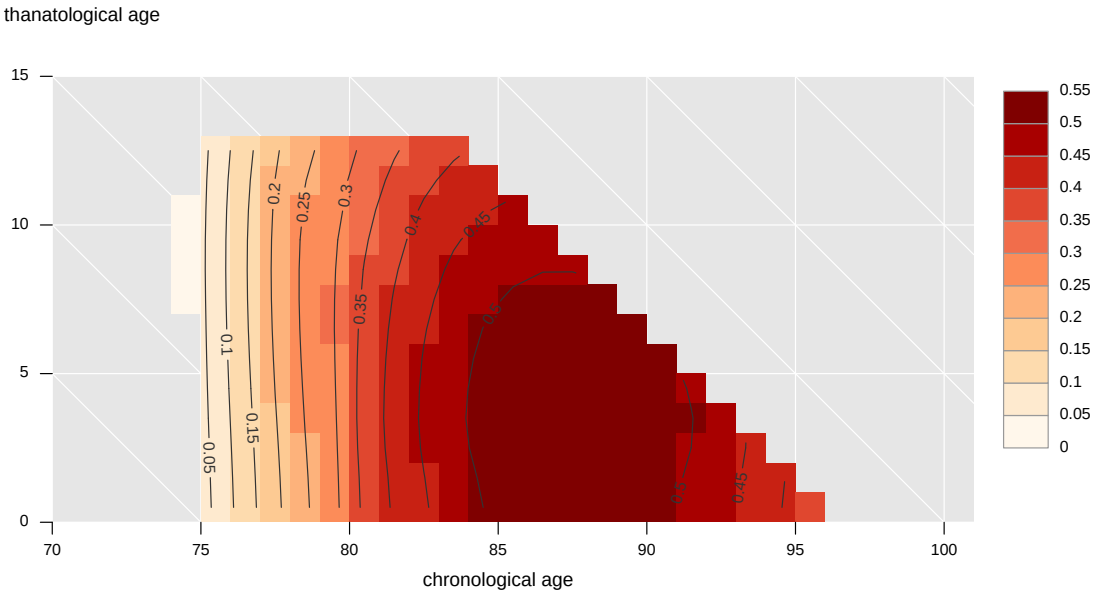


Figure 3: Examples of characteristics that vary along the thanatological and chronological age axes.

(a) Psychological problems (ever) by years lived (x axis) and years left (y axis). Males, 1915-1919 birth cohort.



(b) Back Problems by years lived (x axis) and years left (y axis). Females, 1915-1919 birth cohort.



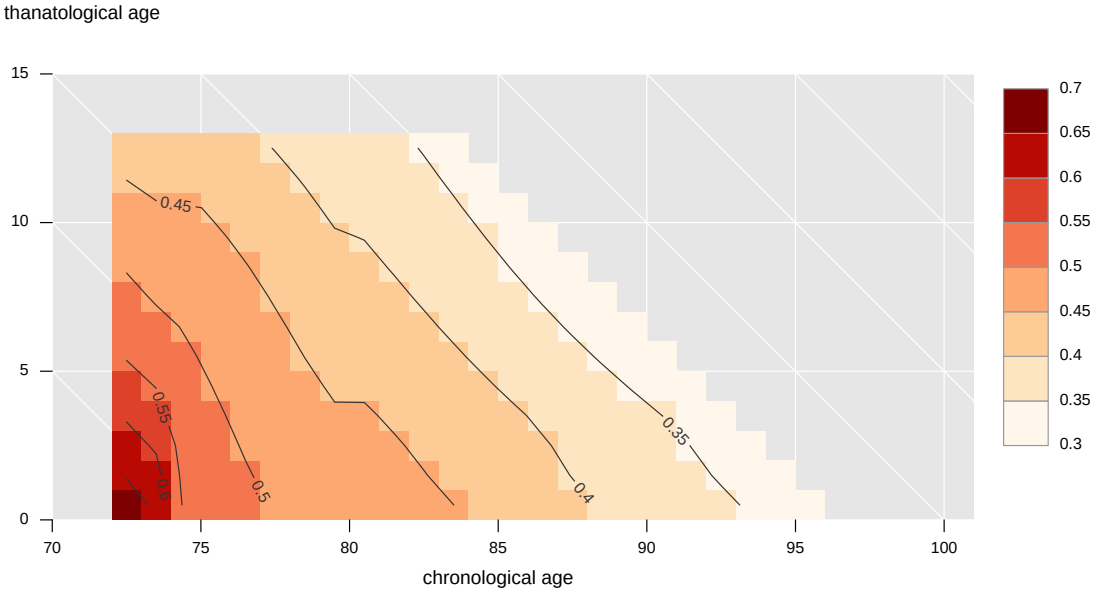
Other informative patterns also exist among the set of characteristics studied. These include characteristics that vary by lifespan. Characteristics that vary by lifespan appear constant within lifespans. These are often characteristics that *determine* lifespan. Ever smoking displays such a pattern, as seen in Figure 4a for females of the 1915-1919 cohort. This pattern is also a corroboration of science and common sense: smoking kills (at least in this range of lifespans). Other variables that display similar patterns in this window of the lifespan include lung disease among males (this is largely redundant with the former), dental visits in the previous year (both sexes), and diabetes among females. Sometimes such patterns combine in complex ways worthy of further study.

The fourth major axis of contour variation runs perpendicular to lifelines. One characteristic that clearly displays this pattern is ever having been diagnosed with high blood pressure among males. This characteristic varies by lifespan, and thanatological age within lifespan within the window of study. In other words, longer lifespans display later onset but greater eventual odds of having been diagnosed with high blood pressure. Arithmetically, $years\ lived - years\ left$ is the operative predictor of blood pressure. For example, for such characteristics, the condition of a 70-year old with five remaining years of life may resemble that of an 80-year old with 15 remaining years of life. Such characteristics are not very useful alone for predicting eventual lifespan.[11] Contours such as this imply that variation for a characteristic

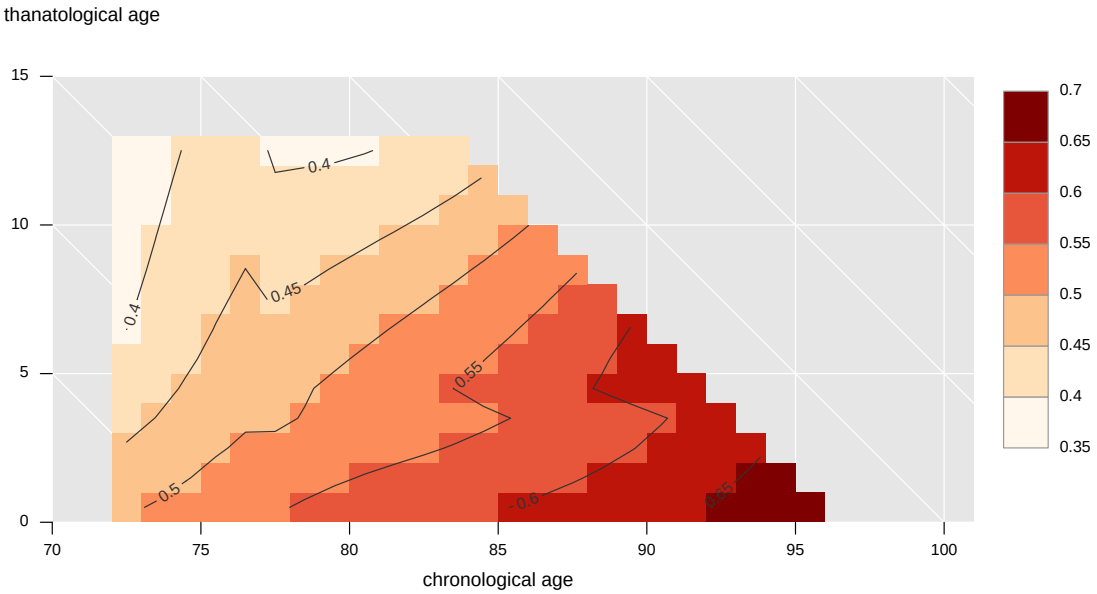
[11] We do not have expertise to comment further on blood pressure, but instead only provide an interpretation of the surface presented.

Figure 4: Examples of characteristics that vary by lifespan only or by thanatological age within lifespan.

(a) Smoking (ever) by years lived (x axis) and years left (y axis). Females, 1915-1919 birth cohort.



(b) Blood Pressure by years lived (x axis) and years left (y axis). Males, 1915-1919 birth cohort.



Summary of results for all characteristics Surfaces such as those in Figures 3 and 4 were produced for all 78 variables and each sex. We distill these surfaces into four Pearson correlation coefficients, each designed to capture the variation along one of the axes explained above. We call the four axes thanatological, chronological, lifespan ($chrono + thano$), and mixed ($chrono - thano$). For a given surface, we calculate the correlation coefficient of the matrix elements against the four margin indices one at a time (rather than using the survey microdata). Most characteristics are summarized nicely by either one or two of these axes. We display these correlation coefficients by juxtaposing perpendicular axes in scatter plots separately for males and females.

Figure 5: Thanatological versus chronological correlation, with selected characteristics labeled. 1915-1919 birth cohort, chronological ages 72-95.

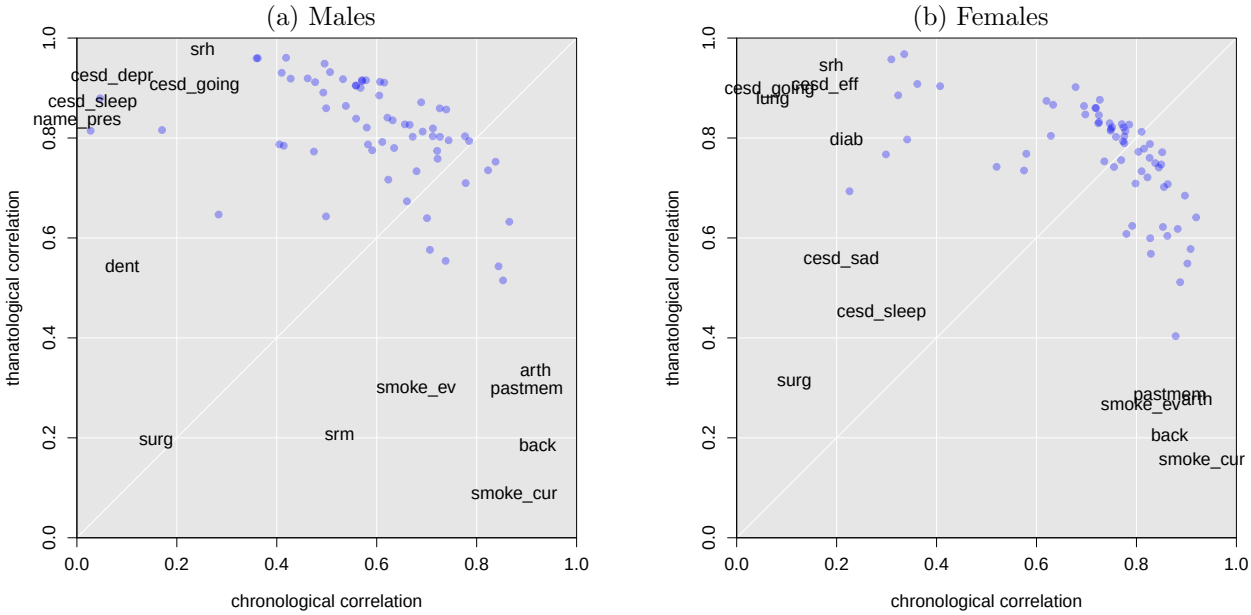


Figure 5 displays the simple correlation coefficients that belong to the chronological and thanatological axes. For males, 45 variables display thanatological correlations of greater than 0.8, versus nine chronological correlations above the same threshold. For females, the figures are 32 and 29, respectively, a different picture overall. Both point clouds are in high thanatological ages, but females lean further towards chronological variation within this cohort. That thanatological patterns are somewhat less accentuated for

females may corroborate aspects of the existing literature on sex differences in morbidity and mortality (Case and Paxson 2005).

Figure 6: Lifespan versus mixed correlation, with selected characteristics labeled. 1915-1919 birth cohort, chronological ages 72-95.

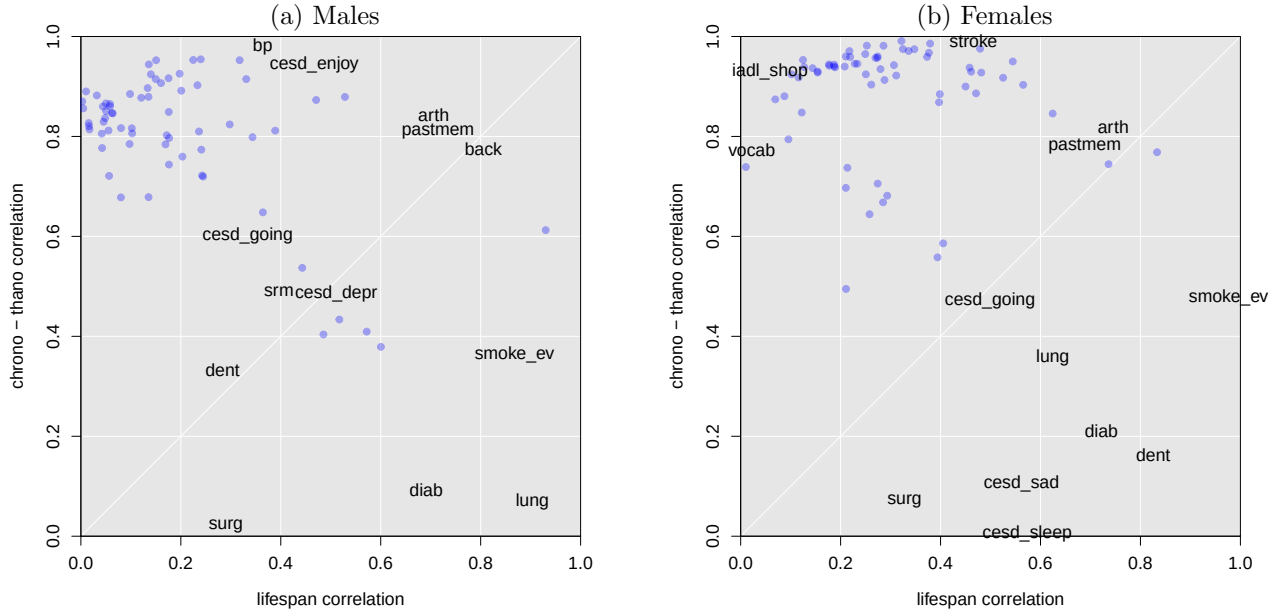


Figure 6 displays correlation coefficients that belong to the lifespan and "mixed" axes. Among males, four variables display lifespan correlations of greater than 0.8, versus 49 mixed correlations above the same threshold. For females, the respective figures are three and 55. The mixed axis ($chronoage - thanoage$) is the winner among the set of characteristics tested. The appendix provides detailed results.

Discussion

The distribution of tested characteristics with respect to the four axis orientations described here is striking. However, these findings must be tempered by noting that 1) the summary measure (correlation coefficient) used here blends out some information, 2) these results may not necessarily extrapolate to the set of all testable questions in the HRS, and 3) this relationship does not necessarily hold in other windows of the lifespan or other birth

cohorts. Further, the patterns presented here are valid for the whole population (of a given sex) taken together, but were the target population split by causes of death (for instance), the patterns may change. For example, imagine hypothetically that the strong thanatological pattern shown in Figure 3 (psychological problems) were driven by strong patterns within individuals that eventually die of suicide, but that other causes of death displayed entirely different patterns with respect to psychological problems. Such cases are easily imaginable for other characteristics and causes of death. At the time of this research, we did not have access to cause of death information from the HRS mortality followup. For detailed investigations of particular characteristics, cause-conditioning surfaces would clearly aid in disentangling morbidity processes.

Research to better document the multidimensional age variation of particular characteristics would benefit from more careful measurement than that conducted here. Despite such shortcomings, the principle aim of this study has been satisfied: this survey of characteristics highlights the complex variety of age and lifespan dimensions over which some key aspects of the aging process unfold. All of the indicators we tested are commonly used to describe population ageing, and very few of them are exclusively a function of chronological age. If this finding is sustained in other cohorts and populations, and if other indicators here untested also display similar temporal complexity, we submit that the common discourse and debate on the nature and impacts of ageing ought to be better informed by more judicious measurement and description in terms of thanatological and chronological age.

We hope that the conceptual model of the lifespan presented here, which complements the Lexis diagram, will be of use to demographers. Other combinations of lifespan time dimensions are also possible, and these would highlight different patterns in data. The variety and availability of such options, perhaps now placed in starker relief, demands a more nuanced understanding of the temporal accounting that relates demographic time perspectives. Further exploration and experimentation with these formal demographic concepts will lead to a more judicious toolkit for demographic measurement and the practice of demography, and ultimately a wiser contribution to the discourse on population ageing. A series of direct applications and implications derive from the concepts and results presented in this paper.

First, if compared between two timepoints, demographic work such as this will provide a more precise answer to the question or morbidity compression. Given the chronological age ruse exemplified in the case of psychological problems (see Figures 2 versus 3a), it is safe to say that unless

retrospective thanatological measurements of morbidity dimensions are undertaken, we do not have direct information about whether compression is (or has been) happening or not. Using the techniques shown here, the researcher may directly estimate the varieties of end-of-life profiles often seen in the literature on morbidity compression (e.g., Fries et al. 2011). That is, changing chronological age patterns may be coincidental.

Second, large scale panel studies may be motivated to implement, increase, or improve mortality follow-up modules. Information on the full age dimensions of health outcomes will be valuable. The good news is that many unlinked panel studies may be linked to death registers in retrospect. A few populations with long-running and fully linked population registers already preside over such information, and we encourage a more thorough exploration of the temporal richness in population change and population characteristics. Underused as it is, the Lexis surface does not tell the whole story!

Third, health care providers and the public may better situate the association of certain health outcomes with stages of the ageing process. This is both a question of allocating resources and a question of how individuals conceive of themselves with respect to age. In this regard, we add to the chorus of researchers working to change the measurement of age to reflect the changing experience of age (see e.g., Sanderson and Scherbov 2013).

Fourth, this material highlights important sex differences in the onset and trajectory of some aspects of morbidity. Some of these differences may corroborate extant findings, and others may provide new understanding to sexual dimorphism in morbidity. In general, these methods and measurements are applicable to describe any between-group disparity in demographic or social outcomes, most of which directly or indirectly relate to remaining years of life.

We do not, at this time, attempt to thoroughly cluster characteristics based on the scores of the four different correlation coefficients, but this may be a fruitful exercise for further work. It is our hope that these results are strongly suggestive and orient future investigation.

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A Variables and correlations

For tables displayed in this appendix we use a shorthand to identify axis types. T indicates the correlation coefficient along the thanatological age axis. C indicates the chronological age axis. $C + T$ indicates the lifespan axis (right-downward slanting isolines). $C - T$ indicates the mixed axis, the

most common type in this data. Results are available on request as machine readable data, and the code used to generate these and all other results is available freely in a repository:

`https://github.com/timriffe/ThanoEmpirical`

Results are grouped by several major morbidity categories.

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Table 1: Activities of Daily Living (ADL)

Variable		Males				Females			
Short	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
adl3	ADL 3-point	0.83	0.67	0.10	0.89	0.80	0.76	0.21	0.94
adl5	ADL 5-point	0.83	0.63	0.06	0.86	0.81	0.75	0.19	0.94
adl_walk	Diff. walking across room	0.86	0.54	0.06	0.81	0.83	0.72	0.15	0.93
adl_dress	Diff. dressing	0.82	0.71	0.15	0.92	0.82	0.75	0.19	0.94
adl_bath	Diff. bathing or showering	0.84	0.62	0.04	0.86	0.82	0.75	0.19	0.94
adl_eat	Diff. eating	0.80	0.67	0.12	0.88	0.76	0.77	0.25	0.92
adl_bed	Diff. getting in/out bed	0.82	0.58	0.02	0.82	0.83	0.72	0.15	0.93
adl_toilet	Diff. using toilet	0.84	0.56	0.02	0.81	0.73	0.81	0.31	0.94

Table 2: Instrumental Activities of Daily Living (IADL)

Variable		Males				Females			
Short	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
iadl3	IADL 3-point	0.89	0.60	0.00	0.87	0.77	0.80	0.27	0.96
iadl5	IADL 5-point	0.90	0.57	0.05	0.85	0.83	0.75	0.18	0.94
lim_work	Health limits work	0.93	0.51	0.08	0.82	0.97	0.34	0.27	0.71
iadl_map	Diff. using map	0.73	0.82	0.32	0.95	0.79	0.83	0.29	0.98
iadl_tel	Diff. using telephone	0.80	0.78	0.23	0.95	0.70	0.85	0.37	0.96
iadl_money	Diff. managing money	0.91	0.56	0.06	0.85	0.81	0.78	0.22	0.96
iadl_meds	Diff. taking meds	0.92	0.46	0.17	0.78	0.79	0.77	0.23	0.94
iadl_shop	Diff. shopping for groceries	0.92	0.58	0.05	0.87	0.90	0.68	0.06	0.93
iadl_meals	Diff. preparing hot meals	0.92	0.57	0.06	0.86	0.82	0.77	0.21	0.96

Table 3: Health behaviors

	Variable	Males				Females			
[redacted]	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
alc_ev	Alcohol, ever	0.78	0.41	0.08	0.68	0.62	0.79	0.40	0.89
alc_days	Alcohol nr of days / week	0.82	0.17	0.44	0.54	0.80	0.34	0.26	0.64
alc_drinks	Alcohol nr drinks per drinking day	0.89	0.49	0.18	0.80	0.75	0.84	0.27	0.96
smoke_ev	Ever Smoker	0.30	0.68	0.87	0.37	0.27	0.81	0.98	0.48
smoke_cur	Current Smoker	0.09	0.88	0.93	0.61	0.15	0.93	0.83	0.77

Table 4: Functional limitations

	Variable	Males				Females			
[redacted]	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
bmi	BMI	0.92	0.53	0.05	0.83	0.74	0.76	0.29	0.91
back	Back problems	0.18	0.92	0.81	0.78	0.21	0.87	0.74	0.74
mob	Mobility difficulty index	0.91	0.61	0.01	0.89	0.88	0.73	0.12	0.95
lg_mus	Large muscle difficulty index	0.86	0.74	0.15	0.95	0.81	0.81	0.25	0.98
gross_mot	Gross motor difficulty index	0.91	0.56	0.06	0.85	0.86	0.72	0.13	0.94
fine_mot	Fine motor difficulty index	0.80	0.73	0.18	0.92	0.79	0.78	0.23	0.94

Table 5: Chronic conditions

Variable		Males				Females			
Short	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
bp	High blood pressure, ever	0.75	0.84	0.36	0.98	0.87	0.63	0.09	0.88
diab	Diabetes, ever	0.65	0.28	0.69	0.09	0.80	0.22	0.72	0.21
cancer	Cancer, ever	0.93	0.41	0.18	0.74	0.96	0.31	0.29	0.68
lung	Lung disease	0.64	0.50	0.90	0.07	0.88	0.07	0.62	0.36
heart	Heart problems, ever	0.96	0.36	0.24	0.72	0.83	0.77	0.25	0.96
stroke	Stroke, ever	0.95	0.50	0.10	0.82	0.69	0.90	0.47	0.99
psych	Psych problems , ever	0.96	0.36	0.24	0.72	0.61	0.78	0.40	0.87
arth	Arthritis, ever	0.34	0.92	0.71	0.84	0.28	0.92	0.75	0.82
cc	Nr chronic conditions	0.91	0.61	0.03	0.88	0.76	0.83	0.35	0.97

Table 6: Cognitive function

Variable		Males				Females			
[redacted]	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
srm	Self-rated memory	0.20	0.53	0.40	0.49	0.51	0.89	0.56	0.90
pastmem	Memory compared to past	0.29	0.90	0.71	0.81	0.28	0.87	0.69	0.78
ss	Serial 7s	0.76	0.72	0.20	0.89	0.86	0.69	0.10	0.92
c20b	Backwards counting	0.80	0.71	0.16	0.91	0.71	0.80	0.31	0.92
name_mo	Naming month	0.77	0.47	0.06	0.72	0.62	0.88	0.46	0.94
name_dmo	Naming day of month	0.79	0.79	0.24	0.95	0.85	0.70	0.12	0.92
name_yr	Naming year	0.80	0.74	0.20	0.93	0.64	0.92	0.48	0.97
name_dwk	Naming day of week	0.73	0.68	0.18	0.85	0.74	0.84	0.34	0.97
name_sci	Naming scissors	0.79	0.41	0.14	0.68	0.55	0.90	0.53	0.92
name_cac	Naming cactus	0.54	0.84	0.47	0.87	0.71	0.86	0.38	0.97
name_pres	Naming president	0.83	0.00	0.57	0.41	0.85	0.72	0.14	0.94
name_vp	Naming VP	0.79	0.58	0.04	0.81	0.74	0.52	0.01	0.74
vocab	Vocabulary score	0.79	0.61	0.02	0.83	0.73	0.57	0.02	0.77
tm	Mental status summary	0.81	0.69	0.13	0.90	0.83	0.79	0.22	0.97
dwr	Delayed word recall	0.83	0.66	0.14	0.88	0.80	0.78	0.27	0.96
twr	Total word recall	0.77	0.72	0.23	0.90	0.78	0.81	0.33	0.97
iwr	Immediate word recall	0.71	0.78	0.33	0.92	0.75	0.85	0.38	0.99

Table 7: Psychological wellbeing

Variable		Males				Females			
[redacted]	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
cesd	Depression score	0.96	0.42	0.24	0.77	0.89	0.32	0.28	0.67
srh	Self-reported health	0.98	0.25	0.36	0.65	0.94	0.19	0.41	0.59
cesd_depr	Felt Depressed	0.92	0.07	0.51	0.49	0.69	0.23	0.21	0.49
cesd_eff	Everything an effort	0.81	0.03	0.48	0.40	0.91	0.18	0.39	0.56
cesd_sleep	Sleep restless	0.87	0.03	0.52	0.43	0.45	0.29	0.57	0.01
cesd_happy	Was happy	0.72	0.62	0.17	0.80	0.91	0.36	0.21	0.70
cesd_lone	Felt lonely	0.87	0.69	0.14	0.92	0.57	0.83	0.47	0.89
cesd_sad	Felt sad	0.91	0.48	0.10	0.79	0.56	0.21	0.56	0.11
cesd_going	Could not get going	0.90	0.24	0.33	0.60	0.90	0.07	0.50	0.47
cesd_enjoy	Enjoyed life	0.63	0.86	0.47	0.94	0.87	0.62	0.07	0.87

Table 8: Healthcare utilization

Variable		Males				Females			
Short	Long	T	C	$C+T$	$C-T$	T	C	$C+T$	$C-T$
hosp	Overnight hospital: 24 mo	0.78	0.59	0.10	0.81	0.75	0.74	0.26	0.90
hosp_stays	Nr hospital stays: 24 mo	0.86	0.50	0.04	0.78	0.80	0.63	0.12	0.85
hosp_nights	Number nights in hospital: 24 mo	0.88	0.05	0.60	0.38	0.77	0.58	0.10	0.79
nh	Overnight stay nursing home: 24 mo	0.64	0.70	0.30	0.82	0.62	0.85	0.46	0.93
nh_stays	Nr nursing home stays: 24 mo	0.67	0.66	0.24	0.81	0.60	0.86	0.48	0.93
nh_nights	Nr nights nursing home: 24 mo	0.58	0.71	0.34	0.80	0.60	0.83	0.45	0.90
nh_now	Nursing home at interview	0.78	0.64	0.05	0.84	0.72	0.82	0.28	0.94
doc	Dr visit: 24 mo	0.52	0.85	0.53	0.88	0.40	0.88	0.62	0.85
doc_visits	Number Dr visits: 24 mo	0.55	0.74	0.39	0.81	0.58	0.91	0.54	0.95
hhc	Home health care: 24 mo	0.91	0.57	0.01	0.86	0.86	0.72	0.18	0.94
meds	Prescription drugs regularly: 24 mo	0.92	0.43	0.20	0.76	0.90	0.41	0.21	0.74
surg	Outpatient surgery: 24 mo	0.19	0.16	0.29	0.02	0.31	0.12	0.33	0.07
dent	Dental visit: 24 mo	0.55	0.09	0.28	0.33	0.77	0.30	0.83	0.16
shf	Special health fac visit: 24 mo	0.86	0.73	0.14	0.94	0.77	0.85	0.32	0.99