What cannot be cured must be endured: the long-lasting effect of a COVID-19 infection on workplace productivity

What Cannot be Cured Must be Endured: The Long-Lasting Effect of a COVID-19 Infection on Workplace Productivity*

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Abstract

The COVID-19 pandemic has triggered economic shock waves across the globe. Exploiting a natural experiment, this paper estimates how being infected with the virus shapes individual-level productivity after having recovered. Studying the performance of professional athletes in Germany and Italy and applying a staggered difference-in-differences design, we find that individual performance drops by around 6 percent after a previously infected athlete returns to the pitch. This striking deterioration remains persistent over time – amounting to 5 percent eight months after the infection. The effect increases with age and infection severity, and is spread disproportionally over the course of a match. We detect no productivity effects for other respiratory infections. We take these findings as first evidence that the pandemic might cause long-lasting effects on worker productivity and economic growth.

Keywords: Labor Productivity, Economic Costs of COVID-19, Public Health

JEL Classification: H12, I18, J21, J24, J44, O47

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1 Introduction

To counteract the spread of COVID-19, governments have introduced a wide range of non-pharmaceutical interventions (NPI), such as distancing rules or work-from-home directives. Though indispensable from an epidemiologist's perspective, measures such as the closing of schools and universities and the general reduction in economic activity has come with high direct and indirect costs for society as early economic evaluations emphasize (see, for example, the review articles by Brodeur et al., 2021; Padhan and Prabheesh, 2021) To date, most of this research has primarily considered the costs of NPIs. Besides the obvious effect of fewer infections and deaths in total, research quantifying the individual and economic benefits of infection prevention is hitherto missing. This paper addresses this gap in the literature by documenting the significant and persistent effect of a COVID-19 infection on individual labor productivity.

We contribute these novel findings by studying the performance of professional soccer players after a COVID-19 infection – a setting unrivaled in data quality: We use a highly granular nested panel data set that encompasses a sub-panel for every single match for nearly two years in the top-tier male soccer leagues of Germany and Italy. Thereby we take advantage of an institutional setting that is unique across occupations not only in terms of data availability. First, professional soccer is an industry that quickly resumed business, mostly unaffected by NPIs, after only a short interruption in spring 2020 – differently from many other industries. Second, the top European leagues implemented a uniquely rigorous testing procedure: Every player was PCR-tested at least once per week and often several times.

Our findings hardly suffer from measurement errors caused by unknown positives. Thus, we circumvent the issue of true case numbers being much higher than reported ones – a problem most occupations face (Hortaçsu et al., 2021; Manski and Molinari, 2021). Hence, we are actually able to estimate a population effect and not only the impact of COVID-19 of those showing up in a hospital. Given the popularity of the sport, we can exploit extremely detailed records of all players in every match. This allows us to disentangle individual and team productivity and to detect short- and long-run effects which would remain unobserved outside this industry. Eventually, medical studies on 'long COVID' are subject to methodological problems due to a reliance on patients' self-reported health or subsamples with strong symptoms (Yelin et al., 2020; Maxwell, 2021). By solely considering observational data, we avoid this issue.

To estimate the effects of a COVID-19 infection, we apply a staggered difference-in-differences framework. We compare infected with non-infected players before and after the infection and exploit the arguably idiosyncratic timing of infections with the virus for identification. In the context of our analysis, we consider productivity as a function of various individual health aspects, such as acceleration, condition, and endurance, but also cognitive capability. Our empirical analysis addresses two questions: Does a COVID-19 infection affect the probability of a player participating in a match and the length of time he stays on the pitch? This extensive margin captures general absence effects related to the infection but also takes up the non-consideration of post-infected players by the team managers. Second, is the performance of previously infected players lower once they play again? Here, our interest lies in productivity across matches as well as within a match – the intensive margin effects.

At the extensive margin, we find that once players are cleared to play by a team's medical staff, their time on the pitch decreases by more than 5 percent. At the intensive margin, we are able to identify a significant deterioration in infected players' productivity of 5–7 percent after an infection. This effect becomes visible right after a player's return to the pitch but remains persistent for more than eight months – a notable difference from what we find for common respiratory infections.

Exploiting the very rich nature of our data, we further assess players' performance throughout every single match. We identify a disproportional decrease in productivity toward the end of a game. This pattern might even be underestimated as the weakest players are likely to be substituted off. Our analysis reveals notable heterogeneity across age groups. Players above the age of 30 are twice as severely hit as players aged 26 to 30. For younger players up to 25 years of age there exists no significant effect at all.

Our paper also contributes to the strand of research which addresses differences between COVID-19 infections and other respiratory infections. For example, Briggs and Vassall (2021) approximate the costs of continuing health deterioration to amount up to 30 percent of the overall costs caused by the disease including fatalities. In our setting, we highlight that an infection with COVID-19 is indeed different from other respiratory infections, because a productivity deterioration of around 5.1 percent persists over the course of more than eight months. In contrast, we do not find productivity effects originating from colds and similar illnesses.

As in many industries, soccer is a team production. Therefore, we investigate how the individual performance deterioration affects the overall group outcome. A priori, it is unclear whether non-infected players might overcompensate the weakness of their colleagues or suffer from lower performance as well. Our findings support the latter. Players' joint performance tends to be even lower than the accumulated individual deterioration of infected team members.

Of particular interest for this paper is research on productivity effects during the pandemic. For example, Bloom et al. (2020) investigate firm-level productivity using a large panel from the UK and identify a decline in total factor productivity of 3–5%. Morikawa (2021) has shown that low-productivity firms in particular have drawn from public subsidy schemes. Regarding working from home, the findings for productivity are mixed. While Barrero et al. (2021) find an overall positive effect on worker productivity in the UK, Etheridge et al. (2020) do not find significant differences for the UK, Morikawa (2022) identifies a decline for the Japanese economy. Using chess tournaments, Künn et al. (2022) identify a deterioration for cognitively demanding tasks.

Altindag et al. (2021) find that online learners during COVID-19 shutdowns have significantly worse outcomes compared to fellow students in classrooms. Particularly among academics, Deryugina et al. (2021) find that womens' productivity was much more affected through the channel of lockdowns and the related burdens of childcare. In a broader sense, Adams-Prassl et al. (2020, 2022) show that workers have been unequally affected by the COVID-19 pandemic due to different possibilities to move their work to their homes. In contrast to those and many other economic papers, our primary focus lies on the *direct* effect of an infection itself on individual productivity and not indirect channels such as NPIs, which are exploited above.

By analyzing soccer players, we also contribute to a large body of economic research, which has frequently applied sports data to uncover otherwise hidden economic mechanisms (Bar-Eli et al., 2020). Among others, this concerns the testing of theoretical hypotheses from game theory (e.g., Bhaskar, 2008; Chiappori et al., 2002; Kassis et al., 2021), identifying psychological drivers of cognitive performance (e.g., Apesteguia and Palacios-Huerta, 2010; González-Díaz and Palacios-Huerta, 2016), or deriving conclusions for public and labor economics (e.g., Caselli et al., 2022; Kahn and Sherer, 1988; Kleven et al., 2013; Lichter et al., 2017; Parsons et al., 2011; Principe and van Ours, 2022).

The remainder of this paper proceeds as follows. In Section 2, we provide background information on the setting of this natural experiment and explain the data used. In Section 3, we outline our empirical analysis. Section 4 presents and discusses our results at the individual and the team level. Section 5 concludes with a summary, discusses limitations, and provides an outlook for future research.

2 Institutional Setting and Data

Germany's governmental agency for infectious diseases, the 'Robert-Koch-Institut' (RKI), registered about 26.3 million cases and more than 137,000 deaths related to a COVID-19 infection (up to June 2, 2022, within an overall population of 83 million).[1] A similar but worse pattern can be found in Italy. The governmental health agency 'Istituto Superiore di Sanità' reported (up to July 28, 2021) 17.5 million cases and some 164,000 casualties (within an overall population of 60 million).[2]

[1] Source for data on case numbers: https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Daten/Altersverteilung.html (incl. individuals with multiple infections). Source for data on casualties: https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Projekte_RKI/COVID-19_Todesfaelle.html, last update June 2, 2022.

[2] Source for data on cases and casualties: Report Esteso ISS – COVID-19: Sorveglianza, impatto delle infeziomi ed efficacia vaccinale (national update): https://www.epicentro.iss.it/coronavirus/bollettino/Bollettino-sorveglianza-integrata-COVID-19_31-maggio-2022.pdf, published June 3, 2022, data up to May 31, 2022 (incl. individuals with multiple infections).

Only the registered cases in both countries make up for more than 25 percent of the overall population. As COVID-19 affects people of all age groups, millions of those with a cured infection are part of the labor force, which might potentially affect their productivity at work. Moreover, the discussed countries are just two examples and many countries face similar magnitudes of cases among their citizens. The problem of potentially persistent negative effects of an infection on subsequent productivity may be sizable given the large numbers of infected and recovered individuals.

We construct a novel dataset consisting of data on player and match statistics, as well as data on COVID-19 infections of players in Germany's Bundesliga and Italy's Serie A. Both leagues are their country's highest division in men's soccer and among the most successful five leagues worldwide.[3] The two leagues have characteristics that make them particularly appropriate to study. The Bundesliga was the first major soccer league to resume its season in 2020 after the suspension of almost all leagues in spring.[4] Italy was hit severely by the virus in Spring 2020 (as shown by the 7-day incidence rates of Italy on the LHS of Figure 1), but continued its season in June, too.[5] Hence, for both leagues we have players that have been infected in early stages of the pandemic. This allows us to estimate persistent and long-run effects among the infected individuals as we cover a time span of more than 12 months after the outbreak of COVID-19 in Germany and Italy.

We have granular data at the match and at the minute level, allowing for overall but also match phase-specific analyses. We amend this data with information on the injuries and sicknesses that forced players to miss matches. In addition, we include information on player nominations for the national teams during this period of time.[6] We collect data on all COVID-19 infections in both leagues since the outbreak of the pandemic. While every infection has to be communicated to the local authorities by the club carrying

[3] In the European Football Association's five-year ranking, the Serie A is ranked #3 and the Bundesliga #4, see https://www.uefa.com/memberassociations/uefarankings/country/#/yr/2021.

[4] https://www.theguardian.com/football/2020/may/06/bundesliga-set-for-go-ahead-to-resume-season-in-second-half-of-may, published 07.05.20.

[5] https://football-italia.net/official-quarantine-rule-softened/, published 18.06.20.

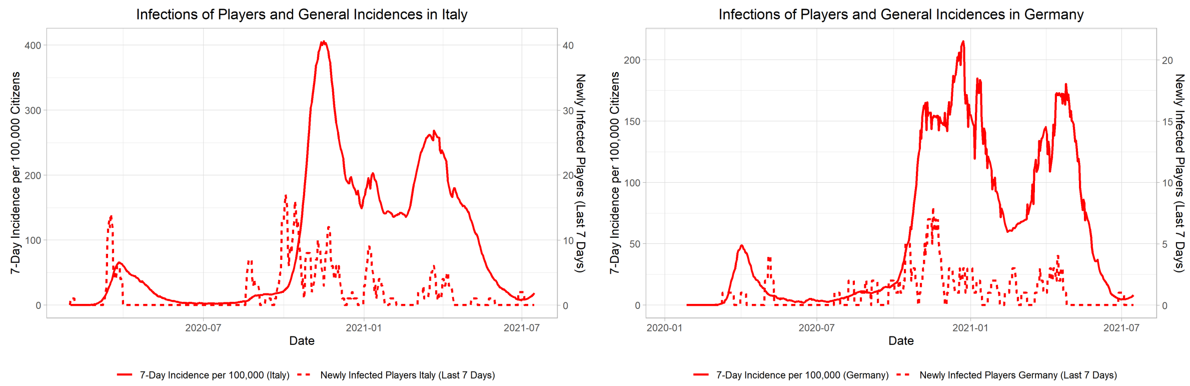
[6] Injury and national team data is obtained from *transfermarkt*, the largest database on soccer players globally.

out the testing, clubs may prefer to keep an infection anonymous, only announcing the number of cases. We identified the large majority of all infected players via a meticulous review of newspapers, reliable websites, and statements from the clubs, players, and soccer associations.

There had been 81 true-positive tests among players in Germany and 176 in Italy by mid-July 2021. We can identify 76 players in Germany and 157 in Italy.[7] Hence, we build our analysis upon the 233 identified players from a sample of 257 positive cases in total. This results in a coverage of over 90 percent. The higher case rates in Italy are likely to be driven by more registered cases in the overall population, and because Italy's Serie A includes more teams (20 compared to 18 in Germany). The high coverage of identified cases should comfortably exceed the knowledge on infections in most industries and allows us to consider our results to be representative of the dataset at hand. We also conduct our analysis for a subsample of the data, in which we drop the observations of teams with anonymous cases. By doing that, we obtain a dataset of perfectly identified players. Our results are highly similar in this case. To further illustrate the case numbers among footballers relative to the overall population, Figure 1 provides information on player infections and 7-day incidences over time – i.e., the number of newly infected persons per 100,000 inhabitants. Infections evolve similarly over time. Figure 1 also highlights incidences close to zero in the summer break between the two seasons. This probably would have been the only period in which clubs could have kept an infection secret without the media recognizing the absence of a player. As the overall incidences were very low during this time, we suspect the number of non-identified but infected players to be, if anything, very low.

The 257 infections among 1,406 players imply that by mid-July 2021, 18 percent of all players had been infected. This exceeds the general incidence of cases in the age group of young adults in both countries. It is likely a consequence of persistent testing and extensive traveling. Additionally, both leagues implemented rigid rules for club and player behavior. The Bundesliga set in place compulsory testing once or twice a week

[7] A full list of all positively tested and known players is available on request.



The plots show the seven-day incidence for Italy (LHS) and Germany (RHS) over time (left y-axis). The seven-day incidence counts all cases over the last seven days and scales them on 100,000. Also, cases among players are given (right y-axis). Source country incidences: (Ritchie et al., 2021, data downloaded: 16.07.2021).

Figure 1: General Incidences and Player Infections

and before a match.[8] The Serie A required a PCR test before a match.[9] Hence, we are confident that we have a true picture of the overall infections.

For player and match statistics, we apply data from *Opta Sports*. The company is one of the leading firms for statistics in sports and has an official partnership with the Bundesliga and the Serie A.[10] The company tracks every player and all of his actions during a match using software that analyzes video records. Every action on the pitch is recorded and registered with the coordinates showing where it happened. We were able to gather information on which players participated in each match of the 2019/2020 and the 2020/2021 seasons, and how these players performed in a match. Hence, we are confident that we have the best data available to track the productivity of all the players.

Our dataset consists of 72,938 records from 1,406 players ranging over both seasons and leagues. These data encompass all players who played on at least one matchday of a season. Among these observations, 40,607 records track players who played in a certain match, i.e., we can construct within-match work performance for them. The remainder

[8] https://www.dfb.de/fileadmin/_dfbdam/226090-Task_Force_Sportmedizin_Sonderspielbetrieb_Version_3.0.pdf – one or two PCR tests per week depends on the severity of the infection process. One PCR test per week was only allowed in case the region or district of a club had a 7-day incidence < 5 per 100,000 people, which was hardly ever the case during the seasons. PCR testing is the most accurate form of testing for a virus with almost 100 percent sensitivity (Guglielmi, 2020).

[9] https://www.figc.it/media/123076/circolare-quarantena-calcio-def-2.pdf

[10] https://www.statsperform.com/team-performance/leagues-federations/. There also exists some literature that validates the quality of the data from Opta, see Liu et al. (2013).

covers players who were not nominated or substituted on the pitch at a particular match. Their observations will be included in the analysis at the extensive margin, i.e., whether a player plays. Table A1 in the appendix provides descriptive statistics.

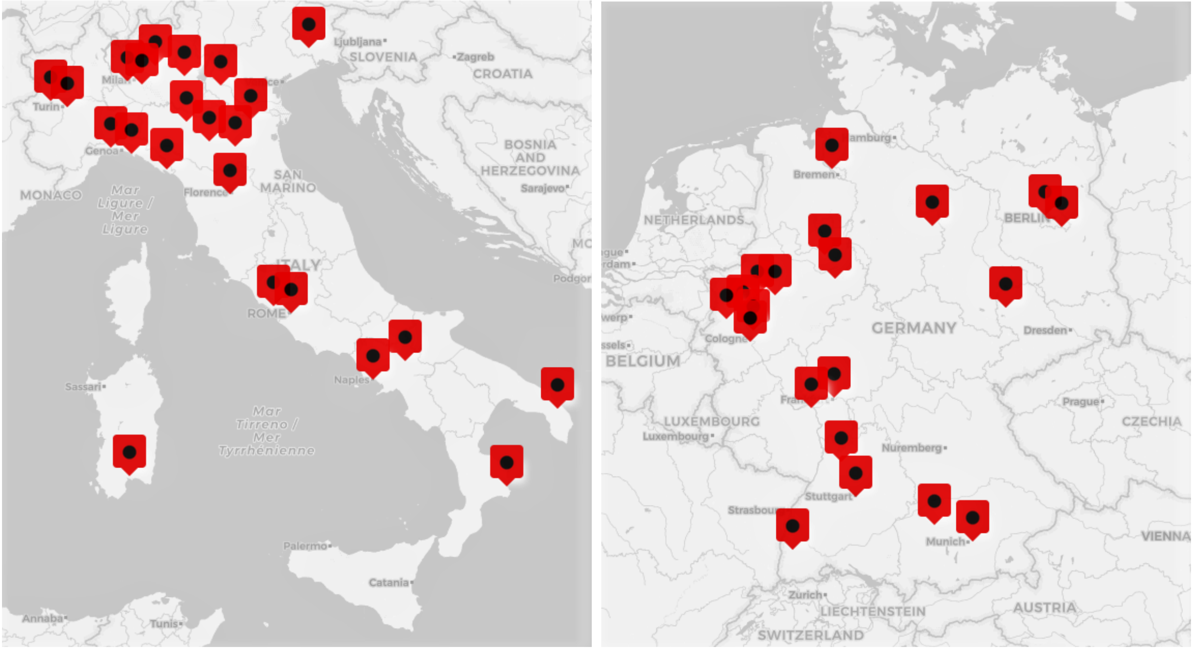
We further extend this already rich dataset with information on wages for Italy. Here, we build upon data collected by the 'La Gazzetta dello Sport,' the largest Italian daily newspaper. It provides data on the seasons 2019/2020 and 2020/2021 for all teams in the Serie A. For the earlier season, we fill missing information on teams with the salary reports for the season 2018/2019. In total, we are able to cover 78 percent of the player×season observations with salary data. We are not the first to use this source, Principe and van Ours (2022) have used 'Gazzetta' salary data as well. This source tends to truncate weaker players, but we still consider the data as valid and highly valuable for our analysis.

With an increasing likelihood of soccer players being or getting vaccinated in the season 2021/22, our analysis of the previous two seasons brings two advantages: First, we can track the unbiased effect of infections without the 'distortion' due to vaccinations, as the Serie A and the Bundesliga started vaccinations only after the end of the 2020/2021 season.[11] Second, vaccinations are likely to increase the degree of self-selection into treatment if some players prefer to remain non-vaccinated. Moreover, our still relatively short treatment period of 15 months (since the beginning of the pandemic) enables us to disregard sample selection issues, for example, that severely hit players may drop out of the top leagues. Contract rigidity in elite soccer ensures that most players remained with their clubs for the whole period.[12]

The comparison between infected – treatment group – and non-infected players – control group – is relevant in our setting. We match both groups and their characteristics with each other in Table 1. While the infection timing is arguably random for each player, there might be some selection into who gets infected or not. Indeed, we find some disparities in the performance measures. Players from Italian clubs are slightly over-represented in the sample of infected players. This might be due to the overall

[11] See for the Serie A (19.07.21): `https://football-italia.net/figc-wants-serie-a-and-serie-b-players-to-get-vaccinated/` and the Bundesliga (in German, 18.05.21): `https://www.kicker.de/dfl-empfiehlt-impfung-der-profis-fan-rueckkehr-realistisch-806070/artikel`.

[12] Older work by Frick (2007) reports an average contract length of 3 years in the Bundesliga.



The maps give the clubs' location (left: Italy, right: Germany). The maps capture clubs being part of the respective league in one or both seasons. Underlying maps by `www.openstreetmap.org`.

Figure 2: Location of the Leagues' Clubs in the Dataset

incidences, which have been much higher in Italy compared to Germany (as shown in Figure 1). Case numbers were particularly high in Northern Italy, where most of the clubs in the Serie A are located (see Figure 2). Furthermore, infected players seem to have played more often and longer prior to the treatment. They also performed better in terms of passes and touches per minute. There are no significant differences in age or other demographics, which might be important for the severity of the symptoms. Concerning positions, it seems that midfielders are over-represented.

We address the differences between the treatment and control groups by controlling for the player- and position-specific effects later on. We then also restrict samples to similar levels of quality to avoid weaker players in the control group biasing our findings and we find our results to be robust for using solely the subsample of treated individuals. Eventually, we perform propensity score matching to create a comparison group that is statistically indifferent to the group of infected players based on observables. All approaches lead to comparable results, so we are confident that sample selection does not drive our findings.

Statistic	Units	Non-Infected	Infected (Pre-Infection)	Δ (p-value)
Match Involvement/Performance				
Played at all	yes/no	0.539	0.659	0.000***
if played...				
Minutes Played	min	66.340	71.509	0.000***
Played Full-time	yes/no	0.484	0.564	0.001***
Passes/min	#/min	0.511	0.546	0.023**
Ball Recoveries/min	#/min	0.057	0.057	0.778
Touches/min	#/min	0.681	0.713	0.042**
Possession/min	#/min	0.491	0.526	0.017**
Dribbles/min	#/min	0.019	0.019	0.402
Aerials/min	#/min	0.038	0.033	0.021**
Shots/min	#/min	0.015	0.017	0.103
Demographics				
Age	years	26.550	26.886	0.351
Height	cm	183.350	184.268	0.049**
Weight	kg	77.273	77.754	0.339
Body Mass Index (BMI)	kg/m^2	22.966	22.879	0.376
Others				
Italian League	yes/no	0.541	0.636	0.013**
Goalkeeper	yes/no	0.043	0.052	0.567
Defender	yes/no	0.223	0.251	0.281
Midfielder	yes/no	0.150	0.225	0.001***
Striker	yes/no	0.077	0.087	0.519
Substitute	yes/no	0.506	0.384	0.000***

Columns (1) and (2) show the means of the respective variable for all observations of non-infected players and infected players (pre-infection). Column (3) reports the p-value of a two-sided t-test. Significant differences are indicated by stars as follows: *p<0.1; **p<0.05; ***p<0.01. The significance of differences between infected and non-infected players is obtained from simple regressions of each outcome on an intercept and a dummy for infected players pre-infection. Standard errors of these regressions are clustered on the player level. Variables which give performance per minute omit observations with zero minutes on the field which make up 232 out of 36,671 observations (approximately 0.6%).

Table 1: Descriptive Statistics for Non-Infected and Infected Players

3 Empirical Strategy

Infections can be modeled as a staggered treatment across players. To disentangle the effect of the infection from other shocks that may limit work performance, we compare infected players' performance before and after their positive test results with the evolution of outcomes of non-infected players. Hence, we apply a difference-in-differences estimation that controls for variation over time and across individuals.[13]

For this setting to be valid, several assumptions need to hold. Within our simple difference-in-differences setting, we need parallel trends of the treatment and control group in the absence of the infection. We have no reason to question this because there is no conceivable cause for the diverging evolution of productivity without COVID-19. Within the dynamic event study setting outlined later on, this corresponds to the requirement that treatment cannot predict outcomes before treatment. As our event study plots will show flat pre-trends, we consider the parallel trends assumption as not violated.

There may exist endogenous drivers of the individual infection risk, for example matches of the national teams that require more and particularly international traveling. The same holds for continental tournaments such as the UEFA Champions League or the UEFA Europa League. In both cases, for obvious reasons, stronger players are more affected than weaker ones. Furthermore, there might be higher exposure to infected people depending on the individual's social predilection for attending parties or public events. Hence, the risk of infection might not be completely idiosyncratic. However, random selection into infection is not necessary for identification, as identification is drawn from the timing of the infection. This should be exogenous for all types of players and unanticipated in the short run. Potential differences between treated and non-treated players will nevertheless be addressed in our robustness checks.

For reliable estimations in the simple difference-in-differences setting, we need no

[13]For this methodology a rapidly developing literature has emerged, which mainly addresses the distortions arising from staggered treatments in plain two-way fixed effects settings (see, e.g., the recent survey by de Chaisemartin and D'Haultfoeuille (2022) on this literature). The main critique is that treatment effects at a certain relative point of time to the treatment event might change with heterogeneity in real time. Related to the critique we later show that there is no significant change in the treatment effect between early and recent infections.

variation in the effect size of the treatment over time. This would not be true if, for example, a new medication had been developed that would have changed the impact of an infection. In general, there is no reason to believe that the work performance effects of an infection are constant over time, so we will analyze dynamic patterns in event studies.

Eventually, a difference-in-differences estimator requires that the treatment only causes partial equilibrium effects, or else we need the stable unit treatment variable assumption (SUTVA) to be fulfilled. As we find spillover effects within a team, there might be some confounding, which collides with the SUTVA. This does not invalidate but strengthens our empirical findings. In theory, it is a priori unclear whether a deterioration in a player's performance either causes an overall lower performance of the team or leads to an (over-)compensation of this deterioration. Indeed, we find strong evidence of the former. An increasing number of recovered players on the pitch decreases their team's performance disproportionately. This implies a negative effect on the control group. Hence, our estimates underestimate the true effect in absolute terms.

As we consider the identifying assumptions as fulfilled and the spillovers as innocuous, our model allows us to extract the treatment effect. We implement the regression setup

$$\text{Performance}_{pm} = \beta \, \text{Post-Infection}_{pm} + X'_{pm}\gamma + Z'\zeta + \epsilon_{pm} \,. \tag{1}$$

Performance_{pm} on the LHS refers to a set of performance or involvement measures of player p in match m. In our setting, this is, for example, a dummy capturing whether a player played at all, or the exact number of passes (in logs).

We use the number of passes as the main productivity measure for the intensive margin estimations. Individual performance in soccer depends on various physical health measures such as acceleration, condition, and endurance, but also the cognitive capability to position oneself optimally on the pitch. The number of passes is related to all of these measures and thereby suitably proxy the involvement of players in a match. Former papers on work performance in sports have also exploited the number of passes as a measure of interest (for example Carmichael et al., 2001; Lichter et al., 2017; Oberstone, 2009). Descriptive statistics on this measure can be found in Figure A1 in the appendix.

Results on other measures (e.g., touches and possession) – which also account for slightly different behavior – are provided later on as a robustness check. Hence, cross-validation with different measures should give a thorough picture of player performance.[14]

Post-Infection$_{pm}$ is the treatment dummy that takes the value 1 for all observations of a player after he has tested positive. Hence, β is our coefficient of interest. To account for variation in the cross-section and over time, we control for a large set of covariates X_{pm} and fixed effects (FE) Z. The vector X_{pm} contains a player's age, the plain and squared number of minutes played to capture non-linearities in time on the pitch, a dummy variable for a home match and one that distinguishes matches before and after the interruption of the leagues in Spring 2020. The vector Z includes player fixed effects, team-season, and opponent-season fixed effects as well as matchday fixed effects and an FE capturing variation before and after the interruption in Spring 2020. Doing this, we control for a general underlying performance effect during the COVID-19 pandemic for all players, as Santana et al. (2021) find a worse running performance after the restart in 2020 but an improved passing accuracy. Also, the exclusion of fans might have impacted player behavior during this period (Bryson et al., 2021). All of these FEs shall capture performance differences unrelated to an infection.[15] ϵ_{pm} is the idiosyncratic error term. We use heteroskedasticity-robust standard errors clustered on the player (i.e., the treatment) level to account for correlated residuals across a player's observations. To test the identifying assumption of parallel trends absent a treatment and to understand the dynamic nature of effects, we also apply an event study setting as a dynamic model:

$$\text{Performance}_{pm} = \sum_{\tau=\underline{k},\tau\neq 0}^{\bar{k}} \beta_\tau \text{ Post-Infection}_{pm,\tau} + X'_{pm}\gamma + Z'\zeta + \epsilon_{pm} \,. \tag{2}$$

This leads to several β_τ coefficients of interest. Subscript τ is the running index of leads

[14]Running performance would be another natural measure to study as COVID-19 is a respiratory infection. However, Lichter et al. (2017) find running and pass performance to be highly correlated. We obtain player-match level running data for the Bundesliga and measure a correlation of 0.64. Later on, we use a variable similar to running performance that shows a significant drop after an infection as well.

[15]We experimented with several reasonable FE combinations. All results go in the same direction. A battery of FE combinations is discussed later on in the paragraph on robustness checks. Plots are provided in Fig. A10 in the appendix.

and lags. We bin these one-day binary variables to group dummies of 75 days. Endpoints are binned and hence include all observations which lie beyond the second-last bins on either side (Schmidheiny and Siegloch, 2020). Our results are robust to different specifications of the effect window size. We mainly plot bins up to 225 days before and after infections and bin all observations beyond these thresholds in the outer bins to have sufficiently many observations in each bin. As infections are hardly anticipated and voluntary precautions are only possible with limitations in the world of professional soccer, we do not struggle with a number of identification challenges that have been addressed in the context of COVID-19 studies, such as voluntary precautions, anticipation, and variation in policy timing (see, e.g., Goodman-Bacon and Marcus, 2020).

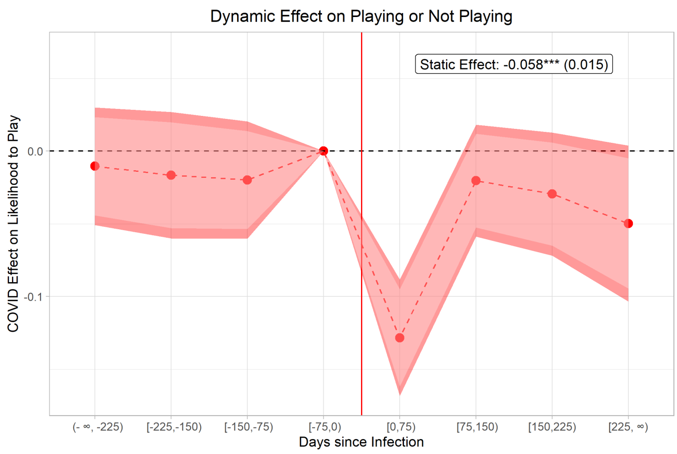
4 Results

Our analysis takes two steps. First, we investigate whether a COVID-19 infection has a short- or long-term impact on the participation of players. Subsequently, we look at within-match performance after an infection. The intensive margin could underestimate the persistent effects of a COVID-19 infection as players hit the most might not play at all. Hence, analyzing both effects is indispensable and might offer some intuition on performance-related mechanisms. While the main measure of interest is within-match work performance, the effect at the extensive margin helps to understand the severeness of the post-infection work performance drops.

Extensive Margin: First, we analyze the effect of a COVID-19 infection on the probability of playing and the number of minutes played. Figure 3 reports the corresponding estimates. From the simple effect, in the upper right of the plot, we infer that players have a 5.7 percentage-point lower probability of playing. However, effects appear to be mechanical, mainly driven by the initial weeks after an infection, when quarantine breaks do not allow a player to participate in a match. The observed drop in playing frequency becomes quickly insignificant again, but does not fully return to its former level. These results indicate that players marginally experience persistent effects on their likelihood to play. A flat pre-trend validates our finding.

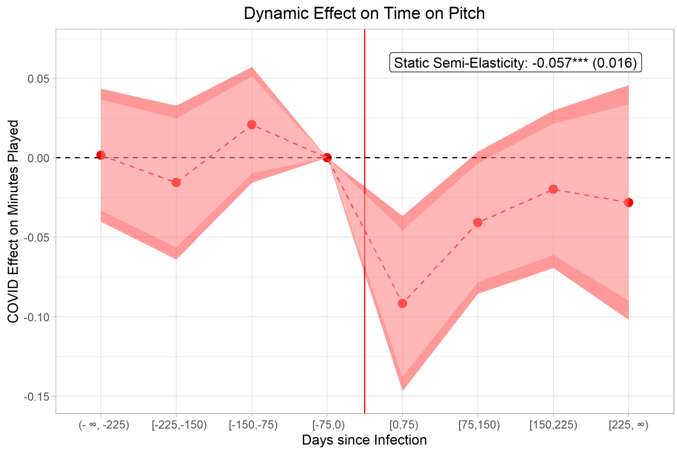
Figure 4 shows the corresponding effect on minutes played by players who play. Immediately after the infection and his return on the pitch, a player spends an average of six minutes less on the field than before – this corresponds to a decrease of almost ten percent. It indicates that several players might have been used only as substitutes leaving the pitch earlier or entering it later. This might point to a general fitness problem of the players and make it more likely that work performance effects at the intensive margin might be underestimated as the player might be substituted off before the severest effects kick in. The effect is visible right after an infection but is quite long-lasting. Only after approximately 150 days or five months of play does fitness return to a level that does not significantly differ from pre-infection match times.

Our findings at the extensive margin are confirmed by an increasing likelihood of being substituted on and off the pitch after an infection. On average, players play for a shorter time which may signal insufficient fitness to participate for 90 minutes. The respective event studies can be found in Figure A2 in the appendix. In general, our



This figure plots the OLS (LPM) estimated coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. An equivalent plot with a 30-day bin size (for a better description of short-run effects) can be found in Figure A3 in the appendix. Standard errors are heteroskedasticity-robust and clustered at the player level. 90 and 95% confidence intervals are given by the red-shaded areas. The dependent variable is a dummy indicating whether a player played or not.

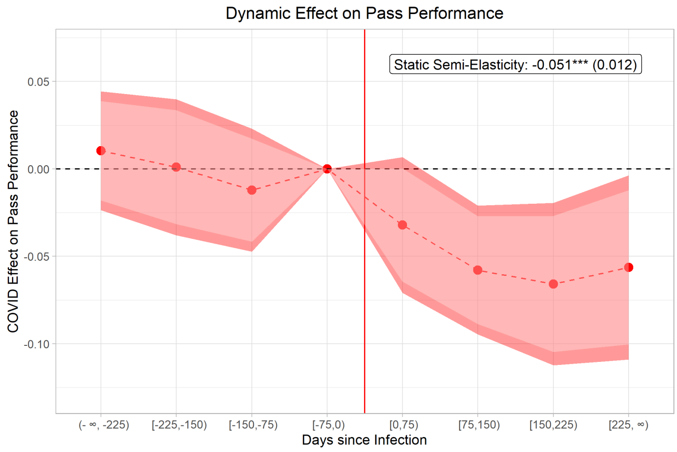
Figure 3: Dynamic Effect on Likelihood to Play



This figure plots the OLS estimated coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. An equivalent plot with a 30-day bin size (for a better description of short-run effects) can be found in Figure A3 in the appendix. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variable is ln(minutes played) conditional on having played.

Figure 4: Dynamic Effect on Minutes Played

results on the effects at the extensive margin indicate a return to initial levels of infected players over time. Either the players return to the former work performance levels or badly performing players re-enter the subsample of players on the pitch. This would shift the treatment effect from the extensive to the intensive margin, such that worse work performance effects should be observed over time in within-match data.

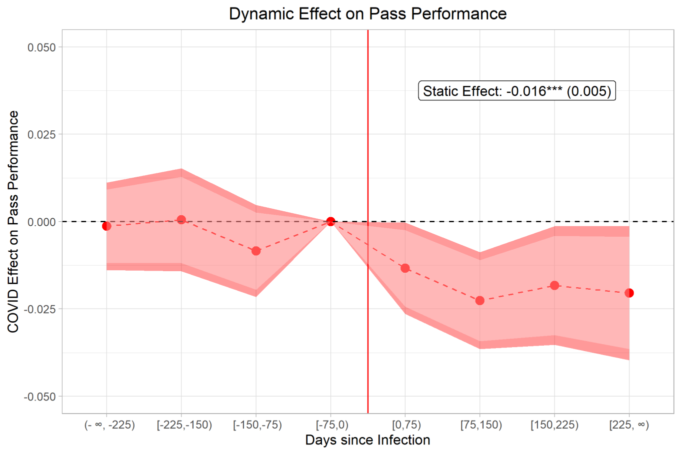


These figures plot the OLS coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variable is ln(passes). Additional work performance measures can be found in figures A5 and A6 in the appendix.

Figure 5: Dynamic Effect on Within-Match Work Performance

Intensive Margin: We next take a nuanced look at a player's performance conditional on being on the pitch. As previously outlined, the main building block of our productivity analysis is the number of passes, as shown in Figure 5. Besides that, we provide results on two related performance measures, possession and touches in Figure A4 in the appendix. Figure 5 presents the corresponding event study providing the dynamic estimates of a COVID-19 infection on within-match performance. This plot, as well as the additional measures in the appendix, show rather flat pre-trends. We find a highly significant simple difference-in-differences effect of -5.1 percent. Thus, we can precisely identify deterioration in productivity following a cured COVID-19 infection. This effect is not transient but remains notably negative over the course of time. We consider this as causal evidence of COVID-19 infections causing long-lasting productivity drops for infected individuals.

This finding is surprisingly coherent with medical research from Switzerland that finds 'long COVID' symptoms to be persistent over seven to nine months for a third of all infected persons in the analyzed sample population (Nehme et al., 2021). As an alternative outcome measure, Figure 6 presents the effect over a COVID-19 infection on an observation's rank in the pass distribution. It is apparent that the persistent deterioration combined with flat and insignificant pre-trends remains in place. A player slides down in the relative productivity ranking by 1.6 percentiles.



These figures plot the OLS coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. Instead of ln(passes), this regression uses the ln(rank) of the amount of passes played by a player during a match.

Figure 6: Dynamic Effect on the Ranking of Infected Players

Interestingly, we also see work performance partly fall over time, while the effect stabilizes after some months post-infection. This gives rise to two remarks: First, players do not return to their former level within the period of observation. Second, the reduction over time also captures the return of infected players to the pitch as there is more involvement at the extensive margin after several months. It may be possible that players who still suffer from weakened performance, eventually return to the pitch and negatively affect the treatment effect over time.

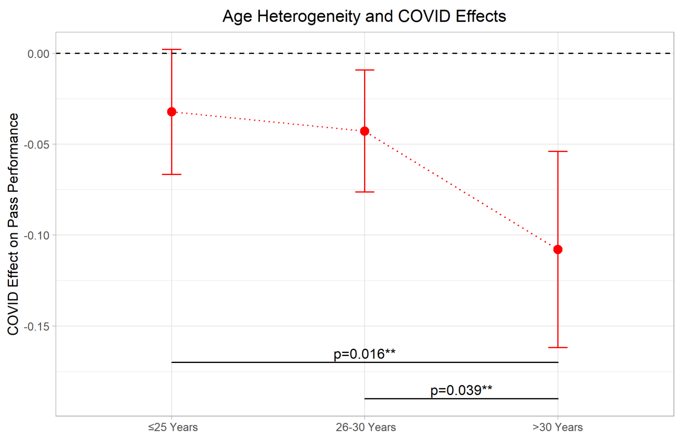
Furthermore, Table 1 provides some evidence on differences between infected and non-infected players. To ensure that the intensive margin effect is not driven by this, we address the potential issue of sample selection with a propensity score matching procedure. We do this by nearest neighbor matching without replacement. The matching takes place within matchday and position, i.e., for a midfielder infected right before matchday 16 in season 20/21, we look for a midfielder equivalent on matchday 15 of this season. We include team×season, matchday×season and position FEs in the matching probit regression. We do not allow a matching of infected players with those players who become infected at a different point in time or with players who have not played a single minute up to the respective matchday.

Table A2 in the appendix shows that the generated control group does not differ in any observable dimension from the treatment group. Re-estimating our main regression for the intensive and the extensive margin using the fully balanced sample, we get results that are very similar to our baseline results (as shown in Figure A16 in the appendix).[16]

Effect Heterogeneity: We do not only find a significant and persistent deterioration in work performance but also heterogeneity in several dimensions. While an infection's effect on the underlying health status should be quite homogeneous in the homogeneous group of players, the consequences of changes in health might impact player performance differently. First, throughout the COVID-19 pandemic, age has been one of the main determinants of how likely an infected person is to develop symptoms or to even die (e.g., Gallo Marin et al. 2021). It seems natural to investigate whether older players also suffer more from an infection. Even though professional athletes in their thirties cannot be compared to the overall elderly population, their recovery may take longer and symptoms may be more persistent. Figure 7 provides some intuition that especially players aged 30 and over face the strongest performance drops of over 10 percent. In comparison, younger players up to 25 years of age are only affected marginally. Both effects are statistically significantly different from each other as the Wald test provided in Figure 7 highlights.

Second, COVID-19 infections are often associated with additional fatigue. Therefore, we investigate whether players need more time to recover from a match post-infection. Put differently, it may be that post-infected players perform worse if the rest break between two matches they played in is insufficient. We compare the treatment effect for different lengths of rest breaks. In Figure 8, we show that the treatment effect is especially strong for short breaks of up to three days. The shortest breaks are in terms of the productivity effect statistically significantly worse than longer gaps from four days

[16]Figure A16 also provides the extensive margin effect on the likelihood to play.



The plot displays OLS interaction effects between the post-infection dummy and age groups included in equation (1). Dependent variable: ln(Passes). SEs: Heteroskedasticity-robust and clustered at player level. The 95% confidence bands are given. p-values: Wald tests for difference between the respective estimates.

Figure 7: Effect Heterogeneity: Age Effects

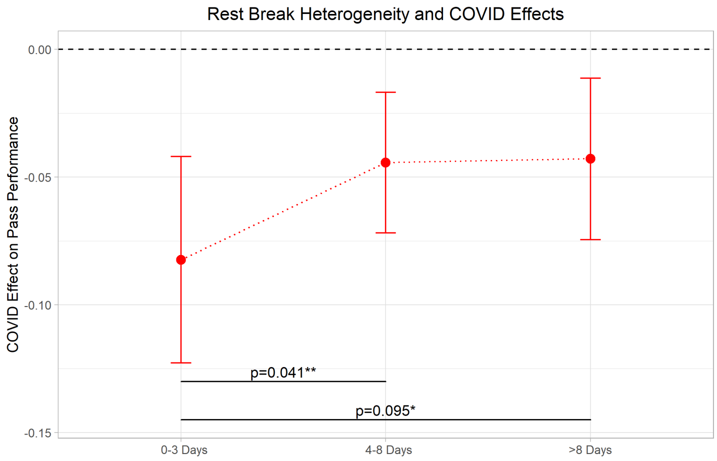
onward. Our results indicate that post-infected players perform better - though not as well as before the infection - if there is enough regeneration time.

We also analyze heterogeneity with regard to positions, team and player strength, and infection timing. Results are intuitive as we find stronger effects for more enduring positions or weaker players.[17] Also, there seems to be no difference between effects from early or late COVID infections, i.e., there tends to be no treatment effect heterogeneity in real time. All plots can be found in Figure A8 in the appendix. There, we also provide equivalent heterogeneity analyses for the extensive margin (Fig. A9). The results are very similar.

Lastly, we study the impact of the severity of symptoms on the subsequent performance. As we cannot measure or observe the exact symptoms infected players suffered from when being isolated at home, we use the length of the break between a player's infection and his return to the *squad* as an approximation. This is because the return to the squad (without necessarily having played) implies that the player is able to play again, i.e., free of symptoms.[18] Moreover, there is less of a quality-based selection issue

[17] The heterogeneity analyses on positions also show that the treatment effect is not driven purely by substituted players but also by starters.

[18] A squad typically consists of 20-22 players (depending on the league) and, hence, encompasses more players than actually play (typically the starting line-up (eleven players) plus 0-5 substitutes).



The plot displays OLS interaction effects between the post-infection dummy and different recovery breaks included in equation (1). The length of a break is calculated on the player level, i.e., the number of days between two matches the player has played in. Dependent variable: ln(Passes). SEs: Heteroskedasticity-robust and clustered at the player level. The 95% confidence bands are given. The first observation of every player is dropped as no recovery break to previous matches can be calculated. p-values: Wald tests for difference between the respective estimates.

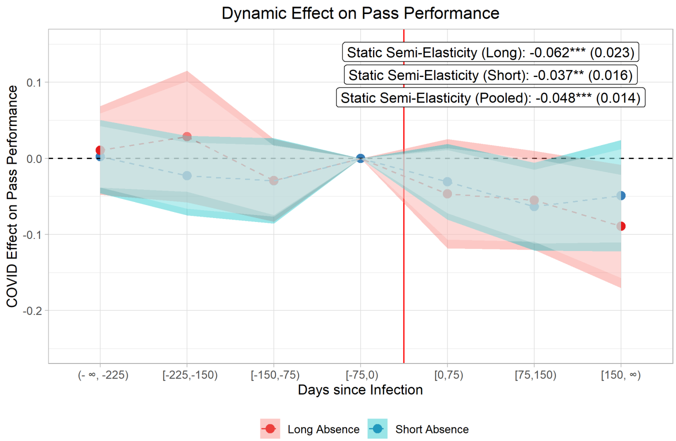
Figure 8: Effect Heterogeneity: Recovery Break Effects

into the squad than into the players on the pitch. We also restrict the sample to those players who could actually play a match soon after the official end of quarantine. In addition, we drop players who suffered a different injury right after their infection.

Finally, we drop the 'worst' 10 percent of the remaining, infected players with regard to playing time as for them selection into the squad might have been an issue. We conducted several tests to ensure that there is no strong linkage between player quality and return velocity in the remaining squad. As one would expect, the point estimate for players with a longer break is higher in absolute terms than for shorter absences, as Figure 9 shows.[19]

Comparison to Other Injuries: It may be that players and teams treat COVID-19 just like any other injury – a player rests for a while and returns to team practice afterward. If COVID-19 infections have performance effects beyond typical injuries and illnesses,

[19]Though, the point estimates are not significantly different from each other in this reduced sample (Wald test: $p = 0.345$). Also note that the effect of the pooled sample differs from the effect shown in Fig. 5 as we exclude those players who suffered an injury directly after the infection, never played again after being infected or got infected directly before the long interruption in Spring 2020, a summer or a winter break as this would distort the approximation. Hence, we only consider players who actually had the chance to play in a match in the two weeks after the end of quarantining.



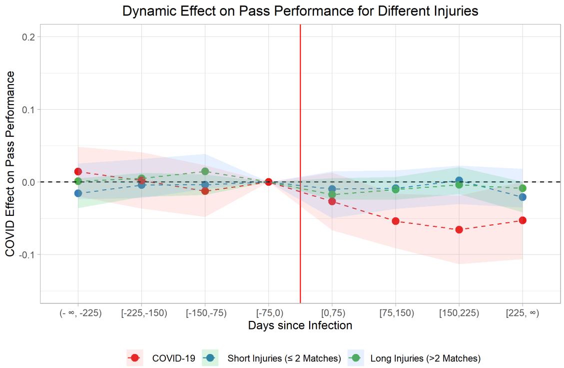
The plot displays the intensive margin effect of an infection depending on the severity of an infection, approximated by the length of the interval between infection and return to the squad. Sample split at the median. $N = 140$ infected players. The last two bins $[150, 225)$ and $[225, \infty)$ are pooled to a joint endpoint due to the otherwise very small number of observations. Dependent variable: ln(Passes). SEs: Heteroskedasticity-robust and clustered at the player level. The 90 and (darker) 95% confidence bands are given.

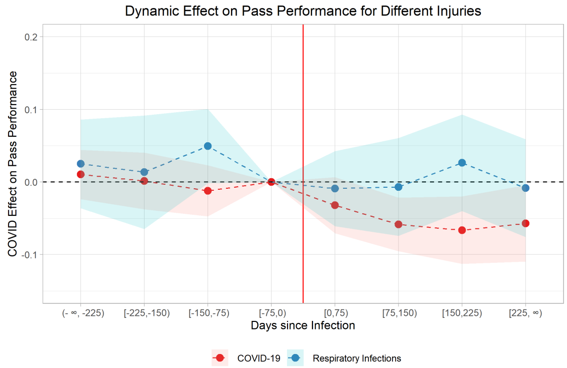
Figure 9: Effect Heterogeneity: Severity of an Infection

this would emphasize the relevance and uniqueness of this particular virus infection. We investigate this by analyzing the work performance effects of all other injuries which happened during our sample period. They range from muscle and ligament injuries to simple colds. In Figure 10, we distinguish the effects of a COVID-19 infection from both short and long injury breaks. We split the data at the median injury duration (2 matchdays) to investigate heterogeneity in injury length. Unlike for COVID-19, we find no comparable work performance effects for other injuries – neither after short nor long ones.

Moreover, we are able to exploit information on the exact type of injury in our sample. We specifically identify absences that are related to similar diseases and infections like COVID-19, such as colds, influenza, and respiratory ailments.[20] This gives us some 100 occasions where players are absent owing to such reasons. The right plot of Figure 10 provides the comparison between the COVID-19 repercussions and the respective effect of other respiratory infections over the course of time. Again, it is evident that the COVID-19 infection causes more severe productivity effects than sicknesses affecting

[20]Note that COVID-19 infections are sometimes free of any symptoms, while the other respiratory infections are likely to be symptomatic as there has not been any testing.





The plots give the time-specific COVID-19 or injury effects on the number of passes on the player and match level estimated by OLS. SEs: Heteroskedasticity-robust and clustered at the player level. We take the first match missed by an injured player as the starting date of the injury. The regression set-up follows equation (2). Under 'respiratory injuries' we subsume colds, influenza infections, pneumonia, and bronchitis (data from www.transfermarkt.de). We report 95% confidence bands in both plots.

Figure 10: Time-Specific COVID-19 and other Injuries' Effects on Performance

similar parts of the organism.[21] This corresponds to earlier research of Keech et al. (1998), who find a work performance deterioration for influenza-like sicknesses after returning to the workplace only over 3.5 days on average.

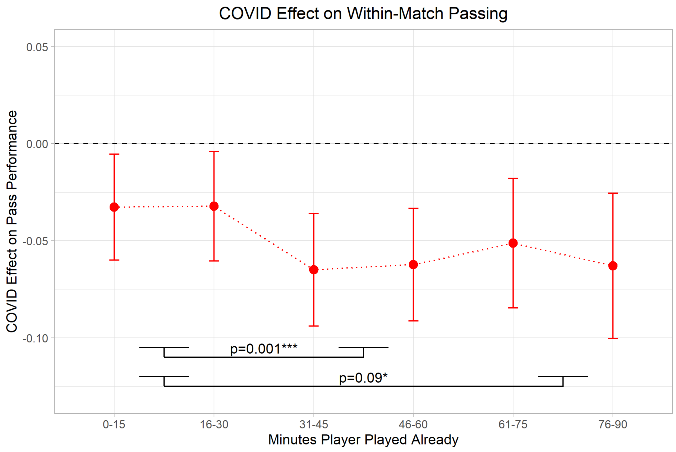
Eventually, our findings also support the external validity of our setting. There are no effects among professional soccer players subsequent to an 'ordinary' respiratory infection and hardly any among the general labor force. Hence, the singularity of professional athletes does not immediately imply differences in productivity outcomes. In turn, the significant deterioration following a COVID-19 infection may not reflect an overstatement of the effect in the general population due to players' stronger dependence on their respiratory system.

[21] To test for such differences for the long-run effect, we conduct Wald tests on the pooled last two bins in each event study regression. As there can be multiple injuries per players, so that the treatment is non-absorbing, we cannot compare simple difference-in-differences coefficients. For the differences between a COVID-19 infection and short and long injuries (as shown on the LHS of Fig. 10), the pooled estimates of the last two bins differ signficantly from each other for COVID-19 infections in comparison to short injuries ($p = 0.031$) and to long injuries ($p = 0.039$). On the RHS of Fig. 10, the long-term difference between the effect of COVID-19 and other respiratory infections is significant with $p = 0.075$.

Within-Match Mechanism: We can identify substantial and persistent effects of a COVID-19 infection on player performance. Our granular data allow us to study not only outcomes by matchday but also performance within a match. As COVID-19 is a respiratory infection and soccer requires endurance in physical activity, it may be likely that players perform worse in the later stages of a match. We investigate this in Figure 11, in which we plot time-specific COVID-19 effects by decomposing the match of a player into a maximum of six parts of 15 minutes in length each.

The results show decreased physical work performance from the first minute on the pitch onward in cases in which a player has recovered from an infection. Furthermore, performance declines throughout a match. While the effect seems to be stable at around -3 percent in the first 30 minutes, post-infected players face a deterioration of some additional 3 percentage points in later phases. This deterioration is statistically significant on the 1% level for the second 30 minutes relative to the first 30 minutes played and significant on the 10% level for the last 30 minutes relative to the first thirty minutes played by an individual previously infected. Such a downward trend would be in line with COVID-19 affecting the player's endurance. Note that Figure 11 shows relative time. Hence, especially the first two bins capturing match time up to 30 minutes also encompass players that have been substituted *on* the pitch in the second half of a game. Even though they play for a shorter length of time and know this in advance, i.e., they do not need to manage their physical energy to last the full 90 minutes, their performance is lower compared to their non-infected peers.

Again, this emphasizes that we are likely to estimate a lower threshold of the treatment effect in absolute terms and that a COVID-19 infection causes a non-negligible deterioration in performance. Additionally, it addresses the external validity of this study, as fewer minutes played might better correspond to 'real world' occupations. Also, players who perform worse during later parts of a match might be substituted off earlier, so that their negative contribution at the end of matches might not be observable. Hence, the extensive margin effects might hide an even steeper downward trend throughout the match.



The plots show the time-specific COVID-19 effects on ln(passes). The x-axis shows the number of minutes a player has already been on the field. The y-axis documents the effect on the outcome variable. Standard errors are heteroskedasticity-robust and clustered at the player level. The 95% confidence bands are given. The regression setup is very similar to (1) estimated via OLS except for additional interactions of the COVID-19 term with the 15-minute time slots, which also results in up to six observations per player and match (for each time category if on the field) instead of one aggregate observation. p-values: Wald tests for difference between the respective estimates. The upper p-value compares the first 30 minutes (bins 1 and 2) with the second thirty minutes (bins 3 and 4). The lower p-values compares the first 30 minutes with the last 30 minutes (bins 5 and 6).

Figure 11: Time-Specific COVID-19 Effects on Within-Match Performance

Spillovers on Team Performance: Essential in team collaborations is the aggregate outcome of all individuals while the aggregate performance may differ from the sum of its components. A crucial question is whether the deteriorated productivity of post-infected players creates spillover effects on other players on the pitch. Hence, is a player's performance also affected by others' health shocks? We noted that treated players make fewer passes. Hence, teammates might be less involved in the match as well. Alternatively, they could compensate for the decreased performance of infected fellow players by taking more responsibility and being more involved in the match.

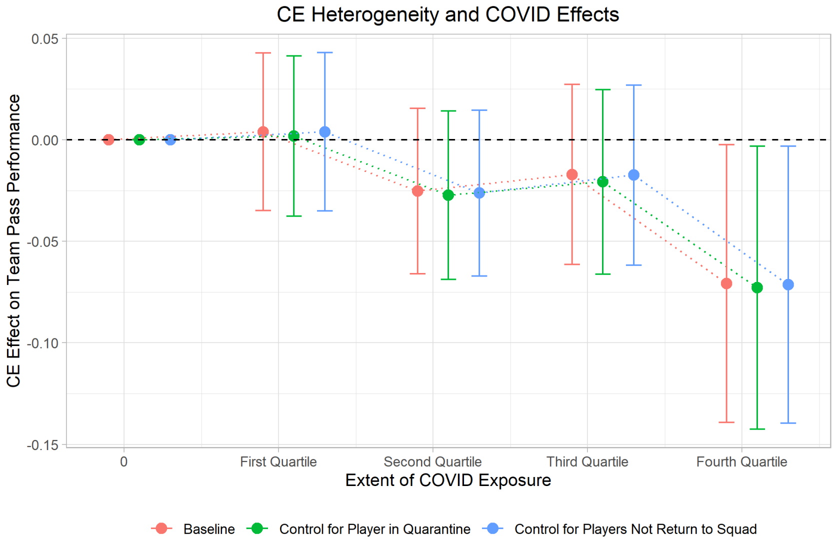
Stepping back, this issue directly corresponds to economic research on team production or else how a worker's effort affects her coworkers. To only name a few papers, Ichino and Falk (2005) find positive spillovers in a lab experiment, Mas and Moretti (2009) confirm the magnitude of this effect in a quasi-experimental setting with field data. They particularly investigate the arrival of highly productive coworkers. In contrast, Azoulay et al. (2010) as well as Waldinger (2011) study negative productivity spillovers. The former analyze the sudden deaths of academic 'superstars,' the latter investigates the forced expulsion of Jewish researchers in Nazi Germany. While Azoulay et al. (2010) find a significant negative effect in quality-adjusted publication rates, Waldinger (2011) does not. In the field of sports, Guryan et al. (2009) investigate the impact of a golf player's performance on the partner he plays with but do not find a sufficient impact. However, this is no team but rather rival performance. Our paper adds to this literature a large-scale natural experiment for workers that need to collaborate strongly in a high-stakes environment with large group sizes.

Technically, we address the spillover issue by analyzing a team's performance depending on its exposure to COVID-19 infections. More specifically, we proxy a team's exposure to COVID-19 by the number of players recovered from an infection as a share of the overall team size (at any point in time before a match) at the match level. We construct the variable 'COVID-19 Exposure' as:

$$CE_{tm} = \frac{\sum_{p \in t} \text{Post-Infection}_{pm}}{\#\text{Players}_{tm}} \,. \tag{3}$$

The numerator is the number of infected players of team t on matchday m. The denominator is the overall number of players of team t at match m, i.e., the squad size. Figure A13 in the appendix displays the distribution of the positive values of this variable. In almost half of the team-match observations, recovered players were involved.

Figure 12 displays the simple reduced-form effect of CE on the logarithmic cumulative pass performance of a team (red). We separate CE into four equal quartiles for $CE > 0$. The decline in performance is increasing but is only significant for the last quartile – $CE \in [0.241, 1]$, which encompasses an exposure of, on average, 35.2 percent recovered players (out of an average team size of 26.58 players). Hence, one additional infection does not have a constant marginal effect. This could be relevant for other industries relying on collaboration in team tasks, too. Research on the direct health effects of COVID-19 typically does not consider such indirect mechanisms. Observable deterioration in team performance in the largest quartile corresponds to the finding that performance losses due to sickness absenteeism of employees exceed their wages (Pauly et al., 2002; Zhang et al., 2017).



The plot shows the effect of CE on team performance measured in ln(passes) estimated by OLS. We compare teams with $CE = 0$ to an exposure in four quartiles, which have the intervals $(0, 0.077)$, $[0.077, 0.130)$, $[0.130, 0.241)$, and $[0.241, 0.500]$ empirically or else $[0.241, 1]$ theoretically. The means are $\overline{CE}_{(0,0.077]} = 0.050$, $\overline{CE}_{(0.077,0.130]} = 0.096$, $\overline{CE}_{(0.130,0.241]} = 0.191$, and $\overline{CE}_{(0.241,1]} = 0.352$. The red bars capture the baseline CE effect on team performance, the green bars additionally control for #players currently in quarantine, the blue bars control for #players not being part of the squad following a COVID-19 infection. All regressions includes controls for home/away matches, ghost matches, the opponent's COVID exposure (transformed by the inverse hyperbolic sine transformation) and team-season FE, opponent-season FE and matchday FE.

Figure 12: Effects of COVID-19 Exposure (CE) on Team Performance

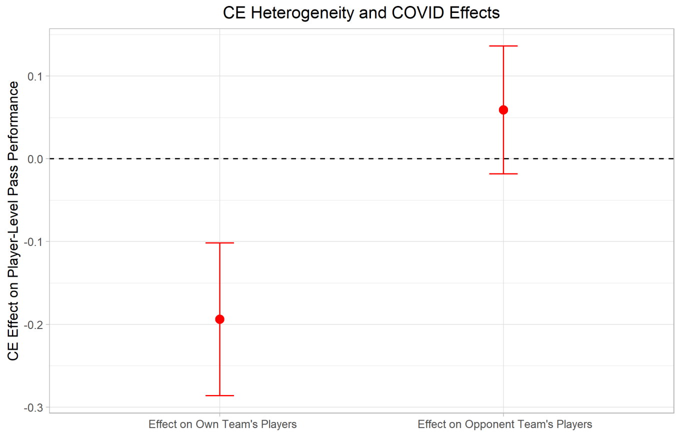
Other than in that research, our treated individuals are not necessarily absent but are often on the pitch. In the blue and green case, we also control for different measures of how many players missed a match due to quarantining. This should isolate the spillover effects of infected players on the pitch from pure composition effects due to missing quarantining players. If anything, the green and blue-colored estimates in Figure 12 highlight that the negative team spillovers are most likely related to productivity decline, when infected players are on the pitch, and are not caused by a composition effect. The latter captures a potential decline caused by the pure absence of players and their replacement with weaker substitutes. As we control for the number of players currently in quarantine (green) and not being part of the squad following a COVID-19 infection, one can see that the estimates hardly change. Overall, it might be the case that a team can compensate for small declines in their team members' contributions but not for larger ones. The deterioration in performance for a $CE \in [0.241, 1]$ amounts to 7.08

percent, while the mean exposure in this interval is only roughly one-third. This is strong suggestive evidence of spillover effects well beyond the individual effect.

Our variable definition allows us to capture the direct effect of weaker performance *on* the pitch as well as performance deterioration due to missing players because they have been hit severely by the infection. Hence, CE captures both extensive and intensive effects. As we argue that non-infected players perform worse due to their underperforming teammates, we have an affected control group, which confounds the estimates of a difference-in-differences setup. However, this only implies that our results on individual effects should be interpreted as lower bounds as there might be performance drops related to the treatment in the control group as well. Interestingly, previous research on performance deterioration on the soccer pitch due to external influences did not find such spillover effects (Lichter et al., 2017). These effects might be unique to COVID-19.

Figure 13 reports the elasticity of an increase of the own team's COVID exposure to own player and opponent player performance. Individual-level productivity is negatively affected by the own team's exposure while opponents' performance is not significantly changed. An increase of about 4 percent COVID exposure (corresponds roughly to one additional infection in an average squad) again reduces player performance by about 0.8 percent. The individual-level COVID effects of infected players remain unaffected by the inclusion of the exposure measures in the individual-level data.

The results over the course of a match for individual players displayed beforehand in Figure 11 hold for the aggregate team performance as well. Figure A14 in the appendix provides estimates of the time-specific effect of higher exposure to COVID-19 infections within a team on pass performance. We again find that the effect of more post-infected players on the field especially arises in later stages of the game, even though the simple semi-elasticity for the own team is only significant at the 10% level. The marginal effect of -0.199 describes a hypothetical change of $\Delta CE = 1$ and corresponds to the basic effect in Figure 13. Overall, Figure A14 also provides no evidence of relevant spillover effects on the performance of the opponent team. Jointly with the basic difference-in-differences results that confirms spillover effects to only occur within a team. As we find a negative effect of COVID-19 across teams, this is additional evidence of the individual-level findings since the SUTVA applies at the team level.

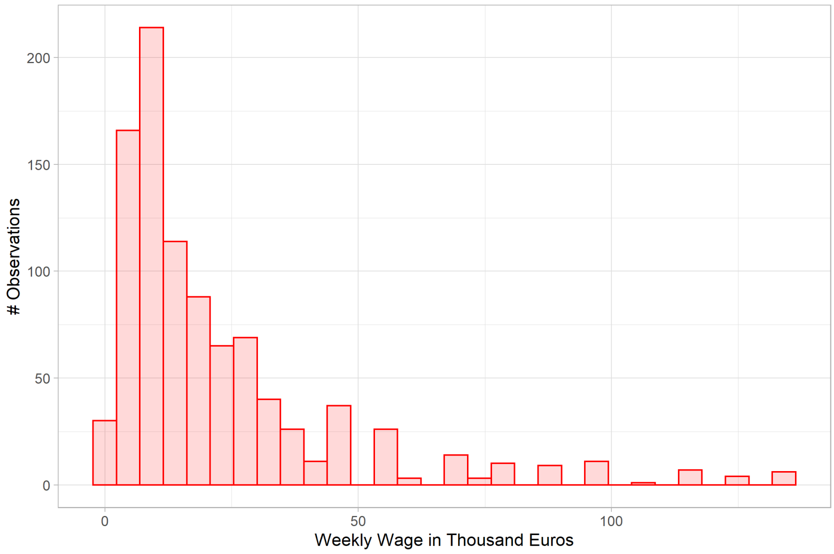


These figures plot the OLS coefficients β_τ of the simple difference-in-differences regression following Equation (1). The 95% confidence intervals are given. The dependent variables are ln(passes). The logarithmic specification excludes observations with zero passes. The independent variables added to the standard model used above is the hyperbolic sine transformation of the players' team COVID exposure.

Figure 13: Basic Effect on Team-Level Performance

Relevance of Productivity Measures: While our performance measures such as the time on the pitch and the number of passes are highly relevant for soccer players, it is less clear to which extent a change in these measures corresponds to more general labor market outcomes. The most prominent criterion is wages. On the one hand, they reflect worker productivity. On the other hand, they essentially determine living standards. We subsequently provide evidence that the number of passes (intensive margin), as well as the likelihood to play (extensive margin), correlate with players' wages.

To do this, we use wage data of Serie A players at the player-season level from the 'La Gazzetta dello Sport', Italy's most prominent sports newspaper. We assemble annual wages for 78% of all player-season observations in the Italian league. The relationship between wages and passes per minute or the likelihood to play is positive and statistically significant conditional on player demographics, player-specific measures such as his position, or the share of matches a player was part of the starting line-up. Figure A11 in the appendix visualizes these correlations.[22] A 5.1% decrease in the pass performance (as in our baseline results) translates into around a 10% decrease in wages across teams and a 2% decrease in wages within a team (including team fixed effects). To address potential non-linearities in the wages of soccer players, we show that the conditional correlations also hold for a nonlinear specification.



The plot shows the logarithmic weekly wages of players per season in the Italian Serie A as reported by the La Gazzetta dello Sport up to the 99% percentile.

Figure 14: Weekly Wages of Serie A Soccer Players

Given the correlation between our main productivity measures and players' wages, we investigate how the decline in performance would translate into monetary terms. As wages are typically rigid, this is a rather hypothetical exercise, but it helps to quantify the effect accordingly. Figure 14 shows the distribution of weekly wages for the Italian Serie A. The median *weekly* wage is EUR 15,384.60 but wages are widely dispersed – the 25th and 75th percentiles are EUR 7,692.30 and EUR 28,846.20, respectively. At the median, a downgrading of 1.6 percentiles in the productivity ranking as in Figure 6 approximately corresponds to a 6.25 percent decrease in the actual wage. This is in the range of our estimates from the partial correlation analysis above and would relate to a weekly loss of EUR 961.54 or EUR 50,000 per annum.

[22]We can additionally show that the conditional correlation remains significant after including team fixed effects (as shown in Fig. A12). Thus, passes per minute continue to be a significant predictor of wages even within a team.

Generalization of Results: A concern regarding our novel findings is that elite soccer players may differ from the general population. They are younger and fitter than the average worker, however, they work in a physically demanding occupation. While the former might imply more severe productivity effects of an infection for the average – less fit – individual, the latter might imply a milder effect. Therefore, assessing whether our findings constitute a lower or an upper bound for the population effect seems inaccurate. Yet, we carefully lay out why the existing differences may not have a sizable impact on observed workplace productivity.

Our paper studies long-run effects of COVID-19 infections after recovery. The prevalence of *post-acute* COVID-19 symptoms, as well as their development over time across age groups and fitness, thus, might be more relevant than differences between athletes and average workers during the actual infection. We focus on research that is not purely based on hospitalized patients, since these patients are a very selective sample of mostly elderly people who had left the workforce (Halpin et al., 2021; Huang et al., 2021; Nalbandian et al., 2021; Group, 2022). Research particularly conducted among athletes reveals that such well-trained individuals can also suffer from post-acute, persistent, non-cognitive symptoms (Brito et al., 2021; Hull et al., 2022; Ribeiro Lemes et al., 2022). More general studies on the long-run impact on non-hospitalized patients cannot identify a monotone relation between age and the prevalence of symptoms in a sample of symptomatic and asymptomatic cases (Whitaker et al., 2022). Similarly, Bliddal et al. (2021) and Tran et al. (2022) do not find a difference in the level and process of symptoms over time between individuals below and above 40 years. Blomberg et al. (2021) and Moreno-Pérez et al. (2021) do not find a robust relation between age, fitness, and persistent symptoms either. Thus, the evidence on non-hospitalized patients does not support that the prevalence of 'long COVID' robustly differs with age and fitness.

While not every job is as physically demanding as the occupation of soccer, there still exists a wide range of industries that rely on physical work as well. The construction sector alone employs millions of workers, 1.8 million in Germany, 1.3 million in Italy. The physically demanding sector of health and social work encompasses 4.9 million or

15 percent of all German jobs.[23] Despite technological advances, these sectors continue to be vastly fueled by the physical labor input of their employees that cannot be easily substituted by machines.

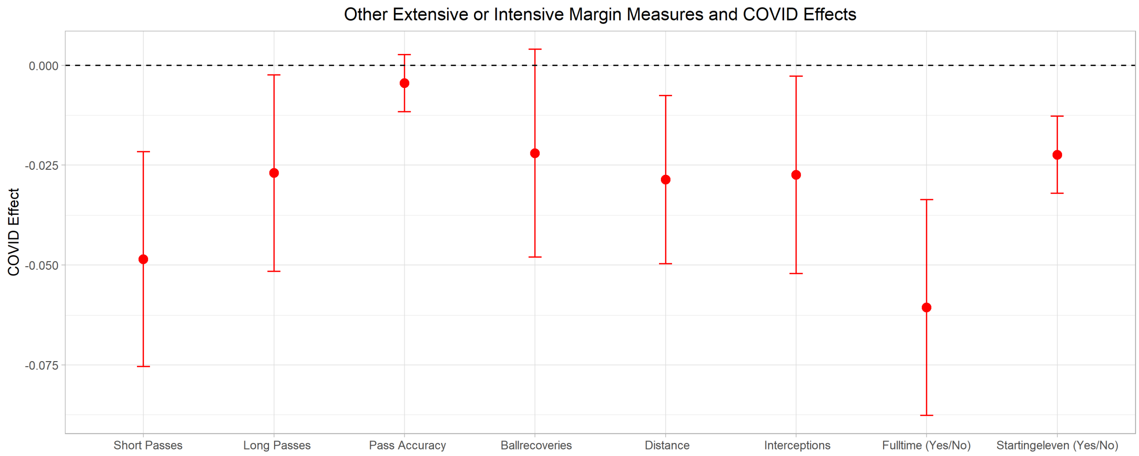
We carefully conclude that professional soccer players are not as different from the average population regarding COVID-19 infections and their consequences as one might expect. If anything, the consequences of a COVID-19 infection may be similar – in qualitative terms – for soccer players and the average worker. One should keep in mind that our sample differs from most medical papers as we observe all *asymptomatic* infections. Given that the level of acute infections impacts post-acute symptoms (Peter et al., 2022; Whitaker et al., 2022), we estimate a real population effect for our sample which differs from results based on samples with unobserved infections or selection on symptoms.

Robustness Checks: We find similar results for several other performance measures at both the extensive and intensive margin. Figure 15 reports significant performance drops also for measures like running distance or the number of interceptions and reveals that performance effects are neither purely driven by short or long passes. Moreover, clear effects on the likelihood to play full-time or be part of the starting line-up are evident.

Moreover, even though passes appear to be a reasonable and strong measure for productivity, one might wonder about the effect of COVID-19 on goals as this is the ultimate purpose of all match effort. However, the average amount of goals scored per team and match in our dataset is $\bar{x}_{tm} = 1.54$. This value is not only low, other than in most industries, a larger supply of input factors or higher productivity does not necessarily translate directly into higher output, i.e., into scoring more goals. A more appropriate measure is the number of shots. Figure A15 in the appendix shows for the individual level that an infection leads to fewer shots for strikers and a higher COVID exposure leads to a significant decrease in shots at least for high values of CE. Insignificant effects for defenders and midfielders are not an objection to our results. If anything, these two types of players mainly try to serve the strikers with good passes to enable them to make

[23] See Statistisches Bundesamt (Destatis) 2021, GENESIS database #13111-0003; Italian National Institute of Statistics/Istituto Nazionale di Statistica (Istat) 2021.

shots from a promising position. Again, the number of passes is the ultimate driver of productivity and outcomes.



The plot shows the OLS estimates of the post-infection dummy included in the baseline regression (1) for different performance measures. Dep. variable: given on the x-axis of the plot (always in hyperbolic sine transformation form – partly due to a high number of zero observations - except for the dichotomous variables 'full-time' and 'starting eleven'). SEs: Heteroskedasticity-robust and clustered at player level. Confidence bands of 95% are shown.

Figure 15: Robustness Check: Other Performance Measures

Our dependent variable is the logarithm of passes such that observations with zero passes etc. are dropped. This relates to players who participated for just a few minutes of the match and hence make up around 0.5 percent of the observations. To demonstrate that our results do not depend on the functional form, in the appendix we provide results for a specification in levels (Figure A5) and for using the inverse hyperbolic sine transformation for the dependent variable (Figure A6).[24] Our results do not change. Also, note that our results are not driven by one specific league. Figure A7 presents extensive and intensive margin effects for both leagues separately. Next, we test whether performance changes are induced by adapted coaching and changing tactics by inserting detailed information about the main team formation at the match level. Formation in this context means how the squad on the pitch is 'arranged', i.e., how many players act as defenders, how many as midfielders and so on. In our data, we observe nineteen different formations. By replacing the team FE with an interacted team $\times$ formation FE, we take

[24]The hyperbolic sine transformation of x is $sinh(x) = ln(x+\sqrt{x^2+1})$ and approximates a logarithmic transformation of the variable in a way that zero values do not get lost.

formation-specific performance patterns into account. As one can see in Figure A10, the static semi-elasticity is again slightly lower but remains significantly negative. The event study pattern remains the same.

As additional robustness checks, we vary the vector of fixed effects (Z). First, we replace the matchday fixed effect with an augmented matchday×season fixed effect. The static semi-elasticity is slightly lower (-4.4% instead of -5.1%) but remains highly significant. Second, we replace the player fixed effect with an interacted player × position fixed effect, which also captures the respective position of a player (goalkeeper, defender, midfielder, striker). Again, the static semi-elasticity is slightly lower (-4.5% instead of -5.1%) as the augmented fixed effect captures a bit more variation but is still highly significant. However, the event study estimates are highly similar to those from the baseline regression in Figure 5 as one can see in the plots provided in Figure A10 in the appendix.

5 Conclusion

This paper analyzes the causal effect of a COVID-19 infection on the productivity of high-performance workers, utilizing a uniquely granular panel data set of elite soccer players. We are the first to quantify COVID-19-related productivity effects at the individual level and find a persistent deterioration of about 5 percent. This does not diminish swiftly but remains prevalent over months. As hundreds of millions were infected around the globe, this is not a problem for a handful of people but is likely to accumulate to an effect size that could be felt by the economy in total. Additionally, we find some mutually reinforcing effects among groups.

This is a novel and thought-provoking result as our findings correspond directly to recent policy debates. A ‘zero COVID’ strategy that aims for complete elimination of the virus in a country or region has been suggested, for example, by Aghion et al. (2021). Bianchi et al. (2020) and Helliwell et al. (2021) highlight the indirect long-run effects of lockdowns on unemployment and health outcomes. Particularly, the latter group finds that rigid NPI strategies leading to zero transmission rates have had superior outcomes in more dimensions than just case rates and mortality. We relate our research to this debate with direct effects.

We are confident that our findings are fairly robust and generalizable. Still, we are aware that our analysis has limitations. That professional soccer players are only a subsample of society has already been discussed. Even though it is ambiguous whether the effects for the 'average' individual might be even worse, it would be helpful if our analyses were more diverse. Future research should, therefore, address gender, a broader age range, and various job profiles. Also, we are not capable of distinguishing the different variants of the virus or the severity of symptoms, since we are fully reliant on test results, which is a dichotomous outcome. Additionally, our results are based on non-vaccinated people. Even though our event study methodology encompasses a fairly long time horizon, for obvious reasons we cannot account for the effects over several years. It would be interesting to re-examine this setting in a few years.

Eventually, our back-of-the-envelope computations for wages of the median worker in Western countries suggest that there may be non-negligible long run costs for individuals infected with COVID-19. Even though these are preliminary considerations, COVID-19 may become not only endemic in a epidemiological sense but also in economic terms.

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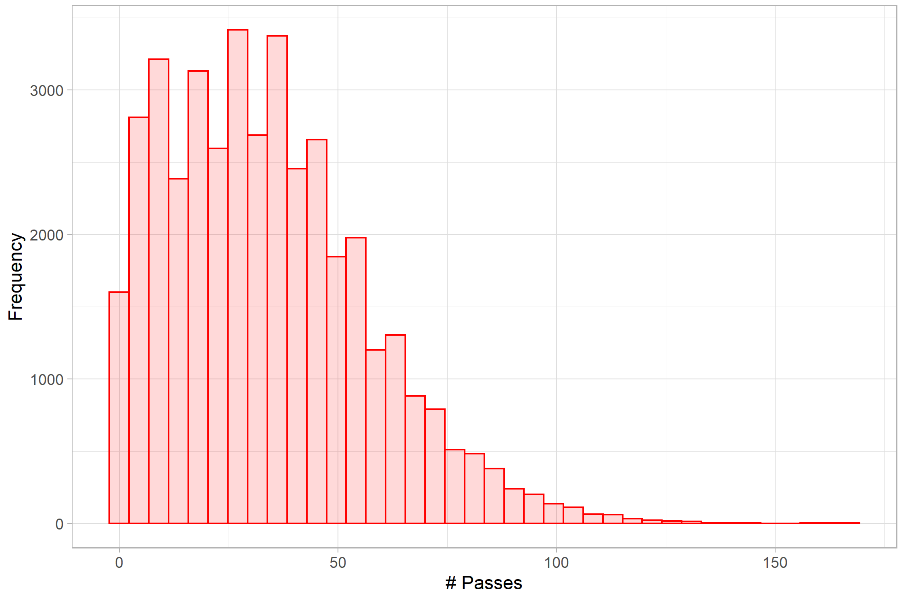
Appendix

Table A1: Descriptive Statistics on Players and Matches

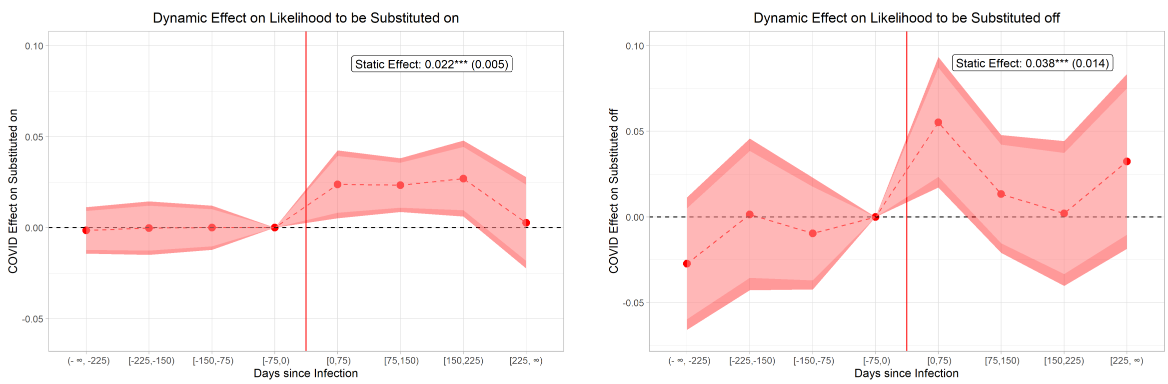
	N	Mean	St. Dev.	Min	Max
Treatment Indication					
Infected Player	72,938	0.195	0.396	0	1
Post-Infection	72,938	0.086	0.280	0	1
Player Characteristics					
Height (in cm)	72,938	183.502	6.139	163	202
Weight (in kg)	72,938	77.322	6.460	58	101
Age	72,938	26.596	4.683	15	43
COVID Game (Yes/No)	72,938	0.659	0.474	0	1
Matchday	72,938	18.592	10.515	1	38
Home (Yes/No)	72,938	0.500	0.500	0	1
Extensive Margin: Player Involvement					
Played (Yes/No)	72,938	0.557	0.497	0	1
Injured (Yes/No)	68,577	0.126	0.332	0	1
Suspended (Yes/No)	68,577	0.024	0.154	0	1
Substituted Off (Yes/No)	40,607	0.257	0.437	0	1
Substituted On (Yes/No)	40,607	0.257	0.437	0	1
Played Full-time (Yes/No)	40,607	0.488	0.500	0	1
Starting Eleven (Yes/No)	40,607	0.743	0.437	0	1
Minutes on Field	40,607	66.741	30.122	0	90
Intensive Margin: Player Performance					
1. General Measures					
Passes	40,607	33.884	22.946	0	167
Passes (Successful)	40,607	26.976	20.313	0	165
Short Passes	40,607	24.212	18.532	0	158
Short Passes (Successful)	40,607	19.624	16.629	0	157
Long Passes	40,607	9.672	6.867	0	50
Long Passes (Successful)	40,607	7.351	5.617	0	41
Distance Covered	40,607	1,175.5	642.9	0	3,878.6
Possession	40,607	32.541	22.152	0	167
Touches	40,607	43.916	26.027	0	177
Aerials	40,607	2.124	2.495	0	28
Aerials (Successful)	40,607	0.022	0.155	0	3
2. Defensive Measures					
Ball Recoveries	40,607	3.581	2.915	0	23
Defensive Aerials	40,607	1.063	1.543	0	17
3. Offensive Measures					
Shots	40,607	0.888	1.290	0	14
Shots (on Target)	40,607	0.311	0.654	0	7
Offensive Aerials	40,607	1.062	1.735	0	26

Figure A1: Distribution of #passes per match



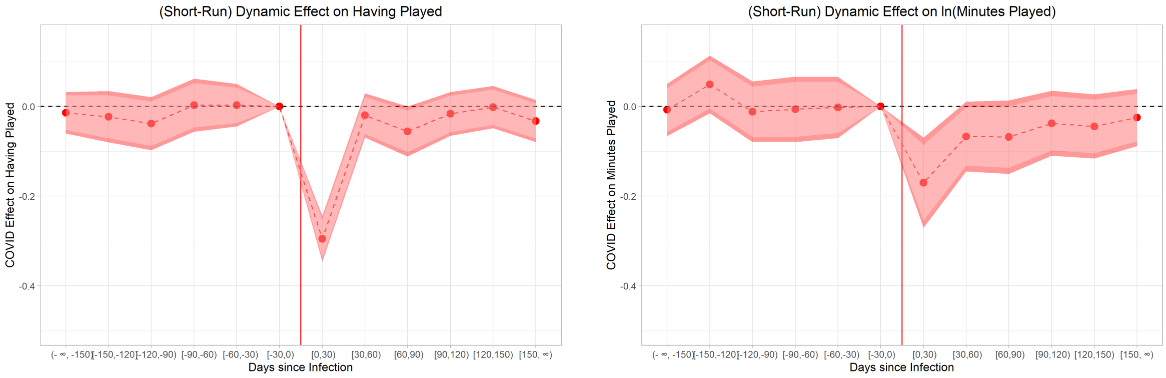
This figure plots the absolute frequency of passes played by a player during his individual time on the pitch in a particular match. Mean = 33.88, median = 31, first quartile = 16, 3rd quartile = 48, minimum = 0, maximum = 167.

Figure A2: Dynamic Effect on On and Off Substitutions



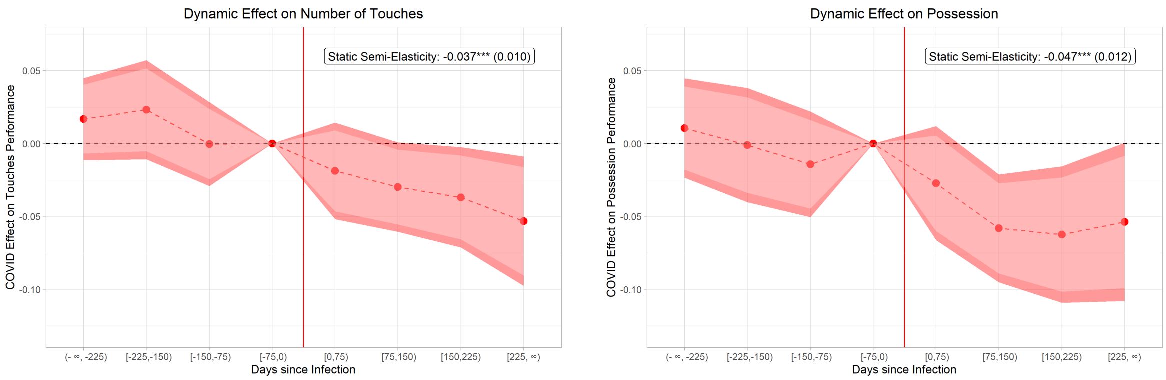
These figures plot the OLS (linear probability model) estimated coefficients β_τ of the event study regressions following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variables is a binary variable indicating to be substituted on (LHS) or off the field (RHS).

Figure A3: Dynamic Effect on the Likelihood to Play and Minutes Played: Smaller Bin Size



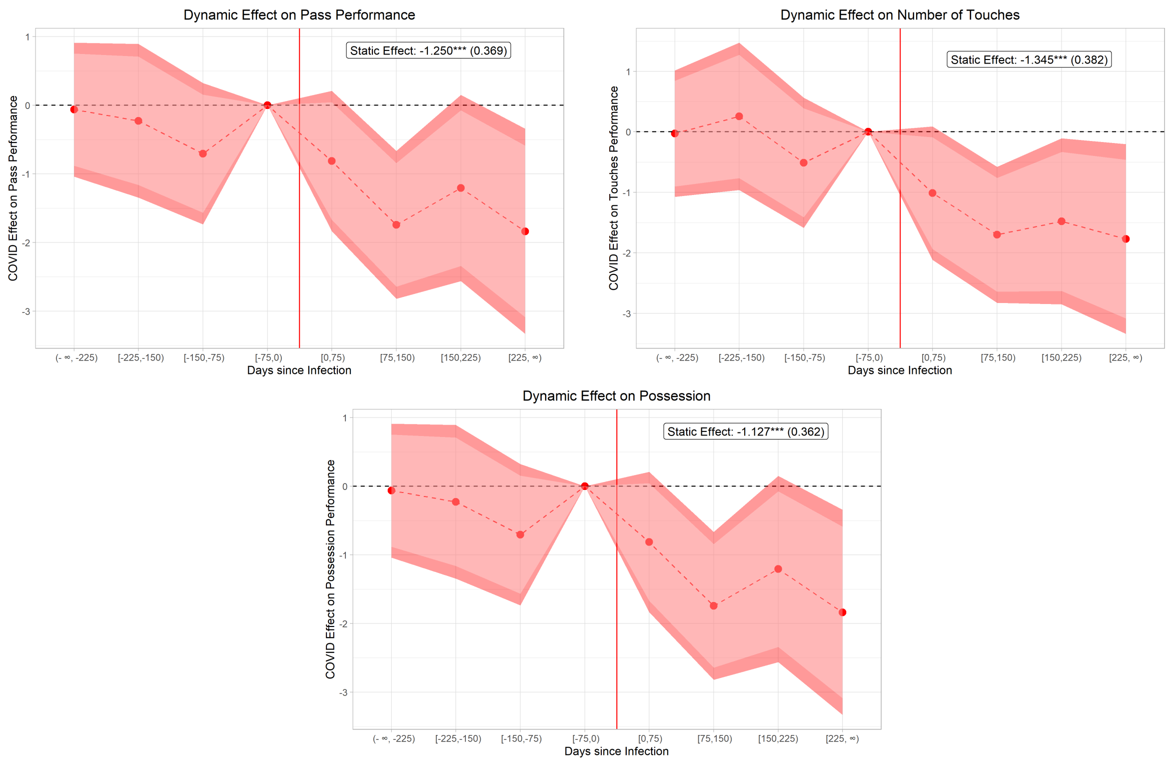
This figure plots the OLS estimated coefficients β_τ of the event study regression following Equation (2). The bin size is now 30 days, i.e. one month. The reference time period is one to 30 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the red-shaded areas. As the bin size is much smaller compared to the baseline setting in figures 3 and 4, this affects the confidence bands. Due to much fewer observations within one bin, we severely lose statistical power, which leads to mostly insignificant results at a 5% significance level. Dependent variable LHS: A dummy indicating whether a player played or not. Dependent variable RHS: ln(minutes played) conditional on having played.

Figure A4: Dynamic Effect on Within-Match Performance: Additional Work Performance Measures



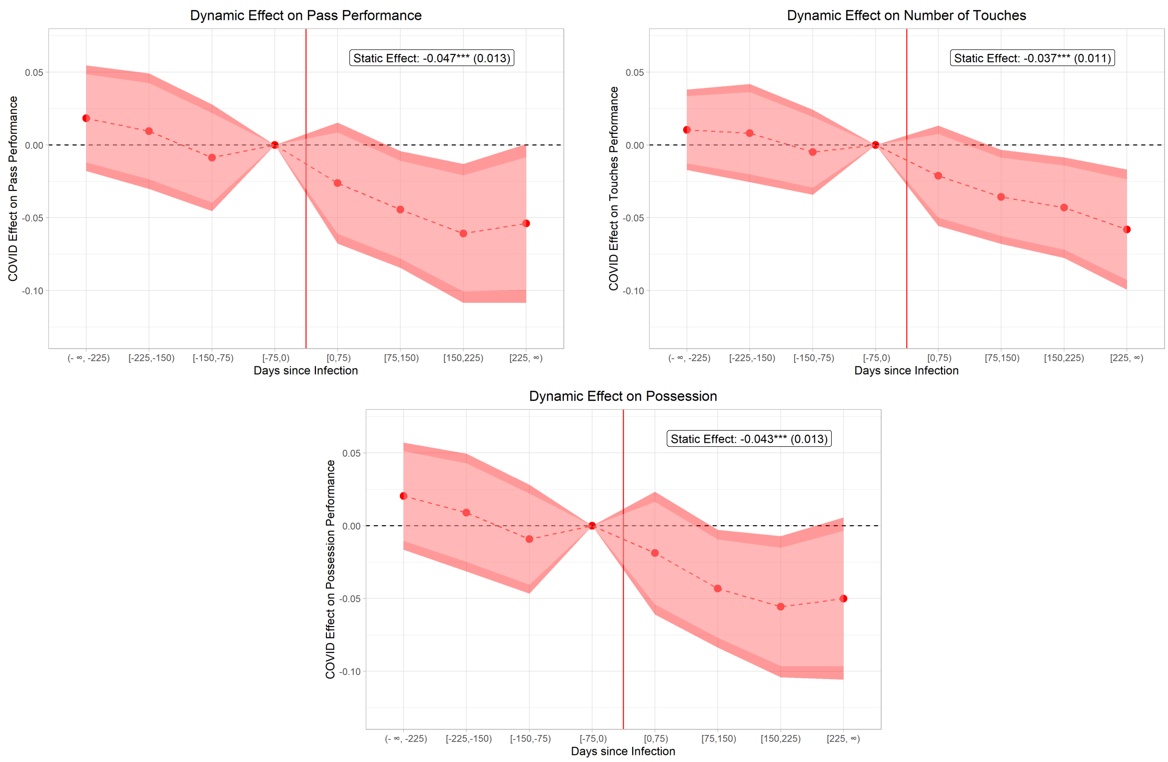
These figures plot the OLS coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variables are ln(touches) as ln(possession) as additional work performance measures. The logarithmic specification excludes observations with zero touches or possessions. Robustness checks in figures A5 and A6 show that these results also hold for settings taking zero values into account.

Figure A5: Event Studies for Different Outcome Specifications: *Levels*



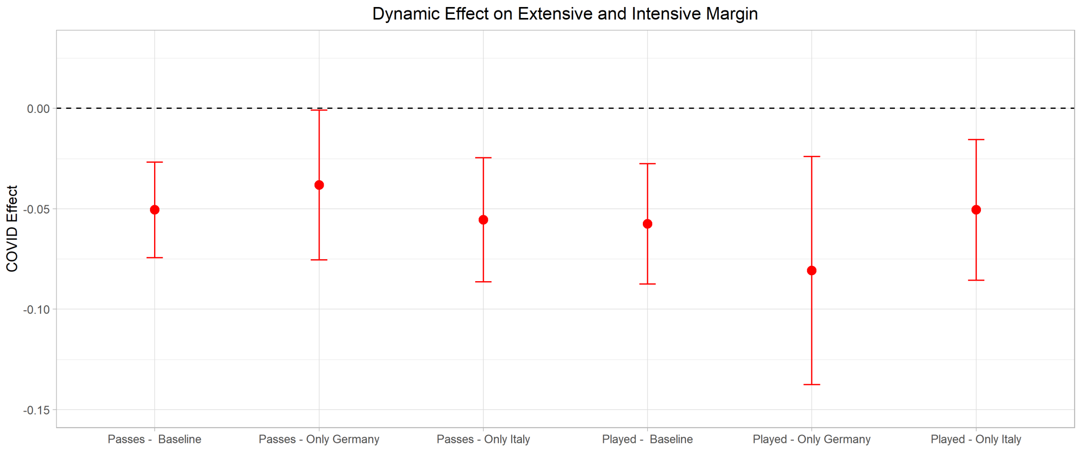
These figures plot the OLS estimated coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. Outcomes winsorized at the 5 and 95% level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variables are passes, touches and possession in their level specification.

Figure A6: Event Studies for Different Outcome Specifications: *Inverse Hyperbolic Sine Transformation*



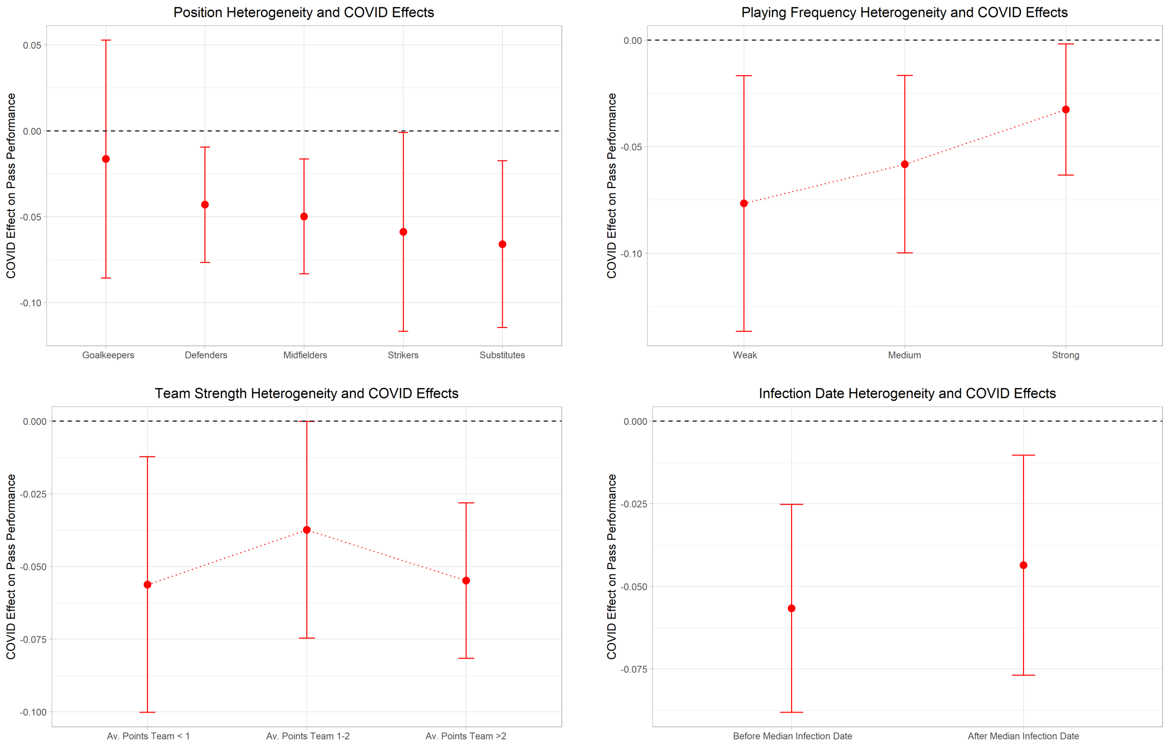
These figures plot the OLS estimated coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variables are the variables passes, touches and possessions transformed via the inverse hyperbolic sine transformation to account for zero-values in the dependent variables. See for a critical assessment of this technique, e.g., Bellemare and Wichman (2020).

Figure A7: League-Specific Effects



The plot shows the effects of the post-infection dummy included in the baseline equation (1) for the extensive and intensive margin estimated by OLS. The x-axis gives more precise information on the choice of the control group. Dep. variable: ln(passes) and a dummy which takes the value 1 if a player has played. SEs: Heteroskedasticity-robust and clustered at the player level. The 95% confidence intervals given.

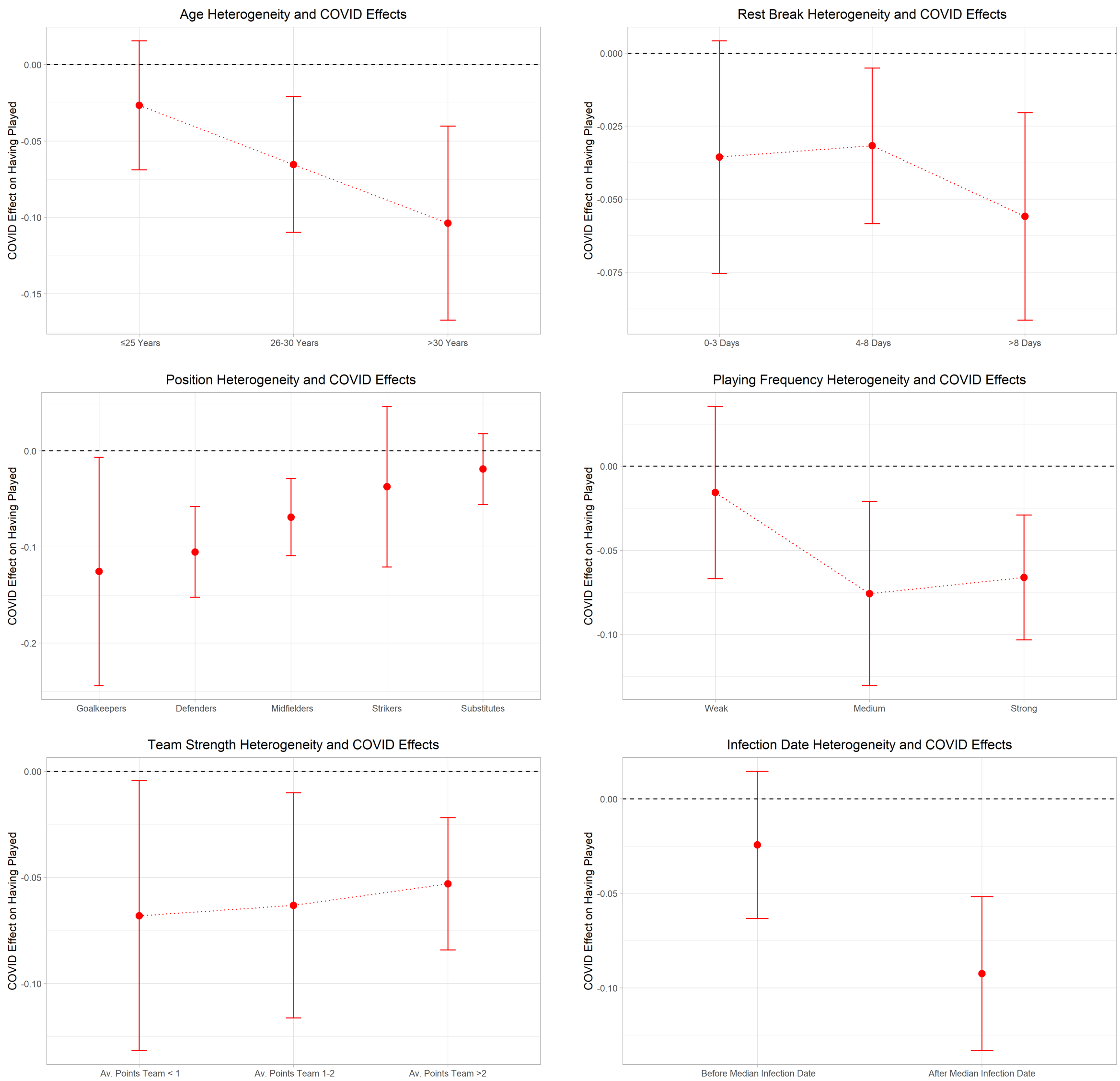
Figure A8: Additional Heterogeneity Analyses for Intensive Margin Effects: Position on the Pitch, Playing Frequency, Team Strength, and Infection Timing



These figures plot the OLS estimated heterogeneous semi-elasticity of a COVID-19 infection on pass performance. Standard errors are heteroskedasticity-robust and clustered at the player level. The 95% confidence bands are displayed.

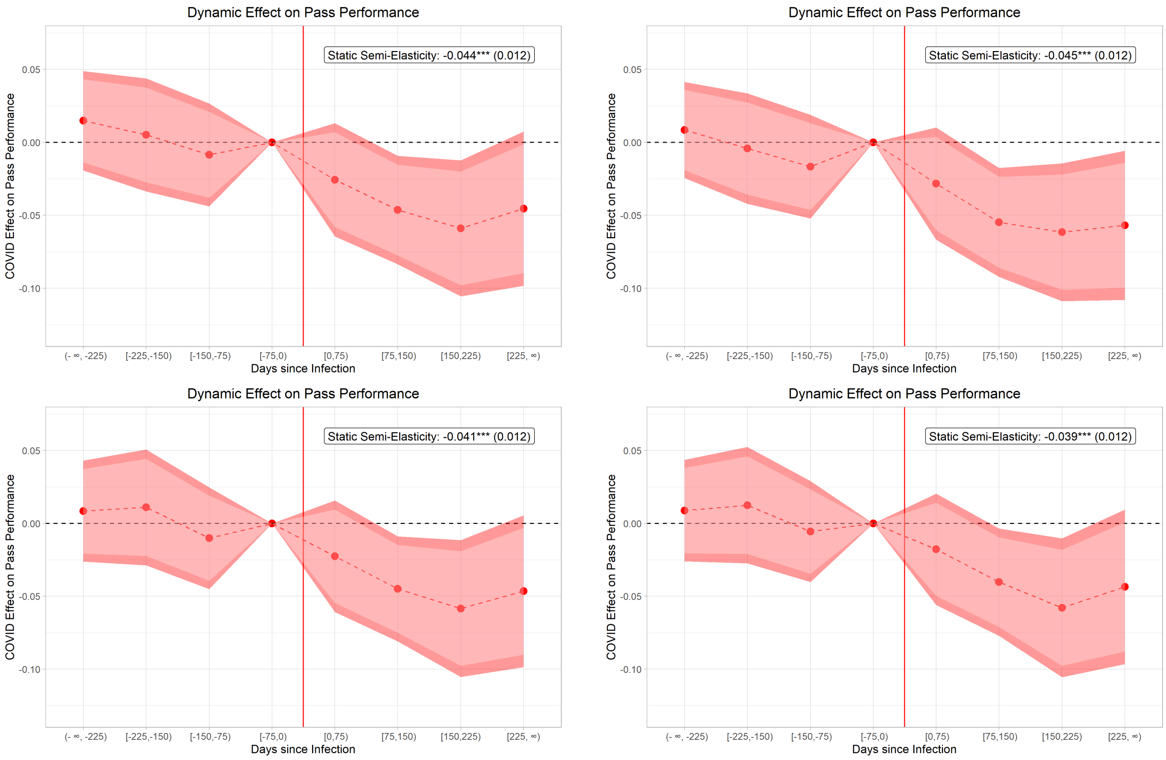
- *Position* addresses the effects on different types of positions a player might have on the pitch. ‘Substitutes’ captures all players that did not play from the beginning but have been substituted on the pitch during a match.
- *Playing Frequency* addresses differences in a player’s quality and significance for his team. To capture this, we calculate the share of available matches a player played in before his infection took place and construct three groups for different terciles (from weak to strong) of this match-share distribution.
- *Team Strength* is the equivalent calculation at the team level. Better teams might have better medical support available while also allowing recovering players to not take on full responsibility immediately. Contrary, above-average teams might perform on a level which is harder to come back to again. We test this relationship by looking at heterogeneous treatment effects for teams which earned a different number of points up to a certain match in a season. Teams can earn zero (defeat), one (draw), or three points (victory) per match, so we group them into clusters of low-performing (average points < 1), medium (average points $1 - 2$) and well-performing teams (average points > 2).
- *Infection Timing* tests whether early infected players show different work performance effects than players who got infected later during the pandemic. The plot at hand shows two groups of infected players which have been divided at the median infection date. One can see that the work performance effect is significant for both groups and not statistically different from each other.

Figure A9: Heterogeneity Analysis for Extensive Margin Effects



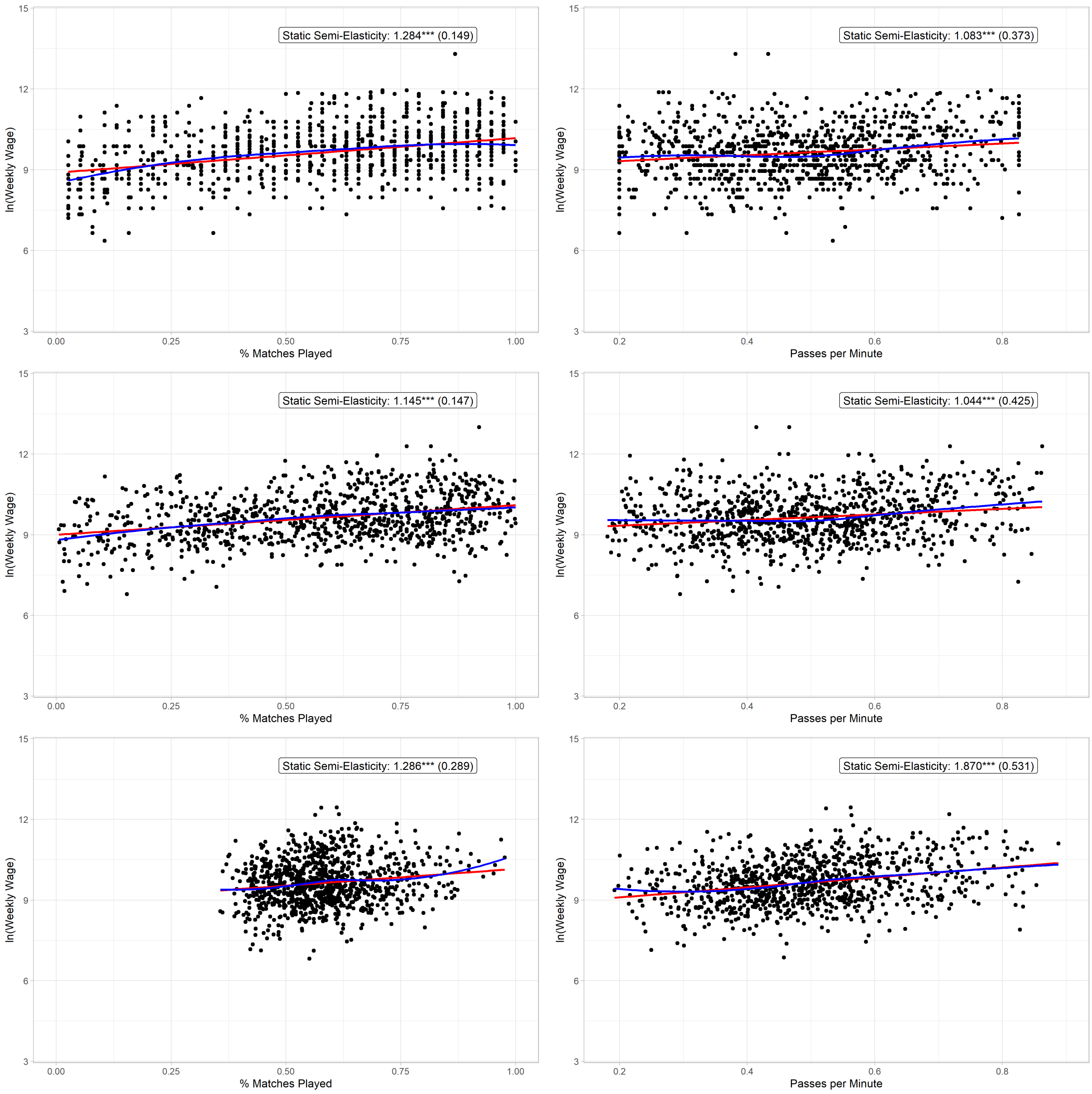
These figures plot the OLS (partly linear probability model) estimated heterogeneous semi-elasticity of a COVID-19 infection on pass performance. Standard errors are heteroskedasticity-robust and clustered at the player level.The 95% confidence bands are displayed.
Heterogeneity in Age and Rest Breaks correspond to the intensive margin effects shown in figures 7 and 8. Position Heterogeneity, Playing Frequency, Team Strength and Infection Timing correspond to the intensive margin effects shown in Figure A8 above. The difference in Infection Timing is driven by technical reasons as for late infections there exists much fewer observations after the infection happened compared to early infections, such that missed matches have a higher weight causing a significant estimate.

Figure A10: Dynamic Effect on Within-Match Work Performance using different sets of fixed effects.



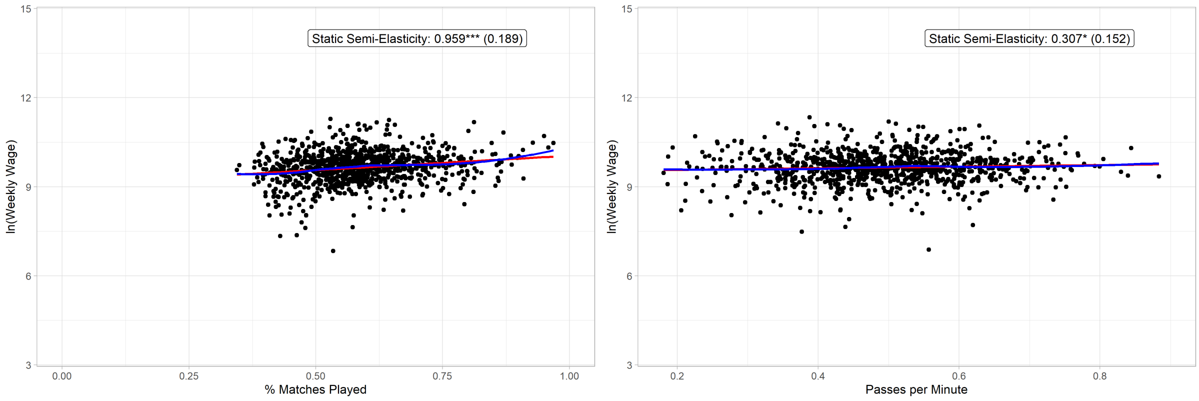
These figures plot the OLS coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variable is ln(passes).
The plot on the upper LHS shows the event study results using a matchday $\times$ season FE instead of the matchday FE used in the baseline regression (shown in Fig. 5). The plot on the upper RHS shows an event study specification that uses a player $\times$ position FE instead of the player FE used in the baseline regression.
Both lower plots show a variation in the team FE. The plot on the lower LHS shows event study results using a team $\times$ formation FE instead of the plain team FE. The lower RHS shows results using not only the team $\times$ formation FE applied on the LHS, but also an opponent $\times$ formation FE.

Figure A11: Partial Correlation Analysis



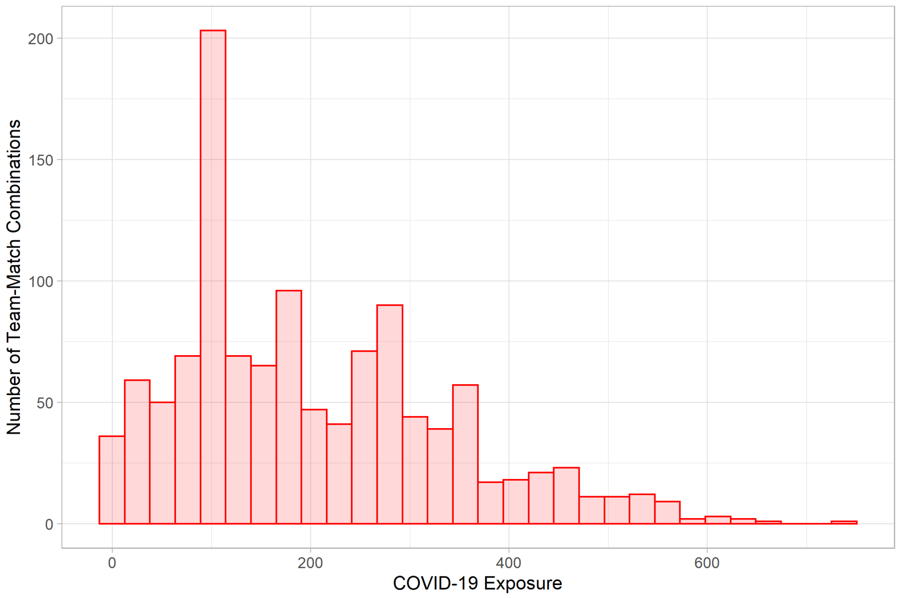
The left column plots the correlation between wages and the share of matches played, the right column plots the correlation between wages and passes/min. Top row: Pure correlation, middle row: correlation controlled for age, weight, and height. Bottom row: additional controls for position, share of starting eleven, share of fulltime matches. The variable passes/min is winsorized at 2.5 and 97.5% to correct for outliers. Standard errors are heteroskedasticity-robust and clustered at the team level. Data from the La Gazzetta dello Sport. We report a linear regression fit (red) and a fit from a local polynomial estimator (blue). *p<0.1; **p<0.05; ***p<0.01.

Figure A12: Partial Correlation Analysis Including Team Fixed Effects



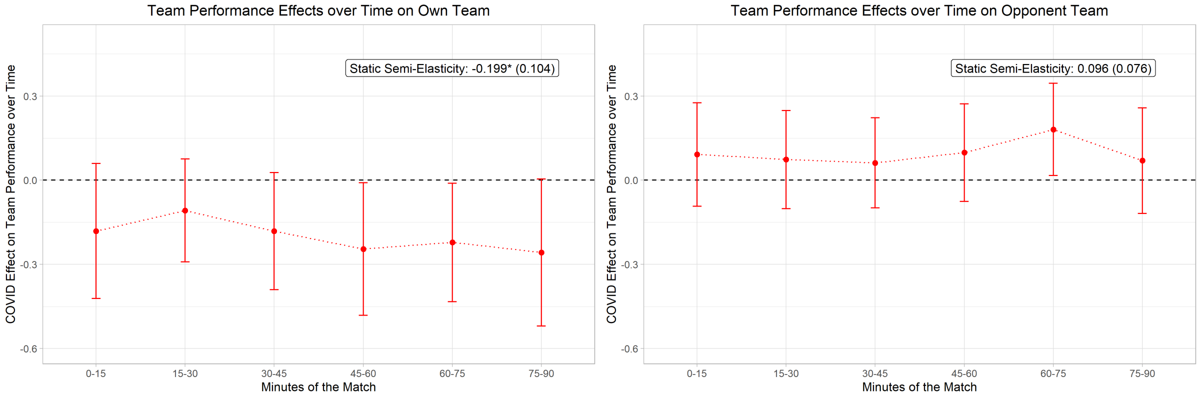
The left column plots the correlation between wages and the share of matches played, the right column plots the correlation between wages and passes/min. Different to Fig. A11, we additionally include team fixed effects. *p<0.1; **p<0.05; ***p<0.01.

Figure A13: Distribution of COVID-19 Exposure (CE) for $CE > 0$



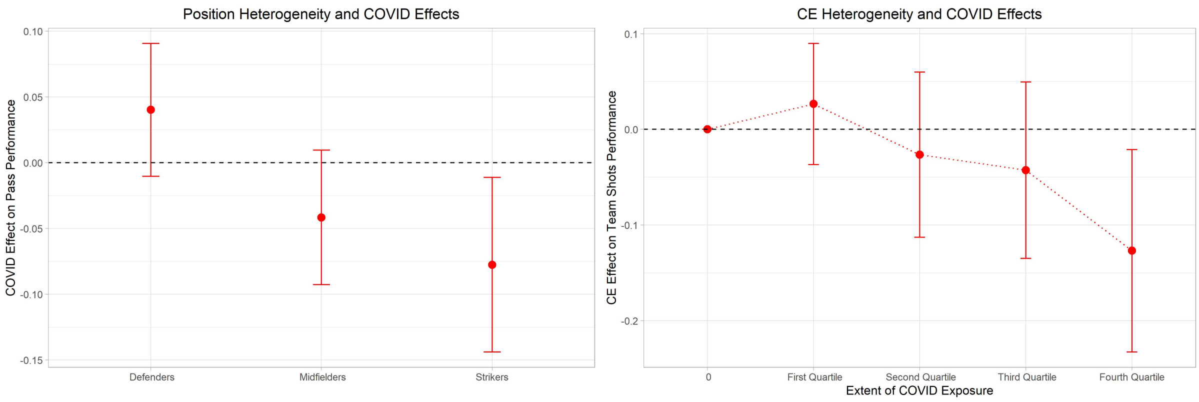
This figure plots the absolute frequency of COVID-19 Exposure (CE) realizations for $CE > 0$ – as defined in eq. (3) – observed in the team data.

Figure A14: Effects on Within-Match Team Performance (Own vs. Opponent Team)



The plots show the time-specific COVID-19 effects on ln(Passes) on team and match level of CE on own (LHS) and opponent team (RHS) performance estimated by OLS. SEs: Heteroskedasticity-robust and clustered at the team level. The 95% confidence bands are given. The regression set-up is equivalent to (1) except for additional interactions of the COVID-19 term with 15-minute time slots, which results in up to six observations per player and match. In contrast to Fig. 11, the time slots capture overall match time and not the minutes a player has been on the field. The regression includes controls for home/away matches, ghost matches and team-season FE, opponent-season FE and matchday FE, and time category FEs.

Figure A15: Effect of COVID-19 on Shots (Individual and Team Level)



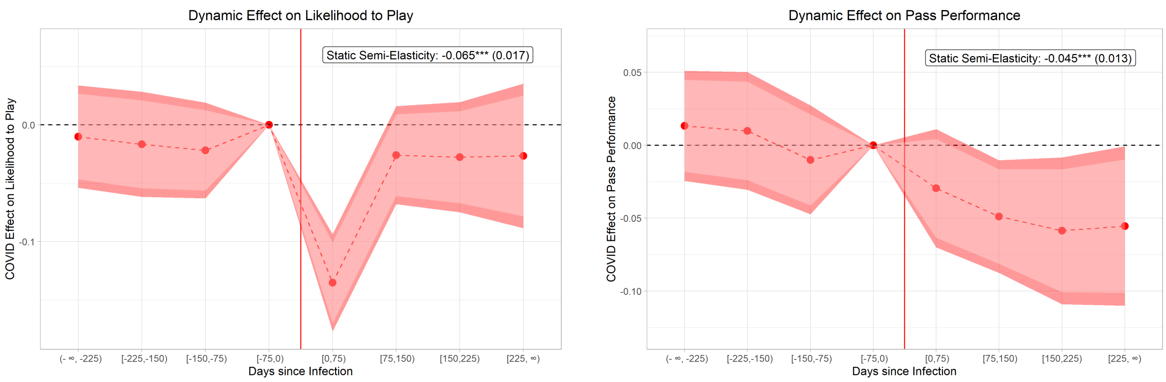
The plots show the effect of a COVID-19 infection on goals on the individual (LHS) and the team level (RHS).
The plot on the left displays OLS interaction effects between the post-infection dummy and age groups included in equation (1). Dependent variable: shots, transformed via the inverse hyperbolic sine transformation to account for zero-values. SEs: Heteroskedasticity-robust and clustered at player level. Goalkeepers are omitted due to the position-specific importance of shots.
The plot on the right shows the effect of CE on team performance measured in the logarithm of shots estimated by OLS. We use the hyperbolic sine transformation due to zero shots observations at the team level. We compare teams with $CE = 0$ to an exposure in four quartiles, which have the intervals $(0, 0.077)$, $[0.077, 0.130)$, $[0.130, 0.241)$, and $[0.241, 0.500]$ empirically or else $[0.241, 1]$ theoretically. The means are $\overline{CE}_{(0,0.077]} = 0.050$, $\overline{CE}_{(0.077,0.130]} = 0.096$, $\overline{CE}_{(0.130,0.241]} = 0.191$, and $\overline{CE}_{(0.241,1]} = 0.352$. The regression includes controls for home/away matches, ghost matches, the opponent's COVID exposure (transformed by the inverse hyperbolic sine transformation) and team-season FE, opponent-season FE and matchday FE. In both plots, 95% confidence bands are given.

Table A2: Test of Balancing Condition

	Before Matching			After Matching		
	Treated	Non-Treated	p-value	Treated	Non-Treated	p-value
Propensity Score	0.034	0.003	0.000***	0.034	0.032	0.534
Recent Match Involvement						
Played	0.702	0.612	0.005***	0.702	0.746	0.321
Played×Fulltime	0.351	0.296	0.102	0.351	0.405	0.264
Played×Starting Squad	0.546	0.452	0.007***	0.546	0.590	0.371
Past Match Involvement						
Played	0.664	0.598	0.000***	0.664	0.643	0.444
Minutes if Played	67.073	61.798	0.000***	67.073	65.191	0.357
Fulltime if Played	0.514	0.450	0.005***	0.514	0.480	0.287
Distance/min	19.038	18.724	0.643	19.038	18.616	0.586
Passes/min	0.516	0.492	0.098*	0.516	0.513	0.884
Ballrecovery/min	0.052	0.056	0.030**	0.052	0.054	0.347
Possession/min	0.497	0.472	0.077*	0.497	0.494	0.879
Touches/min	0.676	0.657	0.182	0.676	0.680	0.857
Shots/min	0.015	0.014	0.556	0.015	0.013	0.309
Aerials/min	0.035	0.037	0.828	0.035	0.034	0.780
Demographics						
log(Height)	5.214	5.210	0.102	5.214	5.211	0.322
log(Weight)	4.344	4.344	0.955	4.344	4.334	0.259
log(Age)	3.273	3.284	0.359	3.273	3.253	0.214
Others						
1[NT COV19]	0.483	0.322	0.000***	0.483	0.463	0.693
1[NT COV19] ×ln(# Matches)	0.782	0.500	0.000***	0.782	0.776	0.947

*p<0.1; **p<0.05; ***p<0.01. p-value report coefficients from two-sided t-tests.
1[NT COV19] = part of the national team during COVID-19. The matching regression also includes team×season, matchday×season and position FE. The matching is conducted within position-matchday cells. We do not match infected players who ever got infected after our sample period or for who we do not observe the last match before the infection. We also drop these players from the final estimation sample. 'Before Matching' compares those observations which are treated in the probit regression, with all other included observations of non-infected players. 'After Matching' compares the matched observation-couples. All variables in the subgroup 'Past Match Involvement' give cumulative averages up to the matchday of the observation for a respective player.

Figure A16: Dynamic Effects Using a Fully Balanced Control Group



These figures plot the OLS coefficients β_τ of the event study regression following Equation (2). The reference time period is one to 75 days before treatment. Standard errors are heteroskedasticity-robust and clustered at the player level. The 90 and 95% confidence intervals are given by the two red-shaded areas. The dependent variables are the logarithm of the likelihood to play (LHS) and the logged number of passes (RHS). These regressions use a fully balanced sample based on propensity score matching as described in Table A2. $N = 205$ infected-counterfactual pairs.