

**Full title: Living longer but not necessarily healthier: The joint progress of health and mortality in the working age population of England**

**Short title: Living longer but not necessarily healthier**

**Disclosure statement**

None

**Data availability statement**

Health Survey for England data are available from the UK Data Service  
<https://www.ukdataservice.ac.uk/>

## Abstract

Despite improvements in life expectancy, there is uncertainty on whether increase in years of healthy life expectancy has kept pace. In this paper we explore whether there is empirical support for the compression of morbidity hypotheses in the population aged 25 to 64 living in England. Synthetic cohorts born between 1945 and 1980 are constructed from repeated annual cross-sections of the Health Survey for England, 1991-2014. Younger cohorts at a given age have poorer or the same prevalence of self-reported poor general health, self-reported high blood pressure (in men), diabetes and circulatory illnesses, clinical hypertension and diabetes, and an overweight BMI. We also find that healthy life expectancy at age 25 has increased at a slower pace compared to life expectancy between 1993 and 2013. Our findings lend support to the expansion of morbidity hypothesis and point to an increased future demand for certain healthcare services at younger ages.

**Keywords:** expansion of morbidity, Health Survey for England, population health, healthy life expectancy, Sullivan's method.

## Introduction

The 20th century witnessed significant improvements in health in most countries, including substantial increases in survival to older ages. Life expectancy at birth in the United Kingdom increased by more than half, and in other high income countries it almost doubled (Leon, 2011; Mackenbach & Looman, 2013), and according to recent projections will continue to increase (Kontis et al., 2017). The rapid increase in life expectancy across generations has raised the question of how healthily the gained years of life will be spent.

The evidence from a number of developed countries suggests that on the one hand the presence of leading risk factors of mortality, such as coronary heart disease and clinical hypertension (Rice, Lang, Henley, & Melzer, 2010), and harmful health-related behaviours such as smoking (O.N.S, 2013) are less common in those born after 1945 than those born earlier. This may in part explain why later born cohorts have gained years in life expectancy (O.N.S, 2013), because the prevalence of life threatening disease is lower. On the other hand, a growing, but piecemeal, literature suggests that in other dimensions of health, those born since 1945 have progressively poorer health at the same age. This has been shown for subjective measures of health, such as self-rated health (Idler, 1993; Jlvraj 2020) and disability (Soldo, Mitchell, Tfaily, & McCabe, 2007), self-reported health conditions, including diabetes (Reynolds, Crimmins, & Saito, 1998; Rice et al., 2010), diagnosed high blood pressure (Rice et al., 2010), and mental health, (Ploubidis, Sullivan, Brown, & Goodman, 2017; Rice et al., 2010; Spiers et al., 2011) and other risk factors of poor health, such as obesity (Andrade, Lebrao, Santos, & Duarte, 2011; Cabrera et al., 2003; Cámara & Spijker, 2010; Johnson, Li, Kuh,

& Hardy, 2015; Reynolds & Himes, 2007; Rice et al., 2010; Shaw, Green, Popham, & Benzeval, 2014).

The possibility of a greater prevalence among successive generations of non-fatal disease reflected in poor subjective health as well as chronic conditions associated with morbidity has important implications for public health, and in particular for the concept of healthy life expectancy (Murray et al., 2013), namely whether the additional years of life that the most recently born cohorts will gain are years of good health or years of disability and frailty. A desirable scenario is for healthy life expectancy to grow faster than total life expectancy, so compression of morbidity will be achieved (Fries, 1980; Fries, Bruce, & Chakravarty, 2011). In the absence of recovery from poor health, a necessary condition for this is that the onset of morbidity is postponed in more recently born cohorts, for whom life expectancy is longer. If instead life expectancy grows faster than healthy life expectancy and there is no recovery from poor health, expansion of morbidity will occur (Gruenberg, 1977; Manton, 1982; Olshansky, Rudberg, Carnes, Cassel, & Brody, 1991). This hypothesis will be supported if, at the same age, younger cohorts have worse or similar health as older cohorts, or if life expectancy increases faster than healthy life expectancy. A third theory proposed by Manton (1982) suggests a sort of continuation of the status quo, where the increase in life expectancy is accompanied by an equivalent postponement of the onset of morbidity that implies an equivalent increase in healthy life expectancy. An alternative interpretation suggests that dynamic equilibrium occurs when the years spent with mild or moderate disability expand, but years spent with severe disability compress (Jagger et al., 2017). We note, that if dynamic equilibrium is realised, the burden on the health care system is likely to increase,

assuming interventions are required at all levels of severity of a health condition, despite the stabilisation of the years lived with morbidity, since due to population ageing more people will be surviving at older ages. This in turn would have potentially profound implications for the development of health systems in terms of costs and design, as well as for the focus of public health policy.

In this paper we empirically test the hypotheses of compression versus expansion of morbidity using two methods with different underlying assumptions. First, we explore cohort differences in subjective and objective indicators of health among working age adults in England, using repeated cross-sectional data from the last twenty years. We adopt a regression-based approach in which we estimate gender-specific age-adjusted year of birth health associations, including a formal measurement model to test for invariance across generations, to take into account the possibility of changes in reporting styles over time. Second, we calculate gender-specific period healthy life expectancy for 1993, 2003 and 2013 for people at aged 25 and test whether the absolute number of years expected to live in good health has increased. We replicated this analysis by calculating partial life expectancy and healthy life expectancy between age 25 and 64 (results available in the appendix). This triangulation approach (i.e. use of more than one method to answer the same research question) (Lawlor, Tilling, & Davey Smith, 2016) enables us to make a substantive contribution to the existing literature by testing for compression of morbidity to a broad set of objective and subjective health measures to more recent generations (Chen, Cohen, & Kasen, 2007; Rice et al., 2010; Shaw et al., 2014).

## Methods

### Data & Sample

The data for this study was constructed from repeated cross-sections of the Health Survey for England (HSE). The HSE is a nationally representative cross-sectional household survey which is collected annually, and includes subjective health measures, self-reports of specific conditions, and objective measures taken by nurse examination and from blood samples in a follow-up visit to the main interview (Mindell et al., 2012).

We use comparable measures from the 1991 to 2014 surveys inclusive. The household response rate during this period ranged from 62 to 79% with the majority of adult sample members taking part in a nurse visit. We use data on individuals born between 1945 and 1980, excluding those aged less than 25 years and more than 64 years to ensure there are enough people of the same age in multiple birth cohorts in the study sample. The total sample size derived from repeated cross-sections is 135,189. The estimation sample varies according to the outcome due to the fact that not all health measures in the HSE are available in every year. Table 1 shows the inclusion pattern, annual sample size, and prevalence of health outcomes in this study. Sample weights were used in the analysis where they are available since 2002. The use of sample weights did not change the substantive findings.

To test whether we could replicate our findings from the HSE data, we ran the same analysis described below using the General Lifestyle Survey (previously the General Household Survey), 1977-2011 for a limited number of subjective measures: long-term illness and general health. Due to the longer timeframe covered, these analyses also enable a comparison across generations of these health outcomes at a wider span

of ages – specifically at younger ages for the older cohorts. The results (available from authors) were not substantively different from those using more recent HSE data.

## *Measures*

### *Health outcome measures*

The study uses subjective and objective measurements that are known to predict later life mortality and morbidity. The choice of measures also enables a distinction between diagnosed and/or treated conditions, and undiagnosed or untreated conditions to be drawn. The subjective health measures are self-rated health (SRH), long-term illness (LTI) and whether an illness or disability limits activities in any way (LLTI). We use a binary indicator of SRH categorising good to fair and bad health. The long-standing illness question in the HSE was slightly modified from 2012 to refer to physical or mental conditions or illnesses lasting or expected to last 12 months. Including or excluding data since the discontinuity did not change our findings.

Self-reports of ever having (yes/no) high blood pressure (HBP) or diabetes (DIA), and a circulatory illness (CVD) provide outcomes of specific chronic conditions. Self-reported conditions were included in selected HSE surveys between 1991 and 2014 (see Table 1). A latent summary of self-reported measures was derived using SRH, LTI, HBP and DIA.

Objective measures of hypertension (HYP), body mass index (BMI) and glycated haemoglobin (HbA1c) are used. Respondents are defined as hypertensive if they have systolic pressure above 140 or diastolic pressure above 90 or both from nurse-assessed measurement (see Zhao et al. (2019) for detailed information on how blood pressure is measured in HSE). Respondents were categorised as hypertensive if they

reported the use of blood pressure related medication. Body-mass index (BMI) (weight in kilograms over height in metres squared) is derived from objective measurements taken during the nurse visit and used to determine whether someone is overweight ( $BMI \geq 25$ ). Hypertension and BMI are major risk factors of coronary heart disease (He et al., 2001). HbA1c is assessed from blood analytes taken during the nurse visit and shows the percentage of haemoglobin in circulation to which glucose is bound and has been suggested as a screen for diabetes (Moody, 2013). There is no agreed diagnosis threshold, however, Banks et al (2006) take a value equal or greater than 6.5% to indicate clinical diabetes. An adjustment of +1 percentage point was added to respondents taking diabetes related medicines. Sensitivity analysis using the full range of the distributions of BMI and HbA1c in a linear model provided substantively similar results and therefore logistic regression models are presented to aid comparison between outcomes.

### *Explanatory variables*

The main explanatory variable in the regression analysis is birth year, which we use to determine whether younger post-war born cohorts had better, or worse health compared with older post-war cohorts (which, when adjusted for age enables hypotheses related to expansion/ compression of morbidity to be tested). We define 12, three-year birth cohorts, retaining observations ranging between the age of 25 and 64. The oldest cohort in the analysis was born between 1945 and 1947 and the youngest cohort was born between 1978 and 1980. Thus, for example, the synthetic 1945-47 birth cohort is constructed between ages of 44 and 64 years, while the 1978-80 cohort is covered between the ages of 25 and 36 years. Each cohort in our

estimation sample overlaps in age with at least 12 others, thus allowing comparisons over age and cohort of health outcomes. The cohorts are referred to hereafter by their mid-point in the analysis (e.g. we refer to the 1945-1947 cohort as 1946).

### **Analytic plan**

With the cohort samples described above we estimate regression models of subjective and objective health as a function of year of birth and age, including a quadratic term for age. Age is centred at its mean of 41 and year of birth is centred at 1961. The quadratic age term takes account of the non-linear relationship between many of the health outcomes and age. Quadratic terms for cohort were only significant for diagnosed diabetes in men and diagnosed high blood pressure in women, and only the former estimate suggested a reversal of a linear trend. We therefore report models excluding quadratic cohort terms in the interest of parsimony. Estimated parameters on the adjusted-for-age association between year of birth and health allows us to indirectly test the expansion of morbidity hypothesis, given the observed decline in mortality rates in England between 1991 and 2014. This is because a necessary condition for compression of morbidity, in the absence of recovery, which is not likely for most of the outcomes we analyse here, is that after controlling for age differences, more recently born cohorts (that are expected to live longer) are healthier compared to older cohorts, so the average onset of morbidity is postponed. If more recently born cohorts are found to be less healthy, or just as healthy as older cohorts, so the age adjusted year of birth association with poor health is positive or null, then the onset of morbidity will not be postponed and the absolute number of years spent

with ill health will increase, a scenario consistent with the expansion of morbidity hypothesis.

To test for measurement invariance across generations in the way that questions how subjective health is answered, we also estimate a two parameter probit unidimensional latent variable measurement model (B. Muthén, 1984; Rabe-Hesketh & Skrondal, 2008) to derive a latent health metric that combines information from self-reported health indicators, allowing us to determine whether health outcomes are measured equivalently across cohorts (Ploubidis et al., 2017). Measurement equivalence implies that systematic sources of error such as response styles, expectations and social desirability are controlled for. Therefore, any observed between cohort differences manifested as year of birth – health associations in our regression models can be attributed to true differences in health status (Meredith, 1993; Bengt Muthén & Asparouhov, 2017).

Advantages of the regression method adopted here include flexible specification of appropriate linear and logistic models, and the ability to correct for measurement error. More specifically, by using a latent construct for self-reported health, which serves to correct for measurement error in self-reported health. The usual assumptions of correct model specification and no omitted variables or unmeasured confounders apply. We believe the no-unmeasured confounders assumption to be reasonable for the year of birth – health associations, since it is difficult to conceive of a situation where a third variable would simultaneously cause someone being born in specific year as well as their health status in later stages of their life course. We have assumed no unmeasured confounding in the regression models. It is possible, though unlikely, that the composition of birth cohorts captured in this study by year of birth

will be shaped, for example, by macro-economic growth and decline which may also influence health. For example, there is evidence to suggest that men and women's hourly wage, house prices and child allowances affect fertility rates (Ermisch, 1988) and one could assume these differentially affect socioeconomic groups. However, the extent to which they exert influence on year of birth at the individual level is unknown. Despite its flexibility and related advantages, the regression-based approach does not allow for the exact quantification of the number of years individuals are expected to spend in good or poor health. Furthermore, whereas it is straightforward to interpret results pointing to expansion of morbidity, it may be difficult to disentangle compression or dynamic equilibrium without explicitly modelling mortality and morbidity jointly. This is because a negative estimated relationship between ill health and year of birth is consistent with both (i.e. dynamic equilibrium and compression of morbidity). For example, a later born cohort may be less likely to be in poor health at the same age but spend the same amount of years (or more) in poor health because change in healthy life expectancy does not keep pace with change in life expectancy. To further test between these hypotheses, we calculate gender-specific healthy life expectancy with Sullivan's method (Sullivan, 1971), using each outcome as the indicator of poor health. Sullivan's method employs a relatively simple modification of conventional life tables, in which expected years of life are scaled by the average fraction of the population of each age group that are free of a specific illness or disability, in order to compute the expected duration of certain defined conditions of interest (e.g. number of years not in self-reported poor health) among the living population. The age-specific health prevalence for each health outcome was obtained directly from HSE surveys in 1993, 2003 and 2013 for 5-year groups aged 25 and over,

and 5-year age-specific mortality in England for these years available from the Office for National Statistics (O.N.S, 2014). The health expectancy component reflects the current health of a real population adjusted for mortality levels independent of age structure. Sullivan's method relies on the assumption that a specific cohort observed at a certain age in a given year will be experiencing the same health status and prevalence rates observed among the other age groups (i.e. other cohorts) in the same year. This is an extra stationarity assumption, in addition to the age-specific hazard and birth rates assumed to be constant over time, as well as the net migration rates being zero for all ages (Imai & Soneji, 2007). Problems of under- or overestimation may occur when health prevalence changes over time, although several studies demonstrate that Sullivan's method can be extended to estimate health expectancy relaxing these stationarity assumptions (Imai & Soneji, 2007; Mathers & Robine, 1997). Comparing health expectancy in different periods is a direct test of the compression or expansion hypotheses, since the years expected to be lived in good health are explicitly quantified. However, Sullivan's method requires a health outcome observed without measurement error that correctly distinguishes healthy from not healthy individuals and does not allow corrections for measurement error.

In both approaches we consider the decrease, increase or stability in the absolute number of years expected to be spent in poor health as a test for the three hypotheses of population health change. The absolute number – as opposed to the proportion – of person years can inform us about how long people, on average, can expect to live with poor health or disability, and whether the overall burden on health systems and families will increase. We note that in both approaches the between-cohort differences indicated by the year of birth – health association as well as differences in

healthy life expectancy in various periods may be due to period or cohort effects, but disentangling these would require additional assumptions (Keyes, Utz, Robinson, & Li, 2010) and lies beyond the scope of this paper since distinguishing between them is not required for valid tests of the expansion or compression of morbidity hypotheses.

## Results

Table 1 shows the descriptive trend in each outcome over time for a) women and b) men aged 25 to 64. The prevalence of poor health according to SRH, LTI, LLTI, HBP, DIA, CVD, HYP, BMI and HbA1c have increased in the working-age population during the period 1991 to 2014.

Table 2 shows results from the regression method for the odds ratio of having a health condition for being born in a later post-war cohort compared to the mean among those aged 25-64 and from the Sullivan's method for a ratio between the change in unhealthy life years and the increase in life expectancy of people at aged 25 during the periods 1993 to 2003 and 2003 to 2013. A value above 1 in the regression method suggests later born cohorts compared with earlier born cohorts are more likely to report poor health at the same age. In the Sullivan's methods, given that the denominator is always positive (i.e. life expectancy has increased for both sex in each time period), a negative ratio indicates less years spent in poor health (compression); a positive ratio denotes expansion of morbidity, and we distinguish i) a value between 0 and 1 that would indicate more years spent in poor health, but a smaller number compared to the total gained years of life, ii) a value of 1 would indicate every additional year of life expectancy will be spent in poor health; iii) a value above 1

indicates more than the additional years of life expectancy will be spent in poor health. These estimates combined can indicate whether there is an expansion or compression of morbidity. The results support an expansion of morbidity for SRH, HBP (in men), DIA, CVD, BMI and HbA1c. The results were inconclusive for LTI, LLTI, HBP (in women) and HYP. The following section describes the results by health outcome with reference to the regression and Sullivan's methods. The full regression and Sullivan's output is available from the authors on request.

### *Self-rated health (SRH)*

The regression method shows that later born post-war cohorts were more likely to report bad SRH in men or women at the same age. Figure 1 shows the predicted probabilities for three selected cohorts (1946, 1958 and 1970) and demonstrates how at the overlapping ages bad health was more prevalent in later born cohorts. This finding is confirmed by Sullivan's method that shows for every year of additional life expectancy gain between 1993 and 2003, at age 25, more than half of those years will be spent in bad SRH in men and women. More than a fifth of the additional life years gains between 2003 and 2013 will be spent in bad SRH in men and women. The regression and Sullivan's findings combined provide support for an expansion of morbidity in relation to bad SRH.

### *Long-term illness (LTI)*

Women were no more or less likely to report a LTI by year of birth in the regression model, whereas men were marginally less likely to report a LTI if they were born later during the period 1945 to 1980 when taking into account age. The number of years expected to live with a LTI increased between 1993 and 2003 and accounts for almost

two times the additional years of life expectancy in men and more than two and half times in women. The reverse was the case between 2003 and 2013. These findings suggest no clear support for either expansion or compression of morbidity in LTI.

#### *Limiting long-term illness (LLTI)*

The regression method showed that men and women were both less likely to report having a LLTI if born later during the post-war period. For example, 16% of men born in 1970 were predicted to have a LLTI at age 44 compared with 20% of men born in 1958 (see Figure 1). In this instance, the smaller prevalence of LLTI in the more recently born cohorts was not enough to overtake the decline in age specific mortality in men or women between 1996 and 2003 and only in men between 2003 and 2013, suggesting little support for either expansion or compression of morbidity.

#### *Self-reported diagnosed high blood pressure (HBP)*

The regression method shows high blood pressure diagnosis was less common in later born women and more common in later born men during the period 1945 to 1980. At age 56, more than 33% of women born in 1946 were predicted to have been diagnosed with high blood pressure compared with 26% of those born in 1958 (see Figure 1). According to Sullivan's method, there was an increase in the number of years expected to live with self-reported diagnosed hypertension in men and women across all working age groups between 1993 and 2003 and between 2003 and 2013 in men only. The opposite is the case for women between 2003 and 2013. Figure 2 shows that women aged 25 in 2003 were expected to spend 35% of their life expectancy with self-reported hypertension compared with 31% for women in the same age group in

2013. These results indicate expansion of morbidity with respect to high blood pressure diagnosis in men but are inconclusive in women.

#### *Self-reported diagnosed diabetes (DIA)*

Men and women were considerably more likely to report a diabetes diagnosis in younger cohorts born between 1945 and 1980. Figure 1 shows that less than 4% of women born in 1946 were predicted to report a diabetes diagnosis at age 56 compared with 8% of women born in 1958 at the same age. Sullivan's method shows that the majority of additional years of life expectancy to be lived by a 25-year-old man (59%) or woman (85%) between 1993 and 2003 and 2003 and 2013 were expected to be lived with a diabetes diagnosis (see Figure 2). The results strongly support an expansion of morbidity.

#### *Self-reported heart or circulatory illness (CVD)*

The regression method shows there are no differences between post-war cohorts in reports of CVD in men or women at the same age. This is exemplified by the predicted probabilities in Figure 1 showing overlapping 95% confidence intervals at the adjoining ages in the selected cohorts. Considering that more recently born cohorts are expected to live longer, this 'null' result suggests expansion of morbidity on this outcome (as explained in the statistical methods section of this paper). This is confirmed by Sullivan's method that shows for every year of additional life expectancy gain between 1993 and 2003, at age 25, more than one times will be spent with CVD in men and women. These results point to an expansion of morbidity for CVD.

### *Clinical hypertension (HYP)*

According to the regression results the prevalence of clinical hypertension was the same across post-war birth cohorts at a given age in women and men in the regression analysis. According to the Sullivan's method, around a third of the number of years gained in life expectancy between 2003 and 2013 were with hypertension in men. The proportion of life expectancy in men spent with hypertension was marginally higher in 2003 (38%) compared with 2013 (37%). Women were expected to spend fewer years with hypertension in 2013 (34%) compared with 2003 (36%). The findings combined indicate inconclusive findings for expansion or compression of clinical hypertension in women and men.

### *Clinical diabetes (HbA1c)*

The regression analysis shows greater predicted probability in high blood sugars in later born post-war male and female cohorts at the same age. Figure 1 shows that 16% of men at aged 56 born in 1958 were predicted to have blood sugars above 6.5% compared with 9% born in 1946 at the same age. The additional years gained in life expectancy for a 25-year-old between 2003 and 2013 were dwarfed by more than a one times increase in the number of years spent with clinical diabetes in women and almost one in men. The findings point to an expansion of morbidity in HbA1c.

### *Overweight BMI*

The regression method shows later born cohorts were considerably more likely to be overweight at the same age in men and women. More than 78% of men aged 44 born in 1970 were predicted to be overweight compared with 71% born in 1958 (see Figure 1). The Sullivan's method shows that a man age 25 in 1993 was expected to spend

53% of their life expectancy with an overweight BMI compared with 61% by 2013 (see Figure 2). Both methods suggest strong support for an expansion of morbidity in BMI. Similar findings are the case (not shown here) for obesity (i.e. BMI  $\geq 30$ ).

## Conclusion

Previous work has shown that over the last 20 years, despite increases in life expectancy, (O.N.S, 2013), the working age population in England has a greater prevalence of poor health on several outcomes of health (Samaranayaka & Gulliford, 2013). This is not simply a consequence of an ageing population, but a trend of greater prevalence of poor health outcomes in later born generations at the same age (Rice et al., 2010). In this paper we have built on such cross-cohort comparative work to test competing theories of compression or expansion of morbidity. We have shown that successively younger post-war cohorts at the same age report more bad self-rated health, the same in men and marginally less in women long-term illness, and less limiting long-term illness. More recently born post-war cohorts at the same age have more or the same self-reported high blood pressure (in men only), diabetes and heart or circulatory illnesses and more likely to have objectively measured diabetes and an overweight BMI and the same clinical hypertension but, in women, less likely to self-report high blood pressure. We have also shown that in the 20-year period between 1993 and 2013 the number of years expected to live with each of these health conditions has increased relative to change in life expectancy. The only expectations are self-reported high blood pressure and clinical hypertension in women, which have declined relative to change in life expectancy. When we considered partial life expectancy (between age 25-64), changes were smaller in

absolute and relative terms compared to changes in life expectancy at 25 because the improvements in mortality occurred mostly at older ages during the period 1993-2013 (Public Health England, 2018). As a consequence, the dynamics in health expectancy between age 25 and 64 was mostly driven by changes in the prevalence in health conditions over the two decades, and the ratio of changes in HE and LE is magnified by very small denominators due to modest increases in LE between ages 25 and 64 in the past two decades.

Both approaches (regression and Sullivan's method) point to an overall expansion of morbidity in the health of the post-war working age population in England.

The trend presents challenges for healthcare service providers who already have to meet the demands of more people living longer. It appears in future more people will require health care services at a younger age and consequently depend on them for longer (Murray et al., 2013). Our findings confirm previous observations from the Global Burden of Disease Study which has shown that in all age groups the UK is heading towards expansion of morbidity, with healthy life expectancy increasing at a slower pace compared to total life expectancy between 2000 and 2010 (Murray et al., 2013). They are also in accordance with previously reported cross-cohort comparisons, where more recently born cohorts were found to be less healthy (Johnson et al., 2015; Ploubidis et al., 2017).

Our findings are in contrast to those on older pre-war born cohorts who have been shown to experience absolute compression of cognitive morbidity, relative compression of self-rated health and dynamic equilibrium of disability (Jagger et al., 2017). Furthermore, more recently born cohorts in those aged over 65 in England had lower prevalence of cognitive impairment and lower inflammation and cholesterol,

suggesting compression of morbidity in these age groups (Martin, Schoeni, Andreski, & Jagger, 2012). The more complex pattern observed in the older population (compression or dynamic equilibrium), compared to our findings on the working age population where expansion is mainly supported, indicate a possible structural break between generations on the joint progress of health and mortality. While each subsequent generation of pre-war cohorts were typically spending less years in poor health, this trend seems to have mainly reversed in post war generations.

In relation to self-rated health, it might be the case that people report poorer health if they are in low socioeconomic groups and in later born cohorts because there is greater social inequality within their generation, which makes people more likely to think of themselves as being less well off than those in a similar economic position born in earlier generations (Jivraj, 2020). Furthermore, later born cohorts may have a higher expectation of their health. A test for strong (“scalar”) measurement invariance (Meredith, 1993; Bengt Muthén & Asparouhov, 2017) in self-reported measures of health across cohorts showed that subjective health assessment is equivalent, indicating that what is thought of as poor health is not different across generations and the observed between cohort differences were due to true differences in health status.

It is important to note that the health differences uncovered in successively born post-war generations might not point to poorer health per se, if they are in fact caused by improvements in diagnosis of treatable conditions, and greater treatment of them (Samaranayaka & Gulliford, 2013). For example, the results showed a lower prevalence of clinical hypertension in later born cohorts and showed all additional years of life expectancy were spent without a clinically hypertensive reading when

only including those with an objectively reported hypertensive blood pressure reading as hypertensive (i.e. including those taking medication with controlled hypertension in the non-hypertensive category). This could explain why the rates of difficult to treat health conditions, such as any limiting illnesses generally and heart and circulatory illness specifically were similar or lower at the same age across cohorts, but that more easily treatable conditions, such as diabetes were more prevalent.

Alternatively, it might be the case that greater prevalence of overweight BMI and clinical diabetes has not manifested into difficult to treat conditions in the youngest post-war cohorts, but they will as they enter older age. Either way, treatment is expensive, diabetes costs the NHS £23.7 billion per annum (10% of its annual budget) (Hex, Bartlett, Wright, Taylor, & Varley, 2012). Our findings suggest treatment costs for high blood pressure and diabetes will increase as more people live for longer with these and other conditions.

There are a number of limitations of this study that the findings should be set against. We have used cross-sectional data from the HSE to compare the health profile of different birth cohorts born after 1944 in our analysis. This means that we can compare the health of working age post-war cohorts at equivalent ages over a relatively long period of time. The HSE is nationally representative and therefore should provide an accurate measure of health in England through time, for the non-institutionalised population. Considering that the prevalence of individuals aged under 60 in institutions is less than 3% in the UK, we are confident that even with extreme differences in the distributions of health outcomes as well as mortality rates between those in institutions and the non-institutionalised population our results would not materially change, an interpretation which is reinforced by findings in the

missing data literature that less than 5% prevalence of missingness does not influence parameter estimates even if ignored (Carpenter & Kenward, 2013). However, the cross-sectional nature of the underlying data used for this analysis means that we are limited in what we can say about the trajectories of individual people in term of the development of their health. Future research should consider older age cohorts (actual or synthetic) to explore the risk of health conditions at times during the life course when poor health and risk of mortality is considerably greater.

In summary, healthy life expectancy has increased at a slower pace compared to total life expectancy in the 20 years between 1993 and 2013 for the working age population in England according to an array of morbidity outcomes, especially in the first decade of observation, 1993-2003. Younger post-war birth generations appear to have poorer health or, at best, the same health as their older counterparts at the same age according to subjective and objective measures of health. Assuming that life expectancy in England will either continue to increase, or remain the same (rather than reduce), our findings lend support to the expansion of morbidity hypothesis. This trend points to a greater demand for public healthcare at younger ages. Future work using longitudinal data, for example from the British birth cohort studies, is warranted to understand the aetiological mechanism that underlies the observed between birth cohort differences, as it is crucial to identify policy modifiable factors that lead to compression of morbidity, the only scenario that has the potential to offset the effects of population ageing. Furthermore, considering that in recent years a slowdown in the increase of life expectancy in the UK has been observed (David A. Leon, Jdanov, & Shkolnikov, 2019), work that investigates the potential impact of the long term

generational differences in health we report in this paper on mortality is warranted. This, we believe, is crucial, since stagnating life expectancy may be due to the association between health and mortality becoming stronger in more recently born cohorts. This, if confirmed, goes against the basic premise of the age of degenerative and man-made [*sic*] diseases phase of the first epidemiological transition (Omran, 1971) and observed in the UK trends in the 20<sup>th</sup> and early 21<sup>st</sup> centuries, where the association between health and mortality became weaker over time and life expectancy increased.

## Declarations

### **Acknowledgments**

Not applicable

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Table 1 Health outcome, by prevalence rate (%) and HSE year

HSE year	Un-weighted Sample aged 25-64 <sup>1</sup>	Subjective health			Self-reported diagnosed conditions			Objective health		
		Bad SRH	LTI	LLTI	High BP	Diabetes	CVD	Hypertensive	BMI>=25	HbA1c =>6.5
1991	2,654	2.2%	27.8%	-	14.9%	0.0%	2.3%	-	44.1%	-
1992	1,685	2.2%	28.9%	-	18.6%	0.0%	2.9%	-	45.6%	-
1993	7,947	2.4%	30.3%	-	17.5%	1.3%	3.1%	-	50.0%	-
1994	7,375	2.8%	29.5%	-	15.5%	1.2%	2.7%	-	50.0%	-
1995	7,668	3.0%	32.1%	-	-	-	3.3%	-	52.2%	-
1996	8,143	3.6%	33.1%	18.3%	-	-	3.5%	-	54.8%	-
1997	4,456	3.8%	35.5%	18.9%	-	-	4.4%	-	56.2%	-
1998	8,352	4.1%	35.7%	19.1%	18.4%	1.7%	3.8%	-	57.0%	-
1999	4,242	5.0%	35.9%	19.5%	-	-	5.0%	-	58.0%	-
2000	4,467	4.5%	36.3%	19.8%	-	-	5.4%	-	59.1%	-
2001	8,838	4.6%	37.4%	19.8%	-	-	6.1%	-	61.8%	-
2002	4,194	5.3%	38.5%	21.6%	-	-	6.3%	-	62.7%	-
2003	8,685	4.8%	39.6%	20.4%	22.2%	2.8%	6.6%	20.7%	62.5%	2.5%
2004	3,917	5.2%	40.2%	20.4%	-	-	6.4%	-	64.3%	-
2005	4,644	5.2%	38.6%	20.3%	-	-	7.3%	22.1%	63.3%	-
2006	8,590	5.3%	38.1%	20.1%	23.6%	3.7%	7.3%	22.1%	64.7%	3.3%
2007	4,139	5.3%	40.3%	21.1%	-	-	8.4%	24.0%	64.3%	-
2008	9,114	5.6%	39.3%	20.4%	-	-	9.0%	25.7%	65.9%	-
2009	2,771	6.5%	39.7%	20.4%	22.8%	5.1%	8.8%	26.0%	66.1%	4.9%
2010	4,946	7.0%	41.4%	22.4%	22.7%	5.4%	9.7%	27.5%	68.6%	5.2%
2011	4,943	6.1%	38.9%	20.7%	26.1%	5.6%	8.5%	26.5%	67.6%	6.6%
2012	4,476	6.7%	37.0%	20.1%	20.3%	5.8%	8.1%	26.1%	67.9%	6.5%
2013	4,716	6.7%	38.1%	21.1%	21.6%	5.8%	9.5%	26.5%	68.2%	6.8%
2014	4,227	7.2%	39.1%	21.6%	22.2%	6.8%	8.3%	28.2%	68.3%	6.3%
Un-weighted N	135,189	134,642	134,640	107,809	70,720	70,800	134,576	36,052	120,480	27,701

Notes: <sup>1</sup> sample size range across each health measures. Dashes indicate measure was not included in HSE survey year. Rates are adjusted using survey weights available since 2003.

Table 2. Regression and Sullivan methods results testing for expansion, or compression of morbidity, or dynamic equilibrium

	Regression method (Odds ratio) <sup>a</sup>	Sullivan method (ratio of years) <sup>b</sup>		Regression method (Odds ratio) <sup>a</sup>	Sullivan method (ratio of years) <sup>b</sup>		Regression method (Odds ratio) <sup>a</sup>	Sullivan method (ratio of years) <sup>b</sup>	
	1991-2014	1993-2003	2003-2013	1991-2014	1993-2003	2003-2013	1991-2014	1993-2003	2003-2013
	<u>Not good self-rated health</u>			<u>Long-standing illness (self-report)</u>			<u>Limiting illness/disability (self-report)</u>		
Women	<b>1.01 (1.01, 1.02)</b>	0.77	0.36	1.00 (0.99, 1.00)	2.72	-0.67	<b>0.99 (0.98,0.99)</b>	0.43	0.23
Men	<b>1.02 (1.01, 1.02)</b>	0.54	0.21	<b>0.99 (0.99,0.99)</b>	1.95	-0.59	<b>0.98 (0.97,0.98)</b>	0.36	-0.02
	<u>High blood pressure (self-report diagnosis)</u>			<u>Diabetes (self-report diagnosis)</u>			<u>Cardiovascular disease (self-report)</u>		
Women	<b>0.97 (0.97,0.98)</b>	2.00	-0.50	<b>1.07 (1.06,1.08)</b>	0.76	0.92	1.00 (0.99,1.01)	1.37	0.07
Men	<b>1.02 (1.01, 1.02)</b>	2.33	0.43	<b>1.05 (1.04, 1.06)</b>	0.41	0.74	1.00 (1.00,1.01)	1.11	0.12
	<u>Hypertensive (objective measure)</u>			<u>Glycated haemoglobin (HbA1c)</u>			<u>BMI &gt;=25 (overweight)</u>		
Women	0.99 (0.98,1.00)	na	-0.30	<b>1.06 (1.03, 1.09)</b>	na	1.08	<b>1.03 (1.02, 1.03)</b>	3.24	0.92
Men	0.99 (0.98,1.00)	na	0.32	<b>1.06 (1.03, 1.08)</b>	na	0.98	<b>1.03 (1.02, 1.03)</b>	2.44	1.18

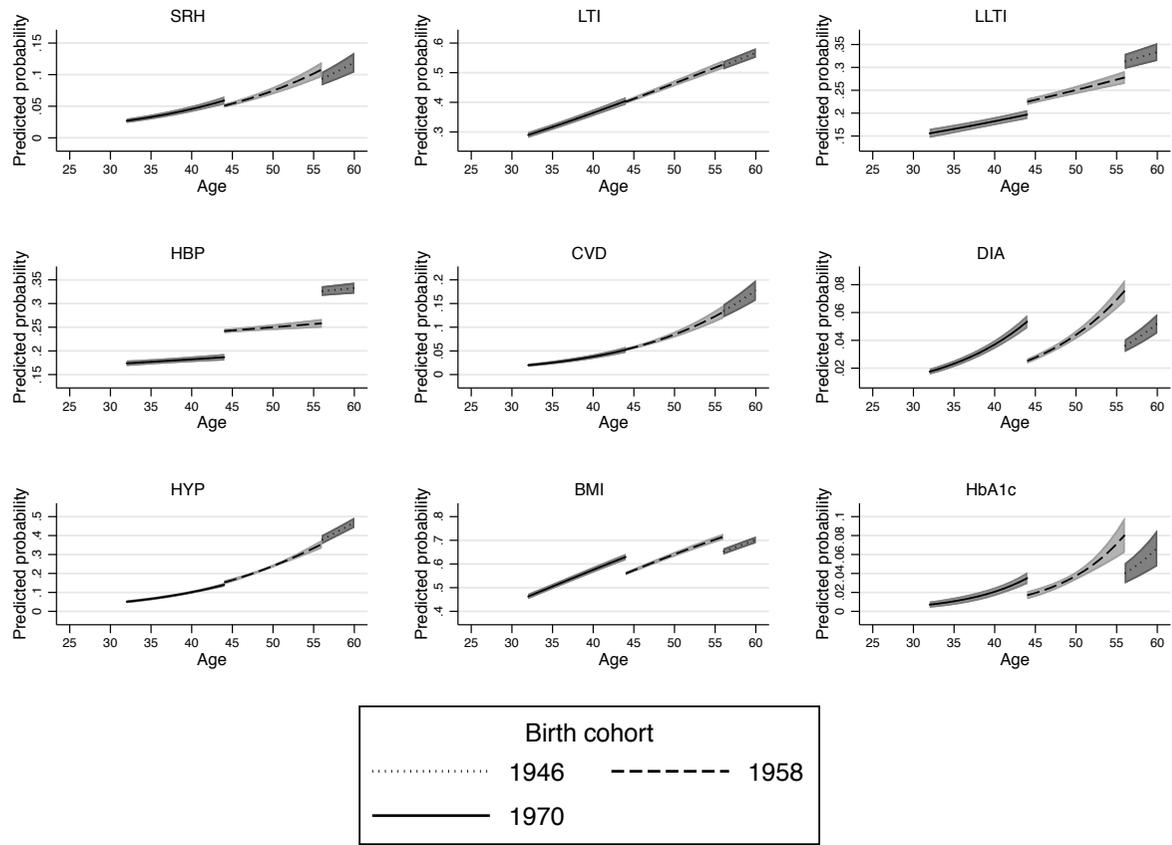
<sup>a</sup> Regression model: Shows estimated odds ratios associated with being born into a later cohort between 1945 and 1980, results are adjusted for age, age<sup>2</sup> and cohort. A value higher than 1 indicates expansion of morbidity. Bold denotes p<0.001

<sup>b</sup> Sullivan method: Shows ratio of increased years of unhealthy life, to increase in life expectancy, between 1993 and 2003 and between 2003 and 2013. LLTI estimates 1996-2003 and 2003 and 2013; Positive ratio denotes expansion of morbidity.

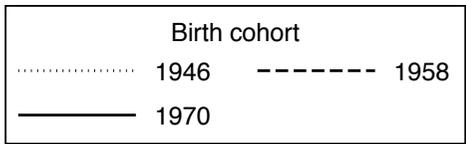
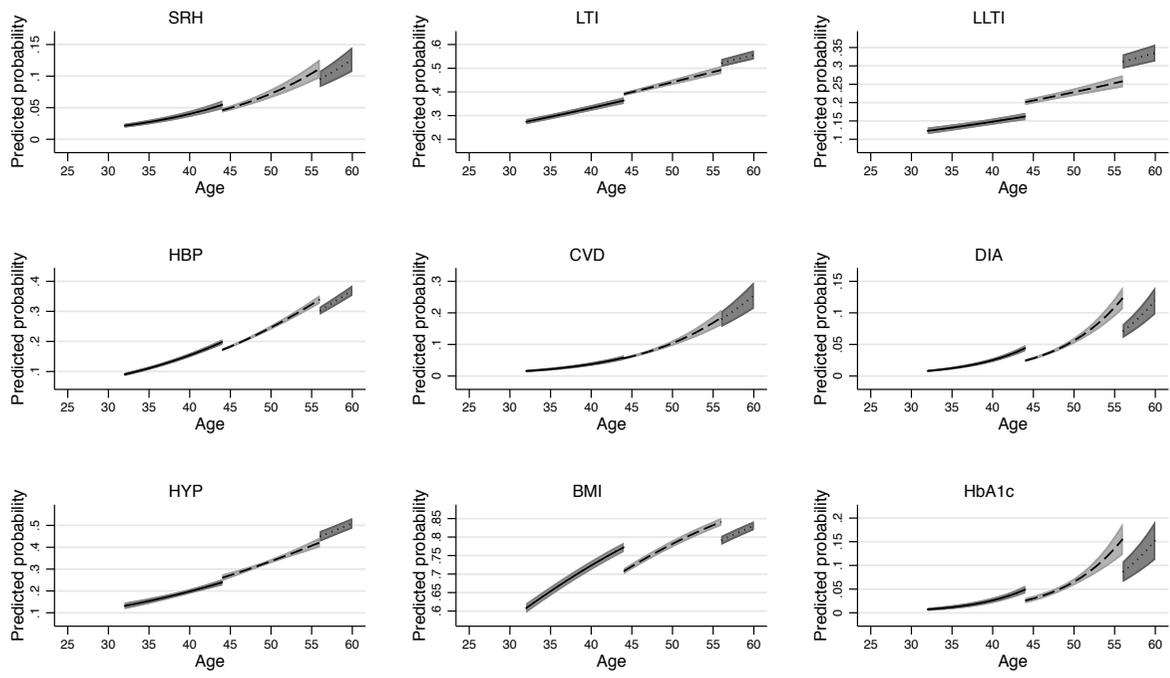
Supports expansion of morbidity
Inconclusive

Figure 1. Regression predicted probabilities by health outcome for selected years of birth, by age

a) Women

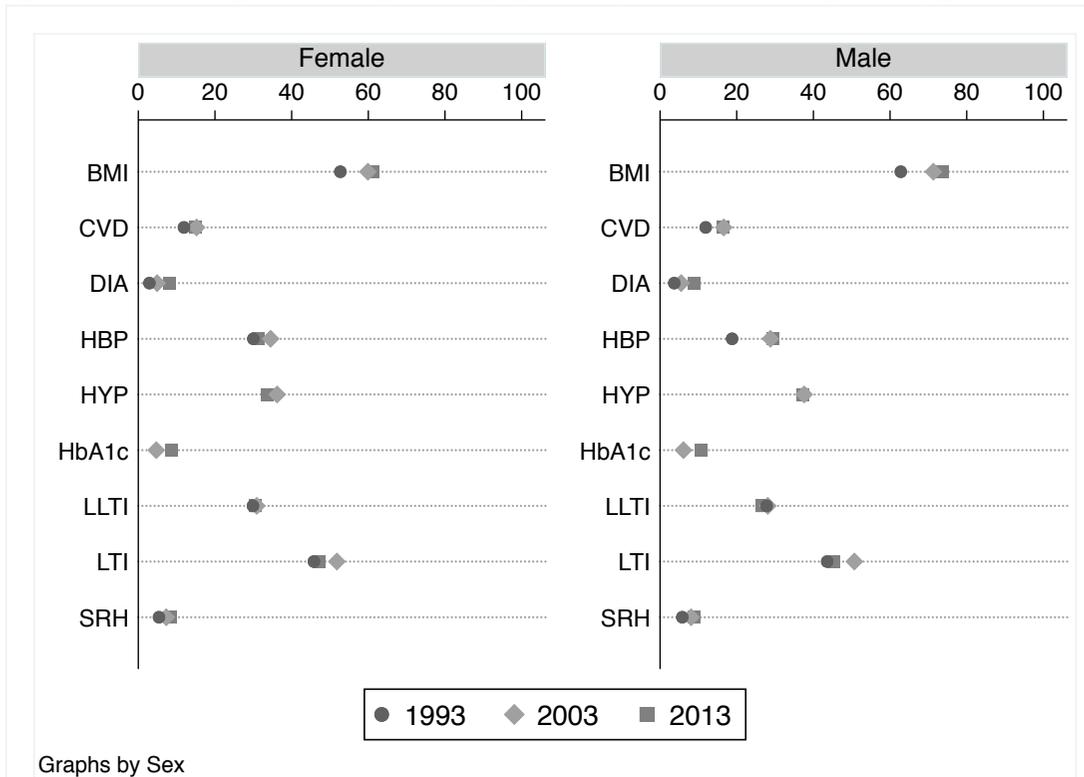


b) Men



Notes: Adjusted for age, age<sup>2</sup> and cohort. Shaded area represents 95% confidence interval. SRH – poor self-rated health; LTI – long-term illness; LLTI – limiting long-term illness; HBP – doctor diagnosed high blood pressure; DIA – doctor diagnosed diabetes; CVD – self-reported heart or circulatory system illness; HYP – objective measure hypertension; BMI – body mass index >=25; HbA1c – glycated haemoglobin >= 6.5%

Figure 2. Percentage of life expectancy spent in poor health at age 25 by gender and health outcome, 1993, 2003, 2013



Notes: Gender-specific healthy life expectancy with Sullivan’s method using each outcome as the indicator of poor health. Age-specific health prevalence for each health outcome obtained from HSE surveys in 1993, 2003 and 2013, and age-specific mortality in England for these years available from the Office for National Statistics. SRH – poor self-rated health; LTI – long-term illness; LLTI – limiting long-term illness; HBP – doctor diagnosed high blood pressure; DIA – doctor diagnosed diabetes; CVD – self-reported heart or circulatory system illness; HYP – objective measure hypertension; BMI – body mass index  $\geq 25$ ; HbA1c – glycated haemoglobin  $\geq 6.5\%$

## Appendix

Table A1. Life expectancy, health expectancy and unhealthy life expectancy at age 25 by sex, 1993, 2003 and 2013

Type of health	Males						Females					
	1993		2003		2013		1993		2003		2013	
	Est.	95% CI	Est.	95% CI	Est.	95% CI	Est.	95% CI	Est.	95% CI	Est.	95% CI
LE	49.9		52.3		55.1		54.9		56.3		58.6	
SRH good	47	(46.7 - 47.3)	48.1	(47.7 - 48.5)	50.3	(49.8 - 50.8)	51.9	(51.6 - 52.2)	52.3	(51.9 - 52.6)	53.7	(53.2 - 54.2)
SRH poor	2.9	(2.6 - 3.2)	4.2	(3.9 - 4.6)	4.8	(4.3 - 5.3)	2.9	(2.7 - 3.2)	4.1	(3.7 - 4.4)	4.9	(4.4 - 5.4)
LTI without	28.1	(27.6 - 28.7)	25.8	(25.2 - 26.4)	30.2	(29.4 - 31.1)	29.7	(29.2 - 30.3)	27.2	(26.6 - 27.8)	30.9	(30.1 - 31.7)
LTI with	21.7	(21.2 - 22.3)	26.5	(25.9 - 27.2)	24.9	(24 - 25.7)	25.1	(24.5 - 25.7)	29.2	(28.6 - 29.8)	27.7	(26.9 - 28.5)
LLTI without*	36.2	(35.7 - 36.7)	37.6	(37 - 38.2)	40.4	(39.7 - 41.2)	38	(37.4 - 38.5)	38.9	(38.3 - 39.5)	40.7	(39.9 - 41.4)
LLTI with*	13.9	(13.4 - 14.4)	14.7	(14.1 - 15.3)	14.7	(13.9 - 15.4)	17.4	(16.9 - 18)	17.5	(16.9 - 18.1)	17.9	(17.1 - 18.7)
BP without	40.5	(40.1 - 41)	37.2	(36.7 - 37.8)	38.8	(38.1 - 39.6)	38.4	(37.8 - 38.9)	36.9	(36.3 - 37.5)	40.2	(39.5 - 41)
BP with	9.4	(8.9 - 9.8)	15.1	(14.5 - 15.7)	16.3	(15.5 - 17)	16.5	(15.9 - 17)	19.5	(18.9 - 20.1)	18.3	(17.6 - 19.1)
Diab without	48	(47.8 - 48.2)	49.4	(49.1 - 49.8)	50.2	(49.7 - 50.7)	53.2	(53 - 53.5)	53.6	(53.3 - 53.9)	53.8	(53.3 - 54.3)
Diab with	1.9	(1.6 - 2.1)	2.9	(2.6 - 3.2)	4.9	(4.4 - 5.4)	1.6	(1.4 - 1.8)	2.7	(2.4 - 3)	4.8	(4.3 - 5.3)
CVD without	43.9	(43.5 - 44.3)	43.6	(43.2 - 44.1)	46.1	(45.4 - 46.7)	48.3	(47.9 - 48.7)	47.8	(47.3 - 48.2)	49.8	(49.2 - 50.5)
CVD with	6	(5.6 - 6.3)	8.7	(8.2 - 9.2)	9	(8.4 - 9.7)	6.5	(6.1 - 7)	8.6	(8.1 - 9.1)	8.7	(8.1 - 9.4)
BMI>=25 w/o	18.6	(18 - 19.1)	15	(14.4 - 15.7)	14.5	(13.6 - 15.4)	25.9	(25.3 - 26.6)	22.6	(21.9 - 23.3)	22.8	(21.8 - 23.7)
BMI>=25 with	31.3	(30.7 - 31.9)	37.3	(36.6 - 38)	40.6	(39.7 - 41.5)	28.9	(28.3 - 29.6)	33.8	(33.1 - 34.5)	35.8	(34.9 - 36.8)
Hyp (+medications)without			32.7	(31.9 - 33.4)	34.6	(33.5 - 35.6)			35.9	(35.3 - 36.6)	38.8	(37.9 - 39.7)
Hyp (+medications)with			19.7	(18.9 - 20.4)	20.5	(19.5 - 21.6)			20.4	(19.8 - 21.1)	19.8	(18.8 - 20.7)
Hyp without			46.7	(46.2 - 47.2)	51.4	(5.1 - 6.2)			49.8	(49.2 - 50.3)	55.1	(54.5 - 55.6)
Hyp with			5.7	(50.8 - 51.9)	3.7	(3.2 - 4.3)			6.6	(6.1 - 7.1)	3.5	(3 - 4.1)
HbA1c (+medications) without			49.2	(48.7 - 49.6)	49.2	(48.4 - 50)			53.7	(53.3 - 54.1)	53.5	(52.8 - 54.3)
HbA1c (+medications) without			3.2	(2.7 - 3.6)	5.9	(5.1 - 6.7)			2.6	(2.2 - 3)	5	(4.3 - 5.8)

HbA1c without	49.5	(49.1 - 49.9)	50.1	(49.4 - 50.8)	54	(53.6 - 54.4)	53.9	(53.2 - 54.7)
HbA1c without	2.8	(2.4 - 3.2)	5	(4.3 - 5.7)	2.3	(2 - 2.7)	4.6	(3.9 - 5.4)

\*For LLTI, the first observation year is 1996 instead of 1993, hence for that year years with LLTI + years without LLTI do not sum up to total life expectancy.  
95% CI in brackets

Table A2. Partial life expectancy, health expectancy and unhealthy life expectancy between age 25 and 64 by sex, 1993, 2003 and 2013

Type of health	Males						Females					
	1993		2003		2013		1993		2003		2013	
	Est.	95% CI	Est.	95% CI	Est.	95% CI	Est.	95% CI	Est.	95% CI	Est.	95% CI
LE	38.2		38.4		38.7		38.9		39		39.2	
SRH good	36.5	(36.3 - 36.7)	36	(35.8 - 36.3)	36.1	(35.7 - 36.4)	37.4	(37.2 - 37.6)	36.9	(36.6 - 37.1)	37	(36.7 - 37.3)
SRH poor	1.6	(1.4 - 1.9)	2.3	(2.1 - 2.6)	2.6	(2.2 - 3)	1.5	(1.3 - 1.7)	2.1	(1.9 - 2.4)	2.2	(1.9 - 2.5)
LTI without	23.9	(23.4 - 24.4)	21.8	(21.3 - 22.4)	24.9	(24.2 - 25.6)	23.9	(23.4 - 24.4)	22.4	(21.9 - 22.9)	25.2	(24.6 - 25.9)
LTI with	14.3	(13.8 - 14.8)	16.6	(16 - 17.1)	13.8	(13.1 - 14.5)	15	(14.5 - 15.5)	16.6	(16.1 - 17.1)	14	(13.3 - 14.6)
LLTI without*	29.5	(29.3 - 29.8)	29.8	(29.3 - 30.3)	31.2	(30.6 - 31.7)	29.5	(29.1 - 29.9)	29.9	(29.5 - 30.4)	31	(30.5 - 31.6)
LLTI with*	8.7	(8.4 - 9)	8.6	(8.1 - 9)	7.5	(6.9 - 8.1)	9.4	(9 - 9.9)	9.1	(8.6 - 9.5)	8.2	(7.6 - 8.7)
BP without	32.5	(32.2 - 32.9)	29.6	(29.2 - 30.1)	30.9	(30.4 - 31.5)	28.3	(27.8 - 28.7)	28.5	(28 - 28.9)	31.9	(31.4 - 32.4)
BP with	5.6	(5.3 - 6)	8.7	(8.3 - 9.2)	7.7	(7.2 - 8.3)	10.6	(10.1 - 11)	10.5	(10.1 - 11)	7.3	(6.8 - 7.8)
Diab without	37.1	(36.9 - 37.3)	37	(36.8 - 37.2)	36.8	(36.5 - 37.1)	38	(37.9 - 38.2)	37.8	(37.6 - 37.9)	37.1	(36.8 - 37.4)
Diab with	1	(0.9 - 1.2)	1.4	(1.2 - 1.6)	1.9	(1.6 - 2.2)	0.9	(0.7 - 1)	1.2	(1.1 - 1.4)	2.1	(1.8 - 2.4)
CVD without	35.4	(35.1 - 35.6)	34.6	(34.3 - 34.9)	35	(34.6 - 35.4)	35.9	(35.6 - 36.2)	35.8	(35.6 - 36.1)	36.5	(36.2 - 36.9)
CVD with	2.8	(2.5 - 3.1)	3.8	(3.5 - 4.1)	3.7	(3.3 - 4.1)	3	(2.7 - 3.3)	3.2	(2.9 - 3.5)	2.6	(2.3 - 3)
BMI>=25												
without	14.3	(13.8 - 14.8)	11.3	(10.8 - 11.8)	10.7	(10 - 11.5)	19.4	(18.9 - 19.9)	16.9	(16.3 - 17.4)	16	(15.2 - 16.7)
BMI>=25 with	23.9	(23.4 - 24.4)	27.1	(26.5 - 27.6)	27.9	(27.2 - 28.7)	19.5	(18.9 - 20)	22.1	(21.6 - 22.7)	23.2	(22.5 - 23.9)
Hyp (+medications)without			27.5	(26.9 - 28.1)	28.1	(27.2 - 28.9)			30.8	(30.3 - 31.3)	32	(31.4 - 32.7)
Hyp (+medications)with			10.9	(10.3 - 11.5)	10.6	(9.8 - 11.4)			8.2	(7.8 - 8.7)	7.1	(6.5 - 7.8)

Hyp without	35.4	(35.4 - 35)	36.4	(36.4 - 35.9)	36.7	(36.7 - 36.4)	37.7	(37.7 - 37.4)
Hyp with	3	(2.6 - 3.4)	2.3	(1.9 - 2.8)	2.3	(2 - 2.6)	1.4	(1.1 - 1.8)
HbA1c (+medications) without	36.9	(36.7 - 37.2)	36.1	(35.6 - 36.6)	38.2	(38 - 38.4)	37.5	(37.1 - 37.8)
HbA1c (+medications) without	1.4	(1.2 - 1.7)	2.6	(2.1 - 3.1)	0.8	(0.6 - 1)	1.7	(1.3 - 2.1)
HbA1c without	37.1	(37.1 - 36.8)	36.2	(36.2 - 35.8)	38.2	(38.2 - 38)	37.6	(37.6 - 37.3)
HbA1c without	1.3	(1 - 1.5)	2.4	(2 - 2.9)	0.8	(0.6 - 1)	1.6	(1.2 - 1.9)

\*For LLTI, the first observation year is 1996 instead of 1993, hence for that year years with LLTI + years without LLTI do not sum up to total life expectancy.