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The effect of risk factors on disability

Reuser, M.

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Chapter 3

3. Smoking kills, obesity disables

Abstract

Increasing BMI causes concerns about the consequences for health-care. Decreasing cardiovascular mortality has lowered obesity related mortality, extending duration of disability. We hypothesized increased duration of disability among overweight and obese individuals.

We estimated age, risk and state dependent probabilities of ADL disability and death and calculated multistate life tables, resulting in the comprehensive measure of life years with and without ADL disability. We used prospective data of 16,176 white adults of the Health and Retirement Study (HRS). Exposures were self-reported BMI and for comparison smoking status and levels of education. Outcomes were years to live with and without ADL disability at age 55. The reference categories were high normal weight (BMI 23-24.9), non smoking and high education.

Mild obesity (BMI 30-34.9) did not change total life expectancy (LE) but exchanged disabled for disability free years. Mild obesity decreased disability free LE with 2.7 [95% confidence limits 1.2:3.2] year but increased LE with disability with 2.0 [0.6:3.4] years among men. Among women, BMI 30-34.9 decreased disability free LE with 3.6 [2.1:5.1] years but increased LE with disability with 3.2 [1.6:4.8] years. Overweight (BMI 25-29.9) increases life expectancy with disability for women only, by 2.1 [0.8:3.3] years). Smoking compressed disability by high mortality. Smoking decreased LE with 7.2 years, and LE with disability with 1.3 [0.5:2.5] years (men), 1.4 [0.3:2.6] years (women). A lower education decreased disability free life, but not duration of ADL disability. In the ageing baby boom population, higher BMI will further increase care dependence.

Introduction

In 2001-2004, 30% of men and 34% of women in the U.S. aged 20-74 year were obese (BMI 30 and over) (Centers for Disease Control and Prevention 2006). Many countries follow the U.S. lead (York et al. 2004). Several studies suggest that obesity could overtake smoking as actual cause of death (Mokdad et al. 2004; Peeters et al. 2003a). However, more recent

studies suggest otherwise, demonstrating that at middle and old age, overweight lowers mortality, especially for men (Bonneux and Reuser 2007; Flegal et al. 2005; McGee 2005; Reuser, Bonneux and Willekens 2008). The so called obesity epidemic is put into question (Basham and Luik 2008; Jeffery and Sherwood 2008). Even mild obesity does not increase the hazard to die among the 65 and older (Al Snih et al. 2007). The major obesity related cause of death, cardiovascular mortality, has been spectacularly declining, partially due to improved therapies and cardiovascular risk management (CDC 1999; Gregg et al. 2005; Lawlor, Lean and Sattar 2006; Wild and Byrne 2006). In Norwegian surveys, the relative risk of death of diabetes, one of the main health consequences of obesity remained constant over time, but the absolute risk halved in a single decade (Dale et al. 2008). Obesity is related to increased blood pressure, dyslipidemia and diabetes. These can now be controlled, but at a cost (Gregg et al. 2005; Lawlor et al. 2006; Wild and Byrne 2006). High weight increases the mechanical stress on joints, particularly knee and back, increasing back pain and osteoarthritis and limiting mobility (Sach et al. 2007; Sturmer, Gunther and Brenner 2000). If disability is increased but not mortality, numbers of obese survivors expand morbidity, increasing life years lived with disability, care dependence and health-care costs (Andreyeva et al. 2004; Reynolds et al. 2005).

We describe the disability free life expectancy and expected duration of disability at age 55 in a recent large U.S. prospective study of the middle aged and the elderly (Juster and Suzman 1995), using multistate life tables. To classify weight we use self-reported BMI and we compare the effects of BMI with the other important risk factors smoking and educational attainment. The life table translates hazard rates into transition probabilities and calculates life expectancies with or without disability, conditioned by risk factors.

Population and methods

We used the RAND user-friendly version F of the Health and Retirement Study (HRS) data file containing the HRS and the Asset and Health Dynamics Among the Oldest Old (AHEAD) which began in 1992 and 1993, respectively, and were merged in 1998 (Juster and Suzman 1995). The HRS and AHEAD surveys include a nationally representative sample of initially non-institutionalized persons born in 1931-1941 (HRS, aged 51-61 in 1992) and in 1923 or earlier (AHEAD, aged 70 and older in 1993). Sampled persons were re-interviewed

biannually and the sample was replenished in 2004 by individuals aged 51 to 56 that year. Response was on average 86% (HRS) and 90% (AHEAD). The combined dataset is also called HRS, to which we will refer from now on. We selected white non-Hispanic men and women of whom date of birth, gender, level of education, Body Mass Index (BMI), smoking status, and ADL score were available. Data on vital status and month and year of death are obtained through the mortality register (the National Death Index) and exit interviews. Only 1% of the population (166/16,176 individuals) experienced recovery. Ignoring recovery simplified the multistate life tables without changing the results. Our sample covers survey rounds from 1992 to 2004.

Self-reported weight and height at baseline are used to calculate BMI (kg/m^2), classified as low normal weight (18.5-22.9), high normal weight (23-24.9), overweight (25-29.9), mildly obese (30-34.9) and severely obese (35+). We split normal weight into two classes, divided at BMI 23, because previous analyses suggested important heterogeneity at middle age (Reuser et al. 2008). We excluded underweight (BMI < 18.5), not being part of our study of normal and excess weight. We use the first length and weight reported. To avoid confounding by individuals with very bad health conditions at baseline, we start counting exposures and events after three years follow up. Smoking status is included as ‘never smoked’, ‘stopped smoking’ and ‘currently smoking’ based on the first reported information on smoking status. We distinguish three groups of educational attainment: Less than high school or General Educational Development (GED), High school graduate, and College graduate and above.

Outcomes are all-cause mortality and disability. Disability is defined by the Katz basic activities of daily living (ADL): Walking, Bathing, Dressing, Toileting and Feeding. (Katz et al. 1963) We classify as ADL disabled anyone answering ‘with difficulty’ to at least one of the ADL items.

We estimated the hazard rates of transitions to death and disability by age for each determinant of interest and for males and females. We estimated Cox proportional hazard ratios by BMI, smoking and education. We chose the Cox model because it is commonly used to assess the effects of risk factors on hazard (transition) rates and because it does not make any assumption on the baseline. Age is used as the timescale for the baseline hazard, accounting for left truncation and right censoring. Schoenfeld residuals with significance

level set at 5% tested the proportionality assumption, which was met for all transitions. To calculate life tables, transition rates are required and exponential smoothing using Poisson regression is applied to reduce the variability of the rates (Mamun 2003). The assumption is that the hazards of death and disability increase exponentially with age, which fitted the transition rates to death and disability very well. The effect of choice of model (Cox or Poisson) on the outcome is small as the increase with age is close to exponential. For ADL disability we assume transitions halfway between two waves. To include covariates in estimating the rates we used both univariate models, stratified by one risk factor of interest, and multivariate models correcting for all risk factors. In order to describe the burden of mortality and disability of BMI, smoking and education we defined multistate life tables by the estimated transition rates. To translate the rates in annual probabilities we assume the rates to be constant in the 1- year intervals because the assumption of transitions halfway the interval cannot be incorporated in the Cox or the Poisson model. The assumption of (piecewise) constant rates can. The main outcomes are total life expectancy, life years with and life years without ADL disability at age 55. Confidence intervals for the life expectancies and differences in life expectancies were calculated using bootstrapping with 250 replicates. Constructing confidence intervals with 1,000 replicates was much more computer-intensive and resulted in negligible differences.

Table 1: Selection of the sample.

	men	women	total
Initial sample	13086	17110	30196
Non-whites	2405	3535	5940
Hispanics	803	1099	1902
BMI < 18.5	93	461	554
Aged < 55	1067	1665	2732
Participated less than 3 year after first report of BMI	1467	1328	2795
Missing data on BMI, smoking or ADL disability	56	41	97
Final sample	7195	8981	16176

Results

The selection of non-Hispanic white individuals aged 55 and over, who participated at least 3 years and reported BMI, smoking, education and ADL resulted in a sample of 16,176 individuals. The selection is shown in Table 1. The distribution of population, exposures and

deaths by sex, age-groups and covariates is shown in Table 2. 30% of males and 36% of females had difficulties performing one of the ADL at some point during observation and were classified as disabled.

Table 2: Population, exposure and deaths by sex, age and risk factor status at baseline.

	Number of individuals			Person Years			Deaths		
	Males	Females	Total	Males	Females	Total	Males	Females	Total
Total	7195	8981	16176	41906	52268	94174	1600	1599	3199
low normal weight	885	2532	3417	4938	14605	19543	303	524	827
normal weight	1335	1668	3003	7872	9815	17687	314	312	626
overweight	3594	3033	6627	21115	17738	38853	730	504	1234
mildly obese	1089	1213	2302	6346	7115	13461	200	174	374
severely obese	292	535	827	1635	2996	4631	53	85	138
never smoked	1872	4633	6505	11164	26865	38029	291	878	1169
stopped smoking	3825	2719	6544	22166	15682	37848	917	447	1364
currently smoking	1498	1629	3127	8577	9721	18298	392	274	666
low education	1965	2321	4286	11069	13196	24265	637	656	1293
middle education	2106	3431	5537	12471	20537	33008	456	533	989
high education	3124	3229	6353	18366	18536	36902	507	410	917
55-64 *)	4036	4768	8804	18147	22105	40252	237	161	398
65-74	1364	1571	2935	12889	12930	25819	364	211	575
75-84	1502	2043	3545	8441	12345	20786	585	552	1137
85+	293	599	892	2429	4888	7317	414	675	1089

*) For number of individuals, the age at entry into observation is used (baseline + 3 year)

Relative risks

A Cox hazard regression model shows the proportional hazards for transitions to death and to ADL disability (defined as failing at least one basic ADL) by BMI, smoking status and levels of education. Table 3 shows both the univariate and multivariate proportional hazard ratios (PHR). When adjusted for the other covariates, some risk factor effects become smaller, e.g. the increased mortality risk for low normal weight men is partly taken over by smoking.

A higher BMI increases the hazard of ADL disability. For mildly obese men and women the PHRs were respectively 1.69 [1.37:2.09] and 1.66 [1.37:2.00] in the multivariate analysis. There was a clear dose response relationship, with increasing obesity causing increasing hazards of disability.

Table 3: Proportional hazard ratios (PHR) of BMI, smoking and education (uni- and multivariate analysis).

	Males				Females			
	Healthy to Death	Healthy to disabled	ADL disabled to Death	ADL disabled to Death	Healthy to disabled	ADL disabled to Death	ADL disabled to Death	
Univariate analysis								
BMI 18.5-22.9	1.39 (1.15; 1.68)	1.18 (0.94; 1.47)	1.46 (1.16; 1.84)	1.09 (0.91; 1.30)	1.01 (0.86;1.19)	1.10 (0.92;1.31)		
BMI 23-24.9 *	1.00	1.00	1.00	1.00	1.00	1.00		
BMI 25-29.9	0.95 (0.82; 1.11)	1.05 (0.89; 1.25)	0.98 (0.80; 1.20)	1.00 (0.83; 1.21)	1.28 (1.10;1.50)	0.75 (0.62;0.89)		
BMI 30-34.9	1.13 (0.91; 1.40)	1.72 (1.39; 2.13)	1.02 (0.79; 1.32)	1.07 (0.83; 1.38)	1.68 (1.40;2.03)	0.76 (0.60;0.96)		
BMI 35+	1.55 (1.06; 2.27)	2.61 (1.88; 3.61)	0.90 (0.61; 1.34)	1.88 (1.33; 2.66)	2.88 (2.28;3.65)	0.99 (0.73;1.34)		
Never smoked *	1.00	1.00	1.00	1.00	1.00	1.00		
Stopped smoking	1.53 (1.31; 1.80)	1.08 (0.93; 1.26)	1.11 (0.92; 1.33)	1.07 (0.92; 1.24)	0.99 (0.88;1.12)	1.11 (0.96;1.29)		
Currently smoking	3.20 (2.66; 3.84)	1.50 (1.24; 1.81)	1.41 (1.12; 1.78)	2.30 (1.92; 2.77)	1.53 (1.32;1.77)	1.75 (1.45;2.13)		
Low education	1.64 (1.42; 1.89)	1.80 (1.55; 2.10)	1.22 (1.03; 1.45)	1.61 (1.36; 1.90)	1.65 (1.45;1.88)	1.27 (1.09;1.48)		
Medium education	1.31 (1.13; 1.51)	1.35 (1.15; 1.58)	1.09 (0.90; 1.32)	1.13 (0.96; 1.33)	1.10 (0.96;1.25)	1.10 (0.93;1.30)		
High education *	1.00	1.00	1.00	1.00	1.00	1.00		
Multivariate analysis								
BMI 18.5-22.9	1.25 (1.04;1.51)	1.11 (0.89;1.39)	1.42 (1.12;1.79)	1.05 (0.88;1.26)	0.98 (0.83;1.15)	1.13 (0.94;1.34)		
BMI 23-24.9*	1.00	1.00	1.00	1.00	1.00	1.00		
BMI 25-29.9	0.95 (0.82;1.11)	1.04 (0.87;1.24)	0.98 (0.80;1.20)	0.99 (0.83;1.20)	1.25 (1.07;1.46)	0.75 (0.63;0.90)		
BMI 30-34.9	1.17 (0.94;1.45)	1.69 (1.37;2.09)	1.01 (0.78;1.31)	1.07 (0.83;1.39)	1.66 (1.37;2.00)	0.79 (0.62;1.00)		
BMI 35+	1.62 (1.11;2.36)	2.54 (1.84;3.52)	0.91 (0.61;1.36)	1.86 (1.31;2.62)	2.81 (2.22;3.56)	1.07 (0.79;1.44)		
Never smoked*	1.00	1.00	1.00	1.00	1.00	1.00		
Stopped smoking	1.50 (1.28;1.76)	1.06 (0.91;1.23)	1.12 (0.93;1.34)	1.09 (0.94;1.27)	1.01 (0.90;1.15)	1.13 (0.97;1.31)		
Currently smoking	2.95 (2.45;3.56)	1.43 (1.18;1.74)	1.34 (1.06;1.69)	2.31 (1.92;2.78)	1.60 (1.38;1.86)	1.69 (1.39;2.05)		
Low education	1.46 (1.27;1.68)	1.69 (1.45;1.97)	1.18 (0.99;1.40)	1.53 (1.30;1.81)	1.49 (1.31;1.71)	1.31 (1.12;1.53)		
Medium education	1.26 (1.09;1.45)	1.32 (1.13;1.54)	1.09 (0.90;1.32)	1.11 (0.95;1.31)	1.04 (0.92;1.19)	1.11 (0.94;1.32)		
High education*	1.00	1.00	1.00	1.00	1.00	1.00		

* Reference category. bold figures are significant at p=0.05

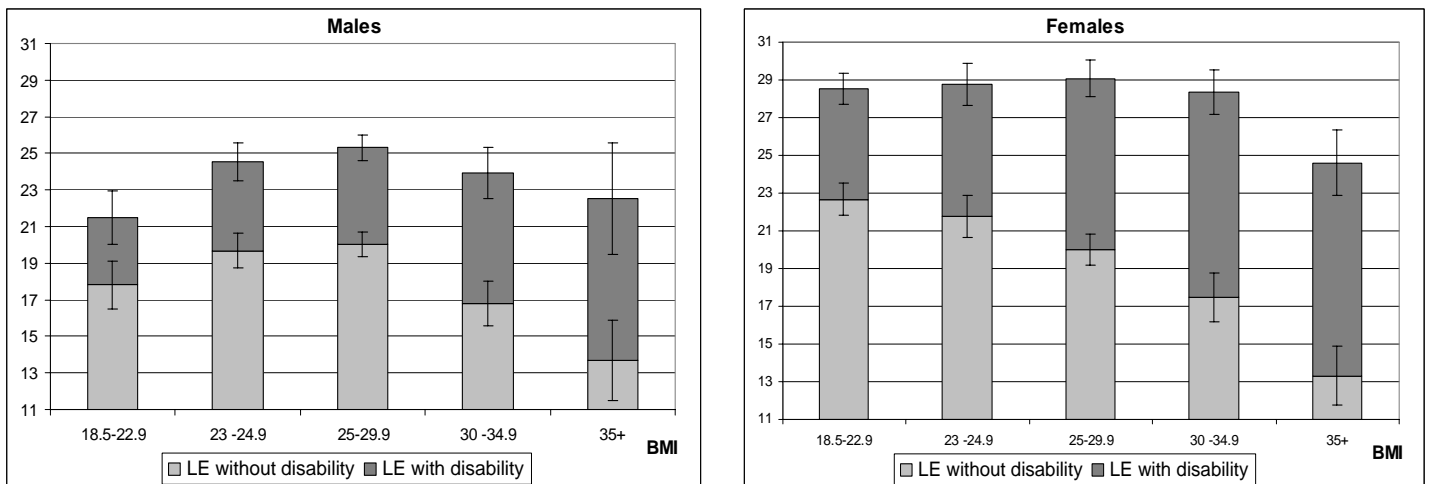
Table 4: Life years yet to live in total, with or without disability at age 55, with gains or losses by risk categories, adjusted for each other.

	Males			Females		
	Total life expectancy	Non-disabled Life	Disabled Life	Total life expectancy	Non-disabled Life	Disabled Life
BMI 18.5-22.9	-1.8(-3.2; -0.4)	-1.1(-2.5; 0.2)	-0.7(-1.8; 0.5)	-0.4(-1.6; 0.8)	0.1(-1.3; 1.5)	-0.5(-1.6; 0.6)
BMI 23-24.9 *	Ref (24.5)	Ref (19.5)	Ref (5.1)	Ref (28.7)	Ref (21.8)	Ref (6.9)
BMI 25-29.9	0.6(-0.6; 1.8)	0.2(-0.9; 1.3)	0.4(-0.6; 1.4)	0.6(-0.7; 1.8)	-1.5(-2.9; -0.1)	2.1(0.8; 3.3)
BMI 30-34.9	-0.6(-2.3; 1.0)	-2.7(-4.2; -1.2)	2.0(0.6; 3.4)	-0.4(-2.0; 1.1)	-3.6(-5.2; -2.1)	3.2(1.6; 4.8)
Never smoked *	Ref (27.6)	Ref (21.4)	Ref (6.2)	Ref (30.4)	Ref (22.0)	Ref (8.4)
Stopped smoking	-2.8(-4.0; -1.6)	-1.9(-3.0; -0.8)	-0.9(-1.8; 0.3)	-1.6(-2.7; -0.5)	-0.7(-1.9; 0.4)	-0.9(-2.0; 0.1)
Currently smoking	-7.7(-9.0; -6.4)	-6.4(-7.6; -5.2)	-1.3(-2.5; -0.5)	-6.6(-7.8; -5.4)	-5.2(-6.3; -4.1)	-1.4(-2.6; -0.3)
Low education	-2.8(-3.9; -1.8)	-3.6(-4.6; -2.5)	0.7(-0.1; 1.5)	-3.2(-4.2; -2.2)	-3.3(-4.4; -2.1)	0.0(-1.0; 1.0)
Medium education	-1.7(-2.8; -0.6)	-2.1(-3.2; -1.0)	0.4(-0.5; 1.2)	-1.0(-2.1; 0.1)	-0.4(-1.5; 0.6)	-0.6(-1.5; 0.4)
High education *	Ref (26.2)	Ref (21.1)	Ref (5.1)	Ref (30.1)	Ref (21.9)	Ref (8.2)

* Reference category. Between parentheses the life years yet to live in total, with or without ADL disability. bold figures are significant at $p < 0.05$

For men, overweight did not increase the risk of disability, but for women it did (PHR 1.25 [1.07:1.46]). Overweight or mildly obese women also face lower hazards of death once disabled, extending life with disability. Smoking and low education both increase risks of ADL disability and death for both men and women. No interaction effects between the other risk factors were significant, the disability hazards by BMI being similar for smokers and non-smokers and for different levels of education. However, the increased risk of death at low normal weight was predominantly observed among current smokers.

Figure 1: Life expectancy with and without ADL disability at age 55 (univariate analysis). Error bars represent 95% confidence intervals of disability free and total life expectancy.



Life expectancy

Translating age, sex and risk factor-specific transition rates (univariate) into life expectancies at age 55 shows the stratified life expectancy with and without disability for each risk group (Figure 1 for BMI). The actual life expectancy of the total unselected white American population in 1997 was 23.6 for men and 27.7 for women at age 55 (Centers for Disease Control and Prevention and National Center for Health Statistics 2006). The comparable life expectancy of our study population was respectively 24.0 and 28.2 years (excluding underweight individuals). A BMI between 18.5 and 29.9 showed the longest life expectancy free from disability for women and for men between 23 and 29.9. Mild obesity (BMI 30-

34.9) did not shorten total life expectancy but, at age 55, mild obesity shortened disability free life with 2.9 [1.4:4.4] years for males and 4.3 [2.6:6.0] for females compared to high normal weight (BMI 23-24.9). Severely obese men live on average 6.0 [3.6:8.4] years shorter free from ADL disability and women 8.4 [6.5:10.4] years. For men, low normal weight (BMI 18.5-22.9) lowers both total and disability free life expectancy.

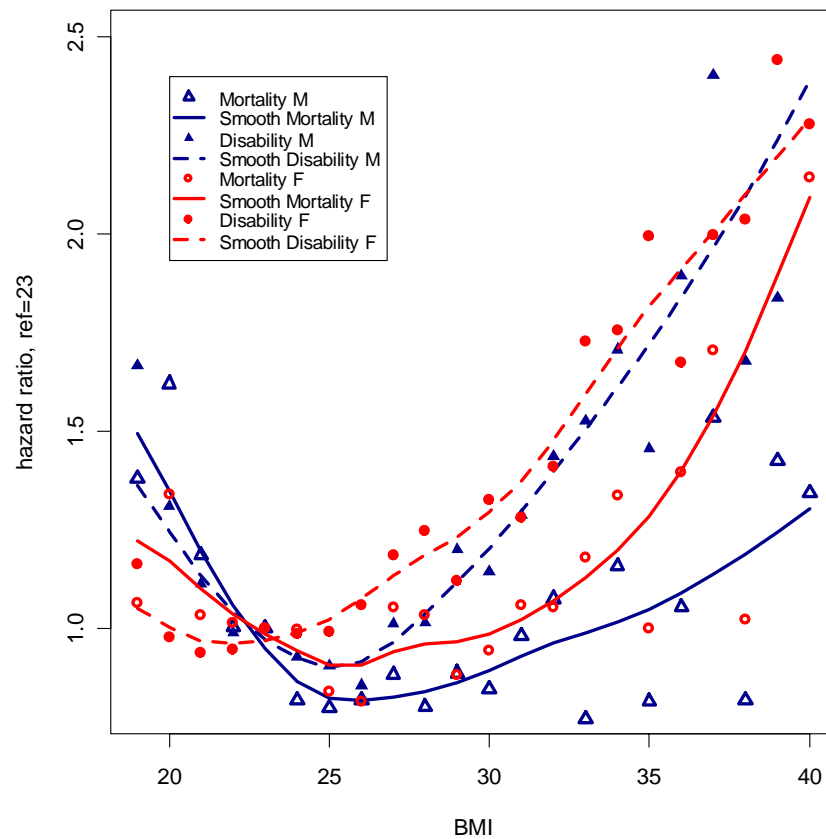
Table 4 shows the gains or losses in disabled and disability-free life expectancy in the multivariately adjusted multistate life table compared to the reference risk category. Differences between the univariate and multivariate life table results are small. Among men, a BMI 30-34.9 compared to BMI 23-24.9 decreased disability free LE with 2.7 [1.2:4.2] year and increased LE with disability with 2.0 [0.6:3.4] years. Among women, a BMI 30-34.9 compared to BMI 23-24.9 decreased disability free LE with 3.6 [2.1:5.2] year and increased LE with disability with 3.2 [1.6:4.8] years. Among women, overweight (BMI 25-29.9) compared to BMI 23-24.9 increased life expectancy with disability with 2.1 [0.8:3.3] years. The sample of severely obese persons was too small to calculate confidence intervals in this multivariate model. Therefore, these results have been omitted. Negligible differences in total life expectancy hid large and significant decreases in disability free life expectancy and increases in life expectancy with ADL disability. In the male HRS cohort, mild obesity increased duration of disability with 40 %, in the female HRS cohort, overweight and mild obesity increased duration of disability respectively with 30% and 46%.

The effect of smoking is very different. Smoking shortens both life expectancy free of disability (6.4 years [5.2:7.6] among men and 5.2 years [4.1:6.3] among women) and years lived with disability (1.3 years [0.5:2.5] and 1.4 years [0.3:2.6]). These results add to previous life course analyses showing decreased health-care costs and cardiovascular morbidity as a consequence of the high mortality of smoking (Barendregt et al. 1997; Mamun et al. 2004). A lower level of education decreases total life expectancy, but does not change life expectancy with disability.

Figure 2 illustrates the differences in hazard ratios by BMI between both sexes, disability and mortality that generated the increased life expectancies with ADL disability. The shape of mortality and disability by BMI is remarkably different, disability being far more BMI dependent than mortality. Among men, mortality shows a broad U-shape with increasing

BMI, increasing at the extremes only, while disability shows a sharp V-shape. A BMI with lower disability risks would be between 22 and 28. Among women, mortality increases earlier (at around BMI 33) and higher, while a BMI with lower disability would be between 20 and 26, two points lower than men. The BMI effect on disability was not different between smokers and non-smokers. Among individuals reporting poor or fair health, BMI had little effect on disability and mortality, among individuals reporting good or excellent health, the correlation was higher.

Figure 2 Mortality and disability hazard ratios for males and females, reference is BMI=23. Lines are discrete splines weighted by personyears ($\lambda=10^{-4}$ and $d=2$).



Discussion

The multistate life table can combine risk- and age-specific incidence and mortality rates into a single outcome, which is the sojourn time in the state reached after incidence e.g. years lived with ADL disability. This allows one to assess changes in the duration of disability, and hence care needs. As in many other more recent cohorts (Al Snih et al. 2007; Flegal et al.

2005; Reynolds et al. 2005), obesity in the white HRS population, men and women of 55 and older, did not shorten total life expectancy at levels of BMI under 35. But high BMI traded life years free from disability for life years with disability. Mild obesity shortened life free from ADL disability with 2.7 years (men) and 3.6 years (women), but increased the duration of ADL disability with 2.0 years (men) and 3.2 years (women). For females, even overweight increases disabled life expectancy by 2.1 years and shortens life free from ADL disability by 1.5 years.

The HRS results disagreed with earlier multistate life tables analyses, with disability and mortality taken from the long standing Framingham Heart Study cohort (Peeters et al. 2003a; Peeters et al. 2004). Peeters et al (2004) found no significant difference between life expectancy with disability of obese, overweight and normal weight individuals in this much older cohort (followed since 1948). The likely reason is the high cardiovascular mortality in the Framingham Heart Study cohort, a cohort that lived through unrestrained high cardiovascular mortality in the 1960s and 1970s. Indeed, obesity decreased life expectancy with 6 years. Lowered cardiovascular mortality, partly by successful risk management changed life expectancy tremendously in more recent cohorts (CDC 1999; Dale et al. 2008; Gregg et al. 2005). EPESE (Established Populations for Epidemiologic Studies of the Elderly) and the same HRS study documented loss of ADL disability free life expectancy among the obese (Al Snih et al. 2007; Reynolds et al. 2005). The older HRS analysis had the disadvantage of a shorter follow-up with few transitions, limiting the potential for a more refined analysis (Reynolds et al. 2005). The EPESE study described people of 65 and older (Al Snih et al. 2007), where we started at age 55.

The study of Dierh et al also studied population of 65 and older and documented largely the same findings, showing worse outcomes among the underweight but not the overweight and obese (Diehr et al. 2008). The sample was smaller, older, and more selected for health. The study started with an entire population of on average 75 years old. Life expectancy was higher (respectively 22 and 18 years among women and men at age 65) and life expectancy with ADL disability was shorter (respectively 5 and 3 years among women and men at age 65). Obese people did live longer with ADL disabilities, but fewer transitions in a smaller sample lacked statistical power to demonstrate these differences.

Obesity is associated with several potential disabling, but non-fatal conditions, such as osteoarthritis of the weight-bearing joints and chronic back pain (Must 1999; Naumann Murtagh 2004). Muscle strength declines with increasing adiposity (Al Snih et al. 2007). The male/female differences are striking. Men and women report disability accurately, women's higher incidence of disability is likely true (Merrill et al. 1997). In the HRS, being female was independently associated with decreased strength and mobility, and the positive association of BMI with mobility difficulty was significantly worse for women than for men (Wray and Blaum 2001). This positive association may be explained by the higher prevalence of osteoarthritis, lower back pain and smaller muscle mass among women than for men, non-fatal conditions which are worsened by obesity (Lean, Han and Seidell 1998).

We used self-reported BMI, which tend to be underreported by 1 BMI point (Nawaz et al. 2001; Visscher et al. 2006). Systematic underreporting of higher BMI underestimates the absolute prevalences of overweight and obesity, and overestimates the true relative risks at higher BMI (the estimated risks for a population with an apparent BMI of 30 to 35 hold for a true BMI of 31 to 36). For comparative studies, the limited bias is acceptable (McAdams, van Dam and Hu 2007).

BMI is but a fair measure of adiposity and it does not reflect fat distribution (Visscher et al. 2001; Visscher et al. 2006). However, it is easy to measure and widely used in health policy. Like many other studies we used the BMI reported at entry into the survey (Al Snih et al. 2007; Reynolds et al. 2005). The follow up of the HRS survey is too short to assess the effect of duration and change by age and cohort, which might be important (Peeters et al. 2003b). Self-reported limitations on ADL compared to medical evaluation of activity performance have shown good correlations ($r=0.88$) (Hubert et al. 2002). Multistate life tables need a sufficient number of transitions between states in small age bands to allow for statistically meaningful calculations. Therefore, we had to limit the ADL states to one only, describing the inability to perform one basic ADL. Changing our definition of ADL disability to two basic ADL did not result in relative changes in the effect of obesity on disability.

Previously, Barendregt et al. and Nusselder et al. showed that smoking 'compressed' morbidity and health-care costs by 'expanding' mortality (Barendregt et al. 1997; Mamun et al. 2004; Nusselder et al. 2000). Comparable to these previous analyses, we found that

smoking shortened both disability free life and life with disability, the latter with 1.3 to 1.4 year. Education then showed a third scenario of compression or expansion of morbidity. Higher levels of education were correlated to higher life expectancy free of disability, but did not extend duration of disability.

The primary aim of this paper is observational and descriptive. We did not try to disentangle the intimate relationships of physical activity and obesity. Physical activity is not a confounder, being causally related to obesity. The limited data available on physical activity did not support a thorough analysis of physical activity as a cause of disability. Fatal or debilitating disease causing weight loss instead of the reverse, can never be fully excluded in observational studies. We assessed potential reverse causation by various sensitivity analyses concerning weight loss during observation and self-reported health at baseline. We recalculated the proportional hazards excluding individuals who lost 10% or more during follow-up or excluding those who reported poor health at baseline. None of these altered the results materially. Longer term weight loss was associated with an increased, not a decreased life expectancy.

The role of pre-existing diseases can confound the relation between BMI and mortality. Healthier people at baseline show lower mortality risks at lower BMI than less healthy people: among women, non-smokers, or those free from disease, the level of BMI related to lowest mortality is lower than among men, smokers or diseased (Adams et al. 2006; Calle et al. 1999; Kuriyama et al. 2004; Manson et al. 1995). Partly, this can be explained by the obesity paradox, a higher BMI extending survival among the less fit and diseased (McAuley et al. 2007). Partly, this can be an effect of confounding by a prudent life style. Non-smokers and people who watch their weight show other risk aversive behaviour, lowering mortality. There was no interaction between BMI, smoking and education as cause of disability. In the HRS population reporting poor or fair health (20% of population), BMI ceased to predict disability or mortality. For the population reporting good or excellent health at baseline, the correlation between increasing BMI and disability was stronger.

The obesity epidemic may be both exaggerated and underestimated (Basham and Luik 2008; Jeffery and Sherwood 2008). The burden of spoiled years by obesity is now more important than the burden of lost years. Overweight and mild obesity ceased to be fatal, but a

paradoxical consequence of lowered mortality is increased disability and care dependence. This holds particularly among women for whom increased disability goes hand in hand with increased survival, both sharply increasing the numbers of years lived with disability. Care needs increase even more, as heavy people with disabilities are more difficult to handle. Fewer smokers and more quitters are another paradoxical source of future disability, adding life years with care dependence. The combination of ageing and increasing population BMI will increase long term care needs severely. Less smoking will increase care dependence, too, but expands life free from disability even more and extends the healthy life of potential care givers. Happily, higher levels of education deliver truly a win-win situation: high levels of education extend healthy life and compress disabled life as a share of total lifespan.

In the recent past, technological innovation has decreased cardiovascular mortality and extended the lives of many overweight and obese people. This is obviously a great success. The failure of success is that the extended survival is increasing care dependence. ADL disability is relatively easy to measure and to interpret: extending disability free life may be a new worthy target. The mortality consequences of the obesity epidemic have been exaggerated, but the consequences for long term care needs are severely underestimated.

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