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Lifestyle Habits and Compression of Morbidity

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Background. There has been much debate regarding the degree to which healthy lifestyles can increase longevity and whether added years will be offset by increased morbidity at older ages. This study was designed to test the compression of morbidity hypothesis, proposing that healthy lifestyles can reduce and compress disability into a shorter period toward the end of life.

Methods. Functional status in 418 deceased members of an aging cohort was observed between 1986 and 1998 in relationship to lifestyle-related risk factors, including cigarette smoking, physical inactivity, and under- or overweight. Three risk groups were created based on the number of these factors at study entry. Disability scores prior to death were modeled for each risk group to compare levels and rates of change, as well as to determine if and when acceleration in functional decline occurred.

Results. The risk-factor-free group showed average disability scores near zero 10–12 years before death, rising slowly over time, without evidence of accelerated functional decline. In contrast, those with two or more factors maintained a greater level of disability throughout follow-up and experienced an increase in the rate of decline 1.5 years prior to death. For those at moderate risk, the rate of decline increased significantly only in the last 3 months of life. Other differences between groups provided no alternative explanations for the findings.

Conclusions. These results make a compelling argument for the reduction and postponement of disability with healthier lifestyles as proposed by the compression of morbidity hypothesis.

WITH the increase in longevity experienced in most western countries during this past century, there has been growing concern about the quality of life that will be experienced by seniors who live well beyond their seventh decade (1). Questions also remain as to whether healthy lifestyles can continue to increase longevity and whether added years will be offset by increased morbidity and disability at older ages. It has been argued that healthy lifestyles and other preventive measures now will exert their greatest influence by postponing the onset of debilitating morbidity while increasing the life span by only a small amount. This proposition, known as the compression of morbidity hypothesis, suggests that the net effect of primary prevention will be to reduce and compress disability into a shorter period toward the end of life, to decrease overall lifetime disability, and, consequently, to reduce the associated health care burden (2–4).

Studies have shown that healthy lifestyles are associated with less disability in an aging population (5–13) and that certain health habits including regular exercise and abstinence from smoking may increase average life span by several years (14–17). Earlier reports also suggest that the onset of disability can be postponed with a more beneficial risk factor profile (5) and that greater physical activity, in particular, may lead to a shorter period of disability at the end of life (17,18). The present study builds on these findings and directly tests the compression of morbidity hypothesis by examining physical disability in deceased members of a large cohort during their final years. The aims are to describe the longitudinal course of disability prior to death and to determine if those with healthier lifestyle habits experience less, as well as later, functional decline.

Methods

Study Population

In 1986, we initiated a longitudinal study of risk factors for physical disability in an older group of university alumni who were participating in a larger study of chronic disease directed by Dr. Ralph Paffenbarger (19). Six thousand ninety-five individuals who had been at school between 1939 and 1944 and were known through alumni records were sent an invitation to participate. Of those who were locatable (N = 5983), 2843 (47.5%) returned a study questionnaire. Respondents (average age = 68) were 77% male and 99% white, reflective of the student body at that time. Prior data showed that nonresponders compared to participants were 1.5 years older, more likely to smoke, and had more heart disease, pulmonary disease, cancer, and arthritis.

For these analyses, 2328 participants who provided longitudinal disability data (i.e., two or more questionnaires) were followed between 1986 and 1998. There were 418 deaths, including 73 women and 345 men whose observation times varied from 1.5 to 11.5 years.

Data Collection

Data on lifestyle habits, medical history, physical impairments, and health care utilization were collected annually by a mailed, self-administered questionnaire. Deaths were ascertained through the participants' families or friends as well as through the National and Social Security Death Indices. This combination of methods ensured more accurate mortality follow-up, particularly for those who no longer returned study questionnaires. Death certificates were also obtained. Of particular interest were lifestyle-related causes

due to coronary heart disease, stroke, lung cancer, and chronic obstructive pulmonary disease.

Physical disability was measured annually using the Health Assessment Questionnaire, a widely used and well-validated instrument for ascertaining functional status (20–23). Studies have shown correlations of 0.85 on repeat assessments and of 0.88 on comparison of scores obtained by questionnaire versus medical evaluation of activity (20). Individuals were asked how much difficulty they had (0 = no difficulty, 1 = some difficulty, 2 = much difficulty, and 3 = unable to do) in performing 20 activities comprising eight functional categories: dressing and grooming, arising, eating, walking, personal hygiene, gripping, reaching, and doing errands and chores. Each category was scored using the maximal response. The disability score, ranging from 0 to 3, was equal to the average of the eight category scores. Some difficulty in only one of the eight categories, the minimal level of disability, was scored 0.125. A score of 0.375 represented either complete inability in one domain or lesser difficulties in two or three categories. Thus, seemingly small numeric differences or changes in scores could have a major impact on function.

Chosen risk factors were those that were lifestyle-related, potentially modifiable, and associated with functional status in other studies of aging. They included body mass index (weight in kilograms divided by height in meters squared), cigarette smoking, and vigorous physical activity (jogging, brisk walking, swimming, bicycling, racquet sports, or exercise that worked up a sweat). Cigarette smoking, a lack of vigorous physical activity, and being under- or overweight (body mass index 20 or less, or more than 25 kg/m^2) were considered unhealthy attributes. The criteria for body mass were developed from data showing their association with greater disability (24). Early in this study, only "vigorous activities that worked up a sweat" were ascertained. Thus, the guideline of "30 or more minutes of moderate activity most days per week" could not be assessed. No vigorous activity, reported by 48% of these seniors, was therefore used to denote a less active lifestyle.

Three risk groups were created based on the number of unhealthy factors at study entry: none (low-risk), one (moderate-risk), and two or more (high-risk). This parameterization provided a simple approach to looking at the predictive power of characteristics as they occurred in combination with one another. No attempt was made to control for changes in risk status over time.

Statistical Methods

In keeping with the compression of morbidity hypothesis, spline regression models were fit to disability over time for each risk group. With this method, two straight lines were fit, joined at a point before death (called the "knot point"). Differing slopes for these two lines would imply different rates of change in disability. Compression of morbidity was tested by ascertaining if there was a steeper slope (acceleration in the rate of change) for the line segment closest to death compared with the earlier segment.

Generalized estimating equations (25) were used to fit the spline regression models. This approach was chosen to estimate parameters because it requires no assumptions about the distribution of scores and accounts for the varying number of data points per individual as well as the within- and between-person correlations. Models were fit allowing each 3-month time-point to be defined as the knot. The model with the largest explained variance determined the best-fitting knot point for the risk group.

Bootstrap methods (26) were used to statistically evaluate whether or not acceleration in disability was evident and with what confidence it occurred at the chosen time-point. For each risk group, spline regression on 2000 independent bootstrap samples was performed. The rate of change in disability (i.e., the slope) was compared before and after the identified knot point in each analysis. The proportion of times that the slope did not increase on the segment closest to death defined the one-sided p value testing compression of morbidity. The 2000 points also described the variability in the estimated time when a steeper slope occurred.

Analyses also were done by risk category for men separately. However, there were too few deaths to obtain statistically reliable estimates for women. Software packages included SAS version 6.12 (27) (SAS, Cary, NC) and S-Plus version 4.5 (28) (Mathsoft Inc., Seattle, WA).

Results

Of the 418 deaths, 81 were risk-factor–free, 184 had only one factor, and 153 had two or more factors at study entry. Average age at death (76 years) was the same among the groups. However, rates were 1.9 times greater in high-risk than low-risk participants (Table 1), unexplained by the 0.6-year age difference at study entry. When only lifestyle-related causes of death were considered, the difference increased 2.8 fold. Although there were no gender differences in rates in the high-risk group, low-risk women, who were 1

Table 1. Death Rates per 10,000 Person-Years by Risk Status: Participants Followed for Mortality From 1986–1998

	Low Risk (n = 612)			Moderate Risk (n = 1062)			High Risk (n = 654)			Total (N = 2328)		
Participant	Deaths	Person-Years	Rate (CI)*	Deaths	Person-Years	Rate (CI)*	Deaths	Person-Years	Rate (CI)*	Deaths	Person-Years	Rate (CI)*
Women (n = 533)	6	1305.5	46.0 (20.7–102.3)	26	2484.8	104.6 (71.2–153.7)	41	1815.8	225.8 (166.3–306.7)	73	5606.1	130.2 (103.5–163.8)
Men (n = 1795)	75	5147.4	145.7 (116.2–182.7)	158	8529.8	185.2 (158.5–216.5)	112	4744.3	236.1 (196.2–284.1)	345	18,421.5	187.3 (168.5–208.1)
Total (N = 2328)	81	6452.9	125.5 (101.0–156.1)	184	11,014.6	167.1 (144.6–193.0)	153	6560.1	233.2 (199.0–273.3)	418	24,027.6	174.0 (158.1–191.5)

Note: CI = confidence interval.

*95% confidence interval around the rate.

Table 2. Characteristics at Study Entry and at Death by Risk Status: Participants Who Died Between 1986 and 1998*

Characteristic	Low Risk (n = 81)	Moderate Risk (n = 184)	High Risk (n = 153)	Total (N = 418)
Male gender	92.6%	85.9%	73.2%	82.5%
Age at entry (y)	69.1 ± 0.5	69.5 ± 0.3	69.2 ± 0.4	69.3 ± 0.2
Age at death (y)	76.2 ± 0.6	76.3 ± 0.4	75.8 ± 0.4	76.1 ± 0.3
Lifestyle-related causes of death†	27.2%	40.2%	40.5%	37.8%
Disability score (0–3)	0.06 ± 0.02	0.11 ± 0.02	0.21 ± 0.03	0.14 ± 0.01
No disability (0 Score)	77.8%	67.9%	52.3%	64.1%
Body mass index (kg/m^2)	23.8 ± 0.1	24.8 ± 0.2	25.6 ± 0.4	24.9 ± 0.2
Cigarettes/day	0	0.9 ± 0.4	12.0 ± 1.5	4.8 ± 0.6
Ever smoked	55.6%	60.3%	79.1%	66.3%
Aerobic exercise (min/wk)	220.4 ± 23.4	74.9 ± 9.6	14.5 ± 5.1	81.0 ± 7.4
Alcoholic drinks per wk	7.0 ± 0.8	6.4 ± 0.6	6.4 ± 0.7	6.5 ± 0.4
Saturated fat (g/wk)‡	121.5 ± 11.5	109.7 ± 5.7	153.6 ± 8.5	128.0 ± 4.7
No. of major medical conditions (lifetime)	3.0 ± 0.2	3.1 ± 0.1	3.2 ± 0.2	3.1 ± 0.1
No. of hospital days (lifetime)	30.3 ± 6.6	26.3 ± 2.9	40.6 ± 9.1	32.3 ± 3.8
No. of doctor visits (past year)	6.3 ± 0.8	5.9 ± 0.5	7.9 ± 1.0	6.7 ± 0.4

*Characteristics are presented as a percent or mean ± the standard error, as indicated.

†Deaths due to heart disease, stroke, lung cancer, and chronic pulmonary disease.

‡Estimated from the number of servings of eggs, whole milk products, cream, cheese, ice cream, butter, and red meat only.

year younger than comparable men, had one-third the male death rate. Risk status appeared to have a greater impact on women than on men. High-risk women had five times the death rate of low-risk women compared with a 1.6-fold difference between these groups in men.

Seven percent of deaths in the low-risk group were among women, compared with 14% and 27% in the moderate and high-risk groups, respectively (Table 2). Although there were no age differences by group at baseline, initial disability doubled with risk from low to moderate and from moderate to high (trend test, $p = .001$). Saturated fat intake also showed a significant positive trend by risk group ($p = .001$). Health care utilization was greater in the high-risk group, but the trend was not significant ($p = .32$). However, the proportion of lifestyle-related deaths increased significantly by group (trend test, $p = .05$).

The spline regression models for disability prior to death are presented in Figure 1. Although the data were analyzed in a continuous fashion, average disability scores at 3-month intervals are superimposed on the fitted models. Irrespective of risk group, time-before-death was a significant predictor, with disability clearly increasing as death approached ($p < .0001$).

Under the compression of morbidity hypothesis, disability would be lower and acceleration in the rate of change (increased slope) closer to the time of death in a lower compared with a higher risk group. The fitted line for the risk-factor–free group shows disability scores starting near 0, rising steadily to approximately 0.4, but without an accelerated rate of functional decline in the final years (Figure 1, Table 3). Those in the high-risk group had greater levels of disability throughout, reached an average score of 1.3, and experienced a significant increase in the rate of decline 1.5 years prior to death. For those at moderate risk, a significant increase in the rate of decline occurred only in the last 3 months of life. Average disability reached 0.7, in between the low- and high-risk individuals.

Prior to acceleration near death, the rate of change in disability was similar for the three risk groups (slopes = 0.012, 0.012, and 0.013; Table 3). The difference, however, was the 1.5 to 3.0 times greater level of disability in the high-risk group evident years before the time of death. Thus, not only did the high-risk group experience an accelerated decline 1 to 2 years before death, but also continually maintained a greater level of disability.

Analyses of the bootstrap samples validated these results. For the low-risk group, 773 (of 2000) samples showed no increase in the rate of functional decline (one-sided p value = 0.387). In contrast, only five moderate-risk samples indicated no increase ($p = .003$). All high-risk replicates showed increased declines ($p = .000$), 95% of these within 2 years before death, and predominantly centered at 1.5 years. In the moderate-risk group, 77% of the point estimates were in the last 3 months of life.

The above analyses by risk category were repeated for men separately to eliminate the possible confounding effects of gender. High-risk men exhibited greater levels of disability than low- or moderate-risk men, and findings remained the same with regard to whether and when an increased rate of functional decline occurred in the risk groups.

Discussion

These results make a compelling argument for the compression of morbidity hypothesis, that is, the reduction and postponement of disability with healthier lifestyles. Those with fewer risk factors experienced less overall disability as well as delayed acceleration in functional decline before death. Individuals with two or more risk factors not only reached a greater level of disability earlier in life and maintained that increased level, but also experienced an earlier acceleration in functional decline, about 1.5 to 2 years prior to death. The delayed decline for those at moderate risk and the lack of any identifiable increase in the risk-factor–free

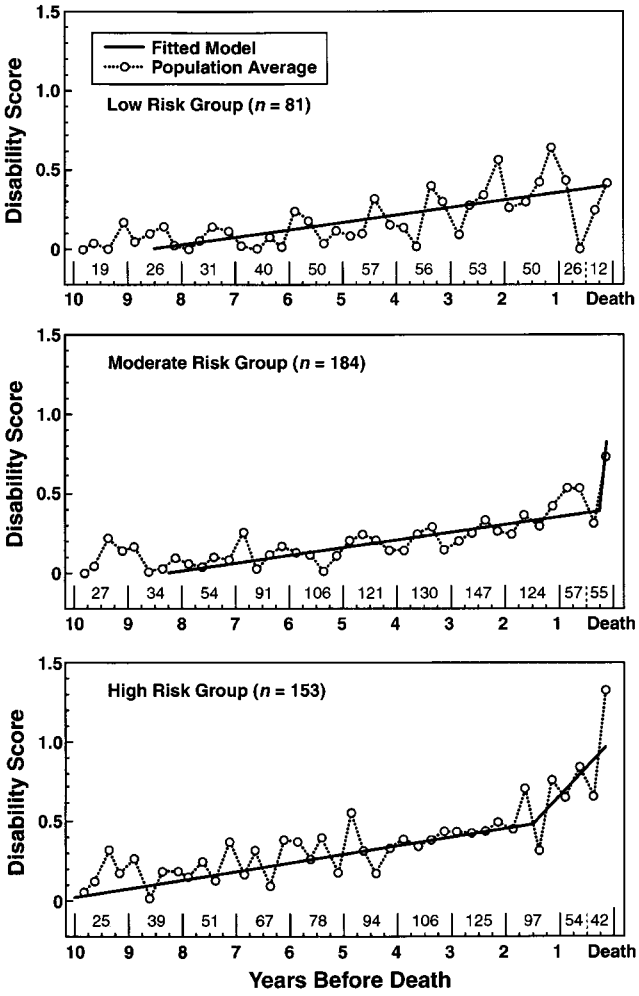

Figure 1. Average disability scores prior to death by risk group. Average scores are plotted within 3-month intervals along with fitted values from the regression models. Immediately above the x-axis are the actual numbers of individuals contributing disability data within a given year or half-year. These numbers are all smaller than the total for each risk group because participants died at different times following study entry and may not have completed a questionnaire within each time interval while alive.

group further illustrate the advantage of these lower risk groups.

With only 18% of the cohort deceased, there was no notable impact of risk group on longevity (0.4–0.5 year). Other studies suggest that differences may continue to be modest as the cohort ages, with healthier lifestyles or risk reduction adding a few years to the life span, on average, while increasing active-life expectancy (14,17,29). Thus, preventive health measures, including intervention on modifiable risk factors, have the potential to offset increases in disabling morbidity as well as the concomitant burden of care resulting from greater longevity among today's aging populations.

The lack of accelerated functional decline in the low-risk group is a key finding supporting the compression of morbidity hypothesis. If accelerated decline in this group occurred closer to the time of death than our 3-month window could detect, it would still argue in favor of a reduction and postponement of disability with healthier lifestyles. The smaller number of decedents who completed questionnaires in the last year of life may have made an increase more difficult to detect. However, the plot of disability (Figure 1) presents no obvious acceleration toward the end of life and, thus, is not consistent with this notion. Furthermore, comparisons of status in the last 3 months of life show no impairments (0 disability score) in 67%, 35%, and 13% of the low-, moderate-, and high-risk participants, respectively, illustrating that healthier lifestyles can prolong disability-free life expectancy.

Alternative explanations for these findings, including differences between groups in age, gender, ethnicity, and educational attainment, are not apparent. The study population is very homogeneous—college educated and 99% Caucasian with no differences between risk groups in average age at baseline or death. While homogeneity may limit the generalizability of these results, it has served to strengthen our ability to draw inferences from the risk group comparisons. Analyses that address the question of confounding by gender do not change the study findings.

With longer follow-up of this cohort, compression of morbidity related to modifiable health behaviors will continue to be a compelling question. Given that physical disability is the most prevalent major health problem of older adults in the United States (30,31), public health policies aimed at reducing this burden are of great importance. Understanding how risk behaviors can effectively decrease morbidity and slow functional decline will help focus initiatives to improve quality of life and diminish the health care burden in aging populations.

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Table 3. Rate of Change (Slope) and Acceleration in Disability by Risk Status: Results of Spline Regression Analyses

Risk Group	Average Disability Score, 3 Mo Before Death	Acceleration Point, Time Before Death	Rate of Change in Disability Over 3 Mo (Slope), Before Acceleration (95% CI)*	Rate of Change in Disability Over 3 Mo (Slope), After Acceleration (95% CI)*
Low	0.4	None found	0.012 (0.006–0.0017)	0.012 (0.006–0.017)
Moderate	0.7	3 months	0.012 (0.001–0.025)	0.958 (0.226–1.691)
High	1.3	1.5 years	0.013 (0.009–0.017)	0.089 (0.055–0.124)

Note: CI = confidence interval.
*95% confidence interval around the slope.

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