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The age-related blood pressure trajectories from young-old adults to centenarians: A cohort study

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ABSTRACT

Background: Blood pressure (BP) trajectories among older adults, especially among the oldest-old, are still poorly characterized.
Objective: To investigate the longitudinal trajectories of four BP components with age and their potential influential factors.
Methods: This population-based prospective cohort study included 3315 participants (age 60–105 years, 64.6% women) who were regularly examined from 2001 to 2004 through 2013–2016. The longitudinal trajectories of systolic BP (SBP), diastolic BP (DBP), pulse pressure (PP), and mean arterial pressure (MAP) with age were estimated using linear mixed-effects models.
Results: Overall, SBP and PP increased with age until ~80 years and then declined, whereas DBP and MAP decreased constantly after 60 years of age. The age-related BP trajectories varied by survival time, birth cohort, use of antihypertensive drugs, and heart disease. Specifically, people who survived <2 years after the last visit showed higher levels of BP components before ~80 years, followed by steeper declines in SBP and PP. At the same age, people who were born earlier showed higher BP than those who were born later. People who used antihypertensive drugs had higher BP than those who did not until ~80–90 years old, thereafter BP showed no significant difference. After ~80 years old, people with heart disease showed steeper declines in SBP and PP than those without.
Conclusions: The late-life longitudinal BP trajectories with age vary with demographics, clinical conditions, and contextual factors. These findings may help better understand the age-dependent relationship of BP with health outcomes as well as help achieve optimal BP control in older people.
Perspectives: Competency in medical knowledge: Understanding the age-related blood pressure trajectories and potential influential factors may help improve blood pressure management in older people.
Translational outlook 1: Blood pressure trajectories with age in older adults vary by birth cohort, survival time, antihypertensive therapy, and heart disease. The age-related blood pressure trajectories by birth cohorts are featured with lower blood pressure levels at the same age in more recent birth cohorts, which may partially reflect the improvement of blood pressure control over time.
Translational outlook 2: The age-related blood pressure trajectories in the oldest old (e.g., age ≥ 85 years) are characterized by steeper and faster blood pressure declines associated with heart disease and short survival (e.g., <2 years). This may have implications for the optimal management of blood pressure as well as for the interpretation of the relationships between blood pressure and health outcomes (e.g., death) among the oldest old.

1. Introduction

High blood pressure (BP) is a major risk factor for cardiovascular disease, cognitive decline, and poor survival, but its deleterious effect may decrease with advancing age [1–4], and among the oldest old low BP may anticipate an increased risk of all-cause

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mortality [5–7]. Thus, capturing the longitudinal patterns of BP trajectories with age in older adults, and detecting their influential factors may help better understand the potential pathways linking BP levels with adverse health outcomes.

Beyond systolic BP (SBP) and diastolic BP (DBP), mean arterial pressure (MAP) and pulse pressure (PP), the two hemodynamic parameters derived from SBP and DBP, can be extremely informative in predicting the risk of cardiovascular events and mortality. MAP measures vascular resistance and cardiac output, and PP reflects artery stiffness and wave reflections. It has been suggested that models combining various BP components are superior to those of single components in predicting the risk of cardiovascular disease [8]. However, population-based cohort studies investigating the longitudinal BP trajectories in older people have either missed the oldest old people (e.g., ≥85 years) [2,9–11], or only focused on SBP and DBP [12,13]. Studying the age-related BP trajectories that involve all 4 BP components among the oldest old is important because the relationship between various BP components and health outcomes is much more complex in the oldest old than in middle-aged and young-older adults.

Furthermore, data from the USA (age 18–74 years), the UK (age 35–80 years), and Norway (age 20–89 years) showed that distribution of both SBP and DBP shifted downward from earlier to more recent birth cohorts [14–16]. This suggests the potential birth cohort effects on BP trajectories with age in young and older adults. Whether a birth cohort effect could prejudice patterns of age-related BP trajectories in a population that includes also the oldest old remains to be clarified.

In addition, a population-based study of older adults in northern Sweden suggested that improvement in antihypertensive therapy might partly explain a downward trend in SBP and DBP over time [17]. Moreover, increased variability in SBP from midlife to old age is associated with a higher risk of heart disease and mortality [18]. Conversely, heart disease may also cause fluctuations in BP with age in late life. Finally, age might modify the relationship between BP and mortality in older adults [6,19], suggesting that the age-related trajectories of late-life BP may vary with the length of survival time. However, whether these factors can affect age-related BP changes later in life remains uncertain.

Therefore, in this population-based prospective cohort study of people aged 60–105 years in central Stockholm, Sweden, we aim to investigate the age-related longitudinal trajectories of SBP, DBP, PP, and MAP by sex, birth cohort, and survival status, and further to explore whether and to what extent heart disease and use of antihypertensive drugs can modify the age-related trajectories of late-life BP.

2. Materials and methods

2.1. Study participants

Study participants were derived from the population-based Swedish National study on Aging and Care in Kungsholmen (SNAC-K), as fully described elsewhere [20,21]. Briefly, SNAC-K is an ongoing multidisciplinary study of aging and health in older people who were living either at home or in institutions in the Kungsholmen district, an area of central Stockholm, Sweden. At baseline (March 2001–June 2004), an age-stratified random sample of 3363 (73.3% of all eligible) persons were examined for SNAC-K. The sample consists of 11 age-specific cohorts, with a 6-year interval for young-old cohorts (60–72 years), and a 3-year interval for old-old cohorts (≥78 years). Follow-up examinations took place every 6 years for the young-old cohorts, and every 3 years for the old-old cohorts. In this study, we used data that were collected at baseline, 3-, 6-, 9-, and 12-years' follow-up examinations until June 21, 2016. Of the 3363 participants, 48 were excluded due to missing information on BP at baseline, leaving 3315 persons for the current study. The flowchart of the study population was shown in eFigure 1 (see online-only figures).

The SNAC-K study was approved by the Regional Ethical Review Board in Stockholm, Sweden. Written informed consent was obtained from all participants or from a proxy if the participant was cognitively impaired.

2.2. Data collection and assessments

At baseline and follow-up examinations, trained nurses and physicians collected data on demographics (e.g., age, sex, education, and living place), lifestyle factors (e.g., smoking, alcohol consumption, and leisure activity), medical history (e.g., diabetes and heart disease), and use of medications through face-to-face interviews, clinical examinations, and laboratory tests, as fully described elsewhere [21,22]. Information on health history for all participants was also obtained from the computerized National Patient Register. Information on vital status until September 30, 2016 for all participants was collected from the Swedish Cause of Death Register and through follow-up telephone calls after the last interview.

In a quiet room with constant temperature, arterial BP was measured twice on the left arm in a sitting position with a 5-min interval, using a calibrated digital sphygmomanometer, and the mean value of the 2 measurements was used in the analysis. We calculated PP as SBP-DBP, and MAP as $(1/3 \times \mathrm{SBP}) + (2/3 \times \mathrm{DBP})$ [8]. Antihypertensive drugs include antihypertensives, diuretics, beta blocking agents, calcium channel blockers, and agents acting on the renin-angiotensin system. Use of antihypertensive drugs was dichotomized into no use and use of the medications at any of the study visits during the observation period.

Heart disease included coronary heart disease (CHD), atrial fibrillation, and heart failure, which were ascertained through clinical examination at baseline and follow-ups, and the patient register from 1969 until December 30, 2011. Smoking was categorized as current vs. noncurrent (never or former) smoking. Alcohol consumption was assessed on the basis of frequency and amount of alcohol intake on a typical drinking day and was classified into heavy vs. no or light-to-moderate drinking [23]. Leisure-time physical activity was dichotomized into physically inactive and active. We defined diabetes as having self-reported history of diabetes, records of diabetes in the patient register, use of antidiabetic agents, or HbA1c ≥6.5%; high cholesterol as non-fasting total serum cholesterol ≥6.22 mmol/l or use of cholesterol-lowing agents; and obesity as a body-mass index (BMI) ≥30 kg/m^2. Living place was categorized into institution vs. non-institution. We assessed global cognitive function at baseline and follow-ups by using the Mini-Mental State Examination (MMSE) [23].

2.3. Statistical analyses

Baseline characteristics of study participants by age groups (60–72 vs. ≥78 years) were compared using Chi-square test for proportions and independent Student's *t*-test for means. We used the mixed-effects models with age as the time scale to explore the longitudinal changes of BP components with age, in which both random intercept and random slope were considered. To test possible nonlinear relationship between BP changes and age, we considered quadratic effect of age in the models. Because BP trajectories with age may differ by survival time, we divided all participants into 2 groups according to the length of survival (<2 vs. ≥2 years) after the last visit, as previously reported [24,25]. We plotted graphs by sex and birth cohort to show the sex- and birth cohort-specific trajectories of the predicted values of various BP components derived from the mixed-effects models.

We further investigated the age-related BP trajectories by heart disease and use of antihypertensive medications, by entering the interaction term of predictor × age into the mixed-effects models. We presented the results by plotting graphs of the predicted mean BP (95% confidence interval, CI). In the mixed-effects models, we adjusted for sex, education, cardiovascular risk factors (current smoking, physical inactivity, heavy alcohol drinking, diabetes, and high total cholesterol) at baseline, and if applicable, for birth cohort and time-varying variables of heart disease and use of antihypertensive drugs. Additional analyses were performed to investigate whether living in institutions would modify the age-related BP trajectories in older individuals. Stata 14.0 for Windows (StataCorp., College Station, TX, USA) was used for all analyses.

3. Results

3.1. Baseline characteristics of study participants

At baseline, the mean age of the 3315 participants was 74.2 (SD, 11.1) years, 64.6% were women, and 8.1% were living in institution. The average BP was 142.6 (SD, 20.5) mmHg for SBP, 80.6 (11.2) mmHg for DBP, 62.0 (16.2) mmHg for PP, and 101.3 (12.8) mmHg for MAP. >40% of the participants reported to have used antihypertensive medications.

Compared with young-old groups (60–72 years, $n = 1771$), those in the old-old groups (≥78 years, $n = 1544$) had higher SBP and PP, but lower levels of education, DBP, MAP, BMI, and total cholesterol ($P < 0.01$). People in the old-old groups were less likely to smoke, drink alcohol heavily, and participate in physical activity, but more likely to live in institution, use antihypertensive drugs, and have heart disease than those in the young-old groups

Table 1
Baseline characteristics of the study participants by age groups.

Characteristics	Total sample	Age at Baseline, y		*P* value
		60–72	≥78	
No. of subjects	3315	1771	1544	
Female, No. (%)	2141 (64.6)	1016 (57.4)	1125 (72.9)	<0.01
Institutionalization, No. (%)	269 (8.1)	13 (0.7)	256 (16.6)	<0.01
Education[a], No. (%)				
Elementary or middle school	574 (17.5)	152 (8.6)	422 (27.8)	
High school	1632 (49.6)	811 (45.9)	821 (54.0)	
University or above	1083 (32.9)	806 (45.6)	277 (18.2)	<0.01
Current smoking[a], No. (%)	435 (13.5)	316 (18.0)	119 (8.1)	<0.01
Heavy alcohol consumption[a], No. (%)	504 (15.6)	358 (20.4)	146 (9.9)	<0.01
Physical inactivity, No. (%)	1124 (33.9)	368 (20.8)	756 (49.0)	<0.01
Obesity[a], No. (%)	385 (12.8)	265 (15.2)	120 (9.5)	<0.01
High total cholesterol[a], No. (%)	1519 (49.4)	909 (53.0)	610 (44.9)	<0.01
Diabetes, No. (%)	315 (9.5)	158 (8.9)	157 (10.2)	0.22
SBP (mm Hg), mean (SD)	142.6 (20.5)	141.6 (18.9)	143.8 (22.1)	<0.01
DBP (mm Hg), mean (SD)	80.6 (11.2)	83.3 (10.3)	77.5 (11.4)	<0.01
PP (mm Hg), mean (SD)	62.0 (16.2)	58.3 (14.2)	66.3 (17.2)	<0.01
MAP (mm Hg), mean (SD)	101.3 (12.8)	102.7 (12.0)	99.6 (13.5)	<0.01
Use of antihypertensive drugs[a], No. (%)	1373 (41.5)	516 (29.1)	857 (55.7)	<0.01
Coronary heart disease, No. (%)	516 (15.6)	151 (8.5)	365 (23.6)	<0.01
Atrial fibrillation, No. (%)	289 (8.7)	57 (3.2)	232 (15.0)	<0.01
Heart failure, No. (%)	306 (9.2)	37 (2.1)	269 (17.4)	<0.01

BMI indicates body mass index; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; and SBP, systolic blood pressure.
[a] Information was missing in 26 persons for education, 87 for smoking, 79 for alcohol intake, 303 for obesity, 241 for high total cholesterol, and 5 for use of antihypertensive drugs.

($P < 0.01$). The 2 groups did not differ significantly in the distribution of diabetes ($P > 0.05$) (Table 1).

3.2. Blood pressure trajectories with age by sex and survival time

Overall, women had significantly higher mean MAP than men, with the age- and education-adjusted β-coefficient (95% CI) of SBP, DBP, PP, and MAP being 1.11 (−0.06, 2.28; $P = 0.064$), 0.57 (−0.05, 1.18; $P = 0.071$), 0.50 (−0.37, 1.37; $P = 0.259$), 0.80 (0.07, 1.53; $P = 0.032$), respectively. The SBP and PP trajectories with age showed a pattern of increase first and then fall, with a transition age ranging between 80 and 90 years, yet DBP and MAP showed a consistent decline with age.

Of the 3315 participants, 1653 (49.9%) died during the follow-ups, and 745 (22.5%) died within 2 years after the last visit. The BP trajectories with age varied by sex and survival time (Fig. 1). Both men and women who died <2 (vs. ≥2) years after the last visit had higher levels of all 4 BP components until ~80 years of age, followed by steeper declines, especially for SBP and PP. In addition, SBP and PP peaked ~10 years earlier in people who died within 2 years than those who survived ≥2 years.

3.3. Blood pressure trajectories with age by birth cohorts

All 4 BP components in all birth cohorts, except PP in the cohort born in 1939, showed a decline with age, and the decline was steeper for people in the earlier than in later birth cohorts (Fig. 2). Furthermore, at a given age, the mean BP was lower in the more recent birth cohorts. For example, at the age of ~65 years, people born 6 years later (birth cohort 1939) had lower average BP than those born 6 years earlier (birth cohort 1933), with the difference being 6.6 mm Hg for SBP, 2.8 mm Hg for DBP, 3.8 mm Hg for PP, and 4.1 mm Hg for MAP.

Similar BP trajectory patterns were observed either when people who died within 2 years after the last visit were excluded or when we only included people who had BP records at all follow-up visits (data not shown). Further, people who died within 2 (vs. ≥2) years showed faster declines in all BP components across all birth cohorts (data not shown).

Blood pressure trajectories with age by antihypertensive therapy and heart disease.

The age-related BP trajectories varied by use of antihypertensive drugs. Among people who did not use antihypertensive drugs during the study period, SBP, PP, and MAP all increased with age until 80–90 years, and then declined (Fig. 3-A). Specifically, SBP increased from ~130 mmHg at age 60, reached a peak of ~140 mm Hg at 80–90 years of age, and declined thereafter, whereas DBP decreased slowly from ~80 mm Hg at the age 60 to ~70 mm Hg at the age of over 100 years. Among people who used antihypertensive drugs, SBP, DBP, and MAP declined consistently with increasing age, starting from higher levels at the age 60, compared to those who did not use the drugs. After ~80–90 years of age, BP trajectories were overlapped between persons who did and did not use antihypertensive drugs. The patterns of age-related BP trajectory by use of antihypertensive medications remained similar after additionally controlling for follow-up survival status and MMSE scores at baseline and follow-ups.

The BP trajectory patterns with age also varied by the presence of heart disease (CHD, atrial fibrillation, and heart failure) (Fig. 3-B). Overall, compared to people without heart disease, those with 1 or ≥2 of the 3 types of heart disease had lower levels in all 4 BP components and showed faster declines in SBP and PP after 80 years of age, but the declines in DBP and MAP were parallel across all age groups.

When examining the 3 types of heart disease separately, similar patterns of BP trajectory with age were observed by CHD, atrial fibrillation, and heart failure (see eFigure 2).

3.4. Blood pressure trajectories with age by living place

Finally, we examined the age-related BP trajectories by living place (institution vs. non-institution). There were significant variations by living place in the age-related trajectories of DBP (P for interaction = 0.007) and MAP (P for interaction = 0.039) trajectories, but not SBP (P for interaction = 0.277) and PP (P for

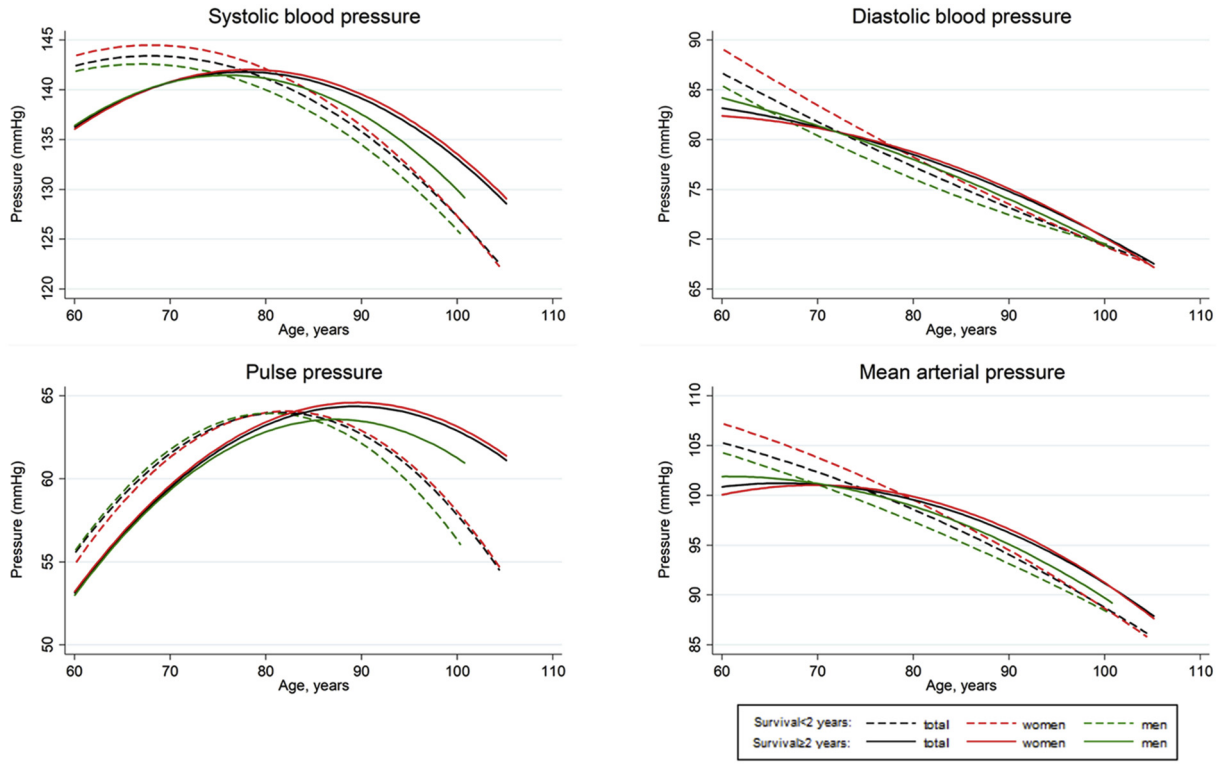

Fig. 1. Longitudinal blood pressure trajectories with age by sex and survival status.
Note. Dash lines are for participants who died within 2 years after the last visit; solid lines are for participants who survived ≥2 years after the last visit.

interaction = 0.653). Compared with non-institutionalized older individuals, those living in the institutions showed a lower level of all 4 BP components, a parallel age-related trajectory pattern in SBP and PP, and a trajectory pattern of a slower decline with age in DBP and MAP (see eFigure 3).

4. Discussion

The main findings from this cohort study of older adults are: (1) SBP and PP increase with age until ~80 years and then decline, while DBP and MAP constantly decrease with age after 60 years. The average level of BP is slightly higher in women than in men. (2) All BP components decline with age across birth cohorts, and the declines are steeper in earlier than in more recent birth cohorts. BP levels at a given age are lower in the more recent birth cohorts. (3) Shorter survival after the last visit (<2 years) is associated with higher levels of all BP components until ~80 years of age; thereafter shorter survival is related to steeper declines and lower BP, especially in SBP and PP. (4) Heart disease is accompanied with lower levels of all BP components, and faster declines in SBP and PP after 78 years of age. (5) Among users of antihypertensive medications, BP increases with age until 80–90 years of age and then declines, whereas among non-users, BP declines consistently with age; after 85 years of age, the trajectories of the BP components are overlapped between users and non-users of antihypertensive medications.

In line with our findings, the population-based studies of middle-aged and older adults have previously shown that SBP increases with age and reaches peak until ~80 years of age, whereas DBP decreases with age after 60 years of age [2,13,26–28], We further demonstrated that MAP plateaued along with a concomitant increase in PP between 60 and 70 years of age, and a decrease in all BP components after 80 years of age.

Several cohort studies have shown that men have slightly higher average BP than women throughout the lifespan, especially in early life and middle age [2,26,27]. However, the sex-specific patterns of late-life BP trajectories with age remain debatable. For instance, the 8 UK cohorts showed that women had higher SBP from 64 to 72 years, whereas men had higher SBP thereafter [13]. The US Third National Health and Nutrition Examination Survey showed that the average SBP in women was as high as or higher than men from the seventh decade of life [29]. We found that women and men had similar patterns of BP trajectories with age. The mean MAP was slightly higher in women than in men, whereas men appeared to have faster declines in DBP and MAP than women after the seventh decades of life.

Population-based cohort studies of older adults frequently reported that the association of high BP with increased mortality was attenuated with advancing age, depending on cognitive and physical functioning [6,19,30,31]. We found that the patterns of age-related BP trajectories differed considerably by survival status, such that the BP trajectories in older adults with a shorter survival (<2 vs. ≥2 years) were characterized by high BP before 80 years of age as well as a faster decline and low BP thereafter. This is in good agreement with the 2 medical record-based studies of primary care settings in the UK, which showed that there was a steeper decrease in SBP in the last 2 years of life, especially among those aged >80 years [24,25]. Our study additionally revealed that among the oldest old, the trajectory of DBP and MAP with age appeared to be similar for people with short-term vs. long-term (<2 vs. ≥2 years) survival. Findings from these studies are in line with the view that the associations of low SBP to the subsequently increased risk of mortality in oldest old people may reflect reverse causality if BP is measured near the end of their life.

Previous studies have reported the birth cohort effects on BP trajectories with age [13–17]. Consistent with these reports, we

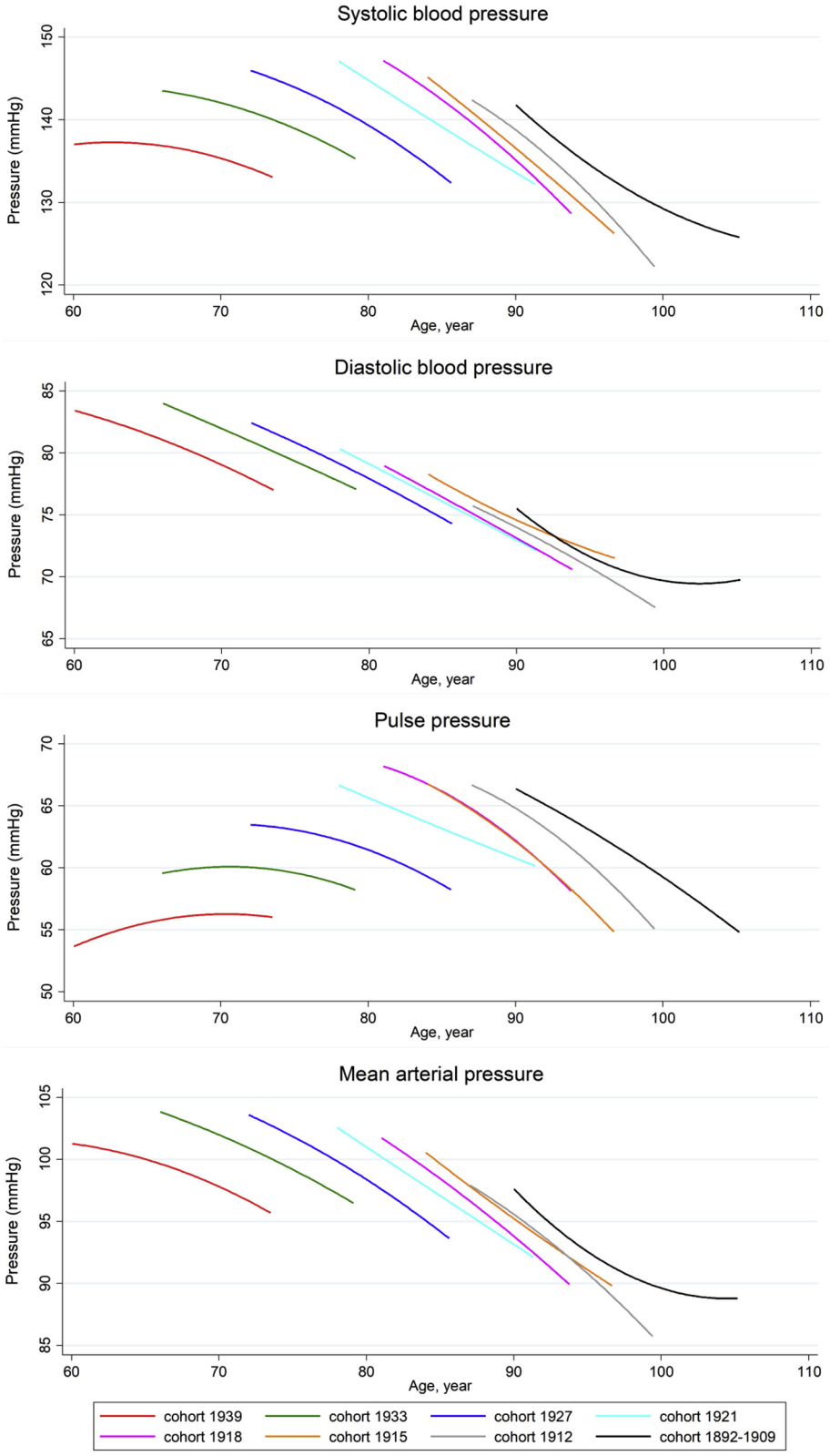

Fig. 2. Longitudinal blood pressure trajectories with age by birth cohorts.

confirmed that the mean BP was lower in the more recent birth cohorts. In addition, we extended previous findings by showing that SBP, DBP, and MAP decreased with age in all birth cohorts born from 1892 to 1939, and that the declines were steeper in more recent birth cohorts. It has been suggested that the downward shift of age-related BP towards more recent birth cohorts was attributable, at least partially, to the improvement of antihypertensive treatment and control of high BP over time [17,32]. This is in line

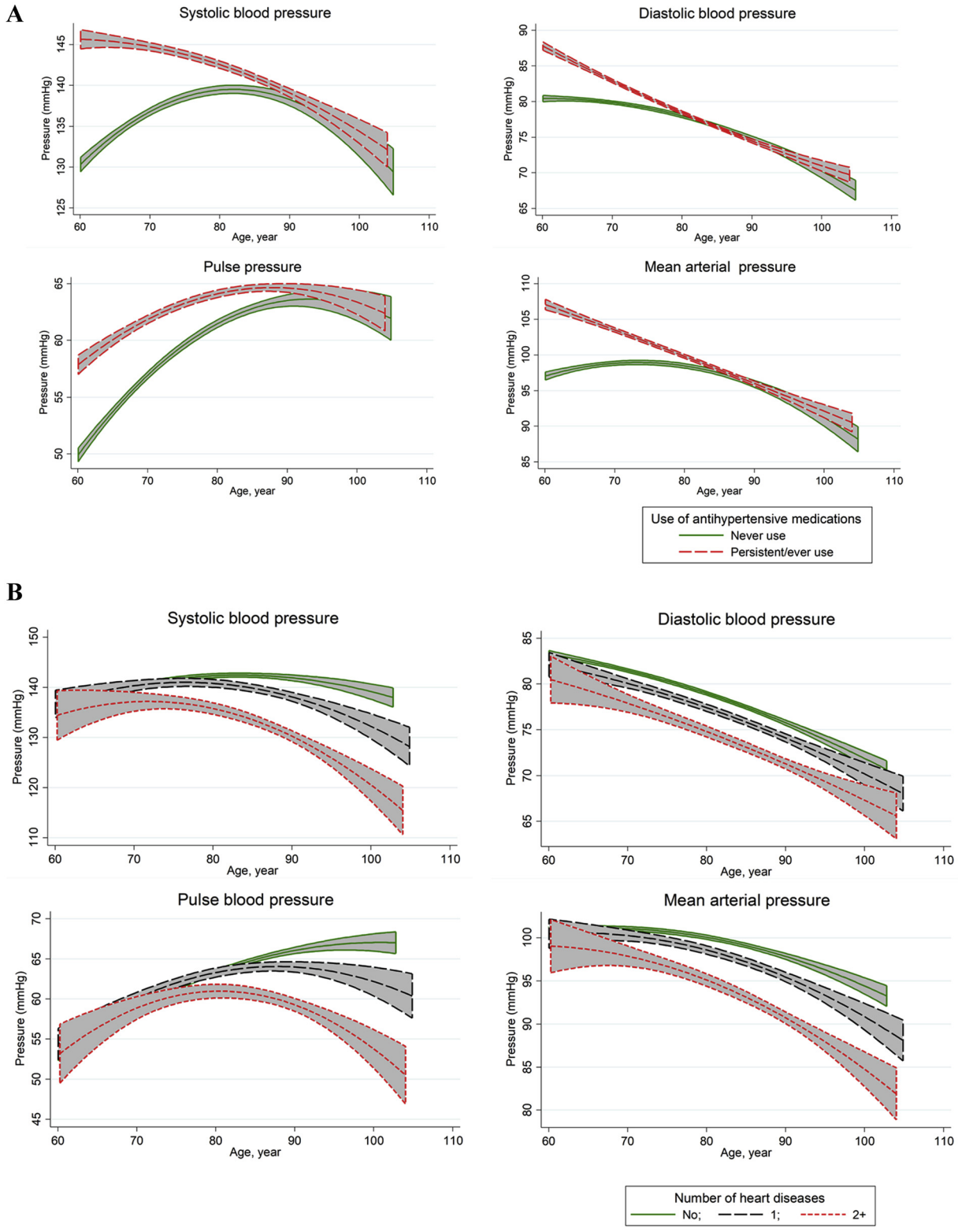

Fig. 3. Longitudinal blood pressure trajectories with age by use of antihypertensive drugs (a) and by number of heart diseases (b).
Note. Data were average levels of blood pressure (95% confidence interval) derived from the linear mixed-effects models that were adjusted for demographic factors, cardiovascular risk factors, birth cohorts, and chronic diseases.

with the fact that clinical guidelines on high BP management in older adults have evolved towards a more aggressive control of high BP in the past decades [33].

The potential impacts of antihypertensive therapy and cardiovascular disease on the age-related longitudinal BP trajectories in late life have rarely been investigated. This is important because despite a large body of evidence for cardiovascular benefits of antihypertensive treatment in middle-aged and young-old people with high BP, whether aggressive BP control benefits the oldest old or frail older persons remains debatable [30,34,35]. Our data

showed that BP trajectories with age were quite similar between older adults who did and did not use antihypertensive drugs after ~80 years, even when additionally controlling for cognitive function and follow-up survival status. This suggests that use of antihypertensive therapy may not affect the BP trajectory patterns with age among the oldest old. However, the influence of antihypertensive therapy on BP trajectories with age in older adults deserves further investigation. In addition, the longitudinal patterns of SBP and PP trajectories with age were almost identical until ~80 years of age between people with and without heart disease, but after 80 years of age, people with heart disease showed faster declines in SBP and PP with increasing age. Further analysis revealed that the overall patterns of age-related BP trajectories were generally similar for people with CHD, atrial fibrillation, and heart failure. This indicates that BP may decline with advancing age as a consequence of impaired ventricular function, especially in the oldest old. However, further research is needed to fully understand the contributing factors and the pathophysiological mechanisms underlying the complex age-related BP trajectories associated with cardiovascular conditions, especially among the oldest old.

Previous studies have not paid much attention to different BP trajectories with age by living place. We found that older adults living in institutions had lower BP levels and a slower age-related BP decline than non-institutionalized older individuals. This suggests that older adults who were living in institutions might have a higher compliance with antihypertensive treatment than non-institutionalized older people. This hypothesis is also in line with the observation that after around 85 years of age, the difference in the age-related BP trajectories appeared to be limited or absent.

This study is based on a large population sample of older adults with a broad age range (60–105 years). Comprehensive data on health conditions, use of drugs, and survival status were available from baseline throughout the follow-up periods. We were also able to integrate data from the national patient register, in which diseases were diagnosed via careful clinical assessments. In addition, the mixed-effects models can properly deal with missing information often occurring during follow-ups, and also take into account the inter- and intra-individual variations, thus, maximizing the use of number of observations in a cohort study with repeated assessments, and accounting for the within individual variability. Our study has limitations. First, residual confounding might still play a part owing to lack or imperfect assessments of covariates (e.g., diet). Second, the SNAC-K participants had socioeconomic advantages over the national average, which should be kept in mind when generalizing our findings to other populations. Third, we did not consider the dosage, length, and use of multiple antihypertensive medications when examining their impact on BP trajectories, although use of antihypertensive drugs was considered as a time-varying variable in our analysis. Finally, selective survival may potentially affect the results because attrition rate due to death is relatively high in a cohort of older adults and people who had poor control of high BP might have a greater risk of cardiovascular mortality.

5. Conclusions

Our findings suggest that the age-related increases in BP among young-old people are potentially modifiable, and that BP decline in the oldest old might be a clinical marker of poor survival. In addition, variations of BP trajectories with age by birth cohorts support the view that management of high BP and general health conditions in older adults may have been improved in the recent decades. Future research is warranted to further clarify the age-related BP trajectory patterns in association with subsequent functional consequences. This will help achieve the goal of maximizing the potential benefits of BP management in older people.

Author contributions

RW and CQ conceived and designed the study. RW analysed the data and drafted the manuscript. All authors contributed to the data interpretation and critical revision of the manuscript. All authors read the final version and approved the submission of the manuscript.

Declaration of Competing Interest

The authors reported no conflict of interests.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2019.08.011.

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