Association of Functional Decline With Subsequent Diabetes Incidence in U.S. Adults Aged 51 Years and Older: The Health and Retirement Study 1998–2010

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OBJECTIVE

We assessed whether functional decline and physical disability increase the subsequent risk of diabetes.

RESEARCH DESIGN AND METHODS

We used a subsample of adults aged 51 years and older with no diabetes at baseline who were followed up to 12 years (1998–2010) in the Health and Retirement Study, an observational study of a nationally representative survey. We assessed baseline disability status and incident disability with subsequent risk of diabetes, accounting for death as a competing risk and controlling for BMI, age, sex, race/ethnicity, net wealth, mother's level of education, respondents' level of education, and time of follow-up. Disability was defined as none, mild, moderate, and severe, based on a validated scale of mobility measures. Diabetes was identified by self-report of a diagnosis from a doctor. Population attributable fraction (PAF) was calculated to assess the percentage of diabetes cases that were attributable to mobility disability.

RESULTS

The sample included 22,878 adults with an average of 8.7 years of follow-up; 9,649 (41.2%) reported some level of disability at baseline, and 8,175 (35.7%) additional participants developed disability during follow-up; 3,546 (15.5%) participants developed diabetes; and 5,869 (25.6%) died. Regression analyses found a statistically significant dose–response relationship of increased risk of diabetes (28–95%) among those with any level of functional decline, prevalent or incident. Among the subanalytic sample, including incident disability only, the PAF was 6.9% (CI 4.2–9.5).

CONCLUSIONS

Our findings suggest those who become disabled, even mildly, are at increased risk of developing diabetes. This finding raises the possibility that approaches to prevent disability in older adults could also reduce diabetes incidence.

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Diabetes has been consistently associated with an increased risk of physical disability in findings from both cross-sectional (1) and longitudinal studies (2). This degree of association might be mediated by multiple factors, including the higher prevalence of obesity, coronary heart disease, peripheral arterial disease, chronic kidney disease, stroke, and depression in people with type 2 diabetes. The burden of diabetes-related physical disability is also concerning because the largest absolute increases in diabetes prevalence are among those aged 65+ years; thus a high rate of disability in this subpopulation has ominous implications for quality of life, subsequent morbidity, and use of health services (3).

Despite this growing concern and attention to disability risk in the population with diabetes, no studies we could identify had considered the converse association: whether functional decline and disability related to aging could increase risk for diabetes in older adults. In 2011, 30.7% of adults aged 65–74 years and 48.0% of adults aged 75+ years had some level of physical difficulty (4). Functional decline in older adults might affect diabetes risk through numerous mechanisms, including the rapid decline in physical activity levels or selective loss of lean muscle mass relative to fat mass; accompanying depression could also affect insulin sensitivity. If true, the high absolute incidence of disability in older adults means that even a modest independent association of disability with diabetes incidence could constitute an important modifiable risk factor for the broad population of older adults. Therefore, for this study, we analyzed longitudinal data to determine the impact of disability on subsequent diabetes incidence among older adults.

RESEARCH DESIGN AND METHODS

We performed secondary analysis on data from the Health and Retirement Study (HRS), a population-based, longitudinal health interview survey of a cohort of adults ≥51 years of age in the U.S. (5,6). These data were collected between 1998 and 2010 from HRS surveys, which are held every 2 years. To ensure adequate sample size, we started with 1998 because it was the first wave that included several cohorts. Baseline response rates during this period ranged from 69 to 81%, and the follow-up response rates were between 87 and 89%. During our study, an average of 18,500 people were interviewed in each wave (7). The HRS is sponsored by the National Institute on Aging and performed by the Institute for Social Research at the University of Michigan. The Health Sciences Institutional Review Board at the University of Michigan approved the HRS design. The data used for this analysis contain no unique personal identifiers and are publicly available.

Study Populations

Responses from persons aged ≥51 years who had been interviewed between 1998 and 2010 were assessed to determine if the respondent self-reported disability or diabetes at the time of first interview. We used two different analytic samples to examine the effect of disability on the subsequent incidence of diabetes. For the first sample, labeled "main study sample," we examined diabetes incidence in adults not self-reporting diabetes at baseline, irrespective of prevalent disability status (n = 22,876). For this analytic sample, the "baseline" refers to the first year from which data were used in our analysis—the first year of participation in the HRS study if 1998 or later. Although HRS began in 1992, only data from 1998 and after were included in the analysis.

We restricted the second sample, labeled "substudy sample" to nondiabetic adults not disabled prior to baseline (n = 12,242) so that we could assess the relationship between incident disability and subsequent incident diabetes. For this analytic sample, "baseline" data are from the second year of the respondent's participation in HRS (but not before 1998). The first year of HRS data was excluded for all respondents in this sample; we did this to ensure that each respondent had no disability prior to or at the time of first participation in HRS and that disabilities, if any, would develop during the study period. Because of the different baselines for the two studies, the substudy is not strictly a subsample of the nondisabled baseline group in the main study. For example, respondents whose first interview with the HRS was prior to 1998 that reported no disability until 1998 would be included in the substudy because they were not disabled prior to baseline. They would also be included in the main study as disabled at baseline in 1998; thus the baseline substudy is not a subsample of the nondisabled baseline group of the main study.

Outcome Variables and Main Exposure

We set up the analysis to provide multinomial outcome variables—incident diabetes, death, or neither—allowing us to assign death as a competing risk (also known as the competing risk model). Incident diabetes was defined as the first self-report by a respondent to the HRS of a diabetes diagnosis (i.e., being told by a doctor that he or she has diabetes or high blood glucose) during the study period (7). Deaths were confirmed using the National Death Index and the Social Security Death Index. To obtain data on the respondent prior to death, proxies for the respondents were interviewed. The response rates for such exit interviews ranged from 84 to 92% during our study period (8).

We calculated the main exposure variable, disability, using responses to a series of questions on the difficulty of performing everyday activities. These activities were *1*) walking one block; *2*) walking several blocks; *3*) climbing one flight of stairs; *4*) stooping, crouching, or kneeling; and *5*) pushing or pulling a large object. Modifying a previously developed four-state model for defining mobility disability among persons with diabetes (9), we classified a respondent's disability as none, mild (difficulty with stooping and walking several blocks or difficulty with at least one or two mobility measures other than climbing), moderate (difficulty with climbing or difficulty with at least three mobility measures), and severe (difficulty with four or five mobility measures). Because disability status changed over time for approximately 57% of respondents, becoming more severe for some and less severe for others, the number of years contributed

at each disability state was used in the denominator of diabetes incidence and mortality rates. The disability state at the time of reported diabetes diagnosis or death was used in the numerator.

Covariates

We controlled for several covariates in the relationship between disability and diabetes. Time-invariant covariates included mother's level of education, respondents' level of education, sex, race (white, black, other), and Hispanic ethnicity, if any (Mexican American and other). Mother's level of education was included because childhood socioeconomic status, in particular, mother's education, has been found to have a strong association with health-related aspects of the aging process and midlife chronic diseases (10). Time-variant covariates included age; net wealth (i.e., sum of all wealth components, including real estate, stocks, mutual funds, income, checking accounts, certificates of deposit, individual retirement accounts, transportation, bonds, business assets, other assets, less all debt, including mortgages, credit cards, amortization, and other loans); depression (based on the Center for Epidemiologic Studies Depression Scale); BMI (calculated from self-reported height and weight); self-reported hypertension; and year of the interview (to account for time in the study).

Statistical Analysis

Due to the longitudinal nature of the data, if a participant did not respond in some waves and data from a previous wave were known, we assumed it did not change and carried it forward. We also used inverse probability weighting to reduce bias related to missing data whether it was due to loss of follow-up or not reported by the respondent in that wave or previous waves (11). In this method, logistic regression is used to determine the predicted value of being a complete case; weights are the inverse of the probability of being a complete case. Binary logistic regression was used for no competing risk modeling in which the outcome was incident diabetes versus no diabetes (censoring those who had died during the study period). Multinomial logistic regression was used for competing risk modeling. Data were modeled with SUDAAN version 11.0.0 (Research Triangle Institute, Research Triangle Park, NC) using PROC MULTILOG, which fits generalized estimating equations, a method that accounts for a multinomial response variable and correlation due to repeated measures within individuals.

Two models were fit for each study analytic sample: the first model included adults without self-reported diabetes, with or without disability, and the second model included adults with neither self-reported diabetes nor disability, within 2 years prior to baseline. The first model assessed the research question regarding the relationship between disability and diabetes, controlling for time-invariant and time-variant covariates that might have confounded the results used to measure relationship. Time-invariant covariates included race (white, black, and other), ethnicity (Mexican American and other), respondent's level of education, baseline BMI, and mother's level of education. Time-variant covariates included age, net wealth, and year of interview. In the second model, we assessed mediation by additionally including hypothesized intermediaries of the association between disability and diabetes; these time-variant variables included depression, BMI, and hypertension. Risk ratios (RRs) are presented from predicted margins estimated from the binary and multinomial analyses.

Finally, using a SAS macro specifically designed to calculate population attributable fraction (PAF) in a cohort study design, we calculated the PAF to quantify the impact of disability on incident diabetes at the population level (12). The resulting PAF estimate indicated the proportion of diabetes attributable to disability by estimating the proportion of diabetes that would not have occurred if no one was disabled at baseline, whether or not they became disabled during the study. Both the RR, which indicated strength of association between disability and subsequent diabetes, and the prevalence of other risk factors were taken into account in the calculation of the PAF. Also, the estimation of PAF was for the time interval 1998–2010 and is adjusted for potential confounding factors and death as a competing risk.

HRS data are weighted so as to be nationally representative of the population ≥51 years of age as a whole. Instead, as we used cohorts of respondents that entered in different years, we could not use weights for any one year or cohort to represent the U.S. population.

RESULTS

Approximately 5% of respondents were lost to follow-up (n = 1,158; 61.1% with no disability at baseline, 27.0% mild, 6.6% moderate, and 5.4% severe). Approximately one-fifth of respondents did not respond at all waves (n = 4,742; 59.0% with no disability at baseline, 26.8% mild, 7.7% moderate, and 6.5% severe). Data that were missing and could not be carried forward from previous waves were minimal (n = 154; 52.6% with no disability at baseline, 27.9% mild; 13.0% moderate, and 6.5% severe).

Among respondents in the main study sample (i.e., no diabetes but includes incident or prevalent disability at baseline), 57.6% of the respondents were female, 82.1% white, 13.3% black, and 5.0% Mexican American. One-fourth of respondents had less than a high school diploma, 39.1% were overweight, and 39.5% had high blood pressure (Table 1). Over an average of 8.7 years of follow-up, 3,546 (15.5%) nondiabetic participants developed diabetes, 5,869 (25.6%) died, and 13,461 (58.9%) neither developed diabetes nor died. Among participants without diabetes at baseline, in the substudy sample, 60.5% reported some level of disability during the study compared with 76.7% in the main study sample.

In the main study sample, cumulative diabetes incidence was 10.8 per 1,000 person-years among those with no disability at any time during the study. Among those with some level of disability, cumulative diabetes incidence was 15.9, 19.0, and 21.7 cases per 1,000 person-years for those with mild, moderate, and severe disability, respectively (Fig. 1, left-hand side). In this sample, mortality rates were also lowest among those with no mobility

Table 1—Baseline descriptive statistics of a nondiabetic population aged ≥51 years in the HRS from 1998–2010

	Main study sample[a] at baseline *n* = 22,876	Substudy sample[b] at baseline *n* = 12,242[c]	Main study sample baseline disability status			
			None *n* = 12,329[c]	Mild *n* = 6,231	Moderate *n* = 2,026	Severe *n* = 2,268
Age at baseline, years						
51–64	56.9	62.6	65.0	52.0[d]	45.1[d]	38.5[d]
65–79	31.8	30.2	29.1	35.6[d]	35.0[d]	32.8[d]
80+	11.3	7.1	5.9	12.4[d]	19.9[d]	28.7[d]
Sex						
Male	42.4	47.3	48.0	38.6[d]	32.0[d]	31.7[d]
Female	57.6	52.7	52.0	61.4[d]	68.0[d]	68.3[d]
Race						
White	82.1	83.1	82.2	84.2[d]	79.7[d]	78.5[d]
Black	13.3	12.1	13.0	11.8	15.7[d]	16.7[d]
Other	4.6	4.8	4.8	4.0[d]	4.6	4.8
Hispanic						
Mexican American	5.0	4.8	4.9	4.5	6.6[d]	5.1
Other	3.4	3.4	3.8	2.6[d]	4.0	3.2
Non-Hispanic	91.6	91.8	91.3	92.9[d]	89.4[d]	91.7
Respondent's education						
No degree	25.1	18.9	19.6	25.3[d]	34.5[d]	46.4[d]
High school graduate/GED	51.6	52.1	51.7	53.9[d]	52.3	43.5[d]
Some college	3.9	4.3	4.4	3.7[d]	2.8[d]	2.4[d]
College graduate	19.4	24.7	24.3	17.1[d]	10.4[d]	7.7[d]
Mother's education						
<High school	62.8	56.2	56.3	65.5[d]	75.9[d]	81.0[d]
High school graduate	26.4	30.6	30.4	25.1[d]	17.3[d]	14.6[d]
Some college	5.9	6.9	7.1	5.4[d]	4.1[d]	2.6[d]
College graduate	4.9	6.3	6.2	4.0[d]	2.7[d]	1.8[d]
BMI						
<18.5	2.6	1.7	1.6	2.2[d]	3.7[d]	8.2[d]
18.6–24.9	36.3	39.1	39.6	32.6[d]	31.1[d]	33.3[d]
25.0–29.9	39.1	41.2	41.3	39.4[d]	34.4[d]	31.3[d]
30.0–34.9	15.7	14.4	13.9	18.3[d]	19.3[d]	15.0
>35.0	6.3	3.6	3.6	7.5[d]	11.5[d]	12.2[d]
High blood pressure						
Yes	39.5	34.1	33.0	42.6[d]	49.1[d]	56.7[d]
No	60.5	65.9	67.0	57.4[d]	50.9[d]	43.3[d]

All data are percentages. [a]The main study sample was adults without self-reported diabetes, irrespective of prevalent disability status. [b]The substudy sample was adults without self-reported diabetes and with no disability prior to baseline. [c]The total substudy sample is not a subset of the main study nondisabled sample due to the different definitions of baseline. [d]Statistically significantly different ($P < 0.05$) from no disability at baseline.

disability at baseline (10.8 per 1,000 person-years); mortality rates were somewhat higher among those with mild, moderate, and severe disability (19.5, 36.9, and 71.5 per 1,000 person-years, respectively). Among the substudy sample (Fig. 1, right-hand side), diabetes incidence of those with no, mild, moderate, and severe disability were 10.7, 17.3, 17.8, and 20.5 per 1,000 person-years, respectively, and mortality rates were 10.8, 18.9, 37.7, and 83.4 per 1,000 person-years, respectively.

In the competing risk models controlling for sex, race/ethnicity, mother's level of education, respondent's level of education, age, baseline BMI, net wealth, and year of report, the risk of incident diabetes was elevated for each level of disability when compared with no disability in the main study sample: mild (RR 1.28; 95% CI 1.17–1.42), moderate (RR 1.43; 95% CI 1.25–1.64), and severe (RR 1.63; 95% CI 1.44–1.85) (Table 2). Also, results of models for nondiabetic persons in the substudy sample were similar to results of the main study sample, except the risks for diabetes were higher: mild (RR 1.40; 95% CI 1.23–1.58), moderate (RR 1.52; 95% CI 1.22–1.90), and severe (RR 1.81; 95% CI 1.42–2.41). Including hypothesized mediators (e.g., time-variant BMI, depression, and blood pressure) in the main study sample yielded lower risk of diabetes by 3–14% and up to 25% in the substudy sample. In general, regardless of analytic sample, the risk of mortality increased with level of disability. In the main study sample, the risk of death from the model assessing the relationship between disability and diabetes (without hypothesized mediators) was 1.41 (95% CI 1.29–1.53) among those with mild disability, 2.16 (95% CI 1.96–2.37) among those with moderate disability, and 3.32 (95% CI 3.06–3.61) among those with severe disability. Similarly, in the substudy sample, risk of death ranged from 1.25 (95% CI 1.12–1.40) to 2.96 (95% CI 2.61–3.35). When death was censored rather than treated as a competing risk, the risk of diabetes was similar but slightly lower

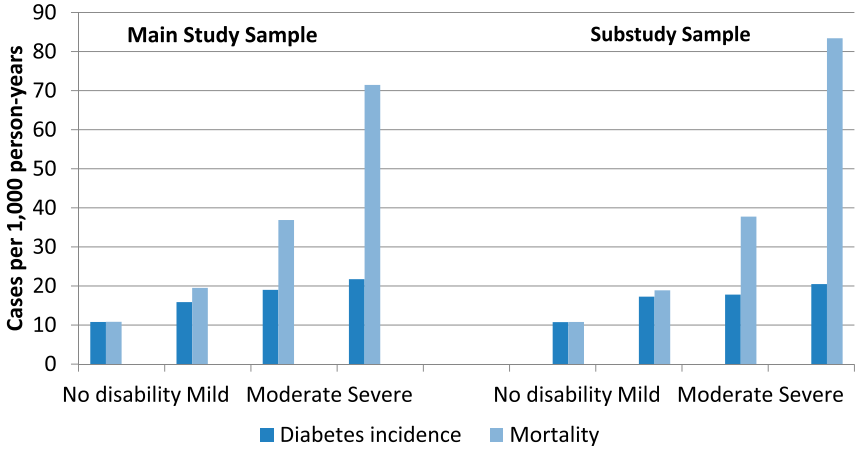

Figure 1—Incident diabetes and mortality by level of disability in U.S. nondiabetic adults aged ≥51 years in the HRS from 1998–2010. The figure shows the number of cases by disability level at time of reported diabetes or death.

among the severely disabled (data were not shown).

The PAF, an estimated proportion of diabetes that would not have occurred if no one had disability prior to baseline or incident disability, was 11.1% (CI 7.6–14.7). For the substudy analytic sample, the PAF was 6.9% (CI 4.2–9.5).

CONCLUSIONS

In this large sample of middle-aged and older adults, there was a dose–response relationship between disability and incident diabetes during a 12-year period. Those with severe disability prior to the baseline or during the study were at 63.0% higher risk of reporting a diabetes diagnosis than those with no disability. Of those who developed diabetes, 6.9% were attributable to incident mobility disability. The difference in excess risk of diabetes by disability status between the two analytic samples was small, likely because the substudy sample was included in the main study sample. The risk of mortality was also strongly associated with disability and increased with level of disability. In general, the risk of mortality was greater than the risk of diabetes, with the largest difference in those with severe disability, indicating the high risk of death may have precluded an even higher incidence of diabetes. In addition, our results suggest that hypothesized mediating factors [e.g., change in BMI (13), depression (14), and hypertension] may account for up to 25% of the effect of disability on diabetes.

Several factors or processes could explain the association of disability with subsequent diabetes. Mobility disability in older adults may be followed by an increase in sedentary behavior, a decrease in physical activity, muscle disuse, and a reduction in the ratio of lean-to-fat mass. Each of these processes have been associated with reduced insulin sensitivity and increased inflammation, which could hasten a deterioration of glucose tolerance in vulnerable older adults (15–17). Physical disability may also influence diabetes risk through its association with comorbid depression, which has similarly been associated with insulin resistance and diabetes risk (18).

Our analyses had limitations. First, although we controlled for change in BMI, we lacked information on body fat

Table 2—Diabetes incidence and mortality by disability status at time of diabetes diagnosis in the nondiabetic population aged ≥51 years in the HRS from 1998–2010

	Without hypothesized mediators[a]			With hypothesized mediators[b]		
	Competing risk model		Noncompeting risk model	Competing risk model		Noncompeting risk model
	RR incident diabetes (95% CI)	Mortality ratio (95% CI)	RR diabetes (95% CI)	RR incident diabetes (95% CI)	Mortality ratio (95% CI)	RR diabetes (95% CI)
Main study sample						
No disability	1.0	1.0	1.0	1.0	1.0	1.0
Mild disability	1.28 (1.17–1.42)	1.41 (1.29–1.53)	1.29 (1.17–1.41)	1.20 (1.09–1.33)	1.29 (1.18–1.41)	1.21 (1.04–1.06)
Moderate disability	1.43 (1.25–1.64)	2.16 (1.96–2.37)	1.43 (1.25–1.63)	1.40 (1.23–1.60)	1.89 (1.70–2.11)	1.40 (1.04–1.07)
Severe disability	1.63 (1.44–1.85)	3.32 (3.06–3.61)	1.60 (1.41–1.88)	1.49 (1.30–1.70)	2.60 (2.36–2.87)	1.46 (1.03–1.07)
Substudy sample						
No disability	1.0	1.0	1.0	1.0	1.0	1.0
Mild disability	1.40 (1.23–1.58)	1.25 (1.12–1.40)	1.39 (1.23–1.57)	1.34 (1.18–1.52)	1.14 (1.01–1.29)	1.34 (1.18–1.52)
Moderate disability	1.52 (1.22–1.90)	1.89 (1.64–2.19)	1.50 (1.21–1.86)	1.46 (1.16–1.82)	1.63 (1.38–1.93)	1.45 (1.16–1.80)
Severe disability	1.81 (1.42–2.31)	2.96 (2.61–3.35)	1.72 (1.36–2.18)	1.56 (1.18–2.06)	2.28 (1.94–2.67)	1.51 (1.15–1.98)

All models allow for change in disability and are adjusted for age, race/ethnicity, respondent's level of education, mother's level of education, person-years, and net wealth. Any disability includes adults without self-reported diabetes, irrespective of prevalent disability status. Incident disability includes adults without self-reported diabetes and with no disability prior to baseline. The noncompeting risk model does not include death as a separate outcome; those who died were dropped from the analysis during the wave of death. The mediators are BMI, self-reported high blood pressure, and depression (based on the Center for Epidemiologic Studies Depression Scale). [a]Also adjusted for baseline BMI. [b]Also adjusted for time-variant BMI, high blood pressure, and depression.

distribution or lean muscle mass, which would have allowed us to determine the degree to which sarcopenic obesity or an increase in the body fat to lean muscle mass ratio could have explained our findings (19–21). Second, we lacked the dates of disability onset or diabetes diagnosis so that we could identify which event occurred first for the 2.5% of respondents (in main study sample, 2.6% in substudy sample) who first reported both during the same wave. We did, however, include those subjects in the model appropriately with the events co-occurring. Third, disability and diabetes status were self-reported, and thus misclassification for undiagnosed cases is possible. However, validation studies have found agreement for the mobility measures used in other aging studies between 69.4 and 84.7% (22) and self-reported diabetes in survey data to range from 70 to 99% (23). Fourth, because we used cohorts of respondents who entered in different years and therefore could not use weights for any one year, results are not representative of the U.S. population in a specific year. It is also important to note that although we hypothesized change in BMI, depression, and hypertension mediated the relationship between disability and diabetes, it can also be argued, physiologically, that these factors could be confounders of the relationship. When interpreting estimates from the model, statistically, these factors could have behaved as mediators or confounders; thus further research to better assess these relationships is warranted. Finally, although physical activity is associated with functional decline as well as diabetes, data were not collected consistently during our study period so that we could include it in our analyses.

A strength of our analysis is that we considered death as a competing risk. Although our findings suggest that the risk of diabetes incidence due to disability would not have been biased by ignoring death as a competing risk, we would have ignored the impact of disability on death. However, by considering death as a competing risk, we found that for those nondiabetic older adults who became disabled, risk of death was even higher than the risk of diabetes. In light of previous work that found more older adults are living with physical difficulty (4), this finding strengthens the case for targeting those at risk for disability for intervention.

Although many studies have associated diabetes with subsequent disability, this is the first prospective analysis we are aware of to examine the association of disability with subsequent diabetes. These findings are important for several reasons. Older adults comprise the fastest growing segment of the U.S. diabetes population because increases in diabetes prevalence have been greatest in older adults (24), mortality rates have decreased most in older adults, and the baby boom generation is transitioning into the age range of high diabetes and disability incidence. We estimated the proportion of diabetes that would not have occurred to be 6.9% if no one had become disabled in our substudy sample of persons aged ≥51 years from 1998–2010. Using unpublished data from the National Health Interview Survey, we estimated that over 9,906,000 new cases of diabetes were diagnosed among persons aged 51–79 years between 1998 and 2010. In terms of the U.S. population, if 6.9% of incident diabetes cases among those aged ≥51 years from 1998–2010 could have been prevented, that would potentially mean 683,514 fewer cases nationally. This suggests that functional decline has contributed to a substantial number of new diabetes cases. This finding raises the possibility that approaches to prevent or modify disability in middle-aged and older adults could also reduce diabetes incidence, and the burden of cost and human suffering that diabetes would cause. A systematic review on interventions to prevent disability in frail (based on at least one of the following: mobility, strength, endurance, nutrition/weight loss/obesity, physical inactivity, balance, and motor processing) community-dwelling adults indicated that relatively long-lasting and multicomponent several-times-weekly physical activity programs for moderately physically frail older persons can be protective for disability (25). In addition, lifestyle-based weight loss interventions have been associated with a slowing of functional decline (9). Hence physical activity programs targeted for those at risk for disability may also benefit the same people who also would later be at risk for developing diabetes. Due to the possible burden of new diabetes cases attributable to functional decline, further research might be done to assess the extent to which *modifiable* mediators between mobility disability and diabetes hasten the onset of diabetes.

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