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ORIGINAL RESEARCH

Biological Aging Exhibits an Inverted J-Shaped Relationship with Stress Urinary Incontinence in Women

Lingyun Wei[1,*], Xinfang Cao[1,*], Shuyu Wang[1], Xiameng Kang[1], Yingjie Ma[1], Jie Shi[2], Jiahui Guo[2], Xiaochun Liu[1]

[1]Third Hospital of Shanxi Medical University, Shanxi Bethune Hospital, Shanxi Academy of Medical Sciences, Tongji Shanxi Hospital, Taiyuan, People's Republic of China; [2]Shanxi Medical University, Taiyuan, People's Republic of China

*These authors contributed equally to this work

Correspondence: Xiaochun Liu, Third Hospital of Shanxi Medical University, Shanxi Bethune Hospital, Shanxi Academy of Medical Sciences, Tongji Shanxi Hospital, Taiyuan, People's Republic of China, Email liuxiaochun@sxbqeh.com.cn

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Background: Biological aging is a systemic driver of chronic disease, yet its relationship with female urinary incontinence (UI) remains unexamined. We leveraged three conceptually distinct metrics-PhenoAge (PA), Klemera-Doubal Method (KDM), and Homeostatic Dysregulation (HD)-to quantify biological age and assessed their associations with stress UI (SUI), urgency UI (UUI), and mixed UI (MUI) in a nationally representative cohort.

Methods: This cross-sectional study included 8,561 women ≥20 years from NHANES 2005–2010 and 2015–2018. After outlier exclusion and covariate selection via LASSO regression, multivariable logistic regression and restricted cubic splines (RCS) were used to estimate ORs and dose-response shapes. Subgroup analyses, mediation analyses, and sensitivity analyses were performed.

Results: PA and KDM were positively associated with UI. The third quartile of PA and KDM conferred the highest risk for SUI (PA: Q3 OR = 1.58, 95% CI 1.33–1.88; KDM: Q3 OR = 1.39, 95% CI 1.20–1.62). Higher quartiles were also linked to greater UI severity. Fully adjusted RCS models revealed inverted J-shaped relationships for SUI (PA inflection: 43.6; KDM: 34.7) and inverted U-shaped relationships for UUI and MUI with KDM (UUI inflections: 40.7 and MUI inflections: 40.5). Associations were robust across most subgroups but attenuated with increasing reproductive risk factors. No significant mediation by sex steroids or α-Klotho was observed.

Conclusion: Biological aging is significantly positively associated with UI, and its relationship with SUI follows an inverted J-shaped curve. Interventions that slow biological aging may offer a novel strategy for UI prevention; however, longitudinal studies are required to establish causality.

Plain Language Summary: We examined whether faster biological aging increases urinary incontinence (UI) in women. Using three aging-metrics (PhenoAge, KDM, HD) in 8,561 NHANES women we found higher biological age raised the odds of stress, urgency and mixed UI, with a clear inverted-J shape for stress UI. Associations persisted across subgroups but weakened with more reproductive risk factors. Slowing biological aging may help prevent UI, yet longitudinal work is needed.

Keywords: aging, prevalence, urinary incontinence, stress urinary incontinence, urgency urinary incontinence

Introduction

Aging is a progressive process characterized by the accumulation of cellular and molecular damage, resulting in functional decline across various physiological systems.[1] Substantial efforts have been directed toward developing robust indicators of biological aging. Exemplary methodologies incorporate epigenetic clock, telomere length, multiple omics prediction tools, and composite biomarker systems.[2] In recent research, the composite biomarker paradigms, exemplified by PhenoAge (PA), the Klemera-Doubal Methodology (KDM), and Homeostatic Dysregulation (HD) indices, have been

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pivotal in exploring the complex nexus between biological aging and age-related disease.[3–5] Including the roles of KDM and PA in the progression of endometrial cancer, and the roles of KDM, PA, and HD in the progression of neurological system diseases.[3,4] PA was developed by modeling survival probabilities using blood chemistry data from National Health and Nutrition Examination Survey (NHANES) III participants. This parameter was derived from a proportional hazards model based on the Gompertz distribution, which integrated nine clinical biomarkers with chronological age.[6] KDM was calculated by Klemera and Doubal using an algorithm derived from regression analysis of biomarkers.[7] HD was a novel analytical method grounded in Mahalanobis distance theory, designed to quantify age-related longitudinal trajectories of biomarker panels. By capturing the progressive increase in Mahalanobis distance with chronological age, HD provided a robust quantitative metric for biological aging.[8]

Urinary incontinence (UI) is typically classified into stress urinary incontinence (SUI), urgency urinary incontinence (UUI), and mixed urinary incontinence (MUI).[9] Due to anatomical differences, the pathogenesis of female UI diverges from that of male UI. Female UI is primarily associated with pelvic floor muscle dysfunction and is influenced by a multifactorial interplay of chronological age, ethnicity, menopausal status, and comorbid chronic conditions.[9,10] Most epidemiological studies have reported a positive association between chronological age and UI prevalence.[9,11] However, two nationwide studies covering the United States and China both show that the relationship between chronological age and SUI is not strictly linear, with prevalence peaking at midlife and then leveling off or decreasing in older women.[12,13] Biological aging may promote urinary incontinence through several interconnected mechanisms. First, cellular senescence-driven extracellular matrix remodelling reduces collagen content and increases elastin fragmentation within the vaginal wall and endopelvic fascia, thereby diminishing urethral support.[14,15] As reported by Zhuang et al (2021), aging also disrupts the structural integrity of the vaginal wall by altering the balance between matrix metalloproteinases and their tissue inhibitors, leading to extracellular matrix degradation and compromised pelvic floor stability.[16] This imbalance further exacerbates the loss of supportive tissue strength, contributing to the development of stress urinary incontinence. Although these studies have contributed to our understanding of aging and urinary incontinence, limited preliminary evidence exists on aging biomarkers and pelvic-floor dysfunction. This study leverages the NHANES database to investigate the association between biological aging and UI for the first time.

Material and Methods

Study Population

NHANES, a nationally representative cross-sectional survey, is meticulously managed by the National Center for Health Statistics (NCHS) within the United States, providing comprehensive health and nutritional data across diverse demographic groups. This survey was conducted with the approval of the NCHS Ethics Review Committee, which is included in the database. Informed consent was obtained from each participant. Given that our study utilizes pre-existing data from NHANES that has already been ethically reviewed and is publicly accessible, the requirement for clinical trial registration does not apply to our research.

This study retrieved data from the NHANES database from 2005 to 2010 and from 2015 to 2018 (50,259 participants); the 2011–2014 cycle was excluded because the serum chemistry biomarkers required to calculate PhenoAge and HD were not fully released during those survey years. We selected women aged 20 years and older for analysis. Initially, outliers in PA, KDM, and HD (defined as values beyond the mean ± 3 standard deviations) and missing values for these three variables were excluded. Subsequently, we excluded cases with missing UI data. Finally, we excluded cases with missing values for the initially selected covariates. Ultimately, 8,561 participants were included in the study (Figure 1).

Assessment of UI

This study analyzed SUI, UUI, MUI and the severity of SUI and UUI. SUI was defined as participants who "leaked urine or lost control of a small amount of urine due to activities such as coughing, lifting, or exercising in the past 12 months". UUI was defined as participants who "leaked urine or lost control of urine due to a sudden urge or pressure to urinate in the past 12 months and could not make it to the toilet in time". Participants who met the criteria for both SUI and UUI

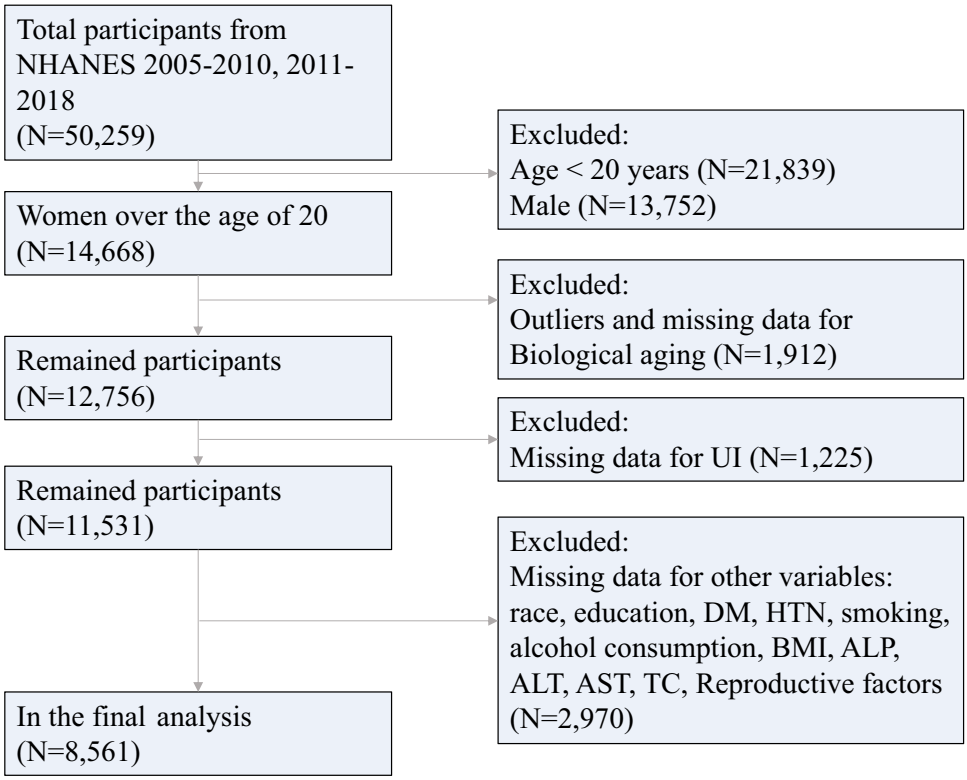

Figure 1 Flow chart for participants.
Abbreviations: NHANES, National Health and Nutrition Examination Survey; UI, urinary incontinence; DM, diabetes mellitus; HTN, hypertension; BMI, body mass index; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TC, total cholesterol.

were classified as having MUI. The severity of SUI and UUI was determined by the frequency of occurrence, categorized as "Less than once a month", "A few times a month", "A few times a week", and "Every day and/or night".

Assessment of Biological Aging

The R package BioAge was employed to calculate three distinct biological aging metrics, namely PA, KDM, and HD.[17] These metrics were analyzed in parallel, as they are constructed from different parameters, thereby ensuring that our results reflected the relationship between biological aging and UI rather than being specific to one aging metric.

Assessment of Covariates

The initial covariates included common variables that may potentially influence UI, including race, education, diabetes mellitus (DM), hypertension (HTN), smoking, alcohol consumption, body mass index (BMI), alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), total cholesterol (TC), age at first menstrual period, menopause status, number of pregnancies, history of hysterectomy, number of vaginal deliveries, and number of live births. The definitions of these variables could be found in Table S1.

Statistical Analysis

In the baseline characteristics, continuous variables were presented as mean ± standard deviation, whereas categorical variables were described by the number of participants and their corresponding frequency (%). Normality tests were first conducted for the three biological aging metrics. We conducted a LASSO regression analysis that included three biological aging metrics and the initially determined covariates. The LASSO regression selected the optimal penalty parameter λ through 10-fold cross-validation and identified the variables with non-zero coefficients. Then extracted the selected variables for further analysis. PA, KDM, and HD, along with their quartiles, were used as independent variables, while SUI, UUI, and MUI were used as dependent variables in three logistic regression models to investigate the

association between biological aging and UI, Model 1 was unadjusted for covariates; Model 2 was adjusted for race and education; Model 3 was adjusted for the remaining variables identified by LASSO regression. As the restricted cubic spline and LASSO algorithms do not incorporate survey weights, the primary analyses were first performed without weighting to ensure methodological consistency across all models. But we also conducted weighted multivariable logistic regression using the NHANES survey weights (WTMEC2YR, SDMVPSU, SDMVSTRA). Additionally, we analyzed the relationship between biological aging and the severity of SUI and UUI. Bonferroni correction was applied to all models to reduce the probability of committing Type I errors. Restricted cubic spline (RCS) analyses were conducted to assess the non-linear relationships between biological aging and SUI, UUI, and MUI, adjusting for the remaining variables identified by LASSO regression. Additionally, subgroup analyses were performed to evaluate the impact of different population characteristics on the study outcomes. Mediation analyses were also conducted, considering sex hormone levels and α-Klotho as potential mediators, to explore the underlying mechanisms through which biological aging affects female UI. Due to the availability of sex-steroid and α-Klotho measurements only in the 2015–2016 NHANES cycle, the mediation analyses were restricted to the 1,045 women with complete biomarker and covariate data from that period.

Finally, sensitivity analyses were performed to examine the robustness of our results. Given that excluding too many missing data points could reduce the sample size and potentially introduce selection bias, we reanalyzed the data after imputing missing values for the biological aging indicators using multiple imputation.

The above statistical analysis mainly used R (version 4.4.2).

Results

Baseline Characteristics

Table S2 summarizes the baseline characteristics of participants stratified by UI subtype: 3,991 with SUI, 2,794 with UUI, and 1,724 with MUI. Across all subtypes, patients exhibited accelerated biological aging and were chronologically older than normal controls. Moreover, the risk of UI rose in direct proportion to the number of pregnancies, vaginal deliveries, and live births.

Normality Test

Normality diagnostics, as depicted by the Q–Q and cubic-moment plots, revealed that PA closely approximates a Gaussian distribution, whereas HD and KDM manifest marked skewness (Figure S1). Therefore, in the subsequent analysis, we mainly focused on the quartiles of PA, HD, and KDM results.

LASSO Regression

The results of LASSO regression showed that 15 variables, including three biological aging indicators, were included in this study (Figure S2). This included PA, HD, KDM, race, education, HTN, smoking, alcohol consumption, BMI, ALP, TC, age at first menstrual period, menopause status, number of vaginal deliveries, and number of live births. We used 12 variables other than biological aging indicators as covariates for further analysis of the fully adjusted model.

Associations of Biological Aging Indicators with UI

Figure 2 shows the results of logistic regression between continuous variables and quartiles of three biological aging indicators and three UI. We observed no statistically significant association between HD and any of the three UI subtypes, while PA and KDM showed a positive correlation with UI. The results of the weighted multivariable logistic regression were materially similar to those of the unweighted analysis (Table S3). Upon full adjustment for potential confounders, the third quartile (Q3) exhibited the highest odds ratios (OR) for the associations of both PA and KDM with SUI (PA: Q3 OR = 1.58, 95% CI 1.33–1.88; KDM: Q3 OR = 1.39, 95% CI 1.20–1.62). The similar, statistically significant patterns emerged for the KDM–UUI and KDM–MUI relationships, whereas no such trend was detected for PA to either UUI or MUI. Furthermore, multivariable logistic regression analyses revealed a significant, positive association between both PA and KDM and the increasing severity of SUI and UUI (all adjusted $P < 0.05$) (Figure S3).

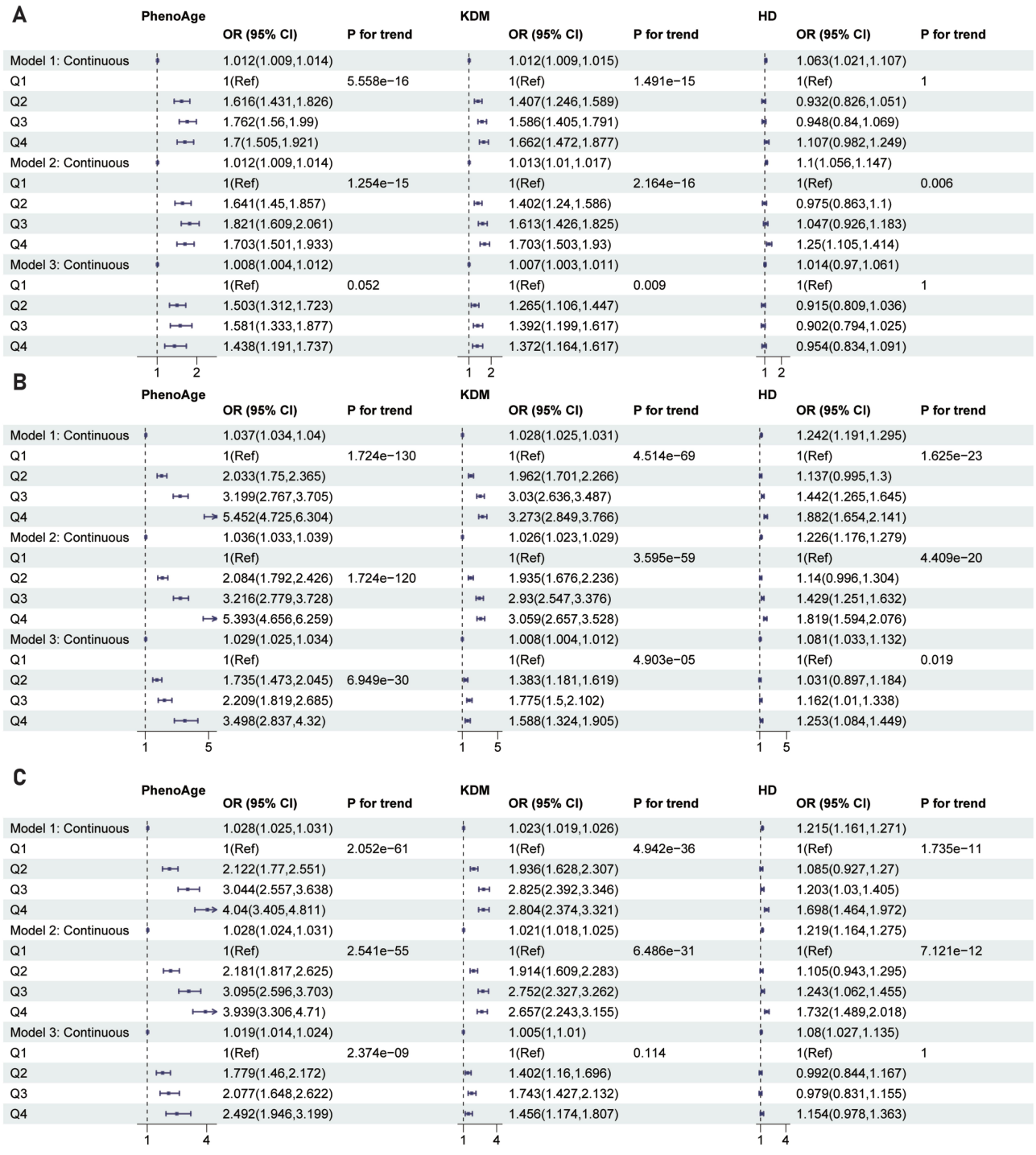

Figure 2 The results of logistic regression between three biological aging indicators and three UI: (**A**) The results of SUI, (**B**) The results of UUI, (**C**) The results of MUI. Model 1 was unadjusted for covariates; Model 2 was adjusted for race and education; Model 3 was adjusted for race, education, HTN, smoking, alcohol consumption, BMI, ALP, TC, age at first menstrual period, menopause status, number of vaginal deliveries, and number of live births.
Abbreviations: KDM, Klemera-Doubal Methodology; HD, Homeostatic Dysregulation.

RCS analyses corroborated the logistic-regression findings, revealing non-linear dose–response relationships of PA and KDM with SUI, and of KDM with both UUI and MUI (Figure 3). RCS results revealed the inverted J-shaped association: the OR of SUI peaked at approximately 43.6 of PA (Figure 3A), whereas the steepest increase in risk for SUI occurred at a KDM level of 34.7 (Figure 3B). However, no significant non-linear relationship was observed for HD

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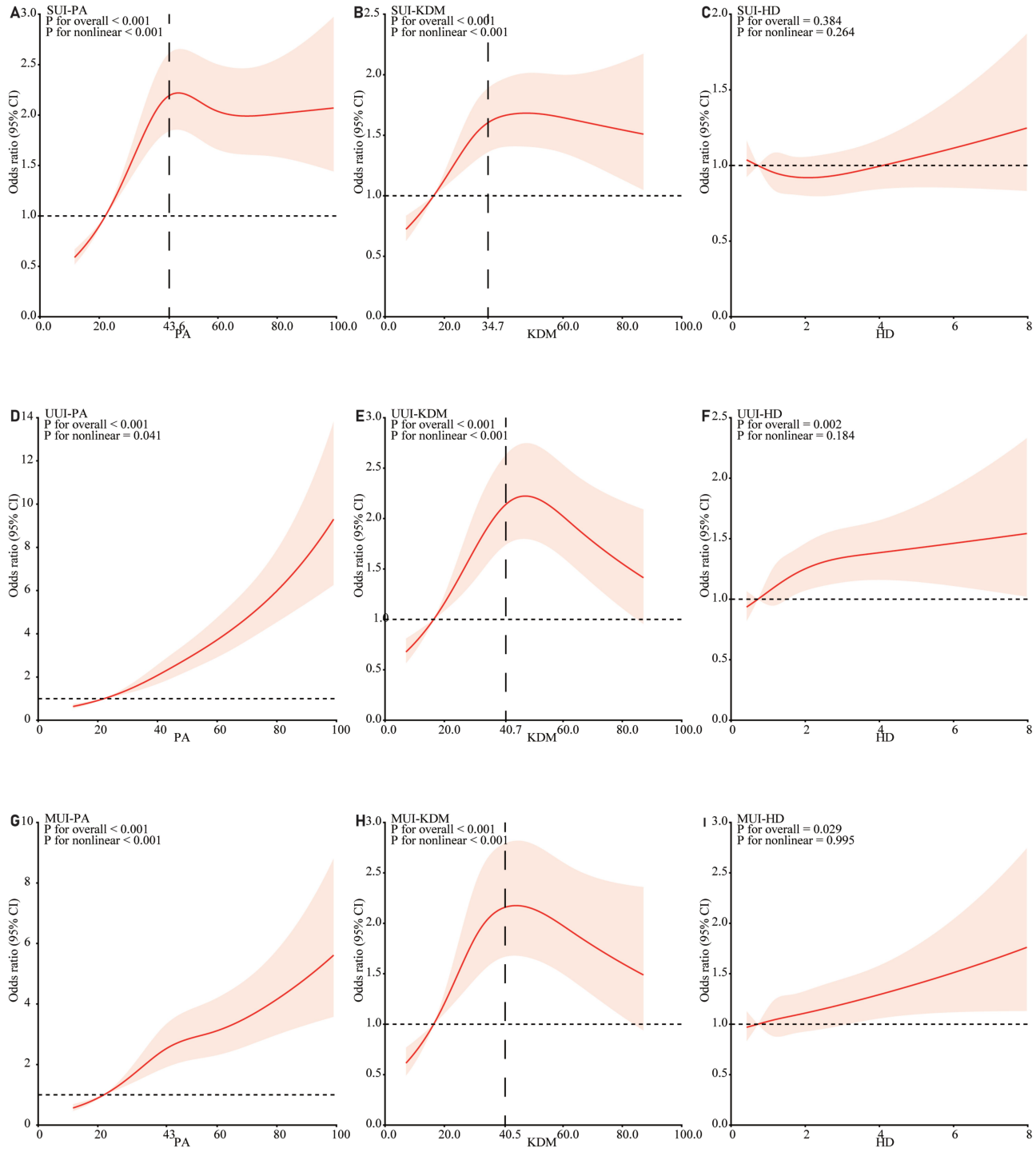

Figure 3 Non-linear relationship between three biological aging indicators and three UI: (**A**) Nonlinear relationship between SUI and PA, (**B**) Nonlinear relationship between SUI and KDM, (**C**) Nonlinear relationship between SUI and HD. (**D**) Nonlinear relationship between UUI and PA, (**E**) Nonlinear relationship between UUI and KDM, (**F**) Nonlinear relationship between UUI and HD, (**G**) Nonlinear relationship between MUI and PA, (**H**) Nonlinear relationship between MUI and KDM, (**I**) Nonlinear relationship between MUI and HD. Adjusted for race, education, HTN, smoking, alcohol consumption, BMI, ALP, TC, age at first menstrual period, menopause status, number of vaginal deliveries, and number of live births.

Abbreviations: KDM, Klemera-Doubal Methodology; HD, Homeostatic Dysregulation; SUI, stress urinary incontinence; UUI, urgency urinary incontinence; MUI, mixed urinary incontinence.

(Figure 3C). Among the non-linear relationships with UUI and MUI, only KDM showed a significant inverted U-shaped association with both conditions (Figure 3D–I).

Subgroup Analysis

Subgroup analyses across PA quartiles showed that, although not every stratum achieved statistical significance, the Q3 level consistently yielded the highest OR for SUI in most subgroups (Figure 4A). Conversely, for UUI and MUI, the OR exhibited a progressive increase across quartiles (Figure 4B and C). In the quartile-based subgroup analyses of KDM, Q3 likewise exhibited the highest OR in the majority of strata across all three UI subtypes (Figure S4). This indicated that the results of logistic regression remain consistent in most subgroups. Nevertheless, when the models were stratified along reproductive factors (menopause status, number of pregnancies, history of hysterectomy, number of vaginal deliveries, and number of live births), the results of associations grew conspicuously intricate—an observation that might intimate the primacy of reproductive factors over biological aging among the factors related to UI.

Mediation Analysis

Due to the large amount of missing data, we could not identify a statistically significant mediating role of estrogen, androgen, and α-Klotho between biological aging and UI (Table S4).

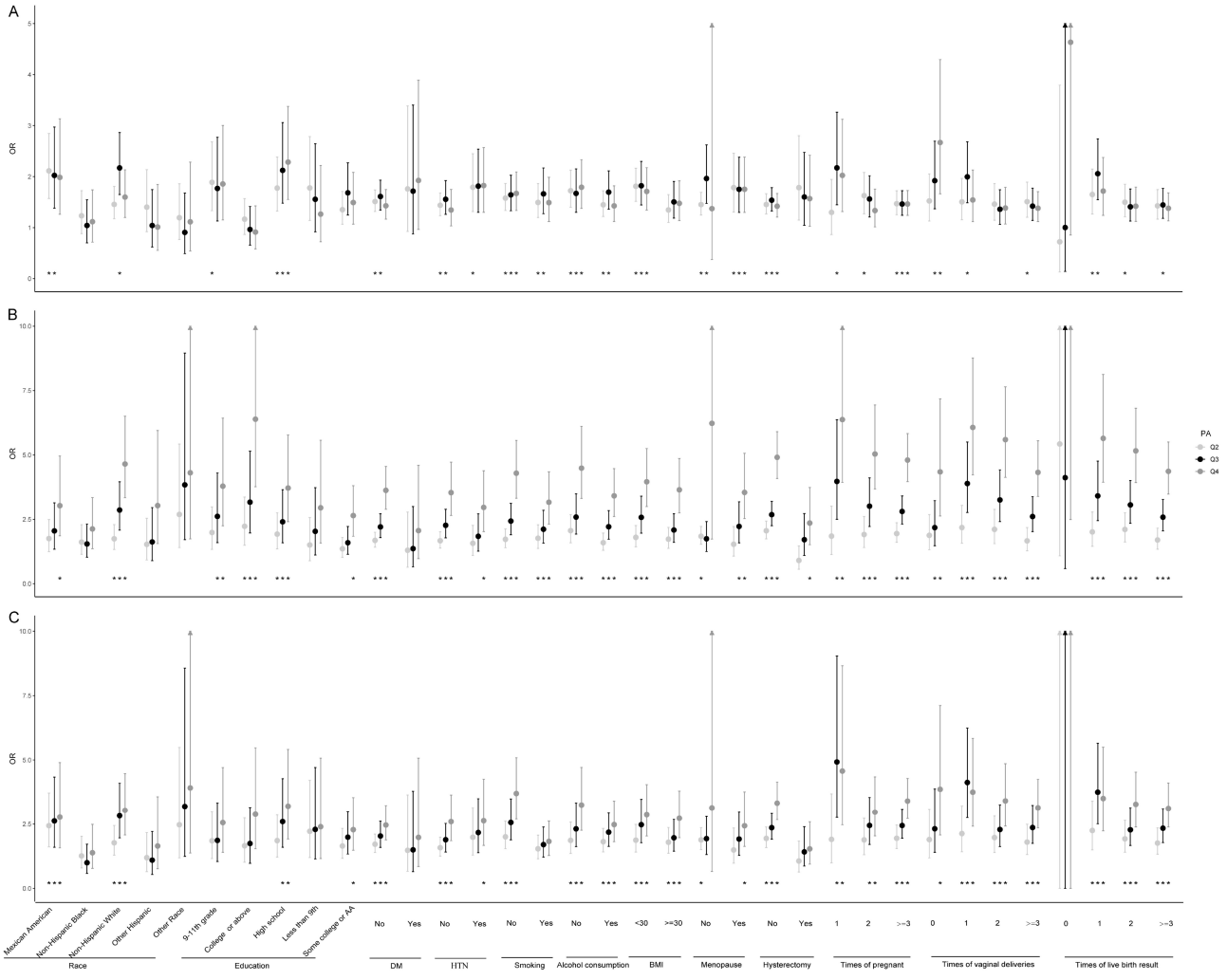

Figure 4 The results of subgroup analyses between PhenoAge and three UI: (**A**) The results of SUI, (**B**) The results of UUI, (**C**) The results of MUI. *P < 0.05; **P < 0.01; ***P < 0.001.

Abbreviations: DM, diabetes mellitus; HTN, hypertension; BMI, body mass index.

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Sensitivity Analysis

After reanalysis of the data after multiple imputation, 12,384 subjects were ultimately included. After conducting logistic and RCS analysis again, we found that the above results still hold (Figure S5–S7).

Discussions

Biological aging was increasingly recognized as a systemic driving factor for many diseases.[3,18,19] By integrating three complementary biomarkers, we provided the first side-by-side evaluation of how PA, KDM, and HD related to SUI, UUI, and MUI in women. In this cross-sectional study, confounder-adjusted RCS models revealed distinctly non-linear relationships between biological aging indices and UI. For SUI, the OR climbed steeply as PA or KDM increased, yet only up to inflection points of approximately 43.6 and 34.7 units, respectively; beyond these thresholds, the upward trajectory flattened and the risk plateaued. For both UUI and MUI, the association with KDM assumed a clear inverted-U configuration: OR rose steadily with increasing KDM until the inflection points of 40.7 and 40.5, respectively, beyond which a marked attenuation was observed. Although HD failed to demonstrate any discernible association with UI, the robust and significant findings for PA and KDM were sufficient to establish a positive link between biological ageing and UI, most notably the inverted-J-shaped relationship between SUI and the burden of biological ageing.

Although prior investigations had not directly interrogated the relationship between biological aging and UI, there was much evidence that demonstrates a progressive rise in UI prevalence with advancing chronological age.[20–23] However, some studies have reported that the relationship between UI and chronological age is not entirely linear. A previous epidemiological study on NHANES showed that the incidence of SUI peaked in the fifth decade.[24] Una J Lee et al similarly reported that the prevalence of SUI peaks among women aged 40–59 years.[13] However, the prevalence of both UUI and MUI exhibited a monotonic increase with advancing age.[13,24] This aligns with our findings regarding the association between the biological aging indicator PA and UI: an inverted J-shaped relationship for SUI, and a linear, positive association for both UUI and MUI.

Whereas excess adiposity has traditionally been framed as the dominant driver of UI,[25] no study has examined how aging relates to UI within different BMI strata. In our subgroup analyses, participants with BMI < 30 exhibited higher ORs than those with BMI ≥ 30, suggesting that the pelvic floor of leaner individuals may be disproportionately vulnerable to senescence-related effects. Although prior studies have documented a positive link between higher BMI and epigenetic indices of biological aging,[26] the "obesity paradox" simultaneously demonstrates that excess adiposity can coexist with metabolic health and favourable disease trajectories,[27] collectively underscoring the intricate relationship among BMI, aging, and disease. Within this complexity, low BMI precipitates atrophy of the pelvic-floor musculature, leaving the supportive scaffold thinner and intrinsically frailer; this structural vulnerability renders leaner adults distinctly more susceptible to the cumulative toll of aging, thereby markedly amplifying their risk of urinary incontinence. Nevertheless, many investigators regard the "obesity paradox" as a statistical or methodological artifact rather than a genuine biological protection. Therefore, longitudinal studies are still required to clarify the complex interplay among BMI, biological aging, and UI before clinical inferences are drawn. Critically, the inverted-J and inverted-U trajectories linking PA and KDM to UI remained intact across every BMI stratum, underscoring the robustness of these relationships beyond adiposity-related pathways. Within strata defined by reproductive risk, the associations exhibited heterogeneity.[28] Nevertheless, as the burden of traditional reproductive risk factors escalated, the OR linking PA and KDM with UI progressively attenuated. This graded diminution implies that, relative to obstetric insults, biological ageing contributes modestly to UI pathogenesis. While oestrogen, androgen, and α-Klotho represent biologically plausible mediators, their availability was limited to a single NHANES cycle, resulting in >30% missingness and constraining both statistical power and generalisability. Future investigations should therefore pool multiple cycles or leverage prospective cohorts with complete biomarker data to validate and extend these mechanistic pathways.

A large body of evidence now links accelerated biological aging to a spectrum of chronic disorders, ranging from endometrial cancer to major depressive disorder.[3,29] Our findings extend this landscape by demonstrating that heightened biological age—captured by PA and KDM—also confers an increased risk of UI, particularly SUI in an inverted-J fashion. Importantly, recent studies have shown that modifiable lifestyle factors can decelerate epigenetic and phenotypic aging, such as diet and sleep.[30,31] Taken together, these observations raise the possibility that

lifestyle interventions aimed at slowing biological aging—such as improving diet and sleep quality—may not only mitigate cardiometabolic and neoplastic risk, but also alleviate the burden of UI in women.

This study was the first to provide a head-to-head comparison of three mechanistically distinct biological-aging indicators—PA, HD, and KDM—to SUI, UUI, and MUI in a large, population-based cohort. Besides, we conducted comprehensive sensitivity analyses, demonstrating that our findings are robust to potential bias. Finally, the use of RCS analyses allowed us to capture non-linear dose–response relationships that would have been obscured by conventional linear models. Despite these strengths, several limitations merit consideration. The cross-sectional design precludes causal inference; longitudinal or Mendelian-randomisation studies are required to establish temporality and directionality. Although we adjusted for a wide array of confounders, residual or unmeasured confounding cannot be excluded. Because survey-weighted estimation is not supported by the RCS and LASSO algorithms, we report unweighted results; this choice may modestly diminish the representation of minority groups and could limit generalisability, although weighted analyses produced materially similar effect estimates.

Conclusion

Biological aging shows a significant positive association with UI, and its relationship with SUI follows an inverted J-shaped curve. Interventions that slow biological aging may offer a novel strategy for UI prevention; however, longitudinal studies are required to establish causality.

Abbreviations

PA, PhenoAge; KDM, Klemera-Doubal Methodology; HD, Homeostatic Dysregulation; NHANES, National Health and Nutrition Examination Survey; UI, Urinary incontinence; SUI, Stress urinary incontinence; UUI, Urgency urinary incontinence; MUI, Mixed urinary incontinence; NCHS, National Center for Health Statistics; DM, Diabetes mellitus; HTN, Hypertension; BMI, Body mass index; ALP, Alkaline phosphatase; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; TC, Total cholesterol; RCS, Restricted cubic spline; OR, Odds ratios.

Data Sharing Statement

Publicly available datasets were analyzed in this study. These data can be downloaded from: https://www.cdc.gov/nchs/nhanes/. Additional data files used for analysis are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study is a secondary analysis of anonymized public data. The NHANES survey protocols were approved by the NCHS Research Ethics Review Board, and all participants provided informed consent (https://www.cdc.gov/nchs/nhanes/about/erb.html). In accordance with Article 32, Items 1 and 2 of the Measures for Ethical Review of Life Science and Medical Research Involving Human Subjects (China, 2023), this study is exempt from local IRB review.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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