



Short Communication

Associations of body mass index trajectories, weight change with mortality among the oldest old: do they differ from general older adults?

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Body mass index (BMI) is widely recognized as a reasonably good measure of general adiposity and an important predictor of mortality risk [1]. Meta-analyses of cohort studies [2] have demonstrated a “U” or “J”-shaped correlation, wherein both low and high BMI values are associated with an increased risk of mortality. The association between BMI and mortality may vary depending on age. Among young and middle-aged adults, obesity is associated with higher mortality risk [3]; but for older adults aged over 65 years, “obesity paradox” is more likely to be observed [4]. One recent study observed that compared with normal weight in the oldest old (aged ≥ 80 years), overweight and obese were associated with significantly decreased risk of non-cardiovascular disease (non-CVD) and all-cause mortality, but not cardiovascular disease (CVD) mortality [5]. However, many of these findings are based on limited BMI measurements that were measured at one time point. Some investigations have found that repeated measurements of weight or BMI status are better in predicting mortality than one-time measurement of weight or BMI status, especially among older adults [6]. The use of dynamic measurements could provide more information on changes of body weight over time, and help to clarify the true association of BMI with mortality risk [7].

One systematic review article reported that weight fluctuation, along with weight gain or weight loss, was associated with increased mortality risk among community-dwelling residents aged ≥ 65 years [8]. Most studies which reported BMI (or weight) changes focused only on the associations of the changes with death events between two visit times during the study period [5]. Since there are limitations to defining subgroups of individuals based on short-term weight changes, this method ignores the heterogeneity in which body weight may be fluctuating [9]. A number of studies have investigated the associations of BMI trajectory with a variety of outcomes [10,11], including diabetes and cognitive impairment, have been reported. These studies mainly focused on young adults or adults aged ≥ 60 years, very few involved the oldest old. BMI trajectories of older adults are different from young or middle-aged adults [12], and BMI trajectories of the oldest old may also be different from that of adults aged 60 to 79 years.

Using data from the Chinese Longitudinal Healthy Longevity Survey, we aim to explore the association of potential BMI dynamic changing trajectories of the oldest old with the risk of all-cause, CVD and non-CVD mortality. The study was approved by the biomedical ethics committee of Peking University (IRB00001052-13074). All participants provided written informed consent. Detailed method description can be found in the [Supplementary materials](#).

A total of 10,883 participants aged ≥ 80 years satisfied inclusion criteria (Table S1; Fig. S1 online) were included in the analyses. The

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mean age was 89.7 years (Standard deviation = 6.9) and 4411 (40.5%) were male. The median follow-up year was 4.8 (interquartile range: 3.1–6.2) years.

We identified 3 distinct trajectories of BMI by the latent class growth mixed model in these individuals (Fig. 1a), named as low-stable ($n = 10,124$, baseline BMI = 18.8 ± 3.0 kg/m²), decreasing ($n = 376$, baseline BMI = 26.9 ± 2.6 kg/m²) and increasing ($n = 383$, baseline BMI = 24.1 ± 3.9 kg/m²). The optimal model was selected based on lower Bayesian information criterion (BIC = 143908.2), relatively higher posterior probabilities (ranged in 0.705–0.922), and proper percentage of group memberships (93.03%, 3.45% and 3.52% for each BMI trajectory, respectively). The model fitting process is shown in Table S2 (online). The low-stable group shows a slightly decreasing trend. BMI decreased gradually from 19.9 kg/m² at age 80 years to 18.2 kg/m² at age 105 years. In the decreasing group, BMI decreased more dramatically, ranging from 25.4 kg/m² to 18.1 kg/m² between age 80 to 105 years. While in the increasing group, BMI gradually increased from normal weight to overweight between age 80 to 105 years. The increasing trend was initially rapid but tended to stabilize after age 90. The proportion of male and participants with hypertension, diabetes, and cerebrovascular disease in the decreasing group were higher than other groups (Table S1 online). The baseline characteristics by initial BMI level are shown in Table S3 (online).

Table 1 shows hazard ratios (HRs)/ subdistribution hazard ratios (SHRs) and 95% confidence intervals (CIs) for the associations of the 3 trajectory groups with mortality. 7701 deaths occurred during 50,513 person-years of follow-up from 1998 to 2018. Among these decedents, 4730 (61.4%) participants had a recorded cause of death, of which 885 died from CVD and 3845 died from non-CVD. Compared with the low-stable group, counterparts in the increasing group had a significantly lower risk of all-cause and non-CVD mortality after adjusting for some demographic factors and healthy factors. The HR and SHR (95% CI) of all-cause and non-CVD mortality for the increasing group were 0.87 (0.76, 0.99) and 0.82 (0.67, 0.99), respectively. While the decreasing group had a significant higher risk of non-CVD mortality. The HR and SHR were 1.10 (0.94, 1.28) for all-cause mortality and 1.30 (1.07, 1.58) for non-CVD mortality. Neither of the two groups had significantly higher or lower risk for CVD mortality.

Fig. 1b shows odds ratios (ORs) and 95% CIs of predicted BMI levels and slopes for mortality, with adjustment for sex, smoking, baseline BMI, activities of daily living (ADL), cognition impairment, diabetes, hypertension and cerebrovascular disease. For all-cause mortality, the ORs of predicted BMI levels slightly decreased from 0.89 (0.87, 0.91) to 0.83 (0.81, 0.86) during 80 to 101 years, then increased gradually to 0.86 (0.83, 0.89) at age 105. The ORs of BMI slopes increased from 0.83 (0.79, 0.86) to 1.25 (1.2, 1.31) between ages 80 to 105 years, with positive association between slopes and all-cause mortality at age 95 years (OR (95%CI): 1.05 (1.01, 1.09)). The association of BMI slopes with non-CVD mortality became positive over the age of 95 years. No significant associations were found for both BMI levels and slopes for CVD mortality (Fig. 1c). Differences in BMI levels and slopes for all-cause, CVD and non-CVD mortality between survivors and deceased at each age point can be found in Tables S4–S6 (online).

Weight loss was associated with high risk of all-cause and non-CVD mortality for all initial BMI levels (Fig. S3 online). The maximum values of HRs were 1.76 (1.31–2.36) in underweight, 1.25 (1.08–1.46) in normal weight, 1.53 (1.24–1.90) in overweight or obese for all-cause mortality, and for non-CVD mortality were 1.79 (1.33–2.41), 1.33 (1.01–1.77) and 1.78 (1.28–2.50), respectively. Weight gain had a significant association with low risk of non-CVD mortality in underweight individuals. There are no significant associations between weight change and CVD mortality.

We did sensitivity analyses by testing models with at least three BMI measurements (Fig. S4 online), as LCGMMs are flexible enough to deal with different observation times between participants. The trends of BMI trajectories were similar with the main results. Predicted trajectories of BMI for the oldest old by sex are shown in Fig. S5 (online). Confounding factors such as diabetes, hypertension, and cerebrovascular disease in different BMI trajectories were balanced after using propensity score matching (Table S7 online). The association of BMI trajectories with decreased CVD, non-CVD and all-cause mortality was essentially unchanged (Table S8 online).

In this cohort study, 3 BMI trajectory subgroups were identified among the older adults aged 80 and above and found significant associations of distinct trajectories with all-cause mortality and non-CVD mortality. More than 90% of the membership were in the low-stable group, followed a steady decreasing pattern, predicting the average BMI decreased from normal weight to underweight. The increasing group had lower all-cause and non-CVD mortality than the low-stable group, while the decreasing group had higher non-CVD mortality.

Previous studies have identified trajectories of BMI at specific ages over the life course in general population or patients with chronic diseases, such as type 2 diabetes [10]. In these studies, different group numbers (3 to 5) and changing patterns of BMI trajectories were ascertained by follow-up time or age, with association of mortality or different health outcomes. To our knowledge, this is the first analysis for the association of BMI trajectories with mortality risk in Chinese oldest old. One Japanese study [13] identified four BMI trajectory groups over a 19-year period among older adults (age ≥ 60 years) and found that older Japanese who stayed overweight had the lowest mortality risk. Another study in Republic of Korea [14] classified participants (age ≥ 65 years) into 4 groups: low-normal, high-normal, overweight and obesity, and all the groups showed a slight decline in BMI during 10 years follow-up period. But these studies failed to find significant changes in BMI. Different from the two studies on Asian older adults, we identified an increasing BMI trajectory group, which increasing from underweight to a high-normal level, and a rapidly decreasing BMI trajectory group, with BMI decreasing from a high level to underweight. The BMI trajectory of young or middle-aged adults mainly showed an increasing trend [6], while the BMI of the elderly, especially the oldest old, mainly showed a decreasing trend [14]. BMI enters a decline after age 65 in general, and the average BMI level of adults over 80 years was lower than that of the adults over 65 years. The observations from this and other studies [10,13] suggest that changes in BMI are important for predicting death.

Weight loss of older adults was associated with an increase risk in mortality [8], extending to the death cause of cancer, CVD, and a range of other life-limiting conditions. The human body tends to lose muscle mass with advancing age, which can have profound effects on overall health and functionality. In our study, weight loss was associated with elevated all-cause and non-CVD mortality risk at all initial BMI levels, but not in CVD mortality. The association of weight loss with all-cause mortality was primarily due to a reduction in non-CVD mortality, which was similar to the previous study [5].

Our study found a notable period at age 95 years and above by the analyses of BMI slopes. The association analyses presented that the ORs for slopes of BMI changing were higher than BMI levels after age 85 for all-cause and non-CVD mortality, and the association became positive at age 95 and above. This result indicated that the rate of BMI change maybe a better predictor of mortality than BMI level in late life. The reason maybe that changes in BMI manifest the physiological adaptability and resilience of the body,

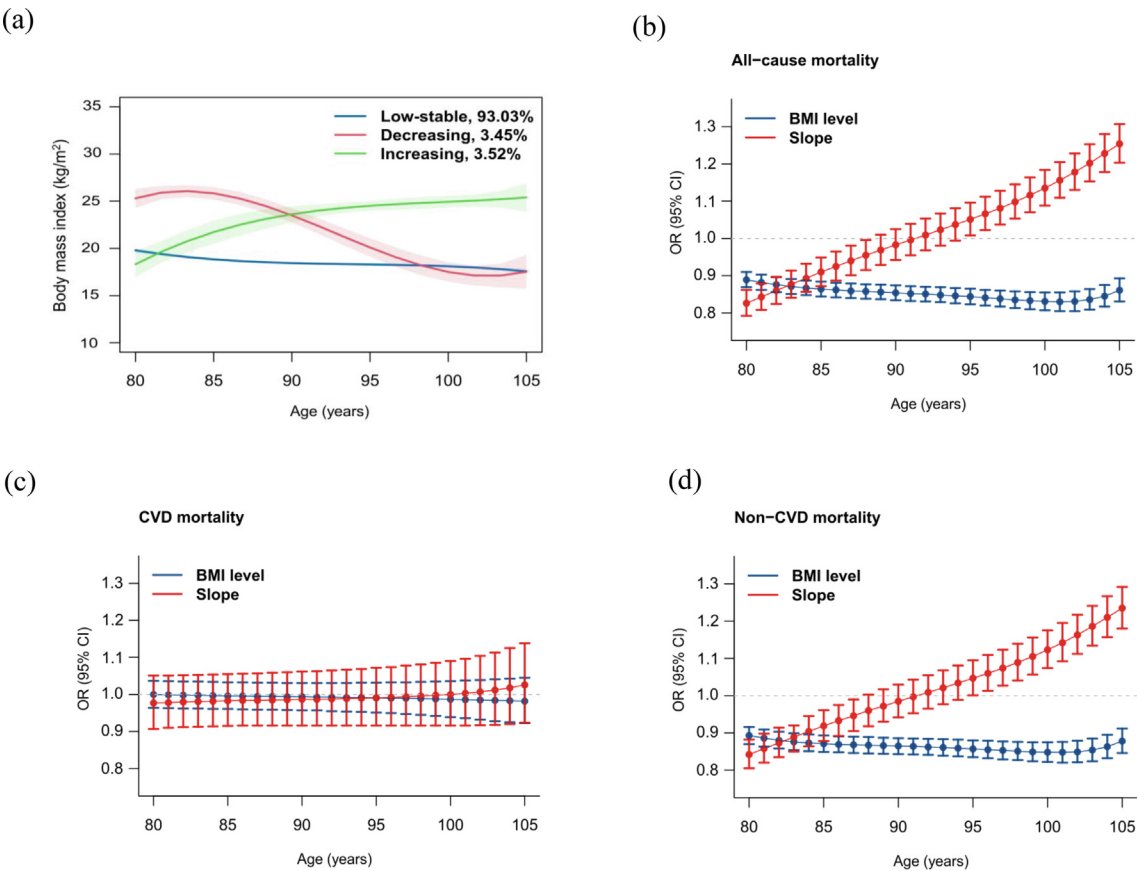


Fig. 1. Trajectories of body mass index for the oldest old and standardized odds ratios and 95% CIs of predicted levels and level-adjusted linear slopes of body mass index by age for all-cause, cardiovascular disease and non-cardiovascular disease mortality. (a) The solid lines show the estimated values of body mass index for members in the groups. Shading around the lines represent confidence bands for the calculated trajectory. (b), (c) and (d) The levels and slopes of BMI estimated by the model at each year of age was fitted to mortality of the individuals by logistic regression model. The dots represent standardized odds ratio of body mass index levels and slopes by age. The line segments indicate 95% CIs. Data were adjusted for sex, smoking, baseline body mass index, activities of daily living, cognition impairment, diabetes, hypertension and cerebrovascular disease.

Table 1
Hazard ratios/subdistribution hazard ratios and 95% CIs of body mass index trajectory groups for all-cause, cardiovascular disease and non-cardiovascular disease mortality among oldest old.

	No. of death (%)	Model 1	Model 2	Model 3	Model 4
All-cause mortality	7701 (70.8)				
Low-stable	7250 (71.6)	1 (Ref.)	1 (Ref.)	1 (Ref.)	1 (Ref.)
Decreasing	207 (55.1)	0.73 (0.64,0.84) ^a	0.92 (0.80,1.06)	1.10 (0.95,1.29)	1.10 (0.94,1.28)
Increasing	244 (63.7)	0.85 (0.75,0.97) ^a	0.80 (0.70,0.91) ^a	0.87 (0.76,0.99) ^a	0.87 (0.76,0.99) ^a
CVD mortality	885 (21.8)				
Low-stable	810 (22.0)	1 (Ref.)	1 (Ref.)	1 (Ref.)	1 (Ref.)
Decreasing	33 (16.3)	1.02 (0.72,1.44)	0.93 (0.65,1.33)	0.80 (0.55,1.19)	0.80 (0.54,1.17)
Increasing	42 (23.2)	1.29 (0.95,1.76)	1.33 (0.98,1.81)	1.24 (0.89,1.71)	1.24 (0.90,1.71)
Non-CVD mortality	3845 (54.7)				
Low-stable	3608 (55.7)	1 (Ref.)	1 (Ref.)	1 (Ref.)	1 (Ref.)
Decreasing	118 (41.1)	0.73 (0.61,0.87) ^a	0.93 (0.78,1.11)	1.31 (1.07,1.59) ^a	1.30 (1.07,1.58) ^a
Increasing	119 (46.1)	0.75 (0.62,0.90) ^a	0.69 (0.57,0.83) ^a	0.82 (0.68,0.99) ^a	0.82 (0.67,0.99) ^a

Model 1 was crude model. Model 2 was adjusted for age and sex. Model 3 was additionally adjusted for material, residence, education, smoking, drinking, exercise, dietary diversity, socio-economic level, psychological condition, baseline body mass index. Model 4 was additionally adjusted for activities of daily living, cognition impairment, diabetes, hypertension, cerebrovascular disease. CI: confidence interval; CVD: cardiovascular disease.
^a $P < 0.05$.

with the crux being their predictive nature of shifts in cardiovascular health status, and their close association with the progression of frailty and sarcopenia in the oldest-old population. This result is similar to a cohort study on community-dwelling older adults which observed that BMI decline is earlier and faster in decedents than survivors [12]. There is a growing difference of BMI for

decedents from that of survivors in the last years of life after adjusting for relevant covariates. While a decline in BMI may a feature of aging, accelerated weight loss may be the result of pathological processes that lead to mortality [15]. The observations of this study propose new perspective on mortality associated with long-term changes in BMI in the oldest old and emphasize the

importance of BMI changing patterns for assessing mortality risk in late life. The velocity of BMI change has a greater impact on all-cause and non-CVD mortality than BMI levels, especially after 95 years.

Our findings underscore the important implications of monitoring and managing BMI change for identifying health impairment and predicting demand for nutritional support and medical care among the oldest old. For the oldest old with relatively low BMI level, measures should be taken to increase their weight appropriately. The study contributes to the development of policies for healthy aging promotion and weight management strategies for the oldest old in China, and also contributes to improving health care for the oldest old by medical and health organizations, as well as medical and nursing service institutions at clinical level. Primary-level medical and long-term care institutions are obligated to establish health records for the elderly, organize regular health examinations, monitor long-term dynamic weight changes, and conduct health education, screening, and follow-up management.

The present study has some strengths. First, we used data from the prospective cohort with a large sample size of the oldest old. This is a long-term stable cohort study followed to 20 years with a 2- to 3-year interval. Furthermore, the LCGMM model, which can identify heterogeneity of individual trajectories, was conducted to identify trajectory subgroups and analyze the association of BMI trajectories with mortality. Additionally, the predicted levels and slopes of BMI were calculated to explore the effect of BMI changing rate and its levels on mortality in the oldest old. Finally, no previous studies have concurrently analyzed changes in weight and BMI trajectories in the oldest old.

However, there remain several limitations. First, Mortality among the very oldest old is high. We only included subjects with at least twice BMI measurements. Participants who died or lost to follow-up at the first follow-up visit were excluded from analysis, which may present survival bias. Second, cause of death information was only recorded for 61.4% of deaths. The missing 38.6% could lead to an underestimation of total CVD or non-CVD mortality, which may misinterpret the actual associations of BMI trajectories with specific cause mortality risk. We may try to supplement this information through the cause of death surveillance database in the future. Third, residual or unmeasured confounding such as undiagnosed chronic diseases may also distort the present findings.

In summary, this prospective cohort study emphasized that the significance of weight gain should be noticed in the oldest old. The current appropriate range and management model of BMI for general adults may not be applicable to the oldest old due to the special physiological changes and nutritional requirements. Strategies aimed at moderately increase body weight might be beneficial to promote longevity of older adults. Future studies such as comparing with the 65–80-year-age group in the same region may be required to validate the findings, and investigate the roles of muscle and fat mass in cause-specific mortality. This could involve using bioelectrical impedance analysis or dual-energy X-ray absorptiometry to assess body composition in the oldest-old, and longitudinal studies to monitor changes in muscle and fat mass, providing insights into their temporal relationship with mortality risk.

Conflict of interest

The authors declare that they have no conflict of interest.

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Author contributions

Xiaoming Shi and Yuebin Lv designed the study and took responsibility for the integrity of the data and the accuracy of the data analysis. Li Qi and Chen Chen did the statistical analysis, data interpretation and drafted the manuscript. Jinhui Zhou, Yuebin Lv, and Xiaoming Shi accessed and verified the data. Jinhui Zhou, Sixin Liu, Jun Wang, Yuan Wei, Wenhui Shi, Yang Li and Tao Zhang provided critical revisions to the manuscript for important intellectual content. All authors read and approved the final manuscript.

Appendix A. Supplementary material

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.scib.2025.01.040>.

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