## Original Articles

# Association Between Occupational High-Temperature Exposure And The Biological Aging of Workers

Yan Guo[1,2,&], Rui Zhao[1,&], Weichao Wu[3], Jinru Chen[3], Xiangkai Zhao[2], Bin Yang[2], Zhiguang Gu[2], Dongsheng Hu[1], Ming Zhang[1,#], and Wei Wang[2,#]

*1. Department of Biostatistics and Epidemiology, School of Public Health, Shenzhen University Medical School, Shenzhen 518060, Guangdong, China; 2. Department of Labor Health and Occupational Diseases, School of Public Health, Zhengzhou University, Zhengzhou 450001, Henan, China; 3. Public Health Service Center of Bao'an District, Shenzhen 518102, Guangdong, China*

**Abstract**

**Objective** To investigate the association between occupational high-temperature exposure and accelerated biological aging.

**Methods** A total of 140 male workers exposed to occupational high-temperatures and 207 male non-exposed control workers were selected as study subjects. Questionnaire surveys and health examinations were conducted. Biological age and organ-specific biological age were calculated using the Klemera–Doubal method. Generalized linear models were used to analyze the effects of occupational high-temperature exposure, body mass index (BMI), smoking, alcohol consumption, and sleep duration on biological age (BA) acceleration and organ-specific biological age.

**Results** Significant differences were observed between the exposed and control groups in length of service, systolic blood pressure, red blood cell count, albumin levels, urea, creatinine, BA acceleration, and liver–kidney BA acceleration ($P < 0.05$). Compared with the control group, which showed a BA acceleration of 0.04 ± 1.34 years, the exposed group demonstrated significantly higher BA acceleration of 0.62 ± 1.31 years. After adjustment for covariates, workers exposed to high-temperatures exhibited significantly higher BA acceleration and liver–kidney BA acceleration than controls ($P < 0.001$). High-temperature exposure and BMI were associated with BA acceleration, with a significant interaction between the two factors ($P < 0.05$). High- temperature exposure, BMI, and smoking were identified as risk factors for BA acceleration, whereas sleep duration was a protective factor ($P < 0.05$).

**Conclusion** Occupational high-temperature exposure may accelerate biological aging. An interaction exists between occupational high-temperature exposure and BMI in relation to BA acceleration.

**Key words:** Aging; Occupational Exposure; High-temperature; Biological aging

*Biomed Environ Sci, 2026; 39(x): 1-8* *doi: 10.3967/bes2026.016* *ISSN: 0895-3988*

*www.besjournal.com (full text)* *CN: 11-2816/Q* 

[&]These authors contributed equally to this work.

[#]Correspondence should be addressed to Ming Zhang, PhD, Tel: 86-13265565637, E-mail: zhangming@szu.edu.cn; Wei Wang, E-mail: ww375@zzu.edu.cn

Biographical notes of the first authors: Yan Guo, Bachelor, majoring in the direction of labor health and environmental health, E-mail: gyzz@gs.zzu.edu.cn; Rui Zhao, Bachelor, majoring in the direction of labor health and environmental health, E-mail: rui2745@gs.zzu.edu.cn

## INTRODUCTION

Aging is a physiological process mediated by numerous biological and genetic pathways. The senescence of tissues, organs, and cells primarily manifests as a gradual decline in adaptability and resilience. Aging is also one of the major risk factors for many age-related diseases, including cancer, cardiovascular disorders, and others[1,2].To better assess the extent of human aging, current research predominantly utilizes biomarkers such as biological age, epigenetic alterations, telomere length, mitochondrial dysfunction, and aging clocks to measure the progression of senescence[3,4]. Environmental exposure is linked to human diseases and is also an important factor in accelerating biological aging. Cohort studies have shown that higher lifetime exposure to air pollutants is associated with shorter telomeres, while age exhibits a significant inverse association with telomere length[5]. Microplastics and nanoplastics have been demonstrated to act as potent inducers of cellular senescence by promoting mitochondrial dysfunction, impairing autophagic flux, and activating DNA damage response pathways, thereby exacerbating senescence[6]. The impact of environmental factors on organismal aging has become an increasingly prominent focus in public health research.

High temperature has become an important public health concern. Previous studies have demonstrated that exposure to extremely high-temperature environments significantly increases the risk of damage to vital organs such as the heart, kidneys, liver, and brain, serving as a major contributor to rising morbidity and mortality from cardiovascular and multi-system diseases[7]. Occupational workers are recognized as one of the most vulnerable groups affected by high temperatures. Globally, more than 1 billion workers are exposed to high-temperature environments, which adversely affect both physical and mental health. Elevated workplace temperatures not only reduce work efficiency and productivity but also increase the risk of occupational health complications. Environmental exposure is closely associated with human disease. Studies have shown that high-temperature environments can induce cellular senescence, reduce the proportion of long telomeres, and decrease mitochondrial DNA copy number (mtDNAcn)[8,9]. In studies examining the combined effects of exposure to high PM2.5 levels and high temperatures on DNA methylation, analyses of five epigenetic aging indicators revealed that short-term exposure to high temperatures under low air pollution conditions may accelerate epigenetic aging[10]. However, the relationship between occupational high-temperature exposure and accelerated biological aging in workers remains poorly understood.

We calculated biological age acceleration (BA acceleration) and organ-specific age acceleration indicators to explore the impact of occupational high-temperature exposure on accelerated biological aging among workers in related industries, thereby providing epidemiological evidence to support further investigation into the molecular mechanisms underlying high-temperature–induced biological aging.

## MATERIALS AND METHODS

### *Research Subjects*

The exposed group comprised workers in Shenzhen engaged in food processing, textile manufacturing, and special-material production, whose tasks included material processing, melting, and casting. The control group consisted of workers from a precision machinery company in Shenzhen who were not exposed to industrial heat sources, with a Wet Bulb Globe Temperature (WBGT) index not exceeding the occupational exposure limit. High-temperature work was defined as tasks in which the average WBGT index at the worksite was equal to or greater than 25 °C.

The inclusion criteria for the exposure group were as follows: length of service ≥ 1 (years); WBGT index ≥ 25 °C (in accordance with GB/T4200–2025); no history of exposure to other occupational hazards; and age between 20 and 60 years. The inclusion criteria for the control group were no exposure to occupational hazards and age between 20 and 60 years old. The exclusion criteria for both groups included the presence of occupational contraindications; diseases affecting vital organs such as the heart, liver, or kidneys; a diagnosis of malignant tumors; or incomplete health examination or questionnaire data.

Based on the following sample size calculation formula, with a two-sided significance level of $\alpha$ = 0.05, a statistical power of 0.8, and parameters derived from preliminary studies and experimental results ($S \approx 1.5$, $d = 0.6$), the minimum required sample size was calculated to be 98 subjects per group. A total of 347 participants were ultimately

included in this study, comprising 140 in the exposure group and 207 in the control group, as shown in Supplementary Figure S1.

$$n = \frac{2 \times \left(Z_{\alpha/2} + Z_{\beta}\right)^2 S^2}{d^2}$$

### *Data Collection and Index Testing*

Demographic characteristics, including age, sex, race, residence, marital status, education, and occupational history, as well as lifestyle information such as smoking history, alcohol consumption, physical activity, and sleep duration, were collected by trained professional investigators using a structured questionnaire. Physical examinations, including measurements of height, weight, and blood pressure, were conducted by professional medical staff in accordance with standardized procedures. Participants were stratified into non-overweight and overweight groups based on body mass index (BMI), using a cutoff value of 25 kg/m$^2$ (non-overweight: BMI < 25; overweight: BMI ≥ 25). All participants were informed of the study objectives and voluntarily signed informed consent forms prior to enrollment.

### *Clinical Blood Biomarker Detection*

Blood samples were collected by professional medical staff, and routine blood tests and blood biochemical analyses were performed by qualified laboratory technicians. The clinical blood biomarkers assessed included red blood cell count (RBC), platelet count (PLT), albumin (ALB), urea (UREA), creatinine (CREA), triglycerides (TG), total cholesterol (TC), glycated hemoglobin (HbA1c), and high-sensitivity C-reactive protein (Hs-CRP).

### *Biological Age (KDM-BA) and Organ Age Calculation*

Based on previous studies[11,12], this study incorporated 10 serum biochemical indicators, including systolic blood pressure (SBP), RBC, PLT, ALB, UREA, CREA, TG, TC, HbA1c, and Hs-CRP. The Klemera–Doubal method (KDM) was employed to construct the KDM biological age (KDM-BA) indicator[13,14]. Samples from the 2009 China Health and Nutrition Survey (CHNS) database were used to train the model parameters. After obtaining the final model formula, the measured clinical biomarker values of each participant were substituted into the formula to calculate individual KDM-BA. BA acceleration was defined as the difference between KDM-BA and chronological age (CA). Using the same approach, the 10 biomarkers were further categorized into three organ-specific BA groups: liver–kidney BA (ALB, UREA, CREA), cardiometabolic BA (SBP, TG, TC, HbA1c), and immune BA (RBC, PLT, Hs-CRP). Organ ages and corresponding organ age acceleration indices were then calculated for each category[15].

### *Statistical Analysis Methods*

All statistical analyses were conducted using SPSS version 27.0.1.0 and R software (version 4.1). Differences between the high-temperature exposure group and the control group were evaluated using independent-samples *t*-tests, while chi-square tests were employed to compare ratios or proportions of baseline characteristics. Logarithmic transformation was applied to TG and Hs-CRP to approximate a normal distribution prior to the calculation KDM-BA and organ age. After adjusting for covariates including length of service, educational level, smoking status, alcohol consumption, physical exercise, sleep duration, and BMI, generalized linear models were applied to analyze factors associated with BA acceleration and organ age acceleration. The significance level was set at $\alpha = 0.05$ for two-tailed tests.

## RESULTS

### *General Information of the Research Subject*

The WBGT index for the high-temperature environments encountered by exposed workers ranged from 26.9 to 29.3 °C, with a mean value of 27.9 °C. The mean age of workers in the exposed group was 36.32 ± 8.12 years, while that of the control group was 35.78 ± 8.66 years. Working tenure was 7.54 ± 6.10 years in the exposed group and 10.29 ± 8.38 years in the control group. Biological age (BA) measurements showed that the exposed group had a BA of 36.94 ± 8.09 years, compared with 35.82 ± 8.82 years in the control group. Compared with the control group, which had a BA acceleration of 0.04 ± 1.34 years, the exposed group demonstrated significantly higher BA acceleration of 0.62 ± 1.31 years ($P < 0.05$). Statistically significant differences ($P < 0.05$) were observed between the two groups in serum parameters, including SBP, RBC, ALB, UREA, and CREA. Additionally, liver–kidney BA acceleration differed significantly between the two groups ($P < 0.05$). No statistically significant differences were

observed in other demographic characteristics ($P$ > 0.05). The results are presented in Table 1.

### *Association of High-Temperature Exposure with BA Acceleration and Organ Age*

After adjustment for length of service, smoking, alcohol consumption, education level, BMI, physical exercise, and sleep duration, the analysis showed significant differences in BA acceleration and liver–kidney BA acceleration between the exposure and control groups. High-temperature exposure was associated with accelerated biological aging ($\beta$ = 0.60, 95% $CI$ = 0.32, 0.89, $P$ < 0.001) and increased liver–kidney BA acceleration ($\beta$ = 0.60, 95% $CI$ = 0.44, 0.93, $P$ < 0.001). The results are shown in Table 2.

### *Influencing Factors of BA Acceleration and Liver-Kidney BA Acceleration*

Generalized linear models were used to further identify factors influencing BA acceleration and liver–kidney BA acceleration, including high-temperature exposure, length of service, smoking, alcohol consumption, education level, BMI, exercise frequency, and sleep duration. As shown in Table 3, high-temperature exposure and BMI were significantly associated with BA acceleration ($\beta$ = 0.59, 95% $CI$ = 0.31, 0.88, $P$ < 0.001; $\beta$ = 0.36, 95% $CI$ = 0.08, 0.65, $P$ = 0.013). High-temperature exposure, smoking, and sleep duration were significantly associated with liver–kidney BA acceleration ($\beta$ = 0.69, 95% $CI$ = 0.45, 0.93, $P$ < 0.001; $\beta$ = 0.25, 95% $CI$ = 0.01, 0.50, $P$ = 0.042; $\beta$ = −0.25, 95% $CI$ = −0.45, −0.04, $P$ = 0.021). Notably, sleep duration acted as a protective factor against liver–kidney BA acceleration.

### *Combined Effects of High-Temperature Exposure And BMI on BA Acceleration*

The combined effects of high-temperature exposure and BMI on BA acceleration are shown in Table 4. After adjustment for length of service, smoking, alcohol consumption, education level, frequency of physical exercise, and sleep duration, the generalized linear model revealed a significant combined effect of high-temperature exposure and BMI on BA acceleration ($P$ < 0.001). In addition, a significant interaction between high-temperature exposure and BMI on BA acceleration was observed ($P$ = 0.020).

## DISCUSSION

With the intensification of global climate warming and the increasing frequency of extreme high-temperature events, the potential health impacts of occupational heat exposure on workers have attracted growing attention from the scientific community and occupational safety authorities. Healthy aging has emerged as a critical focus in public health and gerontological research. However, current research includes relatively few studies examining the association between high-temperature environments and organismal aging. The results of the present study indicate that occupational high-temperature exposure accelerates biological aging in workers.

In this study, parameters for the KDM-BA model were derived from a Shenzhen-based reference population and subsequently applied to workers exposed to occupational high-temperatures to estimate biological age. BA acceleration and organ-specific age acceleration were further assessed. The results demonstrated a significant acceleration of biological aging in the exposed group, with a value of 0.62 ± 1.31 years, compared with 0.04 ± 1.34 years in the control group. These findings indicate that workers exposed to high temperatures exhibit more pronounced aging characteristics and greater BA acceleration. From an epigenetic perspective, previous research has shown that medium- and long-term exposure to high-temperature environments is associated with increased epigenetic age acceleration[16]. Other studies have demonstrated that elevated temperatures disrupt physiological responses, thereby inducing oxidative stress, DNA damage, and cellular apoptosis[17,18]. Toyoki Maeda et al[19]. reported that exposure to 42 °C inhibited the growth of human umbilical vein endothelial cells, reduced the proportion of long telomeres, and tended to decrease telomerase activity. This observation is consistent with the role of telomerase in maintaining telomere length, suggesting that elevated temperatures may accelerate telomere shortening by suppressing telomerase activity or augmenting telomere damage. For occupational populations, the implementation of effective protective measures is recommended to mitigate the long-term health effects of heat exposure.

Further analyses revealed that elevated temperatures also affected liver–kidney BA acceleration, with more pronounced aging observed in workers exposed to high-temperatures. Under high-temperature conditions, the liver–kidney system plays a central role in metabolism and circulation, and its aging process may be accelerated by heat stress responses. The liver and kidneys

**Table 1.** General data of subjects in two groups

| Characteristics | Exposure group (n = 140) | Control group (n = 207) | $t/\chi^2$ | *P* |
|---|---|---|---|---|
| age | 36.32 ± 8.12 | 35.78 ± 8.66 | −0.587 | 0.558 |
| Length of service (years) | 7.54 ± 6.10 | 10.29 ± 8.38 | 3.541 | < 0.001 |
| SBP (mmHg) | 125.51 ± 13.60 | 128.42 ± 10.89 | 2.112 | 0.036 |
| RBC ($10^{12}$ cells / L) | 5.20 ± 0.45 | 5.32 ± 0.46 | −2.156 | 0.032 |
| PLT ($10^9$ cells / L) | 264.55 ± 50.62 | 262.62 ± 59.43 | −0.325 | 0.746 |
| ALB (g/L) | 47.81 ± 2.15 | 49.77 ± 2.39 | 7.962 | < 0.001 |
| UREA (mmol / L) | 4.49 ± 1.12 | 4.88 ± 1.26 | 3.053 | 0.002 |
| CREA (μmol / L) | 80.00 ± 11.13 | 83.24 ± 9.97 | 2.771 | 0.006 |
| TC (mmol / L) | 4.84 ± 0.94 | 4.92 ± 0.94 | 0.846 | 0.398 |
| HbA1c (%) | 5.60 ± 0.88 | 5.53 ± 0.74 | −0.802 | 0.423 |
| Ln Hs-CRP (mg / L) | −0.22 ± 1.12 | −0.12 ± 1.54 | 0.833 | 0.405 |
| Ln TG (mmol / L) | 0.30 ± 0.71 | 0.24 ± 0.59 | −0.885 | 0.377 |
| BA (years) | 36.94 ± 8.09 | 35.82 ± 8.82 | −1.207 | 0.228 |
| BA acceleration (years) | 0.62 ± 1.31 | 0.04 ± 1.34 | −4.027 | < 0.001 |
| Liver - kidney BA | 37.03 ± 7.95 | 35.82 ± 8.81 | −1.295 | 0.196 |
| Liver - kidney BA acceleration | 0.70 ± 1.21 | 0.05 ± 1.12 | −5.219 | < 0.001 |
| Cardio - metabolism BA | 36.16 ± 8.31 | 35.70 ± 8.65 | −0.498 | 0.619 |
| Cardio - metabolism BA acceleration | −0.16 ± 0.60 | −0.08 ± 0.50 | 1.327 | 0.185 |
| Immune BA | 36.40 ± 8.10 | 35.85 ± 8.67 | −0.602 | 0.548 |
| Immune BA acceleration | 0.08 ± 0.52 | 0.07 ± 0.59 | −0.219 | 0.827 |
| Smoking | | | 1.7693 | 0.183 |
| No | 60 (42.90) | 105 (50.70) | | |
| Yes | 80 (57.10) | 102 (49.30) | | |
| Drinking alcohol | | | 3.74 | 0.053 |
| No | 76 (54.30) | 135 (65.20) | | |
| Yes | 64 (45.70) | 72 (34.80) | | |
| Educational level | | | 0.062 | 0.804 |
| Low | 131 (93.60) | 191 (92.30) | | |
| High | 9 (6.43) | 16 (7.73) | | |
| Physical exercise (times / week) | | | 0.595 | 0.743 |
| < 1 | 45 (32.10) | 73 (35.3) | | |
| 1–4 | 59 (42.10) | 79 (38.2) | | |
| ≥ 4 | 36 (25.70) | 55 (26.6) | | |
| Sleep duration (h) | | | 1.148 | 0.564 |
| < 6 | 12 (8.57) | 15 (7.25) | | |
| 6–8 | 82 (58.6) | 133 (64.30) | | |
| ≥ 8 | 46 (32.9) | 59 (28.50) | | |
| BMI (kg / $m^2$) | | | 1.397 | 0.237 |
| Non-overweight | 81 (57.9) | 134 (64.7) | | |
| Overweight | 59 (42.1) | 73 (35.3) | | |

***Note.*** Low: High school or below; High: Associate degree/university or higher; BMI: Body Mass Index, kg/$m^2$; Non-normally distributed biomarkers (Hs-CRP and TG) were log-transformed.

interact through multiple mechanisms under both physiological and pathological conditions. Experimental studies have shown that heatstroke exacerbates hepatic injury in mice subjected to thermal stress, corroborating the organ-specific aging patterns observed in occupational populations[20,21]. Exposure to high temperatures may induce more severe oxidative stress and inflammatory responses, thereby accelerating liver dysfunction. Under chronic heat stress, high temperatures are more likely to cause structural liver damage and downregulation of genes involved in hepatic lipid metabolism[22]. Wisit Kaewput et al.[23] evaluated the prevalence of heat-related heatstroke in the United States and found that, among hospitalized patients, renal failure due to rhabdomyolysis was the most common complication. In addition, most sugarcane workers in the United States reportedly develop irreversible kidney dysfunction[24]. In high-temperature environments, excessive dehydration, electrolyte imbalance caused by sweating, and physiological responses such as rhabdomyolysis may contribute to chronic kidney disease or renal failure. Therefore, enterprises should place greater emphasis on integrated strategies combining environmental improvements, behavioral interventions, and medical monitoring to enhance workers' self-protection awareness and reduce occupational risks through multidimensional measures.

**Table 2.** Generalized linear model results of high temperature exposure on BA acceleration

| Outcome | *β* (95% *CI*) | *Wald*$\chi^2$ | *P* |
|---|---|---|---|
| BA acceleration | 0.60 (0.32, 0.89) | 17.321 | < 0.001 |
| Liver − kidney BA acceleration | 0.68 (0.44, 0.93) | 29.355 | < 0.001 |
| Immune BA acceleration | 0.03 (−0.09, 0.15) | 0.249 | 0.618 |
| Cardio − metabolism BA acceleration | −0.10 (−0.22, 0.01) | 3.102 | 0.078 |

***Note.*** The model includes exposure, length of service, education level, smoking, drinking alcohol, physical exercise, sleep duration, and BMI.

**Table 3.** Influencing factors of BA acceleration

| Outcome | Variables | *β* (95% *CI*) | *Wald*$\chi^2$ | *P* |
|---|---|---|---|---|
| BA acceleration | Expose | 0.59 (0.31, 0.88) | 17.097 | < 0.001 |
| | BMI | 0.36 (0.08, 0.65) | 6.209 | 0.013 |
| Liver − kidney BA acceleration | Expose | 0.69 (0.45, 0.93) | 30.757 | < 0.001 |
| | Smoking | 0.25 (0.01, 0.50) | 4.154 | 0.042 |
| | Sleep duration | −0.25 (−0.45, −0.04) | 5.345 | 0.021 |

***Note.*** The model includes exposure, length of service, education level, smoking, drinking alcohol, physical exercise, sleep duration, and BMI.

**Table 4.** Combination and interaction of high temperature exposure and BMI on BA acceleration

| Exposure | BMI | *β* (95% *CI*) | *Wald*$\chi^2$ | *P* |
|---|---|---|---|---|
| − | − | Ref | | |
| − | + | 0.64 (0.28, 1.01) | 11.901 | < 0.001 |
| + | − | 0.87 (0.51, 1.23) | 22.354 | < 0.001 |
| + | + | 0.84 (0.45, 1.24) | 17.310 | < 0.001 |
| Exposure*BMI | | −0.67 (−1.24, −0.10) | 5.429 | 0.020 |

***Note.*** Exposure: ” +” represents accelerated aging; ”–” represents no aging acceleration; BMI: ” –” represents normal; ”+” represents overweight; Ref: The reference group; the model was adjusted for length of service, education level, smoking, drinking alcohol, physical exercise, sleep duration.

Our findings also indicate that sleep duration significantly influences liver–kidney BA acceleration, consistent with previous research. Hiromichi Imaizumi et al.[25] followed 1,862 Japanese adults and found that women with sleep durations of less than 6 hours had the highest prevalence of nonalcoholic fatty liver disease, whereas those with 7–8 hours of sleep had the lowest prevalence, indicating that adequate sleep may be a protective factor against liver injury. Numerous studies have also shown that insufficient sleep adversely affects kidney health and increases the risk of chronic kidney disease[26,27]. Consistent with previous findings, smoking and BMI were also significantly associated with accelerated aging. Grace Joshy et al.[28] reported that, compared with non-smokers, tobacco use increased mortality from chronic pulmonary diseases, liver cancer, and renal cancer, with mortality rising proportionally with smoking intensity. These findings underscore the importance of maintaining a healthy lifestyle to delay aging processes.

We also identified an interaction between high-temperature exposure and BMI, whereby both overweight status and high-temperature exposure contributed to accelerated aging. Overweight status often exerts adverse effects on multiple physiological systems, is associated with common metabolic diseases and chronic inflammation, and has been linked to an increased risk of cancer[29,30]. Previous studies have shown that BMI is positively associated with accelerated aging[31]. Obesity has also been demonstrated to share key pathophysiological features with aging, such as telomere shortening and mitochondrial dysfunction[32]. A multi-cohort study further highlighted the role of obesity in promoting cellular senescence and the development of age-related diseases[33]. Under high-temperature exposure, workers may experience reduced physical activity and increased BMI, leading to enhanced secretion of pro-inflammatory factors from adipose tissue. This process can exacerbate inflammatory responses and metabolic dysregulation, forming a “high temperature–inflammation” vicious cycle that accelerates functional decline in organ systems.

Notably, the results also suggested that overweight status attenuated the detrimental effect of high-temperature exposure on accelerated aging. This attenuation may be attributable to greater metabolic reserves in overweight individuals, which may better meet the increased energy demands imposed by heat stress. Previous studies have similarly reported a protective effect of moderate adiposity in mitigating accelerated aging induced by toxicant exposure[4]. Therefore, maintaining an appropriate BMI may help reduce physiological damage associated with high-temperature exposure among workers.

Several limitations of this study should be acknowledged. The cross-sectional design allows for the identification of associations between occupational high-temperature exposure and accelerated aging but does not permit causal inference. In addition, the sample may lack representativeness, as aging assessments were restricted to male workers, which limits the generalizability of the findings and precludes their application to female populations.

In summary, occupational high-temperature exposure may accelerate biological aging in workers, and an interaction exists between occupational high-temperature exposure and BMI in relation to BA acceleration.

*Funding* This work was supported by the Shenzhen Science and Technology program (202208183000115).

*Competing interests* The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

*Ethics* Ethical approval for this study was granted by the Medical Ethics Committee of the Health Science Center of Shenzhen University (Approval No. M202200528). All procedures involving human participants were performed in accordance with the ethical standards of the committee. Informed consent was obtained from all individual participants included in the study.

*Authors’ Contributions* G. and R. Z. searched and archived the literature and wrote the draft. W. W. and J.R. sorted out the data. X. Z. B. Y. and Z. G. revised and reviewed the manuscript. D. H. and W. W. obtained funding and revised versions of the manuscript. W. W. and M. Z. conceived, designed, and supervised this project and submitted the manuscript.

*Data sharing* Due to sensitive reasons such as the privacy of the research subjects, the data of the research results of this study are not publicly available. It should be reasonably requested that they be obtained from the corresponding author. The supplementary materials will be available in www.besjournal.com.

Received: September 23, 2025;
Accepted: December 23, 2025

## REFERENCES

1. Li Z, Zhang ZK, Ren YK, et al. Aging and age-related diseases: from mechanisms to therapeutic strategies. Biogerontology, 2021; 22, 165−87.
2. Sundaresan S, Rajapriya P, Lavanya SK. Aging and cancer: clinical role of tumor markers in the geriatric population (Review). Med Int (Lond), 2024; 4, 21.
3. Wang QY, Hou TY, Wang QW, et al. An evaluation of aging measures: from biomarkers to clocks. Biogerontology, 2023; 24, 303−28.
4. Yang B, Jia YY, Yan MQ, et al. Moderate BMI accumulation modified associations between blood benzene, toluene, ethylbenzene and xylene (BTEX) and phenotypic aging: mediating roles of inflammation and oxidative stress. Environ Pollut, 2024; 360, 124669.
5. Kramna D, Machaczka O, Riedlova P, et al. Exploring the relationship between air pollution and telomere length: baseline findings from a comprehensive ambispective cohort study. Int J Hyg Environ Health, 2025; 267, 114577.
6. Mahmud F, Sarker DB, Jocelyn JA, et al. Molecular and cellular effects of microplastics and nanoplastics: focus on inflammation and senescence. Cells, 2024; 13, 1788.
7. Ebi KL, Capon A, Berry P, et al. Hot weather and heat extremes: health risks. Lancet, 2021; 398, 698−708.
8. Abdelnour SA, Yang CY, Swelum AA, et al. Molecular, functional, and cellular alterations of oocytes and cumulus cells induced by heat stress and shock in animals. Environ Sci Pollut Res, 2020; 27, 38472−90.
9. Maeda T, Guan JZ, Koyanagi M, et al. Altered expression of genes associated with telomere maintenance and cell function of human vascular endothelial cell at elevated temperature. Mol Cell Biochem, 2014; 397, 305−12.
10. Van Dang K, Choi EY, Crimmins E, et al. The joint effects of exposure to ambient long-term air pollution and short-term heat on epigenetic aging in the Health and Retirement Study. J Gerontol A Biol Sci Med Sci, 2025; 80, glaf092.
11. Li MH, Gao CY, Qin L, et al. Construction of biological age model based on multiple routine clinical biomarkers for the Chinese population. J Zhengzhou Univ (Med Sci), 2025; 60, 452−5. (In Chinese)
12. Wu WC, Guo Y, Zhao XK, et al. Correlation analysis on occupational acid fog exposure and accelerated biological aging in workers. J Jilin Univ (Med Ed), 2024; 50, 1741−50. (In Chinese)
13. Gao CY, Li MH, Gu ZG, et al. Progress in biological age research. J Zhengzhou Univ (Med Sci), 2025; 60, 445−51. (In Chinese)
14. Liu ZY. Development and validation of 2 composite aging measures using routine clinical biomarkers in the Chinese population: analyses from 2 prospective cohort studies. J Gerontol A Biol Sci Med Sci, 2021; 76, 1627−32.
15. Gu ZG, Zhang LM, Zhao XK, et al. Accelerated biological aging among chemical plant workers. Environ Res, 2025; 285, 122709.
16. Ni WL, Nikolaou N, Ward-Caviness CK, et al. Associations between medium- and long-term exposure to air temperature and epigenetic age acceleration. Environ Int, 2023; 178, 108109.
17. Bai HK, Kawahara M, Kusama K, et al. Heat stress induces oxidative stress and activates the KEAP1-NFE2L2-ARE pathway in reproduction-related cells. Anim Sci J, 2025; 96, e70023.
18. Cheng CH, Guo ZX, Luo SW, et al. Effects of high temperature on biochemical parameters, oxidative stress, DNA damage and apoptosis of pufferfish (*Takifugu obscurus*). Ecotoxicol Environ Saf, 2018; 150, 190−8.
19. Maeda T, Guan JZ, Koyanagi M, et al. Alterations in the telomere length distribution and the subtelomeric methylation status in human vascular endothelial cells under elevated temperature in culture condition. Aging Clin Exp Res, 2013; 25, 231−8.
20. Yanagisawa K, Miyamoto K, Wakayama Y, et al. Exacerbation of hepatic damage in endothelial aquaporin 1 transgenic mice after experimental heatstroke. Biomedicines, 2024; 12, 2057.
21. Zhang W, Zeng HJ, Xie SY, et al. Activation of autophagy with PF-06409577 alleviates heatstroke-induced organ injury. Environ Int, 2025; 196, 109285.
22. Yan HW, Du JX, Li SJ, et al. Chronic heat stress is capable of reducing the growth performance, causing damage to the liver structure, and altering the liver glucose metabolism and lipid metabolism in largemouth bass (*Micropterus salmoides* L. ). Fish Physiol Biochem, 2025; 51, 24.
23. Sorensen C, Garcia-Trabanino R. A new era of climate medicine-addressing heat-triggered renal disease. N Engl J Med, 2019; 381, 693−6.
24. Roncal-Jimenez C, García-Trabanino R, Barregard L, et al. Heat stress nephropathy from exercise-induced uric acid crystalluria: a perspective on Mesoamerican nephropathy. Am J Kidney Dis, 2016; 67, 20−30.
25. Imaizumi H, Takahashi A, Takahata Y, et al. Association between sleep duration and a new onset of nonalcoholic fatty liver disease. Intern Med, 2024; 63, 3277−82.
26. Sun HM, Qin K, Zou C, et al. The association of nighttime sleep duration and quality with chronic kidney disease in middle-aged and older Chinese: a cohort study. Sleep Med, 2021; 86, 25−31.
27. Zhang Y, Zhong ZZ, Tang ZF, et al. Insomnia and sleep duration for kidney function: Mendelian randomization study. Ren Fail, 2024; 46, 2387430.
28. Joshy G, Soga K, Thurber KA, et al. Relationship of tobacco smoking to cause-specific mortality: contemporary estimates from Australia. BMC Med, 2025; 23, 115.
29. Mol T, Nauwynck E, Donckerwolcke K, et al. Prevalence and determinants of elevated high sensitivity c-reactive protein concentrations in Belgian children and adolescents with overweight or obesity. Obes Sci Pract, 2025; 11, e70063.
30. Renehan AG, Tyson M, Egger M, et al. Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. Lancet, 2008; 371, 569−78.
31. Oblak L, van der Zaag J, Higgins-Chen AT, et al. A systematic review of biological, social and environmental factors associated with epigenetic clock acceleration. Ageing Res Rev, 2021; 69, 101348.
32. Tam BT, Morais JA, Santosa S. Obesity and ageing: two sides of the same coin. Obes Rev, 2020; 21, e12991.
33. Kivimäki M, Frank P, Pentti J, et al. Obesity and risk of diseases associated with hallmarks of cellular ageing: a multicohort study. Lancet Healthy Longev, 2024; 5, e454−63.