

Original Article



Association between Occupational High-Temperature Exposure and the Biological Aging of Workers

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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.

Graphical Abstract available in www.besjournal.com.

Key words: Aging; Occupational exposure; High-temperature; Biological aging

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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 PM_{2.5} 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.

$$n = \frac{2 \times (Z_{\alpha/2} + Z_{\beta})^2 S^2}{d^2} \quad (1)$$

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/m² (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 Klemm-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 (IBM Corp, Armonk, NY, USA) and R software (Version 4.1, R Core Team, Vienna, Austria). 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 interact

Table 1. General data of subjects in two groups

Characteristics	Exposure group (n = 140)	Control group (n = 207)	t/χ^2	P
Age (year)	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 (years)	37.03 ± 7.95	35.82 ± 8.81	-1.295	0.196
Liver - kidney BA acceleration (years)	0.70 ± 1.21	0.05 ± 1.12	-5.219	< 0.001
Cardio - metabolism BA (years)	36.16 ± 8.31	35.70 ± 8.65	-0.498	0.619
Cardio - metabolism BA acceleration (years)	-0.16 ± 0.60	-0.08 ± 0.50	1.327	0.185
Immune BA (years)	36.40 ± 8.10	35.85 ± 8.67	-0.602	0.548
Immune BA acceleration (years)	0.08 ± 0.52	0.07 ± 0.59	-0.219	0.827
Smoking			1.769	0.183
No	60 (42.90)	105 (50.70)		
Yes	80 (57.10)	102 (49.30)		
Drinking alcohol			3.740	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–	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/m²; Non-normally distributed biomarkers (Hs-CRP and TG) were log-transformed; SBP, systolic blood pressure; RBC, red blood cell count; PLT, platelet count; ALB, albumin; UREA, urea; CREA, creatinine; TG, triglycerides; TC, total cholesterol; HbA1c, glycated hemoglobin; Hs-CRP, high-sensitivity C-reactive protein; BA, biological age.

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 χ^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. BMI, body mass index; BA, biological age.

Table 3. Influencing factors of BA acceleration

Outcome	Variables	β (95% CI)	Wald χ^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. BMI, body mass index; BA, biological age.

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

Exposure	BMI	β (95% CI)	Wald χ^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. BMI, body mass index.

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.

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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 The literature was searched and the initial draft was written by Yan Guo and Rui Zhao. The data were curated and organized by Weichao Wu and Jinru Chen. The manuscript was reviewed and revised by Xiangkai Zhao, Bin Yang, and Zhiguang Gu. Funding was obtained and subsequent manuscript versions were refined by Dongsheng Hu and Wei Wang. The project was conceived, designed, and supervised, and the final manuscript was submitted, by Ming Zhang and Wei Wang.

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.

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