Original Article

# Are Workers with Type 2 Diabetes at Greater Risk of Complications From Working in a Hot Environment? A Field Study in the Steel Industry

## Abstract

**Background:** The occurrence of heat stress in healthy individuals is different from those with chronic diseases like diabetes. While exposed to heat, complications caused by diabetes may lead to problems in body temperature regulation. Due to the fact that diabetic workers are less efficient in hot environments, researchers are encouraged to evaluate this condition. **Methods:** The current study incorporates 30 young males. In the first stage, individuals with the history of type 2 diabetes mellitus (T2DM), who frequently had exposure to heat at work, were selected and compared with non-diabetic workers as the control group. Indicators like deep body temperature, skin temperature, heart rate, physiological strain index (PSI), and perceptual strain index (PeSI) were measured and recorded. Eventually, data were evaluated and analyzed using repeated-measure design, independent $t$ test, and its nonparametric equivalent, The Mann–Whitney U test. **Results:** In the two groups of type 2 diabetes and control group, following one hour of heat exposure, the median heart rate was 115 (18) and 99 (21) ($P = 0.008$), respectively, mean deep body temperature was 37.6 (0.37) and 36.95 (0.41) ($P < 0.001$), mean PSI was 3.01 (0.93) and 2.08 (1.0) ($P = 0.021$), and mean PeSI was 7.82 (1.43) and 6.12 (2.14) ($P = 0.032$), which were significantly different. In addition, no significant difference was observed between the skin temperatures of the two groups after one hour of exposure to heat. **Conclusions:** According to the results, workers with type 2 diabetes are exposed to more heat strain than the control group in the same hot environment.

**Keywords:** *Diabetes mellitus, heat stress reaction, heat shock response, type 2, occupational diseases*

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## Introduction

In 2011, based on estimations, the prevalence of diabetes in Iran was 11.4% in the adult population (approximately 4.5 million individuals), and it is projected that this trend will even soar to 9.2 million by 2030.[1] One of the most considerable reasons for premature retirement is the unexpected prevalence of diabetes. This is aligned with an increase in the number of diabetic cases in the workers' population.[2] Despite this premature retirement, there are still many diabetic workers in the workplace. According to the statistics derived from the Occupational Medicine Centre of Mobarakeh Steel Company in Isfahan, 2.6% of the workers in this company have diabetes in 2022.

Industrial workplaces including the steel industry contain several physical ambient factors. One of the most remarkable factors is heat stress.[3] Although determining the health quality of workers of steel companies is affected by several factors, exposure to excess heat near smelting furnaces, especially in summer, plays a major role in the occurrence of heat stress.[4] Yet, vulnerability to excessive heat either due to environmental or seasonal factors is quite inevitable. Constant exposure to the aforementioned factors would lead to health threats, a reduction in individuals' productivity, and thus a reduction in organization productivity.[5,6] Inability to adjust the core body temperature (CBT) as well as insufficient heat dissipation could lead to heat stress. The wet bulb globe temperature (WBGT) is considered a practical index that assesses different factors, leading to heat stress.[7-9]

During the heat stress condition, several endogenous responses, namely, heat strain try to maintain the physiologic thermal balance.[7] Heat strain indices include physiological and perceptual indices. The international organization for

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standardization 9886 (ISO 9886, evaluation of ergonomics of thermal strain) has asserted that physiologic parameters like CBT, skin temperature (ST), and heart rate (HR) determine the physiological heat strain index.[10] PSI is an indicator that measures thermal strain using heart rate and body temperature. PSI represents both the amount of load on the cardiovascular system and the regulation of body temperature simultaneously.[11] Moreover, the measurement of individual thermal sensation (TS) and perceived exertion (PE), on the other hand, assesses the perceptual strain index (PeSI).[12]

Heat stress in healthy individuals greatly varies from those with chronic diseases.[13,14] One of these chronic diseases is diabetes, which impairs the body's ability to regulate blood glucose.[15] While in type 1 diabetes mellitus (T1DM) there is insufficient insulin production, in type 2 (T2DM) there is tolerance to insulin and glucose uptake.[16] It is worth mentioning that T2DM is more prevalent than T1DM (approximately 90—95% of whole diabetic patients).[17]

Prolonged accumulation of glucose in diabetic patients would lead to microvascular injury.[18,19] In other words, it leads to the following complications through three main pathways. Firstly, suppressed uptake of glucose enhances its transformation to sorbitol. Secondly, increase in levels of advanced glycation end-products (AGEs) like glycation of serum albumin. Finally, prolonged enhancement of fructose 6-phosphate production and consequently, narrowing microvessels and initiation of ischemic status.[20]

The reasons presented above bring us to the conclusion that diabetic individuals might confront serious problems regarding temperature regulation and responsive system of heat dissipation.[14,21] Normally, the endogenous regulatory system of body temperature attempts to increase superficial skin perfusion in order to enhance dissipating of excess heat through sweating, vaporization, or radiation. Failure of physiologic vasodilation in diabetic patients is not aligned with the aforementioned mechanism; consequently, these individuals might experience the unsatisfied amount of heat dissipation and superficial skin perfusion.[14,15,22-24]

Moreover, staying in environments with a higher temperature than CBT necessitates the combination of sweating and vaporizing as the only practical alternatives.[7] It has been demonstrated that poor control of blood sugar (HbA1C >6.5%) might alter the possibility of compensatory sweating.[14,25]

Furthermore, diabetic patients as a consequence of higher blood sugar and polyuria are more prone to dehydration.[17] Dehydration itself plays a key role in heat stress and heat-related disease.[13]

Apparently, diabetic workers with higher occupational exposure to heat are more susceptible to have lower job efficacy.[26] Therefore, more attention should be paid to workers who experience long exposure to heat. There are also few studies on workers with T2DM and their reactions to heat stress, So in this study, we aimed to evaluate the physiological and perceptual strain index in diabetic industrial workers compared with non-diabetic workers as the control group.

## Material and Methods

The present study incorporates 30 middle-aged working men (aged 40–52, mean = 46.26, SD = 3.26) who were naturally exposed to heat in their workplace (from September to October). The study was a case–control type. The number of samples was 10 people with type 2 diabetes and 20 healthy people, taking the formula $n = [(z1 + z2)^2\ (2s)^2]/d^2$, and the strict entry criteria into account. At first, 10 people with T2DM were selected. The inclusion criteria for the T2DM group were all working workers with type 2 diabetes whose diseases have been confirmed by an industrial medicine physician, job positions that are exposed to heat for more than 2 hours per shift, the absence of diabetes-related and cardiovascular diseases, thyroid and blood pressure, not consuming alcohol, caffeine, cigarettes and tobacco 12 hours before measuring the individuals' parameters, and finally not taking drugs that affect the cardiovascular system, metabolism, and body dehydration. It should be noted that there was no problem with taking diabetes medications. According to the entry criteria, the total number of samples that were available was selected and the sampling method was the available type. The exclusion criterion for all participants was the lack of proper cooperation in measuring the parameters.

Selected job positions for the study were: casting mold operator, electrode assembly operator, smelter, crane repairman, etc. The average WBGT of the resting places and work environments is listed in Table 1. The study subjects were exposed to heat caused by both external (work environment heat) and internal (activities that increase metabolism) sources.

According to the statistician's opinion, for better matching in terms of environmental conditions (WBGT, rest, and work environments), load of work, type of clothing, and personal protective equipment, for each person with T2DM from a job position regarding the mean and standard deviation of age and BMI, two healthy people were selected from the same job position, which was the same entry criteria for the control group. Ten percent of the study subjects had a light workload (metabolism of up to 200 kcal/hour), 80% had a medium workload (metabolism of up to 200 to 350 kcal/hour), and 10% had a heavy workload (metabolism of up to 350 to 500 kcal per hour). Informed consent was obtained from all participants. Also, this research approved the

**Table 1: Demographic information of participants**

| | T2DM (*n*=10) | | Control (*n*=20) | | *P* |
|---|---|---|---|---|---|
| | Mean (SD) | Range | Mean (SD) | Range | |
| Age (years) | 46.9 (4.01) | 40-52 | 45.9 (2.89) | 42-51 | 0.46 |
| Height (m) | 177.7 (4.85) | 170-185 | 173.5 (6.06) | 162-182 | 0.068 |
| Weight (kg) | 85 (10.06) | 70-99 | 81.5 (8.38) | 65-98 | 0.32 |
| BMI (kg/m$^2$) | 26.85 (2.3) | 22.6-30.56 | 27.03 (1.85) | 24-29.91 | 0.82 |
| Body surface area (m$^2$)* | 2.02 (0.13) | 1.81-2.19 | 1.95 (0.12) | 1.69-2.18 | 0.178 |
| Duration of diabetes (years) | 6.6 (4.9) | 3-20 | - | - | - |
| Resting WBGT (◦C) | 18.14 (0.87) | 16.67-19.2 | 18.14 (0.84) | 16.7-19.2 | - |
| Workplace WBGT (◦C) | 33.77 (1.2) | 31.87-38.8 | 33.77 (1.2) | 31.87-38.8 | - |

For measuring body surface area, the equation below was used (26): 0.20247* height (m) 0.725* weight (kg) 0.425. Independent *t*-test indicates that there is no significant demographic difference between the T2DM group and the control group (*P*>0.05)

code of ethics on October 3, 2020 (IR.MUI.RESEARCH.REC.1399.410).

WBGT device (QUESTemp 32) assessed WBGT indices for each workplace, once in the resting place and once in the workplace. Since all participants used the same equipment, the same adjustment clothing factors were employed in order to modify environmental WBGT based on the structure and heat resistance of clothing.[27]

Heart rate, CBT, and ST were measured, first, during the resting time or at least 15 min prior to any heat exposure, then, after initial 30 min of exposure to heat, and finally, after 60 min of exposure. Heart rate was recorded by pulse oximeter (fingertip LK87) which also shows real-time oxygen saturation (O2 sat%) and heart rate (HR). Participants were asked to hold their arms flexed so that fingertips can maintain the same height as the hearts. Thirty seconds after that the device displayed numbers, the value of HR was obtained. Core body temperature and skin temperature were measured via a non-contact infrared thermometer (WF-1000; B.Well Swiss). Assessment of CBT took place by inserting its probe into the external ear while the ear is pulled upward and back. These stages were repeated three times for each participant and the mean of three values was calculated. After each time, the device was disinfected using an alcoholic pad. Moreover, ST of chest, arm, thigh, and calf regions were separately measured three times. The mean of three observations was considered as the input value. Then, according to the following equation,[28] mean total skin temperature was acquired.

$$\mathbf{T_{sk} = (0.3\ T_{chest}) + (0.3\ T_{arm}) + (0.2\ T_{thigh}) + (0.2\ T_{calf})}$$

PSI and PeSI both were separately measured two times, 30 min and one hour after exposure to heat, respectively. PeSI score was evaluated through a subjective rating assessment of thermal sensation (TS) and perceived exertion (PE). TS was calculated using a five-scale rating of 1 (comfortable), 2 (slightly warm), 3 (warm), 4 (hot), and 5 (very hot). Participants were asked to rate PE qualitatively and subjectively ranging from 1 (extremely easy) to 10 (extremely hard). The following formulas present how PSI and PeSI were calculated.[29] $T_{c0}$ and $T_{ct}$ represent CBT during resting and activity, respectively. Likewise, $HR_{c0}$ and $HR_{ct}$ show HR during resting and activity, respectively.

$$\mathbf{PSI = [5(T_{ct} - T_{c0})/(39.5 - T_{c0})] + [5(HR_{ct} - HR_{c0})/(180 - HR_{c0})]}$$

$$\mathbf{PeSI = 5[(TS - 1)/4] + 5(PE/10)}$$

In this study, SPSS 25.0.0 software was used for data analysis. Independent *t* test was used to compare all parameters of thermal, physiological, and perceptual strain indices in case of normal distribution. Otherwise, this comparison was done by Mann–Whitney U test. For the three parameters, namely ST, CBT, and HR, apart from the mentioned tests, the repeated-measure test was also performed. Additionally, to describe the variables with normal distribution, mean and standard deviation were used, and to describe the variables with non-normal distribution, the median and interquartile range were used.

## Results

Demographic features of study subjects are included in Table 1.

### Core body temperature

CBT results in both T2DM and control groups are shown in Table 2. These parameters were measured in three separate times. In the T2D and control groups, the average core body temperature at resting stage was 36.76 (0.3) and 36.29 (0.47). Thirty minutes after exposure, it was calculated 37.2 (0.28) and 36.98 (0.51), respectively. And 60 min after exposure, it was 37.6 (0.37) and 36.95 (0.41), respectively. The table below indicates that the mean $T_{core}$ was higher in T2DM group constantly. Moreover, an independent *t* test revealed that while mean $T_{core}$ is significantly higher in participants with T2DM prior to exposure ($P = 0.008$) and after 60 min of exposure ($P < 0.001$), there was no significant difference between groups after thirty minutes of exposure. Repeated-measure test also revealed that CBT increased more in the T2DM group compared to the control

**Table 2: Core body and skin temperature findings**

| Time | T2DM (*n*=10) | | Control (*n*=20) | | *P* |
|---|---|---|---|---|---|
| | Mean (SD) | Range | Mean (SD) | Range | |
| Tcore (°C) | | | | | |
| Rest time | 36.76 (0.3) | 36.3-37.3 | 36.29 (0.47) | 35.7-37.3 | 0.008 |
| 30 min after heat exposure | 37.2 (0.28) | 36.8-37.6 | 36.98 (0.51) | 35.9-37.9 | 0.213 |
| 1 h after heat exposure | 37.6 (0.37) | 37.1-38.2 | 36.95 (0.41) | 36.36-37.7 | 0.000 |
| Tsk (°C) | | | | | |
| Rest time | 34.65 (1.08) | 32.47-36 | 34.69 (0.85) | 33.06-36.14 | 0.9 |
| 30 min after heat exposure | 36.2 (1.18) | 35.12-37.25 | 36.39 (1.35) | 33.43-37.65 | 0.914 |
| 1 h after heat exposure | 36.33 (0.9) | 34.14-37.48 | 36.63 (0.85) | 33.7-37.63 | 0.559 |

In this table, T2DM is type 2 diabetes mellitus, SD is the standard deviation, IQR is the interquartile range, Tcore is the core body temperature in centigrade, and Tsk is the skin temperature in centigrade

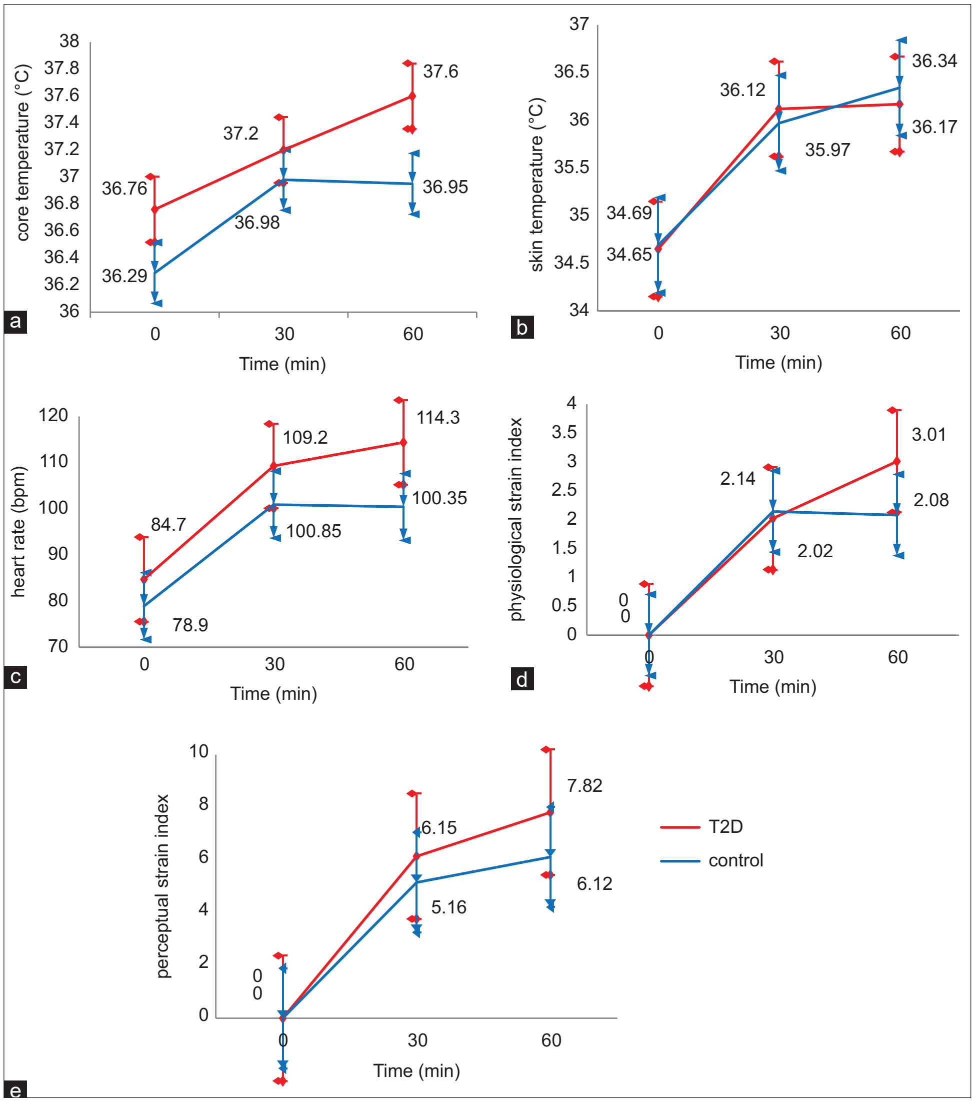

**Figure 1: Patients with T2DM have been shown with a red line and the control group with a blue line. All data are reported as mean collected at rest or before heat exposure (time= 0), 30 minutes (time=30) and 1 hour (time=60) after heat exposure. According to equation, values of PSI compose CBT and HR either at resting stage or during work. This semi-horizontal line with a very slight decreasing slope in the PSI chart of the control group shows that by passing from 30 minutes to one hour, the core body temperature and heart rate in this group not only did not increase, but also decreased slightly. However, since the core body temperature and heart rate in T2D people have increased over time from 30 minutes to one hour, the PSI graph for this group has an increasing slope. (a) Comparison of mean CBT between the T2DM and control groups at three points of time (0, 30, 60), (b) Comparison of mean ST between the T2DM and control groups at three points of time (0, 30, 60), (c) Comparison of mean HR between the T2DM and control groups at three points of time (0, 30, 60), (d) Comparison of mean PSI between the T2DM and control groups at three points of time(0, 30, 60), (e) Comparison of mean PeSI between the T2DM and control groups at three points of time (0, 30, 60)**

group, and this difference was significant (*P* = 0.003). Diagram A in Figure 1 illustrates variations of CBT after exposure to heat in both groups.

### Skin temperature

As it has been indicated in Table 2, skin temperature has increased after exposure in both groups. The mean and median measures of ST in the control group were slightly higher compared to T2DM. In the T2DM group and the control group, the average skin temperature at the resting stage was 34.65 (1.08) and 34.69 (0.85), respectively. After 30 min of exposure to moderate heat, it was 36.2 (1.18) and 36.39 (1.35), respectively, and after 60 min of exposure, it was 36.33 (0.9) and 36.63 (0.85), respectively. Also, the independent *t* test demonstrated that mean ST was not significantly different between groups prior to exposure. Furthermore, Mann–Whitney U test confirmed that mean ST after both 30 and 60 min of exposure were not significantly different between groups. Repeated-measure test also demonstrated that there is no significant difference between the two groups during the time (*P* = 0.940). Diagram B in Figure 1 indicates changes of ST over time.

### Heart rate

Table 3 depicts the evaluation of HR over time. Results of both T2DM and control groups at the resting stage, after 30 min, and after 60 min of exposure are depicted. In the T2DM and control groups, the average resting heart rate was 84.7 (10.56) and 78.9 (7.41), respectively. After 30 min of exposure, the averages were 109.2 (12.95) and 100.85 (13.42), respectively. And after 60 min of exposure, the averages were 115 (18) and 99 (21), respectively. Independent *t* test indicated that mean HR before exposure and after 30 min of exposure are not different significantly between study groups. Nonetheless, Mann–Whitney U test revealed that the mean of HR is different significantly between the T2DM and control groups (*P* = 0.008).Repeated-measure test also revealed that there was a greater increase in HR in the T2DM group compared to the control group over time, and this difference was significant (*P* = 0.019). Diagram C in Figure 1 illustrates variations of mean HR among groups over time.

### Physiological strain index (PSI) and perceptual strain index (PeSI)

Table 4 shows the analytic features of strain indices. In the T2DM and control groups, the mean PSI 30 min after exposure was 1.55 (1.81) and 2.3 (1.83), respectively, and after 60 min of exposure, the average was 3.01 (0.93) and 2.08 (1), respectively. Also, in the T2DM and control groups, the average PeSI, 30 min after exposure, was 6.12 (2) and 5 (2.87), respectively, and after 60 min of exposure, the average was 7.82 (1.43) and 6.12 (2.14), respectively. Mann–Whitney U test depicted that PSI is not significantly different between groups after 30 min of heat exposure. However, an independent *t* test demonstrated that after 60 min of heat exposure, PSI is significantly different between the T2DM and control groups (*P* = 0.021). In addition, Table 4 represents a statistical comparison of PeSI between groups. Likewise, Mann–Whitney U test showed that there is no significant difference between groups after 30 min of heat exposure. Meanwhile, the applied independent *t* test indicated that PeSI is considerably different between groups after 60 min of heat exposure (*P* = 0.032).

## Discussion

This study assessed physical and perceptual strain indices as a comparison between T2DM workers and healthy individuals. Results of this study indicated that T2DM participants exhibited significantly higher CBT after

**Table 3: Heart rate findings**

| HR (bpm) | T2DM (*n*=10) | | Control (*n*=20) | | *P* |
|---|---|---|---|---|---|
| | Mean (SD) | Range | Mean (SD) | Range | |
| Rest time | 84.7 (10.56) | 67-104 | 78.9 (7.41) | 69-94 | 0.091 |
| 30 min after heat exposure | 109.2 (12.95) | 93-136 | 100.85 (13.42) | 85-128 | 0.115 |
| 1 h after heat exposure | 115 (18) | 92-138 | 99 (21) | 82-127 | 0.008 |

In this table, T2DM is type 2 diabetes mellitus, SD is the standard deviation, IQR is the interquartile range, and HR is heart rate in beats per minute

**Table 4: Physiological and perceptual strain index finding**

| | T2DM (*n*=10) | | Control (*n*=20) | | *P* |
|---|---|---|---|---|---|
| | Median (IQR) | Range | Median (IQR) | Range | |
| PSI 30 min after heat exposure | 1.55 (1.81) | 1.06-3.47 | 2.30 (1.83) | 0.05-3.46 | 0.812 |
| PeSI 30 min after heat exposure | 6.12 (2) | 4.5-9.5 | 5 (2.87) | 1.5-10 | 0.109 |
| PSI 1 h after heat exposure | 3.01 (0.93) | 1.65-4.37 | 2.08 (1.00) | 0.54-3.98 | 0.021 |
| PeSI 1 h after heat exposure | 7.82 (1.43) | 5.5-10 | 6.12 (2.14) | 3.25-10 | 0.032 |

In this table, T2DM is type 2 diabetes mellitus, SD is the standard deviation, IQR is the interquartile range, PSI is physiological strain index, and PeSI is perceptual strain index exposure to heat in the workplace. Although the ST increased over time, still no significant difference was observed between study groups. It is worth mentioning that the human body needs to maintain its internal temperature within the normal range in order to function normally and steadily. Naturally, following the production of excess heat in response to either exercise (metabolism) or environmental source, endogenous thermo-regulation attempts to enhance the heat dissipation through sweating and vaporization. Substantially, an increase in superficial cutaneous perfusion is crucial.

As it has been mentioned so far, diabetic patients experience micro-vasculopathy and autonomic neuropathy. Therefore, following heat exposure, vasodilation, sweating, and increase in cutaneous perfusion, the body cannot functionally dissipate the excess heat.[14,15,22-24] Wick *et al*. suggested that the threshold of cutaneous vasodilation among T2DM patients is higher compared with control group. This could result in thermoregulation impairment.[21] Additionally, Petrofsky *et al*.[24] revealed that diabetic patients have lower cutaneous perfusion and consequently, lower heat dissipation compared with the control group. Iavicoli *et al*. also asserted that high temperatures could put diabetic workers at risk due to changes in cutaneous perfusion. These conditions are able to change the mechanisms of heat dissipation and thermoregulatory systems.[23]

In high ambient temperatures, the only way to dissipate heat is to evaporate sweat.[30] With regard to this fact, poor control of blood sugar[14] would lead to endothelial injury and damage to sweat glands.[31-33] Petrofsky *et al*. asserted that tolerance to heat stress altered in both T2DM and T1DM patients. They noted that diabetic patients have higher levels of ST and regarding their basal thermoregulatory dysfunction, this increase in ST would cause CBT to increase as well, thus making patients more prone to heat stress.[31] Nonetheless, in the current study, the ST was not different significantly between the groups possibly due to the increase in the sample size, larger inclusion of regional ST, and considering the past medical history of the participants. In another study, Petrofsky *et al*. concluded that sweating eccrine activity in the forehead site is more than in other parts of the body in diabetic patients during physical activity or heat exposure.[34] Furthermore, Kenny *et al*.[15] mentioned that attenuated superficial cutaneous perfusion and reduction in sweating in diabetic patients are correlated with impairment in their thermoregulation.

Our results indicated that the HR in diabetic participants increased following physical activity and heat exposure. It is worth mentioning that this increase was significantly different in comparison with the control group. HR is also another physiologic response to heat stress. Higher HR would increase superficial perfusion and correspondingly, facilitate the total capacity of heat dissipation. Notley *et al*. discussed effects of exercise on tolerance to heat stress in T2DM subjects. It has been shown that participants with T2DM were not able to dissipate the excess heat satisfyingly owing to sweat evaporation disorder. This could bring about higher means of HR and CBT in T2DM study participants.[35] Likewise, Kenny *et al*. concluded that adults with a history of T2DM reach a higher strain index since they have lower heat dissipation.[36] Eventually, Notley *et al*. also mentioned that T2DM patients have lower heat tolerance when doing prolonged and intense exercises. Prolonged exposure to heat and physical activity would aggravate the accumulation of excess heat in diabetic patients.[26]

PSI directly represents effects of CBT and HR before and after heat exposure. Correspondingly, an increase in amounts of HR and CBT would increase the PSI as well. In this study, it was observed that PSI was increased significantly in diabetic workers following one hour of heat exposure. Also, PeSI as a subjective assessment of heat strain was increased in the T2DM group. Possibly, this is as a consequence of disturbed heat dissipation and elevated level of heat capacity in T2DM participants.

## Conclusions

Based on the permissible limits of occupational exposure and the measurements performed, the sites studied in this research were thermally stressful and had the ability to cause stress in all individuals. We evaluated trending patterns of physiological and perceptual strain index among diabetic industrial workers following heat exposure in comparison with the control group. After one hour of heat exposure between two groups, the difference in mean CBT was 0.15°C, the difference in median ST was 0.3°C, the difference in median HR was 16 bpm, and the difference in mean PSI and PeSI was 0.93 and 1.7, respectively. This study indicated that T2DM industrial workers are more prone to heat strain and heat-related illness.

This study included several limitations. First of all, regarding the COVID-19 pandemic and correspondent protocols, we were not able to include seasonal features that might interfere with the effect of heat exposure on the participants. As a future direction, further studies targeting the effects of seasonal divergence on heat stress and strain indices would clarify the impact of the season. Moreover, quantitative features of blood sugar control and surveillance were not feasible in this study. Assessing the role of HbA1C in future studies would strengthen the understanding of this issue. Eventually, environmental noise could have had a disturbing effect on our assessments. A more appropriate and isolated setting of study with exclusion of disturbing factors would present a more clear blueprint of this phenomenon.

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## Authors contribution

- Fatemeh Dehghani contributed to the acquisition and interpretation of data for the work and drafting of the work.
- Habibollah Dehghan contributed to revising the work critically for important intellectual content and final approval of the version to be published.
- Siamak Pourabdian contributed to substantial contributions to the conception of the work and final approval of the version to be published.

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## Conflicts of interest

There are no conflicts of interest.

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