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[1] 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 (Brouard 1986).

Prospectively, decreasing mortality is equivalent to 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 cohort at a given past point in time is a fixed characteristic. Since a closed birth cohort is akin to a stationary population,[2] one may be tempted to assume that since chronological and thanatological age structures are symmetrical in stationary populations (Brouard 1989, Vaupel 2009, Villavicencio and Riffe 2016) that the patterns of demographic characteristics within cohorts might also demonstrate an analogous kind of symmetry. This is not so; even in the case of stationary populations or extinct cohorts, the age profiles of other demographic characteristics in the population are decidedly different when viewed chronologically versus thanatologically. If the demographic characteristics in question are states, such as health states, one can confirm that for cohorts the mean duration spent in each state is indeed identical, no matter whether measured by chronological or thanatological age. Cohort expectancies are immune from age classification biases. However, distinct patterns emerge in period aggregates due to an interaction between lifespan variation and the age profiles of demographic characteristics.

Some life transitions, states, and changes in state intensities are almost exclusively a function of time to death. 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. 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

[1] Lifespan is used throughout as a synonym for chronological age at death, or thanatological age at birth. These concepts are identical with length of life, which is not to be confused with life expectancy, the mean length of life..

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

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 observation is unknown until the date of death is known, and it 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 aging." 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 aging that are based only on chronological age profiles are prone to bias (Stearns and Norton 2004).

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, Wolf et al. 2015, Stearns and Norton 2004). The first kind are mostly studies on cognitive transitions and economic or health behaviors, while the second kind are mostly studies on health expenditure, except Wolf et al. (2015), which proposes a model to separate latent time-to-death trajectories. A third 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 (RAND 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 pattern families, which we use to classify the end-of-life prevalence 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 earlier and later cohorts, we report results only for the cohort born from 1915 to 1919, which contains the most extensive set of observations 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

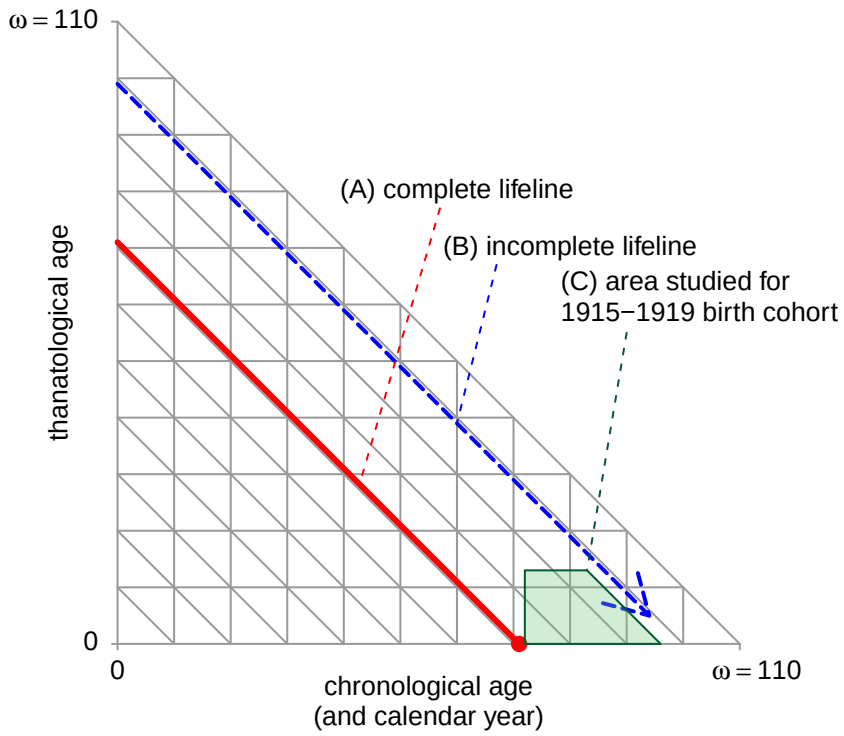
All findings reported in this paper are based on data from the US Health and Retirement Study (HRS). We use version M of the RAND edition of the data, which is conveniently merged across all ten waves available as of 2013. These data are free to download, and all details of data processing and methods are made freely available in an open code repository.[3]

We restrict the sample to only those individuals born between 1900 and 1930 who died between 1992 and 2011, which narrows the dataset down to 37,051 interviews of 9,238 individuals. Of these interviews, 8,137 are from the 1,919 individuals who died from the 1915-1919 cohort. Observations from earlier and later cohorts are kept for the sake of adding information when fitting models to the data.

Underpinning this investigation are a series of demographic surfaces indicating the average prevalence of a given marker along chronological and thanatological time axes 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 rep-

[3]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



resents 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 (or lifespan cohorts), as opposed to the birth cohort diagonals found in the standard Lexis diagram.

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 from the 1915-1919 cohort 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. Since the HRS spans 20 calendar years (1992-2011), the theoretical upper bound of observation of thanatological age is 20. However, individuals in this sample between thanatological ages 13 and 20 (i.e., individuals that entered the study around 1992 and also died around 2011) are scarce, and so we study only thanatological ages less than or equal to 12, ergo the final 12 years of life. As further waves are added to the HRS and mortality linkage continues, the portion of the lifecourse that may be studied in this way will expand.

The 1915-1919 birth cohort was exposed to the 1918 Spanish influenza epidemic as toddlers (1915-1917 cohorts), as infants (1917-1918) cohort, and in-utero (1919 cohort). There is evidence that this exposure manifested in various ways in late life (e.g., Almond 2006, Myrskylä et al. 2013), and so the reader may rightly question whether the results presented here are anomalous. The potential anomalous effects from this cohort are "smoothed-out" in our analysis, due both to the width of the cohort and to the nature of the statistical method we use to estimate aggregate patterns from individual observations. Specifically, loess smoothing borrows information from observations in earlier and later cohorts. Further, at these ages we assume that other risk factors, some of them cumulative over the life course, and senescence itself likely drive health patterns to a much greater extent than might early-life selection or late-life onsets of poor health due to the Spanish influenza. We also verify that patterns for this cohort do not appear visually distinct from those found in earlier and later cohorts. More importantly, our goal here is not to describe the end-of-life experience of this particular birth cohort, but to add resolution to the measurement and description of aging 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.

Version M of the RAND 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 82 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 obtained from these data, 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 focus on 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,[4] and because the HRS provides a good density and spread of data points over this window. The lower and upper age bounds 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 Use: 14 items.

[4] Own calculations based on UN data (United Nations, Department of Economic and 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 2015).

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 exclude 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 ages higher 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 based on selected cutpoints. For example, respondents were asked if they felt depressed. We assigned 0 to answers of "no" and 1 to answers of "yes". As an example of ordinate recoding, self-reported health had possible responses of "excellent", "very good", "good", "fair", and "poor", which we assigned values of 0, 0, 0, 1, and 1, respectively. In this way, population means for this kind of variable can be interpreted as prevalences.

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 body mass index or number of hospital visits were not rescaled. These rescalings are intended to simplify the visual interpretation of surfaces, as a diagnostic, and they do not alter the quantitative summary measures we use later. 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.[5]

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 individuals living in institutions. We try to impute missing person-weights according to some simple assumptions. If the individual was assigned a

[5]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, please consult the annotated code in the open repository.

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 a 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 [6].

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)[7] 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.[8]

[6] 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).

[7] 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.

[8] 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 aging. 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 shaded 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 2a) and back pain for females (Figure 2b). These two surfaces are examples of thanatological and chronological characteristics, respectively.

From the direction of the contours on the surface in Figure 2a, 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 approximate exponential pattern in these same ages, aggregating individual results by chronological age produces an increasing pattern over age for this same characteristic (see Figure 3). In this case, the apparent chronological age pattern is due to an interaction between the thanatological pattern seen in Figure 2a 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). Ceteris paribum, mortality itself ought to be a good proxy for characteristics that are highly thanatological. Some characteristics studied here display patterns that are strongly thanatological.

Figure 2b 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 85. This is the dominant way of thinking about most aspects of the aging 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).

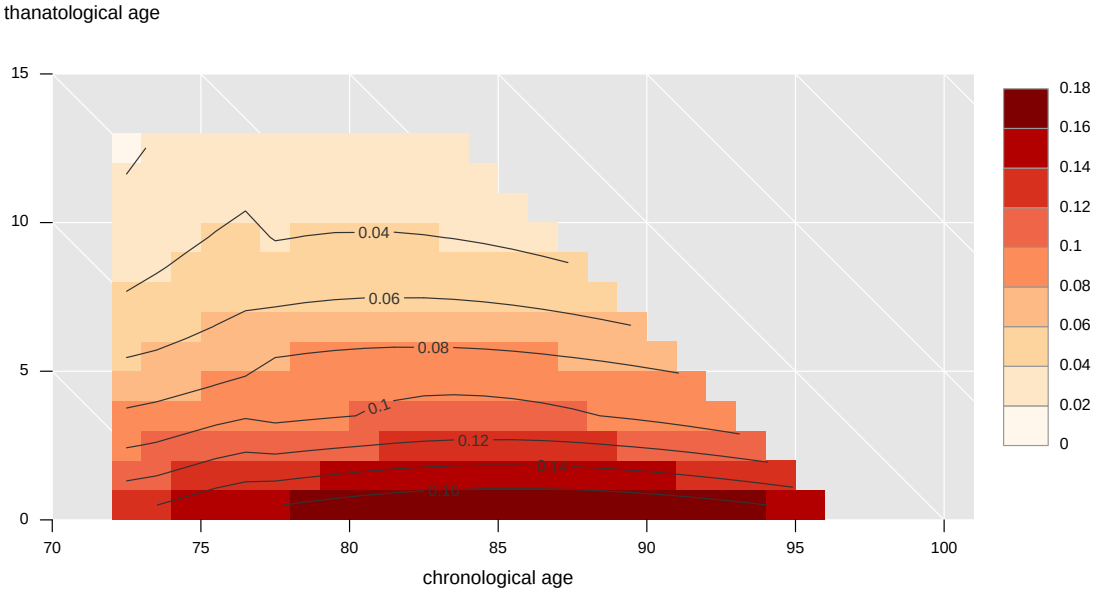
Other informative patterns also exist among the set of characteristics studied. These include characteristics that vary by lifespan, which display downward diagonal contours in surface plots. Characteristics that vary by lifespan appear constant within lifespans. These are often characteristics that *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, eventually (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 two years (females), and diabetes among females. Sometimes such patterns combine in complex ways worthy of further study.

The fourth major pattern 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 for this window of study. In other words, longer lifespans display later onset but greater eventual odds of having been diagnosed with high blood pressure. Arithmetically, *chronological age* − *thanatological age* 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.[9]

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

Figure 2: 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.

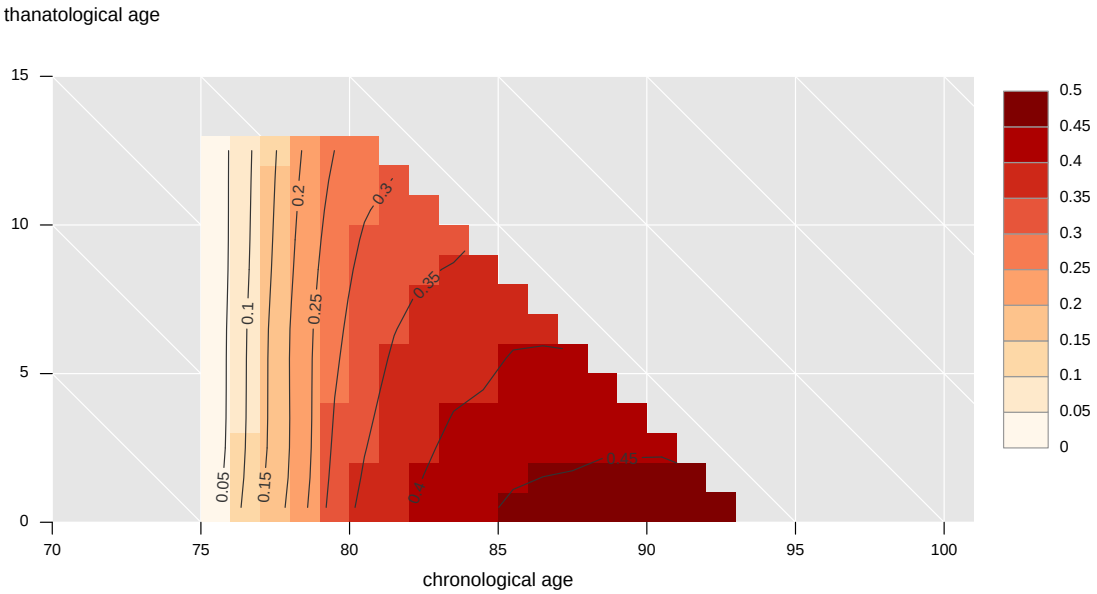


Figure 3: Psychological problems (ever) by chronological age only. Males, 1915-1919 birth cohort. With 95% confidence bands from loess fit.

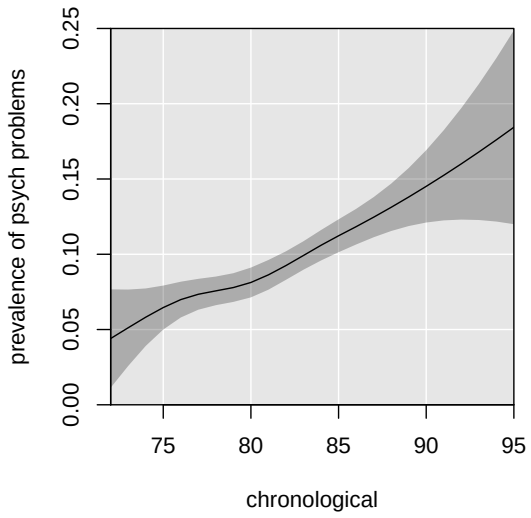
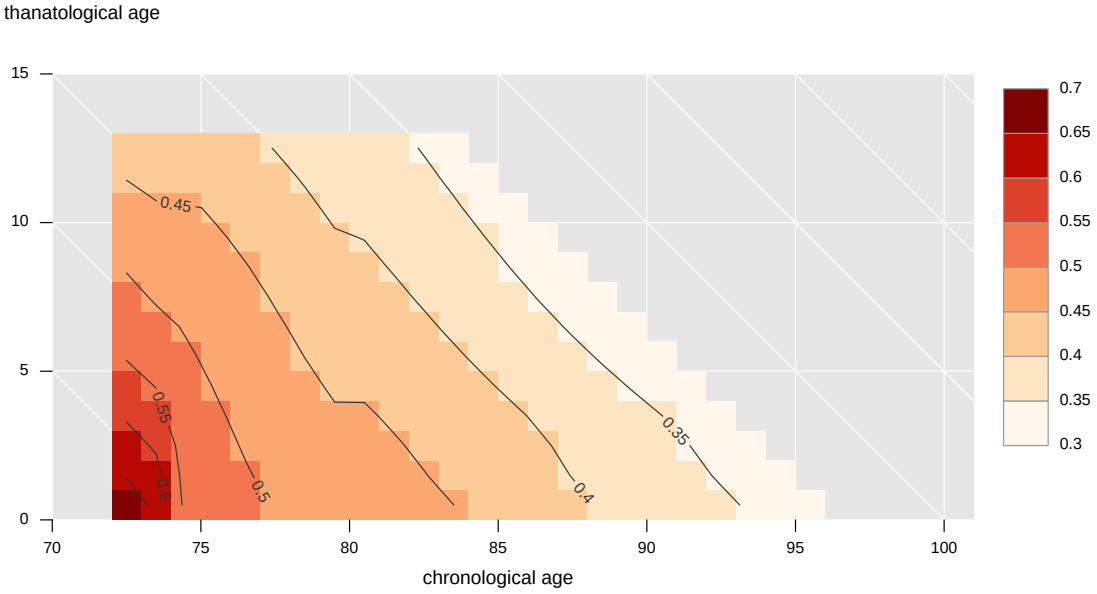
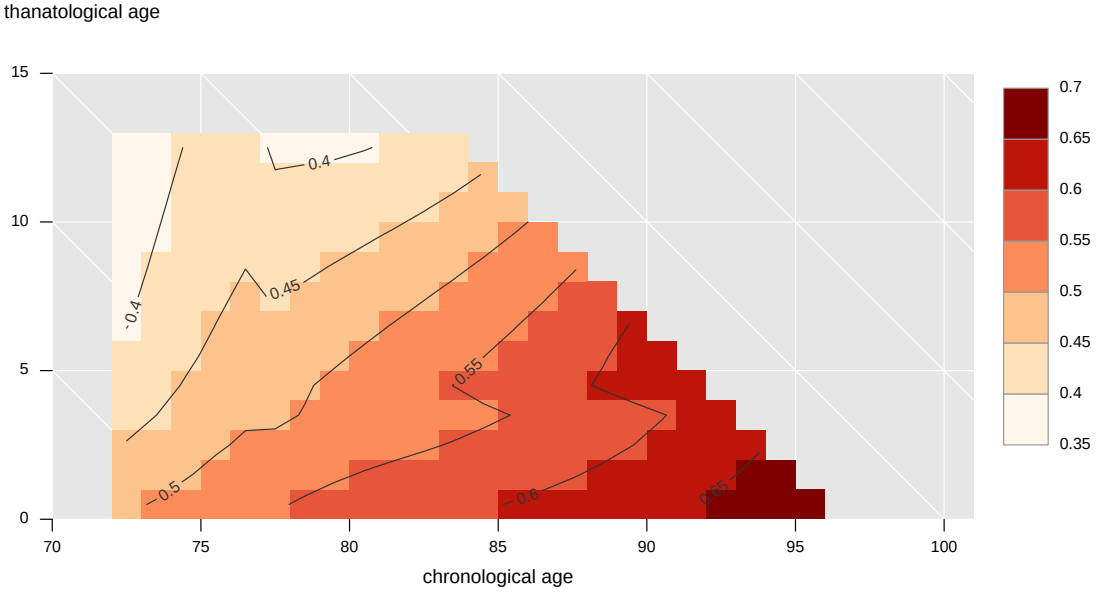


Figure 4: Examples of characteristics that vary by lifespan only or by thanatological age within lifespan.

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



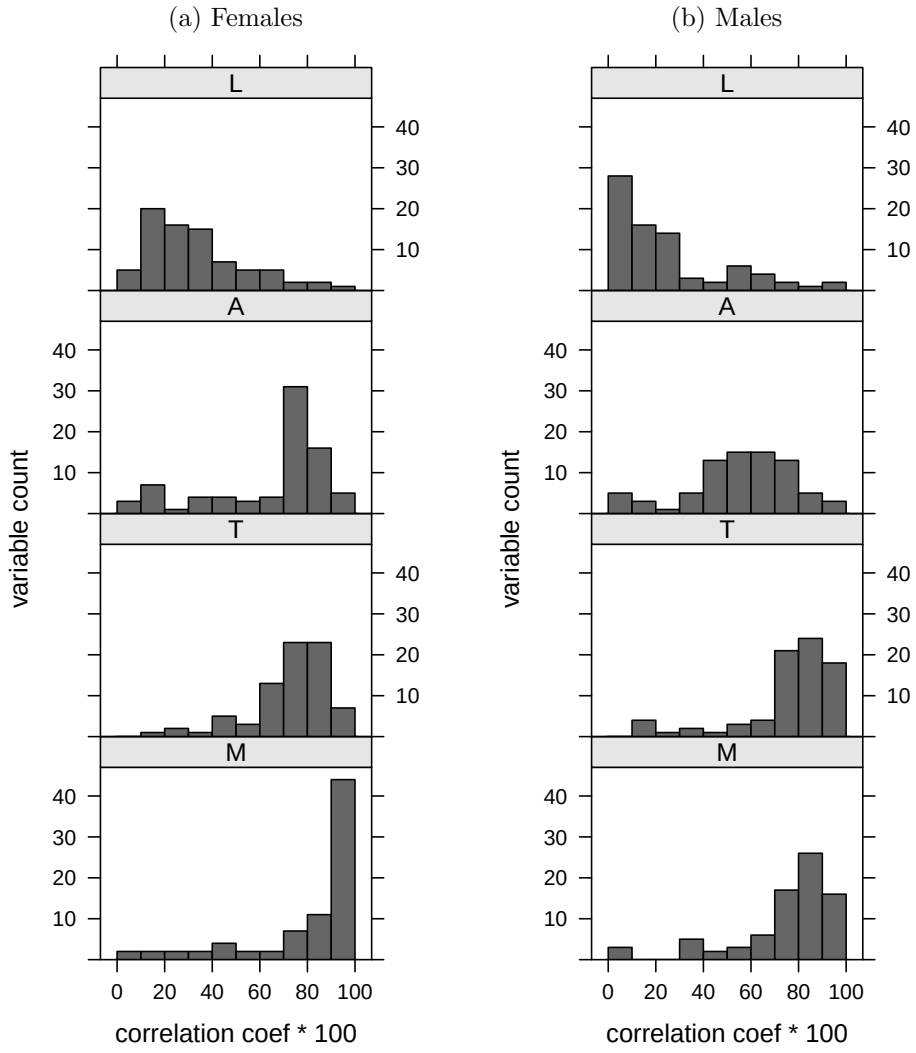
(b) Blood Pressure by years lived (x axis) and years left (y axis). Males, 1915-1919 birth cohort.



Summary of results for all characteristics We produce surfaces such as those in Figures 2 and 4 for all 78 variables and each sex. We distill each of these surfaces into four Pearson correlation coefficients, each designed to capture the variation along each of the four major patterns explained above. We call the four patterns thanatological age (T), chronological age (A), lifespan $(A + T)$ (L), and mixed $(A - T)$ (M). Most characteristics are well-summarized by either one or two of these patterns. Figure 5 shows the correlation coefficients of all 78 variables binned into count histograms for each sex and major variation pattern separately. This view is meant to give a feel for how common each major pattern of variation might be in commonly measured characteristics. This statistic only captures the rough direction of variation in characteristics, and it does not capture differences in levels or gradient steepness.

The first row of this panel shows that variation by lifespan is weak for most variables, and strong for only a few. The second row shows that chronological age is indeed an important aspect of variation for many characteristics, but not all characteristics, and chronological variation is more often strong for females than for males. The third row shows that thanatological age is an important pattern of variation for many variables: the lower tail is thinner than that of chronological age, and there are more cases of strong correlations ($r > 0.80$) in the direction of thanatological variation than of chronological variation. In the distributions over these variables, males tend to more commonly show stronger thanatological age patterns than females, and females tend to show stronger chronological age patterns than males. Finally, the most common pattern in in these data are for characteristics to vary strongly as chronological age increases *and* as thanatological age decreases (M). For females, this is very clearly the dominant pattern among the variables studied. For males, the pattern of variation between characteristics is similar to that of thanatological age. In most cases, for variables with strong patterns of variation in the M direction, there are also strong correlations in the A and/or T directions. Of these, M is most commonly paired with T. Characteristics that show strong correlation in both M and T display surfaces with contour lines slanted less than 45°. A more detailed table of correlation results by variable, pattern, and sex is given in the appendix.

Figure 5: Major patterns of variation, all 78 variables examined



Discussion

The distribution of tested characteristics with respect to the four primary patterns of variation is striking. Chronological age describes prevalence patterns for many conditions well, but time-to-death patterns are more prevalent among the measures tested. For measures that vary both with the increase of age and the approach to death, the approach to death is more often the stronger of the two measures. Characteristics that vary by length of life are few, but their patterns are clear. The upshot, as illustrated by comparing figures 2a and 3, is that representing morbidity or disability variables as chronological age patterns can in many or most cases be misleading as model of morbidity processes, and biased as a basis for prediction.

These empirical findings must be tempered by noting that 1) the summary measure (correlation coefficient) used here blends out some information, 2) these results may not 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. Comparable results for other five-year birth cohorts in the HRS (1905-1925) are given in the manuscript repository.

Further, the patterns presented here are valid for the whole population (of a given sex) taken together, but were the target population broken down by causes of death (for instance), the patterns may change. For example, imagine hypothetically that the strong thanatological pattern shown in Figure 2 (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, both for purposes of understanding and for cause and time of death prediction.

Research to better document the multidimensional age variation of particular characteristics would benefit from more careful measurement and modeling than that conducted here. Despite such shortcomings, the principal 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 aging, 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 aging ought to be better informed by more judicious measurement and description in terms of thanatological and chronological age. This would benefit scientific understanding of health and disability processes, and it would improve the actuarial accuracy of morbidity projections and any policies that count on accurate morbidity projections.

That accounting for time-to-death in predictions of healthcare expenditure reduces bias has already been established in the health economics literature (e.g., Stearns and Norton 2004). A common finding on healthcare expenditure prediction is that in times of mortality improvements, predictions based on chronological age patterns of healthcare expenditure (Sullivan-style predictions (Sullivan 1971)) tend to overestimate total expenditure (e.g., Geue et al. 2014). Since the patterns of variation among the morbidity dimensions we study are similar to those of healthcare expenditure over chronological age and time-to-death, we here infer that Sullivan-style predictions of morbidity are biased in the same direction.[10] The consequences of overestimating future morbidity prevalence are complex and varied, ranging from budget misallocations to lowered expectations on the benefits of lengthening life.

We hope that the conceptual model of the lifecourse presented here, which complements the Lexis diagram, will be of use to demographers, public health researchers, and epidemiologists. Other combinations of lifespan time dimensions are also possible, and these would highlight different patterns in data (Riffe et al. 2015). 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 precise toolkit for demographic measurement and the practice of demography, and ultimately a wiser contribution to the discourse on population aging.

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

[10] Other work in progress treats this point in greater detail (van Raalte and Riffe 2016).

(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 aging 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, the surfaces underlying this study highlight 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. Also 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. `A` indicates the chronological age axis. `L` indicates the lifespan axis (right-downward slanting isolines), the least common in these data. `M` indicates the mixed axis, the most common type in these data. The code used to generate these and all other results, including results for all 5-year cohorts from 1905-1925 and different degrees of smoothing, is available freely the repository. The repository also contains a csv of these summary results.

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

Results are grouped by several major morbidity categories and presented in heatmap tables. In these tables, darker shades of grey indicate higher correlations (black = 1), and lighter grays indicate low correlations (white = 0). Numbers inside the cells indicate the rounded Pearson's correlation coefficient $\times$ 100, and can be interpreted as percents.

Finally, it bears noting that these values say nothing of prevalence levels. They are only intended to be rough gauges of the direction of variation in characteristics.

List of Appendix Tables

Table 1: Activities of Daily Living (ADL)

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
ADL3	ADL 3 point	25	80	80	96	9	67	84	89
ADL5	ADL 5 point	23	79	81	97	5	65	87	89
WALK	Difficulty walking across room	16	73	83	93	6	53	86	80
DRESS	Difficulty dressing	18	75	82	94	8	66	85	89
BATH	Difficulty bathing or showering	17	73	81	94	7	59	83	82
EAT	Difficuty eating	19	70	72	91	15	65	79	85
BED	Difficuty getting in/out bed	14	71	82	93	8	59	80	80
TOILET	Difficulty using toilet	31	81	73	94	0	51	81	78

Table 2: Instrumental Activities of Daily Living (IADL)

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
IADL3	IADL 3 point	28	82	78	97	6	66	88	91
IADL5	IADL 5 point	14	74	87	96	7	68	89	92
WORK	Health limits work	25	36	98	73	6	53	93	84
MAP	Difficulty using maps	24	77	78	94	13	67	80	88
TEL	Difficulty using telephone	33	83	68	96	20	75	78	95
MONEY	Difficulty managing money	21	76	81	95	1	56	90	84
MEDS	Difficulty taking medications	24	75	77	95	3	45	94	73
SHOP	Difficulty grocery shopping	2	65	91	91	8	54	91	84
MEALS	Difficulty prep. hot meals	20	76	82	95	6	60	88	85

Table 3: Health behaviors

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
ALCEV	Alcohol, ever-drinker	40	79	62	88	8	41	78	68
ALCDAYS	Drinking days / week	10	48	77	72	18	40	77	67
ALCDRINKS	Nr drinks per drinking day	28	84	75	96	18	49	89	80
SMOKEEV	Ever-smoker	98	81	27	48	87	68	30	37
SMOKECUR	Current-smoker	83	93	16	77	91	86	10	54

Table 4: Functional limitations

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
BMI	Body mass index	34	79	72	93	4	54	91	83
BACK	Back problems	56	91	43	82	79	92	17	74
MOB	Mobility difficulty index	16	76	86	97	1	64	92	92
LGMUS	Large muscle difficulty index	32	85	77	99	11	72	88	95
GROSSMOT	Gross motor difficulty index	10	71	88	94	5	65	87	89
FINEMOT	Fine motor difficulty index	22	78	81	96	14	70	81	90

Table 5: Chronic conditions

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
CC	Number of chronic conditions	34	82	77	98	7	53	95	84
BP	High blood pressure, ever	14	67	84	89	37	84	75	98
DIAB	Diabetes, ever	72	22	80	21	69	28	65	10
CANCER	Cancer, ever	29	31	96	68	17	41	93	75
LUNG	Lung disease	62	7	88	36	90	50	65	7
HEART	Heart problems, ever	26	78	82	97	23	37	96	73
STROKE	Stroke, ever	46	90	69	99	9	51	95	82
PSYCH	Psychological problems , ever	33	77	69	88	24	37	96	72
ARTH	Arthritis, ever	75	92	28	82	69	91	33	84

Table 6: Cognitive function

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
SRM	Self-rated memory	51	92	65	99	60	70	16	60
PASTMEM	Memory compared to past	61	87	41	85	71	94	36	87
SS	Serial 7s	1	64	92	91	7	48	60	65
C20B	Backwards counting	35	81	66	90	30	79	72	93
NAMEMO	Naming month	33	80	67	90	2	49	72	70
NAMEDMO	Naming day of month	24	78	78	94	21	75	78	92
NAMEYR	Naming year	44	88	64	95	19	74	80	93
NAMEDWK	Naming day of week	16	72	80	91	20	70	73	86
NAMESCI	Naming scissors	50	87	53	88	12	42	78	69
NAMECAC	Naming cactus	39	86	68	95	56	86	45	84
NAMEPRES	Naming president	17	74	82	93	59	3	81	37
NAMEVP	Naming vice president	1	52	74	74	4	58	79	81
VOCAB	Vocabulary score	40	10	67	42	51	13	85	53
TM	Mental status summary	19	76	83	96	10	66	81	87
DWR	Delayed word recall	4	59	87	85	19	71	82	92
TWR	Total word recall	19	71	82	92	27	76	77	93
IWR	Delayed word recall	33	80	76	96	35	80	71	93

Table 7: Psychological wellbeing

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
CESD	Depression score	44	19	91	58	22	43	95	78
SRH	Self-reported health	42	14	90	53	29	33	98	70
DEPR	Felt depressed	55	19	58	13	58	4	86	38
SLEEP	Sleep restless	45	4	65	28	55	3	91	45
HAPPY	Was happy	33	15	76	47	15	60	72	78
LONE	Felt lonely	32	64	50	71	7	64	90	90
SAD	Felt sad	69	39	47	7	22	35	91	69
GOING	Could not get going	70	15	87	30	22	36	92	70
ENJOY	Enjoyed life	13	40	85	70	42	85	67	95

Table 8: Healthcare use (24 months)

Short	Description	Females				Males			
		L	A	T	M	L	A	T	M
HOSP	Overnight hospital	26	73	75	90	11	60	77	81
HOSPSTAYS	Number hospital stays	5	57	80	83	4	50	86	78
HOSP-NIGHTS	Number nights in hospitals	10	40	77	70	61	6	87	36
NH	Overnight stay in nursing home	25	75	67	94	13	64	78	82
NHSTAYS	Nursing home stays	26	76	67	94	10	57	77	78
NHNIGHTS	Number nights in nursing homes	18	70	70	89	13	61	80	80
NHNOW	Nursing home at interview	14	72	71	93	8	46	80	73
DOC	Visited doctor	63	89	40	85	52	85	52	88
DOCVISITS	Number of doctor visits	54	91	58	95	33	70	56	79
HHC	Home health care	18	71	84	94	2	52	90	84
MEDS	Prescription drugs regularly	22	40	90	73	23	41	92	75
SURG	Outpatient surgery	32	11	31	7	30	17	18	3
DENT	Visited dentist	84	33	75	14	27	11	55	35
SHF	Visited special healthcare facility	35	87	75	99	12	71	87	94