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

Tim Riffe, Pil H. Chung, Jeroen Spijker and John MacInnes*

Abstract

We aim to determine the extent to which variables commonly used to describe health, well-being, 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 U.S. Health and Retirement Study to estimate chronological age and time-to-death patterns in 78 such variables. We describe results for the birth cohort 1915–1919 in the final 12 years of life. Our results show that most of the markers used to study well-being in old age vary along both the age and the time-to-death dimensions, but that some markers are exclusively a function of either time to death or chronological age, while other markers display different patterns in men and women.

1 Background

For an individual, age across the life course consists of two components: time since birth, or the *chronological* dimension of age; and time to death, or the *thanatological* dimension of age. In the aggregate, thanatological age is determined 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 estimate this age, an expectation of

* Tim Riffe (corresponding author), Max Planck Institute for Demographic Research, Rostock, Germany
Email: riffe@demogr.mpg.de

Pil H. Chung, Department of Demography, University of California, Berkeley, U.S.A.

Jeroen Spijker, Centre d'Estudis Demogràfics, Barcelona, Spain

John MacInnes, School of Social and Political Science, University of Edinburgh, U.K.

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the lifespan[1] is projected based on scenarios or extrapolations of how mortality rates might change over time. Using this approach, data classified by chronological age, like census population counts, can be reclassified into thanatological age (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 the measure of future remaining lifespan is elastic, and is thus 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] it may be tempting to assume that because the chronological and the thanatological age structures are symmetrical in stationary populations (Brouard 1989, Vaupel 2009, Villavicencio and Riffe 2016), the patterns of demographic characteristics within cohorts might demonstrate an analogous form 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, it can be confirmed that for each cohort, the mean duration spent in each state is indeed identical, regardless of whether age is measured chronologically or thanatologically. The cohort expectancies are thus immune to 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 in which chronological age captures almost all pertinent variation. In cases in which 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. The measurement of the end-of-life trajectories of characteristics is useful in such cases as a way of separating mortality patterns from patterns in the characteristics themselves. Characteristic measurements are taken while the respondent is alive, but as the thanatological age at each observation is unknown until the date of death is known, it is retrospectively

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

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

assigned. This final analytical step lends clarity to our 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 the thanatological rather than the chronological age structure, forecasts of the social and economic "costs" of aging that are based on chronological age profiles only are prone to bias (Stearns and Norton 2004).

Research exploring time-to-death patterns has been done in other domains, and the topics examined can be roughly categorized into two types: (1) phenomena that are a function of apparent or perceived time to death (Hamermesh 1985, Hurd and McGarry 1995, Carstensen 2006, Gan et al. 2004, Bìrò 2010, Salm 2010, van Solinge and Henkens 2010, Cocco and Gomes 2012, Payne et al. 2013, Balia 2013), and (2) phenomena 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). Research in the first category consists mainly of studies on cognitive transitions and economic or health behaviors, while research in the second category consists mainly of studies on health expenditure, except Wolf et al. (2015), who proposed a model to separate latent time-to-death trajectories of disability. A third branch of research relates the perceived and actual remaining life time (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 10 waves of the U.S. Health and Retirement Study (RAND 2013, 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 this end, 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 pattern of variation exhibited by a given characteristic ought to determine how we measure, understand, and respond to the characteristic. We show that while in many cases chronological age ought to be used in conjunction with thanatological age in classifying patterns, chronological age often provides no information at all, and it may even obfuscate true temporal patterns.

Our analytical approach is retrospective rather than prospective, meaning that no life table 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. In the following section, we describe the methods in greater detail. We then demonstrate the four primary age patterns by way of example, and summarize all of the characteristics tested in terms of these four patterns. Finally, we discuss some implications and applications of this work.

2 Data and methods

All of the findings reported in this paper are based on data from the U.S. Health and Retirement Study (HRS).[3] We use version M of the RAND edition of the data, which is conveniently merged across all 10 waves available as of 2013. These data are free to download, and all of the details of data processing and methods are made freely available in an open code repository.[4]

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

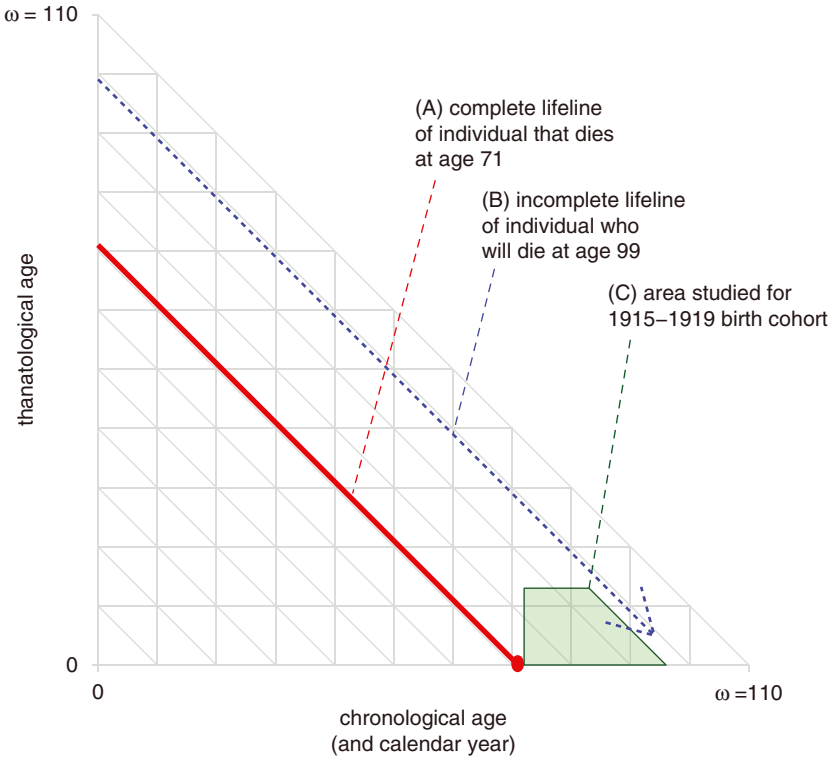
Underpinning this investigation are a series of demographic surfaces indicating the average prevalence of a given marker along chronological and thanatological time axes within a series of quinquennial birth cohorts. However, we focus only on the central 1915–1919 birth cohort. This visual tool is similar to but orthogonal to the familiar Lexis surface. Figure 1 orients the reader by providing 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, whereby 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 (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. In the HRS, enough observations are available from the 1915–1919 cohort to allow us to measure the patterns 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 version used spans 20 calendar years (1992–2011), the theoretical upper bound of observation of thanatological age is 20. However, because relatively few individuals in this sample are between thanatological ages 13 and 20 (i.e., individuals who entered the study

[3] The Health and Retirement Study is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and is conducted by the University of Michigan.

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

Figure 1:
Chronological age and thanatological age over the life course of a birth cohort



around 1992 and also died around 2011), we study only the thanatological ages that are less than or equal to 12; ergo, the final 12 years of life. As further waves are added to the HRS and the mortality linkage continues, the portion of the life course 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 cohorts), and in-utero (1919 cohort). As there is evidence that this exposure manifested itself in various ways in later life (e.g., Almond 2006, Myrskylä et al. 2013), the reader may rightly question whether the results presented here are anomalous. However, the potential anomalous effects from this cohort are "smoothed-out" in our analysis, due both to the breadth 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. Furthermore, we assume that at these ages other risk factors – some of which are cumulative over the life course – and senescence itself likely drive health patterns to

a much greater extent than early-life selection or late-life onsets of poor health due to the Spanish influenza. We also verified that the patterns for this cohort are not 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 to contribute to the practice of demography in general.

Age Thanatological age is calculated for each individual as the lag between the interview and the death dates expressed as decimal years. Chronological age is calculated as the lag between the birth and the interview dates 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 achieves 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 only a few observations at the upper extreme. Results for 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 at 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 the observation window for this cohort is centered on the chronological ages at which most deaths occur, and at which most recent mortality improvements in low-mortality countries have occurred;[5] and because the HRS provides a good density and spread of data points over this window. The lower and upper age bounds vary for questions 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 well-being. For this reason, we have selected a wide variety of questions from the HRS data. These questions can be roughly grouped 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.

[5] 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).

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 well-being: nine items and one composite index.
8. Health care use: 14 items.

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 wave 9. Variables that were not available in the first or second wave have left age bounds at ages higher than 72, whereas items that were not asked in wave 10 have upper lifespan bounds that are below 95.

Each survey question must be in a format suitable for numeric operations. This approach entails 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 cut points. For example, respondents were asked if they felt depressed. We assigned a value of zero to "no" answers and a value of one to "yes" answers. As an example of ordinate recoding, self-reported health had the possible responses "excellent," "very good," "good," "fair," and "poor;" to which we assigned values of zero, zero, zero, one, and one, respectively. Thus, for this kind of variable, population means can be interpreted as prevalences.

Variables with compact or bounded numeric responses were rescaled to range from zero to one. 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 for the purposes of this study. In other words, the surfaces we present are not exact measurements, but are meant to provide *impressions* of how characteristics change over age.[6]

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 that arises when using 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 weight in a previous wave, we carry

[6] The pre-processing of variables is full of details that would clutter this paper. Rather than providing a lengthy and detailed appendix describing the case-by-case treatment of variables, we refer readers to the annotated code in the open repository.

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.[7]

Loess smoothing Direct tabulations of the weighted data are legible if all of the birth cohorts are combined, but doing this distorts the results due to cohort composition bias. To overcome the birth cohort heterogeneity within surfaces, we use birth cohorts as a third time dimension. As tabulations within this three-dimensional space are noisy, 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 of the 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)[8] 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.[9]

3 Results

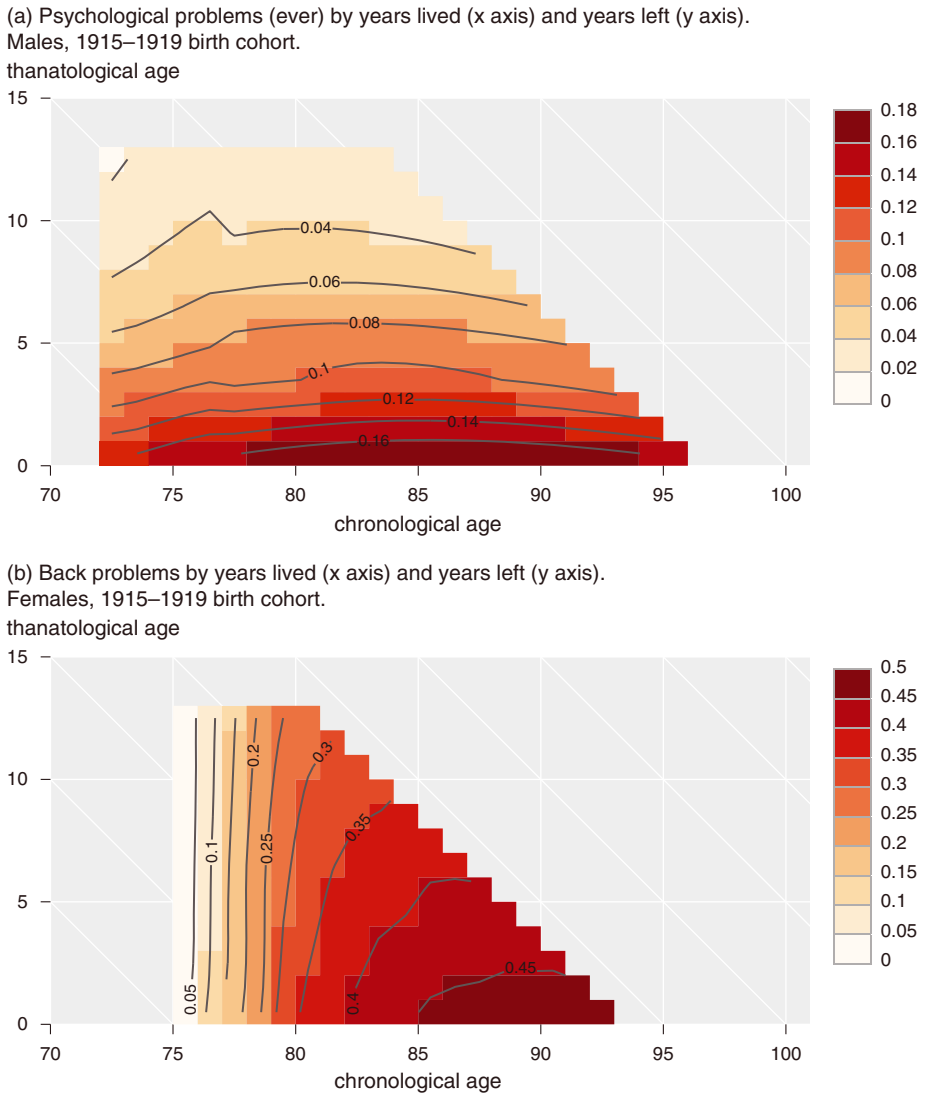
We first present examples of four surfaces that exemplify the major ways in which characteristics tend to vary temporally over the lifespan within a birth cohort. These four major patterns of variation provide a way to categorize and understand markers

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

[8] 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 of the results were also produced using smoothing parameters of .5 and .9, and we concluded that the specific choice of smoothness does not drive results. In order to preserve year units, the three predictor dimensions are not normalized.

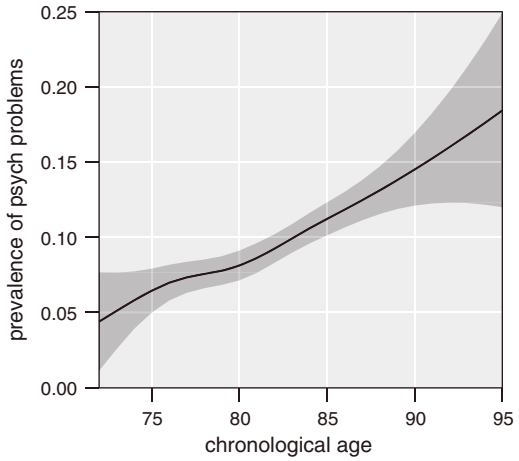
[9] Note that smoothing over these three particular time dimensions is not an overidentification. Within a cohort, smoothing over thanatological age, chronological age, and completed lifespan would be an overidentification, a problem that is similar to the familiar APC problem. The full set of lifespan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

Figure 2:
Examples of characteristics that vary along the thanatological and chronological age axes



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 the results graphically, as well as in an appendix shaded table.

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

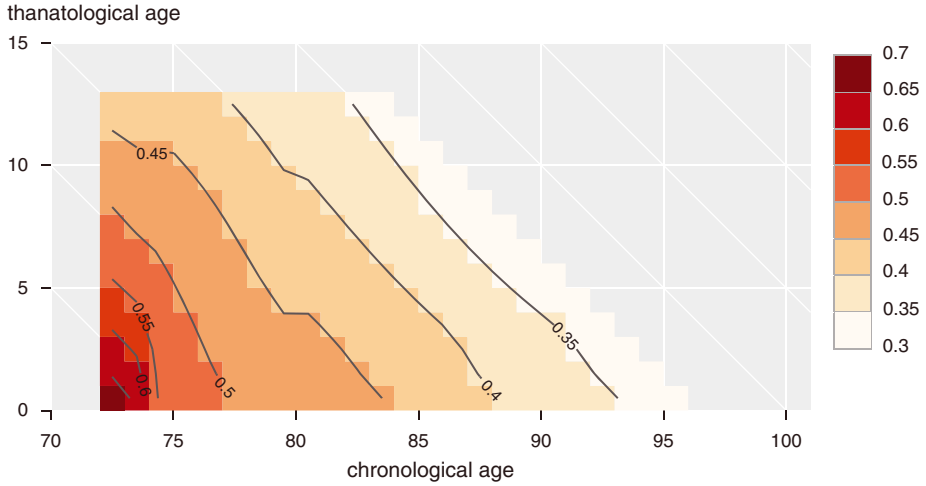


Four major surface axes In most situations, it is obvious to the eye whether a variable operates over thanatological age or over chronological age. There are, however, many instances in which both are at play, or the relationship is complex. We first present surfaces representing psychological problems for men (Figure 2(a)) and back pain for women (Figure 2(b)). These two surfaces are examples of thanatological and chronological characteristics, respectively.

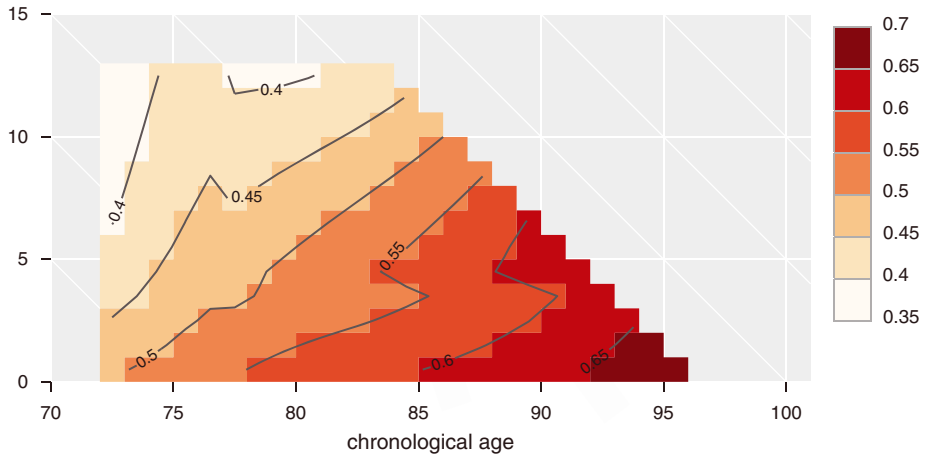
From the direction of the contours on the surface in Figure 2(a), we conclude that the chances of ever having been diagnosed with psychological problems increases with the approach to death, and not with the advancing of chronological age, at least in the window of observation studied here. However, since the risk of death itself also increases according to an approximate exponential pattern at 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 2(a) 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 operate primarily as effects of the body shutting down, or possibly as a signal that, on average, death is not far off. Thus, these characteristics represent 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

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



highly thanatological. Some of the characteristics studied here display patterns that are strongly thanatological.

Figure 2(b) tells the opposite story about back pain for women. 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. At 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 (for both men and women).

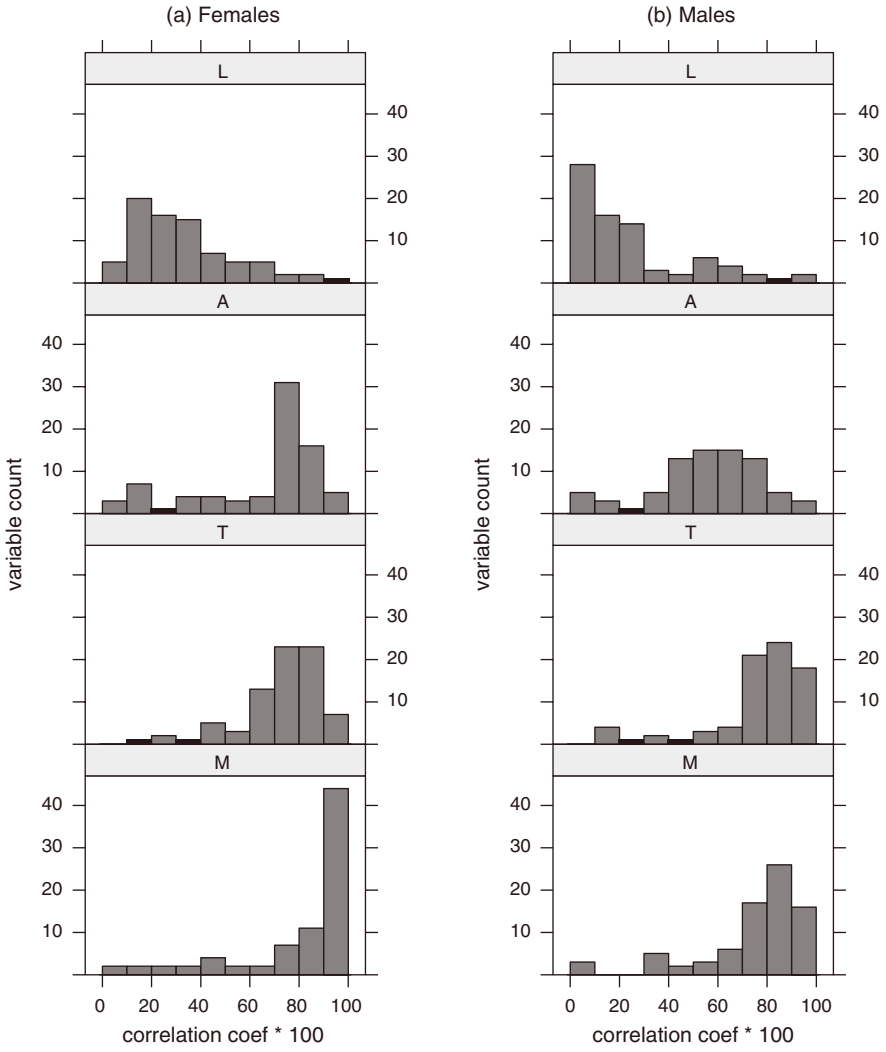
There are other informative patterns among the set of characteristics studied. These include characteristics that vary by lifespan, which display downward diagonal contours in surface plots. Characteristics that vary by lifespan appear to be constant within lifespans, and are often characteristics that *determine* lifespan. Having ever smoked displays such a pattern, as seen in Figure 4(a) for women 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 men (this is largely redundant with the former), dental visits in the previous two years among women, and diabetes among women. Sometimes such patterns combine in complex ways that are worthy of further study.

The fourth major pattern of contour variation runs perpendicular to lifelines. One characteristic that clearly displays this pattern is having ever been diagnosed with high blood pressure among men. This characteristic varies by lifespan, and by thanatological age within the lifespan for this window of study. In other words, individuals with 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. On their own, such characteristics are not very useful for predicting eventual lifespan.[10] Some characteristics appear to follow this pattern, albeit with contour lines at angles less than 45°, which may suggest that thanatological morbidity prevalence is somehow *proportional* to length of life. We do not measure this possibility explicitly.

Summary of the 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 of which is 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 of the 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 provide an impression of how common each major pattern of variation might be in commonly measured characteristics. This statistic

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

Figure 5:
Distribution of correlation coefficients for each of the four major patterns of variation, all 78 variables examined. L indicates lifespan variation (like Figure 4(a)), A indicates chronological age (like Figure 2(b)), T indicates thanatological age (like Figure 2(a)), and M indicates the mixed type variation (like Figure 4(b))



only captures the rough direction of variation in characteristics, and 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 variables (having ever smoked, and having

visited a dentist for women). The second row shows that chronological age is indeed an important aspect of variation for many, but not all characteristics (e.g., having ever been diagnosed with psychological problems); and that chronological variation is often stronger among women than among men. The third row shows that thanatological age is an important pattern of variation for many variables: the lower tail is thinner than the tail 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, men tend to show stronger thanatological age patterns than women, and women tend to show stronger chronological age patterns than men. Finally, the most common pattern in these data are for characteristics to vary strongly as chronological age increases *and* as thanatological age decreases, M (especially for many ADLs, IADLs, functional limitations, and many variables of cognitive function). For women, this is very clearly the dominant pattern among the variables studied. For men, 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 correlations in both M and T display surfaces with contour lines slanted less than 45°. A more detailed table of the correlation results by variable, pattern, and sex is given in the appendix.

4 Discussion

The distribution of the tested characteristics with respect to the four primary patterns of variation is striking. Chronological age describes the prevalence patterns for many conditions quite well, but the time-to-death patterns are more prevalent among the measures tested. For measures that vary both with the increase in age and the approach to death, the approach to death tends to be the stronger of the two measures. Only a few characteristics vary by length of life, and their patterns are clear. The upshot, as illustrated by comparing Figures 2(a) and 3, is that representing morbidity or disability variables as chronological age patterns can in many or in most cases be misleading as a model of morbidity prevalence, and be 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 for other birth cohorts. Comparable results for other five-year birth cohorts in the HRS (1905–1925) are given in the manuscript repository.

Furthermore, the patterns presented here are valid for the whole population (of a given sex) taken together, but if the target population was, for instance, broken down by causes of death, the patterns may change. For example, imagine hypothetically that the strong thanatological patterns shown in Figure 2 (psychological problems)

were driven by strong patterns within individuals who eventually died 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 follow-up. For detailed investigations of particular characteristics, cause-conditioning surfaces would clearly be useful in disentangling morbidity processes, both for the purposes of understanding these processes, and for making cause- and time-of-death predictions.

Research seeking to better document the multidimensional age variation of particular characteristics would benefit from more empirical evidence and further model development. Despite the limitations of this study, we have been able to demonstrate 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 that were untested here are also shown to display similar temporal complexity, we submit that the common discourse and debate on the nature and impacts of aging would be better informed through the inclusion of more judicious measurements and descriptions framed in terms of thanatological as well as chronological age. This approach would contribute to the scientific understanding of health and disability processes, and would improve the actuarial accuracy of morbidity projections and of any policies that rely on accurate morbidity projections.

The claim that accounting for time-to-death in predictions of health care expenditure reduces bias has already been established in the health economics literature (e.g., Stearns and Norton 2004). A common finding in health care expenditure predictions is that in times of mortality improvements, predictions based on chronological age patterns of health care 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 health care expenditure over chronological age and time-to-death, we here infer that Sullivan-style predictions of morbidity are biased in the same direction.[11] The consequences of overestimating future morbidity prevalence are complex and varied, ranging from budget misallocations, to poor design of social health care systems for the elderly, to lowered expectations regarding the benefits of lengthening life.

We hope that the conceptual model of the life course 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 the data (Riffe et al. 2017). Given the variety and the availability of such options – which

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

are perhaps now placed in starker relief – a more nuanced understanding of the temporal accounting that relates demographic time perspectives is needed. Further exploration and experimentation with these formal demographic concepts will lead to the development of a more precise toolkit for demographic measurement and the practice of demography, and, ultimately, to more astute contributions to the discourse on population aging.

We suggest a selection of extensions to the exploration carried out here. The present type of analysis must be replicated for more cohorts and populations. A few countries with long-running and fully linked population registers already preside over such information, and we encourage using such data to engage in a more thorough exploration of the temporal richness of population change and population characteristics. The administrators of large-scale panel studies may be motivated to implement, increase, or improve the quality of 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 retrospectively linked to death registers.

If compared over calendar time, 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 2(a)), it is safe to say that unless retrospective thanatological measurements of morbidity dimensions are undertaken, we will not have direct information about the shape of the morbidity burden in the final years of life. Using the techniques shown here, researchers may directly estimate the varieties of end-of-life profiles often seen in the literature on morbidity compression (e.g., Fries et al. 2011).

There are also consequences for the popular understanding of aging. By using analyses oriented toward the life course diagram, health care providers can better situate the association of certain health outcomes within stages of the aging process. This is both a question of the allocation of resources and a question of how individuals conceive of themselves with respect to age. We therefore 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).

The life course surfaces underlying this study highlight important sex differences in the aggregate onset and the trajectory of some aspects of morbidity. Some of these results may corroborate extant findings, such as results on the male-female health-survival paradox, while others may provide us with a new understanding of sexual dimorphism in morbidity. Specifically, it has been shown that women live longer, but in worse health than men (e.g., Case and Paxson 2005), and that this pattern is consistent with evidence indicating that health patterns vary chronologically more among females than among males. In general, these methods and measurements can be used to describe any between-group disparity in demographic or social outcomes, especially those that directly or indirectly relate to remaining years of life. Numerous other avenues of potential investigation may also be devised from

the present work. It is our hope that these results are strongly suggestive, and help to orient future investigation.

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Appendix: Variables and correlations

For the 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), which is the least common in these data. M indicates the mixed axis, or the upward-right slanting isolines, which is the most common type in these data. The code used to generate these and all other results, including the results for all five-year cohorts from 1905–1925 and different degrees of smoothing, is freely available from 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 gray indicate higher correlations (black = 1), and lighter shades of gray indicate low correlations (white = 0). Numbers inside the cells indicate the rounded Pearson's correlation coefficient × 100, and can be interpreted as percents.

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

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Table A.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	Difficulty eating	19	70	72	91	15	65	79	85
BED	Difficulty 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 A.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 A.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 A.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 A.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 A.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 A.7:
Psychological well-being

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 A.8:
Health care 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
HOSPNIGHTS	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