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The Heat Penalty: Temperature and Injury Risk in Professional Football

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Abstract

We study how ambient temperature affects worker injury incidence using football data from the Italian Serie A. This setting links high-frequency weather observations to precisely timed and located work episodes and injuries. Identification relies on within-team-season variation in kick-off temperature. We find that a one-degree increase in temperature raises the number of muscular/overuse injuries by 1.9%, while traumatic injuries are unaffected, suggesting that heat acts primarily through internal physiological stress. We detect suggestive evidence of larger temperature effects when effort incentives are stronger and among midfielders, whose role involves substantial physical demands. Exposure to high temperatures in the days preceding a match is associated with lower injury risk, consistent with short-term adaptation. These findings show how precisely measured environmental exposure can help identify occupational injury risks in physically demanding work settings and inform targeted prevention policies.

JEL classification

J28, J81, Q51, Q54, Z20, Z22

Keywords

workplace safety, high-resolution gridded data, temperature, professional football

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1 Introduction

Global warming represents one of the most pressing challenges of the twenty-first century, with significant implications for society and living standards. Analysing the costs associated with weather conditions, together with the benefits of mitigation and adaptation policies, is crucial from both a public health perspective and for future economic and labor market outcomes, particularly in light of the steady increase in global temperatures.

As a result, an expanding body of research has sought to quantify the causal effects of rising temperatures on economic activity, labor productivity, and health outcomes. Dell et al. (2014) provide a comprehensive review of the emerging climate-economy literature, documenting recent contributions that analyse the effects of extreme temperatures on economic growth, labor market outcomes, and health. Existing evidence shows that exposure to high temperatures reduces worker productivity and labor supply (Graff Zivin and Neidell, 2014; Adhvaryu et al., 2020; Somanathan et al., 2021; Belloc et al., 2026; Gibney et al., 2026). However, much of the empirical literature on extreme temperature exposure has primarily focused on health outcomes such as mortality, hospitalization rates, and low birth weight (see, e.g., Deschênes and Moretti, 2009; Deschênes et al., 2009; Deschênes and Greenstone, 2011; Masiero et al., 2022; Robben et al., 2026).

As for the role of weather and environmental conditions in workplace safety, a growing strand of the literature has recently begun to investigate the impact of climatic conditions on work-related injuries, as well as the ability of workers and firms to adapt to rising temperatures. The studies that identify the causal effect of temperature on workplace accidents focus on the United States (Dillender, 2021; Park et al., 2021), Australia (Ireland et al., 2023), China (Yao and Cao, 2025), Italy (Marinaccio et al., 2019; Filomena and Picchio, 2024), and Switzerland (Drescher and Janzen, 2025). These studies consistently find that extreme heat increases the likelihood of workplace injuries.

Despite these advances, existing research often provides limited insight into heterogeneous effects across workplaces, worker demographics, and job tasks. For instance, outdoor and manual employees, especially in work environments with poor or costly access to adaptation technologies or where occupational safety regulations may be less stringent, are likely to face disproportionately higher risks compared to indoor and sedentary workers. Understanding these nuances is crucial not only for improving workplace safety but also for informing climate adaptation policies and labor regulations. Identifying the mechanisms through which high temperatures increase occupational injury risk – whether physiological, cognitive, or behavioural – can guide targeted interventions.

By bridging these gaps, our paper contributes to this latter strand of the literature by

estimating the causal effect of ambient temperature on work-related injuries among high-skilled workers in a dynamic and physically demanding labor market, namely the Italian Serie A. In this industry, the overall incidence of injuries is substantially higher than in other industrial occupations generally regarded as high-risk (Hawkins and Fuller, 1999). Moreover, this setting allows us to focus on outdoor workplaces, where workers are directly exposed to weather conditions and have limited opportunities to mitigate their effects. We use hourly data on local temperature, precipitation, wind speed, and air pollution concentrations merged with match-level observations from the Italian Serie A and players' injury histories, covering the period from January 2013 to June 2023.

The use of detailed sports data is particularly valuable for addressing economics research questions (Kahn, 2000; Palacios-Huerta, 2025), as it helps overcome several empirical challenges that complicate the study of environmental risks in conventional labor market settings. In our case, professional football provides a data-rich setting in which workers perform a well-defined and highly comparable task at precisely known locations and times. This allows us to assign environmental exposure to individual work episodes with high spatial and temporal precision. Moreover, elite athletes represent a clean laboratory for studying injury risk under workplace environmental stress like temperature, providing a favorable source of identifying variation. For instance, match scheduling is determined in advance of the season under multiple constraints unrelated to realized weather and environmental conditions and is beyond the control of teams and players. As a result, players' exposure to heat is plausibly exogenous across stadiums and over time. This feature allows us to mitigate endogeneity concerns arising in conventional labor settings from residential sorting and behavioural responses aimed at avoiding exposure to extreme temperatures.

The second main contribution of our analysis lies in leveraging sports data to shed light on the specific types of physical problems and injuries experienced by football players, as well as on the mechanisms through which temperature affects occupational health using match-level statistics. The underlying channels include, among others, the possibility that workers exposed to higher temperatures experience physical and psychological strain, such as increased neuromuscular fatigue and energy expenditure, reduced coordination, dehydration, and altered muscle stiffness or tendon load tolerance. These factors may, in turn, lead to muscle cramps and a higher risk of musculoskeletal and overuse injuries. At the same time, heat exposure may impair cognitive performance and concentration, alter decision-making and situational awareness, and delay reaction times. These effects can increase the likelihood of errors on the pitch, such as mistimed or impulsive tackles, and raise the risk of harmful collisions, thereby increasing the probability of traumatic injuries.

A third contribution of our analysis lies in exploiting detailed player-, team-, and match-level characteristics to conduct heterogeneity analyses that allow us to distinguish how the effect of temperature on workplace injuries may vary across several dimensions. Specifically, we estimate heterogeneous effects along five margins. First, we compare home and away matches to assess the role of effort, pressure, incentives, and match intensity. Similarly, we test whether different stadium attendance, competitive balance, and season rounds present heterogeneous temperature patterns. Second, we test whether context-specific factors may shape the temperature-injury relationship accounting for congested match schedules, organizational differences, and habituation to local climate. Third, we examine differences by playing position to capture variation in job tasks, exposure to risk, and physical strain. Fourth, we analyse heterogeneity by age to assess whether younger players cope better with high temperatures or, alternatively, whether their relative lack of experience limits their ability to adjust effort to ambient conditions. Lastly, we consider injury severity to check whether heat exposure primarily affects the incidence of minor conditions or also increases the likelihood of more serious injuries with longer recovery times.

As a further contribution, we address a gap in the literature on the health effects of heat exposure within a sports framework characterised by substantial physical effort and stress, while also requiring high levels of strategy and sustained concentration. Football is the most popular sport worldwide, with more than 265 million professional, amateur, and youth players globally, according to the FIFA Big Count (FIFA, 2007).[1] From an economic perspective, professional football is a multi-billion-dollar industry involving clubs, leagues, players, supporters, and sponsors. In this context, severe injuries are negative productivity shocks and lead to losses in player availability, reduced team performance, and significant financial costs related to wages and transfer values. Our findings can therefore inform preventive measures, training and work schedules, and match regulations aimed at adapting outdoor sports to a warming climate and ensuring their long-term sustainability, potentially benefiting millions of athletes. Moreover, our results may have external validity beyond football and professional sports. For instance, Spector et al. (2019) show that workers in occupations characterised by high injury risk or complex tasks are also particularly exposed to heat stress, with impaired balance, changes in safety behaviour, muscle fatigue, dehydration, and sleep disruption as potential mechanisms.

Only a few studies provide evidence on the causal effects of ambient temperature on economic outcomes using sports settings. Using data from Major League Baseball (MLB),

[1] No updated census has been published since the FIFA Big Count 2006, the most recent comprehensive global estimate systematically including non-professional and informal football participants.

it has been shown that an increase in game-time temperature reduces umpire accuracy (Fesselmeyer, 2021) and influences players' behaviour by making them more aggressive (Larrick et al., 2011). Picchio and van Ours (2024), using tennis data, analyse the effect of ambient temperature on player performance. They find that higher temperatures reduce first-serve success rates and increase double fault rates, particularly among older players. Using a sample of Fitbit users, Harris (2026) finds that individuals run significantly slower on hot days and shift physical activity within and across days toward more moderate temperatures. Focusing on environmental conditions other than temperature, Lichter et al. (2017) examine the role of air pollution on football players' performance. Their results show statistically significant negative effects of air pollution on productivity, measured by the total number of passes per match. Similarly, Archsmith et al. (2018) find that MLB umpires make more incorrect calls following short-term exposure to pollutants like carbon monoxide or particulate matter, while Humphreys and Ruseski (2023) reveal different effects of air quality on NFL players according to their roles and objectives in team production. Regarding injuries, Burke et al. (2023) show that the probability of retirement in tennis increases by 2.1% per degree Celsius, while Picchio and van Ours (2025) document heterogeneity in the temperature effect among professional male tennis players on the ATP Tour, driven by incentive structures linked to the importance of specific sets. To the best of our knowledge, we are the first to study the occupational health consequences of ambient temperature using professional football data.

This article is organized as follows. Section 2 focuses on data, sample, and descriptive statistics. Section 3 presents the methodology to identify the temperature effect on injuries. Section 4 reports the estimation results and comments on the main findings, along with heterogeneity analysis and sensitivity checks. Section 5 concludes.

2 Data and Sample

2.1 Meteorological and Atmosphere Data

The empirical analysis is conducted on the basis of meteorological and atmosphere data merged with football records. We gathered meteorological and atmosphere data from the Copernicus Climate Change Service and Copernicus Atmosphere Monitoring Service, respectively. More in detail, the meteorological data come from the ERA5-Land, an hourly gridded reanalysis dataset providing a consistent view of the evolution of land variables over

several decades and with a global horizontal coverage.[2] For air quality variables, we used the CAMS air quality reanalyses, which provide air quality data starting from 1 January 2013.[3] We download, at a horizontal grid resolution of 0.1° and hourly temporal resolution, temperature and dew point temperatures 2 meters above the surface, wind speed 10 meters above the surface, total precipitation amount, and ozone and PM2.5 at ground level, from 1 January 2013 to 30 June 2023. The high spatial and temporal resolution of these data allows us to assign environmental conditions to each match based on its specific location and kick-off time, thereby closely approximating the conditions to which players are exposed while performing their work.

2.2 Football Data

We collect football data for Italian Serie A using the `worldfootballR` package,[4] an R package designed to extract various world football results and player statistics from popular football data sites, like Transfermarkt.com (Transfermarkt; Hamburg; Germany) and FotMob.com (FotMob AS; Bergen; Norway). From FotMob.com, we gather information on the geolocation of the stadium where each match was played, the teams involved, and the kick-off date and time of all matches across the seasons from 2012/2013 to 2022/2023.[5]

Over the same period, we obtain from Transfermarkt.com data on all injuries and the characteristics of the injured players. For each injury, we observe the name of the injured player, the diagnosis date, the type of injury (240 different injury types), and its duration. We link injury information with players' bios, containing characteristics like birthdate, from which we derive the player's age at the time of injury, the position on the pitch, and the citizenship. Transfermarkt.com is one of the most popular online databases on professional football players and the only one providing information on injuries per player over time. However, although already used in sports medicine studies (see, e.g., Leventer et al., 2016; Niederer et al., 2018; Schiffner et al., 2018; Della Villa et al., 2020; Grassi et al., 2020; Prieto-Lage et al., 2020), it is an open-collaborated database updated by editors and volunteers. For the section related to injury history, this leads to possible limitations such as

[2]For more details, see https://cds.climate.copernicus.eu/datasets/reanalysis-era5-land (last accessed on March 9th, 2026).

[3]For more details, see https://ads.atmosphere.copernicus.eu/datasets/cams-europe-air-quality-reanalyses (last accessed on March 9th, 2026).

[4]The `worldfootballR` package has been developed by Jason Zivkovic and archived in GitHub at https://github.com/JaseZiv/worldfootballR (last accessed on March 10, 2026).

[5]We downloaded data from FotMob.com in late spring 2023. Following a change in the platform's terms of service in September 2023 (see https://www.fotmob.com/tos.txt), the `worldfootballR` package is no longer able to extract data from the website.

incomplete reporting, missing injury details, and dependency on an active user community and media coverage that may vary between countries, clubs, and years. Its citizen science-based nature is put under scrutiny by Hoenig et al. (2022), who find that minor and moderate injuries are under-reported, whereas severe injuries and injury locations are in general consistent with other collection methods, such as injury data from large insurance databases. We exploit available features from the injury records to take into account the severity dimension and reduce concerns about our results being driven by under-reporting. Furthermore, we apply selection criteria to remove from the sample reporting errors and injuries unrelated to football matches.

Starting from 10,136 injury episodes, we remove 2,634 records that are not directly related to playing activity, such as influenza, depression, stomachache, or traffic accidents. We then apply a conservative procedure to identify injuries related to a Serie A football match. Specifically, we retain an injury only if its diagnosis is recorded on the day a match is played or within the following two days. Injuries that do not satisfy these criteria are not included in our match-level injury measure, as we cannot confidently attribute them to a Serie A match. These episodes include injuries that took place during training sessions or matches of other competitions (e.g., European cups, Coppa Italia, or national-team matches). We then obtain a sample composed by 3,325 records.

The attribution based on the match played on the day of injury diagnosis or within two days preceding the injury diagnosis is grounded on two rationales. First, physiologically, muscle and soft-tissue injuries or soreness as a consequence of intense physical exertion are sometimes characterized by a delayed onset of symptoms, including functional impairment, beginning 24 to 48 hours after the triggering event (MacIntyre et al., 1995; Cheung et al., 2003; Drayton et al., 2025). Second, the formal diagnosis of injuries in professional football clubs typically follows post-match medical evaluations, including physical examinations and imaging, which are routinely conducted after the match. For example, in professional sport, definitive diagnosis is generally established after imaging (magnetic resonance imaging or ultrasound), typically performed within 24-72 hours after the injury event, as scanning too early may underestimate the injury due to hyper-reflective blood components (Palermi et al., 2025; Di Pietto et al., 2025). The two-day window therefore captures the biological delay in symptom onset, accounts for the administrative lag inherent in professional sports injury reporting systems, and does not overlap with the subsequent match, as there are at least two days of rest before the next match.[6]

[6]The robustness of our results to restricting the attribution window only to the match day and the day after is discussed in Subsection 4.4.

As a next step, we remove 233 observations stemming from reporting errors, such as repeated injury episodes for the same player in the same team within the same match, or duplicate records for the same player registered under different teams.[7] We then drop a further 997 observations related to youth team players not participating in the Italian Serie A, as well as records referring to injured players who did not actually take part in the match.[8]

After applying these selection criteria, the final sample comprises 2,095 injury episodes. By focusing on injuries sustained during Serie A matches, which are less likely to go unnoticed by the Transfermarkt.com user community, we mitigate the under-reporting of minor or moderate injuries documented by Hoenig et al. (2022). Moreover, we assess injury severity by distinguishing between mild/moderate and severe injuries based on their duration, using a 28-day threshold (Fuller et al., 2006; Ekstrand et al., 2012). As shown in Section 4, the main effect is driven by severe injuries, which alleviates concerns that our results are driven by the under-reporting of minor physical problems.

For each match and each team, we compute the number of injuries. Following the epidemiological classification introduced in the UEFA injury study model (Hägglund et al., 2005), we distinguish the injury count between *intrinsic* and *extrinsic* injuries. Intrinsic injuries – such as ligament, tendon, muscle, and bone-tendon junction injuries – arise from internal physiological mechanisms rather than external contact. Extrinsic injuries, instead, form a residual category including contusions, bruises, fractures, tendon ruptures, surgeries, and concussions.[9] This distinction is particularly relevant in our setting. There are strong physiological reasons to expect temperature to primarily affect intrinsic injuries: heat exposure increases metabolic fatigue and dehydration, which impair neuromuscular coordination and raise the risk of such injuries. By contrast, extrinsic injuries are more likely to result from external mechanical forces, making it less plausible that ambient temperature directly affects their occurrence. That said, indirect effects may still arise if temperature influences playing style and, consequently, the likelihood of traumatic contact between players.

2.3 Descriptive Statistics

After matching the football dataset with meteorological and air quality variables, we obtain 8,000 team-match observations from the 4,000 matches of Serie A played between January

[7] Such reporting errors are likely to occur in Transfermarkt.com data when players are transferred or loaned between clubs.

[8] For each match, we verify whether injured players were listed as starters or appeared during the game; otherwise, the corresponding records are excluded from the sample.

[9] See Tables A.1 and A.2 in Appendix A for a complete classification of intrinsic and extrinsic injuries in the dataset.

2013 and June 2023.[10] These matches involve 37 different teams. Table 1 reports summary statistics of the main outcome variables and the covariates used in the regression analysis. In total, the sample includes 2,022 injuries, of which 1,366 are intrinsic and 656 are extrinsic. The average number of injuries per team per match is about 0.253, with intrinsic injuries being more frequent than extrinsic ones (0.171 versus 0.082). The average temperature at kick-off is 13.6°C, ranging from −9.4°C in Atalanta-Lazio on 17 December 2017 to 34.6°C in Sassuolo-Genoa on 29 July 2020.

Table 1: Summary Statistics

	Mean	Std. Dev.	Min.	Max.
a) Outcome variables				
Number of injuries per team per match	0.253	0.530	0.000	4.000
Number of intrinsic injuries per team per match	0.171	0.433	0.000	4.000
Number of extrinsic injuries per team per match	0.082	0.289	0.000	2.000
b) Covariates[a]				
Temperature (°C)[b]	13.648	6.884	-9.400	34.607
Wind speed (m/s)[b]	1.987	1.414	0.017	11.387
Precipitation (mm)[b]	2.537	6.180	0.000	82.974
Ozone (μg/m^3)[b]	63.184	32.868	0.000	178.313
PM2.5 (μg/m^3)[b]	15.436	12.584	0.000	89.156
Match kick-off time (hour)[c]	16.812	2.915	12.000	21.000
Playing home	0.500	0.500	0.000	1.000
Post COVID-19 period (June and July 2020)	0.029	0.167	0.000	1.000
Number of observations		8,000		
# of matches		4,000		
# of different teams		37		
# of seasons		11		

[a] In the model specification, we also use month indicators, whose summary statistics are not reported for the sake of brevity.
[b] Meteorological and air pollution variables are measured at the match kick-off times expressed in UTC+2 and rounded to the nearest integer hour.
[c] Match kick-off times are expressed in UTC+2 and rounded to the nearest integer hour.

Figure B.1 in the appendix visualizes the unconditional relationship between temperature and injuries. The outcome variables are counts, as reported in Table 2, with support over non-negative integers ranging from 0 to 4 (and from 0 to 2 for extrinsic injuries). Accordingly, we estimate this relationship using a Poisson regression, where the dependent variable is the number of injuries per team per match (total, intrinsic, or extrinsic), modeled as a flexible function of temperature using a second-degree fractional polynomial (Royston and Altman, 1994). The preferred specification is selected based on the deviance, defined as twice the negative log-likelihood. Comparing the temperature profiles for total, intrinsic,

[10] We exclude matches played in 2012 because, as discussed in Subsection 2.1, the air quality variables obtained from the CAMS air quality reanalyses are available only from 1 January 2013 at the time of writing.

and extrinsic injuries reveals distinct patterns in the raw data. The incidence of intrinsic injuries increases monotonically with temperature (panel b of Figure B.1), with the expected number of injuries more than 20% higher at temperatures above 21°C relative to 0°C. In contrast, the relationship for extrinsic injuries is U-shaped (panel c), with extreme temperatures increasing their frequency by approximately 20% relative to average temperatures. These estimates are purely descriptive and do not account for potential confounding factors. The subsequent regression analysis addresses this limitation by introducing control variables and multiple sets of fixed effects (FE) to more rigorously isolate the effect of temperature.

Table 2: Distribution of Count Outcome Variables

	Total injuries		Intrinsic injuries		Extrinsic injuries	
Number of injuries per team per match	Absolute frequency	Relative frequency	Absolute frequency	Relative frequency	Absolute frequency	Relative frequency
0	6,306	0.7883	6,801	0.8501	7,377	0.9221
1	1,404	0.1755	1,048	0.1310	590	0.0738
2	256	0.0320	137	0.0171	33	0.0041
3	30	0.0038	12	0.0015	0	0.0000
4	4	0.0005	2	0.0003	0	0.0000
Total	8,000	1.0000	8,000	1.0000	8,000	1.0000

3 Estimation Strategy

We estimate the effect of temperature on the number of injuries per team per match using a Poisson quasi-maximum likelihood model.[11] Specifically, we estimate:

$$\mathbb{E}[y_{imt} \mid temp_{imt}, \mathbf{x}_{imt}, \gamma_{it}, \delta_{ot}, \eta_s] = \exp\left(\alpha \cdot temp_{imt} + \boldsymbol{\beta}'\mathbf{x}_{imt} + \gamma_{it} + \delta_{ot} + \eta_s\right), \quad (1)$$

where y_{imt} denotes the number of injuries suffered by team i in match m during season t, and $temp_{imt}$ is the ambient temperature at kick-off time. The vector $\mathbf{x}_{imt}$ includes wind

[11] In estimating the main equation, we maximize the Poisson log-likelihood function, up to an additive constant, $\ell(\theta) = \sum_{i,m,t} [y_{imt} \log \mu_{imt}(\theta) - \mu_{imt}(\theta)]$, where $\mu_{imt}(\theta)$ denotes the conditional mean specified in Equation (1), and θ collects the regression coefficients and FE. Although this objective function corresponds to the Poisson log-likelihood, Poisson quasi-maximum likelihood estimation does not require the conditional distribution of injuries to be Poisson. Consistency of the estimated coefficients requires the conditional mean to be correctly specified, together with standard identification and regularity conditions. A violation of the equidispersion restriction, namely the equivalence between the conditional mean and the conditional variance which characterizes the Poisson distribution, does not, by itself, bias the estimated coefficients. However, it affects statistical inference. We account for this by using cluster-robust standard errors that allow for heteroskedasticity and within-cluster dependence.

speed, precipitation, ozone, and PM2.5 (all measured at kick-off), as well as month FE, kick-off hour, a home indicator, and a dummy for the post-COVID-19 period (June-July 2020). The terms γ_{it}, δ_{ot}, and η_s denote team-season, opponent-season, and stadium FE, respectively. The parameter of interest, α, captures the multiplicative relation between kick-off temperature and the conditional expected number of injuries per team-match. The exponential conditional mean specification implies that a one-unit increase in temperature changes expected injury counts by a constant proportion, holding other covariates and FE constant. Consequently, the same proportional effect can translate into different absolute changes in expected injury counts across team-matches, depending on their baseline expected injury counts.

The inclusion of team-season FE implies that identification relies on within-team-season variation in match-day temperature, comparing matches played by the same team within the same season under different thermal conditions, while holding constant opponent-season, stadium, month, and other environmental factors. More specifically, the temperature coefficient is identified from short-run variation in kick-off temperature that remains after accounting for the FE and kick-off-time controls included in our preferred specification.[12] The key identifying assumption is that, conditional on the included controls and FE, short-run fluctuations in kick-off temperature are orthogonal to unobserved determinants of injury risk.

Weather and air quality variables – such as wind speed, precipitation, and pollutants – are included because they may be jointly determined with temperature and independently affect injury risk through playing conditions. Controlling for kick-off hour captures systematic diurnal variation in temperature and match conditions, as well as potential time-of-day effects on player performance. The post-COVID-19 dummy captures an atypical period with summer matches, schedule congestion, and temporary rule changes, all of which may have affected injury risk. Stadium FE absorb time-invariant location characteristics (e.g., infrastructure, pitch quality, and local climate), while month FE capture common seasonal patterns. Team-season FE control for all observed and unobserved team-specific factors that remain constant within a season, and opponent-season FE account for persistent differences in opponent strength, intensity, and style of play within each season. Together, these FE

[12] Substantial variation in match-day temperature remains after accounting for the FE and kick-off-time controls included in our specification. The standard deviation of temperature is 6.89°C in the raw data and 2.75°C after residualizing temperature with respect to team-season, opponent-season, month, and stadium FE and the kick-off-time controls used in our baseline specification. Moreover, when we flexibly absorb kick-off-hour FE in panel c of Table B.3, the residual standard deviation remains 2.74°C (with an interquartile range of 3.63°C), indicating that the identifying variation is not primarily driven by systematic temperature differences across kick-off hours.

ensure that identification comes from deviations in temperature across matches that are not driven by persistent team, opponent, or location characteristics.

For inference, we employ the two-way cluster-robust variance estimator proposed by Cameron et al. (2011), clustering at the team-season (TS) and calendar-week (W) levels. Let $\widehat{H}$ denote the negative Hessian of the Poisson quasi-log-likelihood with respect to the estimated coefficients, after partialling out the absorbed FE. Let $\widehat{B}_{TS}$, $\widehat{B}_{W}$, and $\widehat{B}_{TS\cap W}$ denote the sums of the outer products of cluster-level scores, aggregated over the TS clusters, the W clusters and their intersection cells, respectively. Following Cameron et al. (2011) the covariance matrix is $\widehat{H}^{-1}\left(\widehat{B}_{TS}+\widehat{B}_{W}-\widehat{B}_{TS\cap W}\right)\widehat{H}^{-1}$, where the intersection term removes the double-counting of observations belonging to both clustering dimensions. The number of clusters is sufficiently large along both dimensions: in the baseline specification for total injuries, there are 219 team-season clusters and 363 calendar-week clusters. This estimator allows for heteroskedasticity, arbitrary serial correlation within team-seasons, and arbitrary cross-sectional dependence across observations within the same calendar week. The latter is plausible because temperature shocks (e.g., heatwaves or cold spells) affect large geographic areas, generating correlation across matches played in the same week. Moreover, spatial correlation in temperature is likely reflected in our gridded climate data, which are constructed via spatial interpolation from station-level observations (Hsiang, 2016). Serial correlation within team-seasons is also plausible, as injury incidence may persist over time due to factors such as underlying fitness, medical management, training intensity, tactical style, and squad rotation policies. Finally, in presenting the main and heterogeneity results, we also report the step-down adjusted p-values following Romano and Wolf (2005a,b). These, also clustered at the team-season and week levels, are robust to multiple hypothesis testing by accounting for the familywise error rate.

4 Results

4.1 Main Findings

Table 3 reports the estimation results of Equation (1). The dependent variable is the number of injuries per team per match in column (1), and is then decomposed into intrinsic and extrinsic injuries in columns (2) and (3).

Considering total injuries, we find no statistically significant effect of temperature: a one-degree Celsius increase generates an increase of approximately 1% in the number of injuries, but the estimate is not significant at the conventional levels. However, once we

distinguish between intrinsic and extrinsic injuries, a clear heterogeneity emerges. A one-degree increase in temperature leads to a statistically significant rise of 1.9% in intrinsic injuries, while the effect on extrinsic injuries is small and not statistically significant (-0.6%).

To assess the economic magnitude of the estimated effect, we consider an interquartile range increase in match-day temperature. Given our estimated coefficient of 0.0192, moving from the 25th to the 75th percentile of the temperature distribution is associated with an approximately 21.7% increase in the expected number of intrinsic injuries. This suggests that temperature variation within the range commonly observed in our sample has quantitatively meaningful effects on injury risk.

These findings are consistent with a mechanism-based interpretation of the temperature effect. The significant and economically meaningful impact on intrinsic injuries suggests that temperature operates through internal physiological stress. Higher temperatures increase dehydration and thermoregulatory strain, accelerate metabolic fatigue, impair neuromuscular control, and contribute to electrolyte imbalances associated with muscle cramps and soft-tissue injuries (Schwellnus, 2009; Nichols, 2014; Casa et al., 2015; King and Baker, 2020; Périard et al., 2021).

By contrast, extrinsic injuries are primarily driven by external mechanical factors – such as collisions, tackles, and accidental contact – and are therefore less directly affected by ambient temperature. The absence of a significant effect suggests that any temperature-induced changes in playing behaviour or aggression are not large enough to translate into a detectable increase in extrinsic injury risk.

Regarding other covariates, we find that wind speed and playing at home are strong predictors of injuries. A one meter-per-second increase in wind speed is associated with a 3.9% increase in injuries, driven in particular by intrinsic injuries (+4.4%). Playing at home is associated with a 10.6% increase in total injuries,[13] and a 15.1% increase in intrinsic injuries,[14] while no statistically significant effect is found for extrinsic injuries.

A possible explanation for these findings is that wind speed and playing at home are associated with injuries primarily through physiological rather than contact-related mechanisms. Soccer ball trajectories are known to be sensitive to aerodynamic instabilities, whereby even small perturbations in airflow can generate irregular and difficult-to-predict flight paths (Ito et al., 2012). In this context, higher wind speeds may further increase trajectory variability, potentially leading to more frequent misjudgements, delayed reactions,

[13] $\exp(0.1007) - 1 = 0.1059$.
[14] $\exp(0.1409) - 1 = 0.1513$.

Table 3: Estimation Results of Injury Equation (1)

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
Temperature (°C)	0.0098	0.0192**	-0.0060
	(0.0087)	(0.0104)	(0.0147)
	[0.2525]	[0.0425]	[0.5945]
Further covariates			
Wind speed (m/s)	0.0388**	0.0439*	0.0236
	(0.0201)	(0.0223)	(0.0363)
Precipitation (mm)	-0.0077*	-0.0073	-0.0102
	(0.0049)	(0.0055)	(0.0118)
Ozone (μg/m^3)	-0.0013	-0.0020	-0.0001
	(0.0015)	(0.0017)	(0.0023)
PM2.5 (μg/m^3)	-0.0024	-0.0036	-0.0007
	(0.0028)	(0.0032)	(0.0040)
Match kick-off time (hour)	-0.0073	-0.0012	-0.0162
	(0.0114)	(0.0144)	(0.0159)
Playing home	0.1007**	0.1409**	0.0278
	(0.0429)	(0.0525)	(0.0752)
Post COVID-19 period (June and July 2020)	0.0750	0.2791	-0.3934
	(0.2156)	(0.2980)	(0.3102)
# of observations	7,961	7,754	6,682
# of team-season	219	214	195
Log-likelihood	-4,552.2	-3,442.9	-1,972.8
Pseudo R^2	0.0963	0.1082	0.1038

* p-value $<$0.10, ** p-value $<$0.05, *** p-value $<$0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), which are robust to multiple hypothesis testing for the significance of the temperature coefficient across models (1), (2), and (3), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include team-season, opponent-season, stadium, and month FE. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation.

and irregular movement patterns, thereby raising the risk of biomechanical instability and intrinsic injuries.

The higher injury risk in home matches may be explained by the fact that home teams typically play at higher intensity and exert greater physical effort (Carmichael and Thomas, 2005),[15] potentially driven by crowd support (Dawson et al., 2007; Philipson, 2026) and more offensive and structured tactical choices (Staufenbiel et al., 2015). This may lead to overexertion and fatigue-related injuries. Moreover, performing in front of a supportive crowd may increase motivation, but excessive pressure can impair performance in skill-based tasks (Harb-Wu and Krumer, 2019).[16] This, in turn, may result in suboptimal decisions during the match, potentially increasing injury risk.

In our baseline specification, we impose a linear functional form on the relationship between temperature and injuries. Figure 1 relaxes this assumption by allowing for non-linearity through a fractional polynomial specification. More specifically, we estimate a Poisson regression model for the number of injuries per team per match as a function of a second-degree fractional polynomial in temperature, alongside the full set of covariates and FE included in the benchmark model.[17] The figure shows that the relationship between temperature and extrinsic injuries is essentially flat and very well approximated by a linear specification.[18] By contrast, the relationship between temperature and intrinsic injuries is increasing and exhibits mild concavity, indicating slightly decreasing marginal effects at higher temperatures. However, this deviation from linearity is limited in magnitude, and the linear specification cannot be rejected against the best-fitting fractional polynomial model at conventional significance levels (p-value $= 0.070$).[19]

The support for a linear relationship, along with the lack of evidence for a convex one, may reflect the interaction of two opposing mechanisms. On the one hand, as temperature rises, injuries may become increasingly likely, conditional on effort. On the other hand,

[15] According to Carmichael and Thomas (2005), playing at home significantly boosts individual performance, especially in offensive metrics, outperforming away teams on several non-outcome measures such as shots on target, corners, and possession.

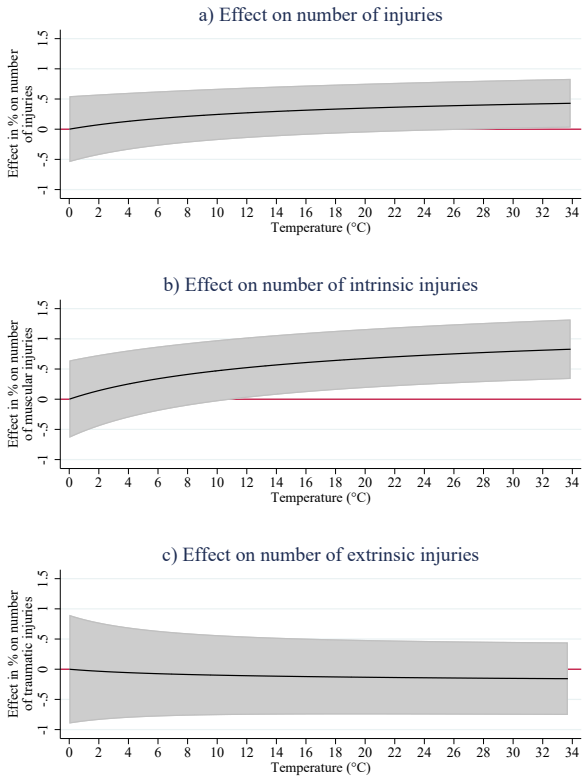
[16] The phenomenon whereby excessive pressure impairs performance is commonly referred to as "chocking under pressure" (Baumeister, 1984).

[17] The powers are selected from the set $\{-2, -1, -0.5, 0, 0.5, 1, 2, 3\}$. Model selection is based on the deviance, defined as twice the negative log-likelihood, with the pair of powers that minimizes the deviance chosen as the preferred specification. We use the Stata command `fp` combined with `ppmlhdfe` for the high-dimensional FE estimation of the Poisson quasi-maximum likelihood model.

[18] For extrinsic injuries, we cannot reject the linear model against the best-fitting fractional polynomial model, with a p-value equal to 0.296.

[19] Using a third-degree fractional polynomial in temperature to allow for greater flexibility, we likewise cannot reject the linear specification against the best-fitting model (p-values of 0.490, 0.091, and 0.510 for total, intrinsic, and extrinsic injuries, respectively).

Figure 1: Fractional Polynomial Temperature Effect



Notes: The estimates reported in these graphs are obtained by fitting a Poisson regression model for the number of injuries per team per match as a function of a degree-2 fractional polynomial in temperature and all the covariates and FE used in the benchmark model. The temperature effect is normalized to be 0 at 0°C. The powers are selected from the set $\{-2, -1, -0.5, 0, 0.5, 1, 2, 3\}$. Model selection is based on the deviance, defined as twice the negative log-likelihood, with the pair of powers that minimizes the deviance chosen as the preferred specification. In all the graphs, the preferred fractional polynomial specification is $(-2, -0.5)$. The gray areas represent 95% confidence intervals computed using heteroskedasticity-robust standard errors clustered at the team-season level. The tests of the linear model against the best-fitting fractional polynomial specification yield the following p-values: 0.320 in panel a, 0.070 in panel b, and 0.296 in panel c.

higher temperatures may induce players to exert less effort, thereby reducing injury risk. This interpretation also suggests that the temperature gradient may vary with the average level of effort players are expected to exert during a match, an issue we explore in the next Subsections. More in detail, in the next Subsections we investigate: match-level heterogeneity driven by differences in incentives and game intensity (Subsection 4.2.1); team-level heterogeneity driven by differences in cumulative fatigue, team organization, and climatic adaptation (Subsection 4.2.2); and player-level heterogeneity associated with different physiological and risk profiles (Subsection 4.2.3).

In what follows, in light of the evidence presented in Figure 1 and given that we cannot reject the linear specification in favor of a more flexible alternative, we retain the linear specification for the temperature effect and report only the corresponding coefficient estimates.

4.2 Effect Heterogeneity

4.2.1 Match-Level Heterogeneity: Incentives and Game Intensity

We start by investigating heterogeneity in the estimated effect across several match-level dimensions related to effort, competitive balance, and incentives to shed light on how contextual factors may amplify or mitigate the impact of temperature on injury incidence.

In the previous Subsection, we showed that injuries, particularly intrinsic injuries, are more likely when teams play at home. We speculated that this pattern may be driven by crowd support and more offensive tactical choices, which could lead players to exert greater physical effort. If this mechanism is at work, one may also expect the effect of temperature to be amplified in home matches, as higher exertion could increase susceptibility to the internal physiological stress induced by heat. Panel a of Table 4 reports the estimated temperature effects separately for teams playing at home and away. Consistent with this hypothesis, the effect of temperature on intrinsic injuries is primarily driven by home teams: for these teams, a one-degree increase in temperature is associated with a statistically significant 2.4% increase in intrinsic injuries. However, although the point estimates suggest a much stronger effect for home teams, the difference between the temperature gradients for home and away teams is not statistically significant.

A supportive crowd is widely regarded as a key driver of home advantage, influencing effort, performance intensity, and game dominance.[20] Therefore, we investigate whether the

[20] Almeida and Leite (2021) and McCarrick et al. (2021) conclude that, in the absence of fans, home teams created significantly fewer attacking opportunities, as measured by the number of corners, shots, pass success, and game dominance. The absence of crowds also affects referee decisions, with fewer cards awarded to away teams (Bryson et al., 2021; Reade et al., 2022; Philipson, 2026).

Table 4: Effect Heterogeneity by Match-Level Characteristics

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
a) Playing home vs. away			
Temperature (°C) if playing home	0.0111	0.0235**	-0.0112
	[0.2574]	[0.0305]	[0.8486]
Temperature (°C) if playing away	0.0082	0.0142	-0.0006
	[0.4193]	[0.2084]	[0.9815]
F-test of significance of the difference, p-value:	[0.8166]	[0.2254]	[0.6987]
b) Open doors and 3 substitutions (before COVID-19) vs. open doors and 5 substitutions (season 2021/2022 onward) vs. close doors and 5 substitutions (from COVID-19 outbreak until May 2021)			
Temperature (°C) if open doors & 3 substitutions (1)	0.0148	0.0261**	-0.0052
	[0.1509]	[0.0140]	[0.9590]
Temperature (°C) if open doors & 5 substitutions (2)	0.0160	0.0257	-0.0042
	[0.2574]	[0.2159]	[0.8991]
Temperature (°C) if closed doors & 5 substitutions (3)	-0.0191	-0.0167	-0.0148
	[0.2574]	[0.1324]	[0.9815]
F-test of significance of difference between (3) and (1), p-value:	[0.0070]	[0.0010]	[0.8686]
F-test of significance of difference between (3) and (2), p-value:	[0.0160]	[0.0160]	[0.8686]
c) Stadium attendance			
Temperature (°C) if high stadium attendance	0.0143	0.0282**	-0.0110
	[0.1999]	[0.0105]	[0.8486]
Temperature (°C) if low stadium attendance	0.0074	0.0158	-0.0054
	[0.4313]	[0.1989]	[0.9590]
F-test of significance of the difference, p-value:	[0.5412]	[0.1684]	[0.8686]
d) Competitive balance			
Temperature (°C) if high competitive balance	0.0109	0.0228**	-0.0097
	[0.2574]	[0.0405]	[0.8511]
Temperature (°C) if low competitive balance	0.0089	0.0160	-0.0025
	[0.3968]	[0.1664]	[0.9815]
F-test of significance of the difference, p-value:	[0.8166]	[0.2299]	[0.7891]
e) First part vs. latter part of the season			
Temperature (°C) if 1st part of the season (August-January)	-0.0034	0.0075	-0.0238
	[0.6527]	[0.4053]	[0.3118]
Temperature (°C) if latter part of the season (February onwards)	0.0269**	0.0353**	0.0143
	[0.0145]	[0.0120]	[0.8191]
F-test of significance of the difference, p-value:	[0.0150]	[0.0915]	[0.1539]
# of observations	7,961	7,754	6,682
# of team-season	219	214	195

* p-value <0.10, ** p-value <0.05, *** p-value <0.01. For each model, in brackets we report the step-down adjusted p-values of Romano and Wolf (2005a,b), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. In panel b, we also include a dummy for closed-door matches with 5 substitutions (period from 06/2020 to May 2021) and open-door matches with 5 substitutions (from August 2021 onward). In panel c, we also include a dummy for stadium attendance above the median. In panel d, we also include a dummy for match competitiveness above the median. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation. In panel a, the number of observations which played at home is 3,982 in model (1), 3,878 in model (2), and 3,348 in model (3). In panel b, the number of observations if "open doors & 5 substitutions" is equal to 838 in models (1) and (2) and 760 in model (3); the number of observations if "closed doors & 5 substitutions" is equal to 1,024 in models (1) and (2) and 896 in model (3). In panel c, the number of observations with high (low) stadium attendance is 3,836 (3,850) in model (1), 3,749 (3,716) in model (2), and 3,338 (3,173) in model (3). The number of spectators is missing for 238 observations, which are not used in panel c. In panel d, the number of observations for which the match competitiveness is above the median is 3,982 in model (1), 3,858 in model (2), and 3,287 in model (3). In panel e, the number of observations for which the match was played in the first part of the season is 4,316 in model (1), 4,222 in model (2), and 3,658 in model (3).

effect of temperature differs across matches played: before the COVID-19 outbreak (up to March 2020), characterized by open stadiums and a maximum of three substitutions; during the COVID-19 pandemic (from July 2020 until May 2021), when matches were played behind closed doors with a maximum of five substitutions; and after the COVID-19 pandemic (from the 2021/2022 season onward), when spectators were readmitted while the five-substitution rule was maintained. By conducting this analysis, we extend the literature on crowd effects by focusing on a physiological outcome rather than performance metrics. Specifically, we examine whether crowd-induced effort, interacting with temperature, affects the incidence of intrinsic injuries. Additionally, we assess whether the five-substitution rule mitigates the adverse effects of heat on injuries by alleviating fatigue and physiological stress. Our findings in panel b of Table 4 indicate that, in matches played with open stadiums and the three-substitution rule, the effect of temperature on intrinsic injuries is significantly positive (+2.6%) and significantly larger than in matches played behind closed doors with the five-substitution rule. Notably, after stadiums reopened while maintaining the five-substitution rule, the point estimate of the temperature effect (+2.6%) returns to its pre-COVID level. This pattern suggests that the effort channel associated with crowd support is attenuated in empty stadiums and that expanding the substitution limit from three to five is not sufficient to offset the positive relationship between temperature and injuries.

To further explore these channels, we extend the analysis by interacting temperature with an indicator for high versus low stadium attendance (defined relative to the median, which is 17,528 spectators). This additional dimension provides a more granular proxy for the intensity of social pressure and effort incentives induced by crowd support. Consistent with the previous interpretation, the estimates in panel C of Table 4 reveal a substantially larger temperature effect in high-attendance matches, whereas the effect is not statistically significant when attendance is low. Although the difference between the two coefficients is not statistically distinguishable from zero, the sizable gap in point estimates suggests that the baseline temperature effect on intrinsic injuries is primarily driven by matches with larger crowds. This pattern reinforces the view that crowd-induced effort plays a key role in mediating the impact of temperature on injuries by shaping players' behavior and physiological strain.

Fourth, under the hypothesis that more evenly matched games require greater physical exertion, we examine whether the impact of temperature varies with the degree of competitive balance, as measured by probabilities implied by BET365 pre-match betting odds. Specifically, we construct an index of match competitiveness defined as one minus the absolute difference between the probabilities that the home and away teams win. Higher values

of this index correspond to more balanced matches. We then interact temperature with indicators for whether this index is above or below its median, which is 0.71. The results in panel d of Table 4 indicate that the temperature effect is larger in more balanced matches, which are expected to require greater physical exertion, push players closer to their physiological limits, and reduce opportunities for pacing. This pattern highlights a complementarity between environmental stress and competitive intensity. By contrast, in less balanced matches, where outcomes are more predictable and the pace of play may be less demanding, players may be better able to modulate effort, thereby attenuating the impact of temperature. Although the difference between the estimated effects is not statistically distinguishable from zero, the gap in point estimates suggests that the baseline temperature effect on intrinsic injuries is primarily driven by more balanced matches.

Finally, in panel e, we estimate heterogeneous effects of temperature by distinguishing between matches played in the first part of the season (August to January) and those played in the latter part (February onwards). This distinction allows us to account for dynamic physiological adaptation, fatigue accumulation, and changing incentives over the course of the season. Indeed, different phases of the season are likely characterized by varying physical conditions. According to Carrieri et al. (2020), the highest percentage of injuries occurs in September, the first month after the summer break, likely due to suboptimal fitness levels and residual effects of summer conditions. By contrast, players are more likely to reach peak conditioning during the middle of the season, benefiting from improved muscular adaptation, whereas late-season matches may reflect cumulative fatigue and a higher incidence of overuse injuries. At the same time, different stages of the season involve heterogeneous incentives, motivations, and playing styles, depending on teams' realistically attainable objectives. For instance, toward the end of the season, some teams compete to avoid relegation, qualify for European competitions, or win the league, while others positioned mid-table may have little at stake. These differences can affect players' attitudes, concentration, intensity, aggression, and, consequently, injury risk.[21] We find that the temperature effect is concentrated in, and essentially driven by, the latter part of the season. A one-degree increase in temperature is associated with a statistically significant 2.7% (3.5%) increase in (intrinsic) injuries. In contrast, injury risk is not affected by temperature in the first part of the season. Extrinsic injuries are never significantly affected, neither in the early nor in the later part of the season. This pattern suggests that, when fatigue accumulates and/or competitive stakes

[21] Consistent with this hypothesis, Paserman (2023) show that tennis players adjust their behaviour in high-pressure situations. Picchio and van Ours (2024) and Picchio and van Ours (2025) find that higher temperatures reduce performance and increase match retirements in professional tennis, although the temperature effect is attenuated when the stakes are higher.

are higher, temperature exacerbates underlying physiological stress, thereby increasing the risk of intrinsic injuries.

4.2.2 Team-Level Heterogeneity: Exposure, Organization, and Adaptation

We now examine heterogeneity in the temperature effect along team-level dimensions, including congested match scheduling, a proxy for team organization, and the climatic conditions of the team's location. This approach provides a more comprehensive assessment of how context-specific factors shape the temperature-injury relationship, accounting for short-run exposure to environmental stress, longer-run adaptation to local climate, and organizational differences in workload management, recovery constraints, and fatigue accumulation.

In panel a of Table 5, we begin by examining the interaction between temperature and congested match schedules, defined as an indicator equal to one if more than three matches were played in the previous 15 days across all domestic and European competitions (i.e., Serie A, Coppa Italia, Supercoppa Italiana, Champions League, Europa League, and Conference League). Fixture congestion may capture cumulative workload and fatigue, as well as recovery constraints, but it is also likely to reflect systematic differences across teams. In particular, teams facing more congested schedules are typically those that progress further in multiple competitions and are therefore, on average, stronger, deeper, and better organized. Our findings indicate that the adverse impact of heat is not statistically significant in matches played under high fixture congestion. A natural interpretation is that this pattern partly reflects the characteristics of teams exposed to congestion: deeper squads, more advanced conditioning and medical support, and greater experience in managing high-intensity schedules may mitigate the marginal impact of environmental stress on injury risk. In addition, players and teams may adjust behavior under sustained workload pressure – through pacing, effort reallocation, and intensity management – further attenuating the temperature effect. By contrast, the temperature effect is larger and statistically significant in matches with less congested schedules. These matches are more likely to involve teams with thinner squads or more limited organizational capacity, for whom environmental stress may translate more directly into physiological strain and injury risk. Taken together, these results suggest that both selection into congested schedules and endogenous behavioral responses play a role in shaping the relationship between temperature and injuries. However, two cautionary notes should be considered. First, from the statistical point of view, the temperature gradients are not different from each other, although the point estimates are quite different. Second, the estimates should be interpreted as capturing heterogeneity across different competitive and organizational environments, rather than the causal effect of congestion per se.

Table 5: Effect Heterogeneity by Team-Level Characteristics

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
a) Congestion of match scheduling			
Temperature (°C) if congested calendar	0.0058	0.0130	-0.0038
	[0.6042]	[0.2509]	[0.9745]
Temperature (°C) if not congested calendar	0.0116	0.0230**	-0.0090
	[0.2819]	[0.0320]	[0.8441]
F-test of significance of the difference, p-value:	[0.7131]	[0.3853]	[0.8131]
b) Teams in top 5 urban areas vs. other teams			
Temperature (°C) if team in top 5 urban areas	0.0076	0.0147	-0.0045
	[0.4673]	[0.1099]	[0.9745]
Temperature (°C) if team not in top 5 urban areas	0.0119	0.0234**	-0.0077
	[0.2869]	[0.0435]	[0.8936]
F-test of significance of difference, p-value:	[0.7131]	[0.3853]	[0.8131]
c) Teams tangible assets			
Temperature (°C) if team with high tangible assets	0.0008	0.0062	-0.0058
	[0.9135]	[0.4988]	[0.9685]
Temperature (°C) if team with low tangible assets	0.0189*	0.0291**	0.0032
	[0.0750]	[0.0200]	[0.9745]
F-test of significance of difference, p-value:	[0.0035]	[0.0055]	[0.7121]
d) Climate of team location			
Temperature (°C) if team in temperate/humid subtropical climate	0.0105	0.0173	0.0001
	[0.3163]	[0.1019]	[0.9950]
Temperature (°C) if team in Mediterranean climate	0.0083	0.0225**	-0.0188
	[0.4673]	[0.0420]	[0.4138]
F-test of significance of the difference, p-value:	[0.7131]	[0.4323]	[0.1879]
# of observations	7,961	7,754	6,682
# of team-season	219	214	195

* p-value $<$0.10, ** p-value $<$0.05, *** p-value $<$0.01. For each model, in brackets we report the step-down adjusted p-values of Romano and Wolf (2005a,b), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. In panel a, we also include a dummy for congested match scheduling (more than three matches in the last 15 days). In panel b, the top five urban areas are Milano, Roma, Napoli, Torino, and Genova. The teams in these cities are AC Milan, Inter Milan, Lazio, Roma, Juventus, Torino, Sampdoria and Genoa. In panel d, teams located in temperate or humid subtropical areas are AC Carpi, Atalanta, Bologna, Brescia, Cesena, Chievo Verona, Cremonese, Genoa, Hellas Verona, Inter Milan, Juventus, AC Milan, Monza, Parma, Pescara, Sampdoria, Sassuolo, SPAL, Torino, Udinese, and Venezia. Teams located in Mediterranean climatic areas are Benevento, Cagliari, Catania, Crotone, Empoli, Fiorentina, Frosinone, Lazio, Lecce, Livorno, Napoli, Palermo, Roma, Salernitana, Siena, and Spezia. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation. In panel a, the number of observations which played in congested periods is 2,725 in model (1), 2,681 in model (2), and 2,356 in model (3). In panel b, the number of observations of teams located in the top five urban areas is equal to 3,553 in model (1), 3,535 in model (2), and 3,111 in model (3). In panel c, the number of observations of teams with above-median tangible assets is equal to 3,192 in model (1), 3,139 in model (2), and 2,727 in model (3). Tangible assets is missing for 1,558 observations (the last three seasons), which are not used in panel c. In panel d, the number of observations for teams in temperate/humid subtropical climate is 5,036 in model (1), 4,844 in model (2), and 4,170 in model (3).

To shed further light on heterogeneity across organizational environments, we conduct an additional analysis distinguishing between "top teams", characterized by larger market size, and teams with smaller market size, using the population of the urban area as a proxy. We classify as "top teams" those located in the five largest urban areas – Milano, Roma, Napoli, Torino, and Genova – namely AC Milan, Inter Milan, Lazio, Roma, Juventus, Torino, Sampdoria, and Genoa. The underlying idea is that these teams typically have greater financial and organizational resources, which may translate into better training and medical facilities, as well as more effective workload and recovery management. Consistent with this interpretation and in line with what emerged from panel a, panel b of Table 5 shows that the adverse impact of temperature is less pronounced among top teams, whereas it is stronger for clubs with smaller market size.[22]

As an alternative proxy for clubs' resource capacity, we use the book value of tangible assets at the end of the previous season (for data sources, see Hoey et al., 2021; Peeters and van Ours, 2026). This measure captures differences in clubs' pre-existing stock of physical capital, which may include training and recovery facilities, medical and rehabilitation infrastructure, and other physical assets potentially relevant for players' health and working conditions. We classify clubs into high- and low-asset groups based on the season-specific median (1.8 millions of €). The temperature gradient is substantially larger among clubs with lower tangible assets. For these clubs, a one-degree increase in temperature is associated with an approximately 2.9% increase in intrinsic injuries, whereas the corresponding estimate for high-asset clubs is small (0.6%) and not statistically significant. The difference between the two temperature coefficients is statistically significant (p-value $= 0.0055$). In line with panel b, this pattern is consistent with greater infrastructure-related resources and capacity being associated with lower vulnerability to heat, although the available accounting data do not allow us to identify which specific assets or investments account for this difference.

Finally, we examine whether the climatic classification of each team's location leads to heterogeneous temperature effects. Our hypothesis is that players' physiological adaptation to heat may depend on their usual climatic environment. To test this, we split teams into those located in temperate or humid subtropical climates and those based in Mediterranean areas.[23] Teams based in temperate or humid subtropical climates may be less able to cope

[22] In the same vein, we replicate the analysis by defining top teams as the so-called "seven sisters" (AC Milan, Atalanta, Inter Milan, Juventus, Lazio, Napoli, and Roma), i.e., the teams most frequently at the top of the standings over the last years. The results are very similar and are available from the authors upon request.

[23] Similar to Fatima et al. (2021) and Filomena and Picchio (2024), we use the Köppen-Geiger climate classification (Beck et al., 2018) to group team locations into different climatic zones.

with high temperatures than those in Mediterranean areas, as they may be less accustomed to frequent exposure to heat. If such adaptation mechanisms are at work, we would expect a larger temperature effect for the former group. Our results (panel c of Table 5) do not corroborate this hypothesis: the estimated effect on intrinsic injuries is slightly larger for teams in Mediterranean areas and the difference between the two groups is not statistically significant. It should be noted, however, that this exercise provides only a limited test of the adaptation hypothesis, as the climatic classification is a coarse proxy for long-run exposure and does not capture individual-level adaptation, short-run acclimatization, or match-specific thermal conditions.

4.2.3 Player-Level Heterogeneity: Physiological and Risk Profiles

In what follows, we perform heterogeneity analyses on the basis of players' and injuries' characteristics, by distinguishing the count of injuries per match per team on the basis of players' position, their age, and injury severity.

In football, players exert different levels of effort and perform distinct tasks depending on their position and role on the pitch. Midfielders generally cover the greatest total and high-speed distance, perform the highest sprint distance, and exhibit the highest percentage of maximal heart rate (Sarmento et al., 2024). Because they sustain greater physical effort, midfielders are more likely to be affected by fatigue and dehydration than other players, particularly at higher temperatures. Accordingly, we hypothesize that the temperature gradient for intrinsic injuries is steeper for midfielders. By contrast, we expect the effect of temperature on extrinsic injuries to be independent of playing position. Consistent with these hypotheses, Table 6 shows that the temperature effect on intrinsic injuries is strongest and highly significant only for midfielders. For this group, a one-degree increase in temperature is associated with a 4.4% increase in intrinsic injuries, whereas for forwards and defenders the estimated effect is smaller and not statistically significant.[24] Moreover, regardless of position, temperature does not increase the risk of extrinsic injuries.[25]

Second, in Table 7 we examine whether the temperature effect varies by age by splitting

[24]The temperature effects on intrinsic injuries differ significantly across playing positions (p-value $=$ 0.0239). The effects on extrinsic injuries do not (p-value $=$ 0.1531).

[25]The literature on air pollution and performance or workplace injuries similarly finds that workers exposed to greater mental and physical strain are more responsive to environmental stressors. Lichter et al. (2017) shows that, in football, productivity in more physically demanding positions is slightly more affected by air pollution. Lavy et al. (2025), studying construction-site accidents, finds that the effects of pollution are exacerbated when workers' physiological state is challenged, such as under high cognitive strain or fatigue.

Table 6: Effect Heterogeneity by Playing Positions

	Forwards' number of injuries			Midfielders' number of injuries			Defenders' number of injuries		
	Total (1)	Intrinsic (2)	Extrinsic (3)	Total (4)	Intrinsic (5)	Extrinsic (6)	Total (7)	Intrinsic (8)	Extrinsic (9)
Temperature (°C)	0.0242	0.0220	0.0264	0.0261	0.0443**	-0.0125	-0.0174	-0.0078	-0.0433
	(0.0127)	(0.0164)	(0.0250)	(0.0136)	(0.0155)	(0.0267)	(0.0142)	(0.0149)	(0.0302)
	[0.1607]	[0.4388]	[0.5455]	[0.1607]	[0.0127]	[0.7794]	[0.4522]	[0.7794]	[0.3992]
Mean outcome	0.070	0.047	0.023	0.079	0.053	0.026	0.096	0.066	0.030
# of observations	5,810	4,610	2,360	6,468	5,284	2,832	7,085	5,985	3,542
# of team-season	174	152	107	190	165	122	202	180	139

* p-value <0.10, ** p-value <0.05, *** p-value <0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation. The p-values from tests of equality of the temperature coefficients are 0.0342, 0.0239, and 0.1531 for the comparisons among columns (1)-(4)-(7), (2)-(5)-(8), and (3)-(6)-(9), respectively.

injuries between players younger than 28 and those aged 28 or older.[26] On the one hand, younger players may be better able to cope with high temperatures due to superior physiological capacity and a generally lower baseline risk of injury (Cloke et al., 2012). On the other hand, older players may better manage adverse conditions and better regulate physical effort thanks to greater experience. Our point estimates suggest that the temperature gradient is larger for older players. For instance, intrinsic injuries increase by 2.3% per one-degree Celsius rise in temperature among older players, compared with 1.4% for younger players. However, splitting the sample by age reduces statistical power, and none of the coefficients remains statistically significant.

Third, we examine whether the impact of temperature is confined to mild injuries or extends to more severe ones. We classify injuries by severity based on recovery duration. Following Fuller et al. (2006) and Ekstrand et al. (2012), we define "mild and moderate" injuries as those involving up to 28 days of absence, and "severe" injuries as those requiring more than four weeks before returning to play. The results, reported in Table 8, show that the temperature effect on intrinsic injuries is concentrated among severe cases. This finding also alleviates concerns that our results are driven by the mechanical under-reporting in data from Transfermarkt.com discussed in Subsection 2.2, because more serious injuries are less likely to be under-reported, being harder to conceal or overlook.

[26] The median age of injured players is 28.

Table 7: Effect Heterogeneity by Age

	Number of injuries of players aged $\geq$ 28			Number of injuries of players aged $<$ 28		
	Total (1)	Intrinsic (2)	Extrinsic (3)	Total (4)	Intrinsic (5)	Extrinsic (6)
Temperature (°C)	0.0175	0.0232	0.0136	0.0015	0.0137	-0.0194
	(0.0117)	(0.0132)	(0.0227)	(0.0114)	(0.0142)	(0.0192)
	[0.2625]	[0.1405]	[0.6915]	[0.7235]	[0.5490]	[0.5490]
Mean outcome	0.124	0.087	0.037	0.129	0.084	0.045
# of observations	7,343	6,607	3,799	7,558	6,607	4,952
# of team-season	204	188	142	211	194	165

* p-value $<$0.10, ** p-value $<$0.05, *** p-value $<$0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation. The p-values from tests of equality of the temperature coefficients are 0.3090, 0.5980, and 0.2800 for the comparisons between columns (1)-(4), (2)-(5), and (3)-(6), respectively.

Table 8: Effect Heterogeneity by Injury Severity

	Number of injuries with duration $\leq$ 28 days			Number of injuries with duration $>$ 28 Days		
	Total (1)	Intrinsic (2)	Extrinsic (3)	Total (4)	Intrinsic (5)	Extrinsic (6)
Temperature (°C)	0.0090	0.0126	0.0045	0.0146	0.0394**	-0.0382
	(0.0103)	(0.0119)	(0.0170)	(0.0154)	(0.0173)	(0.0320)
	[0.5199]	[0.4779]	[0.7442]	[0.5199]	[0.0382]	[0.4712]
Mean outcome	0.178	0.119	0.059	0.075	0.052	0.023
# of observations	7,790	7,338	5,871	6,517	5,500	2,325
# of team-season	214	206	180	188	169	113

* p-value $<$0.10, ** p-value $<$0.05, *** p-value $<$0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation. The p-values from tests of equality of the temperature coefficients are 0.7558, 0.1599, and 0.2453 for the comparisons between columns (1)-(4), (2)-(5), and (3)-(6), respectively.

4.2.4 The Role of Other Meteorological Conditions

The temperature effect may depend and interact with wind speed and precipitation. For example, the temperature effect may be less relevant with a stronger wind or when it is rainy, because in windy or rainy conditions, the body is better able to cool down and the perceived heat is smaller. To check this, we normalized wind speed and precipitation to have zero mean and we re-estimate Equation (1) after separately including in the model specification the interaction between temperature and either wind speed or precipitation. We find that none of the interaction coefficients is statistically significant at the conventional levels, whether the dependent variable is total injuries or we distinguish between intrinsic and extrinsic injuries. Results are reported in Table B.1 in the appendix.

Finally, since one may be concerned that ambient temperature does not fully capture the temperature perceived by the human body, we assess whether humidity has an independent effect beyond temperature. We use dew point temperature, a direct measure of atmospheric humidity. Because it is highly correlated with ambient temperature (Pearson's correlation coefficient of 0.817), including it directly in the regression makes it difficult to disentangle their separate effects. To address this issue, we residualize dew point temperature with respect to ambient temperature, controls, and FE. The resulting residual captures variation in humidity orthogonal to temperature and not explained by the other covariates, which we interpret as 'excess humidity'. We include this residual in the injury regression to identify the effect of humidity net of temperature-driven co-movements. Table B.2 reports the estimates for temperature and excess humidity. The inclusion of excess humidity does not affect the estimated temperature effects.

4.3 Short-Term Accumulation or Adaptation?

The positive effect of kick-off time temperature on intrinsic injuries is likely due to impaired physiological and muscular conditions when playing the match in warmer conditions. However, alongside the contemporaneous impact, temperature may also have indirect dynamic effects on the injury risk (see e.g. Sexton et al., 2022).

In our setting, players' physical conditions may be influenced by heat exposure in the days leading up to the football match, such as during weekly training sessions. High temperature exposure may accumulate over time, with larger risks of exhaustion, higher physical and mental stress (Heal and Park, 2016), and greater energy usage (Deschênes and Greenstone, 2011), which may slow down both physical and mental recovery with a lagged effect on safety outcomes in the next match. On the other hand, higher temperatures in the pre-

vious days may mitigate the positive heat impact during the match in case of short-term adaptation or habituation mechanisms.

Therefore, prior meteorological conditions may contribute either to cumulative physiological strain or to heat acclimation, thereby modifying how football players respond to match-day climate. Distinguishing between these channels has direct policy relevance: if injury risk responds primarily to sustained heat exposure in the days preceding a match, the implications concern long-run adaptation to a warmer climate; if it is driven mainly, or exclusively, by contemporaneous thermal conditions, policy attention should instead focus on short-term preventive interventions.

To shed more light on potential adaptation or accumulation mechanisms, we extend the baseline model by incorporating three distinct temperature dimensions recorded at teams' training venues: (i) the average maximum temperature in the six days preceding the match; (ii) a measure of extreme heat exposure, defined either as the number of days with maximum temperature above 28°C (approximately the 95th percentile) or as a dummy equal to one if at least three consecutive days exceed this threshold; and (iii) intra-week variability, captured by the standard deviation of daily maximum temperatures over the same period. Table 9 reports the estimated coefficients for both the contemporaneous kick-off temperature and these additional variables.

Across all specifications, the estimated effect of match temperature remains positive and stable for intrinsic injuries, suggesting that the baseline relationship is not driven by short-term temperature dynamics in the training period. In contrast, we find little evidence of a statistically significant effect of kick-off temperature on total or extrinsic injuries.

Turning to the additional temperature measures, the results provide limited support for an accumulation mechanism. The average temperature in the preceding days is generally small and statistically insignificant for total and intrinsic injuries, while it is negative and marginally significant for extrinsic injuries. Similarly, intra-week temperature variability does not appear to play a meaningful role, as its estimated coefficients are consistently imprecise and close to zero across all specifications.

Indicators of extreme heat exposure in the days prior to the match negatively and significantly affect injury risk. This pattern is consistent with short-term adaptation or behavioural adjustments, like precautionary measures during training sessions, following periods of extreme heat, which may reduce injury risk at the time of the match.

Overall, these findings suggest that while kick-off temperature increases the likelihood of intrinsic injuries, prior exposure to sustained high temperatures does not exacerbate this effect and may even attenuate it, pointing to the importance of short-term adaptation mech-

Table 9: Including Average Temperature, Extreme Heat Events, and Intra-Variability in the Preceding 6 Days

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
a) Including average and standard deviation of maximum temperature in the previous 6 days			
Temperature (°C)	0.0159*	0.0197*	0.0109
	(0.0102)	(0.0119)	(0.0184)
	[0.0720]	[0.0680]	[0.4348]
Average of maximum temperature previous 6 days	-0.0150	-0.0019	-0.0406*
	(0.0125)	(0.0139)	(0.0228)
Standard deviation of maximum temperature previous 6 days	-0.0143	-0.0094	-0.0307
	(0.0270)	(0.0357)	(0.0457)
b) Including number of extreme days and standard deviation of maximum temperature in the previous 6 days			
Temperature (°C)	0.0120	0.0220**	-0.0054
	(0.0088)	(0.0107)	(0.0146)
	[0.1484]	[0.0210]	[0.6337]
# of days with maximum temperature $\geq$ 28°C previous 6 days	-0.0601***	-0.0688***	-0.0362
	(0.0228)	(0.0217)	(0.0480)
Standard deviation of maximum temperature previous 6 days	-0.0096	-0.0061	-0.0239
	(0.0267)	(0.0355)	(0.0458)
c) Including number of extreme days and standard deviation of maximum temperature in the previous 6 days			
Temperature (°C)	0.0118	0.0215**	-0.0048
	(0.0087)	(0.0106)	(0.0146)
	[0.1569]	[0.0255]	[0.6702]
3 or more consecutive days with max. temp. $\geq$ 28°C in previous 6 days	-0.3295***	-0.3297***	-0.3306*
	(0.0984)	(0.0862)	(0.1834)
Standard deviation of maximum temperature previous 6 days	-0.0079	-0.0045	-0.0217
	(0.0268)	(0.0356)	(0.0457)
# of observations	7,961	7,754	6,682
# of team-season	219	214	195

* p-value <0.10, ** p-value <0.05, *** p-value <0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), which are robust to multiple hypothesis testing for the significance of the temperature coefficient across models (1), (2), and (3), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation.

anisms rather than cumulative physiological stress.

4.4 Robustness Checks

In the main analysis, injuries are attributed to the match played on the same day as the diagnosis or within the two preceding days, to account for delayed symptom onset and post-match medical diagnosis. Panel a of Table B.3 shows that the estimated coefficients are not sensitive to this choice and remain robust when injuries are attributed only to matches played on the day of diagnosis or one day earlier.

Panel b of Table B.3 shows that the main findings are not driven by the congested match schedule observed during the post-COVID-19 period (June–July 2020). In particular, we rule out the concern that the estimated effect of heat merely reflects a small number of matches played in unusually hot months, combined with reduced player fitness following the prolonged suspension of the Italian Serie A. Moreover, in that period also training was inhibited by the COVID-19 lockdown measures and other restrictions. Nonetheless, even when removing this atypical period, the temperature-injury association remains stable, suggesting that the effect of heat is systematic rather than an artifact of scheduling disruptions or temporary declines in training and physical conditioning.

In panel c of Table B.3 we relax our baseline functional-form assumption and include a full set of kick-off-time FE. This specification absorbs any systematic differences across scheduling slots, including differences in thermal conditions as well as match characteristics that may be correlated with kick-off time. The estimated temperature gradient remains similar in magnitude and statistical significance to that obtained in our baseline specification.

To strengthen the interpretation of our findings and better isolate the underlying mechanisms, we refine the definition of intrinsic injuries by focusing exclusively on strictly muscular injuries and separating them from tendinous, ligamentous, and overuse conditions.[27] This narrower classification allows us to more directly capture injuries plausibly linked to acute physiological fatigue and thermal strain. Reassuringly, Table B.4 shows that the estimated effect of temperature remains statistically significant and economically meaningful under this stricter definition. The persistence of the result suggests that our main findings are not driven by broader or more heterogeneous injury categories but instead reflect a consistent relationship between elevated temperature and muscle-related injury risk.

Finally, we assess whether our inference strategy is robust. In the main analysis, we cluster standard errors at the team-season and week levels, yielding a sufficiently large number

[27] See Table A.3 in Appendix A for a complete classification of strictly muscular injuries in the dataset.

of clusters to mitigate concerns about over-rejection due to few clusters (Cameron et al., 2008). However, while this approach ensures asymptotic reliability, it may understate uncertainty if residual dependence persists across seasons within teams, potentially leading to over-rejection and inflated type I error rates. To evaluate whether team-season clustering induces over-rejection, we implement a permutation-based inference exercise (Fisher, 1935; Box and Andersen, 1955; Welch, 1990). Specifically, we randomly permute (re-sampling without replacement) the temperature variable 10,000 times across team-match observations and re-estimate the full Poisson specification, including all controls and FE, while clustering standard errors multiway at the team-season and week levels. For each replication, we store both the estimated temperature coefficient and its associated analytic p-value. This procedure approximates the distribution of the test statistic under the null hypothesis of no effect. Under the null hypothesis, analytic p-values should be uniformly distributed. In the permutation exercise, analytic p-values are close to uniformly distributed over the unit interval (left column of Figure B.2), indicating that multiway clustering at the team-season and week levels does not appear to generate systematic over-rejection or mechanically understate standard errors. Moreover, the permutation distribution of the estimated temperature coefficients (right column of Figure B.2) is approximately symmetric and bell-shaped around zero. This suggests that the estimator exhibits regular finite-sample behaviour and is not evidently driven by extreme leverage points or instability in the estimation procedure.

5 Conclusions

We estimate the causal effect of ambient temperature on worker injury incidence using data on professional football matches from the Italian Serie A. Elite athletes provide a useful setting for studying injury risk under workplace environmental stress such as temperature, which in our context is a plausibly exogenous working condition.

The results are stark: temperature increases intrinsic injuries by 1.9% per one-degree Celsius, while it does not significantly affect extrinsic injuries. This sharp contrast is consistent with temperature operating through internal physiological stress, for example by increasing dehydration and thermoregulatory strain, accelerating metabolic fatigue, impairing neuromuscular control, and contributing to electrolyte imbalances associated with muscle cramps and soft-tissue injuries. The effect is even more pronounced for severe intrinsic injuries, which increase by 3.9% per one-degree Celsius.

The magnitude of the estimated effect is economically meaningful: moving from the 25th to the 75th percentile of the match-day temperature distribution is associated with an

approximately 21.7% increase in the expected number of intrinsic injuries. In addition, the resulting increase in injury risk may entail broader economic consequences that are not captured by our estimates, including foregone labor input, reduced team performance, medical expenditures, depreciation in player market values, and increased workload for remaining players. More broadly, the potential relevance of these findings to larger labor markets and physically demanding occupations with larger workforces points to potentially sizeable aggregate welfare costs associated with heat exposure.

The heterogeneity patterns provide further evidence consistent with a role for physical demands in shaping the temperature-injury relationship. The temperature gradient is particularly pronounced among midfielders, who typically cover the greatest total and high-speed distances. We also find suggestive evidence of larger temperature effects in match contexts potentially associated with stronger incentives to exert effort, including matches with high stadium attendance and home matches, where crowd support and more offensive tactical choices may increase physical intensity; more balanced games, characterised by sustained exertion and intensity; and matches played in the final part of the season, when cumulative fatigue may peak, recovery capacity may be reduced, and competitive stakes are higher.

By analysing potential adaptation and accumulation effects, we find that prior exposure to heat does not exacerbate the immediate cost of playing in high kick-off temperatures. If anything, prolonged heat exposure slightly dampens this effect, suggesting the importance of short-term adaptation mechanisms rather than cumulative physiological stress.

Our findings highlight the potential importance of heat-mitigation strategies for injury prevention in professional football. By showing that higher ambient temperature increases the incidence of intrinsic injuries, our results suggest that measures aimed at limiting players' thermal strain may help reduce injury risk. Such measures may include enhanced hydration protocols, cooling breaks, and other forms of heat management.[28] While adjusting match schedules to avoid periods of high temperature may represent an additional adaptation strategy, our analysis does not directly identify the effect of rescheduling matches across different times of the day. More broadly, our results highlight the importance of adapting work intensity and recovery opportunities in response to environmental conditions.

Our results may be relevant to other outdoor invasion sports that share football's intermittent, multidirectional play and position-specific physical demands, such as rugby, field hockey, and lacrosse. However, this potential generalizability should be interpreted cautiously given differences in contact, rules, and match structure. Moreover, beyond profes-

[28] As a matter of fact, players at the FIFA World Cup 2026 in the US benefited from three-minute hydration breaks in each half of games (https://inside.fifa.com/organisation/news/hydration-breaks-world-cup-2026-player-welfare, last accessed on April 1, 2026).

sional football, our findings may speak to a broader set of occupations in which heat exposure coincides with substantial physical exertion and sustained attention, such as agriculture, construction, firefighting, delivery and logistics, road and utility work, and heat-intensive manufacturing. Professional football represents an unusually demanding work environment, and the magnitude of our estimates should therefore not be directly extrapolated to the average worker. Rather, our results provide evidence on a mechanism that may operate more broadly: heat increases the injury cost of physical effort, with suggestive evidence that this cost may be larger in high-stake activities and for tasks involving greater physical demands.

Thus, while the proximate mechanism linking temperature to injury is physiological, its interaction with work intensity and the incentives shaping workers' effort has direct labor market relevance. This mechanism may be particularly consequential in workplaces with absent, limited, or costly climate control and in resource-constrained organizations with less capacity to buffer heat shocks through flexible staffing, task reallocation, rest periods, or investments in adaptation technologies. As temperatures continue to rise and extreme heat becomes more frequent, understanding how environmental conditions interact with work intensity, incentives, and adaptation can help inform the design of safer workplaces and more resilient production processes.

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Appendix

A Classification of Injuries

Table A.1: Different Types of Observed Intrinsic Injuries

Type	Absolute frequency	Type	Absolute frequency
Abdominal problems	1	Inner ligament stretch of the knee	4
Achilles tendon irritation	1	Internal ligament strain	2
Achilles tendon problems	4	Internal ligament tear	2
Adductor injury	69	Kidney problems	1
Adductor pain	76	Knee collateral ligament strain	2
Adductor tear	1	Knee collateral ligament tear	1
Arch problems	1	Left hip flexor problems	2
Back injury	4	Ligament injury	4
Back problems	8	Lumbago	7
Calf injury	59	Lumbar vertebra problems	3
Calf muscle tear	23	Meniscus irritation	1
Calf problems	39	Muscle fatigue	119
Calf strain	5	Muscle fiber tear	1
Collateral ligament injury	16	Muscle injury	100
Contortion	2	Muscle stiffness	1
Contracture	16	Muscle strain	13
Cruciate ligament injury	15	Muscle tear	1
Cruciate ligament strain	1	Muscular problems	206
Cruciate ligament surgery	1	Outer ligament tear	1
Cruciate ligament tear	32	Overstretching	15
Dead leg	2	Patellar tendon problems	4
Edema in the knee	3	Patellar tendon rupture	1
Fitness	4	Peroneus tendon injury	1
Groin injury	2	Pubalgia	1
Hamstring injury	139	Rest	10
Hamstring muscle injury	145	Sore muscles	1
Hamstring strain	19	Sprain	13
Heart problems	1	Strain	6
Heel injury	1	Strain in the thigh and gluteal muscles	1
Heel problems	4	Stress reaction of the bone	3
Heel spur	5	Syndesmotic ligament tear	1
Hip flexor problems	2	Tendon irritation	2
Inflammation	12	Tendonitis	3
Inflammation in the knee	9	Thigh problems	76
Inflammation of ligaments in the knee	1	Torn ankle ligaments	2
Inflammation of pubic bone	2	Torn lateral ankle ligament	2
Inflammation of the sole of the foot	1	Torn lateral knee ligament	1
Inguinal hernia	3	Torn muscle fiber	7
Injury to abdominal muscles	2	Torn muscle fiber in the adductor area	4
Inner knee ligament tear	1	Torn thigh muscle	2
Inner ligament injury	7	Total	1,366

Table A.2: Different Types of Observed Extrinsic Injuries

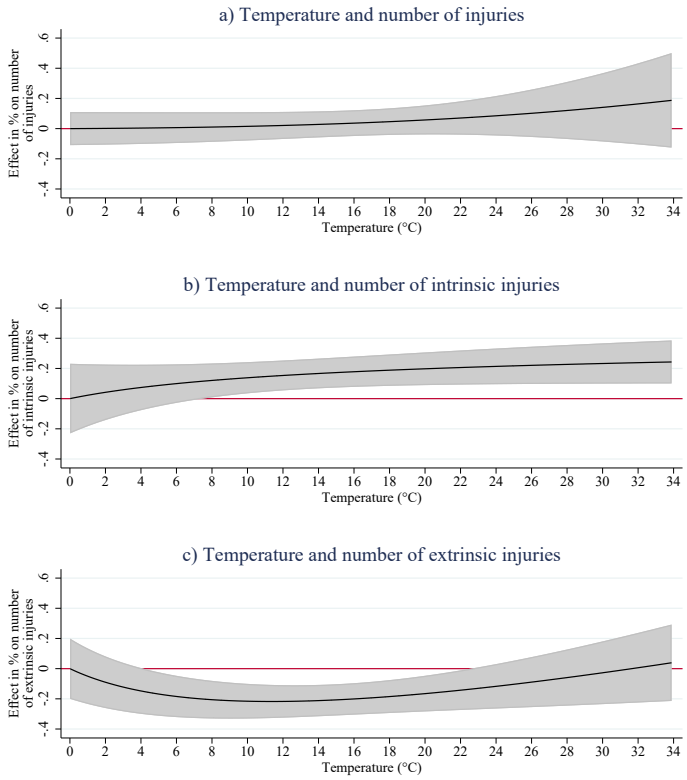
Type	Absolute frequency	Type	Absolute frequency
Achilles tendon contusion	1	Foot injury	14
Achilles tendon rupture	2	Foot surgery	1
Achilles tendon surgery	2	Fracture	8
Ankle injury	39	Hand injury	5
Ankle problems	33	Head injury	15
Ankle sprain	67	Hip bruise	3
Ankle surgery	5	Hip injury	1
Bone bruise	1	Hip problems	2
Bone edema	1	Injury to the ankle	11
Broken ankle	1	Knee bruise	15
Broken cheekbone	7	Knee injury	54
Broken collarbone	1	Knee problems	63
Broken fibula	11	Knee surgery	9
Broken finger	2	Knock	32
Broken foot	3	Leg injury	25
Broken hand	1	Lumbar vertebra fracture	2
Broken jaw	1	Meniscus injury	13
Broken nose bone	10	Meniscus tear	2
Broken toe	2	Metacarpal fracture	1
Bruise	25	Metatarsal fracture	6
Bruise on ankle	7	Minor knock	27
Bruised back	3	Muscle contusion	7
Bruised knee	8	Nose injury	5
Bruised ribs	5	Nose surgery	6
Capsular injury	1	Pelvic injury	2
Cartilage damage	1	Rib fracture	6
Cheekbone bruise	1	Scaphoid fracture	2
Concussion	3	Shin bruise	8
Crack bruise	1	Shoulder injury	35
Elbow fracture	1	Surgery	8
Elbow injury	4	Tendon rupture	2
Eye injury	2	Toe injury	3
Facial fracture	1	Wrist fracture	2
Finger injury	2	Wrist injury	1
Foot bruise	5	Total	656

Table A.3: Different Types of Observed Strictly Muscular Injuries (Subset of Intrinsic Injuries)

Type	Absolute frequency	Type	Absolute frequency
Abdominal problems	1	Injury to abdominal muscles	2
Adductor injury	69	Muscle fatigue	119
Adductor pain	76	Muscle fiber tear	1
Adductor tear	1	Muscle injury	100
Calf injury	59	Muscle strain	13
Calf muscle tear	23	Muscle tear	1
Calf problems	39	Torn muscle fiber	7
Calf strain	5	Muscle fiber in the adductor area	4
Contracture	16	Torn thigh muscle	2
Dead leg	2	Heart problems	1
Fitness	4	Muscle stiffness	1
Groin injury	2	Muscular problems	206
Hamstring injury	139	Sprain	13
Hamstring muscle injury	145	Strain	6
Hamstring strain	19	Total	1,076

B Further Results

Figure B.1: Fractional Polynomial Relationship between Injuries and Temperature



Notes: The estimates reported in these graphs are obtained by fitting a Poisson regression model for the number of injuries per team per match as a function of a degree-2 fractional polynomial in temperature. The temperature impact is normalized to be 0 at 0°C. The powers are selected from the set $\{-2, -1, -0.5, 0, 0.5, 1, 2, 3\}$. Model selection is based on the deviance, defined as twice the negative log-likelihood, with the pair of powers that minimizes the deviance chosen as the preferred specification. The sample consists of 8,000 team-match observations. In panel a, the preferred fractional polynomial specification is $(-2, 3)$; in panel b, it is $(-2, -0.5)$; in panel c, it is $(0.5, 0.5)$. The gray areas represent 95% confidence intervals computed using heteroskedasticity-robust standard errors clustered at the team-season level and at calendar week level.

Table B.1: Estimation Results of Injury Equation (1) with Interaction of Temperature with Wind Speed and Precipitation

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
a) Interaction between temperature and wind speed			
Temperature (°C)	0.0099	0.0192**	-0.0058
	(0.0087)	(0.0104)	(0.0146)
	[0.2464]	[0.0420]	[0.6102]
Temperature (°C) × Wind speed (m/s)	-0.0014	-0.0009	-0.0034
	(0.0024)	(0.0029)	(0.0047)
b) Interaction between temperature and precipitation			
Temperature (°C)	0.0087	0.0198**	-0.0109
	(0.0086)	(0.0105)	(0.0148)
	[0.3153]	[0.0355]	[0.3418]
Temperature (°C) × Precipitation (mm)	-0.0005	0.0003	-0.0027
	(0.0010)	(0.0011)	(0.0016)
# of observations	7,961	7,754	6,682
# of team-season	219	214	195

* p-value <0.10, ** p-value <0.05, *** p-value <0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), which are robust to multiple hypothesis testing for the significance of the temperature coefficient across models (1), (2), and (3), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation. Temperature, wind speed and precipitation are normalized by subtracting the sample means.

Table B.2: Estimation Results of Injury Equation (1) Controlling for Excess Humidity

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
Temperature (°C)	0.0097	0.0191**	-0.0060
	(0.0087)	(0.0104)	(0.0147)
	[0.2574]	[0.0435]	[0.5947]
Excess humidity[a]	0.0022	0.0033	-0.0010
	(0.0100)	(0.0119)	(0.0149)
# of observations	7,961	7,754	6,682
# of team-season	219	214	195
Log-likelihood	-4,552.1	-3,442.8	-1,972.8
Pseudo R^2	0.0963	0.1082	0.1038

* p-value $<$0.10, ** p-value $<$0.05, *** p-value $<$0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), which are robust to multiple hypothesis testing for the significance of the temperature coefficient across models (1), (2), and (3), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation.

[a] We regress dew point temperature on ambient temperature, control variables, and FE. We then use the residuals from this regression, capturing the component of humidity orthogonal to temperature and other covariates, in the baseline injury equation. This residualized measure can be interpreted as 'excess humidity', i.e. variation in humidity not explained by temperature or other controls.

Table B.3: Robustness Checks: Injury Attribution, COVID-19 Pandemic, Kick-off-Time FE

	Number of injuries (1)	Number of intrinsic injuries (2)	Number of extrinsic injuries (3)
a) Injuries attributed to a match up to only one day after the match			
Temperature (°C)	0.0094	0.0188*	-0.0060
	(0.0099)	(0.0117)	(0.0172)
	[0.3498]	[0.0925]	[0.6547]
# of observations	7,831	7,482	6,045
b) Removal of the post-COVID-19 period (June and July 2020)			
Temperature (°C)	0.0102	0.0211**	-0.0080
	(0.0089)	(0.0106)	(0.0149)
	[0.2439]	[0.0215]	[0.4998]
# of observations	7,706	7,499	6,369
c) Match kick-off-time FE			
Temperature (°C)	0.0075	0.0174*	-0.0091
	(0.0086)	(0.0102)	(0.0146)
	[0.4113]	[0.0705]	[0.4143]
# of observations	7,961	7,754	6,672

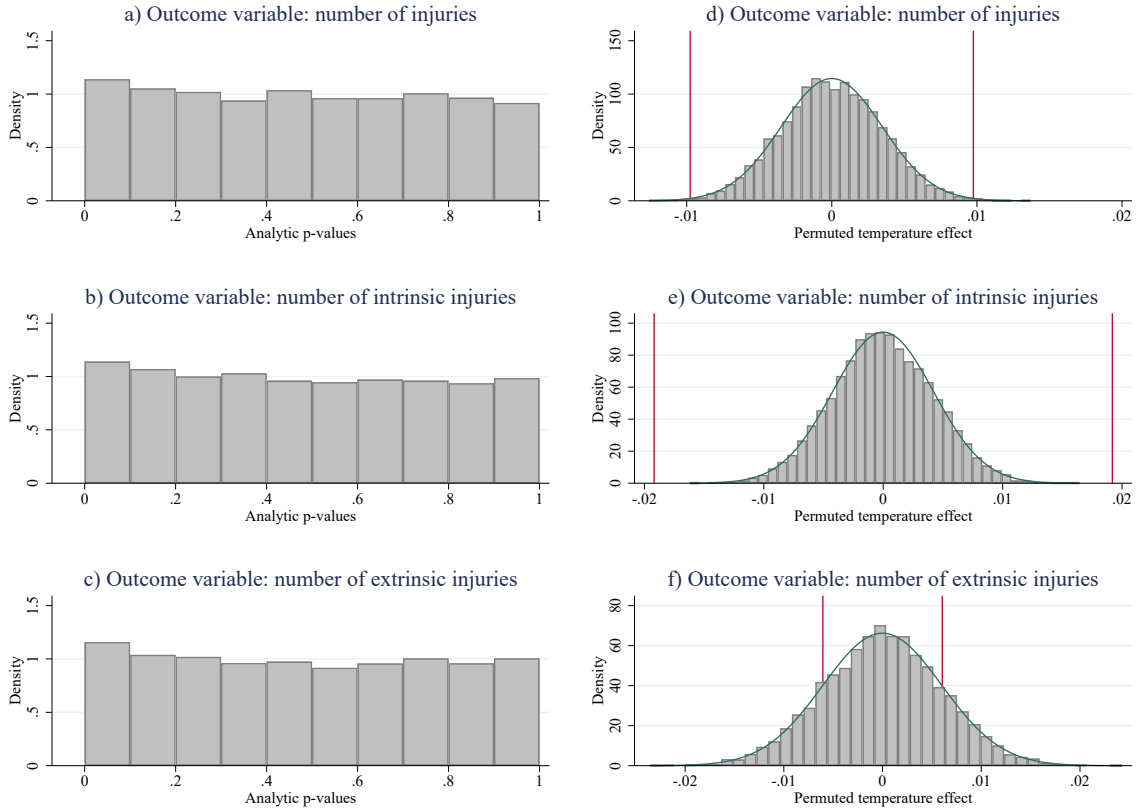
* p-value <0.10, ** p-value <0.05, *** p-value <0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. In brackets, we report the step-down adjusted p-values of Romano and Wolf (2005a,b), which are robust to multiple hypothesis testing for the significance of the temperature coefficient across models (1), (2), and (3), with clusters at the team-season and week levels (2,000 bootstrap replications). All models include the same covariates and the same FE as the baseline model, except for models in panel c where we include kick-off time FE in place of the linear control. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation.

Table B.4: Robustness Checks: Narrower Definition for Intrinsic Injuries

	Number of muscular injuries (1)	Number of overuse injuries (2)
Temperature (°C)	0.0185*	0.0234
	(0.0107)	(0.0239)
# of observations	7,415	4,208

* p-value <0.10, ** p-value <0.05, *** p-value <0.01. Standard errors in parentheses are heteroskedasticity-robust and multiway clustered at the team-season and week levels. All models include the same covariates and the same FE as the baseline model. The number of observations is smaller than what shown in Tables 1 and 2 because singleton groups and observations with no within-fixed-effect variation (i.e., separated by the FE) are excluded from estimation.

Figure B.2: Distributions of p-values and Temperature Coefficients across 10,000 Permutations of the Temperature Variable (under the null hypothesis that there is no effect of temperature on the relevant-injury variable)



Notes:. Graphs a, b, and c (left column) show the distribution of the analytic p-values for the test of the temperature coefficient in the baseline model when standard errors are clustered at the team and week levels. The p-values are obtained by re-estimating the model after randomly permuting the temperature variable 10,000 times. Graphs d, e, and f (right column) show the distribution of the estimated temperature coefficients across the 10,000 permutations, together with the corresponding normal density as a reference. The vertical red lines in the right-column graphs indicate the baseline temperature estimate and its opposite value.