



# Heat Exposure and Oxidative DNA Damage Among Bakery Workers in Duhok Province, Iraq

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## Abstract

**Background and rationales:** There are not much researches on the effects of heat exposure, specifically on oxidative DNA damage, among bakery employees. Physical labor-intensive baking exposes bakers to high temperatures, especially in traditional bakeries with uncovered ovens. An electronic heat measuring device index and serum 8-hydroxy-2-deoxy guanosine(8-OHdG) will be utilized in this study to quantify oxidative DNA damage and heat stress, respectively. The impact of heat exposure on oxidative DNA damage in the Duhok populace has not yet been studied in the literature. This study aimed to measure the impact of heat exposure on serum levels of oxidative DNA damage in bakers who work in high-temperature bakeries. **Method:** A case control study was carried out among 141 participants, 62 individuals exposed to heat (bakery workers) and 79 individuals unexposed to heat exposure as control group. 8-hydroxy 2-deoxy guanosine (8-OHdG) has been analyzed using Enzyme-Linked Immunosorbent Assay (ELISA). **Results:** The mean  $\pm$ SD of 8-OHdG in bakery workers (13.78  $\pm$ 5.89 ng/ml) were higher than those in healthy control individuals (1.55  $\pm$ 0.75 ng/ml) with a statistically significantly differences ( $p < 0.0001$ ). **Conclusion:** High mean levels of 8-OHdG was found in exposed individuals (bakery workers) in comparison with control individuals unexposed to heat, suggesting that high-heat exposures have the risk of causing genetic effects and DNA damage.

**Keywords:** 8-hydroxy-2-deoxy Guanosine, Heat exposure, Oxidative DNA Damage.

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## I. INTRODUCTION

Heat exposure is of growing concern globally with climate change drawing more attention to this issue (Dong *et al.*, 2019) Working in conditions of extreme heat (i.e., temperatures above 35° Celsius), whether outdoors or inside, puts employees' health at risk and lowers labor productivity (Parsons, 2014).

When workers are exposed to high temperatures for an extended period of time, two of the physiological responses that take place are an increase in the risk of DNA damage and a change in the levels of heat shock protein (HSP) in the blood (Al-Horani, 2022). Bakers labor in a hot atmosphere that can cause heat stress, which can have a range of effects from moderate heat cramps to heatstroke (Argaud *et al.*, 2007).

One of the most significant elements that might increase the generation of reactive oxygen species (ROS) is heat stress (Li *et al.*, 2017). The most crucial location for the production of

ROS, a by-product of oxygen metabolism, is in mitochondria (Ray *et al.*, 2012). Additionally, cellular reactions to xenobiotics, cytokines, and bacterial invasion all result in the production of ROS (Starkov *et al.*, 2008). People exposed to high temperatures at work may experience a variety of heat-related diseases (HRIs), including heat rashes, cramps, weariness, syncope, and even heat stroke, which can be fatal (Kjellstrom *et al.*, 2009).

The possibility that exposure to high heat may disrupt a baker's DNA and genes has not received enough consideration. To get to a conclusion on the damage to DNA and the effect of the applied dose, more research and more thorough investigations are required to understand the effects. It's important to comprehend the physiological and molecular causes of heat-related disorders in order to create more potent cures and safeguards (Han *et al.*, 2013). Therefore, this study aimed to determine the effect of heat exposure on levels of

oxidative DNA damage in serum among bakers who are exposed to high temperature bakeries.

## II. MATERIALS AND METHODS

### A. Study design

This case-control study was carried out at bakery workers of Duhok, the interval from January 2022 to April 2022 and it was approved by the ethic committee, the study population was 141 participants (62 cases who exposed to high temperature in bakers and 79 individuals who are healthy people, and not exposed to high environment as a control group) were included in this study. Pre-tested questionnaire was designed to obtain on., age, gender, waist circumferences (WC), Weight, Height, body mass index (BMI), diseases, drug taking, smoking, occupation, and duration of work. 8-hydroxy 2-deoxy guanosine (8-OHdG) has been analyzed using Enzyme-Linked Immunosorbent Assay (ELISA), the levels of (8-OHdG)  $\leq 4.0$  regarded as normal value (Mahmoud and Altimimi, 2019).

### B. Methods

Venous blood samples were being obtained from bakers by an experienced analyst. Then blood sample will be transferred into a vial to separate the serum by centrifuge, the serum then used to measure Oxidative DNA damage [8-hydroxy 2-deoxy guano sine (8-OHdG)] for both groups (exposed and unexposed) using (mybiosource) kits by double antibody sandwich enzyme-linked immunosorbent assay. All measurements were done at university of Duhok, College of health science, medical laboratory department.

### C. Statistical analysis

The general information of the exposed and unexposed groups was presented in mean and Sta. deviation or number and presenter. The homogeneity of the age and gender between exposed and unexposed groups was examined in an independent t-test or Pearson chi-squared test. To make a homogeneity of age and gender between the two study groups, only 62 and 79 persons were included in this study. The comparisons of h8hd between exposed and unexposed groups were examined in an independent t-test. The correlations of DNA damages with heat stress were performed in bivariate regression. A p-value of less than 0.05 was used to establish the significance of the difference. In JMP pro-14.3.0, the statistical computations were carried out.

## III. RESULTS

The Comparisons of general information between exposed and unexposed study groups are summarized in Table 1. The mean age of exposed group was (30.16±8.74) years that is significantly less than of unexposed group (32.82 ±8.52 years). majority of exposed patients (29 ±46.77) and unexposed group (30 ±37.97) were aged from 20-29 years old. More than two third (54 ±87.10) of exposed group were

male comparatively to more than three quarter (66 ±83.54) of unexposed individuals. There was statistically significant difference of body mass index (BMI) and waist circumferences (WC) between studied group. The mean BMI of exposed group was 25.14 ±3.72 that is significantly higher than of unexposed group (24.96 ±5.05). Most of them (exposed (29 ±46.77) and unexposed (33 ±41.77) had normal body mass index according to WHO (BMI) classification. There was no significant difference statistically of diseases and drug taking. majority of exposed patients (37 ±59.68) and unexposed group (46 ±58.23) were smokers. nearly a half of them had 5-10 years' work duration. the abnormal WC (waist circumference) of exposed patients is significantly less than of unexposed group.

Table 1. Comparisons of general information between exposed and unexposed study groups

General information	Study groups no (%)		p-value
	Exposed (n=62)	Unexposed (n=79)	
Age mean ±SD	30.16 ±8.74	32.82 ±8.52	0.0709
Age groups			0.2245
17-19	6 ±9.68	2 ±2.53	
20-29	29 ±46.77	30 ±37.97	
30-39	16 ±25.81a	27 ±34.18	
40-49	10 ±16.13	17 ±21.52	
50-53	1 ±1.61	3 ±3.80	
Gender			0.5565
Male	54 ±87.10	66 ±83.54	
Female	8 ±12.90	13 ±16.46	
BMI mean (SD)	25.14 ±3.72	24.96±5.05	0.8197
BMI category			0.6612
Underweight	2 ±3.23	6 ±7.59	
Normal weight	29 ±46.77	33 ±41.77	
Overweight	24 ±38.71	29 ±36.71	
Obese	7 ±11.29	11 ±13.92	
Smoking			0.8622
Yes	37 ±59.68	46±58.23	
No	25 ±40.32	33 ±41.77	
Occupation			NA
Dough making and forming	21 ±33.87	0	
Baker	28 ±45.16	0	
Counter hand	13 ±20.97	0	
Working duration			NA
<5years	19 ±30.65	0	
5-10years	37 ±59.68	0	
>10years	6 ±9.68	0	
WC			0.2870
Abnormal	7 ±11.29	14 ±17.72	
Normal	55 ±88.71	65 ±82.28	

The Majority of high level of serum 8-OHdG among exposed patients were aged between 20 and 29 years old. Males had more increase in serum 8-OHdG levels than the females when comparing to each other. The greater number of exposed patients (64.04, 48.91%) were overweight and obese with no statistically significant difference between them. The mean

and std Dev of abnormal WC is greater than of the normal WC. most of the exposed patients were non-smokers (21.20, 17.43%). the value of serum 8-OHdG in bakers (24.29 (15.86%) is greater than of dough making (12.09, 5.68%) and counter hand (13.79, 7.28%). majority of high level of serum 8-OHdG (oxidative DNA damage levels) were found in workers who work >10 years. As shown in Table 2.

Table 2. Comparisons of oxidative DNA damage levels in exposed individuals with different characteristics.

Characteristics (n=62)	Level of 8-OHdG in the exposed group		p-value
	Mean	Std Dev	
<b>Age groups years</b>			0.2655
17-19	21.92	16.09	
20-29	27.54	24.10	
30-39	13.98	8.43	
40-49	22.16	13.53	
50-53	12.13	.	
<b>Gender</b>			0.0354
Male	14.97	6.57	
Female	9.46	3.06	
<b>BMI category</b>			0.0051
Underweight	13.60	1.53	
Normal weight	14.06	7.70	
Overweight	24.68	19.77	
Obese	39.36	29.14	
<b>WC</b>			<0.0001
Abnormal	37.76	24.61	
Normal	13.72	5.89	
<b>Smoking</b>			0.1089
Yes	15.62	6.85	
No	21.20	17.43	
<b>Occupation</b>			0.0031
Dough making and forming	12.09	5.68	
Baker	24.29	15.86	
Counter hand	13.79	7.28	
<b>Working duration</b>			0.0981
<5years	11.47	3.61	
5-10years	14.89	6.80	
>10years	18.63	12.21	

The degree of high temperature was measured for all groups include 141 subjects (108 males and 33 females), The mean of oxidative DNA damage in exposed group 13.78 where it was 1.55 A significant difference was found when comparison of oxidative DNA damage levels between cases and controls (exposed and unexposed groups), p-value <0.0001 as shown in Table 3.

The mean of (8-OHdG) was positively correlated with bakery temperature. There was a significant difference in oxidative DNA damage [8-hydroxy 2-deoxy guano sine (8-OHdG)] score between cases and controls mean of case (13.78±5.89 ng/ml) while for controls (1.55±0.75 ng/ml) as shown in Fig.1.

Table 3. Comparisons of oxidative DNA damage levels between exposed and unexposed study groups.

Study groups		p-value
Exposed (n=62)	Unexposed (n=79)	
13.78 ±5.89	1.55 ±0.75	<0.0001

There was a positive correlation between oxidative DNA damage and bakery temperatures at noon stress and evening stress but a negative correlation was found between oxidative DNA damage and bakery temperatures at morning stress r-value = -0.2250 and p-value=0.4200 (non-significant difference), as shown in Table 4.

Table 4. Correlations of oxidative DNA damage levels with bakery temperatures in the exposed group at different time points.

Outcome	Independent factors*		
	Morning stress	Noon stress	Evening stress
8-OHdG			
r	-0.2250	0.0808	0.2623
p-value	0.4200	0.7748	0.3450

\* Bivariate regression was performed for statistical analyses.

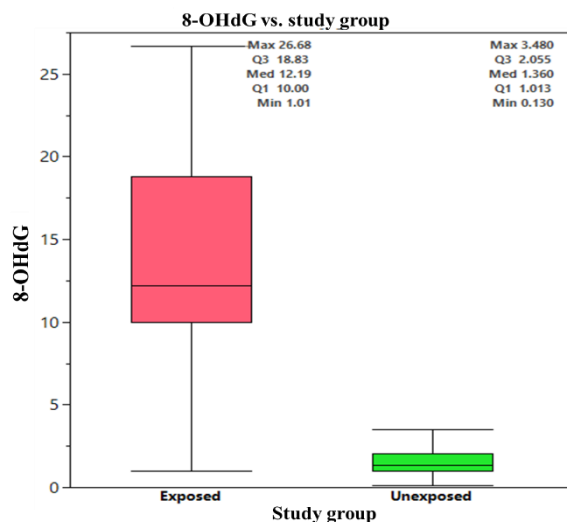


Figure 1. 8-OHdG of exposed and unexposed. Significant difference in oxidative DNA damage [8-hydroxy 2-deoxy guano sine (8-OHdG)] score between cases and controls mean of case (13.78±5.89 ng/ml) while for controls (1.55±0.75 ng/ml).

#### IV. DISCUSSION

According to Pachauri and Reisinger (2008), heat exposures are expected to be one of the primary health impacts on people. Workers are subjected to extreme heat at work as well as to high ambient temperatures, which adds to the health burden (Parsons, 2014). Workers performing hard physical work that requires intense arm and trunk work, carrying,

pushing, and pulling heavy loads throughout their shifts are subjected to high metabolic heat load in addition to the environmental heat that is imposed on them, placing them at a higher danger for heat stress. This risk may be further increased by continuous physical workload to meet production targets with little opportunity for self-pacing (Kjellstrom *et al.*, 2009).

The physiological manifestations of extreme heat exposure often include an increase in CBT, SwR, and USG. The link between these physiological markers of heat stress and the related negative health effects is well documented (Parsons, 2014). Workers are at risk of heat stroke and fatigue owing to a significant workload (Krishnamurthy *et al.*, 2017). According to Rooney *et al.* (1998), the behavioral changes made by the employees regarding their fluid intake and urination patterns also have a detrimental impact on physiological markers.

According to studies of Sawka *et al.*, (2001) and Aragón-Vargas *et al.*, (2009), excessive perspiration and the resulting dehydration worsen heat exhaustion and raise the chance of contracting a heat-related disease. The danger of heat-related diseases is increased for the workers due to the heat and their hard workload (Lucas *et al.*, 2015).

In our study, there was a significant correlation between workers exposed to heat and those who were not exposed (p-value <0.0001), and their DNA damage was ten times more than that of unexposed workers. The investigation of the relationship between work-related heat exposures and DNA damage in structured occupational contexts is novel and preliminary in nature.

In current study the mean levels of 8-OHdG was higher among older individuals, this was consistent with a perior study shown that the levels of 8-OHdG increased with age because of a decline in the ability of cells to repair DNA damage (Goukassian *et al.*, 2000).

From our study we found that the obese individuals have higher levels of 8-OHdG and this was consistent with a study observed in Egypt, shown that the levels of 8-OHdG is increased due to accumulation of triglycerides (Hameed *et al.*, 2012).

The study's key drawbacks are its case-control methodology, limited sample size, and use of data from a single province. To draw firm conclusions on the dose-response relationship between heat exposures and DNA damage, it is necessary to include more baking sectors with a range of exposure patterns and heat levels. The results are consistent with those of other research that have also found links between exposure to extreme heat stress and DNA damage within the study's constraints (Kantidze *et al.*, 2016).

## V. CONCLUSION

As global temperatures increase owing to climate change, heat-related diseases, one of the hidden reasons of morbidity and mortality globally, will become more severe. In addition to harming a worker's health and productivity, heat stress also

prevents DNA repair mechanisms from working properly and has the potential to damage DNA. In conclusion, our early data analysis suggests that high-heat exposures have the risk of causing genetic and DNA damage. It was found through the study and the results that Bakery workers have a significant increase in oxidative DNA damage when compared to unexposed group. A positive correlation exists between oxidative DNA damage. And there is a negative correlation in the bakery temperature at non stress.

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