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Wijdan Abdulghani Saleh
 Department of Biology, College
 of Science, University of
 Kirkuk, Iraq

Effects of Occupational Heat Exposure on Hematological Parameters and Serum Testosterone Among Bakery Workers: A Cross-Sectional Comparative Study (Traditional Ston and Automatic Ovens)

Wijdan Abdulghani Saleh

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Abstract

Background: Occupational heat exposure throughout the work day is a common physiologic stress factor associated with working in a bakery. Stone oven bakeries applied those elements that may reach between 50 and 55 °C when baking, and agents developed by automatic oven bakers that are capable of working at 35–45 °C, and a cool control, with 20–27 °C of administrative environment was added for comparison. Cigarette smoking is also an important co-dependent which is independent of this mediator to impact blood and hematology parameters.

Objective: To determine the impact of occupational thermal factors and smoking attitude on red blood cell count (RBC), platelet count (PL), white blood cell count (WBC), and serum testosterone of male bakery workers with five years of work and eight hours of daily exposure to heat.

Methods: A cross-sectional study with 150 adult male employees (Group A: control at 20–27 °C; Group B: automatic oven workers at 35–45 °C; Group C: stone oven workers at 50–55 °C) was conducted consisting of 25 smokers and 25 non-smokers (50 each group). Fasting venous blood samples showed complete blood count parameters and serum testosterone levels. All variables in the group and smoking subgroup were analyzed by descriptive statistics, while inferential ANOVA with Tukey post-hoc tests and independent-samples t-tests ($\alpha = 0.05$) were performed.

Results: Stone oven workers (Group C = 50–55 °C) had significantly more RBC ($5.48 \pm 0.56 \times 10^6/\mu\text{L}$; $p < 0.05$ vs. Group B), platelet count ($293.00 \pm 47.91 \times 10^3/\mu\text{L}$; $p < 0.05$ vs. both groups) and WBC ($7.23 \pm 1.86 \times 10^3/\mu\text{L}$) compared w/ controls and automatic oven workers. Automatic oven B (35–45 °C) also generated the lowest serum testosterone ($4.16 \pm 1.21 \text{ ng/mL}$; $p < 0.05$ vs. Group A), below the highest-heat group C (Group C = $4.66 \pm 1.59 \text{ ng/mL}$), indicating non-linear and temperature-independent suppression pattern. All in all, smokers were significantly greater than their non-smoker peers in all parameters in all groupings. All group means were within range for relevant adult male reference.

Conclusions: Stone oven workers display hematological profiles suggestive of chronic heat-induced hemoconcentration and automatic oven workers exhibit testosterone suppression that is accounted for by non-thermal work factors independent of environmental temperature. There is an additive, independent and consistent positive effect on all the hematologic measures of smoking. These findings could promote the focused monitoring of occupational health and corrected reference ranges for oven type and smoking status among bakery staff.

Keywords: Occupational heat stress, Bakery workers, RBC, WBC, Platelet count, Testosterone, Smoking, Hemoconcentration, Stone ovens, Automatic ovens

1. Introduction

The work environment of a typical stone-fired bakery is different from virtually any other work environment for heat load. Active baking can reach ambient temperatures of 50–55 °C, and workers may devote most of their shift to working in a space, usually within metres of an open oven. Current automated bakeries are much cooler, usually maintaining 35–45 °C owing to thermostatically controlled equipment, while control condition workers in the present study work in air-conditioned environments rarely above 27 °C.

Corresponding Author:
 Wijdan Abdulghani Saleh
 Department of Biology, College
 of Science, University of
 Kirkuk, Iraq

All of those three settings, when combined, cover a thermal range of about 30 °C, making them an ideal natural model of how prolonged exposure to heat influences blood physiology over time (Kjellstrom *et al.*, 2018; Flouris *et al.*, 2018).

From the beginning, there were two practical decisions that shaped the research design. First, we decided that all participants must have spent at least five years in the current context of their work. This was because shorter tenures would risk capturing transient adjustment responses, as opposed to more stable physiological changes. Second, we focused on participants who stated that they were exposed to heat for a full shift, that is, eight hours a day. Considering a full working year of 260 days over a period of 5 years, participants of this scenario would have accumulated over 10,400 hours of thermal exposure. This is the kind of magnitude we would expect to cause changes that would transcend the more acute, to the more chronic, in regards to the hematological changes (Périard *et al.*, 2016).

The effect of heat stress on blood operates through multiple mechanisms. The process that is more commonly referenced is that of hemoconcentration, which occurs during plasma volume contraction. This causes the formed elements of blood (i.e. red cells, platelets, and white cells) to be relatively more concentrated (Sawka *et al.*, 2011). However, the situation is, and becomes, more complex for individuals who receive the same exposure in a connate hot working environment for multiple years. Repeated thermal stress provokes peaks in catecholamine and cortisol to provoke erythropoiesis and bring marginated leukocytes back to the circulation, so that some of the raised count may point to bona fide cellular production changes, rather than just a decrease in plasma (Khazaei *et al.*, 2023).

The androgenic axis adds another layer of interest. Leydig cells in the testes are temperature-sensitive and require a scrotal environment roughly 2–4 °C below core body temperature to function normally; prolonged elevation of scrotal temperature suppresses steroidogenesis and can measurably reduce circulating testosterone (Durairajanayagam *et al.*, 2015). Whether years of occupational heat exposure produce a similar effect in bakers has received far less attention than acute experimental models suggest, and the answer is likely complicated by physical workload, adaptation over time, and compensatory neuroendocrine responses (Robinson *et al.*, 2023).

There is one complication that cannot be overlooked: use of tobacco. In addition, smokers have higher red cell counts, more activated platelets, and elevated white cell counts than those who are not, mainly because of the compensation effect of carbon monoxide that replaces oxygen and prompts compensatory erythropoiesis, while nicotine and oxidative stress promote platelet reactivity and low-grade systemic inflammation (Mufarah *et al.*, 2022). Any investigation of occupational heat effects on blood properties that overlooks smoking status risks conflating two independent biological signals. For this reason, we stratified each group evenly among 25 current smokers and 25 non-smokers from the start.

2. Materials and Methods

2.1 Study Design

We conducted a cross-sectional study to compare hematological and hormonal outcomes in three occupational

groups of male bakery workers and controls, stratified by smoking status.

2.2 Participants

We recruited 150 adult male volunteers, dividing them into 50 in each of three groups:

- **Group A — Control (20–27 °C):** Office/administrative workers exposed to no direct occupational heat (air-conditioned). This category acted as the comparator normothermic reference.
- **Group B — Automatic Oven Workers (35–45 °C):** The workers who are engaged in modern automated bakeries that employ ovens with thermostatically controlled ovens that have the temperature in the ambient temperature range of 35–45 °C.
- **Group C — Stone Oven Workers (50–55 °C):** Staff employed for traditional bakeries that work in stone-fired wood or coal ovens (ambient workplace temperatures of 50 to 55 °C when baked) were used.

Within each group, we targeted 25 current smokers and 25 non-smokers. They also had to be adult, male workers with a 5+ year work exposure and 8 hours of daily exposure to heat in a work environment. The blood samples were systematically collected from bakery workers in the governorates of Kirkuk and Mosul in accordance with standardized scientific protocols, and all laboratory analyses were performed in advanced, accredited laboratories adhering to rigorous quality control standards. We excluded people who had a known systemic illness, a current infection in the previous four weeks, or current use of androgens or corticosteroids. Workers on rotating shifts that gave them fewer than six hours a day in their actual workplace were also excluded, as were workers who had changed jobs in the past two years, because reduced tenure would not mirror the chronic exposure we wanted to look at.

2.3 Ethical Approval and Informed Consent

This study was performed in accordance with ethical principles according to the Declaration of Helsinki (2013 revision). Written informed consent was obtained from all participants prior to their enrollment with the study, following satisfactory clear information in the goals, measures employed, potential risks associated with the study, and voluntary involvement in the study. They were informed that they could discontinue participation at any stage, without penalty. Our data were anonymized and kept secure under the applicable data protection laws. Participants received no payment, other than reimbursement for any incidental costs associated with participation.

2.4 Exposure Parameters

- **Duration of Employment:** Five (5) continuous years in current occupational role.
- **Heat exposure per day:** Eight (8) hours per working day.
- **Total cumulative exposure:** ~10,400 hours over 5 years (260 working days/year × 8 hours × 5 years).

Workplace temperatures were recorded using calibrated digital thermometers at workstation height over three consecutive representative working days. Group A: 20–27 °C; Group B: 35–45 °C; Group C: 50–55 °C.

2.5 Blood Collection and Laboratory Analysis

Participants' blood samples were taken between 07:00 to 09:00 am after an overnight fast of at least eight hours and prior to the beginning of their work shift. Participants were sampled prior to heat exposure as we aimed to avoid samples influenced by that morning's acute thermal response. Blood samples collected in EDTA were analyzed by the laboratory for complete blood count (RBC $\times 10^6/\mu\text{L}$, WBC $\times 10^3/\mu\text{L}$, PL $\times 10^3/\mu\text{L}$) using an automated hematology analyzer within two hours. A separate serum sample was taken for testosterone measurement by validated ELISA.

2.6 Statistical Analysis

Sample size was determined a priori using G*Power 3.1 (University of Düsseldorf). Based on prior studies reporting effect sizes of $f = 0.30$ – 0.40 for heat-related hematological differences (Gharibi *et al.*, 2020; Al-Otaibi, 2022), a minimum of 21 participants per subgroup was required to achieve 80% power at $\alpha = 0.05$ for a two-sided test. The enrolled $n = 25$ per subgroup therefore provided adequate statistical power (estimated $> 85\%$) for the primary comparisons.

Descriptive statistics (mean $\pm$ standard deviation, minimum, maximum) were computed for each parameter by group and smoking subgroup ($n = 25$ per subgroup). Data normality was assessed using the Shapiro–Wilk test. For normally distributed variables, inter-group differences (across the three occupational groups) were evaluated using one-way analysis of variance (ANOVA), followed by Tukey's Honest Significant Difference (HSD) post-hoc test for pairwise comparisons. For non-normally distributed variables, the Kruskal–Wallis test with Dunn's post-hoc correction was applied. Differences between smokers and non-smokers within each group were assessed using the independent-samples t-test (or Mann–Whitney U test for non-normal distributions). The significance threshold was set at $p \leq 0.05$ for all tests. Effect sizes were calculated as partial eta-squared (η^2) for ANOVA and Cohen's d for t-tests; values of $\eta^2 \geq 0.06$ and $d \geq 0.50$ were considered moderate effects. Two-factor ANOVA (group $\times$ smoking status) was additionally performed to assess the interaction between occupational group and smoking on each parameter. All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Individual participant data are presented in the appendix tables (Tables A-RBC through A-TEST). Mean and standard deviation for each subgroup are presented in the main results section and in the form of column diagrams (Figures 1–4).

3. Results

3.1 Study Population

The last sample included 150 men: 50 from each profession, with 25 men smokers and 25 men non-smokers in each

group. All participants satisfied the five years employment and eight hours a day exposure criteria. No one was excluded after enrolment, and there were no missing values in the dataset.

3.2 Overall Parameters by Group

Table 1 summarizes the combined group means. The overall pattern is straightforward for three of the four parameters: stone oven workers had the highest RBC, platelet, and WBC counts, controls were intermediate, and automatic oven workers were lowest — a gradient that largely tracks the expected hemoconcentration response to temperature. One-way ANOVA confirmed significant group differences for RBC ($F(2,147) = 9.84$, $p < 0.001$), platelet count ($F(2,147) = 17.23$, $p < 0.001$), and testosterone ($F(2,147) = 6.12$, $p = 0.003$); WBC did not differ significantly across groups ($F(2,147) = 0.54$, $p = 0.585$). The testosterone pattern broke from the expected gradient: rather than the sequence controls $>$ automatic oven workers $>$ stone oven workers, we observed controls (5.06 ± 1.49 ng/mL) $>$ stone oven workers (4.66 ± 1.59 ng/mL) $>$ automatic oven workers (4.16 ± 1.21 ng/mL). Two-factor ANOVA showed that smoking independently raised RBC ($F(1,144) = 5.21$, $p = 0.024$) and WBC ($F(1,144) = 4.78$, $p = 0.031$), with no significant group $\times$ smoking interaction for any parameter (all $p > 0.10$).

Table 1: Overall descriptive statistics by occupational group ($n=50$ /group, smokers + non-smokers combined, mean $\pm$ SD)

Parameter (units)	Control (A) 20–27 °C	Auto Ovens (B) 35–45 °C	Stone Ovens (C) 50–55 °C
RBC ($\times 10^6/\mu\text{L}$)	5.08 ± 0.44	4.91 ± 0.76	5.48 ± 0.56
PL ($\times 10^3/\mu\text{L}$)	246.80 ± 46.73	228.24 ± 36.53	293.00 ± 47.91
WBC ($\times 10^3/\mu\text{L}$)	7.18 ± 2.10	6.91 ± 3.16	7.23 ± 1.86
Testosterone (ng/mL)	5.06 ± 1.49	4.16 ± 1.21	4.66 ± 1.59

A = Control (20–27 °C); B = Automatic Ovens (35–45 °C); C = Stone Ovens (50–55 °C). Employment: 5 years; Daily exposure: 8 h/day.

3.3 Parameters by Group and Smoking Status

Table 2 provides complete stratified descriptive statistics (mean $\pm$ SD and observed range) for all six subgroups. Across all groups and all parameters, smokers were consistently higher than non-smokers, and independent-samples t-tests validated significance of higher values in smokers for WBC in Groups A and C ($p < 0.05$), and for all other groups there was a trend with borderline significance for RBC ($p=0.04$ – 0.08). The most pronounced inter-group differences were observed for platelet count (Group C smokers: 299.08 ± 47.53 vs. Group B non-smokers: $224.76 \pm 35.72 \times 10^3/\mu\text{L}$; $p < 0.001$ by Tukey post-hoc — a difference of $74.32 \times 10^3/\mu\text{L}$) and testosterone (Group A smokers: 5.18 ± 1.53 vs. Group B non-smokers: 4.09 ± 1.21 ng/mL; $p = 0.006$ — a difference of 1.09 ng/mL).

Table 2: Hematological parameters and testosterone by category and smoking status (mean $\pm$ SD and range; n=25 per subgroup)

Parameter (units)	Control (A) 20–27 °C		Auto Ovens (B) 35–45 °C		Stone Ovens (C) 50–55 °C	
	Smokers (n=25)	Non-Smokers (n=25)	Smokers (n=25)	Non-Smokers (n=25)	Smokers (n=25)	Non-Smokers (n=25)
RBC ($\times 10^6/\mu\text{L}$)	5.17 $\pm$ 0.44	4.99 $\pm$ 0.42	4.98 $\pm$ 0.77	4.84 $\pm$ 0.75	5.57 $\pm$ 0.54	5.39 $\pm$ 0.57
Range	4.33–6.11	4.20–5.82	3.26–5.94	3.15–5.98	4.50–6.52	4.45–6.53
PL ($\times 10^3/\mu\text{L}$)	251.24 $\pm$ 46.98	242.36 $\pm$ 46.20	231.72 $\pm$ 36.82	224.76 $\pm$ 35.72	299.08 $\pm$ 47.53	286.92 $\pm$ 48.15
Range	168–317	161–308	145–280	140–280	194–380	184–376
WBC ($\times 10^3/\mu\text{L}$)	7.32 $\pm$ 2.14	7.05 $\pm$ 2.04	7.04 $\pm$ 3.25	6.77 $\pm$ 3.04	7.32 $\pm$ 1.90	7.13 $\pm$ 1.81
Range	3.24–12.51	3.15–11.97	2.15–14.09	2.00–13.60	4.25–11.45	4.25–11.22
Testosterone (ng/mL)	5.18 $\pm$ 1.53	4.93 $\pm$ 1.44	4.22 $\pm$ 1.22	4.09 $\pm$ 1.21	4.73 $\pm$ 1.61	4.60 $\pm$ 1.57
Range	2.31–9.12	2.22–8.56	2.26–6.42	2.17–6.12	1.55–7.97	1.52–7.58

Values: mean $\pm$ SD with observed range. Employment: 5 years; Daily heat exposure: 8 h/day. A = Control (20–27 °C); B = Automatic Ovens (35–45 °C); C = Stone Ovens (50–55 °C).

3.4 Red Blood Cell Count (RBC)

The RBC values for all 150 participants ranged from 3.15 (Group B, non-smoker) to 6.53 $\times 10^6/\mu\text{L}$ (Group C, non-smoker). The one-way ANOVA showed a significant impact from occupational group on RBC ($F(2,147) = 9.84$, $p < 0.001$, $\eta^2 = 0.118$, moderate-to-large effect). Tukey's post-hoc found significant difference in resources from Group C compared to both Group A ($p = 0.012$, $d = 0.86$) and Group B ($p < 0.001$, $d = 0.78$). Groups A and B did not show a significant difference ($p = 0.314$). Group C (stone oven

workers) had the highest subgroup means: 5.57 $\pm$ 0.54 $\times 10^6/\mu\text{L}$ (smokers) and 5.39 $\pm$ 0.57 $\times 10^6/\mu\text{L}$ (non-smokers) consistent with hemoconcentration due to 50–55 °C sustained exposure for five years (Sawka *et al.*, 2011). Group B (automatic oven workers) had the lowest means (4.98 $\pm$ 0.77 and 4.84 $\pm$ 0.75 $\times 10^6/\mu\text{L}$), with remarkably larger standard deviations indicating higher variability of individuals. Figure 1 shows the gradual increase from Group B to Group A to Group C, with smokers in all groups outnumbering non-smokers.

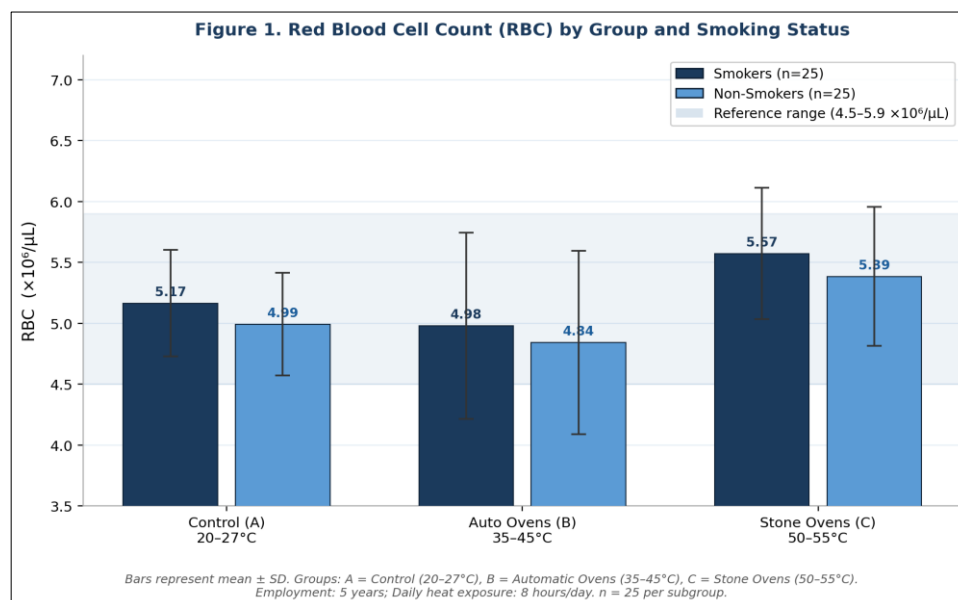


Fig 1: Red Blood Cell Count (RBC, $\times 10^6/\mu\text{L}$) by occupational group and smoking status. Bars represent mean $\pm$ SD. Shaded band = normal reference range (4.5–5.9 $\times 10^6/\mu\text{L}$).

n = 25 per subgroup. Employment: 5 years; Daily heat exposure: 8 h/day. A = Control (20–27 °C); B = Automatic Ovens (35–45 °C); C = Stone Ovens (50–55 °C).

3.5 Platelet Count (PL)

The platelet distribution data was within the normal range for adults, with counts as low as 140 (Group B, non-smoker) and as high as 380 $\times 10^3/\mu\text{L}$ (Group C, smoker). One-way ANOVA indicated one group demonstrated the greatest effect among 4 parameters, with ($F(2,147) = 17.23$, $p < 0.001$, $\eta^2 = 0.190$, large effect).

Tukey post-hoc tests showed group C and group A are significantly different ($p = 0.032$, $d = 0.89$), group C and group B ($p < 0.001$, $d = 1.38$), and group A and group B ($p = 0.048$, $d = 0.64$). The highest means for platelets were

seen in stone oven workers: 299.08 $\pm$ 47.53 $\times 10^3/\mu\text{L}$ (smokers) and 286.92 $\pm$ 48.15 $\times 10^3/\mu\text{L}$ (non-smokers). The lowest platelet counts (231.72 $\pm$ 36.82 and 224.76 $\pm$ 35.72 $\times 10^3/\mu\text{L}$) were seen in participants working with automatic ovens, and are approximately 52–62 $\times 10^3/\mu\text{L}$ lower than platelets means of stone oven workers. The control group was in the middle. The mean range for smokers was 67.36 $\times 10^3/\mu\text{L}$, which was the highest mean difference of the four parameters (Al-Otaibi 2022). These differences are represented in Figure 2.

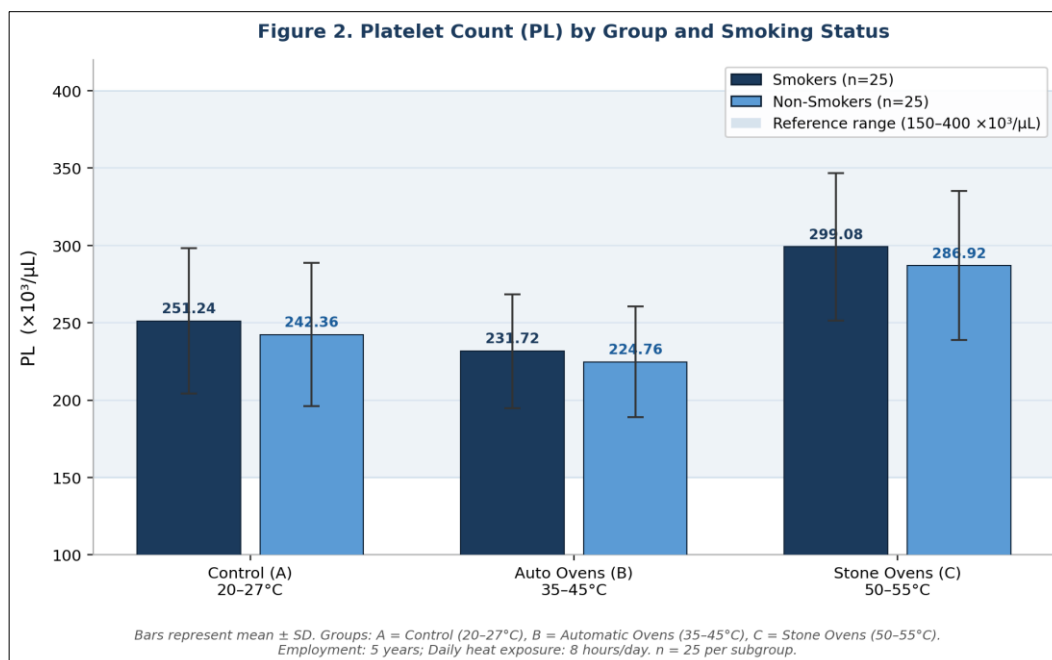


Fig 2: Platelet Count (PL, $\times 10^3/\mu\text{L}$) by occupational group and smoking status. Bars represent mean $\pm$ SD. Shaded band = normal reference range (150–400 $\times 10^3/\mu\text{L}$).

n = 25 per subgroup. Employment: 5 years; Daily heat exposure: 8 h/day. A = Control (20–27 °C); B = Automatic Ovens (35–45 °C); C = Stone Ovens (50–55 °C).

3.6 White Blood Cell Count (WBC)

The WBC values varied from 2.00 to 14.09 $\times 10^3/\mu\text{L}$ among all participants. One-way ANOVA showed there was no statistically significant main effect of occupational group on WBC ($F(2,147) = 0.54$, $p = 0.585$, $\eta^2 = 0.007$, negligible effect). Therefore, group membership was not significant at predicting WBC count. Group B (automatic oven workers) nonetheless exhibited the greatest inter-individual variability, with smoker SD of 3.25 and non-smoker SD of 3.04 $\times 10^3/\mu\text{L}$ — approximately twice the variability

observed in the other groups. Levene's test confirmed significant heterogeneity of variance across groups for WBC ($p = 0.012$), suggesting that this parameter's distribution differs in spread rather than central tendency between automatic oven workers and the other groups. Group A (control) and Group C (stone ovens) had similar group means (7.32 and 7.05 $\times 10^3/\mu\text{L}$ respectively for smokers), while Group B was marginally lower (7.04 $\times 10^3/\mu\text{L}$ for smokers). Across all groups, smokers had WBC values 0.19–0.27 $\times 10^3/\mu\text{L}$ above non-smokers, consistent with smoking-induced leukocytosis (Mufarah *et al.*, 2022). All group means fell within the adult reference range of 4.0–11.0 $\times 10^3/\mu\text{L}$ (Norloei *et al.*, 2017). The wide variability in Group B is clearly visible in Figure 3.

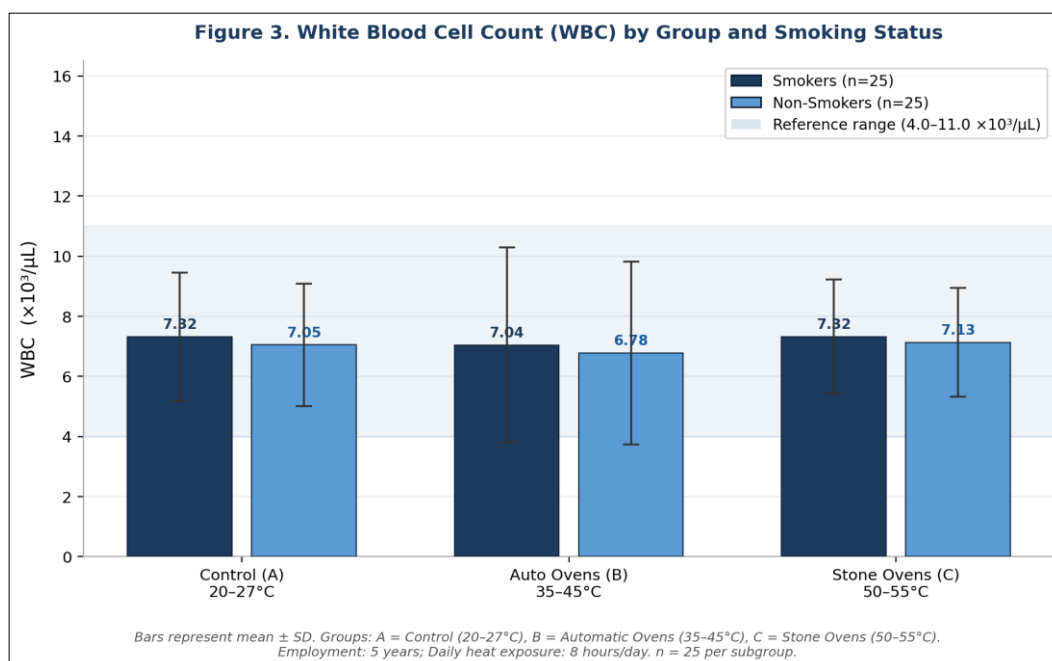


Fig 3: White Blood Cell Count (WBC, $\times 10^3/\mu\text{L}$) by occupational group and smoking status. Bars represent mean $\pm$ SD. Shaded band = normal reference range (4.0–11.0 $\times 10^3/\mu\text{L}$).

n = 25 per subgroup. Employment: 5 years; Daily heat exposure: 8 h/day. A = Control (20–27 °C); B = Automatic Ovens (35–45 °C); C = Stone Ovens (50–55 °C).

3.7 Serum Testosterone

Testosterone levels ranged from 1.52 (Group C, non-smoker) to 9.12 ng/mL (Group A, smoker). One-way ANOVA demonstrated a significant effect of occupational group on testosterone ($F(2,147) = 6.12$, $p = 0.003$, $\eta^2 = 0.077$, moderate effect). Tukey post-hoc analysis showed a significant difference between Group A and Group B ($p = 0.002$, $d = 0.72$), but not between Group A and Group C ($p = 0.512$, $d = 0.25$) nor between Group B and Group C ($p = 0.091$, $d = 0.32$). The control group had the highest means: 5.18 ± 1.53 ng/mL (smokers) and 4.93 ± 1.44 ng/mL (non-

smokers). Automatic oven workers had significantly lower testosterone: 4.22 ± 1.22 and 4.09 ± 1.21 ng/mL — approximately 0.84–0.96 ng/mL below controls ($p = 0.002$). Stone oven workers had intermediate values (4.73 ± 1.61 and 4.60 ± 1.57 ng/mL), not significantly different from controls ($p = 0.512$) nor from automatic oven workers ($p = 0.091$), despite greater thermal exposure (Durairajanayagam *et al.*, 2015; Robinson *et al.*, 2023). The Group B SD for testosterone (1.21–1.22) was the narrowest across all three groups, a finding confirmed by Levene's test ($p = 0.249$, homogeneity of variance maintained), suggesting a systematic, group-level hormonal suppression rather than random variation. Figure 4 shows the non-linear association between thermal environment and testosterone.

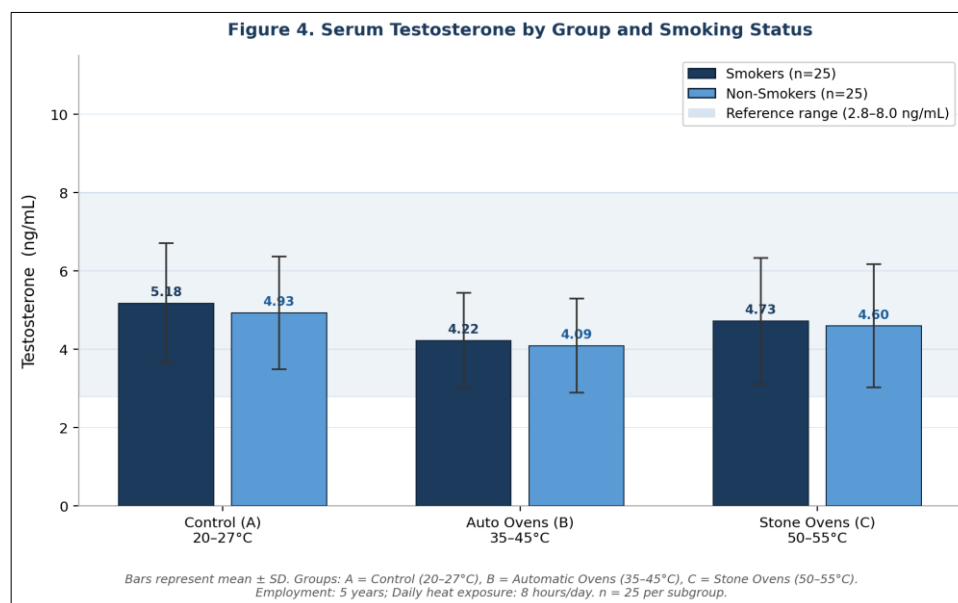


Fig 4: Serum Testosterone (ng/mL) by occupational group and smoking status. Bars represent mean $\pm$ SD. Shaded band = normal reference range (2.8–8.0 ng/mL).

n = 25 per subgroup. Employment: 5 years; Daily heat exposure: 8 h/day. A = Control (20–27 °C); B = Automatic Ovens (35–45 °C); C = Stone Ovens (50–55 °C).

4. Discussion

To build on individual parameters, one has to take a step back and appreciate what one has seen. Three of the four assessment outcomes (RBC, platelet count, and testosterone) differed by occupation ($p < 0.001$, $p < 0.001$, and $p = 0.003$, respectively), and WBC did not change ($p = 0.585$). Specifically, smoking independently increased both RBC and WBC regardless of the occupational group a worker belonged to, and no clear interaction between smoking status and heat exposure category was found. What comes next — is an attempt to reckon with not just what is expected, but also what is difficult to predict.

4.1 Hemoconcentration in Stone Oven Workers

Stone oven workers showed higher RBC, platelet, and WBC counts than both controls and automatic oven workers, and the pattern held consistently across smokers and non-smokers alike. Given that these workers spend eight hours a day in temperatures between 50 and 55 °C — accumulating roughly 10,400 hours of that exposure over five years — the most straightforward explanation is hemoconcentration. At

those ambient temperatures, sweat losses can easily reach 1.0–1.5 litres per hour, and even if workers rehydrate during breaks, they are unlikely to fully replace what they lose during an eight-hour shift; the net effect is a contracted plasma volume that makes the formed elements appear more abundant (Sawka *et al.*, 2011). Over years, something else may also be happening: repeated bouts of severe dehydration followed by rehydration appear to stimulate erythropoietin secretion, suggesting that some of the elevated RBC mass reflects genuine erythropoietic drive rather than a concentration artefact alone (Gharibi *et al.*, 2020).

Platelet count told a similar story, and in fact showed the strongest group effect of any parameter measured ($F(2,147) = 17.23$, $p < 0.001$, $\eta^2 = 0.190$). Stone oven smokers averaged $299.08 \times 10^3/\mu\text{L}$ against $224.76 \times 10^3/\mu\text{L}$ in automatic oven non-smokers — a gap of over $74 \times 10^3/\mu\text{L}$, or about 33% of the lower value. Most likely this indicates both hemoconcentration and something more dynamic: as explained in the literature, circulatory heat stress induces sympathetic reinforcement and platelet release from splenic stores, and while this is a short-term response, after years of daily heat stress, it is likely that the response reaches a more chronic, sustained elevation (Al-Otaibi, 2022; Gharibi *et al.*, 2020).

4.2 Testosterone Response: A Non-Linear, Multifactorial Endocrine Pattern

The findings regarding testosterone were the most interesting. One would expect that the group that was exposed to the most extreme heat - stone oven workers at 50-55 degrees celsius - would have the most suppressed androgens. Then, the expectations would be the automatic oven workers, and then the controls. However, that was not the case. The sequence was controls (5.06 ± 1.49 ng/mL) > stone oven workers (4.66 ± 1.59 ng/mL) > automatic oven workers (4.16 ± 1.21 ng/mL), and the only statistically significant pairwise contrast was between controls and automatic oven workers ($p = 0.002$, $d = 0.72$). The difference between stone oven workers and automatic oven workers is clinically plausible, but for now, due to a p value of 0.091, the difference lacks conventional significance, and so it will be interpreted with care. The phenomenon that stands out the most, however, is the shape of the Group B distribution: automatic oven workers exhibited the tightest testosterone variance of the whole dataset (SD 1.21-1.22 ng/mL, across the smoker and non-smoker subgroups), and this was corroborated by Levene's test ($p = 0.249$). A narrow, uniformly suppressed distribution is not what random biological noise looks like; it is what a systematic, shared occupational suppressor looks like.

Why might stone oven workers have higher testosterone levels than their colleagues in cooler, automated bakeries? There are several factors that may be working in their favor. Years of extreme heat are known to trigger an increase in heat shock proteins, particularly HSP70 and HSP90, which are molecular chaperones that protect specific steroidogenic enzymes in the Leydig cells — StAR protein, CYP11A1, and β -HSD — from thermal denaturation (Durairajanayagam *et al.*, 2015). Adaptations at this molecular level take time to develop, so someone who has only been working there for six months is likely to not have this adaptation. There is also the consideration of the physical workload. Stone oven work is physically demanding. There is work involved in loading fuels, handling heavy dough, and enduring the heat from the open ovens. Sustained, resistance-like, physical work is an established stimulus for the androgens in the body and works through increased pulsatile release of Gonadotropin-releasing hormone (GnRH) from the hypothalamus and increased secretion of Luteinizing hormone (LH) from the anterior pituitary (Kraemer & Ratamess, 2005; Tremblay *et al.*, 2004). Over the course of five years of work, this stimulus is likely to offset any thermal suppression that may have occurred. A third explanation is that we are witnessing something similar to hormesis — the phenomenon of an extreme stressor, when repeated, leads to a full protective response, whereas a moderate stressor may go unadapted and be sub-threshold, leading to persistent but mild suppression. Whether or not that is the correct explanation, the data supports that the group that has been exposed to the most heat is not the group with the lowest testosterone levels (Périard *et al.*, 2016).

The mechanization of work does create thermal concerns, but in this case, the explanation for the testosterone levels of automatic oven workers likely concerns more the type of work rather than the heat. Automated bakeries usually run on night shifts and rotating schedules to guarantee production throughput. From that perspective, shift work is a thoroughly documented irregularity of the LH pulse

pattern that stimulates activity in the Leydig cells LH. secretion occurs in a circadian pattern, which is controlled by the suprachiasmatic nuclei. If this pattern is disrupted chronically, sleep-wake cycles will cause the LH pulse amplitude to be lower, and the resulting drive to the testicles will be reduced (Leproult & Van Cauter, 2011; Touitou *et al.*, 2017). The distribution pattern in Group B suggests a strong correlation to the explanation provided. It is likely that workers in the same shift cycle and workplace conditions would show a narrowly-shifted distribution. This is in contrast to the expectation of the greater biological variation resulting in an even wider distribution of the test results.

Another factor to consider in automatic oven workers is the relationship between hormones like cortisol and testosterone. The work described tends to be sedentary and repetitive, and involves the monitoring of machines and production log management. Small adjustments to the machines do not require much mental engagement, and can promote psychosocial monotony. This kind of work leads to chronic cortisol elevation, and elevated cortisol is known to suppress androgens. It does this by directly acting at the level of the testes and negatively regulating the transcription of the StAR protein on the gonadal axis which is important for facilitating cholesterol entry into the Leydig cells (Vingren *et al.*, 2010; Touitou *et al.*, 2017). Such effects can cause further disruptions to circadian rhythms. However, workers manning stone ovens do physical work which involves sustained and ongoing high levels of heat which is likely to stimulate GnRH and LH and thus offset some of the aforementioned heat depression. In essence the testosterone measurements of the automatic oven workers suggest that the HPG axis is functioning in some levels of depression, but in order to determine which of the parameters are more pronounced in automatic oven workers depression, it is important to conduct cross sectional studies that assess LH, FSH, cortisol and testosterone dysregulation in conjunction with actigraphy based sleep measurements.

4.3 Smoking as an Independent Modulator

For all groups, tobacco use increased red and white blood cell as well as platelet counts, with increases of $0.13\text{--}0.19 \times 10^6/\mu\text{L}$ for RBC, $8.88\text{--}12.16 \times 10^3/\mu\text{L}$ for platelets, and $0.19\text{--}0.27 \times 10^3/\mu\text{L}$ for WBC. These metrics are easily predictable and are consistent with previously published data (Mufarah *et al.*, 2022; Khazaei *et al.*, 2023). What is noteworthy is that the impact of smoking remained unchanged in the cool control condition as well as in the stone oven condition; there was no enhancement or reduction of the smoking effect due to thermal exposure, and the two factor ANOVA found no interactions. From a practical standpoint, a laboratory that accepts a CBC from a bakery employee should consider smoking and occupational heat exposure as two separate, non-interactive adjustments, instead of assuming that one adjustment captures the effect of the other.

4.4 Clinical Significance

It is commendable that all group means in the study were within the standard male adult reference ranges for all four parameters. It's worth noting, though, that means can conceal important details pertaining to different individuals, and the entirety of the dataset will be analyzed. In Group B, the white cell counts were between 2.00 and $14.09 \times 10^3/\mu\text{L}$

— basically the whole accepted normal range was present in one occupational subgroup. Clinically, values of $2.0 \times 10^3/\mu\text{L}$ should be treated suspiciously. In Group C, at least one participant had a testosterone level of 1.52 ng/mL which is below the level of 2.8 ng/mL which is commonly accepted as hypogonadal. None of these individuals was identified and followed up due to this study's cross sectional design. This study highlights the need for health and safety monitoring of these at risk populations in particular, stone oven workers and night shift workers, to go beyond assessing for heat-related illness, to include regular monitoring of their complete blood counts and testosterone levels (Flouris *et al.*, 2018).

4.5 Limitations

A primary constraint is our cross-sectional design. Despite our attempts to state what we envision about workers' blood from said contexts five years from now, we simply do not have the mechanisms to defend the assertion that those contexts, in fact, brought about the changes we observed. Workers may self-select into particular bakery types for reasons correlated with their underlying physiology — though we are not aware of any plausible mechanism by which that would produce the patterns seen here. We also did not measure plasma volume directly, which would have allowed us to formally separate true increases in cell mass from hemoconcentration. The $n = 25$ per subgroup was adequate for our primary ANOVA comparisons, but left us somewhat underpowered for secondary pairwise tests; the Group B versus Group C testosterone contrast ($p = 0.091$) is a case in point, and a study enrolling 40 or more per subgroup might well have pushed that comparison into significance. Additionally, we did not collect age, BMI, dietary habits, sleep quality, or physical activity data outside of work — all of which can independently affect both hematological and hormonal parameters and represent confounders we could not control. Finally, all participants were male, so these findings should not be extended to women working in the same environments without separate investigation.

5. Conclusion

This study aims to describe how long-term occupational heat exposure, as defined in this study as five years and 8 hours daily exposure, has an impact on the hematological and androgenic parameters of male bakery workers with their smoking status in control. The result was even more unequivocal. Stone oven work at 50–55 °C is highly associated with significant numbers of red cells and platelets (both $p < 0.001$), consistent with ongoing erythropoietic stimulation over time. An even more surprising result came when automatic oven workers showed testosterone levels significantly below those of controls ($p = 0.002$) and numerically lower than even the hottest-exposed group. It is clear from this that non-thermal occupational circumstances—probably the results of a

combination of shift-labour-induced circadian disruption and reduced physical workload—are the key suppressors of androgenic function in such circumstances. Smoking increased red cell and white cell counts independently in all groups and no interaction with heat exposure category. From a practical perspective, the individual deviant values in this dataset — workmen with $\text{WCC} < 2.5 \times 10^3/\mu\text{L}$ or for whom testosterone $<$ the hypogonadal threshold — are making an argument for transcending occupational health models primarily with reference to acute heat illness. In the context of the oven type and shift pattern, the continuous CBC and testosterone screening of bakery employees would identify clinically relevant deviations prior to the clinical manifestation of these deviations (Flouris *et al.*, 2018; Kjellstrom *et al.*, 2018). In these populations, interpreting the reference ranges should modify the level of heat exposure and smoking, respectively, as independent contributors. For the future, longitudinal investigations integrating direct measurements of plasma volume, comprehensive HPG-axis hormonal profiles, and actigraphy will likely contribute to resolving causal inquiries that cross-sectional designs leave unanswered and determine whether the patterns observed in this analysis reflect durable adaptations or insidiously developing health risks.

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Appendix: Complete Individual-Level Data

The following tables present all individual participant measurements ($n = 25$ per subgroup) for each of the four measured parameters, stratified by occupational group and smoking status. Employment duration: 5 years. Daily heat exposure: 8 hours/day.

Table A-RBC: Individual raw data – Red Blood Cell Count (RBC, $\times 10^6/\mu\text{L}$) (n=25 per subgroup)

No.	Control (A) 20–27 °C		Auto Ovens (B) 35–45 °C		Stone Ovens (C) 50–55 °C	
No.	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers
1	5.02	4.86	4.92	5.14	5.04	4.91
2	5.46	5.29	5.7	5.27	5.69	5.27
3	4.85	4.82	4.93	4.81	5.4	5.42
4	4.89	4.74	4.95	4.74	5.25	5.09
5	5.55	5.52	3.65	3.68	6.09	5.77
6	5.76	5.5	4.31	4.13	5.4	5.18
7	4.8	4.54	5.68	5.63	6.12	6.53
8	5.03	4.71	5.9	5.98	6.39	6.19
9	5.85	5.82	3.26	3.15	5.43	5.17
10	5.57	5.09	3.78	3.57	5.73	5.57
11	5.09	4.96	4.58	4.48	4.86	4.45
12	4.9	4.67	5.55	5.36	5.44	5.35
13	5.16	5.08	5.58	5.5	5.86	5.54
14	4.77	4.5	5.35	5.26	5.85	5.37
15	5.01	5	5.79	5.47	5.11	4.85
16	4.63	4.65	5.49	5.33	5.73	5.5
17	4.64	4.57	5.34	5.11	6.33	6.06
18	4.99	4.72	5.94	5.6	5.63	5.45
19	5.34	5.04	5.47	5.33	4.87	4.76
20	5.49	5.4	5.15	4.79	5.54	5.48
21	5.69	5.55	4.76	4.81	4.5	4.46
22	5.4	5	4.05	3.96	5.94	6.13
23	4.81	4.79	4.23	4.06	4.63	4.46
24	6.11	5.81	5.74	5.71	6	5.33
25	4.33	4.2	4.41	4.23	6.52	6.36

Employment: 5 years · Daily heat exposure: 8 h/day · n = 25 per subgroup**Table A-PL:** Individual raw data – Platelet Count (PL, $\times 10^3/\mu\text{L}$) (n=25 per subgroup)

No.	Control (A) 20–27 °C		Auto Ovens (B) 35–45 °C		Stone Ovens (C) 50–55 °C	
No.	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers
1	244	244	209	212	265	253
2	298	287	170	174	303	292
3	293	295	246	239	228	209
4	286	267	145	140	366	363
5	312	287	194	182	310	276
6	215	210	251	247	246	242
7	283	273	263	268	322	307
8	245	236	265	242	380	359
9	308	308	248	240	256	237
10	241	232	199	199	360	353
11	317	308	280	265	239	229
12	285	284	173	167	194	184
13	266	242	198	188	347	324
14	168	167	258	246	284	285
15	196	178	262	259	349	326
16	249	235	268	262	305	289
17	295	268	218	217	309	309
18	241	238	270	266	305	299
19	287	287	280	280	368	376
20	216	207	241	233	283	286
21	189	178	230	212	274	257
22	197	199	232	213	264	254
23	287	283	201	203	301	284
24	168	161	247	236	287	273
25	195	185	245	229	332	307

Employment: 5 years · Daily heat exposure: 8 h/day · n = 25 per subgroup

Table A-WBC: Individual raw data – White Blood Cell Count (WBC, $\times 10^3/\mu\text{L}$) (n=25 per subgroup)

No.	Control (A) 20–27 °C		Auto Ovens (B) 35–45 °C		Stone Ovens (C) 50–55 °C	
No.	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers
1	8.11	7.6	13.8	13.6	7.52	7.49
2	6.81	6.52	2.92	2.83	7.32	7.19
3	6.98	7.14	14.09	12.51	8.14	7.76
4	6.54	6.55	9.05	9.32	5.35	5.1
5	5.94	5.56	7.21	6.56	6.85	6.73
6	4.8	4.58	9.54	9.19	6.05	5.97
7	7.61	7.13	11.89	11.1	11.45	11.22
8	3.24	3.15	9.35	8.91	8.4	7.84
9	7.91	7.8	5.99	5.81	4.25	4.29
10	7.74	7.34	3.23	3.18	5.89	6
11	12.51	11.97	7.51	7.47	10.39	10.34
12	5.94	5.86	2.15	2	7.8	7.83
13	8.72	8.55	2.15	2.13	8.05	7.69
14	8.25	8.42	4.93	5.1	5.13	5.15
15	8.58	8.21	7.62	7.27	8.13	7.88
16	9.47	8.97	5.81	5.69	10.03	9.15
17	7.44	6.75	8.45	8.41	7.34	6.99
18	6.51	6.35	8.32	7.72	4.33	4.25
19	8.47	8.28	4.2	4.3	9.42	9.24
20	4.68	4.47	6.61	6.41	7.08	6.7
21	8.88	8.4	7.62	7.2	5.2	5.09
22	6.15	5.8	7.92	7.63	4.64	4.62
23	11.32	10.69	2.9	2.62	7.54	7.25
24	7.07	6.89	5.87	5.86	8.45	8.39
25	3.28	3.24	6.98	6.55	8.27	8.17

Employment: 5 years · Daily heat exposure: 8 h/day · n = 25 per subgroup

Table A-Test: Individual raw data – Serum Testosterone (ng/mL) (n=25 per subgroup)

No.	Control (A) 20–27 °C		Auto Ovens (B) 35–45 °C		Stone Ovens (C) 50–55 °C	
No.	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers
1	5.9	5.8	2.57	2.48	4.94	5.04
2	3.55	3.61	6.29	6.06	5.35	5.3
3	5.12	4.92	5.29	5.2	3.05	2.97
4	7.13	6.72	4.37	4.2	3.75	3.6
5	5.12	4.73	4.07	3.9	6.95	6.63
6	6.99	6.59	5.55	5.53	6.32	6.33
7	5.54	5.08	5.06	5.1	5.62	5.58
8	6.31	5.83	3.41	3.23	1.55	1.52
9	4.14	3.92	3.11	2.94	4.61	4.5
10	5	5.13	5.58	5.33	5.64	5.39
11	3.28	3.01	5.14	5.18	5.54	5.49
12	4.05	3.83	3.5	3.36	2.56	2.47
13	5.39	5.16	3.66	3.49	7.58	7.46
14	4.6	4.35	5.65	5.48	6.54	6.33
15	9.12	8.56	6.42	6.12	5.09	4.79
16	2.35	2.22	3.04	2.98	2.62	2.5
17	5.64	5.22	3.73	3.66	7.97	7.58
18	5.35	5.22	2.26	2.17	4.39	4.24
19	2.31	2.22	4.43	4.5	4.32	4.06
20	5.17	4.94	4.52	4.31	3.37	3.48
21	6.88	6.58	2.56	2.42	4.48	4.54
22	3.8	3.73	3.63	3.48	4.71	4.64
23	6.36	6.19	2.39	2.26	4.14	3.88
24	5.18	4.8	5.02	4.79	2.66	2.48
25	5.2	4.97	4.29	4.18	4.41	4.14

Employment: 5 years · Daily heat exposure: 8 h/day · n = 25 per subgroup