KI REPORTS
KIReports.org

Hyperthermia Predicts Cross-Shift Acute Kidney Injury Risk in Construction Workers

Erica Tourula[1], Jonathan W. Specht[2], M. Jo Hite[1], Charlie Walker[1], Serena Garcia[2], Omkar Khandpekar[3], Hillary A. Yoder[4], Roger S. Zoh[3], Blair D. Johnson[1], David H. Wegman[5,6], Jason Glaser[5], Fabiano Amorim[2,5] and Zachary J. Schlader[1,5]

[1]Department of Kinesiology, Indiana University School of Public Health, Bloomington, Indiana, USA; [2]Department of Health, Exercise, and Sports Sciences, University of New Mexico, Albuquerque, New Mexico, USA; [3]Department of Epidemiology and Biostatistics, Indiana University School of Public Health, Bloomington, Indiana, USA; [4]Department of Kinesiology, New Mexico State University, Las Cruces, New Mexico, USA; [5]La Isla Network, Washington DC, USA; and [6]Department of Public Health, University of Massachusetts Lowell, Lowell, Massachusetts, USA

Correspondence: Zachary J. Schlader, Department of Kinesiology, Indiana University School of Public Health, 1025 E. Seventh St., Ste 112, Bloomington, Indiana 47405, USA. E-mail: zschlade@iu.edu

Received 24 January 2025; revised 15 April 2025; accepted 27 May 2025; published online 3 June 2025

Kidney Int Rep (2025) **10,** 2856–2859; https://doi.org/10.1016/j.ekir.2025.05.044
KEYWORDS: acute kidney injury; dehydration; heat stress

See Commentary on Page 2527

INTRODUCTION

Up to 70% of the global workforce is regularly exposed to excessive heat.[1] The resulting occupational heat stress increases the likelihood of elevations in core temperature (i.e., hyperthermia)[1] that in turn, elevate the risk of heat-related injuries and illnesses.[1] This now includes chronic kidney disease (CKD), where cases do not present with typical risk factors (e.g., obesity, diabetes, hypertension, etc.), and is therefore termed CKD of nontraditional causes.[S1]

Although the etiology of CKD of nontraditional causes is not fully understood, rodent models suggest that single or repeated hyperthermia exposures can cause acute kidney injury (AKI) that, over time, can progress to CKD.[2] This is supported by observations that workers at high risk for CKD of nontraditional causes (e.g., sugarcane workers) frequently encounter occupational heat stress and also exhibit high rates of hospital admission for AKI.[3] Clinical, laboratory-based studies simulating occupational heat stress have demonstrated that AKI risk, estimated from the product of urinary insulin-like growth factor-binding protein 7 (IGFBP7) and tissue inhibitor of metalloproteinases-2 (TIMP-2), [IGFBP7*TIMP-2], a US Food and Drug Administration–approved biomarker for assessing the risk for AKI,[S2] is elevated in a hyperthermia-dependent manner.[S3] This AKI risk is further elevated by the presence of dehydration,[4] which is common during occupational heat stress because of insufficient fluid intake to offset body water losses due to sweating. Although in agriculture workers hyperthermia and dehydration have been shown to increase the likelihood of AKI (determined via cross-shift changes in serum creatinine),[5] whether the laboratory-based observations of urinary [IGFBP7*TIMP-2], which avoids some of the limitations associated with using changes in serum creatinine to define AKI during occupational heat stress,[S4] translate to the field setting is unknown.

Likely because of frequent and simultaneous exposures to both excessive environmental heat and heavy work,[S5,S6] construction workers are the largest employment sector frequently exposed to occupational heat stress in the US, and the construction sector accounts for the highest number of occupational heat-related hospitalized AKI events.[6] Data from 1 study of Saudi Arabian residential construction workers indicate that 18% of workers suffered kidney injury (as measured by an albumin-to-creatinine ratio $>$ 30 mg/g) across the hot summer months.[7] Despite this background, the role of hyperthermia and dehydration exposure in the development of AKI risk (i.e., cross-shift urinary [IGFBP7*TIMP-2]) in construction workers is unknown. Therefore, we tested the hypothesis that during the summer in the Midwest and Southwest regions of the US, cross-work shift AKI risk is predicted by hyperthermia and dehydration.

RESULTS

Outdoor construction workers were observed over 1 working shift on 1 of 4 days at a commercial building construction site in the Midwest (n = 32) and a road

Table 1. Participant and workday characteristics for construction workers

Participant characteristics						
Prescreening variables	**Carpenters (n = 6)**	**Concrete (n = 13)**	**Laborers (n = 8)**	**Roofers (n = 5)**	**Highway (n = 7)**	**Total (N = 39)**
Age (yrs)	33 ± 13	35 ± 12	37 ± 14	43 ± 13	38 ± 13	37 ± 13 (18-64)
Sex	6 M	12 M	5 M / 3 F	5 M	7 M	39 M/ 3 F
Height (cm)	176 ± 9	175 ± 11	170 ± 12	174 ± 3	169 ± 6	174 ± 10 (148–192)
Body mass (kg)	85.1 ± 24.3	86.0 ± 14.0	77.5 ± 14.1	90.1 ± 18.5	86.0 ± 25.3	84.6 ± 18.8 (56.8–139.7)
BMI (kg/m^2)	27.2 ± 5.6	28.3 ± 4.5	26.9 ± 4.5	29.7 ± 5.7	30.1 ± 8.9	28.3 ± 5.6 (18.0–49.0)
Systolic BP (mm Hg)	122 ± 8	122 ± 9	120 ± 8	123 ± 9	129 ± 8	123 ± 9 (102–138)
Diastolic BP (mm Hg)	75 ± 9	76 ± 6	73 ± 8	78 ± 6	86 ± 3	77 ± 8 (60–90)

Workday characteristics			
	Average	**Peak**	***n***
Core temperature (°C)	37.4 ± 0.2	37.9 ± 0.2	34
Change in core temperature [peak minus pre] (°C)		1.0 ± 0.4	32
Estimated metabolic rate (W/m^2)	191 ± 41	358 ± 89	27
Heat index (°C)			
Day 1 (Building construction)	24.0	24.7	8
Day 2 (Building construction)	29.3 ± 0.7	33.0 ± 1.5	10
Day 3 (Building construction)	29.3 ± 0.7	31.1 ± 2.0	14
Day 4 (Road construction)	24.5	34.1	7

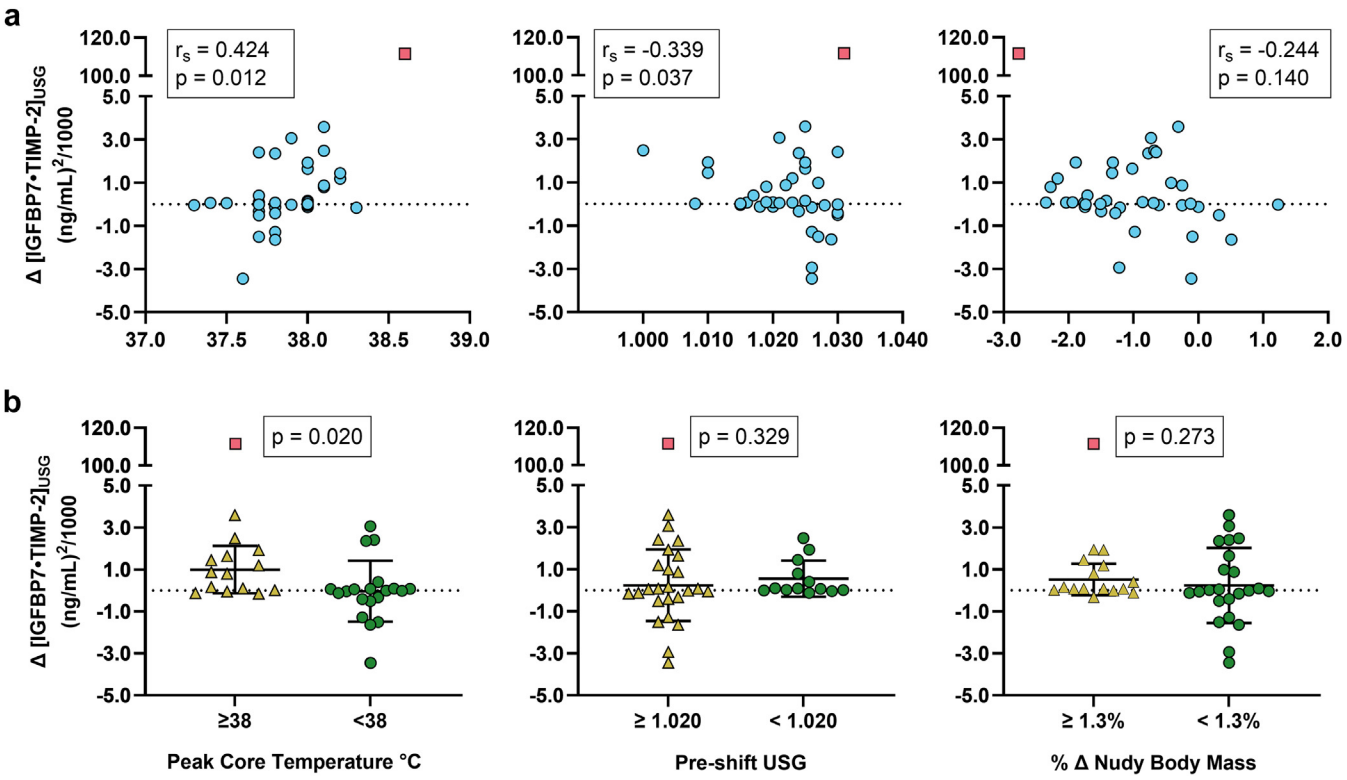
	Pre	**Post**	***P*-value**	***n***
Nude body mass (kg)	85.0 ± 18.3	84.1 ± 18.2	< 0.001	38
Urine specific gravity (USG)	1.021 ± 0.007	1.022 ± 0.007	0.675	38
[IGFBP7*TIMP-2]$_{USG}$	0.68 ± 0.96	1.02 ± 1.20	0.140	38

BMI, body mass index; BP, blood pressure; F, female; IGFBP7, insulin-like growth factor-binding protein 7; M, male; TIMP-2, tissue inhibitor of metalloproteinases-2.
Workday characteristic data is presented as mean ± SD, except average and peak heat index (HI), with subject number (n) reported in the last column. Paired non-parametric Wilcoxon tests were used to compare pre- and post-work.
Participant characteristics presented as mean ± SD, with (minimum, maximum) reported in the Total column.

construction site in the Southwest (n = 7) (Table 1), with spot urine samples being provided before (pre-) and after (post-) work. Prework shift urine specific gravity (USG), a marker of prework hydration status, was 1.021 ± 0.006 (mean ± SD), with 59% of workers starting the workday with a USG > 1.020, indicative of dehydration.[S7] The average peak heat index on these days was 30.8 ± 3.6 °C (range: 24.7–35.2 °C). Average work intensity[8] was light (n = 1), moderate (n = 15), heavy (n = 10), and very heavy (n = 2) (Supplementary Methods). On average, workers lost 1.0% ± 0.8% body weight (range: −2.4% to +1.2%), which is a marker of cross-shift changes in hydration status.[S7] Peak core temperature (telemetry pill, n = 34) was 37.9 ± 0.2 °C (range: 37.3–38.3 °C). Urinary [IGFBP7*TIMP-2],[9] which was calculated as the product of USG corrected urinary IGFBP7 and TIMP-2 divided by 1000, did not change from prework shift (0.68 ± 0.96 [ng/ml]^2/1000) to postwork shift (1.02 ± 1.20 [ng/ml]^2/1000) (Wilcoxon test: P = 0.140; Cohen's d_z = −0.32). The prework to postwork change (Δ) in urinary [IGFBP7*TIMP-2]$_{USG}$ was higher in workers with peak core temperatures ≥ 38 °C (n = 14) versus < 38 °C (n = 20) (0.99 ± 1.13 vs. −0.04 ± 1.45 [ng/ml]^2/1000, Mann-Whitney test: P = 0.020; Cohen's d_z = 0.79). Both a prework USG of 1.020 and a cross-shift body mass loss ≥ 1.3% are indicative of dehydration.[S7] However, there were no differences in Δ[IGFBP7*TIMP-2]$_{USG}$ when preshift USG was above (n = 25) versus below (n = 13) 1.020 (Mann-Whitney test: P = 0.329; Cohen's d_z = 0.23), or above (n = 15) versus below (n = 23) 1.3% body mass loss (Mann-Whitney test: P = 0.273; Cohen's d_z = 0.20) (Figure 1). Δ[IGFBP7*TIMP-2]$_{USG}$ was not significantly correlated (Spearman Rank) with percent changes in body weight (r_s = −0.244; P = 0.139) but was negatively correlated with preshift USG (r_s = −0.339; P = 0.037) and positively correlated with peak core temperature (r_s = 0.424; P = 0.012) (Figure 1). Multiple linear regression indicated peak core temperature (β: 2.37, P = 0.021) but not percent changes in body weight (β: −0.02, P = 0.952) or prework USG (β: −53.19, P = 0.171) was an independent predictor of Δ[IGFBP7*TIMP-2]$_{USG}$ (Figure 1).

DISCUSSION

In partial support of our hypothesis, peak core temperature was an independent predictor of cross-shift changes in AKI risk (i.e., Δ[IGFBP7*TIMP-2]$_{USG}$) (Figure 1) and Δ[IGFBP7*TIMP-2]$_{USG}$ was higher when peak core temperature exceeded 38.0 °C, although only 41% of the workers had a peak core temperature that exceeded 38.0 °C. Nevertheless, markers of before- (prework USG) and during- (percent changes in body mass) work markers of hydration status were not found to be significant predictors of Δ[IGFBP7*TIMP-2]$_{USG}$



c

Multiple Linear Regression	Estimate	Standard Error	p-value
(Intercept)	-34.891	52.749	0.513
Peak Core Temperature	2.367	0.967	0.021
% Δ Nude Body Mass	-0.018	0.290	0.951
Pre-Shift USG	-53.187	37.92	0.171
n	34		
R^2	0.223		
AIC	120.126		

Figure 1. (a) Spearman rank correlations for the change (Δ) in $[IGFBP7*TIMP-2]_{USG}$ versus peak core temperature, preshift USG, and percent (%) Δ in nude body mass. (b) Nonparametric Mann-Whitney tests were used to compare $Δ[IGFBP7*TIMP-2]_{USG}$ values when peak core temperature is > or < 38 °C, preshift USG is > or < 1.020, and % Δ nude body mass is > or < 1.3%. (c) Multiple linear regression was performed on $Δ[IGFBP7*TIMP-2]_{USG}$ and peak core temperature, % Δ nude body mass, and preshift USG. One outlier was identified and excluded from the analysis. These data have been included in the figure, identified by a square symbol. Additional information regarding this outlier can be found in the Supplementary Material. IGFBP7, insulin-like growth factor-binding protein 7; TIMP-2, tissue inhibitor of metalloproteinases-2; USG, urine specific gravity.

(Figure 1). These results support previous findings from laboratory-simulated occupational heat stress[S3] and demonstrate that the development of hyperthermia plays an important role in the risk of developing AKI during occupational heat stress. This conclusion is supported by the fact that the development of hyperthermia is a direct result of occupational heat stress, which is a function of both environmental heat exposure and work intensity.[S1] However, the development of dehydration during occupational heat stress can be mitigated by drinking fluids to offset body water loss because of sweating. Indeed, cool water, sports drinks, and electrolyte-containing ice pops were freely available on the observed worksites. This may explain why measures of hydration status did not significantly predict cross-shift AKI risk.

Notably, 1 worker had an extreme AKI risk value and was excluded from the analysis but is visually

represented by the square symbol in Figure 1. The analysis including this worker (Supplementary Methods, Supplementary Tables S1–S3) potentially offers support for the association of changes in body weight (i.e., dehydration) with increased cross-shift AKI risk. Nevertheless, including this worker's data significantly impacted the model fit; and thus, the model could not be considered to have accurately represented the observed cohort with this worker's data included. However, this worker's data provides a unique case study illustrating the factors underlying cross-shift AKI risk in construction workers. This roofer arrived at work dehydrated (Preshift USG = 1.031) and performed very heavy work (average estimated metabolic rate 303 W/m^2) in a hot environment (average heat index = 30.3 °C). This worker had the highest recorded peak core temperature (38.6 °C), a large reduction in body mass (−2.8%), and a large increase in urinary [IGFBP7*TIMP-2] (111.1 $[ng/ml]^2$/1000). Thus, this unique observation illustrates the importance of occupational heat stress in cross-shift AKI risk and highlights the need for further observations in the construction sector.

This study is limited by the small sample size, the absence of controls for potential confounding variables such as diet and beverage intake throughout the workday, and the acute nature of the study. However, this was a pilot study that aimed to observe workers with minimal interruption to their workday. Future studies should increase the overall number of workers and involve a longer study period to identify if certain job types are at a differential risk for developing hyperthermia and AKI and incorporate blood-based measures used to clinically define AKI (e.g., serum creatinine). In addition, longitudinal measurements may provide insight into how the time of year impacts cross-shift AKI risk and whether cross-shift AKI could translate to changes in kidney function across the summer months.

In conclusion, peak core temperature is an independent predictor of cross-shift changes in AKI risk in construction workers in the US summer.

DISCLOSURE

All the authors declared no competing interests.

ACKNOWLEDGMENTS

This study was supported by CPWR (#22-5-PS to FA) and NIOSH (R01OH011528 to ZJS).

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Supplemental Methods.

Supplemental References.

Table S1. Participant and workday characteristics for construction workers, including all data.

Table S2. Spearman Rank correlations including all data.

Table S3. Normal multiple linear regression and robust linear regression, including all data.

REFERENCES

1. Heat at work: implications for safety and health. International Labour Organization; 2024. Accessed September 22, 2024. https://www.ilo.org/publications/heat-work-implications-safety-and-health
2. Sato Y, Roncal-Jimenez CA, Andres-Hernando A, et al. Increase of core temperature affected the progression of kidney injury by repeated heat stress exposure. *Am J Physiol Ren Physiol*. 2019;317:F1111–F1121. https://doi.org/10.1152/ajprenal.00259.2019
3. Hansson E, Jakobsson K, Glaser JR, et al. Association between acute kidney injury hospital visits and environmental heat stress at a Nicaraguan sugarcane plantation. *Workplace Health Saf*. 2024;72:131–142. https://doi.org/10.1177/21650799241235410
4. Chapman CL, Johnson BD, Vargas NT, Hostler D, Parker MD, Schlader ZJ. Both hyperthermia and dehydration during physical work in the heat contribute to the risk of acute kidney injury. *J Appl Physiol (1985)*. 2020;128:715–728. https://doi.org/10.1152/japplphysiol.00787.2019
5. Moyce S, Mitchell D, Armitage T, Tancredi D, Joseph J, Schenker M. Heat strain, volume depletion and kidney function in California agricultural workers. *Occup Environ Med*. 2017;74:402–409. https://doi.org/10.1136/oemed-2016-103848
6. Shi DS, Weaver VM, Hodgson MJ, Tustin AW. Hospitalised heat-related acute kidney injury in indoor and outdoor workers in the USA. *Occup Environ Med*. 2022;79:184–191. https://doi.org/10.1136/oemed-2021-107933
7. Al-Bouwarthan M, Quinn MM, Kriebel D, Wegman DH. Risk of kidney injury among construction workers exposed to heat stress: A longitudinal study from Saudi Arabia. *Int J Environ Res Public Health*. 2020;17:3775. https://doi.org/10.3390/ijerph17113775
8. National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard: Occupational Exposure to Heat and Hot Environments - Revised Criteria 2016*. Centers for Disease Control and Prevention; 2016. US Department of Health and Human Services. Publication No. 2016-106. Accessed June 20, 2025. https://www.cdc.gov/niosh/docs/2016-106/default.html
9. Vijayan A, Faubel S, Askenazi DJ, et al. Clinical use of the urine biomarker [TIMP-2] × [IGFBP7] for acute kidney injury risk assessment. *Am J Kidney Dis Off J Natl Kidney Found*. 2016;68:19–28. https://doi.org/10.1053/j.ajkd.2015.12.033