Age and Ageing 2021; **50:** 1666–1674
https://doi.org/10.1093/ageing/afab045
Published electronically 26 March 2021

RESEARCH PAPER

Individual changes in anthropometric measures after age 60 years: a 15-year longitudinal population-based study

Jie Guo[1], Ying Shang[1], Laura Fratiglioni[1,2], Kristina Johnell[3], Anna-Karin Welmer[1,4], Anna Marseglia[1,5], Weili Xu[1]

[1]Aging Research Center, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet and Stockholm University, Stockholm, Sweden
[2]Stockholm Gerontology Research Center, Stockholm, Sweden
[3]Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden
[4]Functional Area Occupational Therapy & Physiotherapy, Karolinska University Hospital, Stockholm, Sweden
[5]Division of Clinical Geriatrics, Center for Alzheimer Research, Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Stockholm, Sweden

Address correspondence to: Jie Guo, MSc, Aging Research Center, Department of Neurobiology, Care Sciences & Society, Karolinska Institutet, Tomtebodavägen 18A Floor 10, SE-171 65 Solna, Stockholm. Tel: (+46) 07 392 334 80. Email: jie.guo@ki.se

Abstract

Background: weight loss is commonly observed with ageing. We explored the trajectory of body mass index (BMI) and two proxies of muscle mass—calf circumference (CC) and mid-arm circumference (MAC)—and identified their determinants.
Methods: within the SNAC-K cohort, 2,155 dementia-free participants aged ≥60 years were followed over 15 years. BMI, CC and MAC were measured at baseline and follow-ups. Baseline sociodemographic and lifestyle factors were collected through interviews. Diabetes and vascular disorders were diagnosed by physicians through clinical examination and medical records. Data were analysed using linear mixed-effect models stratified by age (younger-old [<78 years] vs. older-old [≥78 years]).
Results: over the 15-year follow-up, BMI remained stable among participants aged 60 years at baseline ($\beta_{slope} = 0.009$ [95% confidence interval −0.006 to 0.024], $P = 0.234$) and declined significantly among those aged ≥66 years, while CC and MAC declined significantly across all age groups. The decline over 15 years in BMI, CC and MAC separately was 0.435 kg/m^2, 1.110 cm and 1.455 cm in the younger-old and was 3.480 kg/m^2, 3.405 cm and 3.390 cm in the older-old. In younger-old adults, higher education was associated with slower declines in all three measures, while vascular disorders and diabetes were associated with faster declines. In older-old adults, vigorous physical activity slowed declines in BMI and CC, while vascular disorders accelerated declines in BMI and MAC.
Conclusions: CC and MAC declined earlier and more steeply than BMI. Cardiometabolic disorders accelerated such declines, while higher education and physical activity could counteract those declines.

Keywords: ageing, body mass index, calf circumference, mid-arm circumference, older people, trajectories

Key Points

- Body mass index (BMI), calf circumference (CC) and mid-arm circumference (MAC) declined with ageing.
- The declines were more evident among older than younger-older people.
- CC and MAC, suggested as proxies of muscle mass, declined earlier and more steeply than BMI.
- Higher education and being physically active, as modifiable factors, could counteract those declines.
- Type 2 diabetes and vascular disorders accelerated the declines in BMI, CC and MAC.

Introduction

Body composition changes with advancing age [1]. In particular, accelerated weight loss, a common age-related decline in late life, is characterized by loss of muscle mass and concurrently increased fat mass [1–3]. Emerging evidence has shown that muscle mass-related weight loss with advancing ageing is related to a variety of adverse health outcomes [4–7].

Body mass index (BMI) is the most widely used anthropometric measure. BMI is a combination of fat and muscle mass, and muscle mass loss might be masked by increased fat mass [1]. Thus, BMI might have poor sensitivity to detect muscle mass loss, especially early in late life. The loss of muscle mass among older adults is an indicator of sarcopenia [8], which is a predictor for several adverse outcomes including physical function impairment, disability and mortality [9,10]. Therefore, to disentangle the relation of different components of body composition with ageing, other anthropometric measures have been proposed. Calf (CC) and mid-arm (MAC) circumferences have a good correlation with muscle mass [11–14], and the loss of muscle mass is mainly driven by loss of appendicular muscle mass [15], and CC and MAC have been suggested as proxies of muscle mass.

Several studies have shown that lifestyle factors (e.g. smoking, alcohol consumption and physical activity) and chronic conditions (e.g. cardiovascular diseases and diabetes) are associated with changes in BMI or muscle mass measured by bioelectrical impedance analysis or dual-energy X-ray absorptiometry (DEXA) [16–21]. Questions remain about the patterns of changes in different anthropometric measures over the life course and the determinants for such changes among older adults. In the present study, we aimed to assess the trajectories of BMI, CC and MAC over a 15-year follow-up among Swedish older adults and to identify sociodemographic, lifestyle and health-related factors that might be associated with these trajectories.

Methods

Study population

The study population was derived from the ongoing population-based Swedish National Study on Aging and Care-Kungsholmen (SNAC-K) including participants aged ≥60 years living at home or in institutions in Kungsholmen, central Stockholm. A total of 3,363 participants were examined at baseline (March 2001 to June 2004). Given the more rapid changes in health and a higher attrition rate among older participants, follow-up assessments were performed every 6 years for the younger age-cohorts (60, 66, 72 years) and every 3 years for the older age-cohorts (≥78 years). In the current study, follow-up data were available until December 2019.

At baseline, we excluded 310 persons with dementia, 11 with missing information on dementia diagnoses, 133 with missing information on BMI, CC or MAC and 38 with missing information on smoking, alcohol consumption or diabetes status. We additionally excluded 716 with no repeated measurement of BMI, CC or MAC during the follow-up (including 339 died and 329 dropped out before the first follow-up examination, and 48 with missing anthropometric measures). Therefore, 2,155 participants remained for the current study. During the follow-up, 744 (34.5%) died and 403 (18.7%) dropped out (Appendix 1 in Supplementary data).

All phases of SNAC-K data collection have been approved by the Karolinska Institutet Ethical Committee and the Regional Ethical Review Board in Stockholm, Sweden. All participants (or a proxy, such as a close family member, in case of severe cognitive impairment) provided informed and written consent at baseline.

Data collection

Data on sociodemographic (age, sex and education) and lifestyle (smoking, alcohol consumption and physical activity) factors, current medication use (e.g. antihypertensive and glucose-lowering medications), and medical conditions were collected by trained staff (nurses and physicians) following a structured protocol (available at http://www.snac-k.se).

Age was dichotomized into a younger-old (<78 years) and an older-old cohort (≥78 years), according to the SNAC-K study design and given the turning point of fat mass in older adults: fat mass tends to increase with age before the age of 80 years but stabilises or slightly declines thereafter [1]. Educational attainment was categorised as elementary or professional school, high school and university. Smoking status was recorded as never, ex-smoker or current smoker. Alcohol consumption was categorised as no/occasional, light-to-moderate (1–14 drinks per week for men or 1–7 drinks per week for women) or heavy (>14 drinks per week for men or >7 drinks per week for women) drinking [22]. Physical activity was assessed based on the frequency and intensity of physical exercise and categorised into inactive (≤2–3 times/month of light-to-intense exercise), moderate (several times per week or every day of light exercise) and vigorous (several times per week or every day of moderate/intense exercise) [23]. Peripheral blood samples were collected at baseline to measure glycated haemoglobin (HbA1c).

Arterial blood pressure was measured twice on the left arm in a sitting position with a 5-min interval. Hypertension was identified based on systolic and diastolic blood pressure ≥140/90 mmHg or self-report of taking antihypertensive treatment. Ischemic heart disease, heart failure and cerebrovascular disease (registered 5 years prior to the SNAC-K baseline assessment) were ascertained from clinical

examination and identified using the International Classification of Disease 10th version (ICD-10) through the Swedish National Patient Register (NPR). The Swedish NPR covers all inpatients (since 1987) and outpatients (since 2001) in the Swedish health care system [24]. Vascular disorders included ischemic heart disease (ICD-10 codes: I20–22, I24–25, Z951 and Z955), heart failure (I110, I130, I132, I27, I280, I42–43, I50, I515, I517, I528, Z941 and Z943), cerebrovascular disease (G45–46, I60–64, I67 and I69) and hypertension (I10–13 and I15). Type 2 diabetes was identified based on self-reported medical history, glucose-lowering medication use, medical records from the NPR (E11) or HbA1c ≥6.5% [25]. Dementia was diagnosed according to the 'Diagnostic and Statistical Manual of Mental Disorders', fourth edition criteria and using a validated three-step procedure [26].

Data on the participants' vital status were collected via linkage to the Swedish Cause of Death Registry.

Assessment of anthropometric measures

Weight and height were measured with a standard scale in light clothing without shoes by trained staff or self-reported by participants. BMI (kg/m^2) was calculated as weight (kg) divided by squared height (m^2) and categorised in four groups: underweight (<20 kg/m^2), normal (20–25 kg/m^2), overweight (25–30 kg/m^2) or obese (≥30 kg/m^2). MAC was measured at the mid-point between the tip of acromion process and tip of the olecranon process, with the elbow bent at a 90° angle [27]. CC was measured at the point of maximum convexity of the calf with the participants sitting down so that the knee and ankle were each bent to a 90° angle [27]. Both MAC and CC were measured on the right-hand side and rounded to 1 cm.

Statistical analysis

Baseline characteristics by age groups were compared with the *t*-test (continuous variables) or chi-square (categorical variables). Linear mixed-effect models were used to determine the separate trajectories of BMI, CC and MAC as a function of follow-up time. Random effects included a random intercept for individuals and a random slope for follow-up time assuming an unstructured covariance structure. We conducted linear mixed-effect models to assess the associations between baseline age groups (5 groups: 60, 66, 72, 78 and 81+ years; 2 groups: <78 and ≥78 years) and changes in BMI, CC and MAC. To compare the magnitude of changes in BMI, CC and MAC over time by age groups, we computed sex-specific z-scores, using the mean and standard deviation from male and female from the study population. To estimate the associations between each factor (selected based on previous literature) [16–21] and annual changes in anthropometric measures, we conducted separate linear mixed-effect models including the factor, the linear follow-up time and their interaction term (each factor × follow-up time) as fixed effects. In the fully adjusted mixed-effect model, we adjusted for sex, education, smoking status, alcohol consumption, physical activity, vascular disorders and diabetes. All analyses were stratified by age group (younger-old and older-old).

Additional statistical analyses are presented in Supplementary data. Statistical analyses were performed using SAS 9.4 (SAS Institute, Cary, NC). All *P*-values were two-sided, and we defined statistical significance as $P < 0.05$.

Results

Baseline characteristics

At baseline, mean participant age was 71.3 years (standard deviation [SD] 9.6 years), 63.0% were female, and 35.5% had a university education. Compared with the younger-old participants, the older-old participants were more likely to be female, have a lower education level, be more physically inactive and have diabetes and more vascular disorders. However, older-old participants were less likely to be current smokers or heavy drinkers, and had lower BMI, CC and MAC (all *P*-value < 0.05) (Table 1).

Trajectories of BMI, CC and MAC across age groups

In participants aged 60 years at baseline, BMI remained stable over time ($\beta_{slope} = 0.009$ [95% confidence interval {CI} −0.006 to 0.024], $P = 0.234$), while CC (−0.033 [−0.051 to −0.016], $P < 0.001$) and MAC (−0.042 [−0.059 to −0.024], $P < 0.001$) declined statistically significantly. All three measures declined significantly among those aged ≥66 years (Figure 1 and Appendix 2 in Supplementary data). In the younger-old (aged <78 years), the 15-year decline was 0.435 (95% CI: 0.274–0.603) kg/m^2 for BMI, 1.110 (0.938–1.283) cm for CC and 1.455 (1.296–1.615) cm for MAC. In the older-old adults (aged ≥78 years), the 15-years decline was 3.480 (3.081–3.876) kg/m^2 for BMI, 3.405 (3.039–3.760) cm for CC and 3.390 (3.081–3.707) cm for MAC. The z-scores for CC and MAC decreased more quickly than for BMI, especially among the younger-old adults (Figure 2, Appendix 3 in Supplementary data).

Factors related to trajectories in BMI, CC and MAC across age groups

Among the younger-old, participants with university education showed slower declines in BMI, CC and MAC than those with elementary or professional school education. In addition, vascular disorders and diabetes were associated with a faster speed of decline in all three measures over time (Table 2).

Among the older-old, people who were vigorously physical active had a slower decline in BMI ($\beta_{difference\ in\ slope} = 0.087$ [95% CI: 0.005–0.169]) and CC ($\beta_{difference\ in\ slope} = 0.132$ [95% CI: 0.057–0.207]) compared with those who were physically inactive. A similar trend was also observed for MAC ($\beta_{difference\ in\ slope} = 0.044$ [95% CI: −0.021 to 0.109]), though the result was not statistically significant.

Table 1. Baseline characteristics of the study population by age (<78 vs. ≥78 years) (n = 2,155)

Characteristics	Total	Younger-old (<78 years)	Older-old (≥78 years)	P-value[a]
Number	2,155	1,353	802	
Age, years	71.3 ± 9.6	64.8 ± 4.8	82.2 ± 4.4	<0.001
Female	1,358 (63.0)	794 (58.7)	564 (70.3)	<0.001
Education level				<0.001
Elementary/professional school	1,166 (54.1)	591 (43.7)	575 (71.7)	
High school	225 (10.4)	161 (11.9)	64 (8.0)	
University	764 (35.5)	601 (44.4)	163 (20.3)	
Smoke status				<0.001
Never smoker	1,015 (47.1)	548 (40.5)	467 (58.2)	
Ex-smoker	849 (39.4)	581 (42.9)	268 (33.4)	
Current smoker	291 (13.5)	224 (16.6)	67 (8.4)	
Alcohol consumption				<0.001
No or occasional	619 (28.7)	267 (19.7)	352 (43.9)	
Light or moderate	1,152 (53.5)	803 (59.3)	349 (43.5)	
Heavy	384 (17.8)	283 (20.9)	101 (12.6)	
Physical activity				<0.001
Inactive	483 (22.4)	236 (17.4)	247 (30.8)	
Moderate	1,127 (52.3)	689 (50.9)	438 (54.6)	
Vigorous	545 (25.3)	428 (31.6)	117 (14.6)	
Any of vascular disorders[b]	1,625 (75.4)	918 (67.8)	707 (88.2)	<0.001
Ischemic heart disease	265 (12.3)	96 (7.1)	169 (21.1)	<0.001
Heart failure	128 (5.9)	24 (1.8)	104 (13.0)	<0.001
Cerebrovascular disease	111 (5.2)	36 (2.7)	75 (9.4)	<0.001
Hypertension	1,588 (73.7)	904 (66.8)	684 (85.3)	<0.001
Diabetes	163 (7.6)	90 (6.7)	73 (9.1)	0.038
BMI, kg/m^2	25.8 ± 3.9	26.1 ± 3.8	25.3 ± 3.9	<0.001
Underweight (<20)	90 (4.2)	36 (2.7)	54 (6.7)	<0.001
Normal (20–25)	893 (41.4)	542 (40.1)	351 (43.8)	
Overweight (25–30)	886 (41.1)	578 (42.7)	308 (38.4)	
Obese (≥30)	286 (13.3)	197 (14.6)	89 (11.1)	
CC, cm	36.4 ± 3.3	37.1 ± 3.2	35.2 ± 3.2	<0.001
MAC, cm	28.9 ± 3.2	29.5 ± 3.0	27.8 ± 3.2	<0.001

Data are presented as means ± standard deviations or number (proportion). [a]T-test (for continuous variables) or chi-square (for categorical variables) was used to compare the baseline characteristics between younger-old and older-old. [b]Any of vascular disorder was defined as any of ischemic heart disease, heart failure, cerebrovascular disease or hypertension.

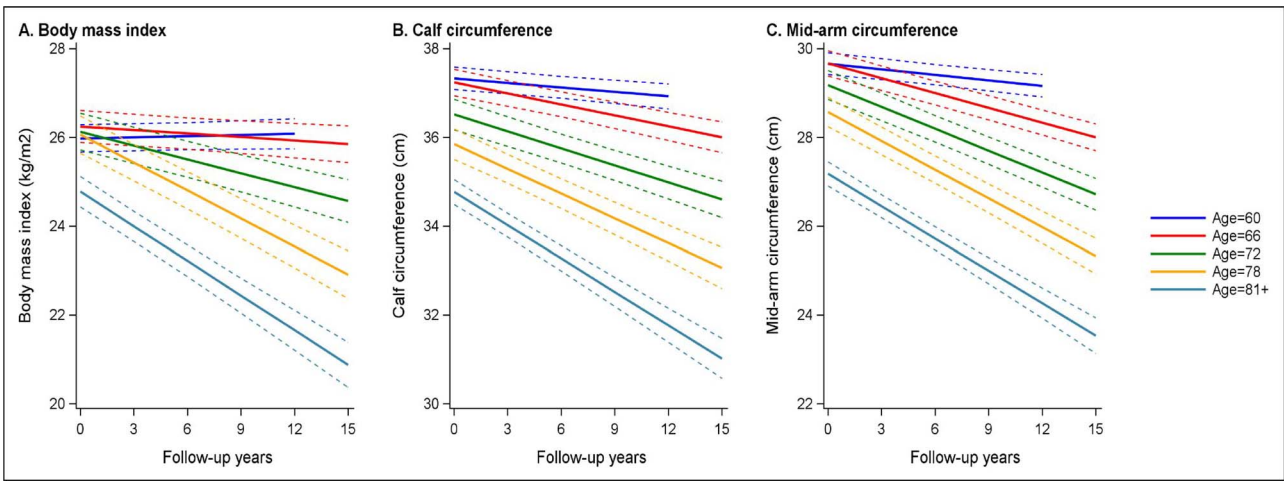

Figure 1. Trajectories of BMI, CC and MAC during 15-year follow-up across age groups.

Moreover, the presence of any vascular disorder was associated with faster decline in BMI ($\beta_{\text{difference in slope}} = -0.110$ [95% CI: −0.191 to −0.030]) and MAC ($\beta_{\text{difference in slope}} = -0.093$ [95% CI: −0.157 to −0.030]), though the result was not statistically significant for CC ($\beta_{\text{difference in slope}} = -0.035$ [95% CI: −0.108 to 0.038]) (Table 3).

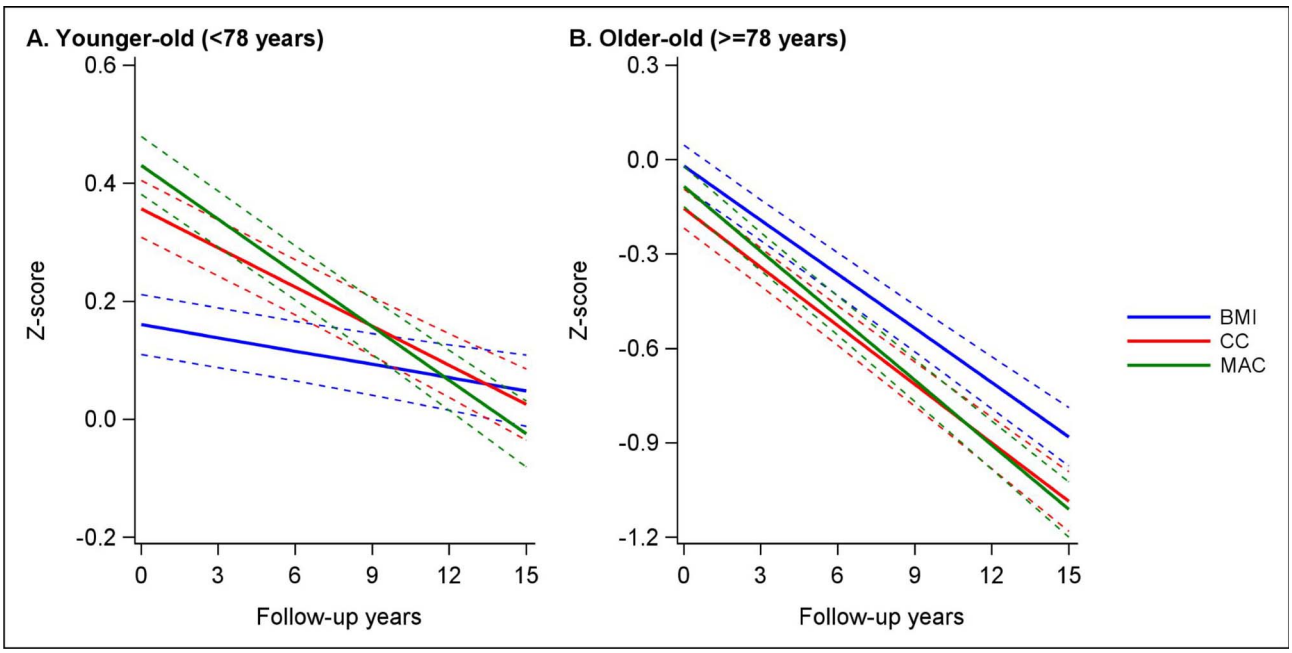

Figure 2. Trajectories of the z-scores of BMI, CC and MAC during 15-year follow-up among younger-old and older-old participants.

Discussion

In this large-scale population-based cohort study of the Swedish older adults, we found that: (i) the trajectories of BMI, CC and MAC declined significantly over the 15-year follow-up, and CC and MAC started declining at an earlier age and had a steeper decline than BMI; (ii) the declines were more pronounced in the older-old vs. younger-old cohorts and (iii) in younger-old adults, vascular disorders and diabetes were associated with accelerated declines in BMI, CC and MAC, whereas higher education decelerated the declines. In the older-old, physical activity was associated with less decline in BMI and CC, while any of vascular disorders was related to faster decline in BMI and MAC though not statistically significant for CC.

Previous longitudinal studies investigating BMI trajectories over the lifespan have indicated that BMI increases until the age of 65 years, thereafter levelling off and starting to decline after the age of 80 [1,3]. In line with these results, we observed a stable or a slight decline in BMI among participants aged 60–75 years and a steeper decline after the age of 78 years over the 15-year follow-up. Our results also showed that CC and MAC declined significantly even among participants aged 60 years and had a steeper decline than BMI over time. Involuntary loss of muscle mass has been reported to start after 50 years, and the decline of BMI in older adults was likely mainly due to the loss of muscle mass [1,2]. However, BMI was not sensitive to detect muscle mass change in younger-old adults, which was also shown in our study. One possible explanation is that the decline in muscle mass might be masked by the increase in fat mass in early late-life [1,28]. CC and MAC have shown high correlations with muscle mass and are easy to perform [11–14]. Moreover, CC was also suggested by the European Working Group on Sarcopenia in Older People to be a proxy of muscle quantity in the absence of other muscle mass methods [8]. These anthropometric measures may be more suitable and practical than using expensive imaging techniques (such as DEXA) in large-scale population-based studies of older adults. To our best knowledge, no previous studies have simultaneously assessed the trajectories of the three indices over time in an older population. The simultaneous decline in the three measures supports the notion of progressive muscle mass loss in ageing [29].

Modifiable factors, such as socioeconomic status and healthy lifestyle, might affect ageing-related body composition changes. Education is commonly used to reflect socioeconomic status. Previous evidence showed that people with less education were more likely to experience weight loss in late life [7,30]. Similarly, we found that university education seemed to be protective against the decline in BMI, CC and MAC, especially for younger-old adults. A prospective study conducted in community-dwelling participants observed that physical activity was associated with the maintenance of muscle mass [31]. The current study further highlighted the possible beneficial effect of physical activity in preserving BMI and CC, especially among older-old adults. Guidelines also recommend physical activity for adults aged ≥65 years to maintain healthy body composition [32]. Existing literature from both human and rodent studies suggests that physical activity could help prevent muscle mass loss by improving mitochondrial function [33,34]. Furthermore, exercise could stimulate numerous signalling pathways that increase protein synthesis [35]. Moreover, we

Table 2. β_{slope} and 95% CI of the associations of sociodemographic, lifestyle and health conditions with changes in BMI, CC and MAC over 15 years: results from linear mixed models among participants aged <78 years

Factors	BMI		CC		MAC	
	β_{slope} (95% CI)[a]	$\beta_{difference\ in\ slope}$ (95% CI)[b]	β_{slope} (95% CI)[a]	$\beta_{difference\ in\ slope}$ (95% CI)[b]	β_{slope} (95% CI)[a]	$\beta_{difference\ in\ slope}$ (95% CI)[b]
Sex						
Male	−0.016 (−0.033 to 0.001)	Reference	**−0.088 (−0.106 to −0.070)**	Reference	**−0.091 (−0.108 to −0.074)**	Reference
Female	**−0.038 (−0.052 to −0.024)**	**−0.026 (−0.049 to −0.003)**	**−0.064 (−0.079 to −0.049)**	0.016 (−0.008 to 0.041)	**−0.101 (−0.115 to −0.087)**	−0.014 (−0.036 to 0.009)
Education						
Elementary/professional school	**−0.048 (−0.065 to −0.031)**	Reference	**−0.090 (−0.108 to −0.073)**	Reference	**−0.112 (−0.128 to −0.096)**	Reference
High school	**−0.047 (−0.079 to −0.016)**	−0.006 (−0.041 to 0.030)	**−0.090 (−0.123 to −0.057)**	0.002 (−0.036 to 0.039)	**−0.117 (−0.148 to −0.087)**	−0.009 (−0.043 to 0.026)
University	**−0.007 (−0.023 to 0.009)**	**0.031 (0.008 to 0.054)**	**−0.054 (−0.071 to −0.037)**	**0.034 (0.009 to 0.058)**	**−0.077 (−0.093 to −0.062)**	**0.029 (0.006 to 0.052)**
Smoking status						
Never smoker	**−0.032 (−0.049 to −0.015)**	Reference	**−0.060 (−0.078 to − 0.042)**	Reference	**−0.099 (−0.115 to − 0.082)**	Reference
Ex-smoker	**−0.028 (−0.045 to −0.011)**	0.001 (−0.023 to 0.024)	**−0.083 (−0.101 to −0.066)**	−0.022 (−0.047 to 0.003)	**−0.101 (−0.117 to −0.085)**	−0.005 (−0.028 to 0.019)
Current smoker	−0.025 (−0.053 to 0.003)	0.008 (−0.025 to 0.041)	**−0.088 (−0.117 to −0.058)**	−0.025 (−0.060 to 0.010)	**−0.082 (−0.110 to −0.054)**	0.019 (−0.014 to 0.052)
Alcohol consumption						
No or occasional	**−0.036 (−0.059 to −0.012)**	−0.023 (−0.053 to 0.007)	**−0.069 (−0.094 to −0.044)**	0.005 (−0.027 to 0.036)	**−0.101 (−0.124 to −0.078)**	−0.010 (−0.039 to 0.019)
Light to moderate	**−0.018 (−0.032 to −0.004)**	Reference	**−0.076 (−0.090 to −0.061)**	Reference	**−0.091 (−0.105 to −0.077)**	Reference
Heavy	**−0.036 (−0.059 to −0.012)**	−0.011 (−0.039 to 0.017)	**−0.069 (−0.094 to −0.044)**	0.007 (−0.023 to 0.037)	**−0.101 (−0.124 to −0.078)**	−0.007 (−0.035 to 0.020)
Physical activity						
Inactive	**−0.040 (−0.067 to −0.013)**	Reference	**−0.093 (−0.122 to −0.064)**	Reference	**−0.102 (−0.129 to −0.076)**	Reference
Moderate	**−0.034 (−0.050 to −0.019)**	0.004 (−0.027 to 0.035)	**−0.073 (−0.089 to −0.057)**	0.017 (−0.016 to 0.050)	**−0.100 (−0.115 to −0.085)**	0.003 (−0.028 to 0.034)
Vigorous	−0.016 (−0.035 to 0.003)	0.018 (−0.015 to 0.051)	**−0.066 (−0.086 to −0.046)**	0.023 (−0.013 to 0.058)	**−0.090 (−0.108 to −0.071)**	0.012 (−0.021 to 0.044)
Vascular disorders [c]						
None	−0.006 (−0.025 to 0.013)	Reference	**−0.046 (−0.066 to −0.026)**	Reference	**−0.073 (−0.092 to −0.054)**	Reference
Any	**−0.041 (−0.054 to −0.027)**	**−0.028 (−0.051 to −0.005)**	**−0.088 (−0.102 to −0.074)**	**−0.035 (−0.060 to −0.011)**	**−0.108 (−0.121 to −0.095)**	**−0.030 (−0.053 to −0.008)**
Diabetes						
No	**−0.021 (−0.033 to −0.010)**	Reference	**−0.068 (−0.080 to −0.057)**	Reference	**−0.091 (−0.102 to −0.080)**	Reference
Yes	**−0.145 (−0.189 to −0.101)**	**−0.118 (−0.163 to −0.072)**	**−0.158 (−0.205 to −0.112)**	**−0.075 (−0.123 to −0.026)**	**−0.186 (−0.229 to −0.143)**	**−0.088 (−0.133 to −0.044)**

[a]β_{slope} (i.e. annual change) for the associations of each factor with changes in BMI, CC and MAC, including the specific factor, follow-up years and their interaction term (factor × follow-up time) in the model. [b]Difference between β_{slope} for the associations of each factor with changes in BMI, CC and MAC, adjusting all factors in the table and their interaction term with follow-up years (factors × follow-up time). [c]Vascular disorder was coded as none, or any of ischemic heart disease, heart failure, cerebrovascular disease or hypertension. Bold values are statistically significant.

found that the effects of physical activity on CC decline were significant among females but not males. This may be because mitochondrial function in females is more sensitive to physical activity [36]. Given the adverse effects of muscle mass loss in older adults, more evidence from intervention studies with sufficient sample sizes and relatively long-term follow-up are warranted to better understand the effect of physical activity in combating muscle mass loss and its role in the promotion of healthy ageing.

The current study also showed that chronic diseases, such as diabetes and vascular disorders, could accelerate the decline in BMI, CC and MAC. These results were consistent with previous literature [3,18,37,38]. A previous study among older Swedish twins reported a steeper decline in BMI in association with type 2 diabetes among female but not male [3]. Research from the Health, Ageing and Body Composition Study also found that older adults (70–79 years) with type 2 diabetes showed more muscle

Table 3. β_{slope} and 95% CI of the associations of sociodemographic, lifestyle and health conditions with changes in BMI, CC and MAC over 15 years: results from linear mixed models among participants aged ≥78 years

Factors	BMI		CC		MAC	
	β_{slope} (95% CI)[a]	$\beta_{difference\ in\ slope}$ (95% CI)[b]	β_{slope} (95% CI)[a]	$\beta_{difference\ in\ slope}$ (95% CI)[b]	β_{slope} (95% CI)[a]	$\beta_{difference\ in\ slope}$ (95% CI)[b]
Sex						
Male	**−0.193 (−0.243 to −0.143)**	Reference	**−0.203 (−0.249 to −0.158)**	Reference	**−0.209 (−0.248 to −0.169)**	Reference
Female	**−0.246 (−0.278 to −0.215)**	−0.030 (−0.094 to 0.034)	**−0.235 (−0.263 to −0.206)**	−0.016 (−0.074 to 0.043)	**−0.233 (−0.257 to −0.208)**	−0.025 (−0.076 to 0.026)
Education						
Elementary/professional school	**−0.246 (−0.277 to −0.214)**	Reference	**−0.238 (−0.266 to −0.209)**	Reference	**−0.234 (−0.259 to −0.209)**	Reference
High school	**−0.174 (−0.268 to −0.080)**	0.029 (−0.073 to 0.131)	**−0.207 (−0.292 to −0.121)**	0.012 (−0.082 to 0.105)	**−0.210 (−0.284 to −0.136)**	0.000 (−0.080 to 0.081)
University	**−0.205 (−0.264 to −0.147)**	0.021 (−0.047 to 0.090)	**−0.196 (−0.249 to −0.144)**	0.025 (−0.036 to 0.087)	**−0.206 (−0.252 to −0.160)**	0.022 (−0.032 to 0.076)
Smoking status						
Never smoker	**−0.241 (−0.275 to −0.206)**	Reference	**−0.240 (−0.271 to −0.208)**	Reference	**−0.226 (−0.253 to −0.199)**	Reference
Ex-smoker	**−0.215 (−0.260 to −0.170)**	0.007 (−0.052 to 0.066)	**−0.209 (−0.250 to −0.169)**	0.020 (−0.033 to 0.074)	**−0.234 (−0.270 to −0.199)**	−0.021 (−0.068 to 0.026)
Current smoker	**−0.257 (−0.358 to −0.156)**	−0.030 (−0.138 to 0.077)	**−0.209 (−0.299 to −0.120)**	0.026 (−0.070 to 0.122)	**−0.198 (−0.278 to −0.119)**	0.015 (−0.070 to 0.100)
Alcohol consumption						
No or occasional	**−0.223 (−0.297 to −0.148)**	−0.043 (−0.103 to 0.018)	**−0.178 (−0.246 to −0.110)**	0.014 (−0.041 to 0.068)	**−0.209 (−0.269 to −0.149)**	−0.000 (−0.048 to 0.047)
Light to moderate	**−0.204 (−0.243 to −0.165)**	Reference	**−0.231 (−0.266 to −0.196)**	Reference	**−0.224 (−0.254 to −0.194)**	Reference
Heavy	**−0.223 (−0.297 to −0.148)**	−0.023 (−0.109 to 0.064)	**−0.178 (−0.246 to −0.110)**	0.048 (−0.031 to 0.127)	**−0.209 (−0.269 to −0.149)**	0.005 (−0.064 to 0.074)
Physical activity						
Inactive	**−0.287 (−0.339 to −0.234)**	Reference	**−0.303 (−0.350 to −0.255)**	Reference	**−0.264 (−0.306 to −0.222)**	Reference
Moderate	**−0.226 (−0.261 to −0.190)**	0.051 (−0.013 to 0.116)	**−0.214 (−0.246 to −0.182)**	**0.088 (0.029 to 0.147)**	**−0.216 (−0.244 to −0.188)**	0.049 (−0.003 to 0.100)
Vigorous	**−0.182 (−0.243 to −0.120)**	**0.087 (0.005 to 0.169)**	**−0.165 (−0.220 to −0.110)**	**0.132 (0.057 to 0.207)**	**−0.215 (−0.263 to −0.168)**	0.044 (−0.021 to 0.109)
Vascular disorders [c]						
None	**−0.137 (−0.210 to −0.064)**	Reference	**−0.189 (−0.256 to −0.123)**	Reference	**−0.152 (−0.208 to −0.095)**	Reference
Any	**−0.246 (−0.274 to −0.218)**	**−0.110 (−0.191 to −0.030)**	**−0.232 (−0.258 to −0.207)**	−0.035 (−0.108 to 0.038)	**−0.238 (−0.260 to −0.216)**	**−0.093 (−0.157 to −0.030)**
Diabetes						
No	**−0.232 (−0.260 to −0.204)**	Reference	**−0.228 (−0.253 to −0.203)**	Reference	**−0.227 (−0.249 to −0.206)**	Reference
Yes	**−0.227 (−0.322 to −0.132)**	0.033 (−0.066 to 0.133)	**−0.214 (−0.301 to −0.127)**	0.029 (−0.063 to 0.121)	**−0.211 (−0.288 to −0.135)**	0.034 (−0.047 to 0.115)

[a] β_{slope} (i.e. annual change) for the associations of each factor with changes in BMI, CC and MAC, including the specific factor, follow-up years and their interaction term (factor × follow-up time) in the model. [b] Difference between β_{slope} for the associations of each factor with changes in BMI, CC and MAC, adjusting all factors in the table and their interaction term with follow-up years (factors × follow-up time). [c] Vascular disorder was coded as none, or any of ischemic heart disease, heart failure, cerebrovascular disease or hypertension. Bold values are statistically significant.

mass loss than participants without diabetes [18]. In line with these results, we found that younger-old adults with type 2 diabetes had faster declines in BMI, CC and MAC, with stronger associations among females than males. The reasons for the lack of associations between diabetes and declines in BMI, CC and MAC in older-old adults could be partly due to selective survival, as diabetes is related to mortality [39]. Moreover, greater muscle mass loss has been observed among older adults with vascular disorders [37,38]. Fülster and colleagues reported a higher prevalence of muscle wasting in participants with heart failure from a population-based study [38]. We also found that vascular disorders could accelerate the decline in BMI, CC and MAC both in younger-old and older-old adults. At the biological level, diabetes and vascular disorders are characterized by an increased level of inflammatory markers [37,40], which had been related to decreased muscle mass [41]. It is plausible that the accelerated decline of muscle mass in these

diseases is partly due to shared pathophysiology pathways [37,40,41].

Strengths of our study include the population-based longitudinal study design with a long follow-up and repeated anthropometric measurements. However, some limitations need to be pointed out. Firstly, participants included in the main analyses were healthier than those who were excluded, which may lead to the underestimation of both the decline over time and associations between factors (socio-demographic, lifestyle and health-related factors) and trajectories of measurements. Secondly, BMI was self-reported but not measured in a sub-sample population ($n = 462$). However, the correlation between self-reported and measured BMI was high (Pearson correlation coefficient's $r = 0.95$, $P < 0.001$). Thirdly, MAC includes subcutaneous fat around the biceps and triceps which might limit its usefulness as a proxy for muscle mass. To this end, previous literature has recommended calculating arm muscle using both arm circumference and triceps skinfold thickness [42], but skinfold thickness was not available in SNAC-K. Fourthly, we were not able to examine the trajectory of visceral fat, which accumulates with ageing and has been associated with several cardio-metabolic chronic diseases and mortality [43]. Fifthly, misclassification may have occurred given that data on lifestyle variables (e.g. smoking habits, alcohol consumption and physical activity) were based on self-report. Sixthly, information on the type of physical activity (i.e. resistance training and aerobic exercise)—which may have different health benefits—was not available [44]. Thus, we could not address the association between muscle mass change and the specific type of exercise. Finally, although we considered a wide range of sociodemographic, lifestyle and health conditions as potential factors, residual confounding could not be completely ruled out.

In summary, BMI, CC and MAC declined significantly over the 15-year follow-up. CC and MAC could detect the decline in muscle mass earlier than BMI, and these declines were more pronounced in older-old (≥78 years) vs. younger-old (<78 years) participants. Furthermore, we found that vascular disorders and diabetes accelerated the declines of BMI, CC and MAC in younger-older adults, but higher education was associated with reduced decline in all three measures. Physical activity seemed to be more beneficial for decelerating the declines in BMI and CC in older-old adults. Our findings underscore the role of social, medical and lifestyle factors in the changes in anthropometric measures in older age.

Supplementary Data: Supplementary data mentioned in the text are available to subscribers in *Age and Ageing* online.

Acknowledgements: The authors would like to express their gratitude to the participants and staff involved in the data collection and management in the SNAC-K study.

Declaration of Sources of Funding: SNAC-K (http://www.snac.org) is financially supported by the Swedish Ministry of Health and Social Affairs; participating County Councils and Municipalities; the Swedish Research Council and Swedish Research Council for Health, Working Life and Welfare. W.X. received grants from the Swedish Research Council (No. 2017–00981), the National Natural Science Foundation of China (No. 81771519) and Demensfonden, the Konung Gustaf V:s och Drottning Victorias Frimurare Foundation (No. 2016–2017). J.G. also received grants from the China Scholarship Council (No. 201808340062) and private foundation (Lindhés Advokatbyrå AB; Stiftelsen För Gamla Tjänarinnor).

Declaration of Conflicts of Interest: None.

References

1. Jackson AS, Janssen I, Sui X, Church TS, Blair SN. Longitudinal changes in body composition associated with healthy ageing: men, aged 20–96 years. Br J Nutr 2012; 107: 1085–91.
2. Ding J, Kritchevsky SB, Newman AB *et al.* Effects of birth cohort and age on body composition in a sample of community-based elderly. Am J Clin Nutr 2007; 85: 405–10.
3. Dahl AK, Reynolds CA, Fall T, Magnusson PKE, Pedersen NL. Multifactorial analysis of changes in body mass index across the adult life course: a study with 65 years of follow-up. Int J Obes (Lond) 2014; 38: 1133–41.
4. Murphy RA, Patel KV, Kritchevsky SB *et al.* Weight change, body composition, and risk of mobility disability and mortality in older adults: a population-based cohort study. J Am Geriatr Soc 2014; 62: 1476–83.
5. Fantin F, Di Francesco V, Fontana G *et al.* Longitudinal body composition changes in old men and women: interrelationships with worsening disability. Journals Gerontol - Ser A Biol Sci Med Sci 2007; 62: 1375–81.
6. Alhurani RE, Vassilaki M, Aakre JA *et al.* Decline in weight and incident mild cognitive impairment: Mayo Clinic study of aging. JAMA Neurol 2016; 73: 439–46.
7. Al Snih S, Raji MA, Markides KS, Ottenbacher KJ, Goodwin JS. Weight change and lower body disability in older Mexican Americans. J Am Geriatr Soc 2005; 53: 1730–7.
8. Cruz-Jentoft AJ, Bahat G, Bauer J *et al.* Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing 2019; 48: 16–31.
9. Brown JC, Harhay MO, Harhay MN. Sarcopenia and mortality among a population-based sample of community-dwelling older adults. J Cachexia Sarcopenia Muscle 2016; 7: 290–8.
10. Wang DXM, Yao J, Zirek Y, Reijnierse EM, Maier AB. Muscle mass, strength, and physical performance predicting activities of daily living: a meta-analysis. J Cachexia Sarcopenia Muscle 2020; 11: 3–25.
11. Kawakami R, Murakami H, Sanada K *et al.* Calf circumference as a surrogate marker of muscle mass for diagnosing sarcopenia in Japanese men and women. Geriatr Gerontol Int 2015; 15: 969–76.
12. Asai C, Akao K, Adachi T *et al.* Maximal calf circumference reflects calf muscle mass measured using magnetic resonance imaging. Arch Gerontol Geriatr 2019; 83: 175–8.

13. Sanchez FF, Faganello MM, Tanni SE *et al.* Anthropometric midarm measurements can detect systemic fat-free mass depletion in patients with chronic obstructive pulmonary disease. Braz J Med Biol Res 2011; 44: 453–9.

14. Santos LP, Gonzalez MC, Orlandi SP, Bielemann RM, Barbosa-Silva TG, Heymsfield SB. New prediction equations to estimate appendicular skeletal muscle mass using calf circumference: results from NHANES 1999–2006. J Parenter Enteral Nutr 2019; 43: 998–1007.

15. Janssen I, Heymsfield SB, Wang Z, Ross R. Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. J Appl Physiol 2000; 89: 81–8.

16. Chao AM, Wadden TA, Tronieri JS, Berkowitz. Alcohol intake and weight loss during intensive lifestyle intervention for adults with overweight or obesity and diabetes. Obesity (Silver Spring) 2019; 27: 30–40.

17. Kyle UG, Melzer K, Kayser B, Picard-Kossovsky M, Gremion G, Pichard C. Eight-year longitudinal changes in body composition in healthy Swiss adults. J Am Coll Nutr 2006; 25: 493–501.

18. Park SW, Goodpaster BH, Lee JS *et al.* Excessive loss of skeletal muscle mass in older adults with type 2 diabetes. Diabetes Care 2009; 32: 1993–7.

19. Sisto IR, Hauck M, Plentz RDM. Muscular atrophy in cardiovascular disease. In: Xiao J. (eds) Muscle Atrophy. Advances in Experimental Medicine and Biology, vol 1088 Singapore: Springer, 2018; 369–91.

20. Al-Sofiani ME, Ganji SS, Kalyani RR. Body composition changes in diabetes and aging. J Diabetes Complications 2019; 33: 451–9.

21. Leite MLC, Nicolosi A. Lifestyle correlates of anthropometric estimates of body adiposity in an Italian middle-aged and elderly population: a covariance analysis. Int J Obes (Lond) 2006; 30: 926–34.

22. Wang R, Fratiglioni L, Laveskog A *et al.* Do cardiovascular risk factors explain the link between white matter hyperintensities and brain volumes in old age? A population-based study. Eur J Neurol 2014; 21: 1076–82.

23. Dohrn IM, Dohrn IM, Gardiner PA *et al.* Device-measured sedentary behavior and physical activity in older adults differ by demographic and health-related factors. Eur Rev Aging Phys Act 2020; 17: 8.

24. Calderón-Larrañaga A, Vetrano DL, Onder G *et al.* Assessing and measuring chronic multimorbidity in the older population: a proposal for its operationalization. Journals Gerontol - Ser A Biol Sci Med Sci 2017; 72: 1417–23.

25. American Diabetes Association. 2. Classification and diagnosis of diabetes. Diabetes Care 2017; 40: S11–24.

26. Association APA-DAP. U. *Diagnostic and Statistical Manual of Mental Disorders IV*. 1994.

27. World Health Organization. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser 1995; 854:1–452.

28. Koster A, Visser M, Simonsick EM *et al.* Association between fitness and changes in body composition and muscle strength. J Am Geriatr Soc 2010; 58: 219–26.

29. Ryall JG, Schertzer JD, Lynch GS. Cellular and molecular mechanisms underlying age-related skeletal muscle wasting and weakness. Biogerontology 2008; 9: 213–28.

30. Tyrovolas S, Koyanagi A, Olaya B *et al.* Factors associated with skeletal muscle mass, sarcopenia, and sarcopenic obesity in older adults: a multi-continent study. J Cachexia Sarcopenia Muscle 2016; 7: 312–21.

31. Scott D, Blizzard L, Fell J, Jones G. Prospective associations between ambulatory activity, body composition and muscle function in older adults. Scand J Med Sci Sports 2011; 21: e168–75.

32. World Health Organization. Global recommendations on physical activity for health. 2010.

33. Zampieri S, Pietrangelo L, Loefler S *et al.* Lifelong physical exercise delays age-associated skeletal muscle decline. J Gerontol A Biol Sci Med Sci 2015; 70: 163–73.

34. Sandri M, Lin J, Handschin C *et al.* PGC-1 protects skeletal muscle from atrophy by suppressing FoxO3 action and atrophy-specific gene transcription. Proc Natl Acad Sci 2006; 103: 16260–5.

35. Bowen TS, Schuler G, Adams V. Skeletal muscle wasting in cachexia and sarcopenia: molecular pathophysiology and impact of exercise training. J Cachexia Sarcopenia Muscle 2015; 6: 197–207.

36. Callahan DM, Bedrin NG, Subramanian M *et al.* Age-related structural alterations in human skeletal muscle fibers and mitochondria are sex specific: relationship to single-fiber function. J Appl Physiol 2014; 116: 1582–92.

37. Scherbakov N, von Haehling S, Anker SD, Dirnagl U, Doehner W. Stroke induced sarcopenia: muscle wasting and disability after stroke. Int J Cardiol 2013; 170: 89–94.

38. Fulster S, Tacke M, Sandek A *et al.* Muscle wasting in patients with chronic heart failure: results from the studies investigating co-morbidities aggravating heart failure (SICA-HF). Eur Heart J 2013; 34: 512–9.

39. Alegre-Díaz J, Herrington W, López-Cervantes M *et al.* Diabetes and cause-specific mortality in Mexico City. N Engl J Med 2016; 375: 1961–71.

40. Kalyani RR, Corriere M, Ferrucci L. Age-related and disease-related muscle loss: the effect of diabetes, obesity, and other diseases. Lancet Diabetes Endocrinol 2014; 2: 819–29.

41. Wei LC, Yu K, Shyh-Chang N *et al.* Circulating factors associated with sarcopenia during ageing and after intensive lifestyle intervention. J Cachexia Sarcopenia Muscle 2019; 10: 586–600.

42. Noori N, Kopple JD, Kovesdy CP *et al.* Mid-arm muscle circumference and quality of life and survival in maintenance Hemodialysis patients. Clin J Am Soc Nephrol 2010; 5: 2258–68.

43. Folsom AR, Kushi LH, Anderson KE *et al.* Associations of general and abdominal obesity with multiple health outcomes in older women: the Iowa Women's health study. Arch Intern Med 2000; 160: 2117–28.

44. Egan B, Zierath JR. Exercise metabolism and the molecular regulation of skeletal muscle adaptation. Cell Metab 2013; 17: 162–84.

Received 21 September 2020; editorial decision 31 January 2021