Grip strength changes over 27 yr in Japanese-American men

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[1]*Epidemiology, Demography, and Biometry Program, National Institute on Aging, National Institutes of Health, Bethesda, Maryland 20892; and* [2]*Honolulu Heart Program, Honolulu-Asia Aging Study, Kuakini Medical Center, Honolulu, Hawaii 96813*

Rantanen, T., K. Masaki, D. Foley, G. Izmirlian, L. White, and J. M. Guralnik. Grip strength changes over 27 yr in Japanese-American men. *J. Appl. Physiol.* 85(6): 2047–2053, 1998.—The aim of this study was to describe changes in grip strength over a follow-up period of ~27 yr and to study the associations of rate of strength decline with weight change and chronic conditions. The data are from the Honolulu Heart Program, a prospective population-based study established in 1965. Participants at *exam 1* were 8,006 men (ages 45–68 yr) who were of Japanese ancestry and living in Hawaii. At follow-up, 3,741 men (age range, 71–96 yr) participated. Those who died before the follow-up showed significantly lower grip-strength values at baseline than did the survivors. The average annualized strength change among the survivors was −1.0%. Steeper decline (>1.5%/yr) was associated with older age at baseline, greater weight decrease, and chronic conditions such as stroke, diabetes, arthritis, coronary heart disease, and chronic obstructive pulmonary disease. The risk factors for having very low hand-grip strength at follow-up, here termed grip-strength disability (≤21 kg, the lowest 10th percentile), were largely same as those for steep strength decline. However, the age-adjusted correlation between baseline and follow-up strength was strong ($r = 0.557$, $P < 0.001$); i.e., those who showed greater grip strength at baseline were also likely to do so 27 yr later. Consequently, those in the lowest grip-strength tertile at baseline had about eight times greater risk of grip-strength disability than those in the highest tertile because of their lower reserve of strength. In old age, maintenance of optimal body mass may help prevent steep strength decrease and poor absolute strength.

aging; muscle strength; prospective study; body composition; chronic diseases

WITH INCREASING AGE, muscle strength decreases and may eventually reach a level at which weakness starts to restrict the ability to perform usual activities (25, 36). In the very old, loss of muscle mass and strength are suggested to be important factors in the process of increased frailty (17). Most of the information about age-related strength changes is based on cross-sectional data (21). In previously published longitudinal studies, the participants have been a selected group of people, or the numbers have been small (1, 2, 12, 13), or the follow-up period has been fairly short (4).

Measurements made in large epidemiologic studies involving older participants have to be cost-effective for the researchers and not too burdensome to participants. Grip-strength tests are convenient, safe, and reliable, and they do not require large or expensive equipment (3, 28). Consequently, grip strength has been used as an indicator of overall muscle strength (4, 16). Use of grip strength as a feasible model to describe overall strength changes is supported by its significant ($P < 0.001$) correlations with other strength measures in older men [$r = 0.638$ for elbow flexion, $r = 0.524$ for knee extension, $r = 0.515$ for trunk extension, and $r = 0.437$ for trunk flexion (29)].

The Baltimore Longitudinal Study on Aging combined cross-sectional and longitudinal data. Hand-grip strength was found to increase up until the thirties and to start to decrease with accelerated speed after the forties (16, 23). Cross-sectionally, lower muscle strength was associated with lower muscle mass as measured by creatinine excretion (16). With aging, muscle mass is lost due to motoneuron death (23) and muscle cell shrinking due to inactivity (14). Also, hormonal changes, particularly decreases in testosterone and growth hormone levels, may be associated with muscle mass decrease (17). Diseases may cause decrease in strength through inactivity, or they may have a direct effect on muscle (4, 32). For example, in stroke, the injury in the central nervous system will affect the descending neural pathways and result in poor motor unit activation (22). However, great interindividual differences are evident in strength decline with increasing age. For example, Kallman et al. (16) found that over an average 9-yr follow-up period, 15% of the subjects ages 60 and over did not show any strength decline.

The aims of this study were *1*) to describe the extent of change in grip strength over a follow-up period of ~27 yr in a large group of initially 45- to 68-year-old Japanese-American men ($n = 8,006$) living in Hawaii and *2*) to study age, body weight changes, and morbidity as predictors of grip-strength change.

METHODS

The participants are from the Honolulu Heart Program and the Honolulu-Asia Aging Study which began in 1965. This program has been described earlier (35). Briefly, the World War II Selective Service Registration file was used to identify 12,417 possibly eligible men of Japanese ancestry (having a Japanese last name and/or listed as of Japanese origin) born from 1900 to 1919 and living in Oahu. These men were sent a questionnaire. In all, 1,269 men were not located, and 1,270 refused to answer the questionnaire. A further 1,692 men who answered the questionnaire refused to participate in the physical examinations, and 180 men who responded to the questionnaire died before they were

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scheduled to have the physical examination. In 1965–1968, 8,006 men participated in *exam 1*. *Exam 2* took place ~3 yr later, in 1968–1970, with 7,498 men participating. Hand-grip strength was measured in *exams 1, 2, 4*, and *5* (Fig. 1). If the person participated in both *exams 1 and 2*, the baseline strength level was determined as the average of the best result of these two. This averaged value represents the midlife strength. If the person participated in only one of these exams, the best result from that examination was chosen for the analyses. The follow-up measurements took place in 1991–1993 (*exam 4*, $n = 3{,}741$) and 1994–1996 (*exam 5*, $n = 2{,}705$). The old age (range, 71–96 yr) strength was determined correspondingly as an average of the maximum in these two later exams, unless the person had participated in only one. In that case, that one result was chosen for the analyses.

Hand-grip strength was measured by using the Smedley Hand Dynamometer (Stoelting, Wood Dale, IL). While each participant was sitting, he extended his arm in front of him on the table and gripped the dynamometer. If necessary, the tester held the dynamometer steady. The width of the handle was adjusted, so that, when the subject held the dynamometer, the second phalanx was against the inner stirrup. Three trials, with brief pauses, were allowed for each hand alternately. Subjects were encouraged to exert their maximal grip. The best result was chosen for analyses.

Body weight and height were measured during *exam 1* and *exam 4*, and values were expressed as kilograms and centimeters, respectively. To calculate body mass index (BMI), height was converted into meters (BMI = weight/height^2).

In *exam 1*, the upper arm circumference and triceps skinfold were measured with the subject standing, arm muscles relaxed, and arms hanging vertically at the side. Recordings were done to the nearest full millimeter. Upper arm circumference was measured by using a standard tape measure midway between the axilla and elbow, without applying excessive pressure. The skinfold thickness over the triceps muscle midway between the axilla and elbow was measured by using a Lange Skinfold Caliper (Cambridge Scientific Industries, Cambridge, MD). Longitudinal fold of skin and subcutaneous tissue was taken between the thumb and the forefinger without applying excess pressure or traction. Caliper tips were applied 1 cm below the fingertips.

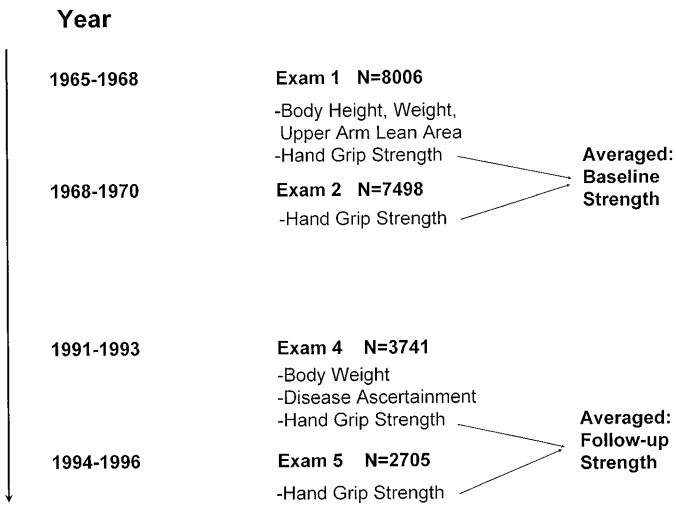

Fig. 1. Design of study; *n*, no. of study participants in each exam period.

Upper arm lean area was estimated from upper arm circumference and triceps skinfold thickness as follows

$$A_l = \pi/4 \cdot (C/\pi - \mathrm{Str})^2$$

where A_l is upper arm lean area, C is upper arm circumference, and Str is triceps skinfold.

The prevalence of angina pectoris, hypertension, coronary heart disease, cerebrovascular disease, chronic obstructive pulmonary disease (COPD), diabetes mellitus, and arthritis was ascertained in *exam 4*. Presence of angina or arthritis was based on self-report ("Has a doctor ever told you that you have...?"). Hypertension was verified if blood pressure was ≥160/95 mmHg or the participant was taking blood pressure medication. Three blood pressure measurements were done 5 min apart on the left arm of the seated subject with the use of a standard sphygmomanometer. The results were averaged. Diabetes was ascertained by asking subjects if they had a diagnosis of diabetes or if they used insulin or pills for diabetes, as well as with a help of a glucose tolerance test using the World Health Organization classification (31). Coronary heart disease was verified on the basis of hospital record surveillance, electrocardiographic findings, and questionnaire data (18). Stroke prevalence was based on hospital record surveillance. COPD was judged on the basis of questionnaire data (cough and phlegm lasting >3 mo consecutively or a physician's diagnosis of emphysema).

Statistical Methods

Means according to age group strata (5-yr groupings) at baseline were compared by using a one-way analysis of variance. The differences in strength at baseline between survivors and nonsurvivors were studied by *t*-tests. Pearson's correlations were used to study the association among age, strength, and anthropometric variables. Age-adjusted associations between grip strength and anthropometric variables were analyzed by using partial correlations. Strength changes over the follow-up in age strata were studied by using *t*-tests for paired samples. Cross-tabulation with χ^2 test was used to study the proportion of those with steep, average, and moderate strength decline in groups based on baseline age. Linear regression analysis was used to study age and weight change as predictors of rate of strength decline. Odds ratios (OR) for steep strength decline and grip-strength disability were computed by logistic regression analyses.

RESULTS

At *exam 1*, one-way analysis of variance showed that the older participants had significantly lower grip strength, body height, weight, BMI, and smaller A_l than the younger participants (Table 1). The age-adjusted correlations between baseline grip strength and anthropometric variables were as follows. *Exam 1*: body height, $r = 0.441$, $P < 0.001$; body weight, $r = 0.401$, $P < 0.001$; BMI, $r = 0.229$, $P < 0.001$; and A_l, $r = 0.347$, $P < 0.001$. The age-adjusted correlation between A_l and body weight was $r = 0.520$, $P < 0.001$.

Figure 2 shows grip strength according to age strata at baseline among those who survived to follow-up measurements, among the nonsurvivors, and among the survivors at follow-up. At baseline, in all age groups, those who died before the follow-up examinations exhibited lower grip strength than did the survivors ($P < 0.012$). However, in the nonsurvivors, the body weight was also on average 0.49 kg lower than in

Table 1. *Baseline height, weight, BMI, upper arm lean area, and grip strength according to baseline age groups*

Baseline Age Group, yr	*n*	Grip Strength, kg	Height, cm	Weight, kg	BMI, kg/m^2	Arm Lean Area, cm^2
45–49	1,831	41.7 ± 0.13	162 ± 0.13	65.7 ± 0.21	24.3 ± 0.07	54.3 ± 0.25
50–54	2,792	40.1 ± 0.11	161 ± 0.11	64.2 ± 0.17	24.0 ± 0.06	53.3 ± 0.19
55–59	1,590	37.9 ± 0.14	160 ± 0.14	63.0 ± 0.22	23.7 ± 0.08	51.1 ± 0.25
60–64	1,334	35.3 ± 0.15	158 ± 0.15	60.6 ± 0.24	23.3 ± 0.08	48.1 ± 0.27
65–68	450	33.3 ± 0.24	157 ± 0.26	59.8 ± 0.43	23.1 ± 0.15	47.6 ± 0.41
F, *P* value		<0.001	<0.001	<0.001	<0.001	<0.001

Values are means ± SE; *n*, no. of subjects. BMI, body mass index.

the survivors ($P = 0.025$). The intra-individual strength changes over time were significant in all age groups ($P < 0.001$). The age-adjusted correlation between baseline and follow-up strength was strong ($r = 0.557$, $P < 0.001$), indicating that those who showed greater grip strength at baseline were also likely to do so 27 yr later. Among the survivors, the absolute grip-strength change was calculated as the difference between the follow-up and the baseline strength. Figure 3 shows the distribution of the absolute strength change over time and demonstrates substantial decline in all age groups. Median change ranged from −9.0 kg in those 45- to 49-yr-old at baseline to −13.5 kg in those 65- to 68-yr-old at baseline.

The grip-strength change was annualized and expressed as percentage of baseline strength. Table 2 shows that average annualized relative decline, as well as the proportion of those with steep decline (>1.5%/year) in strength, was greater in the older men.

Weight change (*exam 4* weight − *exam 1* weight) was associated with age, with the older participants losing more weight ($r = -0.211$, $P < 0.001$). In linear regression analysis with adjustment for weight at *exam 1*, weight change (partial $R^2 = 0.073$) and age (partial $R^2 = 0.068$) were about equally strong independent predictors of annualized grip-strength change. This indicates that those who lost more weight also showed steeper strength decline regardless of their age. To elaborate this observation further, we divided participants into three groups on the basis of their weight change: large weight decrease (5 kg or more), moderate weight decrease (4.99–0.01 kg), or no decrease (including increase). For steep strength decline, those who lost ≥5 kg had OR of 3.24 and 95% confidence intervals (95% CI) of 2.44–4.33, whereas those who lost 4.99–0.01 kg of weight had OR of 1.51 and 95% CI of 1.09–2.09, respectively, compared with those who did not lose any weight.

Chronic diseases of participants were ascertained at *exam 4* (Table 3). The bivariate OR for losing >1.5% of grip strength/yr (adjusted for age and baseline grip strength) were as follows: stroke (OR = 3.44; 95% CI, 2.37–5.01), arthritis (OR = 1.49; 95% CI, 1.14–1.95), diabetes (OR = 1.88; 95% CI, 1.48–2.38), coronary heart disease (OR = 1.49; 95% CI, 1.20–1.82), and COPD (OR = 1.83; 95% CI, 1.09–3.07). Angina and cancer were not significantly more common among those with steep strength decline. Hypertension, however, was protective of steep decline (OR = 0.71; 95% CI, 0.57–0.88).

Finally, we wanted to study the predictors of having very low muscle strength in old age, termed here grip-strength disability. Grip-strength disability was defined as the lowest 10th percentile of old-age grip strength, with the cut-off point being 21 kg. The age-adjusted bivariate OR values are shown in Table 4. The risk of becoming grip-strength disabled was about eight times greater among those who at baseline had grip strength in the lowest tertile and about two times

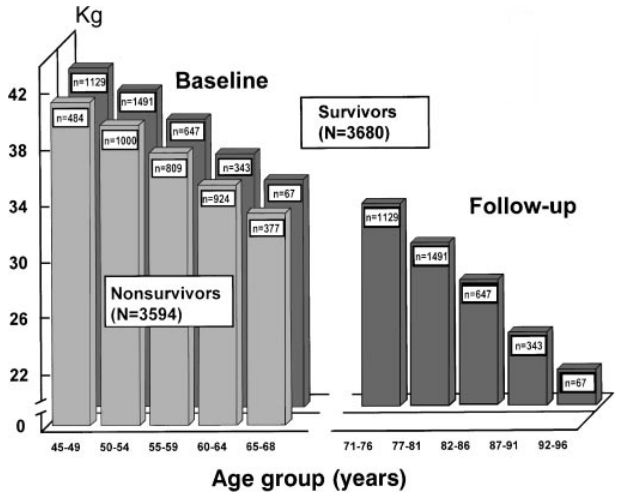

Fig. 2. Mean grip strength (in kg) according to age at baseline among nonsurvivors and survivors and at follow-up 27 yr later among survivors. Survivors in 45- to 49-yr-old age group at baseline are same individuals as in 71- to 76-yr-old age group at follow-up.

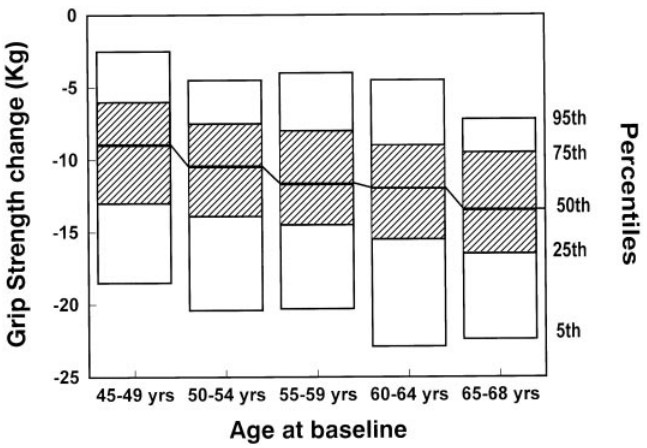

Fig. 3. Absolute change in grip strength over 27 yr according to baseline age. Percentiles are shown in each age stratum.

Table 2. *Annualized grip-strength change over 27 yr and proportion of subjects with steep, average, and moderate decrease according to baseline age category*

Baseline Age Group, yr	*n*	Annualized Change, %	Decreased ≥1.5%/yr, %	Decreased 1.49–0.51%/yr, %	Decreased ≤0.5%/yr, %
45–49	1,129	−0.85 ± 0.01	6	73	21
50–54	1,491	−0.99 ± 0.01	11	77	12
55–59	647	−1.10 ± 0.02	17	76	7
60–64	343	−1.31 ± 0.03	31	65	4
65–68	67	−1.49 ± 0.07	46	54	0
Total	3,677	−1.00 ± 0.008	13	74	13

Values of annualized change are means ± SE in %; *n*, no. of subjects.

greater for those in the middle tertile compared with those in the highest tertile at baseline. Participants in the lowest tertile in body height, body weight, and upper arm muscle area had approximately twice the risk compared with those in the highest tertile. The prevalence of arthritis, coronary heart disease, stroke, and diabetes was greater among those with grip-strength disability than among the rest of the participants. Hypertension, however, was protective of grip-strength disability. Weight change between *exams 1* and *4* as the predictor of grip-strength disability is illustrated in Fig. 4. The participants were stratified into tertiles according to their weight in *exam 1* to study whether weight loss would predict grip-strength disability differently in these tertiles. The reference group was those with *exam 1* weight in the middle tertile and no weight decrease. Regardless of initial weight, those who lost ≥5 kg had about five to six times the risk of grip-strength disability, even after adjusting for age, height, and baseline grip strength.

DISCUSSION

The aim was to study changes in muscle strength from middle to old age by using grip strength as the model. Altogether, 8,006 men (age 45–68 yr) participated in *exam 1*. At the end of the follow-up period (on average, 27 yr), 3,680 men (with ages that ranged from 71 to 96 yr) participated in grip-strength tests. No previous studies have presented strength data of a large group of people over such a long follow-up period.

The average annualized grip-strength decline was 1.0%/yr (SE 0.008). Earlier longitudinal studies have reported declines in average grip strength, ranging from 0.7 to 3%/yr (2, 4, 5, 28). The age groups, however, are not always comparable. In the present study, great interindividual variation in strength changes were found. Older people were more likely to show steeper decline than younger people. This result is consistent with previous studies (2, 4, 5). It has been suggested that strength peaks during the 30s and then starts to decrease gradually. The decrease becomes more pronounced after the 50s and 60s (16).

In previous studies of people, ages 65 yr and older, with follow-up periods varying between 5 and 9 yr,

Table 3. *Unadjusted prevalences of chronic diseases among subjects who participated in hand-grip strength test at exam 4*

Disease	%
Angina	6.5
Arthritis	13.7
Cancer	0.2
Coronary heart disease	29.3
Stroke	4.0
Chronic obstructive lung disease	3.4
Diabetes	17.0
Hypertension	53.9

Total participants in *exam 4*, *n* = 3,610.

Table 4. *Bivariate age-adjusted odds ratios for grip-strength disability in old age*

Predictors	Odds Ratio	95% CI
Baseline		
Grip strength		
Lowest vs. highest tertile	8.18	5.58–11.8
Middle vs. highest tertile	2.08	1.41–3.07
Exam 1		
Body height		
Lowest vs. highest tertile	2.66	1.94–3.63
Middle vs. highest tertile	1.40	1.00–1.98
BMI		
Lowest vs. highest tertile	1.10	0.84–1.45
Middle vs. highest tertile	0.94	0.71–1.23
Upper arm lean area		
Lowest vs. highest tertile	2.10	1.58–2.78
Middle vs. highest tertile	1.43	1.07–1.92
Body weight		
Lowest vs. highest tertile	1.78	1.34–2.38
Middle vs. highest tertile	1.14	0.84–1.54
Exam 4		
Angina	1.27	0.81–1.98
Arthritis	1.40	1.03–1.89
Cancer	2.75	0.61–12.3
Coronary heart disease	1.27	1.00–1.60
Chronic obstructive pulmonary disease	1.66	0.90–3.01
Stroke	3.20	2.12–4.87
Diabetes	1.42	1.08–1.88
Hypertension	0.64	0.51–0.80

Grip-strength disability is grip strength ≤21 kg.

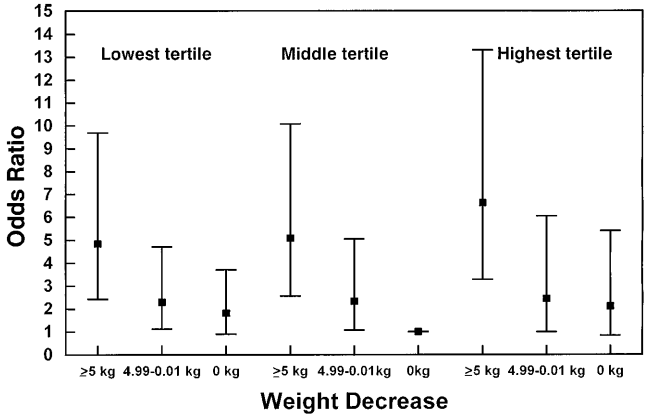

Fig. 4. Odds ratios for incident grip-strength disability according to *exam 1* body weight tertiles and weight decrease between *exams 1* and *4* (5 kg or more; 4.99–0.01 kg; no decrease or increase). Adjusted for age, height, and baseline grip strength.

~15–20% of participants have shown stable or increased strength (16, 28). Using data from *exams 4* and *5*, with approximate length of follow-up being 3 yr, we found that in men ages 71 and over, 22% increased or maintained their previous strength level (*exam 5* strength − *exam 4* strength ≥0). The strength difference between *exams 4* and *5* correlated negatively with *exam 4* strength ($r = -0.300$, $P < 0.001$); this suggests that those who showed poor results at *exam 4* were more likely to improve their strength over the follow-up period. However, over the entire 27-yr follow-up period, only 40 men (1.1% of the survivors) showed no strength decrease. At baseline, these people were younger (85% were 54 yr old or younger) and had lower body weight (47.5% in the lowest tertile) and lower grip strength (55% in the lowest tertile) than the other participants. Over the follow-up period, 70% of them maintained or gained weight. This information suggests that the health of these people may have been compromised at the time of the baseline measurements and that they thus exhibited exceptionally poor strength results. Our findings illustrate that short-term variations in strength go in both directions, even in old age, but the overall long-term trend with age is decline. However, it is worth noting that grip strength is a relatively stable characteristic, as correlation for repeated measures over 27 yr was $r = 0.557$ ($P < 0.001$); this indicates that >30% of the variation in grip strength measured in old age is explained by midlife strength.

Weight loss was a significant determinant of the rate of grip-strength loss and grip-strength disability (follow-up grip strength ≤21 kg) even after adjusting for baseline weight and age. Regardless of the baseline weight, the OR for becoming grip-strength disabled was five to six times greater among those who lost 5 kg of weight compared with those who maintained or increased their weight and were in the middle weight tertile at *exam 1*. It is worth noting that our study population differs from the rest of the US population in that it was more lean, with average BMI at *exam 1* being 23.8 $kg \cdot m^{-2}$. The average BMI in middle-aged black men [25.6 ± 4.9 (SD)] and white men (25.7 ± 4.0) has been found to be considerably greater (7). On the other hand, the fact that weight loss had a similar effect on strength decrease, regardless of initial weight, suggests that this finding may be generalized into more heterogenous populations. In persons of older ages, the majority of lost weight is lean tissue, which mostly consists of muscle (11). For example, in 64 Finnish men followed from the ages of 75–80 yr, the average body mass decreased from 74.0 to 73.0 kg, lean body mass decreased from 57.7 to 56.3 kg, and fat percent increased from 21.7 to 22.2% (34). As part of lost lean tissue is replaced by fat, body weight decrease is probably a valid but conservative estimate of muscle mass decrease. The use of body weight decrease as an indication for muscle mass decrease is further supported by the strong correlation between the upper arm muscle area and body weight observed at *exam 1* (age adjusted, $r = 0.520$, $P < 0.001$). Decline in muscle mass has been suggested as the direct cause of age-related strength decline (9). Our findings suggest that weight loss in old age may lead to loss in muscle strength, because it is likely that a substantial proportion of lost weight was muscle. This observation is clinically important, because poor muscle strength has been found to be associated with poor stair-climbing ability (26), slow walking speed (10), and self-reported mobility difficulties (27). However, it is unclear whether maintenance of body and muscle mass with age would ensure maintenance of muscle strength, because muscle strength per unit of lean body mass has been found to decrease with age (16, 30).

In the present study, stroke, diabetes, arthritis, and coronary heart disease at *exam 4* were associated with steep strength decrease (>1.5%/yr) and grip-strength disability. COPD was associated with steep strength decline, but association with grip-strength disability did not yield statistical significance. Angina and cancer were not associated with steep strength decline and grip-strength disability, and hypertension was found to be protective of these outcomes. The benefit of this design, although retrospective, is that the outcome (steep strength decline) was longitudinal. In a cross-sectional study, it remains unclear whether people with diseases were weak to start with or whether they became weak because of the disease. For example, those who reported arthritis at *exam 4* had significantly lower ($P < 0.001$) grip strength at follow-up [28.2 ± 0.29 (SE) kg, $n = 507$] than did those who did not have arthritis (29.4 ± 0.12 kg, $n = 3{,}173$). Baseline grip strength or age did not differ between these groups; this suggests that low muscle strength did not predispose these people to arthritis. However, temporal relationships of joint pain, disuse, and poor muscle strength that are typical in persons with arthritis require further studies (24). Poor muscle strength as an etiologic factor in knee osteoarthritis is supported by a study in which people with radiographically diagnosed but pain-free knee osteoarthritis had poorer quadriceps muscle strength than did those without the diagnoses, although strength in other muscle groups did not differ (33).

Skeletal muscle is one of the major sites for glucose disposal during carbohydrate loading. Findings of a previous study (19) suggest that poor skeletal muscle strength, which is usually associated with lower levels of physical activity and less muscle mass, may precede and predict the development of insulin resistance. Insulin resistance is associated with the development of non-insulin-dependent diabetes mellitus. However, in the present study, the baseline grip strength or age did not differ between those who had or did not have diabetes at *exam 4*, but the follow-up grip strength was significantly ($P = 0.001$) lower among those with diabetes [28.5 ± 0.26 (SE) kg, $n = 658$] than those without diabetes (29.4 ± 0.12 kg, $n = 3{,}184$). A probable explanation for steep strength decline among those with diabetes is neuropathy, which is a common complication of both insulin-dependent and non-insulin-dependent diabetes. Muscle weakness and atrophy are among the defects experienced by those with neuropathy (8).

Stroke was associated with steep strength decline and grip-strength disability, although the strength of the better, most probably the unaffected, hand was used in the analyses. Muscle weakness is one of the manifestations of impaired neurological function after stroke. Stroke patients have been found to have lower muscle strength also on the unaffected side (22) and to be physically more inactive than healthy controls (6).

Hypertension, however, was associated with lower risk of steep strength decline (OR 0.71; 95% CI, 0.57–0.88). A probable explanation for this is that body weight and grip strength are correlated, whereas higher body weight is a risk factor for hypertension. In the present study, those who had hypertension at *exam 4* had greater body weight ($P < 0.001$) at *exam 1* (64.2 ± 0.19 kg, $n = 2{,}030$) than those who were normotensive at *exam 4* (63.3 ± 0.20 kg, $n = 1{,}767$). In addition, those with hypertension at *exam 4* lost significantly ($P < 0.001$) less weight over the follow-up period (−2.01 ± 0.16 kg, $n = 1{,}936$) than those who did not have hypertension (3.02 ± 0.18 kg, $n = 1{,}658$). However, a further adjustment for body weight did not change the OR meaningfully. Exercise is commonly recommended to those with high blood pressure. Diagnosis of hypertension may make people more aware of their living habits and serve as an incentive for healthier lifestyle.

Diseases and weight loss are treated in these analyses as independent predictors of strength loss. Weight loss and morbidity are, however, related. In the Cardiovascular Health Study (15), those who lost >10% of their weight were more likely to report fair or poor health and mobility disability and to have more medications than those who maintained their weight.

The data in this study suggest that strength decreases at an increasing rate toward the higher age groups and that weight loss and chronic conditions, such as stroke, diabetes, arthritis, coronary heart disease, and COPD are associated with a steeper decline. Although these analyses were restricted to one ethnic and gender group, men with Japanese ancestry, the findings can probably be generalized to other populations. These physiological processes are unlikely to differ materially, for example, according to race. Weight loss in old age may predispose people into accelerated strength loss. Consequently, good disease management and maintenance of optimal weight may help prevent steep strength decline in older people.

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