

Peer Health Shocks and Labor Supply

Joshua C. Martin

Vanderbilt University

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Abstract

I provide novel evidence on how workers respond to peer health shocks within high-risk occupations by leveraging two nested natural experiments within professional hockey and American football. First, I compare differences in labor supply between characteristically similar athletes who differ only in their exposure to a colleague who died of chronic traumatic encephalopathy (CTE) – a deadly neurological disease causally linked to continued workplace participation. Though the information about these deaths is widely publicized, I find that their occurrence differentially increases the probability for former teammates to retire. This effect is greater for those with longer periods spent as teammates and diminishes with time since they were last on the same team. Second, I leverage quasi-random differences in the monetary compensation that workers would forgo upon retiring at the time of this peer health shock. I show these retirements are highly responsive to opportunity costs – estimating that teams would have to increase worker compensation \$1 million to prevent their exit. Remaining treated workers display a heightened sensitivity to health risks by exchanging salary for larger signing bonuses and shorter contracts in their subsequent employment negotiations. The finding that labor supply decisions are highly responsive to the health status of peers suggests that workers substantially underestimate utility loss from work-related health damages even in environments where such risks are highly publicized.

Keywords: Labor Supply, Health Shocks, Peer Effects, Occupational Risk, Compensating Wage Differentials, Opportunity Costs, Behavioral Economics

JEL Codes: J28, J24, J33, I12, Z22

“Let us suppose that ...[a great, distant empire], with all its myriads of inhabitants, was suddenly swallowed up by an earthquake, and let us consider how a man of humanity [...], who had no sort of connection with that part of the world, would be affected upon receiving intelligence of this dreadful calamity. He would, I imagine, first of all, express very strongly his sorrow for the misfortune of that unhappy people, he would make many melancholy reflections upon the precariousness of human life, and the vanity of all the labours of man, which could thus be annihilated in a moment... And when all this fine philosophy was over, when all these humane sentiments had been once fairly expressed, he would pursue his business or his pleasure, take his repose or his diversion, with the same ease and tranquillity, as if no such accident had happened. The most frivolous disaster which could befall himself would occasion a more real disturbance. If he was to lose his little finger to-morrow, he would not sleep to-night; but, provided he never saw them, he will snore with the most profound security over the ruin of a hundred millions of his brethren, and the destruction of that immense multitude seems plainly an object less interesting to him, than this paltry misfortune of his own.”

- Adam Smith, *The Theory of Moral Sentiments*

email: joshua.martin@vanderbilt.edu

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1 Introduction

The factors that influence how individuals perceive and respond to changes in risk have long been a question of central importance to social scientists. While neoclassical models of human behavior often provide valuable benchmarks, they frequently rely on simplifying assumptions that may overlook complex realities. A prominent example in economics is the theory of equalizing differences, which predicts that prices will adjust to reflect all relevant (dis)amenities of a good or service in markets with perfect information and numerous buyers and sellers. Yet, undesirable outcomes persist even in market settings where information is abundant, transaction costs are low, and markets are thick (Thaler and Shefrin 1981). This paper explores whether the labor supply decisions of high-wage workers in occupations with highly publicized and well-known risks are better explained by neoclassical or behavioral models.

By leveraging two natural experiments within two professional sports, this study provides unique insight into crucial factors influencing how individuals perceive and respond to the heightened salience of work-related injury. To do so, I first exploit quasi-random variation in individuals' exposure to chronic traumatic encephalopathy (CTE), a deadly neurological disease that is causally linked to continued workplace participation.

I begin by constructing a unique panel dataset of information on all athletes employed on the rosters of teams within the National Football (NFL) and Hockey Leagues (NHL) from 1970 to 2023. This data contains credible measures of worker productivity, which I combine with data on workers' employment contracts over time. These data allow for a more comprehensive examination of multiple aspects of labor supply while making possible the construction of plausible counterfactual groups via commonly employed matching methods.

I show, using stacked difference-in-differences estimators, that the occurrence of a CTE death of a former teammate leads to sharp increases in these workers' probability of exit from the profession by 2.2-2.5 percentage points (8-9%) relative to a group of characteristically similar workers. This effect size is about 17% of the effect of a one-standard deviation reduction in worker productivity.

I discover evidence of two important channels through which this difference in the rate of exit emerges. First, I show that the probability of exit increases with personal familiarity (measured by the time spent as teammates). Second, I show these effects decrease in magnitude as the time since individuals were last colleagues increases. Estimates from models which jointly incorporates these two mechanisms (athletes who were teammates for longer, one of whom died recently) reveal their interaction is greater than the sum of their individual effects. These findings suggest that informational health shocks' immediacy and personal relevance play crucial roles in shaping

workers' perceptions and responses to occupational health risks.

These findings also help explain the heterogeneity in labor supply responses across sports. Hockey players were more likely to exit the profession following a peer health shock, likely due to smaller average team sizes that foster increased familiarity, closer average temporal proximity to these CTE deaths, and a reduced ability to internalize the heightened workplace risk in one's wages. Hockey players' reduced ability to secure higher wages likely stems from significantly weaker bargaining power, driven by pronounced differences in superstar effects and labor demand relative to football.

Next, to rule out the possibility that these findings primarily arise due to factors unrelated to changes in risk perception, I replace the variables indicating exposure to a former teammate who died of CTE with those indicating exposure to a former teammate who died of causes unrelated to workplace safety. I chose the two most common examples of death among prime-age workers for this task: car accidents and (previously) undetected genetic diseases. Estimates from these models reveal precise null impacts on labor supply, suggesting that factors such as grief are not driving these results.

I then investigate whether wages for these treated workers rose to mitigate the impact of the peer health shock on their exit rates. Although I find economically significant positive effects of treatment on total monetary compensation, these effects are not statistically significant. However, this overall effect conceals considerable heterogeneity in the structure of their subsequent contracts. Treated workers exchange reductions in salary for increased guaranteed compensation and shorter contract durations. This pattern among the remaining workers aligns with the notion of heightened sensitivity to health risks, suggesting that treated workers were willing to stay only if they could secure greater financial guarantees to their future income.

Motivated by the evidence that many of these individuals were willing to remain in this risky profession for greater financial security, I propose an alternative variable for estimating compensating wage differentials: opportunity costs. The contract data, which includes total compensation, annual salary, guaranteed income, and contract length, allows for the precise calculation of the labor income workers would forgo upon voluntary exit (retirement) in any given period.

I find that retirement decisions are highly sensitive to workers' opportunity costs. I then estimate a difference-in-differences model which compares individuals with similar annual wages who differ only in the amount of income they would forgo upon retirement at the time of treatment. Estimates from this model reveal that former teammates of athletes who died of CTE would require a one-time income transfer of approximately \$1 million to be indifferent between exiting the profession and staying.

This paper demonstrates that peer health shocks have a strong impact on workers' labor supply, a finding that is surprising for several reasons. First, professional athletes earn exceptionally high wages, and firm entry into these leagues is highly restricted, resulting in a large excess supply of individuals aspiring to join. This intense competition has led to early and substantial investments in sport-specific skills, which are not easily transferable to other markets. Consequently, workers in these professions have limited leverage to credibly threaten to leave voluntarily. Second, the persistence of these labor supply responses highlights the role of peer effects. Despite widespread media coverage following any diagnosis of CTE in athletes or repeated concussions among superstars, which also reaches individuals in the control group, the observed effects are still pronounced. This suggests that the true influence of peer health shocks on labor supply is even stronger than the findings presented in this paper indicate.

Taken together, these results provide strong evidence of two key phenomena. First, individuals are significantly more responsive to health shocks when they are more likely to have directly associated with the known person. Second, this implies that workers substantially underestimate the utility loss from work-related health damages, even in environments where such risks are highly publicized and widely discussed. This underestimation has potentially wide-ranging implications. For instance, it can lead to suboptimal career choices and excessive exposure to workplace risks that are not adequately compensated. These miscalculations not only endanger individual health and well-being but can also have broader effects on aggregate long-term productivity. This finding underscores the need for more effective communication about occupational health risks, particularly through credible messengers who have personal connections to workers.

2 Literature Review

Social scientists have long studied the effects of peer spillovers on individuals' actions and attitudes. A major force contributing to the sheer volume of this literature was the innovation in empirical methodology for measuring these phenomena introduced by Manski (1993). More recently, the proliferation of methodologies from the causal inference revolution, coupled with a more widespread availability of panel data, has led researchers to increasingly rely upon quasi-random and experimental research designs to identify peer effects empirically.

Research into these effects is especially prevalent in settings where randomization is common, such as classrooms and dorms. This voluminous literature has demonstrated the impact of peer spillovers on a very wide array of outcomes ranging from income, physical fitness, risky behaviors, and attitudes towards minorities (Sacerdote 2001; Marmaros and Sacerdote 2002; Kremer

and Levy 2008; Carrell, Fullerton, and West 2009; Carrell, Hoekstra, and West 2011, 2019; Chetty et al. 2011; Eisenberg, Golberstein, and Whitlock 2014; Yakusheva, Kapinos, and Eisenberg 2014; Feld and Zölitz 2017; Carrell, Hoekstra, and Kuka 2018; Chung and Zou 2023). Another commonly studied setting is the workplace, where peer effects are often observed through learning, productivity, and behavioral influences through social pressure (Falk and Ichino 2006; Guryan, Kroft, and Notowidigdo 2009; Mas and Moretti 2009; Oster and Thornton 2012; Rosaz, Slonim, and Villevall 2016; Cornelissen, Dustmann, and Schönberg 2017; Stevenson 2017; Cohen-Zada, Dayag, and Gershoni 2024).

A large body of literature examines peer effects in financial matters, finding evidence of impacts on topics ranging from retirement savings to charitable giving (Duflo and Saez 2003; Lieber and Skimmyhorn 2018). Recent research also indicates that physical proximity to friends and colleagues is not the only channel through which peer effects occur. For instance, Bailey et al. (2018) and Hu (2022) show that geographically distant friends who experience either increases in the value of their home or exposure to major flooding events are more likely to purchase a home or flood insurance themselves. However, the most relevant strand of research to this paper focuses specifically on how peer health shocks influence behavior. For example, Innocenti et al. (2019) find that vicariously experiencing an acquaintance's negative health shock increases the intention to purchase health insurance more than the impact of one's illness. Similarly, Robertson, O'Neill, and Wixom (1972) demonstrate that having a friend experience (but not die from) a car crash significantly increases seat belt usage through fear of injury.¹

These findings from related literature lead me to investigate the hypothesis that individuals who experience the CTE death of a former teammate will have differentially large labor supply responses relative to characteristically similar non-treated athletes. The first dimension of labor supply studied is at the extensive margin – whether to exit or remain in the profession. Research into the determinants of “early” retirement decisions has highlighted the complex nature of these decisions. Models that incorporate wide arrays of information on workers, such as their wage rate, the state of their finances, their eligibility for pensions, and their current health status, are vastly more predictive of labor supply decisions than models that analyze the impact of these factors separately (Quinn 1977; Anderson and Burkhauser 1985; Giustinelli and Shapiro 2024). Relatedly, empirical work has demonstrated that extensive margin labor supply elasticities are heterogeneous. Some of the highlighted mechanisms driving differences in retirement are threats to finances and health (Brown 2001; Coile and Levine 2007; Brown, Coile, and Weisbenner 2010) and differences in age and education (Coile and Levine 2011).

¹Interestingly, individuals seemingly perceive the probability of their death from a car accident as too improbable to cause changes in behavior whereas seeing a friend injured from a wreck was more salient.

The second dimension of labor supply examined in this paper is the compensating wage differential. This foundational framework in economics posits that observed wage differences equalize all monetary and non-monetary differences in the amenities or desirability of different types of work (and workers) across time and space (Rosen 1986). The grounding of this theory upon the assumption of thick labor markets with many buyers and sellers has led to much research on how workplace safety issues intersect with differences in worker bargaining power. If workers have both imperfect understandings of the probability of workplace accidents and face local labor markets with limited outside options, then the risk of injury will be imperfectly internalized within wages (Fishback and Kantor 1992; Bender and Mridha 2011; Mridha and Khan 2013; Lavetti 2020). A related consequence of non-competitive labor markets is that they can lead to higher professional quit rates, which can have large consequences for both worker finances and aggregate output (Böckerman and Ilmakunnas 2009, 2020; Cottini, Kato, and Westergaard-Nielsen 2011).

The presence of this market failure (whose setting characteristics closely mirror those examined in this paper) led to the rise of unions. Research into the effect of unions on compensating differentials has shown that policies that relaxed the right for workers to unionize helped to increase wages and minimize the pass-through of costs of worker's compensation policies but were less successful in reducing the risks of work-related injuries (Fishback 1986; Fishback and Kantor 1995; Kim and Fishback 1999). Importantly, the presence of compensating wage differentials is not dependent upon a high prevalence of workplace injuries. Hersch (1998) shows that there are large wage premia for women in white-collar jobs who are exposed to small differences in unlikely work-related injury and illness.

Researchers have also long examined the consequences of these wage differences on inequality (Leeth and Ruser 2003). For instance, Lavetti and Schmutte (2023) demonstrates that women and men sort within the labor market differently based on physical risk but similarly on financial risk, which contributes to establishment segregation and can explain a significant portion of the gender wage gap. Hersch (2011) provides evidence of another mechanism driving these differences in occupational segregation by demonstrating that female workers employed in settings with greater risks of sexual harassment earn more in wages, all else equal. While significant stressors such as performance pressure within the workplace have been shown to contribute meaningfully to inequality (Nagler, Rincke, and Winkler 2023), even small differences in preferences for seemingly innocuous factors such as commuting time and driving speed have been shown to have large impacts on aggregate gender wage gaps (Cook et al. 2021; Le Barbanchon, Rathelot, and Roulet 2021).

Research on compensating wage differentials has also been examined in across various sports settings to provide unique insights into the topic. For example, Michaelides (2010) shows that

the wages of professional players are highly elastic with respect to location amenities and non-pecuniary characteristics of the team. Anderson (2022) examines whether wages vary significantly to compensate workers for the risk of re-injury, finding large wage premia between professional boxers who have lost a fight via knockout. Additionally, Dole and Kassis (2010) finds that players with larger bargaining power (as measured by those in the top quartile of the income distribution) receive wage premiums for playing more games on surfaces commonly deemed to increase the probability of injury.

3 Setting

3.a. CTE & Collision Sports

Chronic traumatic encephalopathy (CTE) is an irreversible neurodegenerative condition caused by repeated head impacts over extended periods (Ling, Hardy, and Zetterberg 2015). The symptoms of CTE vary widely and may include motor impairments, emotional regulation difficulties, nerve pain, and often dementia. Individuals suffering from CTE can present symptoms similar to those of advanced Parkinson's and Alzheimer's disease. Treatment options are limited to symptom management for a condition that progressively worsens. Although these symptoms are more common in older populations, the highly salient fear surrounding CTE stems from its propensity to affect individuals as early as their mid-to-late 20s. At the time of this writing, no diagnostic tools, formal or informal, exist to determine one's risk for developing the disease; diagnoses can only occur post-mortem through brain tissue analysis. While CTE can theoretically affect anyone, it has become synonymous with collision sports—particularly American football.

Before the term “chronic traumatic encephalopathy” was formalized, there was considerable variation in the terminology used to describe individuals experiencing short- or long-term symptoms of head trauma. Two groundbreaking papers that documented CTE in former NFL players (Omalu et al. 2005; Omalu et al. 2006) were pivotal in helping to establish the term. Before this, individuals were often referred to with dangerously misleading metaphors such as having their “bell rung” or being “punch drunk” (Martland 1928). These metaphors were harmful, as they implied that head trauma-related injuries were rare, temporary, and mostly experienced by reckless individuals. The metaphor of a bell ringing implies that it will stop eventually, just as a drinker returns to sobriety after ceasing to drink, masking the seriousness and permanence of these injuries.

The discovery of CTE in a former NFL player triggered a wave of diagnoses among other former athletes, all of whom shared similar traits: professional careers spanning a decade or more, disproportionate exposure to repeated head trauma, and aggressive playing styles. While

the emergence of CTE among these athletes was undeniably disturbing, it initially implicated a relatively small group of their peers as being “at risk” for developing the disease, given the limited number of individuals with similar characteristics. Perceptions of who was “at-risk” began to change in 2009 with the discovery of CTE following the accidental death of Chris Henry. Unlike the previous cases, Henry was collision-averse and had played competitive football for fewer than ten years before his death at the age of 26. Then, within a 14 month span from 2010 to 2011, four hockey players – three of whom were still active professionals – died and were diagnosed with CTE at the ages of 45, 35, 28, and 27.

The CTE-related deaths of hockey players came as a shock and brought the disease to the forefront of public awareness regarding the dangers of collision sports. Despite the inherently violent, collision-heavy nature of hockey – which involves players often weighing over 200 pounds colliding at speeds exceeding 20 miles per hour – hockey was thought to carry a significantly lower risk of CTE compared to American football. This belief likely stemmed from the lower incidence of high-profile concussions and instances of players being knocked unconscious during games. Additionally, the sport’s culture, which emphasized aggression as a key strategy for success and privileged toughness over worker safety, further contributed to downplaying the risks. However, these deaths shifted the narrative from focusing solely on concussions to recognizing the dangers of repeated blows to the head and neck.

Figure 1 presents two figures comparing the discovery of CTE among young athletes across two sports.² Figure 1a displays the timeline of CTE diagnoses among former professional athletes. In American football, diagnoses have been relatively evenly spaced, with annual fluctuations between 0 and 2 diagnoses annually for the past two decades. This contrasts sharply with hockey, which experienced a spike of 4 CTE diagnoses in 2010 and 2011, followed by relatively few in subsequent years. Figure 1b shows that, due to the relatively young ages of this first cohort diagnosed in hockey, there are significant differences in the immediacy of these deaths compared to those in football. Among the share of professional athletes who have ever had a former teammate die of CTE, hockey players are twenty percentage points more likely to have experienced this health shock less than one year after they were last teammates.

3.b. Labor Markets for Professional Athletes

There are several key factors to consider when examining differences in worker compensation, with two of the most important being superstar effects and collective bargaining agreements (CBAs). Figure 2 illustrates the distributions of worker productivity and salary across sports. In

²By “young athletes”, I specifically refer to those with at least five former teammates still playing professionally at least one year before their death.

Figure 1. Differences in the Discovery of CTE among Young Athletes across Sports

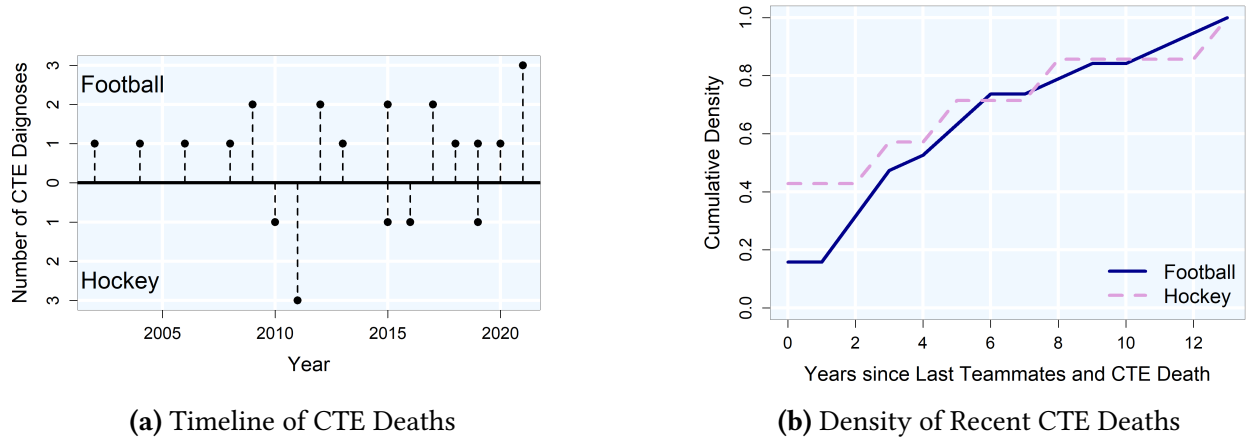
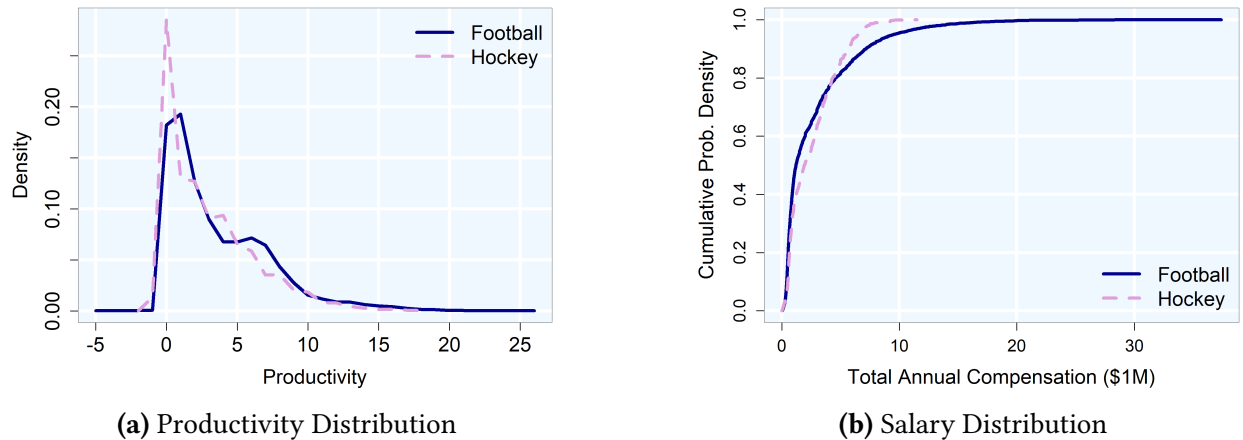


Figure 2a the distributions of productivity are normal and right-skewed across both sports. However, football has a much longer right tail in the productivity distribution, while hockey shows a larger cluster of athletes with productivity values at or near zero. This reflects the somewhat counterintuitive fact that the rules of football favor the emergence of superstar effects, likely due to the greater availability of space and an asymmetry in the rules that encourages offensive play-making.³ Figure 2b shows that the combination of greater potential for productivity and large differences in the popularity of these sports leads to significant disparities in average compensation rates, with the top 1% of earners capturing most of the income differential.⁴

Figure 2. Differences in Productivity & Salary Across Sports

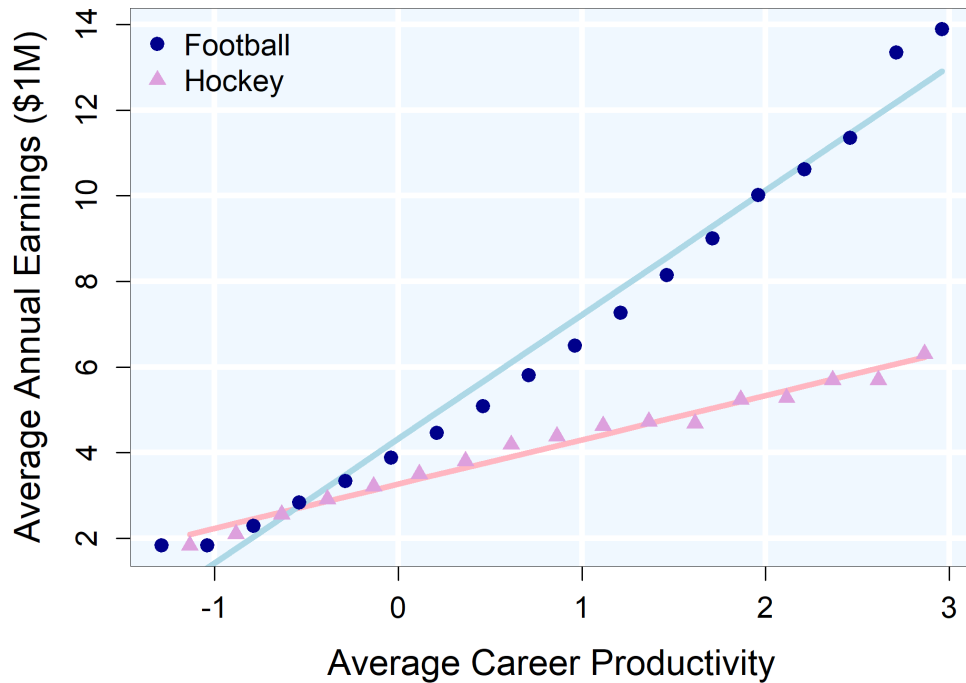


³Hockey rinks are approximately 17,000 sq. ft. with 6 players per team on the ice, while football fields are over 57,000 sq. ft. with 11 players per team on the field.

⁴Figure A7 in the Appendix shows that the championship game for American football regularly draws 20 to 40 times more viewers than the championship series in hockey. This reflects the large disparities in revenue generated by each league.

Figure 3 presents a scatter plot showing the relationship between productivity and wages, revealing a strikingly linear trend for both sports. However, this masks significant differences in the slopes of the lines. For hockey players, a one standard deviation increase in productivity is associated with an approximate \$1M (USD) increase in wages, while the same increase in productivity for football players leads to a \$2.5M (USD) wage increase. This highlights that although the typical (modal) player in both sports has similar characteristics, the rate of return on productivity gains is significantly higher in football than in hockey. This finding is reinforced for asymmetric gains at the top of the ability distribution for football players (Rosen 1981; Adler 1985).

Figure 3. Productivity and Salary



While both sports share several labor market similarities—such as pre-determined “rookie” contract wages, salary caps, and maximum salaries—there are important contextual differences. The sporadic demand for hockey, coupled with generally longer career spans, led to the introduction of largely guaranteed contracts in the NHL’s 2005 Collective Bargaining Agreement (CBA). This contrasts sharply with American football, where an abundance of potential replacement talent undermines union bargaining power. As a result, employment protections in football are weak, and guaranteed compensation is primarily reserved as a tool to attract highly productive free agents. However, the cost of these guarantees for hockey players has been restrictive salary caps, including the possibility of escrowing payments – that is, withholding player salaries if league revenues fall below a certain threshold.⁵

⁵In 2024, the salary cap per worker was \$6.2M (USD) in American football and \$3.8M (USD) in hockey.

4 Theoretical Model

I formalize a mathematical theory of how workers' labor supply decisions can be influenced by the salience of peer health shocks in the context of violent, collision sports. Workers maximize lifetime utility by choosing whether to exit the profession each period, balancing the trade-offs between wage differentials across professions with greater risks to one's health and longevity. This is expressed in Equation 1,

$$U_i = \max_{d_{ist}} \sum_{t=T_0}^T \delta^{t-T_0} (d_{ist} \cdot \omega_{ist} + (1 - d_{ist}) \cdot \eta_{ist}) \quad (1)$$

where workers (i) maximize utility (U) from the present period (t) until retirement (T) by deciding ($d \in \{0, 1\}$) whether to continue earning wages from professional sports (ω) or exit to earn wages in their next best alternative profession (η). The discount factor (δ), reflects time preferences, weighting future wages less than current wages.

The constraining trade-off in this utility maximization problem is shown in Equation 2, which illustrates that non-sport wages are determined by two factors: the worker's human capital in the labor market (μ) and the number of years they can work.⁶ Specifically, in this context, continued participation in professional sports is physically taxing, which reduces the number of years a worker can earn non-sport wages by some amount (θ).⁷ I will broadly refer to this parameter as the longevity penalty.

$$\eta_{ist} = \mu_{it} - \theta_{ist} \quad (2)$$

I allow the longevity penalty to vary across individuals, as shown in Equation 3. The penalty consists of two components: known workplace hazards (γ) and a residual term (ϵ). The first component reflects the risks of workplace participation known to all athletes within each sport by year, while the residual captures all remaining individual-specific disamenities. Common theoretical assumptions – whether workers have complete information about workplace safety or that residual disamenities are randomly distributed – predict that the expected value of the residual term is zero.

⁶It is assumed that one cannot improve their human capital while employed as a professional athlete, so their human capital is fixed prior to period t .

⁷For simplicity, this parameter is modeled as a reduction in annual non-sport wages, rather than in the number of years one can work (T).

$$\theta_{ist} = \gamma_{st} + \epsilon_{ist} \quad (3)$$

Equation 4 illustrates that the second major component of worker utility, sport wages, is determined by two primary factors. The first factor represents the value of the marginal product of labor through the product of market demand (ρ) and worker's productivity (v).⁸ Second, the theory of compensating wage differentials predicts that wages are influenced by the disamenity associated with participating in the sport (θ) which manifests in Equation 3 as long-term health issues. The degree to which these disamenities are reflected in wages depends on worker's bargaining power (β).

$$\omega_{ist} = \rho_{st} \cdot v_{ist} + \beta_{st} \cdot \theta_{ist} \quad (4)$$

The factors influencing worker bargaining power are shown in equation 5. Game theoretically, the credibility of player's threats improve bargaining outcomes. The credibility of the threat to retire from professional sports is captured by the first parameter which measures the value of the worker's human capital in the labor market (μ). The second component in this equation measures superstar effects. There are imperfect substitutes for highly skilled labor. The number of suitable replacement workers shrinks as worker productivity (v) increases, which increases worker bargaining power. This measure of productivity is augmented with a measure of the strength of the collective bargaining agreement (π) binding the worker by sport and year.

$$\beta_{ist} = \mu_i + \pi_{st} \cdot v_{ist} \quad (5)$$

Very simply, each period workers will remain working in sports if their sport wage is higher than their non-sport wage ($\omega_{ist} \geq \eta_{ist}$). Representing this decision by substituting equations 2 and 5 can be seen in 6.

$$\rho_{st} \cdot v_{ist} + (\mu_i + \pi_{st} \cdot v_{ist}) \cdot (\gamma_{st} + \epsilon_{ist}) \geq \mu_i - (\gamma_{st} + \epsilon_{ist}) \quad (6)$$

This model can be simplified by incorporating unit and time fixed effects, helping to isolate relevant factors related to what cause *changes* in individual labor supply decisions. These fixed

⁸An implicit assumption of this equation is that market demand for a sport is not causally related to the risks its workers face. While this assumption is clearly false in a literal sense, the highly inelastic demand for both hockey and football displayed in Figure A7 with respect to the wide-spread health concerns for the athletes at the time suggests that any relationship between the two variables is likely to provide both little explanatory benefit and possibly even obfuscate the model through additional complexity.

effects differences out factors that are constant over time for each individual and for factors that are constant across individuals within the same time period.⁹ The results of this process are displayed in Equation 7.

$$\Delta_{it}(v_{ist} + \epsilon_{ist} \cdot (v_{ist} + 1)) \geq 0 \quad (7)$$

The results from Equation 7 yield two theoretical predictions. First, if workers have full information about workplace safety, or if residual risk perceptions do not vary systematically across workers, changes in productivity are the only relevant factor explaining voluntary exits from risky workplaces. Second, if increased risk perceptions affect different groups of workers unevenly, the impact of such shocks on exit decisions will depend on whether a worker’s productivity is rising or falling. Though this outcome may seem counterintuitive, it is theoretically consistent. For example, more productive workers can expect greater gains from remaining in the sport after a negative health shock, as their higher productivity increases future wages both directly, through the value of their marginal product and bargaining power, and indirectly, through the exit of less productive workers. Conversely, workers with declining productivity will be more likely to exit following a work-related health shock for the same reasons.

5 Data

I gather data from two sources to empirically test the impact of peer health shocks on labor supply. The primary dataset is sourced from [Sports Reference](#), a collection of websites offering comprehensive statistics and historical data across various sports. A key advantage of this dataset is its wealth of athlete-specific information, which allows me to track the movements of these workers both leading up to and throughout their professional careers. Importantly, this enables me to observe not only their former teammates during their professional careers but also occasionally during college, semi-professional teams, and even high school.

For analysis, I construct player-by-season records for all professional American football and hockey players who made the final roster for any team in the premier divisions of these sports from 1970 to 2023. The profiles of these athletes include a variety of details, such as years of participation in university or amateur teams, the name of their high school, city of birth, athlete relatives, recruiting rank, draft order, date of birth, and, when applicable, date of death. Additionally, I gather data on positional information, accolades, preseason championship expectations (as

⁹Unit fixed-effects can be mathematically expressed as $\Delta X_i = X_{it} - \frac{1}{T} \sum_{\tau=1}^T x_{i\tau}$ while time fixed effects can be written as $\Delta X_i = X_{it} - \frac{1}{I} \sum_{t=1}^I x_{it}$. For notational simplicity, I denote unit and time fixed-effects as Δ_i and Δ_t respectively.

measured by implied probabilities from pre-season betting markets), and game participation.

Most importantly, for identification purposes, this dataset contains continuous measures of worker productivity. For hockey, I use player-by-season estimates of “Point Shares,” which attribute a player’s contributions to their team’s total points in a season. In football, I employ the “Approximate Value” metric, which similarly measures a player’s contribution to their team’s chances of winning.¹⁰ These measures are notable for their ability to harmonize statistics across players in vastly different positions within their sport, while also making productivity comparable across time, even as the rules and strategies of the sports evolve.¹¹

To account for differences in the number of games played by teams over time, I standardize these productivity measures within each sport and year, such that a one-unit change reflects a one standard deviation difference in productivity relative to other athletes in the same sport and year. As a result, the mean value of productivity in this analysis is zero, which reflects the threshold below which athletes are likely to be fired and replaced.¹²

I then match this with a secondary dataset sourced from [Spotrac](#), a website that provides financial information about player contracts in various professional sports, including American football and hockey. Though Spotrac is primarily focused on tracking salary caps – that is, the extent to which teams are bound by league-imposed spending limits on player salaries, this platform offers the most detailed and comprehensive data on contracts signed by professional athletes available at the time of this writing. The dataset includes information on the total value of contracts, the duration of employment, and detailed breakdowns of compensation into guaranteed and variable components over time. Additionally, contracts are dynamically updated to reflect extensions and terminations. This rich dataset allows me to calculate the exact dollar amount workers would forgo upon retirement in each year.

After merging these datasets, the final analysis sample consists of 2,312 unique hockey players, of whom 977 (42%) have complete salary information, and 538 (23%) are identified as having had a former teammate die from CTE. The sample also includes 8,817 unique football players, with 4,916 (56%) having complete salary data and 1,244 (14%) are identified as having had a former teammate die from CTE.¹³

¹⁰These measures are slight derivations of the concept of “Win Shares,” introduced in the late 1970s by writer and statistician Bill James, who, perhaps unsurprisingly, holds a degree in Economics as these measures often closely resemble concepts from the discipline such as the marginal product of labor.

¹¹For a detailed analysis of the correlates of worker productivity, see Figure A1 in the Appendix.

¹²For an in-depth explanation of how Approximate Value and Point Shares are calculated, see pro-football-reference.com/about/approximate_value.htm and hockey-reference.com/about/point_shares.html.

¹³For more information regarding missingness of contracts over time and across players, see Figure A12 in the Appendix.

6 Methodology

6.a. Identification

To examine the impact of peer health shocks on labor supply decisions in professional sports, I compile a list of all former athletes reported to have been diagnosed with CTE. A subset of this list, used for analysis, is presented in Table 1. The selection criteria include only those athletes who had at least 30 former teammates still active in professional sports at least four years before their death, and at least five remaining one year before their death.

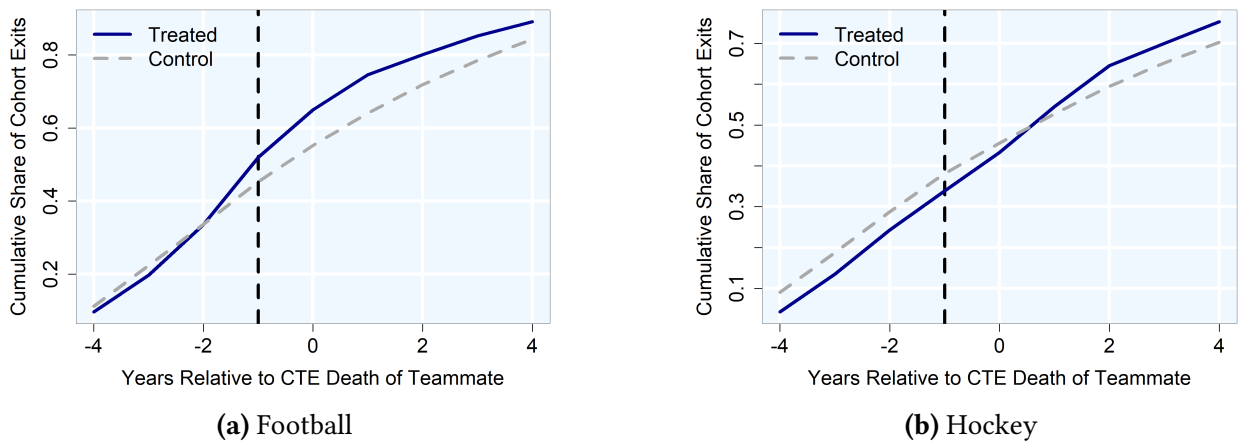
Table 1. CTE-Diagnosed Athletes with Remaining Teammates at Time of Death

Player	Sport	Career	Date of Death	Teammates at $\tau = -4$
Mike Webster*	Football	1974-1990	9/24/2002	22
Justin Strzelczyk	Football	1990-1998	9/30/2004	61
Andre Waters*	Football	1984-1995	11/20/2006	13
Tom McHale*	Football	1987-1995	5/25/2008	16
Shane Dronett	Football	1992-2001	1/21/2009	40
Chris Henry	Football	2005-2009	12/17/2009	137
Bob Probert	Hockey	1986-2002	7/5/2010	53
Derek Boogaard	Hockey	2006-2011	5/13/2011	102
Rick Rypien	Hockey	2006-2011	8/15/2011	92
Wade Belak	Hockey	1997-2011	8/31/2011	151
Junior Seau	Football	1990-2012	5/2/2012	157
Jovan Belcher	Football	2009-2012	12/1/2012	116
Paul Oliver	Football	2008-2011	9/24/2013	115
Steve Montador	Hockey	2002-2012	2/15/2015	141
Adrian Robinson	Football	2012-2013	5/16/2015	115
Tyler Sash	Football	2011-2012	9/8/2015	80
Marek Svatoš	Hockey	2004-2011	11/4/2016	59
Aaron Hernandez	Football	2010-2012	4/19/2017	71
Daniel Te'o-Nesheim	Football	2010-2013	10/29/2017	117
Kevin Ellison*	Football	2009-2009	10/4/2018	16
Greg Johnson*	Hockey	1994-2006	7/7/2019	14
George Atkinson	Football	2014-2016	12/2/2019	117
Max Tuerk	Football	2017-2017	6/20/2020	68
Vincent Jackson	Football	2005-2016	2/15/2021	92
Phillip Adams	Football	2010-2015	4/8/2021	114
Demaryius Thomas	Football	2010-2019	12/9/2021	199

Two key exogenous forces are relevant in this context: the limited ability of workers to endogenously cluster into teams and the quasi-random timing of these deaths. However, exogeneity

alone does not guarantee unbiased estimation of treatment effects. Figure 4 highlights an important concern, showing substantial differences in exit rates between treated and untreated workers leading up to a peer health shock. These differences suggest that the full-sample control group may not serve as a convincing counterfactual for treated workers. This issue likely stems from endogenous selection related to worker turnover. Specifically, treated workers, by definition, have played long enough to experience the death of a former teammate from CTE. In a setting where the typical career length is 3 to 5 years, even small differences in factors that extend career longevity can lead to significant discrepancies in both trends and levels of the outcome.

Figure 4. Unadjusted Cumulative Share of Cohort Exits

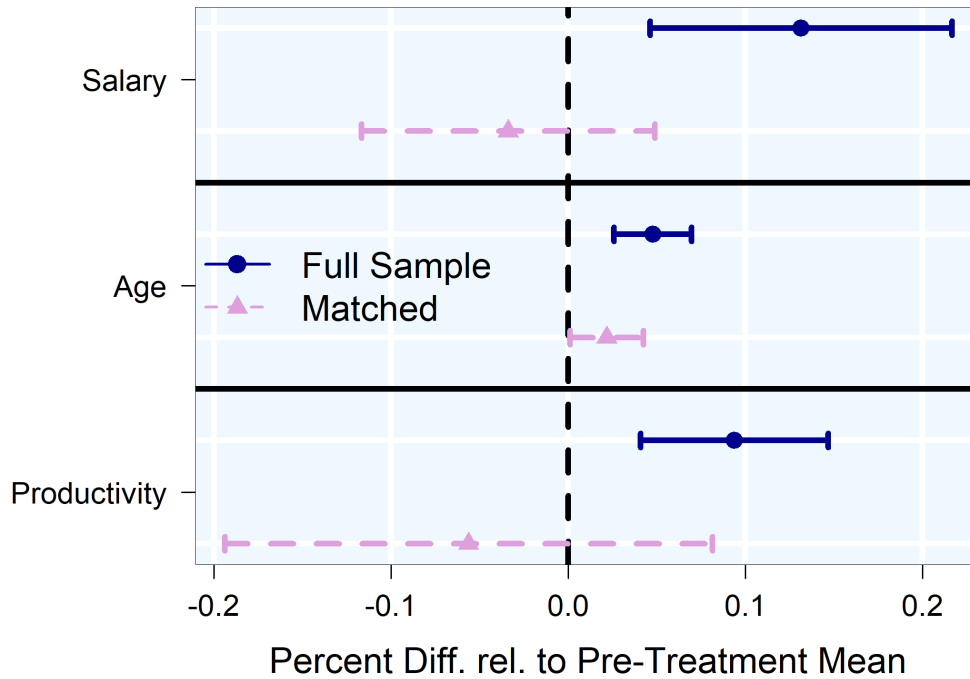


To address concerns over differences between my comparison groups, I use a Coarsened Exact Matching (CEM) method, which adjusts for pre-treatment covariate differences between treated and control groups. The primary advantage of CEM over other matching methods is that it ensures both the mean and distribution of each covariate are similar across groups by “coarsening” their values into small bins, or “strata.” This method generates two key outputs: indicators for poorly matched control units and a numerical weight that accounts for any remaining differences between the groups. I primarily rely on the former output, as the large number of workers in both the treated and control groups often makes weighting unnecessary.

The results from this matching process, which relies solely on two covariates – worker age and productivity – are displayed in Figure 5. As expected, treated workers in the full sample earn approximately 13% more in salary, are 5% older, and are 10% more productive. After matching, no statistically significant differences remain, as the process excludes control group athletes who are the most dissimilar to the treated group in terms of these characteristics.

Figure 6 demonstrates that, in the matched sample, the pre-treatment trends in the outcome

Figure 5. Covariate Balance



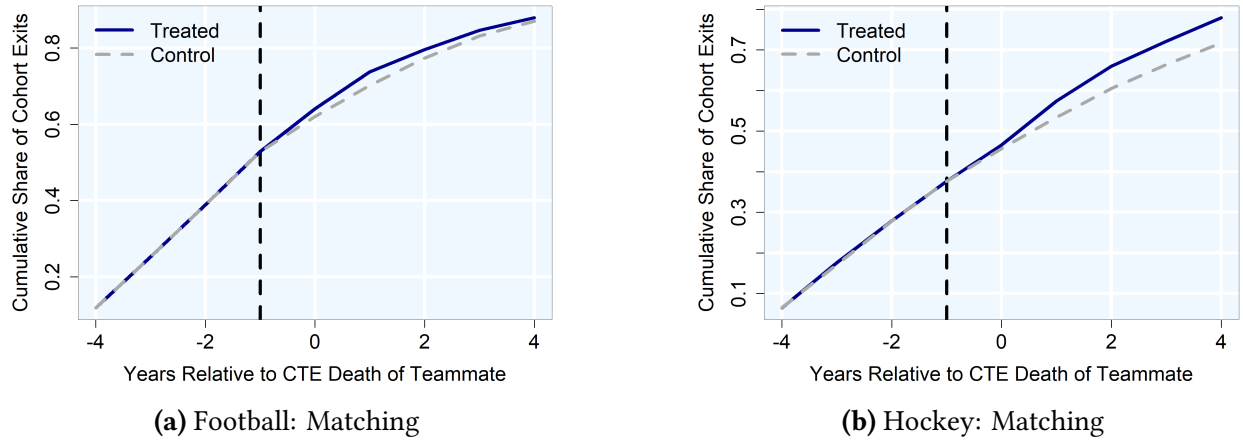
variable are both parallel and approximately equivalent across the two groups.¹⁴ The figure also shows a significant increase in the rate of exit for treated workers, which occurs only after the timing of the treatment.

In order to examine whether the results from Figure 6 are attributable to changes in group-level characteristics after the timing of treatment, I turn to the usage of difference-in-differences models which allow one to more convincingly isolate the effects of treatment through the usage of worker and year fixed effects. Individual-level characteristics that are time-invariant throughout the sample such as players' height, race, educational achievement, position, draft order, and talent are captured by using player fixed-effects. Time-fixed effects, on the other hand, capture variation in changes in the dependent variable that are unique to that period, such as worker strikes, changes in the rules (or enforcement of said rules) about player safety, the signing of new collective bargaining agreements, variation in league revenue, and the establishment of commissions tasked with providing payouts to former athletes suffering from work-related neuro-trauma.

While the two-way fixed-effects models that are estimated using individual-level data allow one more peace of mind that omitted variables are not biasing treatment effects, endogenous behav-

¹⁴This is particularly important given the bounded nature of the outcome variable. For example, the rate of cohort exits is likely to asymptotically approach an upper bound of one over time. This suggests that groups with lower exit rates prior to treatment may close gaps in the outcome due to inherent properties of the variable, rather than treatment effects.

Figure 6. Matched Trends in Cumulative Share of Cohort Exits



ioral responses to CTE deaths, which disproportionately change for treated players over time, would be a major threat to identification. For instance, one might reasonably fear that important unobservable individual or team-level factors, such as increased shirking or greater usage of personal protective equipment while working, might bias the estimate of the “true” treatment effect since workers would be less likely to alter their behavior with less potential for injury.

There are several key points worth highlighting in this sports context that should alleviate valid concerns of threats to identification. First, the wages earned by these workers are many times greater than what these workers would likely earn if not employed as professional athletes. These relatively high wages lead to a massive surplus of workers willing and able to replace shirking athletes. Relatedly, the coach determines the players’ intensive-margin labor supply decisions exogenously. Failure to follow orders from higher-ranking team employees gives teams the legal ground to terminate players’ employment contract.¹⁵ Relatedly, professional sports are extraordinarily competitive, and worker productivity is paramount. Teams will fire players when the marginal value of their labor drops below the marginal cost of their employment.¹⁶

Second, most safety policies and equipment are unlikely to provide any additional protection against the risk of developing CTE. The gradual expansion and professionalization of team sports, coupled with an increasing focus on the importance of health and recovery, has meant that athletes (relatively) seldom experience concussions during practice. The riskiest contact professionals are exposed to occurs during games where their opponents are strongly incentivized to use dangerous force. However, a far greater reason that attempts to limit concussions in collision sports are likely futile, is that helmets and other improvements in protective equipment aid

¹⁵Insubordinate athletes would be correctly identified in my model as having exited.

¹⁶See Figure A1 for a visualization and more lengthy discussion of this phenomenon.

primarily in impact absorption rather than in limiting movement of the brain within the skull. Similar to what has been implanted to reduce concussions in auto-racing, meaningful reductions in concussions are likely unable to meaningfully reduce unless a firm harness holds one’s neck in place at all times to prevent whiplash upon contact (Kaul et al. 2016). Nevertheless, players’ beliefs of these policies’ efficacy will ultimately determine their behavior.¹⁷ Even if every treated player were fully convinced of the efficacy of these interventions, this would bias treatment effect estimates towards zero rather than away from it.

6.b. Estimation

To address the well-known biases associated with using two-way fixed effects (TWFE) models with staggered treatment timing, as identified in the modern econometrics literature, I use a stacked event-time approach by treatment cohort. Commonly referred to as a “stacked” difference-in-difference estimator, this method follows the familiar TWFE estimation process but requires restructuring the data to center it in event-time. Specifically, stacking involves selecting an event-time window of nine years (four years pre-treatment and five years post-treatment for this analysis). After keeping only the information within this period, I store the IDs of all treated workers to ensure they are not later used as control units. Finally, I create a unique “stack” identifier for each cohort. This process is repeated for all unique treatment timings, and the stacks are ultimately combined into one dataframe for analysis.

The process of restructuring the data this way can be seen in the estimating equations below. Equation 8 tests worker i in stack s in year (t) exits (E) from professional sports. This model relies upon worker fixed effects (α) and year fixed effects (δ). A treatment indicator T is equal to zero for all units prior to treatment, and then is set equal to one for all former teammates of a worker who died of CTE after their death. The coefficient β represents the differential impact of the likelihood of exiting the profession after treatment between treated and control groups.

$$E_{sit} = \alpha_{si} + \delta_{st} + \beta T_{sit} + \epsilon_{sit} \quad (8)$$

Equation 9 estimates the dynamics of the effect of a peer health shock on the probability of exit. The period immediately prior to treatment ($\tau = -1$) serves as the reference period. There are two major advantages of this model. First, it allows one to test whether the assumption that the outcomes of the two groups would continue trending parallel to one another in the absence of treatment. Second, it allows one to visualize how the effects evolve over time.

¹⁷Though this is unlikely give the well-documented unintended consequence that safety measures for head injuries lead to increases in lower-body injuries (Hanson, Jolly, and Peterson 2017).

$$E_{sit} = \alpha_{si} + \delta_{st} + \sum_{\tau=-5}^{-2} \beta_{\tau} T_{i\tau} + \sum_{\tau=0}^{-5} \gamma_{\tau} T_{i\tau} + \epsilon_{sit} \quad (9)$$

Equation 10 displays a Poisson Regression model which mirrors equation 8 but for its log transformation of the expected value of the dependent variable. This is done primarily for the salary outcomes which are highly skewed. Thus, the coefficient of interest in this regression (β) measures the percentage (rather than percentage point) change in the dependent variable caused by treatment.

$$\log(E[Y_{sit}]) = \alpha_{si} + \delta_{st} + \beta T_{sit} + \epsilon_{sit} \quad (10)$$

This study employs both balanced panel and unbalanced data. The latter dataset can be conceptualized as a panel of team rosters, which contain detailed information about each worker. Crucially, no new workers are allowed to enter the sample after the treatment timing within each stack. The dependent variable of interest, “exit,” is equal to zero until it takes a value of one the last time a worker is observed in the dataset. Crucially, this setup captures changes in the composition of the workforce over time. As such, only the remaining workers in each period serve as comparison groups for one another. This contrasts with the balanced panel dataset, which requires that individuals remain in the sample for every period. In this case, the dependent variable “exit” is set to zero until it takes a value of one for every period after the worker is last observed in the unbalanced panel.¹⁸

Therefore, differences in estimated treatment effects between these groups can be interpreted as differences in comparison groups: either relative to all workers in the balanced panel or relative to only the remaining workers in each period within the unbalanced panel. This suggests that treatment effects may be more sensitive when estimated using the unbalanced data, where the gradual attrition of workers creates a more dynamic workforce composition, compared to the balanced panel, where comparison groups remain static over time. However, this dynamically changing workforce composition may bring the benefit of potential for more appropriate comparisons if there is endogenous in pre-treatment retirement decisions.

¹⁸Age and productivity measures are used in the matching method. For periods where we do not directly observe worker information on rosters, players’ ages are extrapolated from their birth dates, and productivity is set to zero.

7 Results

7.a. Professional Exit (Retirement)

Table 2 presents empirical estimates of the differential impact of well-publicized deaths related to workplace safety on the probability that their former coworkers exit the profession. Estimating models using data from the entire universe of athletes on NFL and NHL rosters in the years surrounding these deaths, each regression model in Table 2 reveals that CTE deaths significantly increase the probability that former teammates retire.

Table 2. The Effect of the CTE Death of a Former Teammate on Retirement

Dependent Variable:	$\mathbb{1}(\text{Exit}) \times 100$			
Sample:	Full		Matched	
Model:	(1)	(2)	(3)	(4)
Panel A (Balanced)				
CTE (Post) \times Teammate	4.58*** (0.78)	1.79** (0.70)	2.53*** (0.80)	2.24*** (0.72)
Productivity		-13.21*** (0.08)		-13.30*** (0.12)
Pre-Treatment Mean Retire	29.03	29.03	29.03	
% Change	0.16	0.06	0.09	0.08
Observations	409,383	409,383	229,833	229,833
Panel B (Unbalanced)				
CTE (Post) \times Teammate	3.03*** (1.05)	1.89* (1.00)	4.00*** (1.09)	3.97*** (1.04)
Productivity		-10.03*** (0.12)		-10.12*** (0.16)
Pre-Treatment Mean	17.28	17.28	17.28	17.28
% Change	0.18	0.11	0.23	0.23
Observations	190,886	190,797	110,561	110,516

Ordinary Least Squares (OLS) is used to estimate each model which all include stack-by-player and stack-by-year fixed-effects. The dependent variable *Exit* is a binary estimate that is multiplied by one hundred for presentational simplicity. The mean of the dependent variable is calculated from pre-treatment observations of players in the treated group. *Clustered (Stack-by-Player) standard-errors in parentheses.* Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

The binary dependent variable used in Table 2, *Exit*, is multiplied by one hundred to express the coefficients in percentage terms, making the result easier to interpret and preventing the effect sizes from being obscured by small decimal values. Measures of worker productivity are standardized in order to provide a more easily comparable method for comparing the magnitude

of the treatment effect on retirement with that of a one-standard-deviation change in worker productivity.

Panel A of this table analyzes a balanced sample, meaning individuals do not drop out of the dataset after retirement. Thus, the coefficients in this panel represent changes and persistence in the probability of exiting due to treatment compared to all other workers on rosters prior to treatment. Panel B is unbalanced, meaning that coefficients on treatment indicators from this panel reflect changes in the probability of exiting relative to *remaining* workers. Estimates from the later model are inherently more sensitive due to the smaller sample sizes. However, they are included in the table because groups of remaining workers could reflect more suitable counterfactual groups in the periods surrounding treatment.

Models one and two of Table 2 are estimated using data on all workers. Treatment effect estimates across model one in each panel reflect significant treatment effects, which diminish significantly when conditioning on worker productivity. As discussed in the Identification section of this paper, the large change in these estimates from models one to two suggests significant differences in productivity across workers in the treatment and control groups. Thus, my preferred estimates come from models three and four of Table 2 which are estimated using data from a sample of workers who are characteristically similar prior to treatment. Adding a control for worker productivity in model four does not significantly alter the coefficients on the treatment indicators, suggesting the usage of the more appropriate control group, and to stress that the results are not sensitive to the choice of sample that is analyzed.

The results from model four of panels A and B suggest that treatment increases the probability of exit from the profession by 2.24-3.97 percentage points. Along with being statistically significant, these effect sizes are meaningfully large. The magnitude of the treatment indicators suggests that the CTE death of a former teammate increases the probability of retirement by 8-23% relative to the pre-treatment mean. This effect size is approximately equivalent to a 0.16-0.39 standard deviation reduction in worker productivity.

To explore the mechanisms driving this increase in the probability of retiring, I stratify these results by sport in Table 3. Models one and two (three and four) examine the treatment effects for hockey (football), while odd-numbered models use the full control group and even-numbered models use the matched samples. The key finding of this table is that the treatment effect is unambiguously larger in hockey than in football.

The results of Table 3 align with expectations, reflecting important contextual differences across sports, especially if familiarity and proximity are plausible mechanisms driving this outcome. For instance, the smaller team sizes, longer careers, extended periods as teammates, more

Table 3. Peer Work-Related Deaths & Retirement: Heterogeneity by Sport

Dependent Variable:	$\mathbb{1}(\text{Exit}) \times 100$			
Sport:	Hockey		Football	
Sample:	Full	Matched	Full	Matched
Model:	(1)	(2)	(3)	(4)
Panel A (Balanced)				
CTE (Post) \times Teammate	7.08*** (1.39)	4.42*** (1.41)	3.44*** (0.94)	1.61* (0.98)
Pre-Treatment Mean	22.4	22.4	32.21	32.21
% Change	0.32	0.2	0.11	0.05
Observations	74,619	52,893	334,764	176,940
Panel B (Unbalanced)				
CTE (Post) \times Teammate	5.08*** (1.51)	5.15*** (1.53)	1.76 (1.42)	3.21** (1.51)
Pre-Treatment Mean	13.46	13.46	19.13	19.13
% Change	0.38	0.38	0.09	0.17
Observations	39,754	29,311	151,132	81,250

Ordinary Least Squares (OLS) is used to estimate each model which all include stack-by-player and stack-by-year fixed-effects. The dependent variable *Exit* is a binary estimate that is multiplied by one hundred for presentational simplicity. The mean of the dependent variable is calculated from pre-treatment observations of players in the treated group. *Clustered (Stack-by-Player) standard-errors in parentheses. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.*

abrupt CTE-related deaths, and the greater relative ‘surprise’ surrounding the discovery of CTE among former teammates are all more pronounced in hockey than in football. These factors suggest that hockey players should have a more elastic labor supply response than football. Further, the relative time proximity difference in the timing of these deaths means that counter-factual groups can be more easily constructed in the hockey setting than for football. This can be seen in changes in the number of observations employed in the matched samples relative to the full data, falling 25~30% in the hockey setting and nearly 50% for football.

The coefficients of Table 3 reveal large treatment effect sizes. The preferred models, which use a matched control group (models two and four), reveal effect sizes ranging from 4.42-5.15 percentage points for hockey and 1.61-3.21 for football. These effect sizes correspond to 20-38% increases relative to the pre-treatment mean for hockey and 5-17% for football.

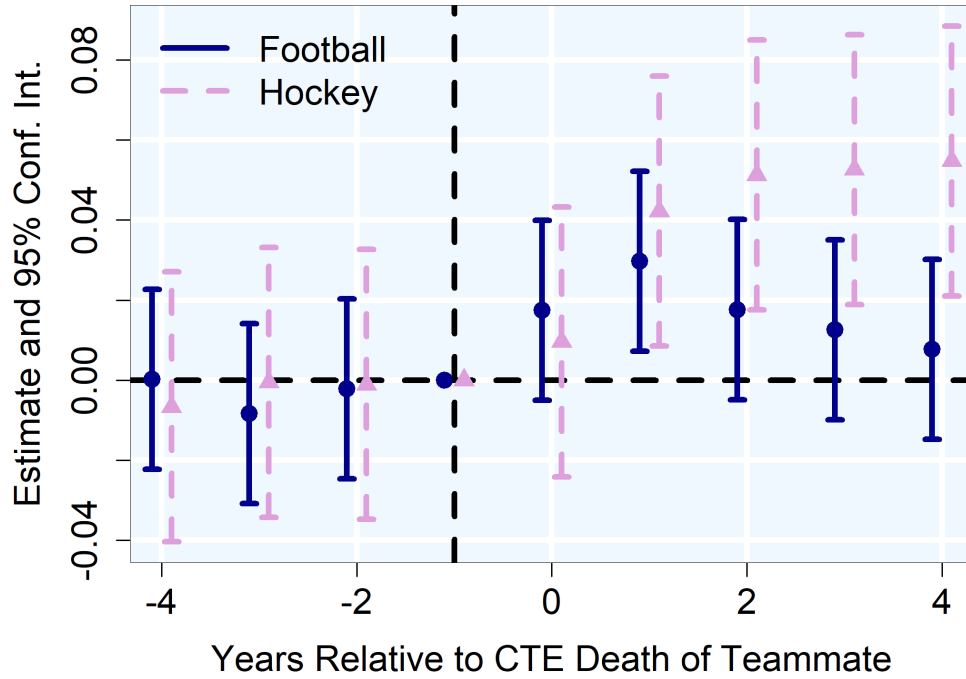
In order to investigate the dynamics of the effect of treatment across sports, I estimate two event study models via Equation 9 using the matched control group for hockey and football players. Estimates of pre-treatment differences in exit between the treated and control players are tightly centered around zero, providing evidence supporting the hypothesis that outcomes between the two groups would continue trending parallel to one another absent treatment. Following the timing of treatment, treated players in both sports see large, statistically significant increases in the probability of exit relative to the control group. However, both the magnitude and the dynamics of this post-treatment increase differ meaningfully across the sports, where the treatment effect for hockey is larger but also increases in magnitude over time. These results contrast with the smaller, transitorily treatment effect seen in football.

Notably, the treatment effect for both sports seems to be delayed one season, particularly in the case of hockey players whose estimates increase from approximately 1 percentage point in period zero to 4 percentage points in the following period. One potential reason for the (relatively) delayed labor supply response is the difference between the timing of a former teammate’s death and their eventual CTE diagnosis.¹⁹ Additionally, hockey contracts are longer on average than those seen in football. Thus, we would expect to see more delayed exit responses in hockey if individuals leave the sport after their contract at the time of treatment. I explore heterogeneity in contract structure as a driving force behind differences in retirement in the following subsection of this paper.

To further explore the mechanisms driving the heterogeneous labor supply responses that

¹⁹For instance, there was a delay of over 200 days between the date of death of Bob Probert (the first hockey player with teammates still in the league to be diagnosed with CTE) and his eventual diagnosis. Though delays of this magnitude are relatively common, speculation that the athlete died with CTE is more common in football, where the salience of the disease is more prevalent. For a more detailed discussion of this topic, please refer to Table A1 and its corresponding section in the Appendix.

Figure 7. Event Study of the Effect of the CTE Death of a Former Teammate on Professional Exit



individuals display in response to the death of a coworker, I examine familiarity as an explanatory mechanism. These results can be seen in Table 4, which indicate that individual labor supply responses to peer health shocks exhibit a more elastic response as the amount of times individuals spent together on a team increases. In panel A of this table, the results are stratified by length of time spent as teammates of periods of one, two, and three years.²⁰ The results increase across each specification from 2.53 percentage points in model one to 4.67 percentage points in model three. This increase is even larger in percentage terms, rising from 9%-19% across the models as individuals who were teammates for longer have marginally lower probabilities of having retired prior to treatment.

As seen at the bottom of panel A of Table 4, individuals treated for longer periods have lower average pre-treatment differences in the share of those who have already exited and are slightly older. These age differences raise the possibility that the observed effect attributed to familiarity may instead reflect reversion to the mean, as older players are generally more likely to retire, all else being equal. Thus, I explore the role of heterogeneity in these findings across the age

²⁰Over 60% of treated units were only teammates with a coworker who died of CTE for one season, as shown in Figure A9 in the Appendix. This magnitude of this relationship can be explained by a combination of high turnover in these sports and the young age at which their former teammates die. Over ninety percent of all treated players were on the same team for four or fewer seasons, which sharply limits the ability to stratify results beyond this cutoff without running into issues of statistical power.

Table 4. Peer Work-Related Deaths & Retirement: Heterogeneity by Teammate Duration

Dependent Variable:	$\mathbb{1}(\text{Exit}) \times 100$		
Model:	(1)	(2)	(3)
Panel A (Length of Time as Teammates)			
CTE (Post) \times Teammate	2.53*** (0.80)	4.30*** (1.22)	4.67*** (1.69)
Pre-Treatment Mean	29.03	25.69	24.87
% Change	0.09	0.17	0.19
Observations	229,833	218,853	215,226
Mean Pre-Treatment Age	27.75	28.59	29.31
Length of Time Teammates (L):	$L \geq 1$	$L \geq 2$	$L \geq 3$
Panel B (Teammates by Age)			
CTE (Post) \times Teammate	0.10 (2.22)	3.70** (1.53)	0.56 (1.72)
Pre-Treatment Mean	10.8	17.31	36.27
% Change	0.01	0.21	0.02
Observations	97,794	131,022	32,643
Pre-Treatment Mean Age	22.36	26.03	32.25
Age Percentile (P):	$P < 0.3$	$0.3 \leq P < 0.6$	$P \geq 0.6$

Ordinary Least Squares (OLS) is used to estimate each model which all include stack-by-player and stack-by-year fixed-effects. Panel A subsets the treated group by the amount of time spent as teammates whereas panel B subsets the treated group by age quantiles. The age ranges for each model are 19-27, 28-31 and 32-48 years old. The dependent variable *Exit* is a binary estimate that is multiplied by one hundred for presentational simplicity. The mean of the dependent variable is calculated from pre-treatment observations of players in the treated group. *Clustered (Stack-by-Player) standard-errors in parentheses. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.*

distribution in panel B. The models from this panel stratify the results by the age percentile that players were in at the time of treatment consisting of individuals aged 18-25 (age percentile (P) $< \frac{1}{3}$), 26-29 ($\frac{1}{3} \leq P < \frac{2}{3}$) and 30 or older ($P \geq \frac{2}{3}$). Unlike in panel A of Table 4 where differences in the pre-treatment mean are negligible across each model, models one and three of panel A have differences in the pre-treatment mean of over twenty-five percentage points. Thus, in order to construct suitable counterfactual groups for each of these newly defined treated groups, I use the same iterative regression-based covariate balancing method described in the Methods section.²¹

The results of panel B of Table 4 reveal that, within this setting, the effect of workplace safety-related peer health shocks on extensive margin labor supply elasticities does not linearly increase with age. The estimates from models one and three, which analyze the youngest and oldest thirty-three percentiles of athletes who were exposed to the CTE death of a former teammate have statistically insignificant 0.1 (1%) and 0.56 (2%) percentage point increases in the probability of exiting the profession after their teammate's death relative to characteristically similar athletes. These effects contrast with the large and statistically significant 3.7 (21%) percentage point increase in the rate of exit among those in the middle of the age distribution. These findings reinforce the plausibility that the findings presented in panel A of this table are attributable to familiarity and not age.

Many factors could explain the findings from panel B of Table 4. For instance, differences in earnings potential between younger and mid-career athletes may play a significant role. Younger athletes, who are often still on their first ("rookie") contracts and whose wages are artificially constrained by binding price ceilings, face high expected opportunity costs of leaving the profession given the asymmetrically large salary benefits which can be earned after entering "free agency" where players can face more competition for their services. Mid-career athletes who have likely already secured contracts in free agency may face lower future expected opportunity costs and thus may be more sensitive to peer health shocks. In contrast, older athletes who have played for long periods may exhibit fatalistic risk preferences regarding CTE. Since CTE develops due to *cumulative* blows to the head, these athletes might feel that their risk is already cemented, regardless of whether they exit the profession.

Another plausible mechanism driving heterogeneity in the labor supply responses between sports may arise due to differences in the temporal proximity of the CTE death of a colleague. As can be seen in Figure 1, the discovery of CTE within hockey not only occurred far more suddenly than in football but also many of these individuals died much closer to the ends of their careers.²²

²¹The event study estimates from these models can be seen in Figure A8 in the Appendix, providing evidence in favor of the hypothesis that the parallel pre-trends assumption required the estimation of treatment effects in difference-in-differences models has been satisfied.

²²In the case of the three NHL players who died in 2011 and were later diagnosed with CTE, none had yet

To examine this hypothesis, I stratify the treated group based on the number of years between the last time an individual was on the same team with someone who died (and is later diagnosed with CTE) and the date of their death. This stratification allows one to test whether there is a gradient of labor supply responsiveness with respect to the temporal proximity of peer health shocks. Results from Table 5 provide evidence in favor of the hypothesis that individuals respond to peer health shocks more strongly when they are teammates more recently.

Table 5. Peer Work-Related Deaths & Retirement: Heterogeneity by Temporal Distance

Dependent Variable:	1(Exit)*100		
Temporal Treatment Distance (D):	$D \leq 2$	$D \leq 4$	$D \leq 6$
Model:	(1)	(2)	(3)
Panel A (Temporal Treatment Distance)			
CTE (Post) \times Teammate	3.14** (1.45)	1.78* (0.96)	1.91** (0.88)
Pre-Treatment Mean	10.45	22.27	28.29
% Change	0.3	0.08	0.07
Observations	95,049	202,824	219,366
Panel B (Temp. Treat. Dist. & 3+ Years Teammates)			
CTE (Post) \times Teammate	7.22* (4.05)	4.62* (2.65)	3.81* (2.29)
Pre-Treatment Mean	10.45	22.27	28.29
% Change	0.69	0.21	0.13
Observations	39,231	68,256	73,035

Ordinary Least Squares (OLS) is used to estimate each model, which includes stack-by-player and stack-by-year fixed-effects. The dependent variable *Exit* is a binary estimate multiplied by one hundred for presentational simplicity. The mean of the dependent variable is calculated from pre-treatment observations of players in the treated group. *Clustered (Stack-by-Player) standard errors in parentheses. Significance Codes: ***: 0.01, **: 0.05, *: 0.1.*

Table 5 examines heterogeneous peer effects of the impact of the CTE death of a former teammate on the probability of exiting the profession by the length of time between when individuals last played together for a team and the date of their eventual death. These treated individuals are stratified into periods of two, four, and six years, where each subgroup is contained in the larger year bin. Relative to a group of characteristically similar athletes prior to treatment, treated athletes who were last teammates with someone who died two or fewer years ago saw a 3.14 percentage point increase in the likelihood of exiting. Relative to a lower pre-treatment retirement retired and each was on an active-roster.

rate, this represents a large 30% % increase. Model two (three) shows that this effect size decays when including individuals that shared a team with at least four (six) years prior to their teammate's death to 1.78 (1.91) percentage points. Relative to the pre-treatment mean, this estimate reflects an 8% (7%) increase in the probability of retiring.

Panel B of table 5 jointly examines the interactions of temporal proximity and familiarity with peers on the responsiveness of their teammates' labor supply. To test this interaction, the treated groups from panel A are further stratified by individuals treated for at least two years across each model in this panel. The results reveal interactions that are greater than the sum of their parts. For instance, the estimates of model one show that athletes who were teammates with a former teammate who died with CTE for three or more years and played for a team with them at least two years before their death are 7.22 percentage points more likely to retire than characteristically similar non-treated players in the same years. Relative to the pre-treatment mean, this represents a 69% increase. This effect size is meaningfully larger than the sum of the 30% effect of proximity of treatment seen in model one of panel A in Table 5 and the 19% effect of familiarity seen in model three of panel A in Table 4. The addition of individuals treated four (six) years ago or fewer reduces the effect sizes to 4.62 (3.81) percentage points which represent a 21% (13%) increases relative to the pre-treatment mean.

To test whether the treatment effects reported thus far can more plausibly be attributed to factors such as the increased salience of risk or any other range of emotional trials which could be experienced following the death of a coworker, I perform a placebo analysis where CTE deaths are replaced with deaths that are unrelated to workplace safety. I chose the two most common causes of death for young athletes: car accidents and rare-genetic (typically cardiac-related) diseases.²³ The effects of this exercise can be seen in Table 6. The results from this table fail to reject the hypothesis that deaths unrelated to workplace safety differentially increase former teammates' likelihood of retiring. These findings bolster the argument that peer health shocks increase retirements through the channel of increases in the salience of risk.

7.b. Contracts

I next turn my attention to studying the subsequent employment contracts of treated athletes who remain playing risky professional sports after the CTE death of a former teammate. Economic theory posits that if individuals perceive types of work as relatively less desirable, then workers will exit this profession or workplace until wages rise to compensate for the undesirability of the job. Alternatively, this theory suggests that workers in more hazardous jobs who are willing to continue working do so because they are able to receive greater compensation which help to

²³Note to Josh: I need a reference to an appendix table here with these names.

Table 6. Peer Deaths Unrelated to Workplace Safety & Retirement: A Placebo Test

Dependent Variable:	$\mathbb{1}(\text{Exit}) \times 100$	
Model:	(1)	(2)
<i>Variables</i>		
Car Accident (Post) \times Teammate	-0.01 (0.01)	
Disease (Post) \times Teammate		0.00 (0.01)
Mean of Dep.	0.19	0.19
% Change	-0.06	0.00
Observations	95,533	70,891

Each model includes Stack-by-Player and Stack-by-Year fixed-effects. *Clustered (Stack-by-Player) standard-errors in parentheses. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.*

offset the utility loss from have to face the workplace risks which have increased in salience.

In a market where wages can freely and rapidly adjust to the changes in preferences of its workers, athletes' voluntary exits from the profession should occur only in settings where the marginal value of the labor of the worker (and thus in the wages offered from the firm) fall below the worker's reservation wage.²⁴ However, in this setting, worker compensation is very heavily regulated.²⁵ These binding price ceilings mute variation in worker compensation relative to what would be expected in less regulated markets, which, in turn, increases the rate at which workers exit the profession.

To study the impact of peer-health-shock-induced increases in risk perception on the details of workers' subsequent employment contracts, I estimate a series of difference-in-differences models in Table 7. These models most notably differ from those in the previous subsection in their use of dependent variables. These new measures include total compensation, guaranteed compensation, salary, and the length of time individuals are contracted with a team. Another important change in these models is the switch from the usage of balanced data to unbalanced data, which can impact the interpretation of some coefficients if these measures capture realized compensation rather than what is enumerated in the contract. These measures of compensation will equal one another if a player is not released by the team before the end of their contract. Finally, I subset this data to only include observations in the contracts that individuals sign immediately before and

²⁴An involuntary exit refers to situations in which workers are cut from team rosters when the marginal cost of their employment is greater than the marginal benefit.

²⁵See the Setting section (Section 3) of this paper for more information on how wage controls differ between the NFL and NHL.

(if applicable) after treatment. Reducing the sample size by this amount helps to isolate treatment effects more accurately by reducing variation from contracts that are signed much earlier or later in one's career.

Each model in 7 is estimated using Poisson regression, whose coefficients are interpreted as the log of the expected count ratio. These coefficients can be closely approximated for small changes as percentage changes in the outcome. Furthermore, I condition the treatment effects in models one, two, and three on the length of the contract. This approach ensures that the treatment effects are more directly attributable to changes in the outcome variable itself rather than being influenced by variations in contract length, as would happen if the estimates were annualized by dividing by the contract duration. The results from this table demonstrate that conditional upon not exiting, treated athletes exchange reductions in annual salary for increased signing bonuses and shorter contracts. On the net, this moderately increases total compensation, albeit statistically insignificant.

Table 7. The Effect of the CTE Death of a Former Teammate on Subsequent Contract Details

Dependent Variables: Model:	Total (1)	Guaranteed (2)	Salary (3)	Length (4)
Panel A (Full Sample)				
CTE (Post) × Teammate × New Ct.	0.07 (0.07)	0.29*** (0.07)	-0.28*** (0.06)	-0.07* (0.04)
Length	0.37*** (0.01)	0.23*** (0.01)	0.13*** (0.01)	
Pre-Treatment Mean	13.84	1.17	1.76	3.84
Change	1.03	0.34	-0.49	-0.29
Observations	77,987	61,207	77,945	78,652
Panel B (Balanced Sample)				
CTE (Post) × Teammate × New Ct.	0.02 (0.07)	0.22*** (0.07)	-0.28*** (0.06)	-0.07* (0.04)
Length	0.37*** (0.01)	0.23*** (0.01)	0.13*** (0.01)	
Pre-Treatment Mean	14.38	1.21	1.86	4.03
Change	0.28	0.26	-0.52	-0.29
Observations	52,912	44,595	52,909	53,271

Poisson regression is used to estimate each model. Each model includes Stack-by-Player and Stack-by-Year fixed-effects. *Clustered (Stack-by-Player) standard-errors in parentheses.* Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

Model one in panel A of Table 7 measures the degree to which total compensation changes in the treated group relative to the control group before and after treatment. This variable reflects

that which is enumerated in the contract, and thus, treatment effect estimates of this variable are not influenced by the amount of compensation that the player ultimately receives. The coefficient reveals that treated players' total compensation rose approximately 7% relative to a control group after treatment. This represents an increase of approximately \$1.03 million. While \$1 million represents a non-trivial increase in compensation, this estimate is imprecisely estimated and masks more interesting variations in the specifics of treated players' contracts.

Model two in panel A of Table 7 measures the amount of additional guaranteed compensation that players receive due to the increased salience of workplace safety. The coefficient reveals that treated players' signing bonuses increased significantly by approximately 30 percent. This substantial increase in signing bonuses, relative to the pre-treatment mean, represents an increase in total compensation of \$340k. This boost in financial security is a direct result of the increased focus on workplace safety. Similar to total compensation in model one, guaranteed compensation is owed to workers regardless of whether they are still employed by the team in the future. Thus, the estimates from this model are not conditional upon the continued employment of the worker beyond any point after which they sign the contract. This contrasts with estimates from model three of this table which measures how total salary changes due to treatment. The coefficient of interest from this model reveals that the total amount of salary earned by players in their contracts signed after the CTE death of a former teammate is approximately 30% lower than expected. In order for the findings from models one, two and three to be simultaneously true, it must be the case that treated players are disproportionately likely to exit the profession before the end of the contract signed after treatment.

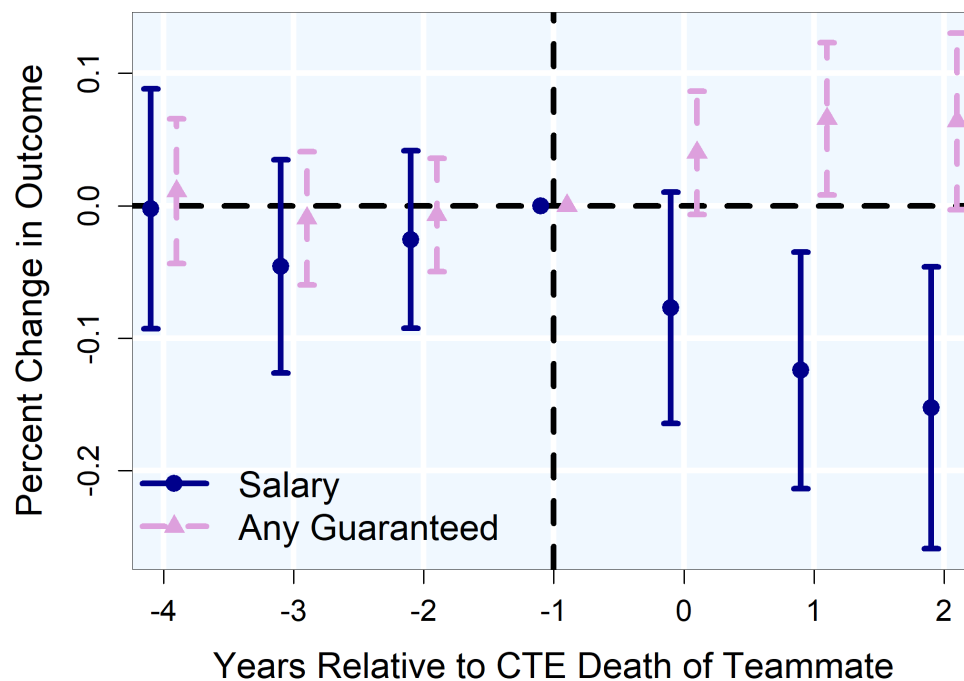
Finally, model four in panel A of Table 7 reveals that treated workers sign contracts that are approximately 7% shorter than individuals in the control group after treatment. Relative to the pre-treatment mean, this corresponds to a reduction in contract length of approximately one-third of a year. The results from these models in panel A present a clear story of risk mitigation strategies on the part of athletes with a greater salience of risk after the CTE death of a former teammate – they sign shorter contracts, receive large increases in their signing bonus, and then exit the profession earlier than individuals in the control group while forgoing less salary for doing so.

In order to test whether the results from panel A of Table 7 are driven by endogenous selection of workers who remain in the profession, I restrict both the treated and control group only to include workers who sign contracts at least once before and after treatment. Though this significantly reduces the sample sizes available for estimation, the magnitude and statistical significance of the estimated treatment effects remain remarkably consistent across panels. The only estimated effect that meaningfully reduces is that for total compensation, which falls from

7% to 2% while remaining statistically insignificant.

As is commonly known, the major identifying assumption of difference-in-differences models is that, absent treatment, the outcomes of treated and control groups would continue to trending parallel to one another. Thus, I test for the plausibility of this assumption via an event study analysis of two contract-related outcome variables – salary and a binary indicator for whether individuals have any amount of guaranteed compensation within their contract. Limiting observations to individual’s employment contracts before and, if applicable, after treatment, Figure 8 that the outcomes between treated and control players were trending parallel to one another prior to treatment. These outcomes diverge after treatment with the probability of treated players having any amount of their compensation guaranteed increasing by approximately 5% and their total earned salary decreasing approximately 10%.

Figure 8. Event Study of the Effect of the CTE Death of a Former Teammate on Subsequent Contract Details



I next separately estimate contract-related treatment effects by sport in Table 8. Panel A of this table estimates effects for hockey players using the same models from Table 7. Each treatment coefficient in this panel is both statistically insignificant and less than ten percent in absolute magnitude, suggesting that treated individuals were unable to meaningfully alter the terms of their subsequent employment contracts in order to satisfactorily internalize the updated salience of the risks of continued workplace participation.

Table 8. The Effect of the CTE Death of a Former Teammate on Subsequent Contract Details (by Sport)

Dependent Variables: Model:	Total (1)	Guaranteed (2)	Salary (3)	Length (4)
Panel A (Hockey)				
CTE (Post) × Teammate × New Ct.	-0.01 (0.09)	-0.08 (0.42)	-0.04 (0.06)	0.08 (0.09)
Length	0.28*** (0.02)	0.40*** (0.07)	0.10*** (0.01)	
Pre-Treatment Mean	16.84	0.15	2.98	4.23
Change	-0.21	-0.01	-0.12	0.33
Observations	10,980	4,093	10,972	11,278
Panel B (Football)				
CTE (Post) × Teammate × New Ct.	0.13 (0.08)	0.29*** (0.07)	-0.38*** (0.08)	-0.14*** (0.04)
Length	0.39*** (0.01)	0.22*** (0.01)	0.14*** (0.01)	
Pre-Treatment Mean	13.42	1.32	1.59	3.78
Change	1.8	0.38	-0.61	-0.55
Observations	67,007	57,114	66,973	67,374

Poisson regression is used to estimate each model. Each model includes Stack-by-Player and Stack-by-Year fixed-effects. *Clustered (Stack-by-Player) standard-errors in parentheses.* Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

The null results from panel A of 8 are potentially unsurprising for two reasons. Most notably, in exchange for more player-friendly concessions such as longer, mostly-guaranteed contracts relative to other professional sports leagues, the salary cap is far more binding in hockey.²⁶ This cap strongly limits player flexibility in contract negotiations due to these caps which restrict annual total team wage expenditures. Second, though only six players on the ice at any one time per team in hockey, the high frequency with which hockey players rotate throughout the game means that there is less ability for any one individual to determine the outcome matches relative to other sports. This limits superstar effects within the sport which, in turn, makes the terms of contracts more uniform.²⁷

The lack of significant results from panel A of 8 contrast strongly with those of panel B which analyze treatment effects for football players. Consistent with previous findings, model one of this panel estimates statistically insignificant effects of treatment on total compensation. However, the magnitude is meaningfully different with an estimate of a 13% increase. Relative to the pre-treatment mean, this represents a \$1.8 million increase. Similar to what was seen in table 7, salaries and contract lengths decrease approximately 40% and 14% (\$610 thousand and 0.55 years) while guaranteed compensation increases 30% (\$380k).

Given that the binding nature of salary caps reduces variation in total compensation needed to estimate compensating wage differentials in model one of Tables 8 and 7, I turn my attention to alternative identification strategy which leverages quasi-random variation in the amount of salary owed to athletes at the time of the CTE death of a teammate.

An analysis of the impact of opportunity costs on player retirement decisions can be seen in Table 9. Model one and two of this table estimate these effects on the full sample, finding that significant impacts of both treatment, opportunity costs and their interaction. Specifically, estimates from model one reveal that, holding all else equal, treatment increases the probability of exit by approximately 3.6 percentage points while a one percent increase in the amount of money individuals would forgo upon retirement decreases this probability by approximately 0.5 percentage points. The coefficient of interest in this table which interacts these two variables reveals treated workers' labor supply decisions are highly sensitive to opportunity costs. The coefficient for this interaction reveals that treated workers see an additional 0.37 percentage increase in the probability of retiring for every one percentage point increase in their opportunity cost.

Model two of Table 9 extends the findings of model one by adding two key control variables:

²⁶See section 3 (Setting) for a more lengthy discussion of the NHL salary cap.

²⁷See Figure 2a for a visualization of non-standardized differences productivity in across hockey and football. The modal player in the hockey is far more likely to have no impact on the game, while the distribution of football player's productivity has a very long right-tail, suggesting a greater number of superstars.

Table 9. Peer Work-Related Deaths & Retirement: Heterogeneity by Opportunity Cost

Dependent Variable:	$\mathbb{1}(\text{Exit}) \times 100$			
Sample:	Full		Hockey	Football
Model:	(1)	(2)	(3)	(4)
<i>Variables</i>				
CTE (Post) \times Teammate	3.59** (1.45)	2.48* (1.46)	1.53 (1.41)	4.08** (1.88)
$\sinh^{-1}(\text{Opp. Cost})$	-0.48*** (0.01)	-0.06*** (0.01)	-0.09*** (0.01)	-0.02 (0.02)
CTE (Post) \times Teammate $\times \sinh^{-1}(\text{Opp. Cost})$	-0.37*** (0.09)	-0.32*** (0.09)	-0.06 (0.08)	-0.52*** (0.12)
$\sinh^{-1}(\text{Total})$		-1.93*** (0.13)	0.95*** (0.16)	-2.92*** (0.18)
Length		-2.83*** (0.10)	-0.66*** (0.08)	-3.06*** (0.13)
Observations	131,424	131,424	18,679	112,745

Ordinary Least Squares (OLS) is used to estimate each model. Each model includes Stack-by-Player and Stack-by-Year fixed-effects. *Clustered (Stack-by-Player) standard-errors in parentheses. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.*

the total amount of compensation agreed to on the contract and the number of years it covers. These control variables help to isolate the effect of the opportunity cost on retirement by comparing individuals with similar contract details but only differ in the amount of money remaining on their contract at the time of treatment. While adding these control variables unsurprisingly reduces the magnitude of the coefficient for opportunity cost, it does little to change the effect size of its interaction of treatment.

Models three and four of Table 9 extend the estimating equation used in model two to separately present the differential impacts of opportunity costs on retirement for athletes in hockey and football. Model three finds little evidence that treated players with larger amounts of money remaining on their contract at the time of treatment in hockey were differentially more likely to exit after the CTE death of a former teammate. This is consistent with the knowledge that in hockey, players' remaining yet-paid salaries are far more likely to be received following their retirement due to the terms of their collective bargaining agreement. This contrasts sharply with football, whose athletes are far less likely to receive their yet-paid salaries upon retiring. Estimates from model four reveal that a one percent increase in treated football players' opportunity cost is estimated to reduce their probability of exiting by 0.5 percentage points relative to non-treated players with similar contracts.

To conclude, I utilize the predicted values from model two of Table 9 to estimate the dollar amount that treated individuals would have to receive to be indifferent between retirement and

remaining within the workforce. This regression model provides me with a continuous measure of the predicted probability of exit from the profession based on the length of their contract, the total amount of compensation specified within it, their opportunity cost and their treatment status. I present the results from this exercise in Figure 9.

Figure 9. Opportunity Costs and the Probability of Exit

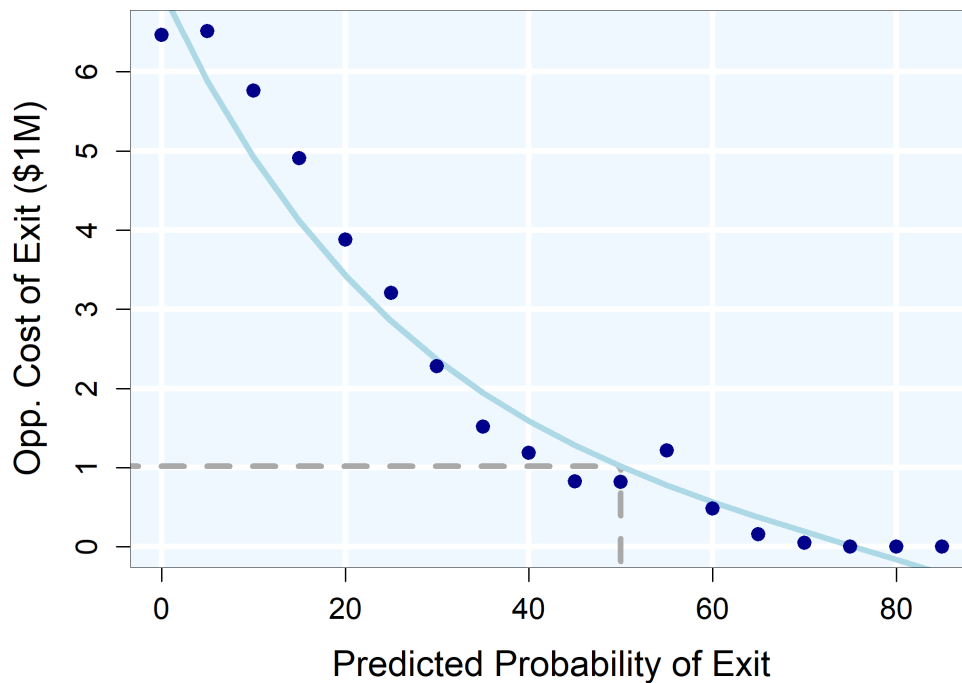


Figure 9 displays the relationship between opportunity cost and the predicted exit probability. The estimates are aggregated into bins, five percentage points in width for visual simplicity. The indifference point on the figure can be seen at the intersection of the line of best fit for these points (seen as a downward-sloping light blue shade in the figure), and the predicted probability of exit value is equal to 50. This intersects the y-axis at a value approximately equal to \$1 million.²⁸ However, reading left-to-right across this entire figure reveals that, on average, athletes would need to be compensated an additional \$6 million annually if teams wanted to know the minimum amount that they would have to pay and be very confident in preventing their workers from exiting.

While the downward-sloping nature of this curve matches theoretical priors, its convexity is potentially surprising. If individuals receive diminishing marginal utility from income, then one would expect that the estimates from Figure 9 would be concave. The intuition behind this

²⁸Interestingly, this estimate almost perfectly equals the compensating wage differential estimate from model one in panel A of Table 7.

reasoning is that moving from zero additional dollars of income to one million additional dollars should increase one's probability of remaining in the profession by a greater amount than moving from one million dollars of income to two million.

On the other hand, the convexity of the curve could be a mechanical artifact of the y-axis since opportunity costs of exit cannot fall below zero, causing the curve to steepen as it approaches this lower bound. Additionally, athletes may perceive risks in a nonlinear fashion, requiring significantly more compensation as the perceived health risks of remaining in the profession grow after a peer-health shock. This heightened risk perception could lead to an exponential increase in the required compensation to deter exit, explaining the convex shape of the relationship.

8 Conclusion

The concept of equalizing differentials, first noted by economists as early as Adam Smith, is foundational to economic theory (Smith 1776). Its widespread influence can be attributed, at least in part, to the numerous fields that rely on this theoretical framework. These range from the adjustment of wage compensation to account for employment uncertainty and health risks (Viscusi 1993; Averett, Bodenhorn, and Staisiunas 2005; Gertler, Shah, and Bertozzi 2005) to the impact of geographic amenities and firm agglomeration on housing markets through migration channels (Roback 1982; Glaeser, Kolko, and Saiz 2001). Although the setting examined in this paper shares broad conceptual overlap with the aforementioned research, the results presented here both align with and diverge from the predictions of standard economic theory in nuanced ways.

In examining the effects of informational health shocks on labor supply, I find that former peers exhibit notably stronger responses than expected. This is surprising for at least two reasons. First, professional athletes face intense competition for their jobs and have invested heavily in sport-specific human-capital to secure this employment. These skills likely have limited transferability to the external labor market which strongly limits their ability to credibly threaten to voluntarily exit the profession. The exogeneity of exposure to a former teammate who dies of CTE rules out concerns that the increase in exit among former teammates which I document is driven by differences in expected wages that one could earn outside of professional athletics. Second, even with extensive media coverage of CTE diagnoses and concussions affecting both "treated" and "control" groups, the pronounced effects observed in both suggest that the impact of peer health shocks on labor supply may be even more substantial than observed.

There might be a number of reasons for this underestimation. First, team owners have purposefully obfuscated fatality rates. The calculation of VSL requires work-place fatality rates that are easily observable to athletes. Second, CTE currently is only diagnoseable post-mortem. This

implies that workers attempting to draw inference on the probability are working with imperfect information. I need to talk about monoposony power here. Even though there is a relatively free-market for labor in the sense that there is a bidding process, wages are still highly suppressed by the strictly binding nature of the salary cap. However, in professional sports, particularly in the NHL and NFL, salary caps and other wage restrictions limit the ability of wages to freely adjust to the increasing risk. The findings show that treated athletes are more likely to exit the profession, with wage increases insufficient to fully compensate for the increased salience of health risks.

This paper also draws upon and contributes to insights from behavioral economics, particularly with respect to biases regarding one's personal health status and risks to it. It is well documented that individuals suffer from optimistic biases regarding personal risks (Weinstein 1989). This bias persists even in settings where information is low cost and errors are likely to have large, negative ramifications to finances and health (Bhattacharya, Goldman, and Sood 2009; Oster, Shoulson, and Dorsey 2013; Golman, Hagmann, and Loewenstein 2017). The prevalence of these behavioral biases has meaningful implications regarding both health and wage inequalities for workers as well as for policy regarding the optimal provision of worker's compensation (Viscusi 1980).

The analysis utilizes difference-in-differences models to examine various components of player contracts, such as total compensation, guaranteed compensation, salary, and contract length. Treated athletes tend to sign shorter contracts, receive larger signing bonuses, but also have lower salaries. These results suggest that while players are receiving increased compensation upfront in the form of guaranteed payments, they may still exit earlier, indicating that the contract structure cannot fully mitigate the risks associated with staying in the profession after a peer-health shock.

More broadly, these findings contribute to the literature on peer effects and labor economics by highlighting the limited ability of wage adjustments to retain workers in risky environments. Even when workers receive higher guaranteed payments, the increased salience of risk can lead to earlier exits, reflecting the difficulty of fully internalizing health risks through compensation alone. This aligns with broader labor market trends where workers in hazardous jobs may need significant compensating differentials to stay, especially after witnessing the consequences of workplace hazards firsthand.

Future research could explore how variations in contract structures across different sports influence athletes' decisions to continue working in high-risk environments. Additionally, examining how changes in collective bargaining agreements or new safety measures impact the trade-off between compensation and risk perception could provide valuable insights into how labor markets respond to evolving workplace risks.

As countries progress economically, there is an increasing emphasis placed by governments and policymakers on strengthening worker protections to ensure that labor markets evolve in ways that safeguard worker welfare and promote equitable economic outcomes. Such considerations become even more crucial in the context of non-competitive labor markets where employers wield significant control over employment conditions. The impact of this dynamic is acutely observable in industries where labor supply decisions are heavily influenced by personal health concerns.

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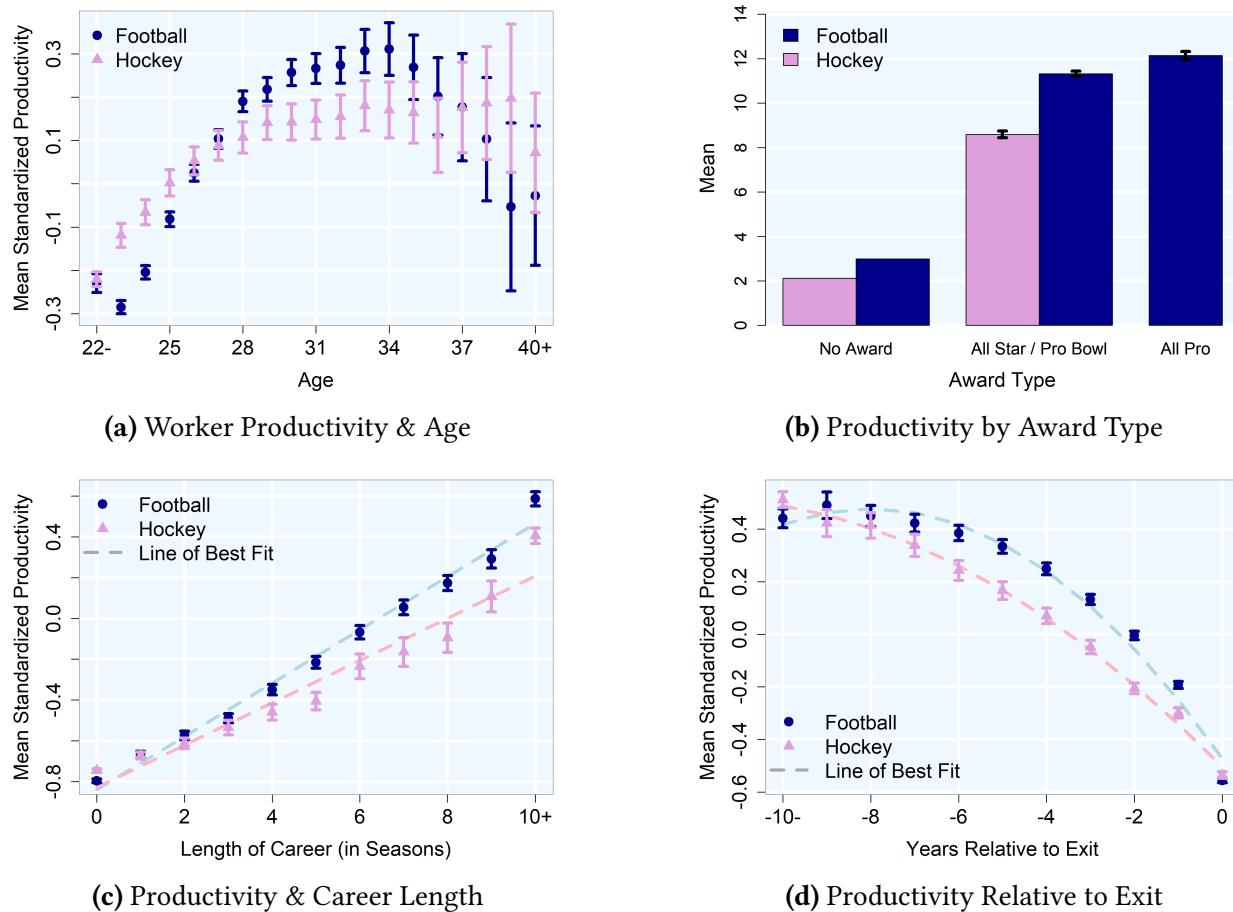
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A1 Worker Productivity

Figure A1. Trends and Distributions in Worker Productivity Across Sports



A2 CTE & Cause of Death

A2.a. American Football

Aside from the discovery of CTE in two former NFL athletes, the strength of the common perception linking the disease and American football is not accidental. Though the commonly-held concerns regarding the long-term consequences of repeated head trauma often far pre-date the professionalization of modern sports, the NFL was the largest sport at the time to have to publicly address concerns of safety for its workforce directly. In response to rising concerns about the prevalence of head injuries, the NFL committed to a strategy of obfuscation, establishing pseudoscientific organizations to serve as the public-facing propaganda arms for the League in their uphill battle to change the public perception of the safety for their sport amid rapidly mounting evidence of the ramifications repeated head trauma (Fainaru-Wada and Fainaru 2013). It took

more than a decade after the publication of the findings CTE in a former professional football player for the NFL to publicly admit to the link between the sport and disease (Breech 2016).

In 2002, decorated and fan-beloved offensive lineman **Mike Webster** died of heart failure at the age of 50. He played professionally for 17 seasons and won 4 Super Bowls. Renowned for his toughness, he notably went over six seasons without missing a single offensive play – earning him the nickname “Iron Mike.” However, the negative health impacts of this longevity in the game became quickly evident. He likened the impact of his career to experiencing 25,000 automobile crashes (Fainaru-Wada and Fainaru 2014). He was declared totally and permanently disabled by a physician before the end of his playing career. His estate later sued the administrative body of the NFL’s retirement plans for discounting his physician’s assessment and denying his repeated claims (“Jani v. Bert bell/pete rozelle NFL player retirement plan, no. 05-2386” 2006).

After his retirement, Mike was diagnosed with amnesia and dementia and suffered from various physical ailments that were severe enough to require the use of a conductive energy device (taser) to shock himself to sleep (Laskas 2015). Even former teammates, coaches, and team owners covered the total share of his housing expenses, not enough to prevent him from experiencing long stretches of homelessness. The severity of the deterioration of his health, coupled with the number of hits to the head he received throughout his professional occupation, led to an inquiry into the physical state of his brain post-mortem. This led to Mike Webster becoming the first former professional American football player to be diagnosed with CTE.

From 2004 to 2009, there were numerous other high-profile cases of early-life deaths of former NFL players who were later confirmed to have had CTE. In each instance, similar to Mike Webster, evidence of significant declines in physical and mental health is present – often resulting in the individuals dying violently. In each instance, there was substantial news coverage of the events and speculation on the causes of their death.

NFL veteran **Justin Strzelczyk** was forced into retirement at the end of the 1998 season after being placed on injured reserve for receiving an injury during a bar fight. In the following four years, he was twice arrested for a DUI and for carrying a firearm without a permit, and he died in 2004 in a car crash while driving against the flow of traffic to flee the police. Notably, toxicity tests revealed he was sober at the time of the accident (Schwarz 2007).

Terry Long retired from a long and successful career in the NFL in 1991 following a failed drug test for a performance-enhancing steroid and for brandishing a firearm at a coach while at work. One decade later, he was found guilty of arson and fraud and was facing foreclosure on his home. In 2005, he died by suicide after drinking a gallon of antifreeze (Finder 2005).

Renowned hard hitting safety **Andre Waters** retired from professional football as an athlete

in 1995 after having played for twelve seasons. After serving as a football coach for the second half of his career, Waters died by suicide from a self-inflicted gun shot wound with many linking his depression to the brain damage he received from his playing career (Schwarz 2007).

Tom McHale and **Shane Dronnett** also both had long careers in the NFL. The former retired from professional football in 1995 and died via a drug overdose in 2008 (Schwarz 2009). The latter retired in 2002, but quickly developed paranoia that often presented in unpredictable fits of rage. The deterioration of Dronnett's emotional and mental health did not improve after removing a benign brain tumor. He died in 2009 of a self-inflicted gunshot wound after a domestic fight where he brandished a gun and threatened to kill his wife (Smith 2011).

However, despite the alarming rise in gruesome, early-life deaths that were being increasingly linked to CTE at the time, the NFL was first hostile and later resistant to accept the fact its product was seriously endangering the longevity of its workers.

There were two important characteristics about each of the individuals above who died (and were later diagnosed with CTE) prior to 2010. First, each of them was *former* player who had career lengths that were significantly longer than the modal NFL player. Second, each specialized in positions that required far greater amounts of physical contact than others. Combined, these factors made them obvious for candidates of developing CTE as they were exposed to a far greater cumulative risk of exposure to head injury.

This changed with the death of *Chris Henry*. Henry died mid-season in 2009 while only aged 28 – becoming the first player to be diagnosed with CTE while on an NFL roster. In addition to his young age, his death directly contradicted the contemporaneous narrative that developing CTE would be rare as he played a position that had significantly lower rates of head impacts and had never been diagnosed with a concussion in either his collegiate or professional career (Schwarz 2010).

A troubled athlete with talent that was difficult to deny, Henry was often penalized on the field for his conduct and was arrested five separate times in his professional playing career for offenses ranging from driving under the influence to multiple instances of aggravated and sexual assault. He died after being ejected from a moving automobile that he was attempting to enter, which was being driven by the mother of his children, who was fleeing from him after a domestic dispute (Martinez 2009).

A2.b. Mortality Risk of American Football & CTE

While many now view it as evident that extended participation in American Football can carry tremendous health risks, only *recently* has there been convincing empirical evidence in favor of

this hypothesis. The majority of empirical evidence relied on matching methods or controlling for observable differences between NFL players and the general population to argue that the benefits of income and elite physical fitness outweighed the health risks of American Football. This narrative began to change in the 2010s. For instance, Koning et al. (2014) confirm the work of earlier literature finding significant advantages in footballer’s longevity, but show that this advantage diminishes as athletes play in more games over their careers.

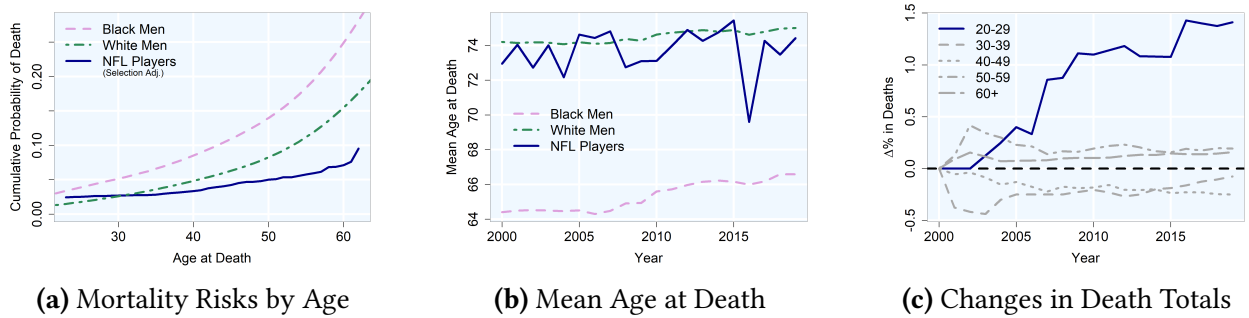
The narrative flipped quickly as soon as researchers began to select alternative comparison groups. Venkataramani, Gandhavadi, and Jena (2018) compared “replacement players” who were temporarily hired to play during a league-wide player strike in 1987, finding higher mortality risks among career NFL players. Nguyen et al. (2019) compared professional baseball players and found significantly higher mortality rates from all causes, with neurodegenerative diseases being particularly pronounced. LeClair et al. (2022) compared elite high-school players and found that professionals are nearly 2.5 times more likely to develop CTE. Daneshvar et al. (2021) compared American males and found four times greater incidence and mortality rates of ALS among NFL players.

To illustrate the mortality risks associated with participation in American football, I gathered data on the timing of deaths of former athletes who played in the NFL. This data is collected from pro-football-reference.com, which contains information on the date of birth and, when applicable, death of all current and former players. Thus, I observe the date of birth and death of all NFL players who died from 1980-2019. I contrast estimates from this data to those from the 2004 Actuarial Life Tables from the United States Social Security Association. When stratifying estimates by race for the general public, I gather data from the United States Center for Disease Control and Prevention’s (CDC) Life Tables within annual National Vital Statistics Reports. Historical estimates of average life expectancy for differential age and race cohorts are calculated using data from the CDC Wonder database. To ensure proper comparisons, I adjust all mortality estimates for black and white men by the percentage change in their life expectancy between the earliest date of data availability and the average cohort birth date of the relevant samples of NFL players.

Figure A2 highlights the mortality paradox for professional American footballers. Figure A2a presents their cumulative probability of death, contrasting this with the same outcomes for black and white men in the general public. The figure underscores the substantial longevity benefits for footballers, which significantly increase over time. By age 30, individuals who played in the NFL are approximately 2 percentage points less (equally) likely to have died than black (white) men in the general population. Thirty years later, at age 60, this gap has expanded to an 18 (8) percentage point gap between footballers and black (white) men.²⁹

²⁹I employ a conservative “selection adjustment” in this figure. This accounts for the fact that the estimation

Figure A2. Comparative Mortality Risk of Professional American Football



Despite this massive head start in longevity, Figure A2b provides evidence that footballers have no meaningful differences in the average age of death compared to white men in the general population. While the finding from Figure A2b partially reflects that a combination of increased wealth, education, and physical fitness that come with being an elite professional athlete aids in closing the racial longevity gap, Figure A2c provides evidence that this paradox in mortality risk is likely increasingly driven by deaths before the age of 40. This pattern is particularly pronounced for those under 30 – increasing by 150% from 2000 to 2019.

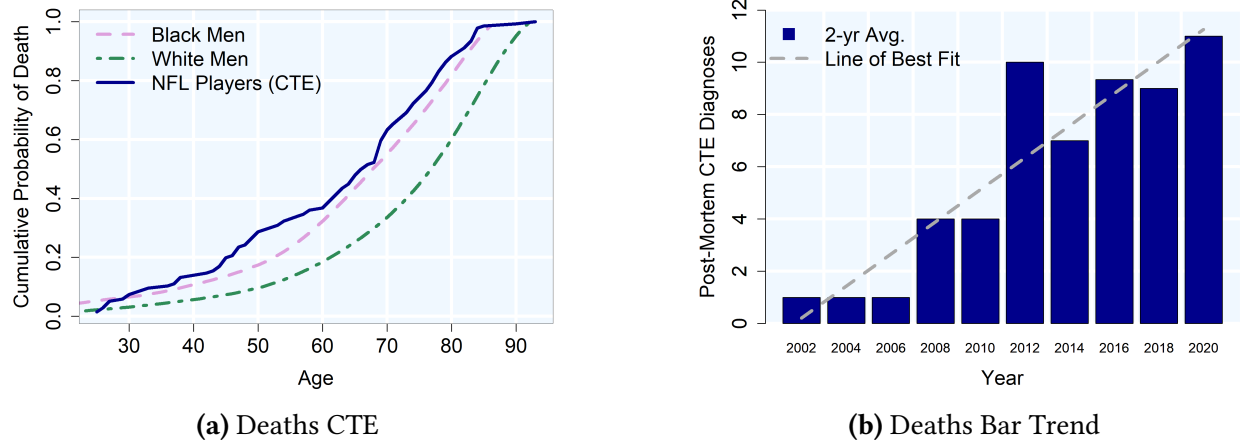
Given the rising increase in early-career deaths of footballers, I examine one of the most salient and dramatic (workplace-related) causes of death – CTE. Figure A3 shows that individuals who die with CTE die young. These individuals have significantly greater probabilities of death at every age than white and black men in the general population.

These young deaths are becoming increasingly common. Figure A3b shows that the number of confirmed cases of CTE cases for former NFL players has increased from an average of 1 per year in 2002 to 11 per year by 2019.³⁰ While we know the number of CTE cases has mechanically increased over time due to testing capacity and knowledge of the disease, it is plausible that this dramatic increase in cases reflects a rise in the actual prevalence of the disease rather than just improvements in surveillance. If so, increases in CTE could be driven by increasing participation in football at all ages (and thus increases in lifetime cumulative head impacts) as rising wages at the professional level incentivize greater parental investment in talented youths. Regardless

of mortality rates is conditional upon observation of employment within the National Football League. Given that employees often begin their careers in their early 20s, NFL players have a conditional mortality rate of zero before the age of 23. Thus, the mortality rate of NFL players at age 23 is approximately the same as that seen in the general public. Therefore, I shift these estimates by the mean of the cumulative probability of mortality for black and white men in the general public at age 23. This is a very conservative assumption, given that individuals who go on to work as professional athletes very likely have significantly lower mortality risk prior to adulthood. If so, the estimates in this figure significantly understate actual mortality differences.

³⁰Given the sporadic timing and relatively low-frequency occurrences of these diagnoses, I take the mean of the outcome in two-year intervals year to most clearly illustrate the increasing time trend.

Figure A3. Trends in CTE Mortality



of the exact mechanism driving these increases, it is clear that the health risks of continued workplace participation in the NFL during this period increased significantly.

Etc text: Only recently has evidence arisen which suggests that the health risks of American football outweigh the benefits of income and elite physical fitness. Many studies relied upon matching methods or controlling for observable differences – finding that playing in the NFL is associated with increased longevity, but that this advantage diminishes as athletes play in more games over the course of their careers (Koning et al. 2014; Owora et al. 2018).

For instance, the CTE Center, an academic research center within Boston University’s Alzheimer’s Disease Research Center, reported a CTE diagnosis rate nearly 92 percent among the 376 brains of former NFL players they had studied (“Researchers find CTE in 345 of 376 former NFL players studied” 2023). This finding contrasts starkly with the a diagnosis rate of less than 1 percent among a studies of 164 in the United States and 310 in Europe (Forrest et al. 2019; McKee et al. 2023).³¹

Mez et al. (2020) shows a dose-response relationship between American football participation and the probability of developing CTE and its severity.

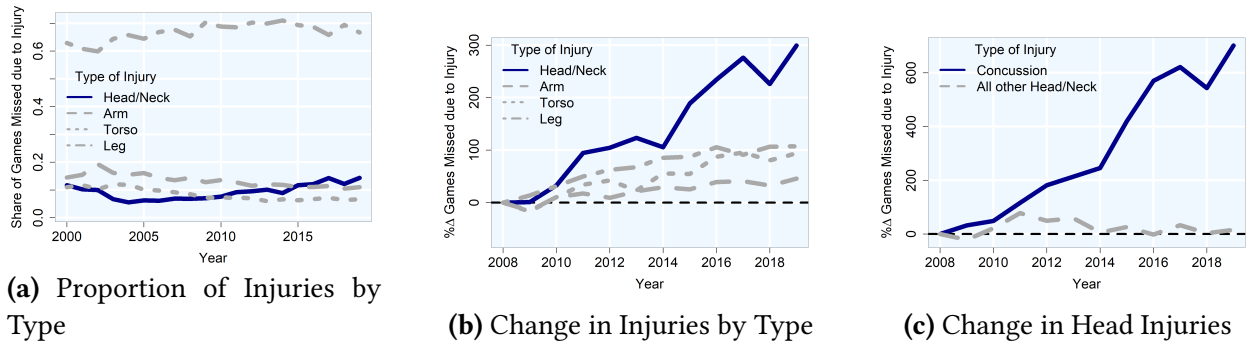
Etc: Kitchens (2015), (Viscus 1979), (Viscusi 1978)

A2.c. Injuries

[Click to Return to Appendix Table of Contents](#)

³¹They report the lone individual with CTE among the general population played collegiate football.

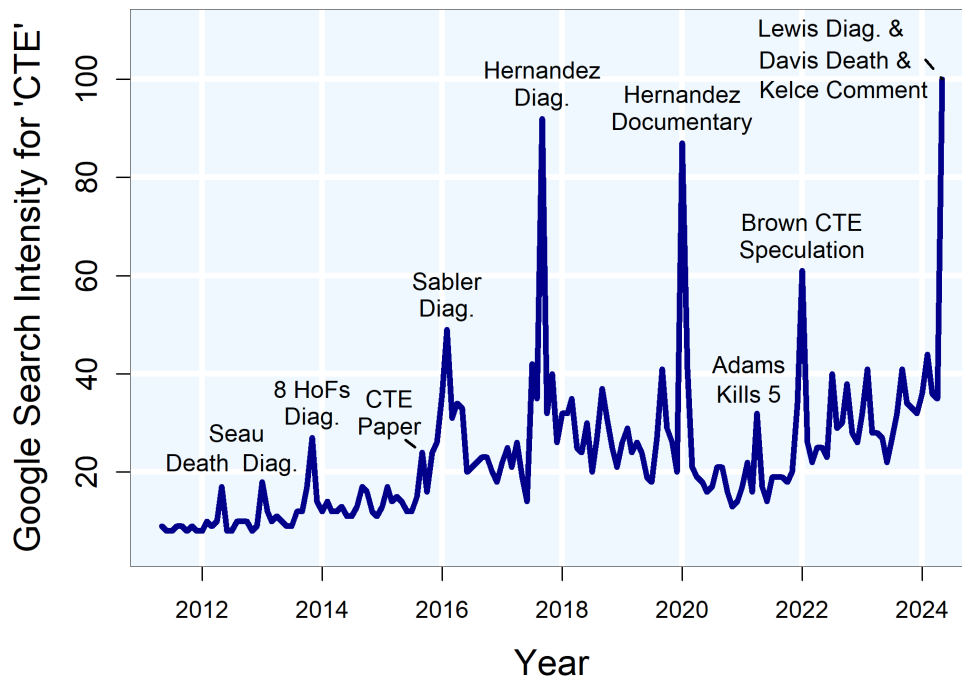
Figure A4. Injuries in the NFL over Time



A3 Relative Popularity of Sports

A3.a. Google Trends

Figure A5. Timeline of Variation in Google Search Intensity for "CTE"



I use individuals' date of death as a proxy for the timing of informational health shocks about workplace safety. One might be concerned with whether one's date of death can adequately serve as a treatment timing for changes in perceptions of safety – particularly if causes of death are seemingly unrelated to the disease or if their CTE diagnosis occurs many months or years after death. To investigate this concern, I gather data from Google Trends which allows me to observe changes in internet search intensity for topics related to the disease.

You can see the raw output from this data in Figure *NAME HERE* searches for CTE have increased exponentially over time. The first notable increase in CTE search intensity occurred in the Winter of 2009 following the violent death of former NFL player Shane Dronett and the release of Alan Schwarz's article *New Sign of Brain Damage in the NFL* in the New York Times which reported on the sixth former NFL player younger than 50 to have been diagnosed with CTE coupled with quote that "...football is clearly strongly related to the presence of this pathology" from researchers at the newly-formed Center for the Study of Traumatic Encephalopathy at Boston University. The national reputation and reach of the both New York Times and Boston University granted a sense of legitimacy to the discovery of CTE and sparked a nationwide conversation regarding the safety of football.

Each of the remaining clearly-visible spikes in search intensity clearly belong to the CTE deaths of football and hockey players. Two years later, in the Winter of 2011, Bob Probert became the first professional hockey player to be diagnosed with CTE. In May 2012, legendary football player Junior Seau died via suicide.

In order to circumvent the well-documented empirical challenges with Google Trends data, I transform the search intensity variable (which is typically bounded from zero to one hundred) into a percentage change so that earlier periods (when CTE was a relatively unknown phenomenon) receive an equal weight to later periods. I next match in an indicator variable to this data such that

There are many well documented issues with Google trends. The primary issue is that certain intensity is bounded from 0 to 100. In order to circumvent this known problem. I estimate the change in certain intensity of the term CTE from month to month this removes the gradual trend upward in the outcome variable of interest that is associated with growing awareness of the disease.

in order to determine the causal impact of a celebrity athletes death on search intensity for CTE I employ a stacked difference and different method where I compare months before and after ACT death in one year to the same months in years, where there was not a CT death. I choose an event time window of eight months in order to allow for long lags between one's death the announcement of their diagnosis.

Table A1 in the appendix shows the results of this exercise. Model one reveals that a CT death increases certain intensity for CTE by 15% model two adds the use of month and year fixed effects revealing the estimate for model. One is not sensitive to their inclusion. Models three and separate the impact of a CT Density for it between the deaths of players and the deaths of football players. Model three reveals a 10% increase whereas model four reveals a 16% increase

these findings reinforce the fact the NFL is much more popular than hockey generally. Figure A4 provides event study evidence. In the search search intensity of CTE increases significantly following the death of a professional athlete.

Table A1. The Effect of Athlete CTE Deaths on Internet Search Intensity for CTE

Dependent Variable:	Search			
Sample:	Full		Hockey	Football
Model:	(1)	(2)	(3)	(4)
CTE (Post)	0.15*** (0.05)	0.13** (0.05)	0.10** (0.05)	0.16*** (0.06)
<i>Fixed-effects</i>				
Month		✓		
Year		✓		
Observations	248	248	88	205

IID standard-errors in parentheses.

A3.b. Nielsen Ratings

[Click to Return to Appendix Table of Contents](#)

Figure A6. Event Study of the Effect of the CTE Death of a Former NFL/NFL Player on Internet Search Intensity for CTE

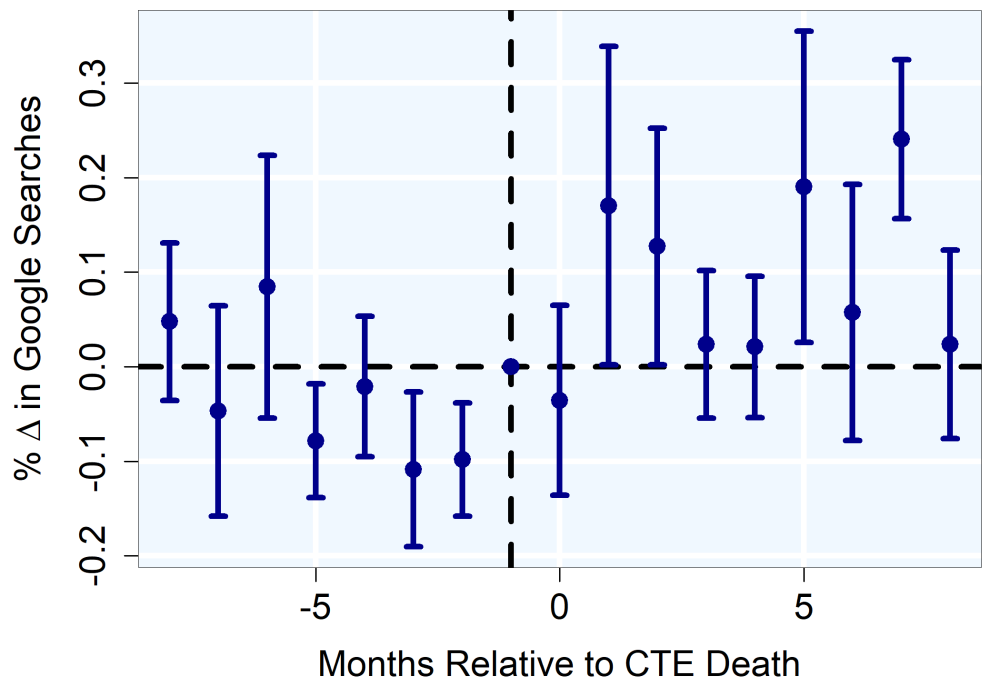
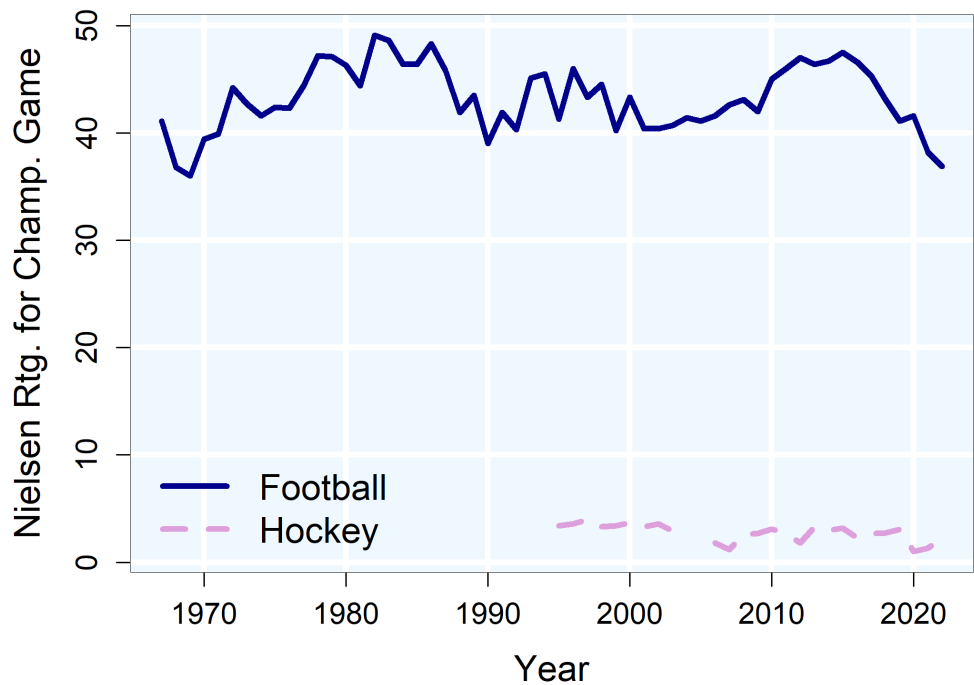


Figure A7. Nielsen Ratings for Championship Game / Series



A4 Early vs. Late Treated

In addition to differences and exposure to former teammates, an additional explanation could be that the risk of CTE was not appropriately priced into hockey relative to football. Explore the hypothesis and table by comparing the model of early versus late-treated groups. Table A2 reveals that excluding lead-treated groups from the analysis, the effect of the early CTE deaths within hockey differentially increased the probability of former teammates retiring by 37% relative to the control group. When looking at early treated cases of CTE for football players from 2004 to 2009. I find no detectable change in the probability of retiring former and non-former teammates. Footnote: This may be unsurprising because CTE has not become common knowledge at this time. When analyzing cases from 2009 to 2012, I see a positive but statistically insignificant effect of the CT form you made on the probability of retiring. This marginally increases the sample from 2009 to 2017, which marks the highest visibility CT death, which is Aaron Hernandez.

Table A2. CTE & Professional Exits: Early vs. Late Treated

Dependent Variable:	1(Exit)*100			
Sport:	Hockey	Football		
Sample:	'10-'11	'04-'09	'09-'12	'09-'17
Model:	(1)	(2)	(3)	(4)
<i>Variables</i>				
CTE (Post) × Teammate	6.87*** (2.14)	-1.79 (3.10)	2.19 (1.46)	2.83** (1.23)
<i>Effect Sizes</i>				
Pre-Treatment Mean	18.63	43.47	30.02	29.52
% Change	0.37	-0.04	0.07	0.1
<i>Fixed-effects</i>				
Stack-by-Player	✓	✓	✓	✓
Stack-by-Year	✓	✓	✓	✓
Observations	25,326	26,757	68,994	105,768

*Clustered (Stack-by-Player) standard-errors in parentheses. Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.*

A5 Matching

Figures a five and a six highlight the strong need for methods which more carefully construct counterfactual for the treated group. Instance figure A5 displays the share of remaining cohorts, which exit from the profession between the treated and control groups in hockey and in football.

On the rate of exit between these two groups does not trend parallel to one another in the pre-period. This can also be seen in figure ASX, which looks at the cumulative share of individuals who have exited within the entirety of the cohort once again between the treated group and the control group. The rate of exit between each group is not trending parallel.

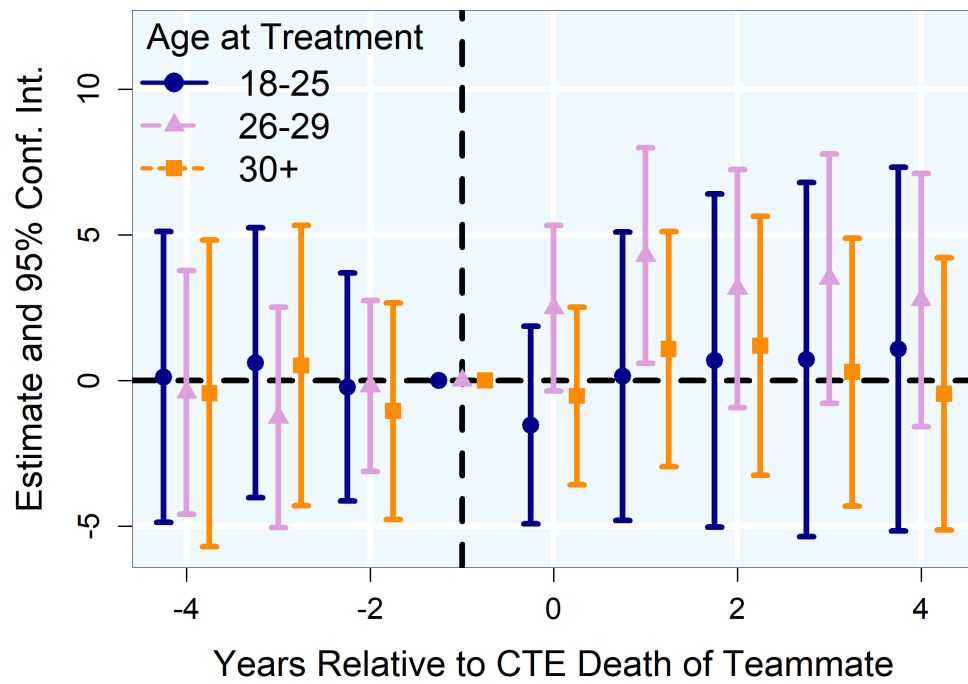
Figure a seven shows suggestive evidence why this match is potentially failing to occur. Shows differences in the pre-treatment means between two key covariates in modeling, the probability of exiting the profession: age and productivity. The upper section of figure eight⁷ reveals that on average the treated group is significantly older than the control group. After using a matching method to reduce the spasm, you can see that the effect size reduces such that the control group becomes marginally older than the treated group. In the bottom section, you can see large and statistically significant differences between the treated and control groups in the pre-treatment. Such that it treated is almost 15% more productive than the control group. After employing a matching method, this effect produces less than 5% difference in productivity and one that is statistically insignificant.

One should note that, though the differences in agent productivity between the treated and control groups in the match sample are not statistically different from one another the fact that the treated group remains both slightly younger and more productive in absolute terms than the control group bias affects towards zero younger and more productive workers. All else equal should be less likely to exit the profession for reasons unrelated to treatment.

Figure 8 reveals the effect of the matching method on the cumulative share of exits between treated and control cohorts across both sports. Both results show virtually no change in the rate of exit between treated and control cohorts prior to the CTE death of a former teammate. These outcomes begin to diverge significantly following one of these deaths suggesting that a workplace related death significantly increases the probability of exiting the profession among former teammates.

Interestingly, the shape of the response to a CT death differs between both hockey and football. In the affect on the probability of exit is immediately clear but diminishes overtime such that four years after a former teammates CT death differences in the probability of exit statistically indistinguishable from the control group. This contrast with the scene in hockey or the impact seems delayed by one year, but then sharply diverges, and the effect sizes continue to grow afterwards. It is worth noting that one potential reason for this phenomenon could be due to the upper bound that is that the convergence that we see in the football labor supply response might be due to the fact that it is rapidly approaching the upper bound in terms of the number of individuals that are capable of retiring in total.

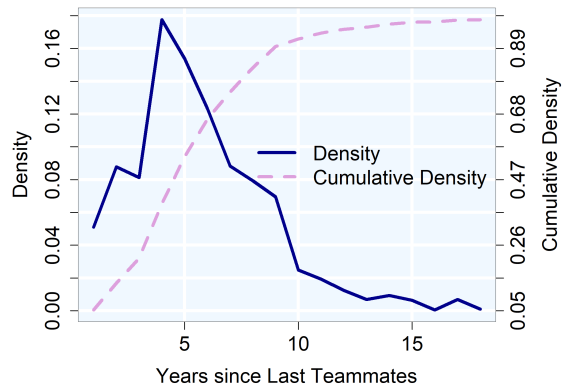
Figure A8. Event Study of the Effect of the CTE Death of a Former Teammate on Professional Exit; Stratified by Age at Time of Treatment



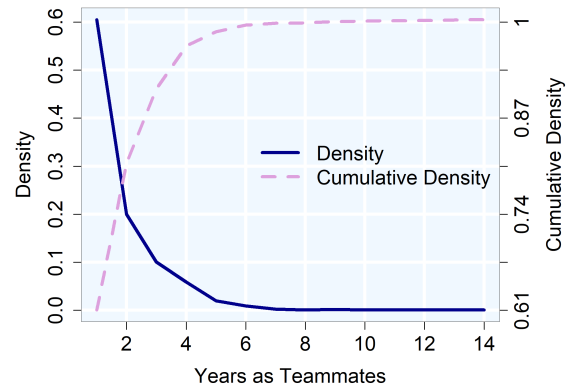
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A6 Teammate Characteristics

Figure A9. Distribution of Time as (and since) Teammates



(a) Distribution of Years since Last Teammates



(b) Distribution of Years as Teammates

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A7 Worker Productivity

Table A3. Highest Career Productivity Totals in the NFL

Ranking	Player	Position	Total AV	Years	Seasons	AV/Season
1	Tom Brady	QB	326	2000-2022	22	14.8
2	Drew Brees	QB	277	2001-2020	19	14.6
3	Peyton Manning	QB	271	1998-2015	17	15.9
4	Brett Favre	QB	259	1991-2010	19	13.6
5	Jerry Rice	WR	251	1985-2004	19	13.2
6	Fran Tarkenton	QB	233	1961-1978	17	13.7
7	Aaron Rodgers	QB	231	2005-2023	18	12.8
7	Reggie White	DE	231	1985-2000	15	15.4
9	Bruce Smith	DE	229	1985-2003	18	12.7
10	Ray Lewis	LB	224	1996-2012	16	14.0
...						
60	Aaron Donald	DT	153	2014-2023	9	17

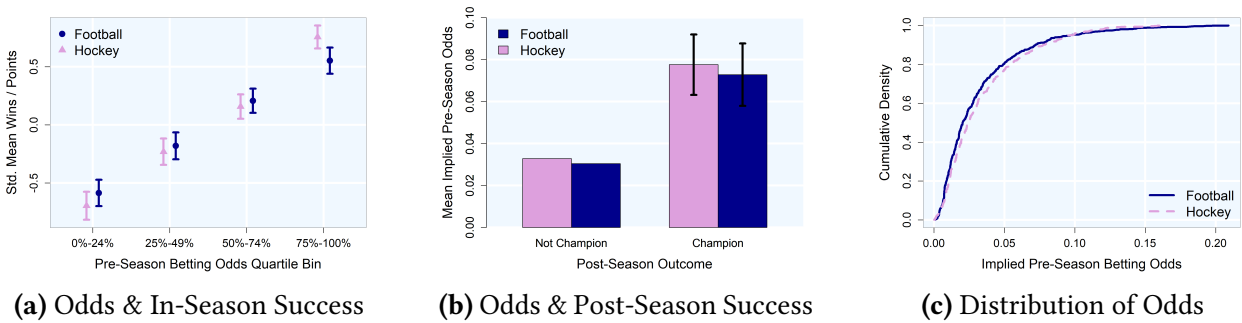
Table A4. Highest Career Productivity Totals in the NHL

Ranking	Player	Position	Total PS	Years	Seasons	Mean PS
1	Wayne Gretzky		251.01	1979-99	20	12.6
2	Ray Bourque		242.69	1979-01	22	11.0
3	Roberto Luongo		217.84	1999-19	20	10.9
4	Gordie Howe		217.11	1946-80	34	6.4
5	Jaromír Jágr		217.06	1990-18	28	7.8
6	Nicklas Lidström		211.77	1991-12	21	10.1
7	Martin Brodeur		206.97	1991-15	24	8.6
8	Alex Ovechkin		203.68	2005-24	19	10.7
9	Patrick Roy		198.34	1984-03	19	10.4
10	Al MacInnis		195.01	1981-04	23	8.5
...						
42	Bobby Orr		150.95	1966-79	13	11.6
...						
129	Connor McDavid		114.76	2015-24	9	12.8

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A8 Post-Season Expectations

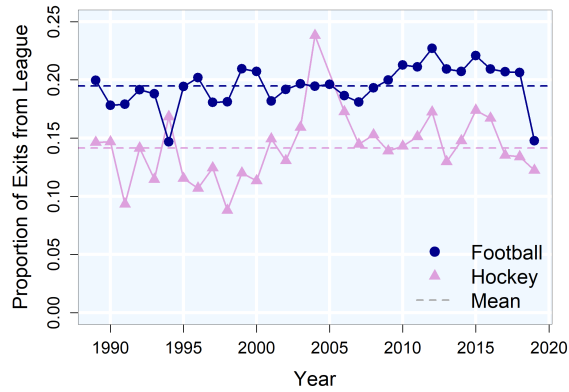
Figure A10. Trends in Betting Odds by Sport



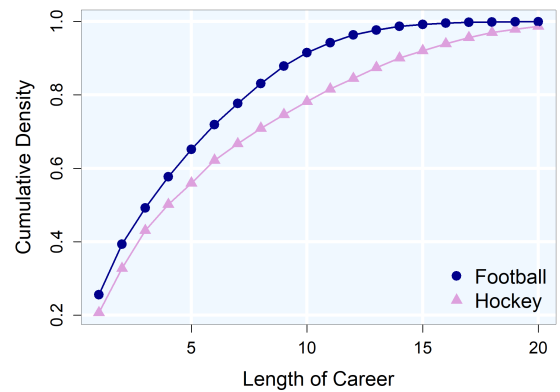
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A9 Career Longevity

Figure A11. Trends in Career Longevity



(a) Timeseries of Worker Turnover

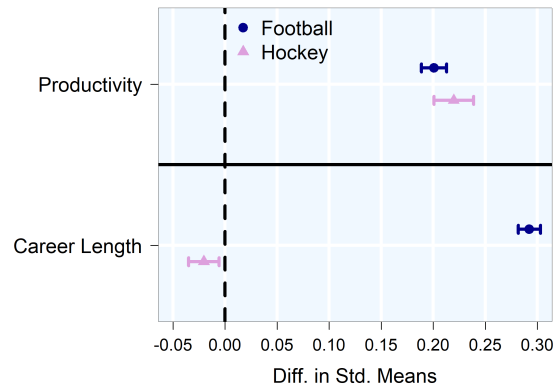


(b) Career Longevity Distribution

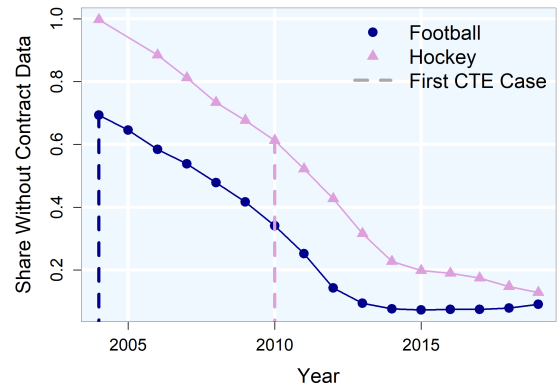
A10 Contracts

The details of the contracts are current as of Summer 2024. Extensions to the contract are counted within the original length.

Figure A12. Trends in Contract Missingness



(a) Predictors of Contract Data



(b) Share without Contract Data

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A11 Summary Statistics

Note to Josh: I need to update this with correct contract information.

Table A5. The Effect of the CTE Death of a Former Teammate on Subsequent Contract Details (OLS Models)

Dependent Variables: Model:	$\sinh^{-1}(\text{Total})$ (1)	$\sinh^{-1}(\text{Guar.})$ (2)	$\sinh^{-1}(\text{Salary})$ (3)	Length (4)
Panel A (Full Sample)				
CTE (Post) ×Teammate ×New Ct.	-0.04 (0.06)	-0.03 (0.29)	-0.17** (0.08)	-0.25* (0.14)
Length	0.49*** (0.01)	1.14*** (0.03)	0.11*** (0.01)	
Pre-Treatment Mean	13.84	1.17	1.76	3.84
Change	-0.54	-0.04	-0.31	-0.25
Observations	78,652	78,652	78,652	78,652
Panel B (Balanced Sample)				
CTE (Post) ×Teammate ×New Ct.	-0.07 (0.06)	-0.10 (0.29)	-0.19** (0.08)	-0.24* (0.14)
Length	0.49*** (0.01)	1.14*** (0.03)	0.11*** (0.01)	
Pre-Treatment Mean	14.38	1.21	1.86	4.03
Change	-1	-0.12	-0.35	-0.24
Observations	53,271	53,271	53,271	53,271

Ordinary Least Squares (OLS) is used to estimate each model. Each model includes Stack-by-Player and Stack-by-Year fixed-effects. *Clustered (Stack-by-Player) standard-errors in parentheses.* Signif. Codes: ***: 0.01, **: 0.05, *: 0.1.

No compensation is ok! Sometimes I observe players who are under contract with a team and their contract is entirely incentive pay related.

Table A6. Summary Statistics by Sport

Statistic	N	Mean	St. Dev.	Min	Max
<i>NFL</i>					
Age	91,343	26.494	3.319	20	48
Games	91,343	11.491	5.113	0	17
Started	91,343	5.788	6.287	0	17
Experience	91,343	3.490	3.261	0	26
Productivity	91,303	0	1	-2.659	6.336
Pro Bowl	91,343	0.056	0.229	0	1
All Pro	91,343	0.027	0.161	0	1
Salary (\$100k)	28,677	14.191	21.446	0	314.090
Bonus (\$100k)	28,677	13.392	55.549	0	2,649.413
Total (\$100k)	28,677	26.681	37.091	0	518.500
Length	28,677	3.086	1.507	1	12
Opportunity Cost (\$100k)	28,677	29.316	74.414	0	2,156.156
<i>NHL</i>					
Age	47,021	26.319	4.346	18	51
Games	47,111	42.557	28.250	1	84
Started	47,111	7.076	9.755	0	92
Experience	47,111	4.527	4.120	0	25
Productivity	47,111	0	1	-1.616	6.676
All Star	39,468	0.044	0.205	0	1
Salary (\$100k)	13,002	21.608	21.185	0	120
Bonus (\$100k)	13,002	3.355	32.039	0	860
Total (\$100k)	13,002	23.722	22.970	0	126
Length	13,002	3.237	2.073	1	14
Opportunity Cost (\$100k)	13,002	41.648	85.521	0	1,112.683