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The Association of Body Temperature with Longevity: Insights from Historical Cohorts

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Abstract: Effective thermoregulation is crucial for maintaining homeostasis. Previous research has suggested a link between lower steady-state body temperature and longevity, particularly in physically healthy, non-obese older adults. However, the exact mechanisms behind this relationship remain unclear. Despite the physiological insights gained from studies on body temperature, limited attention has been given to its potential role as a biomarker of longevity in physically healthy older populations. This study aimed to evaluate the relationship between body temperature and longevity using historical data from two cohorts. The longitudinal cohort consisted of 142 individuals, followed for 25 years beginning at age 45, while the cross-sectional cohort included 204 individuals stratified into four lifespan categories. To examine age-related trends in body temperature, Page's test was employed, and ordinal regression was used. The analysis revealed a significant decrease in body temperature in women with age, while men showed no significant change. The cross-sectional analysis indicated a trend toward lower body temperatures in individuals with longer lifespans. Lower body temperature may reflect a reduced metabolic rate, thereby mitigating oxidative stress and molecular damage, both of which are known to drive aging and limit lifespan. Furthermore, lower body temperatures may signal a favorable inflammatory profile, which could translate into slower aging and increased survival. However, the observed sex-specific differences in thermoregulatory patterns raise important questions about the role of hormonal influences, such as estrogen levels. Overall, these findings suggest that lower lifetime steady-state body temperature may be a biomarker of healthy aging and longevity, warranting further exploration of its mechanistic underpinnings.

Key words: age, aging, biomarker, body temperature, lifespan, longevity, survival

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Introduction

Identifying reliable biomarkers of healthy aging and longevity is one of the central challenges in biogerontology and medical research (Martin-Ruiz et al. 2011; Dodds et al. 2014; Arai et al. 2015; Sayer and Kirkwood 2015; Chen et al. 2016; Davis et al. 2016; Ferrucci et al. 2018; Levine et al. 2018; Smith et al. 2019; Guerville et al. 2020; He et al. 2024). Among the various candidates, core body temperature stands out as an intriguing and potentially informative biomarker (Conti 2008; Lehmann et al. 2013; Keil et al. 2015), as studies have associated lower temperatures with longer lifespan and higher temperatures with shorter lifespan in diverse species, including animal models of aging (e.g., *Caenorhabditis elegans*, *Drosophila melanogaster*, and mice) as well as humans (Rikke and Johnson 2004; Waalen and Buxbaum 2011; Palani et al. 2023 Chmielewski et al. 2025). Reflecting the delicate equilibrium between heat production and dissipation, body temperature not only underpins homeostatic control but also encapsulates the cumulative effects of metabolic, immunological, and environmental influences on aging organisms (Roth et al. 2002; Ruggiero et al. 2008; Åström et al. 2011; Soare et al. 2011; Keil et al. 2015; Geneva et al. 2019; Lee et al. 2023; Kowald et al. 2024; Li et al. 2024).

In healthy individuals, body temperature follows a circadian rhythm, typically reaching its lowest point in the early morning and peaking in the late afternoon. Such diurnal fluctuations underscore the importance of considering the timing of temperature measurements, as sporadic readings may fail to capture the basal set point that is critical for assessing long-term health and survival (Simonsick et al. 2016). The distinction between adaptive and maladaptive alterations in body temperature is further highlighted by the differential responses seen in hyperthermia versus fever. Hyperthermia is characterized by an excessive accumulation of heat that overwhelms the body's dissipative mechanisms, which is harmful to health. In contrast, fever is a regulated increase in the body's temperature set point, which is orchestrated by endogenous pyrogens such as interleukins (e.g., IL-1, IL-6, and IL-8), interferons (e.g., interferon-γ), tumor necrosis factor-β etc., in response to infectious or inflammatory stimuli. The fever response represents an adaptive strategy that evolved to combat pathogens and increase survival.

Previous studies have suggested that lower basal body temperature may be a biomarker of healthy aging and greater longevity, particularly in physically healthy, non-obese older adults (Waalen and Buxbaum 2011; Simonsick et al. 2016; Chmielewski et al. 2025). However, this association remains understudied in the Polish population, and it is unclear whether reduced core temperature directly influences longevity or simply serves as a surrogate marker for other health-promoting processes. Enhanced immune responses, decreased chronic low-grade systemic inflammation (CLSI), and the absence of disease or infirmity may all contribute to a reduced temperature profile, which could also correlate with longevity benefits in the elderly population (Franceschi and Campisi 2014; Nilsson et al. 2014; Proctor et al. 2015; Chmielewski et al. 2016; Chmielewski and Strzelec 2018; Ferrucci and Fabbri 2018).

One should consider whether there are factors and mechanisms that underlie the association between lower life-

time steady-state body temperature and extended longevity, and, if so, identify what they are. For instance, the phenomenon of lower body temperature has been closely linked with caloric restriction (CR), which is a well-established intervention that promotes longevity across a range of species (Colman et al. 2009; Fontana et al. 2010; Anderson and Weindruch 2010; Chmielewski 2017; 2020; Picca et al. 2017; Campisi et al. 2019; Dorling et al. 2020; Speakman 2020; Giacomello and Toniolo 2021; Hoong and Chua 2021; Sultanova et al. 2021; Waziry et al. 2023; Di Francesco et al. 2024; Greenhill 2024). CR is known to induce a metabolic shift characterized by reduced energy expenditure and improved physiological efficiency, which is often accompanied by a modest decline in core temperature (Carrillo and Flouris 2011).

Furthermore, subclinical conditions such as endocrine disorders, latent infections (e.g., tuberculosis, hepatitis B and C, and HIV), autoimmune disorders (e.g., lupus), as well as insulin resistance, metabolic dysregulation, and type 2 diabetes mellitus, have been linked to elevated body temperature and reduced survival. Moreover, unhealthy lifestyle factors, including chronic psychological stress, long-term alcohol consumption, and inadequate sleep, can lead to changes in inflammatory cytokines and white blood cell counts (Mullington et al. 2010; Knutson 2012; Chen et al. 2024). Conversely, progressive sarcopenia and atherosclerosis—conditions commonly observed in older adults—can lead to a decline in body temperature, but they are also associated with increased cardiovascular risk and premature mortality (Barquera et al. 2015; Herrington et al. 2016; Agnelli et al. 2020; Bayraktar et al. 2020; He et al. 2021).

Despite the physiological insights gained from studies on body temperature (Lu et al. 2010; Obermeyer et al. 2017; Diamond et al. 2021), little attention has been devoted to its potential role as an independent biomarker of longevity in physically healthy older populations. Most clinical measurements of body temperature are conducted during acute illness or hospitalization, which restricts our understanding of its normative patterns in the context of longevity among community-dwelling older adults. This gap is especially pronounced in historical cohorts, where comprehensive longitudinal data are extraordinarily scarce. Consequently, key questions regarding the typical profiles of core body temperature and their association with reliable markers of survival (e.g., inflammatory biomarkers and epigenetic 'clocks') in long-lived versus short-lived individuals remain largely unexplored.

This study aims to address this gap by analyzing both longitudinal and cross-sectional data to investigate whether lower body temperature is associated with greater longevity in physically healthy older adults within the Polish population.

Materials and methods

Study Population

The study adhered to the principles of the Declaration of Helsinki. Archival clinical data from physical examinations at the Mental Health Center in the vicinity of Zielona Góra, Lubuskie Province, Poland, were used for this research. Ethical approval for the study was granted by the institutional review board in 2007 as part of a doctoral research project. All medical records were anonymized to protect

patient confidentiality and subsequently used to construct a comprehensive database incorporating both longitudinal and cross-sectional data.

The longitudinal cohort comprised 142 residents (68 men and 74 women), who were monitored continuously from ages 45 to 70 years. These individuals reached the age of 70 years, after which their outcomes were not further tracked. The cross-sectional cohort consisted of 204 individuals, including 98 men and 106 women, who were assessed during periodic clinical examinations at multiple intervals. These participants were stratified into four lifespan categories based on death certificates: (1) short lifespan: 15 men (aged 50–58 years, mean age 53 years) and 12 women (aged 50–58 years, mean age 53 years), (2) medium lifespan: 26 men (aged 58–65 years, mean age 63 years) and 30 women (aged 58–65 years, mean age 63 years), (3) long lifespan: 42 men (aged 65–72 years, mean age 68 years) and 40 women (aged 65–72 years, mean age 68 years), and (4) very long lifespan: 15 men and 24 women (aged 76+). The short lifespan category included only individuals who lived significantly below their life expectancy at birth ($< e_0$), while the medium and long lifespan categories contained individuals with life expectancies close to e_0. The very long lifespan category exclusively included individuals who surpassed 76 years, thus exceeding the e_0 threshold.

Physiological Measurements

Sublingual body temperature (°C) was measured monthly under clinical conditions using a standard thermometer with 0.1°C accuracy. All measurements were taken systematically by trained medical personnel in standardized conditions at the same medical institution, typically in the morning. This study used only averaged data derived from 60 measurements per 5-year period for each individual in the longitudinal cohort, resulting in 300 measurements per person over the entire study period.

In the cross-sectional cohort, each individual contributed at least several dozens of measurements. These rigorous data collection practices ensured statistical robustness. Comprehensive details regarding the study cohorts, including the daily routines of patients and medical staff, as well as the data collection procedures, have been documented in previous publications (Borysławski et al. 2015; Chmielewski et al. 2015; 2016; 2017; 2025).

Statistical Analysis

To calculate reliable estimates of central tendency and variability, we aggregated frequently repeated measurements for each participant, including arithmetic means, medians, percentiles, and standard deviations (SDs). This approach minimized variability and enhanced the reliability of the findings. The normality of data distribution was tested with the Shapiro-Wilk test (Shapiro and Wilk 1965). The significance level was set at 0.05.

To examine whether a trend exists in body temperature with age, Page's test (Page 1963) was employed. This test serves as an alternative to Friedman's test and has greater statistical power. The null hypothesis in Page's test, similar to Friedman's test, assumes equality among the measures of central tendency across all analyzed groups. However, the alternative hypothesis in Page's test differs from that in Friedman's test. It posits that for the measures of central tendency in n studied groups—$\theta_1, \theta_2, \theta_3, \ldots, \theta_n$—the

following sequence of inequalities holds: $\theta_1 \leq \theta_2 \leq \theta_3 \leq \ldots \leq \theta_n$, with at least one strict inequality. This implies the presence of an increasing trend in the measures of central tendency. In the present analysis, this would correspond to an increase in the median values of the studied variables across successive age groups: 45, 50, 55, 60, 65, and 70 years.

Ordinal regression was conducted using the Cumulative Link Model (CLM), which accounts for covariates and provides a robust framework for modeling ordinal outcomes. All statistical analyses were performed using R software (R Foundation for Statistical Computing, Vienna, Austria).

Results

Longitudinal Cohort

The normality of data distribution was confirmed by the Shapiro-Wilk test ($p > 0.05$). In men, no significant change in body temperature was observed over the study period (Table 1, Fig. 1), as Page's test did not reveal any significant increasing or decreasing trend in body temperature for men (test statistic = 4965.5; $p = 0.692$). In contrast, for women, Page's test identified as significant decreasing trend (test statistic = 5876; $p < 0.05$), indicating a significant decline in body temperature associated with aging (Table 2, Fig. 1).

Table 1. Basic descriptive statistics of age-related changes in body temperature in the longitudinal data for men who were examined for 25 years, starting from age 45 onwards

Age	Men					
	Min	Q_1	Median	Q_3	Max	Mean ± SD
45	36.0	36.4	36.6	36.7	37.2	36.6 ± 0.2
50	36.2	36.5	36.6	36.6	37.0	36.6 ± 0.2
55	36.0	36.5	36.6	36.7	37.0	36.6 ± 0.2
60	36.3	36.5	36.6	36.7	37.0	36.6 ± 0.2
65	36.2	36.5	36.6	36.7	36.9	36.6 ± 0.2
70	36.0	36.4	36.6	36.7	37.2	36.6 ± 0.1

Table 2. Basic descriptive statistics of age-related changes in body temperature in the longitudinal data for women who were examined for 25 years, starting from age 45 onwards

Age	Women					
	Min	Q_1	Median	Q_3	Max	Mean ± SD
45	35.8	36.4	36.5	36.6	37.0	36.5 ± 0.3
50	36.0	36.4	36.5	36.6	36.9	36.5 ± 0.2
55	36.0	36.5	36.6	36.6	36.9	36.6 ± 0.2
60	36.2	36.5	36.6	36.6	36.9	36.6 ± 0.2
65	36.2	36.5	36.6	36.7	37.0	36.6 ± 0.2
70	36.2	36.5	36.6	36.8	37.1	36.6 ± 0.2

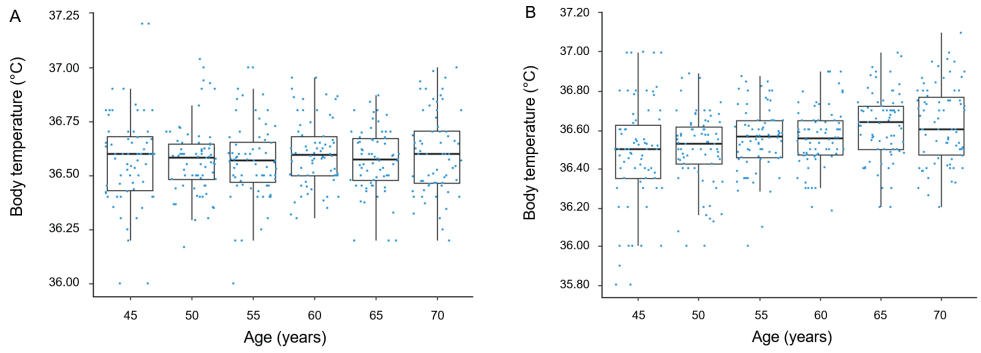

Fig. 1. Age-related trends in body temperature for men (panel A) and women (panel B) based on longitudinal data, stratified into six consecutive age categories. In the box-and-whisker plots, the bold line within each box represents the median, while the lower and upper edges denote the first and third quartiles, respectively. Whiskers extend to the most extreme values within 1.5 times the interquartile range from the quartiles, and values beyond this range are plotted as outlier points

Cross-Sectional Cohort

The basic descriptive statistics for men and women in the cross-sectional cohort are summarized in Tables 3 and 4, respectively. Age-related changes in measures of central tendency, along with standard deviations across consecutive lifespan categories, are presented in Fig. 2. The cross-sectional analysis revealed a trend toward lower body temperatures in long-live men and women, but it was statistically non-significant ($p > 0.05$). The results of the CLM analysis for men and women are provided in Table 5.

Table 3. Basic descriptive statistics of survival-related changes in body temperature in the cross-sectional data for men who were examined for several years until their death

Lifespan category	Men					
	Min	Q_1	Median	Q_3	Max	Mean ± SD
Short	36.3	36.4	36.7	36.8	36.9	36.6 ± 0.2
Medium	36.0	36.5	36.6	36.6	36.8	36.5 ± 0.2
Long	36.2	36.5	36.5	36.6	37.0	36.5 ± 0.2
Very long	36.0	36.4	36.6	36.6	36.7	36.5 ± 0.2

Table 4. Basic descriptive statistics of survival-related changes in body temperature in the cross-sectional data for women who were examined for several years until their death

Lifespan category	Women					
	Min	Q_1	Median	Q_3	Max	Mean ± SD
Short	36.0	36.5	36.6	36.8	36.9	36.6 ± 0.2
Medium	36.3	36.5	36.6	36.7	37.0	36.6 ± 0.2
Long	36.0	36.5	36.5	36.6	37.0	36.5 ± 0.2
Very long	36.0	36.5	36.6	36.7	36.8	36.5 ± 0.2

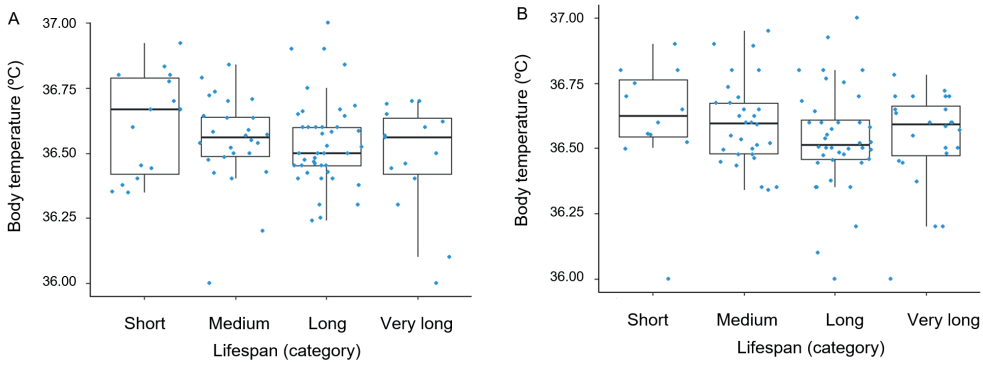

Fig. 2. Survival-related trends in body temperature for men (panel A) and women (panel B) based on cross-sectional data, stratified into four lifespan categories. In the box-and-whisker plots, the bold line within each box represents the median, while the lower and upper edges denote the first and third quartiles, respectively. Whiskers extend to the most extreme values within 1.5 times the interquartile range from the quartiles, and values beyond this range are plotted as outlier points

Table 5. Cumulative Link Model (CLM) outcomes in both sexes

Sex	Estimate	Standard Error	z-value	Pr (>|z|)	Odds Ratio	2.5%	97.5%
Men	–1.892	1.086	–1.742	0.0815	0.1508	0.0172	1.236
Women	–1.486	0.948	–1.568	0.1169	0.2262	0.0344	1.442

Discussion

This study builds on previous research investigating the relationship between resting body temperature and longevity (Chmielewski et al. 2015; 2025) by analyzing historical data from long-term residents of the same mental health center. The findings offer novel insights into the association between body temperature and long-term survival. Specifically, the analysis revealed sex-specific differences in long-term trends, warranting further investigation into the link between lower body temperature and increased longevity.

The longitudinal analysis showed that body temperature declined with advancing age in women, while no significant age-related trend was observed in men. Similarly, the cross-sectional data, which categorized individuals by lifespan, revealed a downward trend, with older individuals tending to have lower body temperatures compared to those with shorter lifespans. Although this difference did not reach statistical significance, it suggests a potential trend worthy of further exploration. For instance, it was claimed that because women generally have a higher body temperature than men—and yet consistently outlive them—it is unlikely that core body temperature affects longevity (see Introduction). However, studies have shown that women have only a slightly higher body temperature than men (approximately 0.5 °C, largely attributable to temperature fluctuations during the menstrual cycle, which diminish after menopause), and our analysis clearly demonstrated

that during the study period—between the ages of 45 and 70—a statistically significant reduction in body temperature occurred in women but not in men. Thus, since only women experienced a significant reduction in body temperature while living longer, the notion that core body temperature does not affect human longevity becomes less tenable.

This serves as an example of the challenges in redefining classical models and views on aging in light of emerging experimental evidence (Chmielewski 2017; 2020). One classical theory on the evolution of aging is the Disposable Soma Theory of Aging (DSTA), formulated by Thomas Kirkwood (1977), which posits that aging evolved as a byproduct of natural selection due to an evolutionary trade-off between resources allocated to somatic maintenance and sexual reproduction—that is, the more an organism invests in sexual reproduction, the less is available for somatic maintenance, and *vice versa* (Kirkwood and Holliday 1979; Kirkwood and Rose 1991; Drenos and Kirkwood 2005). This influential, mathematically rigorous, and elegant theory holds that our bodies can be considered as disposable 'containers' for our genes and that, beyond an 'essential lifespan' (roughly between 35 and 45 years), they begin to deteriorate because evolution did not expect them to function indefinitely or much longer than this critical period, e.g., due to the selection shadow (Chmielewski 2017; 2019).

Although alternative models have been proposed (Maklakov and Chapman 2019; Speakman 2020; Carlsson et al. 2021; Gems 2022; Lemaître et al. 2024; Mitchell et al. 2024), including markedly different perspectives (Longo et al. 2005; Longo and Anderson 2022), the DSTA remains one of the more robust and influential frameworks in current biogerontology (Jasienska 2009; Hammers et al. 2013; Ziomkiewicz et al. 2016; Jasienska et al. 2017; Collins et al. 2023). Indeed, the DSTA can help elucidate our findings: despite investing more in sexual reproduction, women still live longer than men. Furthermore, the higher core body temperature that women experience during their fertile period (e.g., due to hormonal changes during the menstrual cycle) may represent one example of the biological costs of reproduction that women incur. Taken together, these findings suggest that lower lifetime steady-state body temperature may be associated with increased longevity. This finding is in agreement with previous studies (Rikke and Johnson 2004; Waalen and Buxbaum 2011; Simonsick et al. 2016; Palani et al. 2023 Chmielewski et al. 2025).

We hypothesize that a lower body temperature may reflect a reduced basal metabolic rate (BMR), which is a condition that has been associated with decreased production of reactive oxygen species (ROS) and a consequent reduction in cumulative molecular and cellular damage. In animal models, lower metabolic rates have been correlated with extended lifespans, positing that slower metabolic rates may help to mitigate the deleterious effects of oxidative stress. Additionally, the possibility exists that lower body temperature is indicative not only of reduced metabolic activity but also of a more favorable inflammatory profile, as elevated body temperature may signal the presence of chronic systemic inflammation, subclinical diseases, latent infections, or autoimmune processes—conditions that are known to contribute to age-related morbidity and mortality (Chmielewski 2018; Chmielewski and Strzelec 2018). Our ob-

servation that short-lived individuals tend to have higher temperatures suggests that elevated body temperature could indicate an underlying, suboptimal inflammatory state. This may predispose aging individuals to earlier mortality. Conversely, a lower body temperature in long-lived individuals could denote a more robust immune system or an absence of deleterious inflammatory activity, thereby supporting longevity.

The sex-specific divergence observed in our study enriches the discussion. Women not only displayed a significant decline in body temperature with advancing age, but they also, as other studies have shown, tend to have slightly higher baseline temperatures than men, yet women consistently outlive men (McGann et al. 1993; Chmielewski 2015; 2016; 2022; 2024; Keil et al. 2015; Chmielewski and Borysławski 2016; Baum et al. 2021; Öngel et al. 2021; Chmielewski et al. 2023). The dichotomy between the temperature trends observed in men and women raises intriguing questions about the underlying physiological mechanisms at play. It is possible that hormonal differences, variations in body composition, or disparities in the prevalence of autoimmune conditions contribute to these sex-specific patterns. For instance, the higher propensity for autoimmune disorders among women might initially elevate body temperature (Dolgin 2024). However, as adaptive mechanisms evolve, a subsequent decline might reflect a rebalancing that ultimately favors longevity. In contrast, the absence of a similar trend in men could indicate that other compensatory mechanisms, such as differences in metabolic regulation or thermogenic responses, come into play.

The interplay between body mass index (BMI), systemic inflammation, and body temperature should not be overlooked. Prior research has documented a positive association between higher BMI and increased body temperature, as well as between elevated temperature and higher mortality rates (Waalen and Buxbaum 2011; Simonsick et al. 2016; Chmielewski et al. 2025). It is plausible that individuals with a lower BMI, who may also experience reduced systemic inflammation, are more likely to exhibit a lower steady-state temperature and, consequently, a longer lifespan. This hypothesis is further bolstered by the observation that higher white blood cell counts—often reflective of ongoing inflammatory processes—are associated with poorer health outcomes in older adults (Ruggiero et al. 2007; Nilsson et al. 2014; Chmielewski 2018; Chmielewski et al. 2016; Chmielewski and Strzelec 2018). The converging lines of evidence thus suggest that a low basal temperature might be more than a passive marker of metabolic rate; it could also be a surrogate for an overall anti-inflammatory state that is conducive to healthy aging.

Notwithstanding the implications of these findings, several limitations must be acknowledged. First, the reliance on historical data from a specific institutionalized population raises questions about the generalizability of the results to the broader aging population, as clinical data may be affected by confounding variables (Chmielewski et al. 2015; 2016; 2025). Second, the cross-sectional component, while suggestive of a relationship between temperature and longevity, is inherently limited by its observational nature and the potential for confounding variables—such as undiagnosed subclinical conditions or lifestyle factors—that may not have been fully accounted for.

Despite these constraints, our study contributes to a growing body of literature that challenges traditional interpretations

of age-related thermoregulatory decline. Rather than viewing lower body temperature simply as a result of diminished thermoregulatory function in the elderly, our findings raise the possibility that a lower steady-state temperature may be an adaptive trait reflecting a finely tuned balance between metabolic efficiency, immune function, and systemic inflammation. This interpretation suggests that effective interventions targeting the underlying mechanisms of aging could one day offer novel strategies for promoting longevity (Chmielewski et al. 2024; Li et al. 2024; Mahoney et al. 2024).

Future investigations should aim to clarify these relationships by employing prospective, population-based designs with rigorous standardization of temperature measurements. Such studies would benefit from the inclusion of a comprehensive set of biomarkers, including detailed assessments of metabolic rate, inflammatory mediators, and immune function, in order to disentangle the complex interdependencies underlying the observed associations. Furthermore, exploring the molecular and genetic determinants of thermoregulation across different populations could provide insight into why some individuals exhibit lower baseline temperatures and enjoy a survival advantage.

In conclusion, this study offers preliminary evidence suggesting that lower lifetime steady-state body temperature could be a biomarker of longevity. The observed trends, suggesting that long-lived individuals tend to have lower body temperature, support the hypothesis that a lower metabolic rate and reduced systemic inflammation are beneficial for survival. However, the sex-specific differences and the lack of statistically significant differences between lifespan categories caution against oversimplification and highlight the complexity of the relationship between body temperature and longevity. Our findings also emphasize the need for further research to clarify the causal pathways and potential clinical implications of these associations.

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Contributions from individual authors

PPC developed and designed the study, collected the data, performed the statistical analyses, conducted the literature search and collected all pertinent references, interpreted the results, drafted the initial version of the manuscript and all subsequent versions, as well as produced all figures and tables for this manuscript. KC planned and managed the research project, collected the data, and contributed to the critical review of the manuscript. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Conflict of interest

The authors have no conflicts of interest to declare.

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