

**MORTALITY IN PROFESSIONAL ATHLETES: EXAMINING INCIDENCE,
PREDICTORS AND CAUSES OF DEATH**

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Abstract

Objective: The overarching purpose of this dissertation was to provide an evidence-based portrayal of i) incidence, ii) predictors and iii) causes of death in athletes from Major League Baseball, the National Basketball Association and American Basketball Association, the National Football League and the National Hockey League. More specifically, this investigation highlighted i) mortality outcome differences of athletes between and within professional sport(s), ii) potential statistical artifacts that may be empowering biases of risk of certain lifespan predictors and iii) the challenges of contextualizing historical data to answer questions with relevance in the present where socio-contextual factors may be different.

Methods: Data on player lifespan and biological and occupational variables were collected from publically available sources. A majority of the data were collected from wikipedia.org and sports-reference.com, which is a recognized sports archive of aggregated athlete records, and were cross-verified through rigorous web-based and sport encyclopedia archival searches. Several methodological approaches were used across seven studies, including descriptive and Kaplan-Meier and Cox regression survival analyses.

Results: The key findings of this dissertation suggest that elite athletes generally have favourable lifespan outcomes, although numerous characteristics need to be taken into consideration, such as occupational (e.g., required energy system needed for participation) and biological (e.g., height) differences. As well, the leading causes of death in players from the four major sports in North America are similar to the leading causes of death in the age- and sex-matched controls from the Canadian and United States general population.

Conclusions: Statistical limitations and biased reporting may skew public perception of the relationship between participation in high performance sport and lifespan. As such, there is inherent value in scientists critically examining the health outcomes of athletes and to make these

data known to a broader audience, particularly as preconceived notions of health risks from sport participation vocalized through media often distort reality and can adversely affect sport participation rates. In summary, a comprehensive understanding of the implications of involvement in elite sport informs our broader understanding of general athlete health and helps to form evidence-based models of athlete development and care.

Dedication

To my loving parents, Minka and Živko Lemez.

Acknowledgements

I once read an article by an NBA senior writer who credited a coach's circumstance "largely to serendipity – a series of happy accidents marked by sublime timing." That quote can probably be placed next to the "PhD" after my name. Luck has many different interpretations. For me, it manifested itself in different ways over the past six years of graduate school. However, the *happy accident marked by sublime timing* that transcends all and played a critical role in my development as a student and academic was remarkable supervision. I have been incredibly fortunate to have been supervised and mentored by both Drs. Joe Baker and Patricia Weir.

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Four years may not seem like much in the grand scheme of things; ten straight years of post-secondary schooling sure does, though. Now comes the easy part, right?

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CHAPTER ONE: GENERAL INTRODUCTION

Background and Rationale

One of the most linear relationships in behavioural science was described by Oeppen and Vaupel in 2002, where a four-decade increase in life expectancy across 16 decades was found for both women ($r^2 = 0.992$) and men ($r^2 = 0.980$). They noted that this linear-regression trend “may be the most remarkable regularity of mass endeavor ever observed” (Oeppen & Vaupel, 2002; p. 1029). Certainly, the notion of life expectancy continuing to steadily increase evokes feelings of optimism, yet it is deceptively simple. The mercurial nature of lifespan is the result of numerous natural and unnatural confounders impacting an individuals’ age of death. From a population health perspective, Parrish (2010) illustrated a causal web that depicted the complexity of health outcomes in populations, which included interactions among cultural, environmental, political, social, economic, behavioural and genetic factors. The health outcomes were shown to be affected by ‘intermediate’ influences such as proximal factors (e.g., diet, exercise) leading to physiological changes (e.g., blood pressure, cholesterol) and changing prevalence of diseases and injury (e.g., diabetes, falls). Adding to the complexity, ‘reverse causality’ can exist where health outcomes such as morbidity can change a risk factor (e.g., a congenital disability negatively affecting participation in physical activity; Kindig, 2014). Importantly, Kindig (2014) implied that each of these determinants does not contribute equally to health outcomes, and that differences exist depending on the population studied. For example, while Oeppen and Vaupel’s (2002) findings implicitly suggest that we as a species are engaging in healthier practices, Carrera-Bastos, Fontes-Villalba, O’Keefe, Lindeberg and Cordain (2011) reported that today’s greater life expectancy is not due to a healthier diet and lifestyle; but to improved sanitation, vaccinations, antibiotics, better medical care and lower physical trauma. Further, Samaras (2012)

suggested that these improvements in modern medicine have sharply reduced infant, childhood and maternal mortality and increased our ability to keep older people alive; however, we should have a much *higher* life expectancy as these trends do not indicate that people are living healthier. As such, although the upper limit of human life expectancy has incrementally increased, determining the exact impact of each health determinant on lifespan remains a major challenge in health research.

Historical Changes

The World Health Organization has not amended their definition of health since 1948, which states that “health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity” (World Health Organization, 1948). As a variety of socioeconomic, demographic, and epidemiologic factors shape population change (Omran, 2005), so do the magnitude and causal risk factors of health that affect life expectancy. Historical shifts in patterns of health and disease are generally considered the fundamental elements of population change, and have been illuminated through evolving theories and contemporary branches of science that capture the changing population health patterns over the past century. For example, Vaupel’s research in biodemography, an emerging sub-field within human demography that examines biological and demographic determinants of health (Carey & Vaupel, 2005), has used the biodemographic framework to investigate causal factors affecting increased longevity (e.g., Vaupel et al., 1998) and human aging through ‘postponement of mortality’ (i.e., delaying the process of aging; e.g., Vaupel, 2010). In addition, the epidemiologic transition theory (originally posited by Omran in 1971) describes the changes in health of a country undergoing modernization through population growth resulting from medical advancements (Omran, 2005). More specifically, the leading causes of death in the developed world have

changed from predominantly infectious diseases (e.g., tuberculosis) earlier in the twentieth century to non-communicable diseases (i.e., chronic and degenerative diseases such as cardiovascular disease) after 1950 (Omran, 2005).

While studying population health necessitates and facilitates integration of knowledge across the breadth of factors that influence health, it has also been criticized for its broad perspective, which limits its practicality in guiding specific research or policy (Kindig, 2014). Parrish (2010) suggested that understanding that death (or loss of function) can occur as the result of *any* of the aforementioned determinants of health (e.g., negative lifestyle choices such as smoking) may impact those determinants through either behaviour change or have implications on policy changes. Mortality and life expectancy are widely considered as the two basic measures of population health (e.g., Parrish, 2010), and Vaupel proposed in his 2010 *Nature* article that “mortality is by far the most readily and reliably measured index of health” (p. 537). As such, adopting a more focused thanatological (i.e., scientific study of death) approach can be useful for understanding and reducing longevity outcome disparities between the heterogeneous sub-groups of the population.

Athlete Health

Critical and comprehensive investigations of elite athletes’ lifespans can provide fruitful avenues to explore issues of human health through engagement in sport and assist in decoding risk factors of premature mortality (i.e., dying earlier than life expectancy at birth). While elite athletes represent a relatively homogeneous occupational population, North American professional sport leagues dating back at least half a century permit interesting opportunities to explore mortality outcomes of athletes from sports where the demographics of the athlete population have considerably evolved. More specifically, increased anthropometric and racial

heterogeneity in the four major professional team sport leagues in North America, Major League Baseball (MLB), the National Basketball Association (NBA), the National Football League (NFL) and the National Hockey League (NHL), reflect and overlap with i) the epidemiologic transition theory by virtue of the population boom and thus having larger pools of athletes to select from, and ii) the Civil Rights Movement in the United States (US) during the 1950s and 1960s with its goal to end racial segregation and discrimination, thus giving African-Americans a greater opportunity to participate in professional sport. As a result, from the social determinants of health perspective, major paradigm shifts in society over time have underpinned socio-cultural divisions, where exceptional high achievers such as professional athletes generally benefit from relatively higher income, social status and working conditions. Therefore, examining mortality outcomes of athletes from these four leagues given their historical nature and diverse cultural make-up can unveil incidences, predictors and causes of death that help form a more complete understanding of the relationship between participation in elite sport (i.e., participation in national, professional, and international competition such as the Olympics) and health.

While this research can inform our broader understanding of population health through more sophisticated knowledge of what makes and keeps athletes living longer, there is value in examining health of elite athletes from a lifespan perspective as well. For example, Wylleman and Reints' (2010) "whole career/whole person" conceptualization of career transitions of elite athletes addresses transitional challenges that athletes may go through, which can ultimately have important implications on health behaviours later in life, such as long-term involvement in vigorous physical activity.

Availability Heuristic

In Polednak's 1979 book *The Longevity of Athletes*, he prefaced his summary of studies by acknowledging that the data presented aimed to dispel common misconceptions regarding the longevity and health of former athletes. Fast forward to today, this trend of reductive generalizations still persists. It is difficult to ignore the rise in mass media (i.e., news media such as print media, broadcast news and the Internet) coverage of visible tragedies in sport, such as a premature cardiac-related death or suicide of a high-profile athlete. In particular, as social media represents a ubiquitous form of 'direct-to-consumer' communication, there is growing importance and necessity for evidence-based research that dispels social bias of misrepresentative health risks. Parrish (2010) suggested that measuring the function and well-being of a population or society itself (rather than individual members) is the most appropriate approach to measuring population health outcomes since "it focuses on how well the population produces societal-level conditions that optimally sustain the health of all people" (p. 2). Unfortunately, the predominantly public nature of social media can propagate a narrative that suggests athletes are at an increased risk of premature death, skewing public perception of the relationship between sport and health. This concept was indirectly supported in Link and Estes' (2012) study on sudden cardiac death in athletes, where they drew attention to the tendency of athletes' deaths to be nationally noted compared to much less media coverage for non-athletes.

An aggregation of similar causes of death in an alike demographic group in a short period of time, combined with inequalities between reporting deaths in higher profile and lesser known athletes, can produce a psychological phenomenon known as the *availability heuristic*. Tversky and Kahneman (1973), who posited the term, defined it as a cognitive predisposition to rely upon readily available knowledge and the ease with which an event can be brought to mind rather than examining other alternatives. This ideology also has a *serial position effect* (i.e., primacy-

recency) undertone, a cognitive bias where we recall the most recent information (along with primary/first information) better than information received in the middle (Ebbinghaus, 1902). These cognitive biases are corroborated through frequent exposure to trending topics and news stories, creating a susceptibility to overreact to news of a young athlete death. For example, implementing electrocardiograms as part of the pre-participation physical examination for athletes, such as runners prior to a marathon as a result of a previous sudden cardiac death during a race, may not be feasible and has been debated by sports federations and governing bodies (e.g., Harmon, Drezner, O'Connor, Asplund, & Finnoff, 2016).

Popular media that normalizes an overly simplistic view regarding risks of death of athletes can be particularly harmful at the youth levels of sport. Larson and Verma (1999) found that among post-industrial populations, sport was one of the most common leisure-time activities youth participated and spent time in. As such, there is inherent value in scientists critically examining the health outcomes of athletes and to make these data known to a broader audience, particularly as preconceived notions of health risks from sport participation vocalized through media often distort reality and can adversely affect sport participation rates.

Purpose and Objectives

Much attention has been given to the question of whether participation in sport is a healthy pursuit (e.g., see Baker, Safai, & Fraser-Thomas, 2014). This dissertation focuses on a branch of this question by critically examining the ultimate health outcome: mortality. While mass media can play a positive role in raising awareness to issues (e.g., long-term cognitive impact of concussions), it also provided the initial impetus for this research on mortality outcomes of high performance athletes. Thus, the overarching purpose of this research was to provide an evidence-based portrayal of i) incidence, ii) predictors and iii) causes of death in

athletes from MLB, the NBA (and American Basketball Association; ABA), the NFL and the NHL. More specifically, this investigation highlighted i) mortality outcome differences of athletes between and within professional sport(s), ii) potential statistical artifacts that may be empowering biases of risk of certain lifespan predictors and iii) the challenges of contextualizing historical data to answer questions with relevance in the present where socio-contextual factors may be different. The objectives of each study/chapter were as follows:

Chapter Two includes a systematic literature review of mortality and longevity in elite athletes which identified several risk factors within and between sports that may have powerful effects on mortality outcomes (Lemez & Baker, 2015). *Chapter Three* examines the occupational risk factor of career precocity in depth through a more critical methodological lens by providing a historical review of the precocity-longevity hypothesis, along with two studies that examined this relationship in Canadian-born NHL players (Lemez, Wattie, Ardern, & Baker, 2014) and NBA/ABA players. *Chapter Four* directly addresses the notion of growing premature deaths in professional athletes by examining the trends and causes of death in players from MLB, the NBA, the NFL and the NHL who died *during* their playing careers (Lemez, Wattie, & Baker, 2015). *Chapters Five* and *Six* re-visit the framework of cognitive biases manifested through media portrayals by investigating i) a biological mechanism in height and its role on lifespan and ii) the causes and early predictors of death in former NBA/ABA players. In addition, *Chapter Six* illuminates the statistical limitations and artifacts of using historical data for likelihood estimates with relevance in the present. *Chapter Seven* includes the final study of this dissertation which comprehensively examined the lifespans and mortality trends of athletes from MLB, the NBA/ABA, the NFL and the NHL. The key findings and practical implications of these studies

are discussed in *Chapter Eight*, along with future directions and a general conclusion to this dissertation.

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CHAPTER TWO: SYSTEMATIC LITERATURE REVIEW

Do elite athletes live longer? A systematic review of mortality and longevity in elite athletes

*Lemez, S., & Baker, J. (2015). Do elite athletes live longer? A systematic review of mortality and longevity in elite athletes. *Sports Medicine – Open*, 1:16. doi: 10.1186/s40798-015-0024-x

*This article was one of the five most downloaded articles in *Sports Medicine – Open* in 2015

Chapter Two Overview

Background: Understanding of an athlete's lifespan is limited with a much more sophisticated knowledge of their competitive careers and little knowledge of post-career outcomes. In this review we consider the relationship between participation at elite levels of sport and early mortality risk relative to other athletes and age- and sex-matched controls from the general population. Our objective was to identify, collate and disseminate a comprehensive list of risk factors associated with longevity and trends and causes of mortality among elite athletes.

Methods: English language articles were searched using the Web of Science database. Key words *athletes, death, elite, "high performance" life expect*, longevity, mortality, players, professional, and sport* were used to locate research articles. Seventeen additional articles were retrieved from reference lists found in these papers and a general web search. The inclusion criteria were the following: (1) publication year 1980 or later; (2) the study examined elite-level athletes; and (3) outcome data measured mortality/longevity trends and/or causes.

Results: Fifty-four peer-reviewed publications and three articles from online sources met the criteria for inclusion. Baseball, football, soccer, basketball, and cycling had the most reported data on elite athletes' lifespans. A variety of characteristics have attempted to explain risk of early mortality (e.g., handedness, playing position, achievement, etc.). Considerable support was found for superior longevity outcomes for elite athletes, particularly those in endurance and mixed-sports.

Conclusions: Participation in elite sport is generally favourable to longevity. Future research into the mechanisms and occupation-specific risk factors that may adversely affect health is important for a better understanding of life expectancies in both eminent and non-eminent populations.

Key Points:

- A majority of studies included in this review reported superior lifespan outcomes for elite athletes compared to age- and sex-matched controls from the general population and other athletes.
- Several characteristics within and between sports may have powerful effects on the overall lifespans of players (e.g., type of sport, playing position, race, and energy system).
- Future research on mortality in elite athletes would benefit from more comprehensive statistical measures and reliable databases to determine potential mechanisms and risk factors that may influence mortality trends and causes in both athlete and non-athlete samples.

Background

Rationale

Researchers have given considerable attention to the athlete development process (e.g., positive youth development through sport; see Holt, 2008). Interestingly, insight into post-career outcomes is significantly limited. For instance, our knowledge of how participation in elite sport affects lifespan is particularly incomplete. This lack of information about later phases of athletes' lifespans may be attributable to several factors, such as the greater access to athletes during their competitive careers as well as the preponderance of participation- and performance-based theories focusing on elements related to understanding the antecedents of sporting success (e.g., Baker & Horton, 2004). Attaining a *complete* understanding of an athlete's lifespan requires knowledge of the inherent complexity of relationships that link hereditary and environmental characteristics to developmental outcomes. In this study, we focus on mortality of previously-elite athletes.

An important challenge to mortality research in sport is the lack of data on the health behaviours of athletes post-retirement. Much of our current understanding of trends and causes in elite athlete mortality derives from what appear to be 'one-off' studies by small teams of researchers. Past work examining this issue in depth appears to be limited, although the Finnish studies that examined lifespans of former elite endurance, team, and power athletes who represented Finland between the years 1920 to 1965 are exceptions (e.g., Kettunen et al., 2014; Kujala et al., 2001; Sarna, Kaprio, Kujala, & Koskenvuo, 1997; Sarna, Sahi, Koskenvuo, & Kaprio, 1993). In addition, views on elite athlete mortality are largely shaped by popular media sources, which may downplay tenets of the lifespan and create false perceptions of early

mortality in athletes (e.g., MLB's Tony Gwynn at 54 years [cancer], the NFL's Junior Seau at 43 years [suicide], boxing's Tommy Morrison at 44 years [AIDS], sailing's Andrew Simpson at 36 years [drowning] and skiing's Sarah Burke at 29 years [training fall]).

A recent meta-analysis completed by Garatachea et al. (2014) indicated that elite athletes live longer than the general population, with an all-cause pooled standard mortality ratio (SMR) of 0.67 (95% confidence interval [CI] 0.55–0.81; $P < .001$). Further, they found a lower risk of cardiovascular disease (CVD) and cancer in those who participated in high performance sport, which emphasized the health benefits of exercise (Garatachea et al., 2014). While their study makes an important contribution to our knowledge of longevity outcomes in elite sport, the restrictive sampling frame necessary for a meta-analysis excluded numerous studies that could inform our broader understanding of elite athlete health. For example, Garatachea et al. (2014) included 10 studies of elite athlete longevity in their meta-analyses, but their inclusion criteria excluded studies that did not use SMR as a statistical measure of mortality (38 studies were excluded for this reason). As a result, studies with important information regarding longevity in elite athletes were excluded, such as greater longevity in Norwegian professional divers (hazard ratio [HR] = 0.79, 95% CI 0.63–0.997; Irgens, Troland, Thorsen & Grønning, 2013), and important risk factors that may influence longevity, such as sex, as indicated by Olympic medal-winning females having greater longevity relative to Olympic medal-winning male athletes (HR = 0.61, 95% CI 0.51–0.72; Coate & Sun, 2013), and race, as indicated by African-American professional basketball players having a 77% greater risk of death compared with white players (HR = 1.77, 95% CI 1.35–2.32; Lawler, Lawler, Gibson, & Murray, 2012). Further, several studies which reported SMRs were not included in the meta-analysis (e.g., Farahmand, Broman, de Faire, Vågerö, & Ahlbom, 2009; Lehman, Hein, Baron, & Gersic, 2012; Morcet, Perrin,

Trégaro, Carré, & Deugnier, 2012; Poznańska & Gajewski, 2001; Reynolds & Day, 2012). As such, there is an opportunity to grow our current limited understanding of longevity outcomes of elite athletes by examining a wider range of studies and sports through a systematic review.

Similarly, Teramoto and Bungum (2010) completed a narrative review of mortality and longevity of elite athletes; however, a relatively small sample of 14 epidemiological studies was included. Their primary method of literature retrieval was through the PubMed (1950–) and Scopus (1960–) databases, using key words *mortality*, *longevity*, *life expectancy*, *death* and *(elite/professional) athletes* and *players*. While this literature search strategy was appropriate, a large number of studies on elite athlete mortality and longevity were published during or after their publication year of 2010 (e.g., Abel & Kruger, 2010; Baird et al., 2010; Baron, Hein, Lehman, & Gersic, 2012; Clarke et al., 2012; Coate & Schwenkenberg, 2012; Coate & Sun, 2013; Grimsmo, Maehlum, Moelstad, & Arnesen, 2011; Irgens et al., 2013; Kettunen et al., 2014; Koning & Amelink, 2012; Kuss, Kluttig, & Greiser, 2011; Lawler & Lawler, 2011; Lawler et al., 2012; Lehman et al., 2012; Lindqvist et al., 2014; Marijon et al., 2013; Morcet et al., 2012; Reynolds & Day, 2012; Sanchis-Gomar, Olaso-Gonzalez, Corella, Gomez-Cabrera, & Vina, 2011; Smith, 2011a, 2011b; Zwiers et al., 2012). As a result, risk factors such as race have since been validated as more consistent indicators of early mortality (e.g., Baron et al., 2012; Lawler et al., 2012). Currently, it may be premature to make conclusions about the long-term value of being a professional athlete considering the new evidence that has emerged from literature.

Although it appears that there are many unanswered questions concerning athletes' lifespans, Teramoto and Bungum (2010) presented enough empirical evidence to determine some cross-sport and energy systems trends. Teramoto and Bungum (2010) found a trend towards endurance (e.g., long-distance runners) and mixed-sport (e.g., soccer) athletes having more

favourable survival outcomes relative to power sport (e.g., weightlifters) athletes and the general population. Similarly, a meta-analysis performed by Löllgen, Böckenhoff and Knapp (2009) examined 38 studies that measured physical activity and all-cause mortality in samples of physically active individuals (non-elite athletes) and reported an overall significant relationship between physical activity participation and lower all-cause mortality. Light and moderate intensity levels of activity were generally associated with a reduction in early mortality, whereas training at high intensities was not required for the main prevention against all-cause mortality (Löllgen et al., 2009). While Löllgen and colleagues (2009) provided evidence of physical activity positively influencing lifespan independent of age and sex, the relationship between participation in *elite sport* and longevity can enhance our understanding of the benefits of physical activity at the highest levels of competition in unique athletic cohorts.

Teramoto and Bungum's (2010) review suggested that the type and dose of elite sport participation may ultimately determine mortality risk. In addition, Teramoto and Bungum (2010) highlighted the importance of considering elite athletes as a heterogeneous group with respect to mortality trends; differences between and within sports exist. In particular, the differences in health-related behaviours between and within sports may also create modifiable factors that are associated with longevity and mortality. As a result, overall mortality risk is explained by several modifiable factors, such as obesity and physical inactivity, and non-modifiable (unchangeable) factors, such as age and race, that are unique to athletes. For instance, Baron et al. (2012) found an overall decrease in mortality rates in National Football Players (NFL) who were active between 1959 and 1988; however, defensive linemen had increased mortality rates from CVD and cardiomyopathy. Moreover, those with a playing time body mass index (BMI) of $>30 \text{ kg/m}^2$ had a significantly higher risk of CVD, which was also influenced by race/ethnicity (Baron et al.,

2012). Although the effects of playing position on lifespan may be more pronounced in contact sports such as football, we cannot overlook the differences and nuances in health-related behaviours between and within sports that may influence lifespans.

Objectives

A subject such as death rates in professional athletes may be more susceptible to sensationalism when trends begin to emerge; therefore, it is essential that the data being disseminated are transparent and accurate. In this review, we consider the relationship between participation at elite levels of sport and mortality risk relative to other athletes and age- and sex-matched controls from the general population. Our objective was to extend the narrative review of Teramoto and Bungum (2010) with a more comprehensive and up-to-date list of studies on mortality and longevity in previously elite athletes. More specifically, our aim was to advance knowledge in this area by collating athlete mortality/longevity literature that may help refine future analytic methods, form evidence-based models of athlete longevity, and determine whether elite-level participation in high performance sport produces a lifespan advantage. Similar to Teramoto and Bungum's (2010) research questions, we asked the following: (1) do elite athletes have superior longevity outcomes relative to the general population, and (2) which mechanisms and risk factors are associated with longevity and are potential precursors to early mortality?

Methods

Literature Search

Following the PRISMA statement (Moher, Liberati, Tetzlaff, & Altman, 2009), a systematic review of literature was performed using the Web of Science database (1 January 1980–30 September 2014; see Tables 1.1 and 1.2). Web of Science was chosen as our primary

citation index as it contained over 90 million records through its 7 online databases, which would have made searches into smaller citation indexes largely redundant (e.g., EMBASE contains over 28 million records). Further, given this review's objective, Web of Science's databases appeared to be the most relevant to our study (e.g., Science Citation Index Expanded). Key words, including *athletes*, *death*, *elite*, "*high performance*" *life expect**, *longevity*, *mortality*, *players*, *professional*, and *sport*, were used to locate research articles. While a full electronic search strategy for at least *one* database is recommended (Moher et al., 2009), we further located research articles by searching the references of records that were identified through our database search, in addition to performing a general web search through the Google Scholar search engine.

Inclusion Criteria

The inclusion criteria were the following: (1) publication year 1980 or later; (2) the study examined elite-level athletes; and (3) outcome data measured mortality/longevity trends and/or causes. We excluded studies with no full-text availability (abstracts, conference proceedings, commentaries, and editorials), no English-text availability (a small proportion of international articles were unavailable for translation to English), other literature reviews with different inclusion criteria (e.g., non-elite samples), duplicates, case reports, studies on morbidity (i.e., CVD risk factors, etc.), psychosocial measures, collegiate athletes, and studies on the effectiveness of pre-screening strategies and prevention. For consistency, we use "elite" synonymously with any form of high performance participation in sport (i.e., national, professional, and international competition such as the Olympics).

Data Extraction

We identified 1,001 records through database searching using the aforementioned key words. After review of title and abstract, we excluded 961 of those records (identical papers, $n = 31$; inclusion criteria not fulfilled, $n = 930$; most commonly due to studies not examining elite athletes and/or mortality/longevity trends), which left 40 eligible full-text articles from the Web of Science database search. Seventeen additional articles were retrieved from reference lists found in these papers and a general web search. In total, 57 studies filled the criteria for inclusion (Tables 1.1 and 1.2): 54 peer-reviewed publications (see Table 1.1), in addition to three articles from online sources included to investigate findings of mortality risk in elite athletes that may be disseminated to a different cohort of the population (e.g., social media users; see Table 1.2). Specifically, these three additional articles were located through Google Scholar's search engine using the same key words as used in the Web of Science database search (e.g., athletes, death, etc.). We assessed the quality of each of these records through the Newcastle-Ottawa Quality Assessment Scale for cohort studies (Wells et al., 2015). See Fig. 1.1 for the PRISMA statement (Moher et al., 2009).

Measured Outcomes

This review was comprised of elite athlete mortality/longevity studies from 13 different countries: Belgium, Finland, France, Germany, Italy, Japan, Netherlands, New Zealand, Norway, Poland, Sweden, UK and the USA. Measures of mortality included: hazard rate/ratio of death (HR), life expectancy (LE), mortality rate (MR), odds ratio for mortality (OR), relative conditional survival (RCS), relative survival ratio (RSR), SMR, and standardized proportionate mortality ratio (SPMR). All LE and MR data were compared to age-matched controls from the general population and all studies were on male participants, unless stated otherwise in the tables.

Results

Literature Search

From 1 January 1980 to 30 September 2014, we located 54 peer-reviewed studies (Abel & Kruger, 2004, 2005a, 2005b, 2006a, 2006b, 2007a, 2007b, 2010; Aggleton, Bland, Kentridge, & Neave, 1994; Aggleton, Kentridge, & Neave, 1993; Baird et al., 2010; Baron et al., 2012; Beaglehole & Stewart, 1983; Belli & Vanacore, 2005; Bianco et al., 2007; Boren & Erickson, 1998; Clarke et al., 2012; Coate & Schwenkenberg, 2012; Coate & Sun, 2013; Fafian, 1997; Farahmand et al., 2009; Fudin, Renninger, Lembessis, & Hirshon, 1993; Gajewski & Poznańska, 2008; Grimsmo et al., 2011; Halpern & Coren, 1988; Hicks, Johnson, Cuevas, DeHaro, & Bautista, 1994; Irgens et al., 2013; Kalist & Peng, 2007; Kanda et al., 2009; Kettunen et al., 2014; Koning & Amelink, 2012; Kujala et al., 2001; Kuss et al., 2011; Lawler & Lawler, 2011; Lawler et al., 2012; Lehman et al., 2012; Lindqvist et al., 2014; Marijon et al., 2013; Menotti et al., 1990; Morcet et al., 2012; Pärssinen, Kujala, Vartiainen, Sarna, & Seppälä, 2000; Poznańska & Gajewski, 2001; Reynolds & Day, 2012; Saint Onge, Rogers, & Krueger, 2008; Samaras, Storms, & Elrick, 2002; Sanchis-Gomar et al., 2011; Sarna et al., 1993; Sarna et al., 1997; Selden, Helzberg, & Waeckerle, 2009; Smith, 2011a, 2011b; Taioli, 2007; Waterbor, Cloe, Delzell, & Andjelkovich, 1988; Zwiers et al., 2012) and three online articles (Barnwell, 2012; Baron & Rinsky, 1994; Hargrove, 2006) that examined mortality and longevity in elite athletes ($N = 57$). This is an addition of 43 peer-reviewed studies that were not included in Teramoto and Bungum's (2010) review (11 peer-reviewed studies overlap). Three peer-reviewed studies from their review did not match our inclusion criteria (<1980).

Summary of Life Expectancy in Elite Athletes from Literature

This review included a total of 465,575 athletes: 450,295 from peer-reviewed literature (Table 1.1) and 15,280 from online articles (Table 1.2). Of those 465,575 examined, only 5,610 (1.2%) were female athletes. Although it appears that females have been grossly underrepresented in mortality and longevity studies of elite athletes, the extremely low proportion of 1.2% is skewed by separate studies examining the same cohort of players (e.g., Baron et al., 2012 and Lehman et al., 2012, $N = 3,439$; Kettunen et al., 2014, Kujala et al., 2001, and Sarna et al., 1993, 1997, active Finnish athletes from 1920 to 1965), single studies that contained very large sample sizes of male athletes (e.g., Farahmand et al., 2009; $N = 300,818$), and instances where the breakdown of sex in the sample went unreported (e.g., Zwiers et al., 2012). Nevertheless, only 6 peer-reviewed studies on elite athlete mortality and longevity that included females in their samples were identified (Clarke et al., 2012; Coate & Sun, 2013; Gajewski & Poznańska, 2008; Kuss et al., 2011; Menotti et al., 1990; Zwiers et al., 2012), and no studies that investigated females exclusively.

From the 9-point Newcastle-Ottawa Quality Assessment Scale (Wells et al., 2015), one study had a quality score of 6, six studies had a quality score of 7, 32 studies had a quality score of 8, and 15 studies had a quality score of 9 (only peer-reviewed articles were assessed). Therefore, the majority of the studies included in this review were generally of high quality (e.g., representative sample sizes, age- and sex-matched control groups from the general population, etc.).

Of the 54 peer-reviewed studies included, four were responses to the authors of different studies related to mortality and longevity of elite athletes (Fudin et al., 1993; Hicks et al., 1994; Smith, 2011a, 2011b). Aside from the studies or reviews that examined multiple sports, professional baseball players ($n = 16$), football players (4 peer-reviewed, 2 online), soccer

players ($n = 4$), basketball players ($n = 3$), and cyclists ($n = 3$) were identified through our literature search as having the most reported data on elite athletes' mortality outcomes. In particular, MLB players (Abel & Kruger, 2005a, 2006a; Kalist & Peng, 2007; Reynolds & Day, 2012; Saint Onge et al., 2008; Waterbor et al., 1988), NFL players (Abel & Kruger, 2006b; Baron & Rinsky, 1994; Baron et al., 2012; Lehman et al., 2012), cyclists (Marijon et al., 2013; Morcet et al., 2012; Sanchis-Gomar et al., 2011), NBA players (Fafian, 1997; Lawler et al., 2012), and golfers (Coate & Schwenkenberg, 2012; Farahmand et al., 2009) had the most robust evidence of greater longevity. These LE benefits generally ranged from 4 to 8 years (e.g., Abel & Kruger, 2005a and Sanchis-Gomar et al., 2011, respectively).

A majority of studies included in this review examined sport organizations that were primarily located in the USA, and used age- and sex-matched controls that were also American (MLB, NBA, and NFL). The general finding of these studies was a greater longevity for elite athletes relative to their American controls. Notably, the majority of mixed-sport studies included in this review also found support for greater longevity for athletes who competed at elite levels of competition (e.g., Olympics). Similarly, the 'one-off' studies from international researchers whose controls spanned 12 different countries also reflect a general trend towards increased survival rates for a diverse group of athletes relative to their country-specific controls from the general population. For example, elite cyclists, such as French, Italian, and Belgian Tour de France participants, had greater longevities when compared to the pooled general population from their respective countries for the appropriate age cohorts (Marijon et al., 2013; Morcet et al., 2012; Sanchis-Gomar et al., 2011). Further, Norwegian divers (Irgens et al., 2013) and skiers (Grimsmo et al., 2011), and Italian track and field athletes (Menotti et al., 1990), had greater longevities relative to controls from their respective countries as well. Alternatively, less

consistent results were found in soccer players, in which both superior survival rates in Dutch players (Koning & Amelink, 2012) and inferior survival rates in German players (Kuss et al., 2011) were reported, alongside increases in amyotrophic lateral sclerosis (ALS) prevalence in Italian players (Belli & Vanacore, 2005; Taioli, 2007). In addition, elite Finnish powerlifters (Pärssinen et al., 2000) displayed lower lifespans compared to the Finnish general population.

Risk Factors of Early Mortality and Primary Outcomes

Considerable research attention has been given to identifying which mechanisms and risk factors may be precursors to early mortality, including handedness, precocity, names and initials of players/athletes, playing position and weight, education and race, achievement, and energy system classifications. First, the relationship between handedness and mortality in elite athletes has received increased attention within the last two decades. Nevertheless, differences in longevity related to handedness appear to be non-existent in MLB (Abel & Kruger, 2004; Fudin et al., 1993; Hicks et al., 1994) and NBA players (Lawler & Lawler, 2011). While a relationship between longevity and handedness was identified in elite cricketers (Aggleton et al., 1993), the inclusion of a larger sample size appears to have washed out previous significant findings (Aggleton et al., 1994). Second, the precocity-longevity hypothesis (see McCann, 2001), which investigates the relationship between precociousness (i.e., career debut) and early death, has been shown to affect MLB players' longevity (Abel & Kruger, 2007a). Third, MLB players with positive initials in their names (e.g., A.C.E.) were found to live significantly longer than players with negative initials (e.g., D.E.D.; Abel & Kruger, 2007b) while baseball, hockey, football and basketball players with names beginning with D had shorter lifespans than those with names beginning with E to Z (Abel & Kruger, 2010). It is noteworthy that there has been some criticism regarding the methodologies used in these studies, such as using selective data and the

appropriateness of the statistical tests performed (Smith, 2011a, 2011b). Further, while it has been suggested that health is influenced by certain symbolic aspects of the environment (Christenfeld, Phillips, & Glynn, 1999), such as a decreased prevalence of death before birthdays (e.g., Phillips, Van Voorhees, & Ruth, 1992), the scientific rationale behind the initials of a name affecting longevity is limited. In turn, the emergence of a hypothetical name-longevity relationship in elite athletes emphasizes the need for greater replication in this area of research.

Fourth, an athlete's playing position is arguably the most obvious occupational characteristic that influences mortality risk, largely due to differences in exposures and perceived anthropometric differences that are determinants of health (e.g., weight). Longevity was found to be correlated with position in MLB (Waterbor et al., 1988) and NBA (Fafian, 1997) players. In particular, *weight* and position significantly influenced mortality risk in NFL players (Abel & Kruger, 2006b; Baron & Rinsky, 1994; Baron et al., 2012; Hargrove, 2006; Maher & Gill, 1997; Samaras et al., 2002; Selden et al., 2009). Further, weight also influenced the lifespans of baseball players (Samaras & Storms, 1992; Samaras et al., 2002), and played a role in the longevities of Japanese sumo wrestlers, although its influence appeared to be small (OR = 1.08, CI 1.01–1.15; Kanda et al., 2009). Fifth, athlete race has been shown to be associated with longevity in NBA (Lawler et al., 2012) and NFL players (Baron et al., 2012), even after controlling for covariates, as well as with education and longevity in MLB players (Kalist & Peng, 2007). Sixth, high achievement in sport emerged as a determinant of longevity, specifically through winning percentage in Japanese sumo wrestlers (Kanda et al., 2009), handicap in Swedish golfers (Farahmand et al., 2009), and Hall of Fame (HOF) induction in a diverse range of sports (Abel & Kruger, 2005b; Bianco et al., 2007). Although Abel and Kruger (2005b) reported differences in the life expectancies of MLB HOFs compared to non-inductees,

Smith (2011b) did not find a statistically significant difference using corrected data from the same sample.

The last trend that developed from these studies, which served as a classification method for Teramoto and Bungum's 2010 review, was the relationship between the type of sport and required energy systems for participation (i.e., aerobic/endurance, mixed, and anaerobic/power) and mortality. Similar to Teramoto and Bungum's (2010) findings, the largest gains in lifespans were in endurance and mixed-sport athletes. The greatest LE advantages were found in European cyclists (Marijon et al., 2013; Morcet et al., 2012; Sanchis-Gomar et al., 2011), whereas the lowest LEs were found in Finnish power lifters (Pärssinen et al., 2000). Cross-sport analyses uniformly reported LE advantages in endurance and mixed-sport athletes compared to power-sport athletes (Clarke et al., 2012; Kettunen et al., 2014; Kujala et al., 2001; Sarna et al., 1993, 1997; Zwiers et al., 2012), who had some evidence of increased premature mortality from suicide suspected from prior anabolic steroid use (Lindqvist et al., 2014; Pärssinen et al., 2000). The inconsistent mortality outcomes in power sport athletes make it difficult to generalize across all sports. Nevertheless, there was considerable support in the existing literature for superior longevity outcomes for elite athletes compared to the age- and sex-matched controls from the general population.

Discussion

The objective of our review was to advance knowledge on elite athlete mortality and longevity to ultimately determine whether elite-level participation in high performance sport produces a lifespan advantage. As a whole, the empirical evidence suggests several mechanisms and risk factors within and between sports have powerful effects on the overall lifespans of players. Our first research question considered whether elite athletes had superior longevity

outcomes relative to the general population. An overwhelming majority of studies included in this review reported favourable lifespans for athletes compared to their age- and sex-matched controls from the general population. In fact, only two studies reported lower lifespans in athletes relative to the controls: 812 male and female soccer players who participated in international matches for Germany between 1908 and 2006 ($RSR = < 1$) (Kuss et al., 2011), and 62 male Finnish powerlifters who placed first–fifth in their respective weight category in the Finnish championships between 1977 and 1982 ($SMR = 4.6$) (Pärssinen et al., 2000). Our second research question explored the risk factors associated with longevity and whether there were precursors to early mortality. Although our overall understanding of modifiable and non-modifiable factors that contribute to mortality risk in elite athletes remains limited, in part due to methodological and data source inconsistencies (Smith, 2011a, 2011b), some trends emerged from our investigation. In particular, our review supports previous conclusions that aerobic and mixed-sport athletes have superior longevity outcomes relative to more anaerobic sport athletes. In addition, playing position and weight, as well as education and race appeared to be consistent indicators of mortality risk, whereas other risk factors and characteristics such as handedness, precocity, and names and initials appeared to be less consistent and/or examined.

In their review of the relationship between body size and lifespan, Samaras et al. (2002) drew attention to several confounders that may positively influence longevity outcomes, such as higher socioeconomic status, smaller body size, and positive environmental and health benefits. As highlighted in this review, weight is an important predictor of mortality risk. Likewise, significant empirical evidence suggests obesity is one of the major risk factors for premature death (e.g., Bahrami et al., 2008). While it is premature to undervalue the relationship between weight and mortality in athletes, particularly post-retirement, research has also highlighted the

importance of accounting for body composition. For example, it has been suggested that a measure of BMI is likely to overestimate adiposity in muscular athletes (e.g., Okorodudu et al., 2010), particularly in NFL players (Baron et al., 2012). In addition, Koning and Amelink (2012) highlighted that self-selecting to participate in an occupation where health is important may predispose athletes to favourable survival outcomes relative to population comparisons. Factors such as these, in addition to a bevy of other confounders, such as access to high quality medical care (Koning & Amelink, 2012), are what make LE a difficult outcome to accurately predict.

As a variety of confounders may impact longevity, the reasons for the differences in lifespans between elite athletes and the general population are likely to be multifactorial. Teramoto and Bungum (2010) provided possible explanations of increased survival in the elite athlete cohort; namely, participation in higher volumes of exercise training leading to higher physical fitness levels, the likelihood that elite athletes are comprised of the healthiest and fittest individuals, and the maintenance of active and healthy lifestyles later in life (although less is known about longitudinal lifestyle behaviours). The extents to which these confounders contribute to mortality risk are still largely unknown however, as survival statistics may undermine the interplay of complex socioeconomic factors (Omran, 2005). For example, medical care accessibility made available by higher income may improve the LE of athletes when compared to other groups. Further, plenty of corroborating evidence suggests health-care services alone do not result in improved health outcomes, but a variety of social factors such as education and employment produce these widespread biases in health (e.g., Kelly, Morgan, Bonnefoy, Butt, & Bergman, 2007). As a result, the historical investigations of elite athletes and longevity outcomes need to be cautiously interpreted and discussed in the contexts of a variety of possible influential factors of mortality.

Abel and Kruger (2010) discussed two advantages to studying elite athletes with regard to longevity. First, they represent a relatively homogeneous occupational population, similar to Teramoto and Bungum's (2010) classification of a distinct physically fit and healthy group, and, perhaps more importantly, many sports contain detailed statistical and historical databases that track a variety of variables that can influence longevity (e.g., anthropometrics, performance-based indicators such as induction into the HOF, etc.). These advantages help contribute to the growing body of research on elite athlete mortality trends, which in turn can advance research by forming evidence-based models of athlete longevity through investigations into a variety of variables. In contrast, a caveat to this tendency of measuring the effects of sport-specific variables on mortality is that the amount of data available varies from sport to sport, and may be somewhat arbitrary. For example, handedness is unlikely to be measured in soccer players, and the influence of position in non-contact sports may be more relevant in life quality research rather than life longevity. In addition, each sport will have a different inaugural season, which limits the amount of deceased players in the relatively newer sports (e.g., Mixed Martial Arts). Therefore, each sport will have unique statistical variables that may make it more difficult for researchers to draw cross-sport comparisons.

Limitations

The main finding of this systematic review indicates favourable lifespan advantages for elite athletes relative to age- and sex-matched controls from the general population; however, limitations in reviewing literature on mortality and longevity in elite athletes exist. We will first examine the possible biases in epidemiological research in historical samples of athletes.

Databases

The use of accurate and up-to-date databases is extremely important when analyzing a sample longitudinally; however, some statistical databases for past players have been found to be incomplete. For example, Smith (2011b) discovered a substantial portion of missing death date data (e.g., unknown death dates) in former MLB players in the Sean Lahman Baseball Archive (2010). Unfortunately, Smith's (2011b) critique of Abel and Kruger's (2005b) conclusion (cases without death date data were treated as living players) that HOF non-inductees had a five-year longevity advantage was not the only study on MLB player longevity that used the same database (e.g., Abel & Kruger, 2004, 2005a, 2006a, 2007a, 2007b, 2010; Kalist & Peng, 2007). Although it may be premature to conclude that other databases have similar fallibility (e.g., missing death date data), we must be cautious of the possible incongruity between reported and unreported/unknown death date data in other studies that bias lifespan results.

Holistic Health

As a variety of socioeconomic, demographic, and epidemiologic factors dynamically interact to shape population change (Omran, 2005), so do factors that influence holistic health. Arguably, the most objective measurement of elite athlete health is rate of mortality. Using mortality statistics of elite athletes who played in the earlier decades to make inferences regarding holistic health in present-day athletes may be deceptive. The information age has made information on elite athletes easy to access, whereas information on elite athletes from the earlier eras often fails to depict the mental and social well-being characteristics that encompass holistic health. Sorenson, Romano, Azen, Schroeder, and Salem's (2014a) investigation of lifespan exercise among elite intercollegiate athletes is one of few studies that have presented empirical evidence of lifetime health and well-being in modern competitive athletes relative to age- and sex-matched controls. They found that current student athletes reported higher volumes of

weekly exercise, perceived exercise importance, and likelihood of compliance with American College of Sports Medicine (ACSM) exercise guidelines relative to non-athletes (Sorenson et al., 2014a). Interestingly, Sorenson et al. (2014a) found no significant differences between alumni student athletes and non-athletes, suggesting that former athletes failed to maintain higher exercise levels later in life. Further, in their follow-up study on the same sample, Sorenson et al. (2014b) found that relatively older former student athletes (age 43+ years) had a greater risk for joint health concerns later in life compared to a non-athlete control group. These findings seem counterintuitive since physical activity is often associated with a substantial reduction of chronic disease risk and being important to overall health and well-being (e.g., Miles, 2007).

To broaden our knowledge on holistic health outcomes and behaviour in former athletes, it is important to consider the totality of data that have been collected and analyzed to date, particularly as research pertaining to the “whole person” (e.g., physical and psychosocial measures) in modern competitive athletes gains momentum. It is equally important to make the distinction between “quality of life” and “longevity,” as physical health likely moderates psychosocial health. For example, evidence suggests that physical activity plays an important role in managing mental health diseases, such as anxiety and depression (e.g., Paluska & Schwenk, 2000). Thus, factors such as being physically capable to participate in physical activity are important to consider when determining an individual’s quality of life. As such, our current understanding of elite athletes’ quality of life during and after sport is limited relative to their mortality trends.

Statistical Measures

Another criticism of the athlete-mortality literature is on methodological grounds; more specifically, cross-study discrepancies in the statistical tests and/or measures used. Although the

relative paucity of lifespan studies of elite athletes may serve as a temporary explanation for why different measures and control variables are used to analyze mortality, we cannot ignore the impact of possible statistical bias risk. Risk of bias can affect the cumulative evidence of a review of literature (Moher et al., 2009), such as selective reporting within studies, whereby researchers may under-report variables that were found to have less statistical impact on longevity outcomes in the course of reporting results that support the direction of their findings. As a result, the extent of our knowledge about the influence of certain variables on longevity may be restricted. Future work in this area of research would benefit from replication of control variables when analyzing the same or similar athletic populations to better establish important predictors of longevity. Further, meta-analyses on the longevity of elite athletes, such as the review completed by Garatachea and colleagues (2014), can provide more evidence-based data on the benefits of participating in physical activity. Moreover, it is notable that the sports examined have examined periods of different length. Ideally, comparisons would be best when comparing timespans of similar length; however, the timespans investigated have ranged from players being born from as early as 1840 (Aggleton et al., 1994) to being active as recently as 2012 (Marijon et al., 2013). These differences could affect the proportion of those living or dead in a sample, which may affect measures such as SMR. The implication of these biases on the cumulative evidence of this review is unclear, and as a result, it is important to consider how publication bias can under- and over-estimate certain predictors of longevity.

Longitudinal Lifestyle Factors

Given that empirical evidence is necessary for coherent explanations of lifespan outcomes of elite athletes, do risk factors that typically influence longevity become ineffective and/or less powerful if maintenance of physical activity ceases? Although there has been some

evidence that former intercollegiate student athletes fail to maintain higher exercise levels later in life (Sorenson et al., 2014a), our understanding of the impact of different longitudinal lifestyle factors influencing lifespan remains incomplete. For example, former male Finnish world class athletes were found to be more active than their non-competitive controls (Fogelholm, Kaprio, & Sarna, 1994), and participation in physical activity at a young age predicted later life involvement, which reduced the prevalence of coronary heart disease (Kujala, Sarna, Kaprio, Tikkanen, & Koskenvuo, 2000). In addition, former athletes have been found to partake in fewer negative health habits, such as smoking and drinking alcohol (Fogelholm et al., 1994).

Another issue that is emerging from more recent research relates to the possible detrimental effects of high levels of training. In particular, O’Keefe (e.g., Lavie, O’Keefe, Sallis, 2015; O’Keefe et al., 2012) has advocated that ‘excessive’ aerobic training can result in cardiovascular damage (e.g., atrial fibrillation, coronary artery disease, and malignant ventricular arrhythmias). These effects may have particular relevance for studies of mortality in previously-elite athletes. It is important for future research to determine which factors are more robust predictors of longevity and if they continue to be relevant in later life. In turn, these findings will have implications on the generalizability of factors found to predict early mortality and longevity in elite athletes that were measured at one point in time (i.e., active athletic career).

Future Directions

The relationship between sport and health has evolved considerably over the past 100 years. The context in which the historical data are transferable is important to consider, particularly when discussing the social determinants of health (Kelly et al., 2007). Despite its limitations, historical analyses of sport and health shape our present understanding of its relationship and influence.

Reliable Databases, Repetition, Causes of Mortality, and Follow-up Studies

Continued contributions to the growing body of research on longevity outcomes of elite athletes should utilize appropriate statistical testing with reliable and complete databases. Although there are a variety ways to statistically measure and report mortality, research must be substantiated through repetition. An important first step is locating or comprising a reliable and comprehensive database that embodies all accessible and applicable data. To this end, future research of athlete lifespan outcomes can arguably have the greatest impact by determining the *causes* of mortality. Current empirical evidence on the rates of mortality in athletes is far superior to our knowledge on the causes of mortality. Epidemiological studies with long-term follow-ups are also rare (Belli & Vanacore, 2005). For example, available evidence suggests a possible connection between dietary supplements and/or drug use and the high prevalence of amyotrophic lateral sclerosis (ALS) in former soccer players (Belli & Vanacore, 2005; Taioli, 2007). This potential association emphasizes negative long-term neurological outcomes of performance demands that are not reflected in MR occurrence.

Cross-sport Comparisons and Generalizability

Several sports were noticeably absent from the elite athlete mortality literature (e.g., ice-hockey, field hockey, handball, snowboarding, table tennis, volleyball, and motorsports). In addition, sex-related differences in lifespan remain largely unknown due to the paucity of studies on elite female athletes. This raises an important concern about the cross-sport generalizability disseminated in this review. Perhaps more importantly, the applicability of these results to the general population (i.e., non-elite-athletes) can likely be best explained by health risk factors that have not been extensively examined to date in the athlete cohort. Some examples include: smoking and diet/nutrition (as stated by Teramoto & Bungum, 2010), lifespan health (psychosocial and physical, such as the influence of morbidity on life quality, etc.), and the

interplay of nature (hereditary, such as superior genotypes for physical fitness) (e.g., Montgomery & Safari, 2007) and nurture (environmental influences). Presumably, elite athletes possess advantageous genetic traits. Research on the heritability of physical fitness (e.g., Garatachea & Lucia, 2013) suggests that we cannot discount the influence of advantageous genetic inheritance coupled with high levels of participation and competition in sport.

Conclusion

Risk factors such as type of sport, playing position, weight, education, and race can inform our understanding of lifespan, which places increased responsibility on future research to demystify and contextualize mortality risk in both eminent and non-eminent populations. In conclusion, while additional research studies are needed to address quality of life and wellness outcomes, this review highlights mortality trends among elite athletes and concludes that participation in elite sport is generally favourable to lifespan.

Ethical Standards

This manuscript does not contain clinical studies or patient data. Srdjan Lemez and Joseph Baker declare that they have no conflict of interest. This project was not funded.

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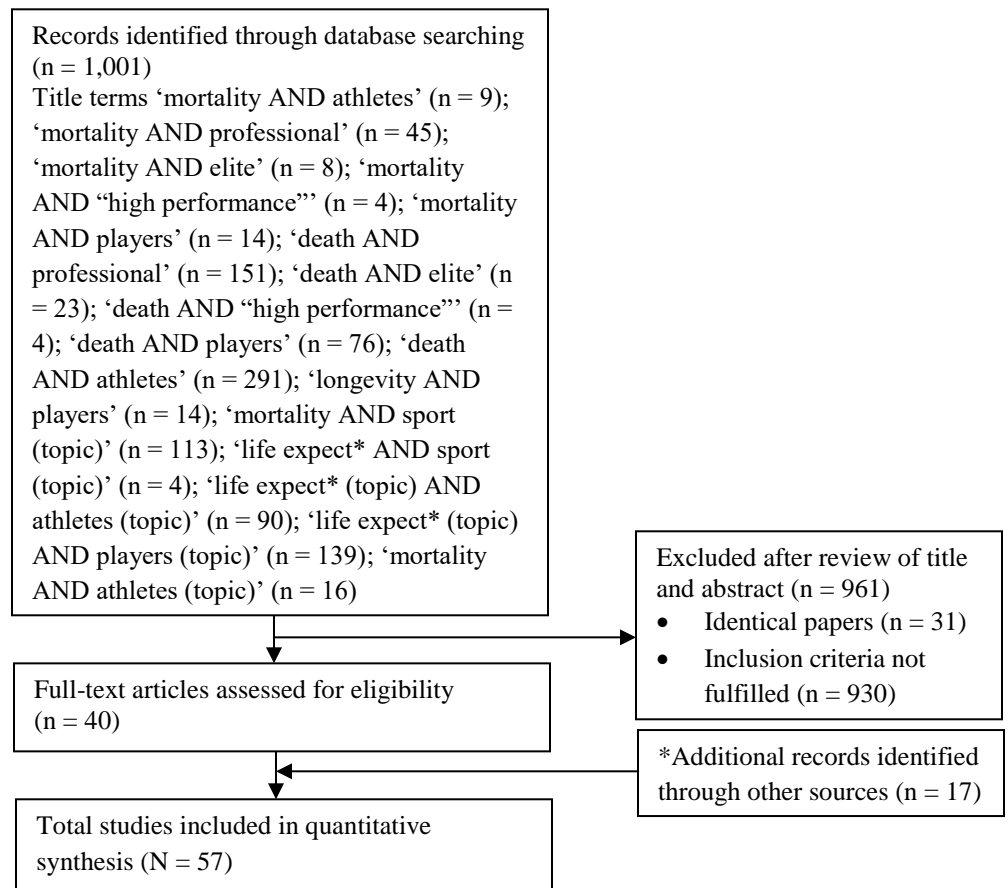
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Figure 1.1: Flow of information through the different phases of a systematic review, as per the PRISMA statement (Moher et al., 2009).



*Additional records were identified through searching the references of records that were identified through database searching and a general web search (i.e., Google Scholar)

Table 1.1: Peer-reviewed elite athlete mortality literature (> 1980; $n = 54$)

Sport/Country	Authors	N	Key finding	LE vs. GP
MLB ¹	Abel & Kruger (2004)	6038	No significant differences in longevity related to handedness ($F[2,6035] = 0.13$); (death < 2001)	--
MLB ¹	Abel & Kruger (2005a)	2604	LE: ~4–5 years longer ($f = 188.0$, $df = 1$, $2,555$, $p < 0.001$); (1900–1950 debut)	↑
MLB ¹	Abel & Kruger (2005b)	3573	Median post-induction survival for HOFs was 5 years shorter than for non-inducted players, 18 years (CI 15.0–21.0) vs. 23 years (CI 22.1–23.9) for matched controls (OR = 1.37, CI 1.08–1.73); (death \leq 2002)	--
MLB ¹	Abel & Kruger (2006a)	4492	LE: 4.8 years longer (SD = ± 15.0); career length increased longevity ($F = 3.4$, $df = 3,4379$; $p < 0.02$); (1900–1939 debut)	↑
MLB ¹	Abel & Kruger (2007a)	3760	Earlier career debut predicted earlier death ($F[8,2898] = 7.78$, $p < 0.001$); (death < 2006; 1900–1935 debut)	--
MLB ¹	Abel & Kruger (2007b)	3835	Players ($n = 11$) with positive initials (e.g., A.C.E.; $80.4 \pm SE = 3.0$) lived significantly longer ($F[2,904] = 3.7$, $p < 0.03$, two-tailed) by 13 years than players ($n = 30$) with negative initials (e.g., D.E.D.; $67.0 \pm SE = 3.1$) or players ($n = 864$) with neutral initials ($67.1 \pm SE = 0.5$); players with positive initials lived significantly longer than their matched controls and those with negative initials ($p < 0.05$); (died before 1950)	--
MLB ¹	Boren & Erickson (1998)	28	The most common toxin that lead to death by poisoning was carbon monoxide; low overall risk (death 1889–1995)	--
MLB ¹	Fudin et al. (1993)	1686	<i>In response to Halpern and Coren's (1988) study:</i> Left-handers ($n = 235$) had a M longevity of 64.46 years (SD = 15.34) compared to 64.56 years (SD = 15.02) for right-handers ($n = 1451$), a difference of 38.57 days ($t[1684] = 0.09$, $p > 0.05$ for	--

years lived; Halpern and Coren (1988) reported a difference of 244.72 days); (considered longevity data through 1978)

MLB ¹	Halpern & Coren (1988)	1708	<i>M</i> age at death for right-handers was 64.64 years (n = 1472; SD = 15.5) and 63.97 years for left-handers (n = 236; SD = 15.4), a significant difference ($Z = 6.63$, $P < 0.001$)	--
MLB ¹	Hicks et al. (1994)	5441	<i>In response to Fudin et al.'s (1993) study:</i> Reliable data were confirmed for 3501 right-handers (64.3%), 1199 mixed-handers (22.0%) and 741 left-handers (13.6%); the differences in <i>M</i> days of life were not significant in each group ($F[2,5338] = .59$, $p = 0.55$) and between just right- and left-handers ($t[4240] = -1.08$, $p = 0.28$; <i>M</i> longevity less for right-handers)	--
MLB ¹	Kalist & Peng (2007)	2641	Greater longevity overall (SMR = 0.31); positive relationship between education and longevity (HR = 0.74, CI 0.56–0.977); black players had a HR 2.47 times greater than white players (1963–1996 debut)	↑
MLB ¹	Reynolds & Day (2012)	14360	Greater longevity rates throughout the last century (1900–1999 debut); (SMR = 0.87, CI 0.85–0.89; 1930–1999)	↑
MLB ¹	Saint Onge et al. (2008)	6772	LE: ~5 years longer, compared to 20-year-old U.S. males; at 20 years, players from the Modern Era can expect to live 65.5 years vs. 52.4 years and 58.3 years from the Early and Golden Eras, respectively (1902–2004 debut)	↑
MLB ¹	Smith (2011a)	--	<i>In response to Abel and Kruger's (2007b) study:</i> No relationship between name initials and longevity ($p > 0.05$)	--
MLB ¹	Smith (2011b)	102	<i>In response to Abel and Kruger's (2005b) study:</i> Corrected data shows that there is no statistically significant difference in the LE of HOF players ($Z = 0.06$, two-sided $P = 0.952$)	--
MLB ¹	Waterbor et al. (1988)	985	Greater longevity overall (SMR = 0.94); longevity was correlated with position and in-game achievement (1911–1925 debut)	↑

NBA ¹	Fafian Jr. (1997)	2810	Greater longevity overall, particularly in guards (active 1946–1994)	↑
NBA ¹	Lawler & Lawler (2011)	3647	Handedness was not significantly related to LE (HR = 0.92, CI 0.54–1.60; $P = 0.76$); (active 1946–2009)	--
NBA ¹	Lawler et al. (2012)	3366	White players lived longer (~1.5 years) than black players (HR = 1.77, CI 1.35–2.32); LE: ~4 year longer overall (active 1946–2005)	↑
NFL ¹	Abel & Kruger (2006b)	1512	LE: 6.1 years longer (SD = ± 11.9); career length ($F = 4.7$, $df = 2, 1504$; $p < 0.01$) and BMI ($R^2 = 0.01$, $p < 0.01$) increased longevity (debut < 1940)	↑
NFL ¹	Baron et al. (2012)	3439	Greater longevity overall (SMR = 0.53, CI 0.48–0.59); BMI, race, and position were associated with longevity (active 1959–1988)	↑
NFL ¹	Lehman et al. (2012)	3439	Greater longevity overall (SMR = 0.53, CI 0.48–0.59); increased overall risk of neurodegenerative MR (SMR = 2.83–3.26, CI 1.36–5.21); elevated ALS (SMR = 4.31, CI 1.73–8.87) and AD (SMR = 3.86, CI 1.55–7.95) subcategories (active 1959–1988)	↑
NFL ¹	*Selden et al. (2009)	--	Review of recent data suggests ↑ CVD risk factors in players, particularly linemen	--
Boxing	Baird et al. (2010)	339	Decline in premature MR after 1983 (rounds reduced from 15 to 12); (death 1950–2007)	--
Cricket ¹³	Aggleton et al. (1993)	3165	There was a significant lifespan difference ($t[3163] = 2.77$, $P = 0.006$, two-tailed) between right-handers ($n = 2580$; 65.62 years) and left-handers ($n = 585$; 63.52 years); left-handers were more likely to die prematurely in accidents or in warfare (players in the British Isles from 1864–1983)	--
Cricket ¹³	Aggleton et al.	5960	No significant relation between mortality and handedness ($P = 0.3$); left-handers	--

	(1994)		were more likely to die from unnatural causes ($P = 0.03$, log hazard 0.37, CI 0.04–0.70), particularly warfare ($P = 0.009$, log hazard 0.53, CI 0.13–0.92); (born between 1840–1960; players in the British Isles from 1864–1992)	
Cyclists ²	Marijon et al. (2013)	786	Greater longevity overall in Tour de France participants (SMR = 0.59, CI 0.51–0.68, $P < 0.0001$); reduction in neoplasms (SMR = 0.56, CI 0.42–0.72, $P < 0.0001$) and CVD (SMR = 0.67, CI 0.50–0.88, $P = 0.004$); (1947–2012 participants)	↑
Cyclists ²	Morcet et al. (2012)	514	Greater longevity overall (SMR = 0.50, CI 0.34–0.71); although higher MR in younger cyclists (active 1960–1990)	↑
Cyclists ²³⁴	Sanchis-Gomar et al. (2011)	834	LE: ~8 years longer for Tour de France participants ($p < 0.05$); (active 1930–1964)	↑
Diving ⁵	Irgens et al. (2013)	3130	Greater longevity overall (HR = 0.79, CI 0.63–0.997), although increased violent deaths (born 1950–1999)	↑
Golf ¹	Coate & Schwenkenberg, (2012)	313	LE: 5.4 years longer (won prize money 1980–2009)	↑
Golf ⁶	Farahmand et al. (2009)	300818	Greater longevity overall (SMR = 0.60, CI 0.57–0.64); greatest longevity in most skilled players (SMR = 0.53, CI 0.41–0.67); (born > 1920, registered < 2001)	↑
PL ⁷	Pärssinen et al. (2000)	62	Increased premature MR (SMR = 4.6, CI 2.04–10.45; $P = 0.0002$), suspected from prior anabolic steroid use (placed 1 st –5 th in Finnish championships, 1977–1982)	↓
Rugby ⁸	Beaglehole & Stewart (1983)	822	LE of All Blacks is the same as GP, although LE (73.0 years) for non-Māori All Blacks was ~10 years longer (CI 71.8–74.5) relative to the Māori All Blacks (1884–1981)	=
Skiing ⁵	Grimsmo et al. (2011)	122	Total MR was 9% lower after a 30-year follow-up ($P = 0.04$); (study participants, 1976–1981)	↑

Soccer ³	Belli & Vanacore (2005)	350	Similar observed and expected MR, although a ten-fold increase of ALS MR (SPMR = 1158, CI 672–1998) was present (active 1960–1996)	=
Soccer ⁹	Koning & Amelink (2012)	371	Greater longevity overall ($P = 0.003$); (active 1970–1973)	↑
Soccer ¹⁰	Kuss et al. (2011)	812	Cumulative observed survival was smaller than cumulative expected survival; therefore, male and female players had reduced longevity (RSR = < 1); (active 1908–2006)	↓
Soccer ³	Taioli (2007)	5389	Greater longevity overall (SMR = 0.68, CI 0.52–0.86), although increased risk for car accident (SMR = 2.23, CI 1.46–3.27) and ALS (SMR = 18.18, CI 5.00–46.55) death (active 1975–2003)	↑
SW ¹¹	Kanda et al. (2009)	73	Deceased wrestlers had higher BMIs (OR = 1.08, CI 1.01–1.15) and winning percentages (OR = 1.29, CI 0.86–1.93); (active 1926–1989)	--
T & F ³	Menotti et al. (1990)	983	Greater longevity in males (SMR = 0.73; n = 700) and females (SMR = 0.48; n = 283); significant when analyzed together ($P = 0.0296$); (active > 1940)	↑
Mixed ¹	Abel & Kruger (2010)	10216	Decrease in longevity associated with names beginning with A to D; linear decrease for baseball and hockey players, but non-linear in football and basketball players; for each sport, A names lived longer than E to Z names, and D names had decreased longevity compared to E to Z names $F(4,10,193) = 4.16, P = 0.002$); football players lived the longest (72.3 ± 12.7) and baseball players lived the least years (70.8 ± 14.7); significant letter differences occurred between A and E-Z (HR = 0.80, CI 0.71–0.91, $p < 0.001$) and D and E-Z (HR = 1.16, CI 1.04–1.30, $p < 0.01$); (born 1875–1930)	--
Mixed	Bianco et al. (2007)	715	Baseball (LE: 76; n = 154), basketball (LE: 78; n = 58), boxing (LE: 73; n = 81), football (LE: 77; n = 81), ice-hockey (LE: 74.5; n = 130), swimming (LE: 74; n = 37), tennis (LE: 79; n = 83), track and field (LE: 75; n = 59), and wrestling (LE: 77; n = 32) HOFs had greater overall longevity ($p < 0.05$); ($M = 76$ years);	↑

(born 1860–1930)

Mixed	Clarke et al. (2012)	15174	11,619 (76.6%) male and 3,555 (23.4%) female Olympic Game medalists' (1896–2010) LE was 2.8 years longer (RCS = 1.08, CI 1.07–1.10); endurance (RCS = 1.13, CI 1.09–1.17) and mixed (RCS = 1.11, CI 1.09–1.13) sport athletes had a more favourable survival advantage relative to power sport athletes (RCS = 1.05, CI 1.01–1.08)	↑
Mixed ¹	Coate & Sun (2013)	2690	Females (n = 1348; HR = 0.61, CI 0.51–0.72) had a ~6 year LE advantage over males (n = 1342), even though they competed in the same Olympic events (1900–2008) and tennis championships (HR = 0.65, CI 0.47–0.91; > 1880); Olympic medalists' LE: 2-3 years longer if born < 1920, but smaller advantage overall; tennis players' LE: 5-6 years longer if born < 1920, and 2-3 years overall; <i>M</i> LE was 82 years for females and 76 years for males	↑
Mixed ¹²	Gajewski & Poznańska (2008)	2113	Greater longevity overall in male (n = 1689; SMR = 0.50, CI 0.44–0.56) and female (n = 424; SMR = 0.73, CI 0.48–1.05) Olympians (1924–2000)	↑
Mixed ⁷	Kettunen et al. (2014)	2363	LE: ~5-6 years longer overall; endurance (79.1 years; CI 76.6–80.6), team (78.8 years; CI 78.1–79.8) and power (75.6 years; CI 74.0–76.5) sport athletes who represented Finland in international competitions (active 1920–1965) had higher LE than controls (72.9 years, CI 71.8–74.3), even after adjusting for socio-economic status, birth cohort, and disease-specific mortality (HR = < 1); boxers had increased risk of dementia mortality (HR = 4.20, CI 2.30–7.81)	↑
Mixed ⁷	Kujala et al. (2001)	2009	Greater longevity overall (SMR = 0.74, CI 0.69–0.79); all-cause mortality was lower for endurance (SMR = 0.57, CI 0.47–0.68), mixed (SMR = 0.68, CI 0.61–0.76), and power (SMR = 0.90, CI 0.81–1.00) sport athletes who represented Finland in international competitions (active 1920–1965); increased risk of hypertension in power sport athletes (SMR = 2.63, CI 1.06–5.42); (SMRs calculated 1971–1995)	↑
Mixed ⁶	Lindqvist et al.	1199	MR was not increased overall in former power sport athletes, except a slight	--

	(2013)		increase at 45 years (CI 2.1–4.2); between 20-50 years, estimated HR = 1.44–1.46; 2.1–3.9 times increased MR from suicide between 30 and 50 years (overall, HR = 1.74, CI 1.08–2.66; $P = 0.025$); however, there was a lower malignancy MR (HR = 0.71, CI 0.50–0.98; $P = 0.036$); (active 1960–1979)	
Mixed ¹²	Poznańska & Gajewski, (2001)	1505	Greater overall longevity (SMR = 0.42, CI 0.35–0.49), particularly between 1992–1998 (Olympians 1981–1998)	↑
Mixed	*Samaras et al. (2002)	--	Modest correlation coefficients indicated that weight is only one risk factor that affected the longevity of baseball players (n = 1278; $r = -0.22$, $p < 0.025$), football players (n = 199; $r = -0.33$, $p < 0.005$), and Finnish elite athletes ($r = -0.51$, non-significant)	--
Mixed ⁷	Sarna et al. (1993, 1997)	2613	LE for endurance (5.7; $M = 75.6$, CI 73.6–77.5), team (4.0; $M = 73.9$, CI 72.7–75.1), and in power (1.6; $M = 71.5$, CI 70.4–72.2) sport athletes was greater than the referents (69.9); decreased CVD in endurance (OR = 0.49, CI 0.26–0.93) and team (OR = 0.61, CI 0.41–0.92) sport athletes (active 1920–1965)	↑
Mixed	Zwiers et al. (2012)	9889	High intensity sport athletes had lower longevity compared to low intensity sport athletes (high risk of bodily collision, HR = 1.11, CI 1.06–1.15, and high levels of physical contact, HR = 1.16, CI 1.11–1.22 (Olympians 1896–1936; sex breakdown unknown)	--

Notes: AD, Alzheimer disease; ALS, amyotrophic lateral sclerosis; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; GP, general population; HOF, Hall of Fame; HR, hazard rate/ratio of death; LE, life expectancy; M, mean; MLB, Major League Baseball; MR, mortality rate; NBA, National Basketball Association; NFL, National Football League; OR, odds ratio for mortality; PL, powerlifting; R², explained variation/total variation (coefficient of determination); RCS, relative conditional survival; RSR, relative survival ratio; SD, standard deviation; SMR, standardized mortality ratio; SPMR, standardized proportionate mortality ratio; SW, sumo wrestling; T & F, track and field
 Samples: ¹USA; ²France; ³Italy; ⁴Belgium; ⁵Norway; ⁶Sweden; ⁷Finland; ⁸New Zealand; ⁹Netherlands; ¹⁰Germany; ¹¹Japan; ¹²Poland; ¹³United Kingdom; Studies with no subscript analyzed multiple countries, or other sports were used as controls. Full citations provided in reference list; all LE and MR data were compared to age-matched controls from the GP and all studies were on male participants, unless stated otherwise; descriptions reflect only the key findings; *reviews met inclusion criteria

Table 1.2: Online elite athlete mortality articles ($n = 3$)

Sport/Country	Authors	<i>N</i>	Key Finding	LE vs. GP
NFL ¹	Baron & Rinsky (<i>NIOSH</i>) (1994)	6848	NFL players had a 46% decreased MR (SMR = 0.54); linemen had a 52% greater risk of death from heart disease than the GP, and three times the risk compared to football players; players had a decreased risk of death from violence (79%) and accidents (39%); (players since 1959; death through 1991)	↑
NFL ¹	Hargrove (<i>Scripps Howard News Service</i>) (2006)	3850	The heaviest NFL players were more than twice as likely to die before their 50 th birthday than their teammates; players are generally not dying sooner than average, but offensive and defensive linemen had a 52% greater risk of dying from heart disease than the GP; out of the 130 players who died before age 50, 1/69 players born since 1955 are dead, 22% of which died of heart diseases (77% qualified as obese) and 19% from homicides or suicides; when compared to 2403 <i>MLB</i> players who have died in the last century, NFL players are more than twice as likely to die before age 50 (born since 1905)	--
Mixed ¹	Barnwell (<i>Grantland</i>) (2012)	4582	12.8% of football players had died ($N = 3088$) compared to 15.9% of baseball players ($N = 1494$) as of 2007 (active from 1959–1988)	--

Notes: GP, general population; MLB, Major League Baseball; MR, mortality rate; NFL, National Football League; SMR, standardized mortality ratio

¹USA

CHAPTER THREE: THE PRECOCITY-LONGEVITY HYPOTHESIS

As discussed by Lemez and Baker (2015), research attention has been given to identifying which characteristics may be precursors to early mortality, such as handedness, anthropometrics and race. One characteristic that has received considerably more attention outside of sport is precocity's role on mortality. The precocity-longevity hypothesis proposed by McCann (2001) asserts that factors related to early career achievement, such as early debut in professional sport, may cultivate early death. Until our two studies (pg.'s 74 and 80), only Abel and Kruger (2007) had examined this hypothesis in athletes. Although researchers began dissecting the relationship of early career achievement and mortality nearly a century ago (e.g., Sorokin, 1925), the subsequent development and investigation of this phenomenon has remained limited. The following section will examine the small body of research that has focused on the association of age at high career achievement and mortality from a historical perspective, and is organized chronologically, since precocity-longevity research has been sparse and the historical changes of samples and methodologies are important for explaining the evolution of the topic and its propagation today.

Body – Part 1

The first investigation linking precocity with longevity was initiated by sociologist Pitirim Sorokin (1925). Sorokin studied the lifespans of hereditary monarchs who belonged to seven groups: Roman Empire (Holy and Eastern), English kings, French kings, Austria, Prussia, Turkish sultans, and Russian czars. Scientists at that time believed that the environment surrounding monarchs produced physical, mental and moral degeneration, ultimately manifesting in shorter lifespans (e.g., Jacoby, 1904). Sorokin (1925) examined the correlation between mean accession age and mean length of life for the seven groups of monarchs and found that ages at

which monarchs inherited the throne was related to their longevity. Generally, it was found that monarchs who became rulers earlier tended to live shorter lives. Explanations for this lifespan discrepancy mainly surrounded the detrimental environment; high stress resulting from responsibility, strenuous workload, and other unfavourable conditions appeared to undermine and shorten lifespans (Sorokin, 1925).

The influence of precocious achievement on lifespan was not revisited again until Harvey Lehman's 1953 book *Age and Achievement*. Lehman (1953) examined the relationship between mean age at time of achievement and longevity in scientists from 10 different fields. A tendency towards greater longevity was found in scientists who made an exceptional contribution to their respective field at older mean ages, relative to scientists whose contributions occurred at younger ages. Thus, the trends shown by Lehman and his colleagues extended the notion that career precociousness may not be beneficial to lifespan, although the understanding of this relationship was still largely incomplete.

Scientific discovery was also used to define precocity 33 years later in Zhao and Jiang's (1986) examination of longevity of scientists. They reported that the lifespans of scientists were closely related to their peak age of achievement, which was approximately half the peak value of their lifespan (e.g., 37 years and 74 years, respectively). Further, Zhao and Jian (1986) noted an interesting trend where the distribution curves of achievements and lifespan were symmetrical, and suggested that declining ability in scientific research may be related to deteriorating health such as senility. While there are outliers to this relationship, where famous precocious scientists such as Albert Einstein, Galileo, Edmond Halley and Sir Isaac Newton exceeded their projected life expectancies, Zhao and Jiang (1986) reported that precocious scientists between the 16th century and 1960 lived shorter on average relative to 'ordinary' scientists. However, when

compared to the general population, the precocious group nevertheless had greater longevity (e.g., the average lifespan for the general population in 1871 was 36 years versus 59 years for precocious scientists). Therefore, these results were interpreted as support to further dispel the common (and outdated by this time) impression that mental labor was too much a tax on one's brain that ultimately reduced lifespan, and supported the precocity-longevity link where precocious scientists died relatively younger compared to 'ordinary' scientists.

In a separate form of career achievement and productivity, Simonton (1991) explored the determinants of creativity and early eminence on the lifespans of 120 famous classical music composers (this deceased sample accounted for 89% of all music heard in the classical repertoire, thus making it highly representative of the entire domain). Although the focus of Simonton's (1991) study was on the relationship between age and productivity, he nonetheless reported a significant correlation between creative precociousness and shorter lifespan. However, he interpreted the precocity-longevity relationship differently; Simonton suggested that precocious output simply *permits* a composer to die young and still earn recognition after death, thereby artificially inflating the precocity-longevity correlation (McCann, 2001). For example, famous composer Franz Schubert died at 31 years, but if he produced his masterpieces at a later age, his fame would have likely weakened (Simonton, 1991). In other words, the younger a composer is at the commencement of formal training the overall accomplishments are likely going to be higher, but the allure of producing a successful composition in early life may override later life achievements. As such, Simonton (1991) concluded that individuals with higher early creative potential may be predetermined to produce a successful/popular composition at a younger age, which may ultimately influence the rhetoric surrounding their death.

While Simonton's (1991) theory highlighted the nuances of understanding the relationship between career achievement and death, McCann (2001) was the first to break the mold of restricted samples and name this phenomenon the *precocity-longevity hypothesis*. McCann (2001) studied 23 samples of eminent persons (N = 1,026) through five studies. In his first study, McCann (2001) examined the longevities of US presidents and vice presidents, French presidents, Canadian, British, New Zealand and Australian prime ministers, British monarchs, popes, US Supreme Court justices, and Nova Scotia premiers (he excluded those who died by assassination). Robust support for the hypothesis was found, but perhaps more interesting was his finding of the average differences in lifespan for those above and below the median age at first becoming a leader. The largest difference was found in British monarchs where the average age to get appointed a monarch in Britain was 30.4 years, and an appointment after that age indicated greater longevity by *18.4 years* relative to those who got appointed during their twenties. The 'smallest' difference was identified in US presidents, who exhibited a 7.2 year longevity advantage if elected into office at a relatively later age.

McCann's (2001) second study extended the findings of his first study and tested whether the hypothesis was generalizable to high-level creative accomplishment. He examined exceptional creativity in the form of Nobel Prize winners and Oscar winners in the best actor and actress and best supporting actor and actress categories. The advantage in examining these two samples was that i) the Nobel Prize is the pinnacle of career peak achievement in several fields, making it one of the best measures of precocity, and ii) Oscars are awarded to males and females equivalently, making it possible to test the precocity-longevity hypothesis in women for what appeared to be the first time (McCann, 2001). Nevertheless, similar to previous findings, there was overall support for precocity negatively influencing lifespan; only in the field of Physics for

Nobel Prize was there an absence of statistical significance (although the correlations and mean differences were in the hypothesized direction). The largest reported difference in longevity for those above and below the median age at first being awarded a Nobel Prize was a 13.5 year lifespan advantage for recipients in the Peace field (above the mean age of 64.8 years). Although overall support for actors and actresses was weaker relative to Nobel Prize recipients, males who won best supporting actor above the mean age of 54.8 years lived an additional 9.2 years. Therefore, McCann (2001) demonstrated that career precociousness affecting lifespan was not exclusive to high-level leadership positions, but to other influential and creative domains as well.

The third study of McCann's (2001) investigation into the precocity-longevity hypothesis examined a single momentous event; the signing of the US Declaration of Independence in 1776 (N = 56). Unlike the previously studied samples, it was impressive *past* accomplishments that largely determined the sample, with the event itself being considered a unique occurrence. McCann (2001) assumed that by participating in this historic event, younger participants must have previously demonstrated superior early achievement. The mean age at signing was 44.8 years (SD = 10.4) and the length of life was 66.4 years (SD = 14.7). McCann (2001) found a significant lifespan advantage of 7.7 years for signers who were above the median signing age, suggesting that precocity has a significant effect on longevity even when measuring precocity as a single event that was marked by past achievements.

The fourth study of McCann's (2001) investigation also examined the precocity-longevity hypothesis from an atypical perspective, where he operationalized precocity as age at lesser/multiple career peaks. He examined the relationship between age at receiving an American PhD in Psychology and the lifespans of eminent American psychologists (N = 86). Similar to previous findings, support was found for the precocity-longevity hypothesis; the mean age of

attaining a PhD was 27.5 years (SD = 3.1), which was significantly correlated with the mean death age of 71.8 years (SD = 11.5). Students who attained a PhD above the median age lived an average of 6.9 years longer, suggesting that the hypothesis is applicable to lesser career peaks as well (McCann, 2001). Alternatively, a person's career trajectory is typically variable, where multiple career peaks may exist and be termed as 'precocious' achievement. McCann's (2001) finding that ages at career peaks that are later surpassed *still* influences lifespan highlights that caution is needed in how we operationalize 'precocity' and begin to explore explanations for its effects in more depth. To date, all work in this area has been descriptive and the underlying risk factors remain speculative as most studies have been performed with the main purpose of establishing a link between precocity and longevity. Two proposed explanations of the precocity-longevity hypothesis are i) statistical biases and artifacts and ii) epidemiological explanations.

Expounding the Precocity-Longevity Hypothesis

Statistical Biases and Artifacts

The first proposed explanation suggests methodological limitations may explain, or at least artificially inflate, the link between precocity and longevity. An artifact is defined in the context of a scientific investigation as something that is observed that is not naturally present, but occurs as a result of the procedure ("Google Dictionary," 2016). Thus, our understanding of the precocity-longevity hypothesis is confounded by the statistical strategy used to measure the effect.

Life expectancy bias

The life expectancy bias arises from older entrants into a sample, or those who reached a career peak after the median age of the cohort, being predisposed to having greater longevity than those who entered the sample at a younger age. Similar to a threshold hypothesis, a person

can only enter a sample by crossing a threshold (in this context, reaching an achievement peak); however, the ages at which it is crossed vary from person to person (McCann, 2001). For example, persons who achieved eminence in a given domain at the age of 25 years will have 10 more years in which they could die compared with those who achieved eminence at 35 years. As Abel and Kruger (2007) describe within the context of sport, “whereas every player potentially has the same life expectancy at a given age, those who achieve success at a relatively late age have already lived longer than those who achieve similar success at an earlier age” (p. 934). Indeed, if life expectancy automatically increases with age if we assume a homogeneous life expectancy at birth, our measurement of the precocity-longevity link may simply reflect this sample inclusion artifact (McCann, 2001).

Selection bias

The basis for the selection bias artifact, initiated by Simonton (1988), is that early peak achievement allows a person to die prematurely and still be retained in an eminent sample. In turn, persons who enter a sample at an early age and die at any early age artificially inflate the precocity-longevity hypothesis. Alternatively, a person who enters an eminent sample at a later age has no chance of dying at an earlier age. This raises the issue of using an exclusively deceased sample to calculate precocity and longevity. When a sample does not include high achievers who are still alive it may result in a skewed average age that supports the precocity-longevity hypothesis. For example, if earlier achievers who die prematurely are included in the same cohort as later achievers, there is a much greater likelihood of finding a significant correlation between the two variables (Abel & Kruger, 2007; McCann, 2001). Interestingly, while studies have attempted to control for the aforementioned artifacts (discussed in the next

section), all of the literature on this phenomenon to date has used samples with only deceased persons.

The Healthy Worker Effect

In epidemiology, the last proposed statistical artifact is known as the Healthy Worker Effect (HWE; McMichael, 1976). Although many factors other than the occupational environment influence longevity, McMichael (1976) was the first to suggest that when comparing the working population with the non-working population, the active workers are presumably healthy enough to have been and remain employable in a typical occupational setting, whereas the same theoretical conclusion cannot be made for the non-workers. Abel and Kruger (2006a) stated that this effect operates on two levels: i) a “healthy hire effect” where an initial selection bias occurs because ill and/or disabled persons are not typically hired, and ii) a “healthy survivor effect” where subsequent selection bias occurs and results in healthier persons remaining employed. In any case, a lifespan bias exists in favour of the working group.

In sport, Abel and Kruger (2006b) suggested that the HWE is a result of three related factors: i) an initial selectivity associated with qualifying for employment, such as becoming a high performance athlete (e.g., Beaglehole & Steward, 1983); ii) the positive effects of regular physical activity on health (e.g., Morris et al., 1980), and; iii) a subsequent selectivity whereby workers/players who remain healthy and fit continue within their respective professions, which consequently results in a healthier occupational cohort (whereas the injury prone/unhealthy athletes become part of the general population; e.g., McNamee, 2003). Within the context of sport, the HWE is measured by career length (or ‘span of service’ in a more general context). Abel and Kruger (2005) examined the longevities of Major League Baseball (MLB) players and found that they lived an average of four years longer than age-matched controls from the general

population. More specifically, a HWE was attributed to a likely initial selection process of becoming a professional athlete, and subsequent maintenance of good health throughout their careers to retain a roster spot each year (e.g., muscular strengthening, conditioning, diet, physicals, etc.; Abel & Kruger, 2005). In revisiting the HWE on a larger sample size of MLB players, Abel and Kruger (2006a) found players to live 4.8 years longer compared to the general population, and more interestingly, robust support was found for the HWE. Career length both significantly and incrementally increased the lifespans of players from playing a single season (+4.1 years) to playing 11 or more seasons (+7.4 years; Abel & Kruger, 2006a). Similarly, strong support was found for the HWE in their follow-up study on professional football players (Abel & Kruger, 2006b). On average, professional football players lived 6.1 years longer than age-matched controls from the general population, and longevity was again found to increase incrementally with the number of seasons played (+5.5 years for one season to 6.7 years for four or more seasons; Abel & Kruger, 2006b). In sum, research on the HWE in sport thus far corroborates findings from other occupations; the longer the span of service, the greater their lifespans appear to be.

Epidemiological Explanations

As the precocity-longevity hypothesis remains relatively under-studied as evidenced by the lack of scientific replication and long durations between studies, explanatory frameworks for its effect remain speculative. Nevertheless, two epidemiological explanations have been at the forefront of discussion (and have existed from the hypothesis' inception, albeit as an undertone in the earlier studies): stress and personality type.

Stress

McCann (2001) suggested that the most obvious explanatory risk factor is the role of stress on lifespan. The strains, challenges, and obligations that accompany precocious achievement have the potential to catalyze physical and mental declines (McCann, 2001). A large body of evidence suggests that when we experience psychological or social stress, we may activate physiological responses where stress-related disease emerges predominantly from repetitive activation of our physiological systems that respond to acute physical emergencies, yet our coping mechanisms generally fail to switch it off (Sapolsky, 2004). In turn, stress has been found to have an influence on individuals' behaviours, such as both objective (e.g., life events) and subjective (e.g., distress) levels of stress that negatively affect physical activity and exercise participation (Stults-Kolehmainen & Sinha, 2014). Plenty of research has shown that stressful experiences may predispose us to disease, and ultimately premature death (e.g., Taylor, 1999); however, the role of stress on longevity is less known.

In sport, four general sources of social, psychological and physiological stress have been highlighted: i) frequent changes in arousal and anxiety related to performance demands (Hoar, 2007); ii) fear of physical injury and the subsequent recovery process (Wiese-Bjornstal, 2004); iii) normative and non-normative transitions out of sport and the robustness of their athletic identity (Taylor & Ogilvie, 2001), where athletes with stronger athletic identities appear more willing to engage in health compromising behaviours such as disordered eating (e.g., Sundgot-Borgen & Torstveit, 2010), and; iv) coping with pressure stemming from early fame/exposure (Schaller, 1997). This last example of athlete stress best relates to the precocity-longevity hypothesis; stressors that complement high performance competition at an early age may promote premature ill health, or in McCann's (2001) view, "the stresses that accompany a rapid drive to achievement peaks may eventually contribute to a shorter life" (p. 1430). The pressure

of precocious achievement, particularly in sports such as gymnastics where gymnasts' 'primes' occur at relatively much younger ages, has also been suggested as a predictor of self-destructive behaviour, where early fame's association with aversive self-consciousness may ultimately result in premature death (Schaller, 1997).

Personality type

The more stable and ingrained form of individual character is personality type, and the Type A-B personality dimension (Friedman & Rosenman, 1959; Friedman, 1969; Friedman & Rosenman, 1974) is the other proposed epidemiological explanation of the precocity-longevity hypothesis (although it can be argued that this is very similar to the stress hypothesis, as ultimately Type A personalities are generally predisposed to more stress). Type A personalities tend to be competitive, self-critical, ambitious, goal-oriented, impatient and aggressive; alternatively, Type B personalities are relaxed and non-competitive (McLeod, 2011). The seminal study by cardiologists Friedman and Rosenman (1959) found that Type A personalities had a higher risk of heart disease and high blood pressure relative to Type B personalities (although the external validity of this study has been questioned, due to its exclusively middle-aged male sample and a lack of control variables such as diet). Since this proposed correlation, research has provided substantial evidence that Type A personality (e.g., greater autonomic arousal and heightened competitive drive required to be an elite athlete), alongside stress, can cause premature disease and death (e.g., Taylor, 1999). As such, research appears to support these two epidemiological frameworks to explain the precocity-longevity effect, although much more work is necessary to appropriately address these purported explanations and their specific relationship to premature death.

Body – Part 2

The second part of this historical investigation of the development of the precocity-longevity hypothesis discusses studies that have attempted to control for the aforementioned confounding statistical artifacts. To conclude McCann's (2001) multi-study exploration into this phenomenon, his fifth and last study controlled for the life expectancy and selection artifacts using a subsample strategy on 22 samples of eminent persons where an initial significant precocity-longevity correlation was found. This strategy created an alternate 'recruitment' age in which all persons were assumed to have an equal life expectancy. As the recruitment age cut-off was arbitrary, it was crucial that the sample did not include any person who died before the age at which any other person reached their achievement peak (i.e., precocity). For example, those who died prematurely (e.g., 25 years) before others made a precocious contribution (e.g., 28 years) would be excluded from the subsample (which places importance in cautiously selecting the cut-off age to retain as many cases as possible). Nevertheless, McCann (2001) found support for the precocity-longevity hypothesis in 21 of the 22 restricted samples after controlling for the statistical artifacts, suggesting through his comprehensive five-study investigation that enough reasonable evidence exists that precocity may adversely affect lifespan across a variety of domains. Further, his follow-up study on state governors, which also controlled for statistical artifacts (i.e., restrictive subsample strategy), also found support for the precocity-longevity hypothesis; relatively younger ages at election resulted in premature death for governors (McCann, 2003).

The last study on the precocity-longevity hypothesis, prior to our two investigations, was a first exploration into its effects in sport. Abel and Kruger (2007) examined 3,760 deceased MLB players who debuted between 1900 and 1935. Precocity was defined as age at debut in MLB, which was a considerably narrower definition of precocity relative to other domains where

the variability in age of achievement was much greater (e.g., elected ages of state governors; McCann, 2003). Conversely, narrower measures of precocity may be inherent to high performance sport; athletic skills needed for peak performance in many sports such as speed, flexibility, and endurance are most prominent during a specific time period (e.g., Schulz & Curnow, 1988 suggested between 19 and 27 years of age). As such, defining precocity as age of entry into elite sport will typically restrict the age range of achievement, which can i) consequently play a role in diminishing the potential of finding a significant lifespan difference between precocious and non-precocious individuals, or ii) constitute impressive support for the hypothesis if a lifespan difference is found.

Similar to previous methodologies, Abel and Kruger (2007) used a restrictive subsample strategy to control for the life expectancy and selection bias artifacts, using 28 years as the cut-off age. They found that every year a player debuted before the average age of 23.6 years (SD = 2.3 years), their lifespan was decreased by nearly a quarter of a year (0.24 years). A secondary analysis comparing the debut ages of Hall of Fame inductees (the pinnacle of sporting achievement in MLB) to non-inductees born in the same year also revealed support for the precocity-longevity hypothesis. Thus, similar to McCann's (2001) fourth study examining multiple achievement peaks (i.e., PhD attainment and later life achievement), Abel and Kruger (2007) indicated that precocious achievement of MLB players (i.e., younger age at debut and Hall of Fame induction) significantly reduced their lifespan.

Conclusion

To date, the precocity-longevity hypothesis remains limited in its generalizability. The consistent trend towards early achievement cultivating premature death is alarming, and there is incomplete understanding of the mechanisms and risk factors explaining this link. A majority of

the previous research has focused on eminence in politics or academics; limited attention has been given to *athlete* mortality. Given the relatively more physically strenuous occupation of being an elite athlete, more research is needed on this cohort of the population to test whether precocious achievement in athletics supersedes the numerous other socially-constructed factors that promote greater longevity (e.g., physical activity, socio-economic status, etc.).

Importantly, two issues have been highlighted that apply directly to how we structured our investigations: i) previous methodologies were fallible; a sample that excludes high achievers who are still alive may result in a skewed average age that falsely supports the precocity-longevity hypothesis, and ii) defining precocity as ‘age of entry’ into elite sport lacks the necessary variability for a typical analysis of age at precocious achievement. Below, **Study 1** (pg. 74) examined the precocity-longevity hypothesis in *living* and deceased Canadian-born NHL players through Kaplan-Meier and Cox regression survival analyses, using a subsample strategy of including those who debuted only between 1917 and 1986 to limit the influence of players who were still living (i.e., censored cases), and excluded players who died within five years of debut and players whose date of death was unknown. A more rigorous methodological approach was undertaken in **Study 2** (pg. 80) which analyzed i) only deceased and ii) deceased and living NBA/ABA players, defining precocity in three different ways: a) age upon entering professional sport, b) age at first All-Star game, and c) age at first All-League selection.

While the priority of our two studies was to establish whether the precocity-longevity hypothesis is generalizable to professional sport (and in two sports with different sport-related injury profiles), more empirical evidence is needed to support the proposed explanations of the precocity-longevity hypothesis. Two methods to empirically test the aforementioned epidemiology explanations within sport are proposed:

i) The *inference method*: Stress levels (e.g., emotional readiness, pressure, arousal, etc.) and personality types are measured in current and recently retired elite athletes within the same sport. The scores would then be contextualized by age of achievement (e.g., debut age) and inferences would be made to the deceased cohort being analyzed. This method estimates the different stressors deceased precocious and non-precocious athletes may have faced that contributed to their death, although these inferences would likely be confounded by decade of play considering the evolving environment of professional sport.

ii) The *longitudinal method*: Stress levels and personality types of current and former elite athletes are also measured; however, this same group of participants will be analyzed longitudinally. While this method may be limited by its impracticality considering the event being measured is death, with a large pool of respondents, mixed-methods analyses on subsamples of recently deceased athletes would provide more accurate explanations of the risk factors that underpin precocity and longevity.

As explanations for why early achievement may negatively impact lifespan remain theoretical, methods such as these may be necessary to investigate whether different sources of stress are experienced and internalized by precocious and non-precocious individuals. From a broader perspective, the fundamental theory behind the precocity-longevity hypothesis can be questioned. Simonton (1988) noted that achievement is very much an attribution made by others. Further, perceptions of precociousness may be individualistic (e.g., receiving a bonus; how does it relate to “ordinary” persons?). Thus, current knowledge imposes a constraint on the range of possible explanations, making it exceedingly important to be cautious when disseminating and generalizing this phenomenon to the general public.

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The precocity-longevity hypothesis re-examined: Does career start age in Canadian National Hockey League players influence length of lifespan?

*Lemez, S., Wattie, N., Ardern, C. I., & Baker, J. (2014). The precocity-longevity hypothesis re-examined: Does career start age in Canadian National Hockey League players influence length of lifespan? *Journal of Sports Science and Medicine*, 13, 969–970.

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The precocity-longevity hypothesis re-examined: Does career start age in Canadian National Hockey League players influence length of lifespan?

Dear Editor-in-chief,

Available data on elite athletes suggests they have longer lifespans than the general population (Teramoto and Bungum, 2010); however, this relationship is likely more nuanced than previously considered. For example, the precocity-longevity hypothesis proposed by McCann (2001) asserts that factors related to early career achievement (e.g., debut age) may cultivate early death. Stress and personality type (e.g., McCann, 2001) have been the predominant explanations because of their purported association with disease states. To date, only Abel and Kruger (2007) have examined this hypothesis in sport, concluding that every year a Major League Baseball player debuted before the mean age of 23.6 years was associated with a decreased lifespan of 0.24 years. Little is known about the extent of this phenomenon among different athlete populations.

This study explored the precocity-longevity hypothesis among Canadian-born National Hockey League (NHL) players. Based on the link between high career achievement and early mortality identified in previous research, we hypothesized that precocious NHL players would have shorter lifespans than those debuting at later ages. There have been 4,583 Canadian-born NHL players who debuted between 1917 and 2010 (Quant Hockey, 2014); however, we only analyzed those who debuted between 1917 and 1986 ($n = 2,971$) to limit the influence of players who were still living (i.e., censored cases). Additionally, we restricted our sample to Canadian players to limit the influence of different developmental systems and socio-cultural factors that might affect longevity. Data were collected through the official website of the hockey hall of

fame (www.hhof.com), and a random sample (~10%) was cross-referenced within *Total Hockey* (Diamond et al., 1998) and www.hockey-reference.com which confirmed complete agreement.

Kaplan-Meier and Cox regression survival analyses were used. Hazard ratios (HR) considered whether the predictor variable (debut age in the NHL) significantly influenced the event (age at death), while controlling for the potential confounders of position (i.e., Center, Wing, Defense, or Goalie) and years played. To limit the effect of statistical artifacts (see McCann, 2001), we excluded players who died within five years of debut and players whose date of death was unknown (~40 cases). All data were evaluated at the $p \leq .05$ level of significance (95% confidence interval; CI), using SPSS 21 (IBM Corp., 2012).

Overall, 910 out of 2,971 Canadian-born NHL players were deceased as of 1986 (30.62%). The mean age of entry into the NHL by decade were as follows: 25.18 (1917–1926), 22.59 (1927–1936), 22.22 (1937–1946), 21.09 (1947–1956), 21.74 (1957–1966), 22.06 (1967–1976), and 20.84 years (1977–1986). Descriptively, 537 players who debuted *above* the median age, respective to their decade of play, were deceased (median life expectancy estimate: 81 years), compared to 373 players who debuted below the median age of entry (82 years). Kaplan-Meier analysis found no significant difference in survival distribution [$\chi^2 (1) = 2.35, p = 0.12$; see Figure 2.1]. In Cox regression models adjusted for player position and years played, no significant relationship between precocity and longevity was found (HR: 0.91, 95% CI: 0.79–1.05).

Our results do not support the precocity-longevity relationship in Canadian-born NHL players, raising the question of its generalizability to sport populations. In addition, our descriptive finding that early high achievers had a lifespan *advantage*, contradicts even the trend of previous precocity-longevity findings. This discrepancy may reflect methodological

differences; in particular, our sample contained both deceased and still-living athletes, while the samples of previous studies have only included deceased high achievers. A sample that excludes non-deceased high achievers may result in a greater age differential that artificially supports the precocity-longevity relationship.

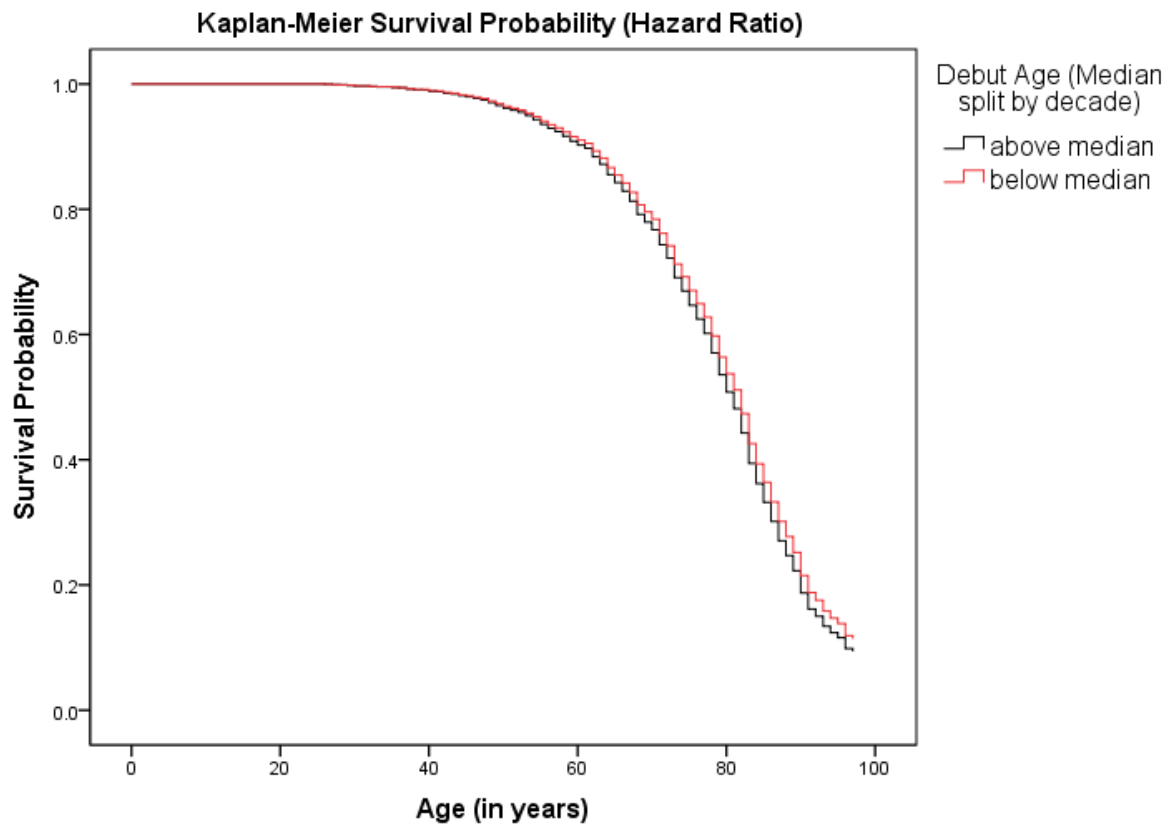
Our results may also reflect some of the complications that arise from the heterogeneity and unique demands of sport. For example, ice-hockey is likely more physically demanding than previously studied professions. In addition, sport typically has a narrower entry age range compared to other domains of eminence (e.g., McCann, 2001). As most skills needed for peak athletic performance are age-specific, finding a significant difference in players whose entry ages into professional sport are relatively narrow would indicate impressive support for the hypothesis (Abel and Kruger, 2007). Lifespans have tremendous variation and are influenced by many underlying etiological factors (e.g., genetics); therefore, it may be challenging to find widespread support for this phenomenon considering the age restrictions imposed by different domains of eminence.

To more fully understand this phenomena, moderators such as sport-specific energy system demands (i.e., aerobic, anaerobic, mixed) may need to be considered. In addition, an important limitation of this study is that cause-of-death data were not considered. Since age-of-entry is relatively narrow in range, future research in sport may also benefit from examining precocity with respect to different accomplishments, such as age at first all-star selection. To date, analyses of the relationship between early high achievement and premature death are sparse, and largely limited to studies of a theoretical and descriptive nature. Given the limitations inherent to previous methodologies and the complexity of athlete populations, the precocity-longevity hypothesis should be interpreted cautiously.

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Figure 2.1: Survival probability of Canadian NHL players



Does early career achievement lead to earlier death? Assessment of the precocity-longevity effect in professional basketball players*

*This article is currently under review for publication in *Frontiers in Public Health*, section *Epidemiology*

Study 2 Overview

Objectives: To examine the precocity-longevity (P-L) effect in North American professional basketball players who debuted between 1946 and 1979, and to determine whether playing position and decade of play influenced the relationship between age of career achievements and lifespan. **Methods:** 1852 players were evaluated from a recognized sports archive (i.e., sports-reference.com), which provided information on date of birth, death, and career debut, playing position, and indicators of achievement (i.e., All-Star team and/or All-League team selection). Athletes were categorized as above or below the median age of professional debut, and median age of selection to first All-Star team and/or All-League team. Analyses of deceased players ($n = 598$) were comprised of bivariate correlations between age of achievement (age of debut, age of first All-Star game and age of first All-League team selection) and age of death, and t-tests to compared the average age of death of early and late achievers ($p < .05$). Survival analyses, using the entire sample (living and deceased players), compared the lifespans between those who debuted above and below the median age of achievement for each indicator of achievement. **Results:** Only the correlation between age of professional debut and age of death ($r = 0.33, p < .001$) and age of first all-star game and age of death ($r = .29, p < .05$) and the t-test comparing the average death age of early (66.4 y) and later (69.3 y) debut age groups ($p = .01$), reached statistical significance. Although, survival analyses demonstrated a trend for lower risk of death for early achievers, with one exception (i.e., age of debut) this trend was not statistically significant. **Conclusions:** Results did not support the P-L hypothesis, suggesting that contextual factors, such as the physical demands of a sport, and measurement methodologies, may influence support for the proposed hypothesis in sport.

Does early career achievement lead to earlier death? Assessment of the precocity-longevity effect in professional basketball players

The precocity-longevity (P-L) hypothesis asserts that those who experience noteworthy high achievements earlier in life have a shorter lifespan than those who attain similar notable career milestones later in life. This hypothesis was first formally described by McCann (2001) in a study of high achievers in politics (presidents and prime ministers), law, entertainment, creative pursuits and academia, which showed evidence that the younger a person is when they achieve a notable peak in achievement (or a landmark contribution) the younger they tend to die. For example, Nobel Peace prize recipients who received the award above the median age of 64.8 years lived 13.5 years longer than those who received the prize before this age [$p < .01$; (McCann, 2001)].

Two explanations have been at the forefront of discussion (and have existed from the hypothesis' inception, albeit as an undertone in the earlier studies): stress and personality type. First, McCann (2001) suggested that the stress, challenges, and obligations that accompany precocious achievement have the potential to be a catalyst for poor health. McCann (2001) further highlighted that the *ascension* to career success may play an important role: "the stresses that accompany a rapid drive to achievement peaks may eventually contribute to a shorter life" (p. 1430). Research does support that stressful experiences predispose humans to disease, which can ultimately lead to premature death (e.g., Taylor, 1999), where repetitive stress may activate physiological responses that cause stress-related diseases to emerge (Sapolsky, 2004).

Personality type, the more stable and ingrained form of individual character, is the other proposed epidemiological explanation of the P-L effect (McCann, 2001). Type A personalities tend to be competitive, self-critical, ambitious, goal-oriented, impatient, and aggressive; alternatively, Type B personalities are relaxed and non-competitive (McLeod, 2011). The

seminal study by cardiologists Freidman and Rosenman (1959) found that Type A personalities have a higher risk of heart disease and high blood pressure relative to Type B personalities. However, contemporary research suggests a more complicated relationship between components of Type A personality (i.e., hostility and anger) and disease (e.g., Myrtek, 2001). Nevertheless, previous studies on the P-L relationship may have examined samples that likely fall in some components of the Type A personality category, such as professional baseball players (Abel and Kruger, 2007) and eminent persons such as presidents, prime ministers, and monarchs (McCann, 2001), making personality type seemingly a valid explanatory theory.

Although etiological studies of the P-L effect will be important, it has been suggested that a more immediate objective should be to determine the existence and generalizability of the phenomenon (McCann, 2004). To date, however, only a few studies have investigated the existence and generalizability of the P-L effect in the athletic disciplines. Abel and Kruger's (2007) investigation of deceased Major League Baseball (MLB) players noted that when precocity was defined as age in which athletes debut professionally, there was a significant relationship with lifespan; for every year a player debuted before the average age of 23.6 years ($SD = 2.3$), their lifespan decreased by 0.24 years (2007). Further, correlations between debut age and longevity were higher in Hall of Fame inductees relative to non-inductees (0.48 versus 0.09, respectively), which further supported the proposition that precocious achievement may have negative effects on longevity. Alternatively, a recent analysis by Lemez and colleagues (Lemez, Wattie, Ardern, & Baker, 2014) on Canadian professional ice hockey players did not demonstrate a clear P-L effect. The authors speculated that the positive relationship between cardiovascular fitness and reduced all-cause mortality may explain the lack of an effect among ice hockey players compared to baseball and non-athletic eminent achievers. However, with only

two studies to-date on high achieving athlete populations there is a clear need to explore the P-L effect in other athlete samples.

In addition to there being a limited number of studies on high performing athletic populations, there are also methodological challenges inherent to research on the P-L effect. First, there are some epidemiological and methodological influences that may affect the P-L effect: life expectancy and selection artifacts. The life expectancy artifact describes the potential that younger death age among high achievers may result from the fact that younger age is associated with shorter life expectancy (McCann, 2001). There is also the possibility that early death (and hence early achievement) simply permits a person to enter a study sample. This ‘selection artifact’ (Simonton, 1994) means that the absence of still-living early achievers may skew effects in favour of a P-L effect. Both of these artifacts describe the inherent potential for bias when studying a sample of *only* deceased eminent achievers, and may increase the likelihood of Type I error (i.e., identifying a P-L effect when one does not exist).

There may also be challenges to studying the P-L effect as a result of how ‘precocity of achievement’ is operationalized. Both Abel and Kruger (2007) and Lemez et al. (2014) defined precocious achievement in athlete samples as the age at which athletes entered professional sport. However, using age of professional sport debut as a measure of achievement may be impacted by the fact that the age range for professional sport debut is typically quite narrow (McCann, 2001). If this range is sufficiently narrow it would be difficult to find clear support for the P-L effect as the age difference between ‘early achiever’ and ‘late achiever’ would be quite small. Lemez and colleagues (2014) suggested that one way to circumvent these issues would be to use an alternate marker of accomplishment more independent of age and more indicative of exceptional achievement, such as age at first participation in an “All-Star” game.

Taken together, it is clear that there is a need to study the generalizability of the P-L effect in different domains (e.g., sport vs. non-sport), and there is a need to start reconciling the methodological challenges inherent to studying this phenomenon. Based on the two studies of the P-L effect among eminent athletes, this study has two aims: 1. To explore the generalizability of the P-L effect among high achievers in a sample of athletes not previously studied (i.e., North American professional basketball players); and 2. To address some of the methodological challenges that are important to consider in this area of research. To that end, the current study aimed to explore the P-L effect using diverse approaches. First, we replicated previous methodologies for analyzing the effect using a sample of only deceased athletes in order to compare this sample to previous research. Second, the unique approach of analyzing an inclusive sample of deceased *and* living athletes allowed more ecologically valid analyses to be performed. Based on previous P-L research on athlete and non-athlete samples, we hypothesized that athletes who had younger ages of notable achievement would have shorter lifespans than those who had notable achievement at older ages.

Methods

Sample and variables

This study did not involve any human participants or animals, and therefore adhered to recognized ethical standards and national/international laws (Harriss & Atkinson, 2013). Data were collected for *all* players who had played professional basketball from 1946 to 2012 (N = 4015) from a recognized sports archive of aggregated publically available athlete records (i.e., sports-reference.com). During this period two professional leagues existed, the National Basketball Association (NBA) and the American Basketball Association (ABA). A random sample of the data (10%) was also cross referenced with an official NBA encyclopaedia

(Shouler, Ryan, Smith, Koppett, & Bellotti, 2003) and showed complete consistency. We censored all players who debuted from 1980 to 2012 because the majority (98.4%) of that cohort was still living. This resulted in a final sample size of 1852. For each player, date of birth, career milestones (i.e., first year of professional debut, last year of play, date of first All-Star game, date of first All-League selection), playing position (i.e., Guard, Center or Forward), and date of death were collected. The All-Star game is an annual mid-season exhibition game where 12 players from each conference (i.e., Eastern and Western) who are playing at a notably high-level are selected by fans (starters) and coaches (reserves). Similarly, an All-League selection honours the best players *following* a season to two teams from 1946 to 1988 (N = 10), which are selected by a panel of sportswriters and broadcasters throughout Canada and the United States (e.g., NBA.com). From the above data, a number of additional variables were derived for the current study, including: i) length of playing career (in seasons played), ii) age at the start of playing career, iii) age at first All-Star game, iv) age at first All-League team selection, v) age at time of death (in years) and vi) age as of January 2013 (days) for living and former athletes. Although age-related variables were computed and analysed in ‘days’, results are presented in years for ease of understanding. Athletes were also coded as either deceased or living for the purpose of analyses.

Precocity of achievement was defined in three different ways. First, we replicated Abel and Kruger’s (2007) definition of achievement as ‘age upon entering professional sport.’ In addition, in order to have a measure of achievement more independent of age, and more indicative of eminent achievement in this population, two other indicators of achievement were included: ‘age at first All-Star game’ and ‘age at first All-League selection’. For each indicator of achievement, variables were computed using a player’s *first* notable achievement. This was

necessary because the ABA only existed from 1967 to 1976, and therefore some players debuted in the ABA and transferred to the NBA (and vice versa). For example, if a player debuted or was nominated to an All-Star game in the ABA then later transferred to the NBA the achievement values were derived from their ABA. Consistent with previous research, we explored career precocity by coding athletes as debuting either below or above the median ‘age of achievement’ for each indicator of achievement. The median ages of achievement were calculated separately for each decade of debut, since these values fluctuate (this was particularly important for debut age, which has declined in more recent decades).

Analyses – Part 1 (Deceased Sample)

The purpose of our first set of analyses was to replicate the methods of previous studies in an unstudied population (i.e., professional basketball players) and with additional indicators of achievement. Specifically, these analyses used a sub-sample of *only deceased* players who had debuted prior to 1980 (n = 598), similar to previous research on eminent athlete and non-athlete samples (Abel and Kruger, 2007; McCann, 2001). As such, all coding of the achievement measures in this portion of the analyses were based on the values (i.e., median ages of achievement) obtained from the deceased sub-sample of NBA players.

We described the study sample by calculating the median age of death, the sample composition according to playing position (Center, Forward, Guard), and the proportion of deceased athletes who had participated in an All-Star game, and who had been selected to an All-League team.

In accordance with previous research (see McCann, 2001), each of the three measures of achievement were correlated, and partially correlated (controlling for decade of debut), with age

at death ($p < .05$). Similarly, t-tests compared the mean ages of death for those who were above and below the median ages of achievement for each of the three measures of achievement.

Two final analyses were included in Part 1. We also categorized athletes as above (later death) or below (early death) the median age of death (where each median death age was decade specific), and categorized athletes as either ‘eminent’ or ‘not eminent’ based on whether or not they had been selected as an All-Star or to an All-League team (respectively). Using these categorizations, two 2x2 chi-squares were run with a $p < .05$ significance criteria: All-Star Eminence (yes/no) x Death age (early/later); All-League Team Eminence (yes/no) x Death age (early/later). Similar to Abel and Kruger’s (2007) distinction between Hall of Fame and non-Hall of Fame athletes, these analyses sought to distinguish any potential differences in longevity associated with *degree* of eminence.

Analyses – Part 2 (Alive or Deceased Sample)

The second set of analyses for this study aimed to address the possibility that a P-L effect observed in a sample comprised of deceased eminent achievers may result from still-living eminent achievers not having entered the study sample (i.e., the selection artifact: Simonton, 1994). Therefore, these analyses used a sample of deceased and still-living players who had debuted prior to 1980 ($n = 1852$) with the aim of testing whether or not those with earlier death age are in fact more precocious achievers than still living athletes. As with the first portion of analyses, achievement was defined as i) age of professional debut, ii) age at first All-Star game, and (iii) age at first All-League team selection. However, when coding athletes as above or below the median age of achievement (for each indicator of achievement), the values of the *entire* sample (living and deceased) of athletes were used for these analyses (with median achievement values calculated separately for each decade of play and pertinent league).

We described the study sample by providing the proportions of each playing position (Center, Forward, Guard), and the proportion of the sample who had participated in an All-Star game, and who had been selected to an All-League team. We also describe the sample by listing the median age (in days) of achievement for the three indicators of achievement (stratified by decade) separately for deceased and still-living players.

Three Kaplan-Meier (KM) analyses were used to visualize the univariate relationship between each career achievement (i.e., precocity) and longevity ($p < .05$). Cox proportional hazards regression was performed to assess the influence of career precocity on lifespan, after adjusting for playing position and decade of career debut. Hazard ratios (HR) and 95% confidence intervals (95% CI) were used to estimate the relationship between precocity and longevity, after adjusting for covariates.

Results – Part 1 (Deceased Sample)

The median age of death among the deceased players was 70.3 years (standard deviation ± 14.4 years). Overall, the majority of this sample was comprised of Guards (42.6%) and Forwards (42.4%), while Centers constituted only 15.1% of the sample. The majority of athletes participated exclusively in the NBA (88%), while smaller proportions played exclusively in the ABA (7.4%) or played in both leagues (4.7%). Of the 598 deceased athletes, 11.5% had participated in an All-Star game, and 6.3% had been selected to an All-League team.

Table 2.1 presents the correlations between each of the achievement measures and age of death, as well as the results of the independent t-tests. Both age of professional debut and age of first all-star game demonstrated statistically significant positive correlations with age of death. The relationship between age of professional debut and age of death was a medium size correlation ($r = 0.33$), but decreased to a small correlation ($r = 0.11$) when decade of professional

debut was adjusted for. The correlation between age of first all-star game and age of death was also medium-sized ($r = .29$), but this correlation was not statistically significant when decade of debut was adjusted for. Age at first All-League selection was not significantly correlated with age of death (regardless of whether decade of debut was adjusted for). T-tests were used to compare the average age of death between earlier and later achievers (see Table 2.2). Although there was a trend for early achievers to have a younger mean age of death for each of the indicators of achievement, the difference in death age was only statistically significant between those with earlier and later ages of professional debut ($p = .01$).

Neither the chi-square test for All-Star Eminence (yes/no) x Death age (above vs. below median; $\chi^2(1) = .13, p = .72$), nor the chi-square test for All-League team Eminence (yes/no) x Death age (above vs. below median; $\chi^2(1) = 1.07, p = .30$) provided any evidence for the P-L hypothesis.

Results – Part 2 (Alive or Deceased Sample)

Of the 1852 players alive or deceased, the majority of the sample was comprised of Guards (42.4%) and Forwards (42.5%), while Centers constituted 15.1% of the sample. The majority of athletes participated exclusively in the NBA (73.4%), while smaller proportions played exclusively in the ABA (15.5%) or played in both leagues (11.1%). Overall, 14.7% had participated in an All-Star game, and 6.7% had been selected to an All-League team. Descriptive comparisons of median age of achievement between living and deceased players revealed that living athletes had younger median ages of achievement on all *Age of Professional Debut* comparisons, and 5 out of 11 comparisons for *Age at first All-Star* and *All-League Team selection* (see Table 2.3).

Age of Professional Debut. The median lifespan for early achievers was higher than it was for late-achievers (82.6 y v 81.0 y), although this trend was not statistically significant [$\chi^2(1, N = 1852) = 3.31, p = .07$]. In a subsequent Cox regression, which adjusted for playing position and decade of playing debut (see Table 2.4), career precocity was associated with a lower risk of death (HR: 0.84, 95% CI: 0.71-0.98). No main effects were observed for decade of debut. However, compared to Guards, Forwards had a higher risk of death (HR: 1.21, 95% CI: 1.02-1.45), but no difference in risk of death among Centers. No two-way interactions were observed.

Age at first All-Star game. Although the median lifespan for early achievers was higher than it was for late achievers (84.1 y v 80.7 y) in the current sample, this trend was not statistically significant [$\chi^2(1, N = 271) = 0.05, p = .83$]. In a subsequent Cox regression (see Table 2.4), which adjusted for playing position and decade of playing debut, career precocity was not independently associated with a lower risk of death. Moreover, no main effects were observed for decade of debut or playing position, and no interactions were observed.

Age at first All-League Team selection. The median lifespan for early achievers was lower than it was for later achievers (82.4 y v 84.1 y), although this trend was not statistically significant [$\chi^2(1, N = 119) = 0.10, p = .75$]. When decade of debut and playing position were adjusted for in a Cox regression (see Table 2.4), mortality risk did not differ between early and later achievers. No main effects were observed for decade of debut or playing position, and no interactions were observed.

Discussion

This study examined the relationship between precocity of achievement and longevity in elite professional basketball players. Previous work by McCann (2001) and Abel and Kruger (2007) observed that earlier achievement is associated with a shorter lifespan across different

domains, including sport. However, the current results present a more complicated picture of the P-L effect. Our interpretation is that the results from the first portion of analyses do not support the P-L hypothesis. First, the only statistically significant correlation and t-test were for the age of debut indicator of achievement. The weight of these results needs to be considered alongside the limitations associated with the age of professional debut indicator of achievement. This measure of achievement may be less than ideal because it is strongly dependent on age, and simply entering professional basketball may not truly describe eminence/highest achievement in professional basketball.

Second, none of the Part 1 analyses on the age at first All-Star game and age at first All-League selection reached statistical significance. The results of the chi-square tests for these two indicators of achievement also did not support the P-L hypothesis. In addition, there is the need to weigh the results of the first series of analyses against the results of Part 2.

The second series of analyses aimed to expand on the methods used in the majority of P-L research. It had previously been suggested that younger death age among high achievers results from lower life expectancies associated with younger age (Taylor, 1999), and that early death may simply permit a person to enter a study sample (i.e., the selection artifact: Simonton, 1994). The inclusion of still living and deceased athletes and the subsequent results of the survival analyses (Part 2) suggest that the concerns related to the above artifacts are well-founded in the current population of high achievers. Not only did the results of the survival analyses not support the P-L hypothesis, the trends for the three indicators of eminent achievement were opposite to those in Part 1 and suggest a *lower* risk of death for precocious achievers (although only the age of debut achievement indicator reached statistical significance). There is of course a notable limitation to using survival analyses in instances where the exposure

event (age of achievement) is itself a potential confounder of the outcome, which cannot be controlled for. In this case, it is not possible to control for the fact that precocious achievers are followed for a greater period of time than late achievers. The results of the current study should be weighed against such limitations (see Hanley & Foster, 2014). However, in light of the notable limitations to the methods presented in Part 1, we argue that survival analyses perhaps represents the lesser of two evils, and that for the time being a triangulation of multiple methods, which includes deceased-only and still-living athletes, may be prudent. It may be worth repeating, however, that one of the biases identified earlier is that precocious achievement and younger age is associated with greater risk of early mortality (McCann, 2001; Taylor, 1999). As such, if the limitations inherent to survival analyses biased our findings, they theoretically should have reflected biased support *for* the precocity-longevity hypothesis. However, the results of the survival analyses do not support the precocity longevity effect. We believe that these trends stem from the differences in age of achievement between living and deceased athletes.

The discrepancy between the trends observed in Part 1 and Part 2 appear to be related to the different median age of achievements between still-living and deceased athletes (Table 2.3). Descriptive trends suggests that in many cases the median achievement age of *deceased* athletes does not accurately reflect the median achievement age for the overall population of athletes. In some cases it appears that the median age of achievement of the still-living athletes is actually younger than that of the deceased-only sub-sample, especially with regard to the ‘age of debut’ measure of achievement. These descriptive differences in median age of achievement suggest there is a precocity-longevity effect in the current sample, but unlike previous research precocity of achievement appears to be related to *longer* lifespan in this population. The fact that the median age of achievement differs for deceased and still-living athletes in many cohorts (i.e.,

decade of entry and league) of athletes suggests that this trend is systematic, not random, and that the Part 2 analyses are a more accurate reflection of achievement and mortality trends within this population. It also introduces the possibility of an additional bias; that the median age of achievement in previous studies of deceased-only samples may not accurately reflect the true median value for the entire population of eminent achievers. As Hanley and Foster (2014) suggested, “Theories such as the just-cited precocity-longevity hypothesis are seductive, and have a certain plausibility. But some of this may be a result of the framing” (p. 8). This seems to be the case for our data; as such, we conclude that the trends observed in Part 1 appear to be less representative of achievement trends than those observed in Part 2.

There are many reasons that the P-L effect does not exist in the current sample of high achievers. As Lemez et al. (2014) noted, there are notable differences between previous research and the current investigation that might explain these discrepancies. First, given the importance of physical fitness for the reduction of all-cause mortality (Marijon et al., 2013), it is possible that the high levels of physical fitness required for elite performance in basketball players compared with the domains examined by McCann (2001) and the baseball players examined by Abel and Kruger (2007) buffered the effect of precocity on longevity. Moreover, beginning a professional career earlier and having the potential for longer exposure to financial rewards, health care, and fitness related health benefits may explain the finding that early achievers have longer lifespans in this sample. A related explanation is that players who eventually make it to play professional basketball, regardless of their age of debut, have a superior genotype for physical fitness than those in other domains, which negates any relationship between early achievement and length of lifespan. The ‘Healthy Worker Effect’ describes a form of selection bias wherein inclusion into a study sample reflects a health-advantage compared to those not

included (see Rothman, 2002). As such, the Part 1 analyses of deceased-only athletes, which appear to support the trend for the P-L hypothesis, may reflect a sort of ‘inverse-healthy worker effect.’

Although the results of the current study add to a relatively limited evidence base regarding predictors of early mortality in elite and professional sport, the current study did not provide any insights on cause-specific mortality. Future investigations that incorporate cause-specific mortality, and any subsequent links to the proposed underlying risk factors of the P-L effect, such as stress and personality, may provide further insight into the long-term risks and benefits of participation in elite sport. The historical nature of this sample, and the possibility of generational differences in mortality trends and life expectancy, also reinforce the need to incorporate cause-specific mortality in future research. It will be important to consider these factors when exploring the generalizability of the current findings in other samples of eminent athletes.

In summary, the results of the current study suggest it is unlikely that the P-L effect exists in NBA athletes. Importantly, the current study also raises a methodological consideration regarding the estimates of average precocity values, and whether or not the average age of precocious achievement among deceased eminent achievers is an accurate reflection of the entire eminent population. An average age of achievement among still-living eminent achievers that is significantly different than that of deceased eminent achievers could skew the results (as was likely the case for our data). Going forward it will be important to consider how the physical demands of a sport, the health and financial benefits associated participation in high performance sport, and measurement methodologies, may influence support for the precocity-longevity hypothesis in sport. Given the significant dedication toward talent identification and

development at early ages in many sports worldwide, the potential relationship between the milestones of athlete development (e.g., age of first participation in organized sport) and athlete health outcomes such as overuse injuries are important to consider.

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Table 2.1: Bivariate and partial correlations between age of achievement measures and age of death

Test	Measure of Achievement	Death Age	<i>p</i> value	Death Age	<i>p</i> value
Correlations	(Age)	(<i>r</i>)		(<i>r</i>)†	
	Age Pro Debut (n = 598)	0.33	< .001	0.11	< .01
	Age 1 st All-Star Game Nomination (n = 69)	0.29	< .05	0.22	.07
	Age 1 st All League Team Nomination (n = 38)	0.10	.54	0.27	.10

Table 2.2: T-test comparisons of the mean death age between earlier and later achievers

Test	Measure of Achievement	Mean Death Age (years)		t (df)	<i>p</i> value
T-Tests	Above vs Below median Achievement age††	Earlier Achiever M(SD)	Later Achiever M(SD)		
	Age of Pro Debut (n = 598)	<i>M</i> = 66.4 (±15.2)	<i>M</i> = 69.3 (±14.3)	t (596) = 2.34	.02
	Age 1 st All-Star Game Nomination (n = 69)	<i>M</i> = 62.2 (±16.7)	<i>M</i> = 67.7 (±9.6)	t (67) = 1.65	.10
	Age 1 st All League Team Nomination (n = 38)	<i>M</i> = 63.1 (±17.4)	<i>M</i> = 69.1 (±10.1)	t (36) = 1.27	.21

†Partial correlation controlling for decade of entry to professional basketball. ††Age of median achievement was calculated for each decade, and median splits were respective to athletes decade of entry to professional sport; *M* = mean; SD = standard deviation. All of the above analyses were repeated using a restricted inclusion criteria that ensured no athlete had died before all the individuals in the sample had accomplished their measure of career achievement.

Table 2.3: Presents the median age (in days) of achievement for age of NBA debut, age of first All-Star game and age of first All-NBA selection, stratified by decade of professional debut, league (if applicable) and mortality status, and the difference (Δ) between the median values for alive and deceased athletes

Indicator of Achievement		Median Age of Achievement (days)		Δ Alive - Deceased (NBA ABA)
		Alive (NBA ABA)	Deceased (NBA ABA)	
Debut Age	1940s	8942 n/a	9351 n/a	-409 n/a
	1950s	8350 n/a	8519 n/a	-169 n/a
	1960s	8309 8521	8561 8734	-252 -213
	1970s	8265 8250	8348 8299	-83 -49
All-League	1940s	9893 n/a	9168 n/a	725 n/a
	1950s	9671 n/a	8883 n/a	788 n/a
	1960s	9107 9303	9923 9258	-816 45
	1970s	9002 8344	9538 n/a	-446 n/a
All-Star	1940s	9091 n/a	9965 n/a	-874 n/a
	1950s	8947 n/a	8880 n/a	67 n/a
	1960s	9201 9277	9528 9077	-327 200
	1970s	8909 8604	9255 8444	-291 160

Table 2.4: Hazard ratios for age of achievement, decade of debut and playing position from Cox regressions for age of debut, age of first All-Star game, and age of first All-League team indicators of achievement

Measure of Achievement	Variables		Hazard Risk	
			HR _{adjusted}	95% CI
Age of Debut	Age of achievement	Early achievement	0.84	0.71-0.98
		Late achievement	-	-
	Decade of Debut	1940s	1.01	0.81-1.51
		1950s	0.95	0.69-1.33
		1960s	0.87	0.63-1.20
		1970s	-	-
	Playing Position	Centers	1.20	0.94-1.52
		Forwards	1.21	1.02-1.45
		Guards	-	-
	Age at first All-Star game	Age of achievement	Early achievement	0.82
Late achievement			-	-
Decade of Debut		1940s	0.96	0.36-2.55
		1950s	1.14	0.45-2.85
		1960s	0.81	0.34-1.91
		1970s	-	-
Playing Position		Centers	1.40	0.71-2.75
		Forwards	1.64	0.95-2.83
		Guards	-	-
Age at first All-League team		Age of achievement	Early achievement	0.80
	Late achievement		-	-
	Decade of Debut	1940s	0.68	0.21-2.19
		1950s	0.57	0.18-1.87
		1960s	0.55	0.17-1.74
		1970s	-	-
	Playing Position	Centers	1.29	0.50-3.34
		Forwards	1.38	0.66-2.88
		Guards	-	-

HR = Hazard ratio; CI = confidence interval; Bold font = statistically significant HR.

CHAPTER FOUR: EARLY DEATH IN ACTIVE PROFESSIONAL ATHLETES

Early death in active professional athletes: Trends and causes

Lemez, S., Wattie, N., & Baker, J. (2015). Early death in active professional athletes: Trends and causes. *Scandinavian Journal of Medicine & Science in Sports*, 26, 544–549.

Chapter Four Overview

The objective of the study was to examine mortality trends and causes of death among professional athletes from the four major sports in North America who died during their playing careers. 205 deceased athletes who were registered as active when they died from the National Basketball Association (NBA), National Football League (NFL), National Hockey League (NHL), and Major League Baseball (MLB) were examined. Results were compared with the Canadian and U.S. general population. The leading causes of death in players reflected the leading causes of death in the Canadian and U.S. general population (i.e., car accidents). Descriptively, NFL and NBA players had a higher likelihood of dying in a car accident (OR 1.75, 95% CI: 0.91–3.36) compared with NHL and MLB players. In addition, NFL and NBA players had a significantly higher likelihood of dying from a cardiac-related illness (OR 4.44, 95% CI: 1.59–12.43). Mortality trends were disproportionate to team size. Overall, death in active athletes is low. Out of 53,400 athletes who have historically played in the four leagues, only 205 died while active (0.38%). Future examinations into the trends and causes of mortality in elite athlete populations will create a better understanding of health-related risks in elite sport.

Early death in active professional athletes: Trends and causes

Public perceptions of the long-term value of being a professional athlete may be somewhat distorted by social media. For instance, deaths of widely known active athletes are known to the public, which may lead to public perceptions of early death in professional sport despite evidence suggesting otherwise. Teramoto and Bungum's 2010 literature review suggested athletes exposed to more aerobic actions had greater lifespan than the general population. Further, athletes are not homogeneous; the relationship between mortality and professional sport is likely more nuanced than previously considered. There is corroborating evidence that suggests it may be a mistake to undervalue professional athlete lifespans. For example, a more contemporary literature review on the relationship between physical activity and lifespan suggests a conservative lifespan expectancy increase of 2–4 years for individuals who are physically active (Reimers, Knapp, & Reimers, 2012). In addition, specific to high-performance sport, there is similar support for lifespan expectancy benefits at high-intensity levels of sport, such as in professional football players (Baron, Hein, Lehman, & Gersic, 2012), cyclists (Marijon et al., 2013), and soccer players (Taioli, 2007).

It is important to consider the potential confounders of mortality, along with the potential outliers that may skew the overall findings of athlete longevity. For example, although Italian male professional soccer players were found to have an overall greater longevity compared with controls from the general population [standardized mortality ratio (SMR) 0.68, 95% confidence interval (CI): 0.52–0.86], they had an increased risk of death from car accidents (SMR 2.23, 95% CI: 1.46–3.27) and amyotrophic lateral sclerosis (SMR 18.18, 95% CI: 5.00–46.55; Taioli, 2007). Active player mortality trends and causes of death provide interesting insight into the survival likelihood of an athlete at a specific time period in their life. Thus, the purpose of this

study was to examine mortality trends and causes of death among professional athletes who died during their playing careers. Given the limited empirical knowledge of causes of death in active athletes, it was hypothesized that the leading causes of death would be similar to those seen in age- and sex-matched controls from the general population.

Methods

Participants

As of June 2014, deceased professional athletes from the four major sports in North America who were registered as active when they died made up the sample for this study ($N = 205$): the National Basketball Association (NBA; $n = 10$), National Football League (NFL; $n = 60$), National Hockey League (NHL; $n = 48$), and Major League Baseball (MLB; $n = 87$). To meet the entry conditions of our sample, all players had to have played at least one game in their sport and been part of an active roster on a professional team when they died. Alternatively, all professional athletes who played in alternative leagues for the entirety of their careers were excluded from the sample. For example, professional football players who died while active that played in the American Football League, Arena Football League, Canadian Football League, NFL Europe, or the X Football League (the “X” in XFL did not stand for anything) without having played a minimum of one game at the NFL level were excluded from the sample. In addition, athletes who had retired from sport were excluded, irrespective of still being considered physically capable of competing at an elite level and having the potential to re-enter professional sport as a player. Player data were analyzed from each sport’s respective inaugural season: NBA, 1946; NFL, 1920; NHL, 1917; MLB, 1871.

Grouping Criteria

There were 43 different causes of death in our sample. As such, causes of death were analyzed independently, with the exception of rare causes ($n = 1$), which were grouped as “other” (e.g., carbon monoxide poisoning, empyema, nephritis, etc.). In addition, some reported cause of death data may have been confounded by changing perspectives on mental health and suicide. For example, an athlete that overdosed on drugs without intent may have had his death classified as a suicide in an earlier time period; however, a more contemporary coroner/police report may classify it as an accidental drug overdose. Therefore, we kept different classified causes of death independent of each other during analysis (e.g., drug overdose and suicide).

Data Collection

Data were collected from www.wikipedia.org¹ using the key word search of “active athlete death.” We verified these data by cross-referencing a number of online sources, such as www.nfl.com and www.football-reference.com for active death in NFL players, and official team press releases. In addition to our search of sport-specific websites, we further verified death date data through an archive search of the official sport encyclopedias/almanacs that were relevant to our sample, such as *Total Basketball* (e.g., Shouler, Ryan, Smith, Koppett, & Bellotti, 2003), which confirmed complete agreement. All data were collected through secondary sources that were publicly accessible.

General Population Comparison

Mortality statistics of our sample were compared with age- and sex-matched controls from the general population. Canadian-born players make up most of the NHL, although the number of U.S.-born players in the league is growing (second highest nationality to participate

¹ Vital statistics may be an example where Wikipedia’s “open access” method provides accessible data that is similar or superior to traditional sources (Coate & Sun, 2013), having been proven accurate in (former) professional golfers (Coate & Schwenkenberg, 2012) and Olympians and tennis players (Coate & Sun, 2013).

all-time at 14.7%; 24.6% of the players in the 2013–2014 season were U.S.-born; Hockey Weekend Across America, 2012; Quant Hockey, 2014). In addition, a significant proportion of players who played in the NFL, NBA, and MLB were U.S.-born (e.g., 97.1% U.S.-born players made up the 2012 NFL season rosters; Kacsmar, 2013). Therefore, we compared the cause of death results from our sample to the leading causes of death in the Canadian and U.S. referents from the general population to limit the influence of different socio-cultural factors that might affect health and mortality rates. In order for survival comparisons to be made, general population mortality data were accessed through the latest mortality reports from Statistics Canada (2014a) and the Centers for Disease Control and Prevention (CDCP, 2014), which provided lifespan trend tables by age (15–44-year range analyzed, as the leading causes of death were organized into 15–24-, 25–34-, and 35–44-year-old age groups).

Proportional Comparison

For a proportional comparison of mortality rates between sports, we developed a numerator and denominator for our data using the overall proportions for the four leagues (i.e., number of active player deaths vs. number of total players). To collect the total number of players who played in each of the four leagues, we used the player indexes from www.sports-reference.com, which provided an encyclopedia of all players who have historically participated in each of the four leagues, while excluding other personnel (e.g., managers, coaches, etc.; Sports Reference, 2014). These data were cross-referenced where accessible, which confirmed complete agreement (e.g., www.quanthockey.com, nfl.com, etc.). Each database was up-to-date as of June 2014, and the proportions of deaths were reported per 1,000 athletes.

Analyses

Descriptive and odds ratio (OR) analyses were performed using SPSS Version 21 (IBM Corp, 2012). The analyses were performed to assess the trends and causes of death in active professional athletes, after adjusting for type of sport, age, career length, decade of play, race, and Hall of Fame induction. All data were evaluated at the $P \leq 0.05$ level of significance (95% CI).

Results

Descriptive

Among the deceased players, the mean (M) death age was lower for NFL (26.05 years) and NBA (26.7 years) players than for the MLB and NHL players (28.6 years). The youngest age at death was 18 years and the oldest was 44 years ($M = 27.7$ years). Overall, the leading causes of death were from car accidents ($n = 50$; 24.4%), homicides (including casualties of war; $n = 19$; 9.3%), plane crashes and cardiac diseases ($n = 18$; 8.8% each), and suicides ($n = 14$; 6.8%). Mortality rates began to increase from 1960 to 2014 ($n = 138$; 67.3%), when compared with the earlier decades (< 1960; likely, in large part because of the expansion of leagues resulting in a greater professional athlete pool). The sample was mainly comprised of deceased players of Caucasian ($n = 125$; 60.9%) and African-American ($n = 55$; 26.8%) descent. Given the variability of age at death [standard deviation (SD) = 4.46], career length did not appear to influence mortality trends in active professional athletes ($M = 4.96$ years; SD = 4.2). In addition, there were few inductees into the Halls of Fame of their respective sport ($n = 14$; 6.8%). See Fig. 3.1 for an illustration of the leading causes of death data, by sport.

As of June 2014, a total of 53,400 athletes have played in the NBA, NFL, NHL, and MLB. With a total of 205 athletes who died while active, the ratio of death was 3.83 per 1,000 athletes (or 0.38%). Specifically, ratios of death for each sport were 2.37/1,000 (NBA; $n =$

4,217), 2.52/1,000 (NFL; $n = 23,763$), 6.75/1,000 (NHL; $n = 7,103$) and 4.74/1,000 (MLB; $n = 18,317$). Relative to the NBA and NFL, the proportion of deceased players from the NHL and MLB was notably larger, although the percentages remained low (0.67% and 0.47%, respectively). Therefore, despite the NFL and MLB containing more athletes who died while active (147 players vs. 58 NBA and NHL players), mortality trends were not universally proportionate to team size. See Fig. 3.2 for the proportionate leading causes of death data by sport, per 1,000 athletes.

When compared with the mortality databases from Statistics Canada and the CDCP, there was little difference between the leading causes of death in males in the 15–24-, 25–34-, and 35–44-year-old age groups between the two databases from the general population, and our athlete sample. For example, the leading cause of death in all samples was unintentional injuries (i.e., car accidents, etc.). Although plane crashes presumably fell into this category for the national mortality databases, we kept road and aircraft accidents separate in our analyses as plane crashes remain a rare occurrence, and the frequency of death from this cause may be more pronounced through its ability to cause death to a greater number at one time. See Table 3.1 for the leading cause of death data, by country.

OR Analyses

Overall, NFL and NBA players had a higher likelihood of dying in a car accident (OR 1.75) compared with NHL and MLB players, although the relationship just missed significance (95% CI: 0.91–3.36). In addition, NFL and NBA players had a significantly higher likelihood of dying from a cardiac-related illness (e.g., hypertrophic cardiomyopathy; OR 4.44, 95% CI: 1.59–12.43) compared with NHL and MLB players. This should be cautiously interpreted, however, as proportional analysis indicated the NHL was the sport with the highest likelihood of having an

active athlete die from a car accident, if the number of participants between the four sports was considered (1.68/1,000). Therefore, the imbalance between sample sizes in the four sports will produce a higher frequency of deaths in sports with larger participant pools (NFL and MLB), suggesting the number of active player deaths is more likely due to team roster size, rather than participation in the sport itself.

Discussion

This study investigated the mortality trends and causes of death among professional athletes who died during their playing careers from the four major sports in North America. It was hypothesized that the leading causes of death would be similar to those seen in the age- and sex-matched controls from the general population. In our sample, the leading causes of death were from car accidents, homicides, plane crashes and cardiac diseases, and suicides. Importantly, there has been a low proportion of death in active athletes from the NBA, NFL, NHL, and MLB. Out of 53,400 athletes who have historically played in the four leagues, only 205 died while active (0.38%). When compared with the age- and sex-matched controls, the leading causes of death in our athlete sample reflected the leading causes of death in the Canadian and U.S. male general population who were between 15 and 44 years old. The leading causes of death were also sensitive to the decade of play, where earlier participating athletes (< 1960) were more prone to die from infectious diseases (e.g., tuberculosis in MLB players) vs. non-communicable diseases (e.g., heart disease) in the more contemporary players (> 1960).

Although this study was able to determine risk and cause of death for active athletes, the inherent complexity of using historical data to answer questions with relevance in the present where socio-contextual factors may be different was a limitation of our study. For example, in a historical analysis, the impact of socio-contextual factors from specific eras may create a

problem in interpreting causal effects (e.g., causes of death). If the control group is from a suppressed socioeconomic developmental period, such as the Great Depression or during a period of international conflict (e.g., casualties of war being classified as a “homicide”), present-day implications of the study will be limited. We can only draw causal inferences from quantitative analyses of historical data with great care (Simonton, 2003); therefore, a leading cause of death such as tuberculosis in active MLB players may not be as relevant today as non-communicable conditions such as cardiovascular disease.

Second, it is difficult to compare risk and cause of death of a historical sample to the present-day’s general population. That is, our data date back to 1871, MLB’s inaugural season, whereas the data provided by Statistics Canada and the CDCP are more contemporary reports. For example, we can determine the risk of death in Canadian males between the ages of 15 and 44 years who died in 2009: 6,527 (deceased; Statistics Canada, 2014a) divided by 16,663,222 (2009 Canadian male population; Statistics Canada, 2014b) results in just a 0.039% chance of death (or 0.39 per 1000). When compared with the active athlete death rate of 3.83 per 1,000, it appears that being an athlete is associated with an increased risk of death when compared with the Canadian male population; however, moderators such as the rate of death in active athletes likely fluctuating annually and the mixed cultural make-up of our sample makes socio-demographic comparisons to the present-day statistics difficult.

Last, caveats will inevitably arise in this research domain as mortality is an enormously complex outcome. For example, this study examined only active athletes who died if they played at least one game in one of the four sports and had been part of an active roster on a professional team when they died. Thus, this criterion excludes those athletes who have played in a farm system their entire careers, or those lacking the ability to move up to a higher level. This

selection bias of selecting arguably the best athletes limits this study's ability to generalize risk and cause of death findings to all athletes who play or have played basketball, football, ice-hockey, and baseball at a recreational or high-performance level. Further, the generalizability of our results to other countries and sports may be limited. Although culturally diverse participants are inherent to contemporary professional sport organizations, our historical sample predominantly contained Canadian and U.S. born athletes. Nevertheless, beginning to understand the role that sport (e.g., combat vs. non-contact) and socio-cultural (e.g., salary, popularity of sport, etc.) differences play in early death may create some transparency to the general unpredictability of death to relatively younger cohorts.

The inclusion criteria for this study created some larger scale questions. For example, a Hall of Fame player, such as "Junior" Seau Jr., who committed suicide within 3 years of retirement was not included in this sample. Evidently, the entry conditions of this study in which early death was restricted to active career limits our discussion of potential consequences of elite sport participation in recently retired athletes. Nevertheless, given the findings we have highlighted above, it is important to begin identifying risk and cause of death among athletes so future studies can uncover the underlying mechanisms and characteristics that may predispose sport participants to specific causes of early death.

Perspectives

Our findings indicate that there has been a low proportion of death in active professional athletes from the NBA, NFL, NHL, and MLB. Further, the leading causes of death in our sample were similar to those in the Canadian and U.S. general population. In a broad sense, a comprehensive understanding of the implications of involvement in professional sport is necessary for the formation of evidence-based models of athlete development and care.

Acquiring a comprehensive understanding of athlete health outcomes will necessitate assessments of high performance athletes prior to, during, and after professional/elite participation in sport. Specifically, future examinations into the trends and causes of mortality in elite athlete populations will create a better understanding of potential health-related risks in elite sport. In summary, given our current lack of empirical understanding, future research should seek to provide new insight on which developmental pathways contribute to longer life expectancies. As such, this research can have important implications at the youth sport level for policymakers regarding how programs should be structured. For example, a larger focus on sampling sports rather than early specialization may reduce the incidence of overuse and acute injuries, which can minimize risk of osteoarthritis manifesting in later life that could inhibit lifelong maintenance of physical activity.

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Figure 3.1: Leading causes of active player death, by sport (percentages represent the percentage of total causes of death in each sport)

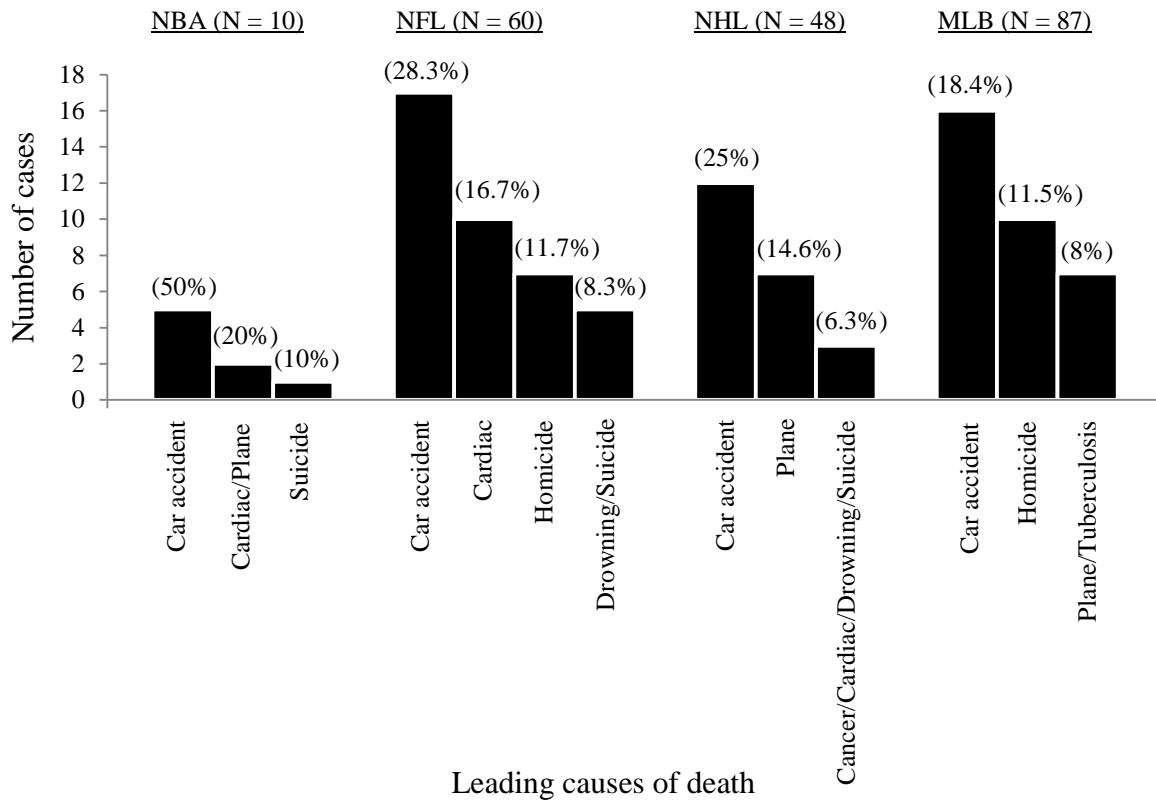


Figure 3.2: Proportionate leading causes of active player death by sport, per 1,000 athletes (total historical players in each sport in brackets; 1871 to June 2014)

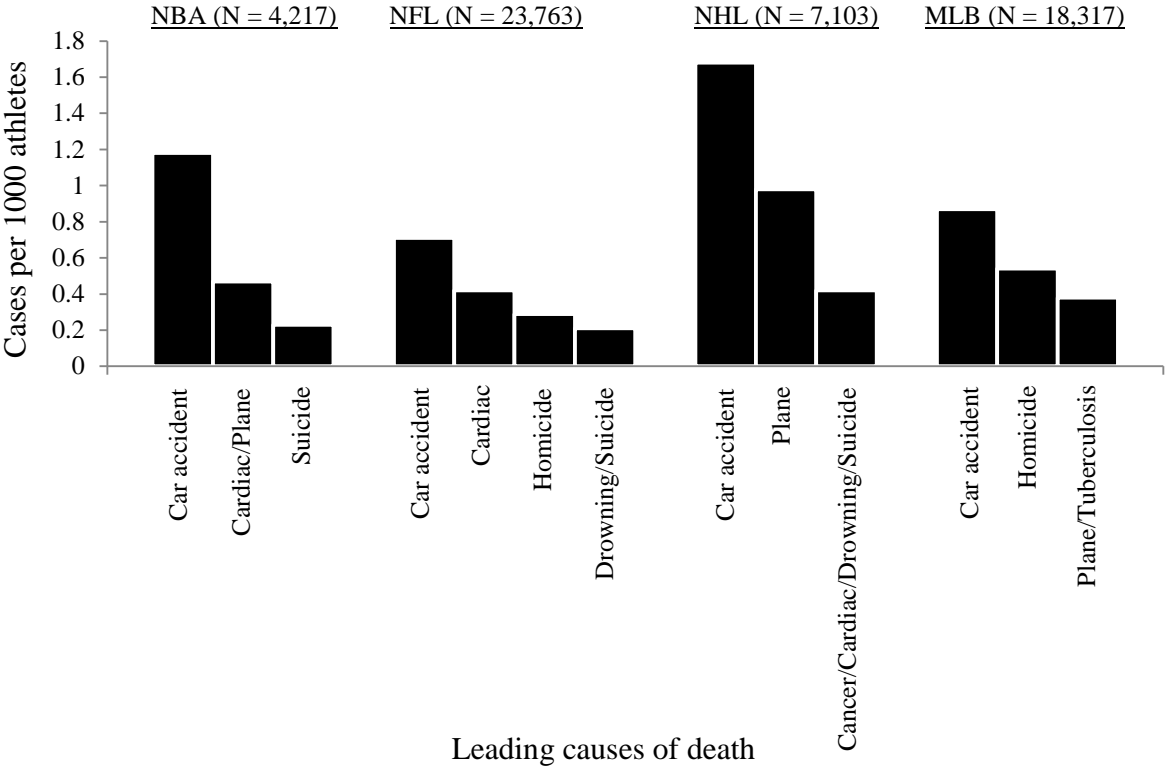


Table 3.1: Leading causes of death, by country (Centers for Disease Control and Prevention, 2014; Statistics Canada, 2014a)

Age group (years)	Leading cause of death in males; percentage of total deaths in age group due to the cause indicated
15–24	<i>Canada, 2009:</i> Unintentional injuries (i.e., car accidents); 41.5%, intentional self-harm (suicide); 23.5%, assault (homicide); 9.1%, malignant neoplasms (cancer); 6.7%, diseases of the heart (cardiac); 2.9% <i>United States, 2010:</i> Unintentional injuries (i.e., car accidents); 41.5%, homicide; 19%, suicide; 17.2%, cancer; 4.6%, cardiac; 3.2%
25–34	<i>Canada, 2009:</i> Unintentional injuries (i.e., car accidents); 34.2%, suicide; 23.6%, cancer; 7.7%, homicide; 5.6%, cardiac; 4.9% <i>United States, 2010:</i> Unintentional injuries (i.e., car accidents); 37%, suicide; 15.9%, homicide; 12.2%, cardiac; 7.6%, cancer; 6.1%
35–44	<i>Canada, 2009:</i> Unintentional injuries (i.e., car accidents); 18.6%, suicide; 18.4%, cancer; 17.1%, cardiac; 13.7%, chronic liver diseases and cirrhosis; 2.4%* <i>United States, 2010:</i> Unintentional injuries (i.e., car accidents); 23.5%, cardiac; 17%, cancer; 11.8%, suicide; 11.6%, homicide; 4.3%

*Chronic liver diseases and cirrhosis were the only dissimilar top 5 leading cause of death from these mortality reports. Homicide was the sixth leading cause of death in the 35–44 year-old group in Canada in 2009.

CHAPTER FIVE: HEIGHT AND LIFESPAN

Do “Big Guys” really die younger? An examination of height and lifespan in former professional basketball players*

*A consolidated version of this article is currently under review for publication in *Scandinavian Journal of Medicine & Science in Sports*

Chapter Five Overview

Background: While factors such as genetics and socio-economic status may mediate the relationship between height and mortality, a sizeable amount of evidence suggests larger body size independently reduces longevity. The purpose of this study was to extend this research and critically examine the relationship between height and longevity in a population of athletes generally regarded as having superior height relative to the rest of the population; professional basketball players.

Methods: We examined living and deceased players who played in the National Basketball Association (debut between 1946 – 2010) and/or the American Basketball Association (1967–1976) using descriptive and Kaplan-Meier and Cox regression analyses. The cut-off date for death data collection was December 11, 2015.

Results: Overall, 787 former players were identified as deceased with a mean height of 193.9 cm (± 8.83 , Range: 167.6–228.6). Descriptive findings indicated that the tallest players (top 5%) died younger than the shortest players (bottom 5%) in all but one birth decade (1941–1950). Similarly, survival analyses showed a significant relationship between height and lifespan [$\chi^2 (1) = 13.04, p < .05$], where taller players had a significantly higher risk of early mortality compared to shorter players (HR: 1.30, 95% CI: 1.13 – 1.50, $p < .05$).

Conclusions: The uniqueness of examining the height-longevity hypothesis in a relatively homogeneous population of tall individuals should be considered when interpreting these results. More research is needed on this relationship in sport, where further evidence can inform and clarify our understanding of potential risks of early mortality to help generate more specific screening programs geared towards at-risk cohorts of the athlete population.

Do “Big Guys” Really Die Younger? An examination of height and lifespan in former professional basketball players

“I tell my wife all the time, 'You don't see many 7-footers walking around at the age of 75,'" says Bird, who's 6-foot-9. "She hates it when I say that. I know there are a few of us who live a long time, but most of us big guys don't seem to last too long. I'm not lying awake at night thinking about it. If it goes, it goes.”

– Larry Bird, Hall of Fame basketball player (MacMullan, 2016)

A recent article by ESPN senior writer Jackie MacMullan entitled “*Larry Bird will die young. Just ask him*” highlights a growing concern and “fatalistic view” of lifespan amongst former professional basketball players (MacMullan, 2016). Certainly, the death of a former teammate or competitor can often initiate one’s own contemplation of mortality, and recent young deaths of former “big guy” National Basketball Association (NBA) players (e.g., Anthony Mason, Darryl Dawkins and Moses Malone) may accentuate this perception.

From a scientific standpoint, much of the work in this area has been dominated by one team of researchers (Samaras and his colleagues), which can have important implications in how an effect is perpetuated and disseminated. One of the main principles of the scientific process entails independent replication, and extensive work by a small number of researchers can be susceptible to a unidimensional viewpoint and places an increased emphasis on conducting rigorous and appropriate analyses. The premise that larger body size leads to reductions in lifespan has been substantiated ubiquitously through this research over the past 40 years (see www.humanbodysize.com for a complete list of Samaras et al.’s publications, in addition to having authored two books, *The Truth About Your Height* (Samaras, 1994) and *Human Body Size and the Laws of Scaling* (Samaras, 2007)). Samaras’ research suggests smaller body size is generally better for one’s health, and is supported by robust cross-cultural findings of average lifespan reduction with increasing height observed in groups such as deceased American male

veterans (Samaras & Storms, 1992), French males and females who died before the year 1861 (Samaras, 1996; Samaras & Heigh, 1996) and males born in Sardinia, Italy between 1866 and 1915 (Salaris, Poulain, & Samaras, 2012). This finding has been supported by several reviews, which also include similar longevity outcomes from animal data (e.g., Samaras, 2009b, 2012a, 2014; Samaras & Elrick, 2002). Of particular interest, Samaras' 2014 review showed evidence that smaller body size is related to greater longevity in eight different types of studies, where life expectancy differences have been found: i) *within* the same species (e.g., dogs; Bartke, 2012), ii) in caloric intake (i.e., caloric restrictions produce smaller body sizes and longer longevity; e.g., Fontana, 2009; Willcox et al., 2004), iii) in centenarians, who are generally smaller (adjusting for shrinkage; e.g., Martinez, Calzadilla, Fonseca, Castellano, & Sceo, 2009), iv) between ethnic groups (e.g., greater longevity in shorter groups such as the Asian population; e.g., Samaras, 2009b), v) among developed countries (i.e., top populations for life expectancy were shorter (e.g., Macao) in relation to the tallest Western Europe populations (e.g., Sweden); Samaras, 2009b), vi) between males and females (e.g., on average, men are approximately 9% taller than women, but have a 9% lower life expectancy; Samaras, 2009b), and in vii) survival and viii) lifespan studies that generally favour shorter individuals (e.g., a Spanish study of 1.3 million males reported a loss of 0.7 years per centimetre (cm) of increased height; Holzenberger, Martin-Crespo, Vicent, & Ruiz-Torres, 1991). Thus, Samaras' overarching conclusion suggests health practitioners should de-emphasize that a taller height is necessary for well-being and success in life (e.g., Samaras, 2012a), despite this being somewhat counterintuitive to society's belief that increased height is a by-product of a healthy lifestyle (Samaras, 2009a). Nevertheless, the relationship between height and quality of life (particularly early to midlife) and eminence/higher

social status may be less clear and different than the height-mortality link, where quality of life is more socially constructed and mortality is more biologically linked to health.

Biological Mechanisms

While the biological reason for the relationship between height and lifespan in humans is not yet fully understood (He et al., 2014), it is difficult to ignore the potential profound effect of genetics on lifespan. A study by He and colleagues (2014) on 8,006 American men of Japanese ancestry found height was positively associated with mortality, and perhaps of more interest, they were the first to conclusively link the “longevity gene” *FOXO3* to smaller body size and greater longevity in humans. In addition, Samaras (2014) provided a summary of studies illuminating proposed biological mechanisms that underpin superior longevity in smaller humans. Various biological and physiological parameters change with increases in body size (Samaras, 2014), such as greater cardiovascular load in bigger mammals, and some of most robust explanations for this apparent inherent detriment of greater height includes reduced cell replication potential in old age to maintain body tissues and organs (e.g., Samaras, 2012a) and higher incidence of DNA and free radical damage (e.g., Giovannelli, Saieva, Masala, Testa, Salvini, & Pitozzi, 2002). For example, an individual with a larger body size has trillions more cells compared to a smaller person (e.g., 6’7” versus a 5’9” individual), and as a consequence, cell replication required after cumulative exposure to stresses can ultimately lower the ability of healthy cells to replace the damaged ones later in life since regeneration is limited, leading to higher incidences of chronic diseases such as cancer and cardiovascular disease from greater exposure to carcinogens (e.g., Samaras, 2007, 2012b). In addition, organs such as the brain, liver and kidneys become disproportionate in size in taller individuals, which reduces their functional capacity relative to body mass (Samaras, 2007, 2012a).

While these biological and physiological changes, accompanied with the *FOXO3* genotype, have been found to influence lifespan, it is also important to recognize confounders of this relationship. Samaras (2012a) has suggested that height generally explains less than 10% of the proportion of variance regarding longevity, and has identified numerous confounders on this relationship (e.g., genetics, socioeconomic status (SES)). He and colleagues (2014) surmise that the lack of consensus on how height affects lifespan is likely due to the impact of these extraneous variables. For example, Gavrilov and Gavrilova (2012) did not find an association between height and survival to age 100 years, and reported that the average height of centenarians at ~50th percentile (although they acknowledged the lack of effect may have been a result of a small sample size). In contrast, from a morbidity standpoint, a more recent study examining 144,701 postmenopausal women found a positive association of height with risk of *all* cancers (Kabat et al., 2013). Thus, conflicting findings in epidemiological studies on height and health reflect the inherent complexity of controlling for confounders and the difficulty gaining access to official death certificates produced by government officials, such as coroner/medical examiner reports, which may limit analyses to “all-cause” rather than cause-specific deaths.

Although a sizeable amount of evidence suggests larger body size *independently* reduces longevity (e.g., Samaras & Elrick, 2002), attention has also been given to the potential influence of other factors that may mediate the relationship that has previously been observed between height and mortality, such as differences in genetics, SES, education, medical care, relative weight, hygienic practices, nutrition, and lifestyle choices such as engaging in regular exercise and avoiding smoking (e.g., He et al., 2014; Samaras, 2012a). In particular, medical practitioners are implementing cost-effective interventions to promote healthier lifestyles such as plant-based diets, which has been defined as “a regimen that encourages whole, plant-based foods and

discourages meats, dairy products, and eggs as well as all refined and processed foods” (Tuso, Ismail, Ha, & Bartolotto, 2013, p. 61). A recent report by the World Health Organization reviewing over 800 studies linked the carcinogenicity of excessive red and processed meat consumption to increased cancer risk (e.g., colorectal, pancreatic and prostate cancer; International Agency for Research on Cancer, 2015). Further, a review examining the effectiveness of plant-based diets found reduced risk of cardiovascular disease and mortality compared to individuals who consumed a non-plant-based diet (Dietary Guidelines Advisory Committee, 2010). From a caloric restriction standpoint, Okinawan Japanese are relatively shorter and consume significantly fewer calories than mainland Japan, and the residents of the island have historically had the longest life expectancy, highest prevalence of centenarians per capita, and the lowest mortality rates from chronic diseases such as cardiovascular disease, cancer, and diabetes in the developed world (e.g., Suzuki, Willcox, & Willcox, 2001; Willcox et al., 2004). The above confounders on height and mortality highlight the complex nature of this relationship, which is likely impacted by various known and unknown biological processes (He et al., 2014), prompting researchers to suggest the relationship may be more complicated than, for example, obesity’s role on longevity (Gavrilov & Gavrilova, 2012).

Athletes

Compared to the general population there has been much less investigation into the relationship of height and lifespan in athletes. Samaras (2009b) provided a summary of studies that have examined athletes, and a decrease in longevity per centimetre of height (years/cm) was reported in Finnish (-0.49) and Harvard (-0.70) athletes, and baseball (-0.35) and football (-0.81) players. While each of these athlete samples had a negative correlation between height and longevity, it is important to recognize the aforementioned concern regarding having

predominantly one team of researchers conducting this work. Lawler, Lawler, Gibson and Murray (2012) examined mortality outcomes in professional basketball players, and Cox proportional hazards survival analyses did not reveal height to be a significant predictor of lifespan in their crude regression model (Hazard Ratio (HR) 1.01, 95% Confidence Interval (CI): 0.96 – 1.05). Therefore, there is conflicting evidence on the relationship between height and lifespan in athletes.

The purpose of this study was to extend this research and provide a more in-depth critical analysis on the time-dependent variable of height on a population of athletes generally regarded as having superior height relative to the rest of the population; professional basketball players. Considering the recent media speculation regarding the concern of NBA “big guys” dying younger, a more up-to-date study to Lawler and colleagues’ (2012) examination into the mortality outcomes of National Basketball Association (NBA) and American Basketball Association (ABA) players was conducted, since they included players as of the 2004-2005 NBA season and calculated deaths as of December 31, 2011. This study exclusively measured height and its role on lifespan, despite the expression of an individual’s longevity profile being affected by several variables. However, if height influences longevity independently, deceased professional basketball players represent a promising group to further investigate this phenomenon given the relative homogeneity of exceptional height and more affluence (particularly in the more recent decades), where the higher SES/social status may result in less confounding by factors such as ethnicity. We hypothesized that when adjusting for birth decade, exceptionally taller players will have died at relatively younger ages.

Methods

Population

The population of this study was comprised of living and deceased players who played in the NBA (debut between 1946 – 2010) and/or the ABA (1967 – 1976). A large proportion of players who debuted in the 2009-10 season were still active at the time of this study. The cut-off date for death data collection was December 11, 2015. Data were collected from publicly available sources.

Data Collection

To compile our data, basketball-reference.com was the primary source used for retrieving players' lifespan and anthropometric information. Wikipedia.org was accessed as a supplemented source in the case of incomplete date of birth and/or death data (<10 cases). Twenty percent of the lifespan and anthropometric data were cross-referenced in the official NBA encyclopedia (Koppett, Shouler, Ryan, Smith, & Bellotti, 2003), which confirmed complete agreement between data sources.

Variables

Athletes were coded as living or deceased. Height was converted into centimetres (cm) and the median height for each birth decade was calculated to categorize players into "below median height" or "above median height."

Statistical Analyses

Data were analyzed using i) descriptive and ii) Kaplan-Meier and Cox regression survival analyses. Since average height was liable to change over the time-course of the athletic population studied, it was analyzed by percentiles for separate birth decades of players. In addition, lifespan results were compared to males from the United States (US) general population using life expectancy data from the latest vital statistics report from the Centers for Disease Control (CDC; "*Deaths: Final data for 2013*," 2016) and from Noymer and Garenne's (2000) study on mortality differences in the US. For the survival analyses, a t-test median split of height

per birth decade was performed to also account for the changing anthropometric birth cohort effects. Analyses were performed using SPSS version 23, and statistical significance was defined as $p < 0.05$, at the 95% CI.

Results

Descriptives

Overall, 787 former NBA/ABA players were identified as deceased as of December 11, 2015. The mean height of the deceased NBA/ABA player population was 193.9 cm (± 8.83 , Range: 167.6 – 228.6). Table 4.1 shows the mean ages of death per birth decade, displayed by percentile height (bottom 5%, bottom 10%, 50th percentile, top 10% and top 5%). The 5th percentile included the shortest players whereas the 95th percentile included the tallest players. A linear increase in mean height was observed for players born in the earliest decades ($M = 188.6$ cm) to the most recent decades ($M = 205.7$ cm), which supports the commonly held view that the evolution of basketball players (and other athletes) is predicated on them becoming bigger, faster and stronger.

Since the inaugural season of the NBA was not until 1946, a high majority of deceased players in this population were subjected to dying prematurely; therefore, the influence of height on lifespan was interpreted by lifespan differences *within* the same decade of birth, rather than between decades. Descriptively, the tallest players (top 5%) died younger than the shortest players (bottom 5%) in all but one birth decade (1941 – 1950). In total, there was a negative linear relationship between height and lifespan; the shortest players lived the longest (75.1 years) and the tallest players lived the shortest (56.6 years). Interestingly, it appears that the robustness of this effect diminished in players born in more recent decades. More specifically, for players born in 1940 or earlier, the mean ages of death for the tallest players were below the mean ages of death for all players born within the same respective decade; however, this effect reversed for

those born in 1941 or later (i.e., the tallest players lived slightly longer on average than the mean age of death for each birth decade). The large overall discrepancy in lifespan between shorter and taller players is likely a result of the larger sample sizes of deceased players born in 1940 or earlier (n = 579) that found shorter players to live longer compared to more recently (≥ 1941 ; n = 208).

Given that our study included deceased former players as of December 11, 2015, only a small proportion had the opportunity to live a full life (i.e., to ‘old age’). Therefore, we compared the mean ages of death for players born in 1920 or earlier and between 1921 – 1930 to the life expectancies at birth and in 2013 (most recent data) for males from the US general population. The life expectancies at birth in the male US general population were 46.3 years in 1900, 48.4 years in 1910, 53.6 years in 1920 and 58.1 years in 1930 (Noymer & Garenne, 2000). Evidently, the mean ages of death of the NBA/ABA player population far exceeded these estimates; players born in 1920 or earlier died at 76.7 years on average, and 75.2 years on average for those born between 1921 and 1930. Further, the tallest players (95th percentile) had similar ages of death to their respective birth decade mean. Interestingly, when compared to the 2013 referent life expectancy of 76.4 years (“*Deaths: Final data for 2013*,” 2016), players’ mean ages of death were very similar, with the largest discrepancies found in players born in 1920 or earlier who were in the bottom 5th percentile in height (lived 80.6 years on average) and those born between 1921 and 1930 that were in the top 5th percentile in height (lived 72.4 years on average).

Survival Analyses

Overall, data from 3,901 living and deceased players who played between 1946 and 2010 were included in this analysis. Figures 4.1 and 4.2 illustrate survival curves which were grouped by above and below median height per birth decade. Kaplan-Meier survival analysis found a significant relationship between height and lifespan in former NBA/ABA players [$\chi^2(1) = 13.04$,

$p < .05$]. In addition, Cox regression analysis found taller players to have a significantly higher risk of early mortality compared to shorter players (HR: 1.30, 95% CI: 1.13 – 1.50, $p < .05$).

Discussion

The purpose of our study was to examine the influence of height on the lifespans of professional basketball players, and we hypothesized that the relatively taller players would die at younger ages. Our hypothesis was supported through our key descriptive finding which indicated that there was an overall negative linear relationship between height and longevity where the tallest players died earliest on average. Interestingly, there was small lifespan *favourability* for the tallest players born in 1941 or later (in relation to the mean ages of death per birth decade); however, we postulate that this counterintuitive trend was likely a result from small sample sizes that may be skewing our findings. More specifically, 45.6% ($n = 359$) of the deceased players were born between 1921 and 1930, whereas only 6.7% ($n = 53$) were born between 1951 and 1960 and 4.7% ($n = 37$) after 1960. In addition to our descriptive analyses, Kaplan-Meier and Cox regression analyses also supported our hypothesis, where taller players had significantly higher risk of early mortality compared to shorter players.

Our descriptive analyses allowed us to explore the nuances of lifespan for the exceptionally tall cohorts (i.e., 95th and 90th percentiles). Therefore, in the present sample, the tallest players appear to die relatively younger than the shortest players, although findings for players born in the earlier decades may have more meaningful significance since they had the opportunity to live a full life. The lifespan differences in the later born players were likely confounded by more unnatural causes of death rather than the suggested negative biological dispositions of increased height. This hypothesis is supported by Lemez, Wattie, and Baker (2015), who reported that the leading cause of death in *active* NBA players was car accidents, although only 10 cases of premature death were noted in this athlete group.

More importantly, a high majority of participants in professional basketball are likely taller than the average height of males in the general population, including players even in the bottom 10th percentile of height. Therefore, although taller players appear to die younger relative to shorter players, we found the mean ages of death in the 95th percentile in height of players born in 1930 or earlier to be similar to the life expectancies at birth in 2013 for males from the US general population (and still far superior to what their life expectancies estimated at the time of birth were, although time of birth estimates include those who die very young). Lawler and colleagues (2012) suggested that the reasons for particular mortality outcomes for basketball players are likely to be multifactorial, such as the income, life experience, and socio-economic status incurred from being a professional athlete which allow for greater life quality such as better medical care. These reasons, accompanied with exceptional height being such a rarity in the general population, complicate our understanding of health in individuals with heights in the highest percentiles. In fact, in David Epstein's *The Sports Gene*, he states,

“An American man who is seven feet tall is such a rarity that the CDC does not even list a height percentile at that stature. But the NBA measurements combined with the curve formed by the CDC's data suggest that of American men ages twenty to forty who stand seven feet tall, a startling 17 percent of them are in the NBA *right now*. Find six honest seven-footers, and one will be in the NBA.”

– (Epstein, 2013, p. 131 – 132)

We calculated only 2% of the entire deceased player population to be seven feet (16/787; 213.36 cm), with a majority being born between 1961 and 1970 (9/16). Therefore, as professional athletes continue to become taller, more data will become available for future research to explore the rates and causes of death in the exceptionally tall athletes, which will provide us with more complete knowledge to delineate the reasons behind the potential risks

involved with increased height, or whether being a professional athlete is enough to offset the apparent health detriments from larger body size.

Limitations

First, a potential limitation of the height variable was that a median split of height by birth decade was used to measure mortality outcomes, which may have been too crude of a measurement of height. However, using a more specific measure (e.g., $.33 \times .33 \times .33$) would have created small sample sizes, particularly in the deceased samples from more recent decades. This was further complicated by the players' overall exceptional height; nearly all data was above the 90th percentile for general US population height, making any-cut off point arbitrary. Second, since living players were included in the survival analysis, 79.8% of the NBA/ABA player population was censored, which may have limited some of the meaningfulness of the survival analyses results, although these findings were supported descriptively. Last, while the focus of this study was to critically examine the relationship between height and lifespan, many other socially constructed or biological variables could have influenced mortality outcomes. Conversely, if height is considered a unique predictor of death, then controlling for variables such as decade of birth may not be as important.

Conclusion

This study found that taller NBA/ABA players died earlier than shorter NBA/ABA players; however, the uniqueness of examining the height-longevity hypothesis in a relatively homogeneous population of tall individuals should be considered when interpreting these results. As many players have superior height compared to the age- and sex-matched average height of the US general population, there appears to be a curvilinear relationship between height and longevity where the magnitude of early risk of mortality decreases past a certain threshold (i.e.,

for players in the 95th percentile of height born in 1941 or later). However, smaller sample sizes in the younger players may have been driving this effect, and it is unknown whether this relationship is more monotonic in athletes. Further, Lemez and Baker's (2015) review suggested that elite athletes live longer, despite most of them likely being taller than individuals from the general population. Thus, more research is needed on this relationship in sport, where further evidence can inform and clarify our understanding of potential risks of early mortality to help generate more specific screening programs geared towards at-risk cohorts of the athlete population (e.g., implementing more frequent cardiovascular screenings for the relatively taller players). From a general population perspective, it is unclear whether there is a threshold for the apparent longevity benefits from having smaller body size (i.e., do the same benefits apply to persons who are in the 5th percentiles of height and weight?). Therefore, continued research on the height-longevity hypothesis is needed in both athlete and non-athlete samples to demystify the nuances of this relationship, such as including cause of death data and examining the mortality outcomes of the smallest individuals.

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Figure 4.1: Kaplan-Meier survival probability, by above and below median height per birth decade [$\chi^2 (1) = 13.04, p < .05$]

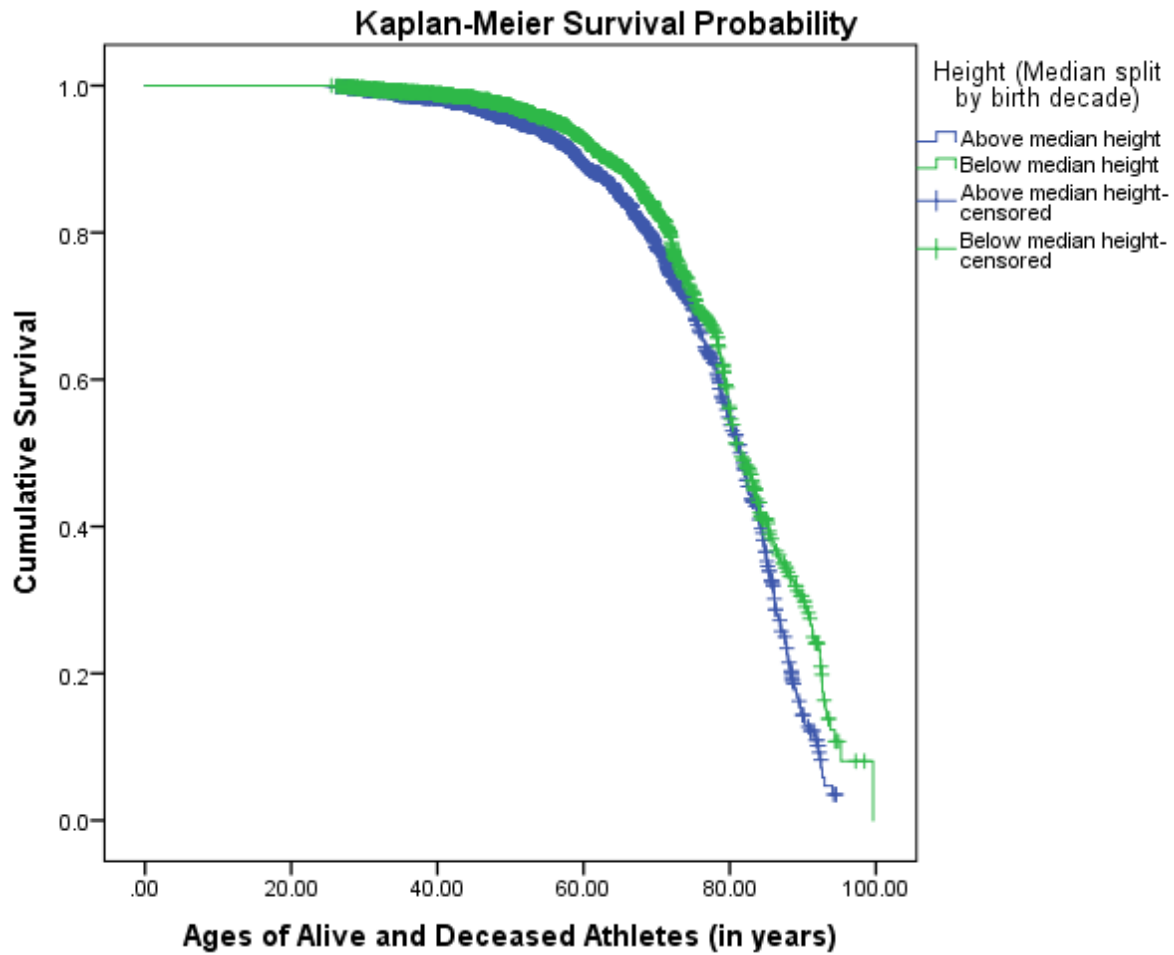


Figure 4.2: Cox regression hazard model, by above and below median height per birth decade

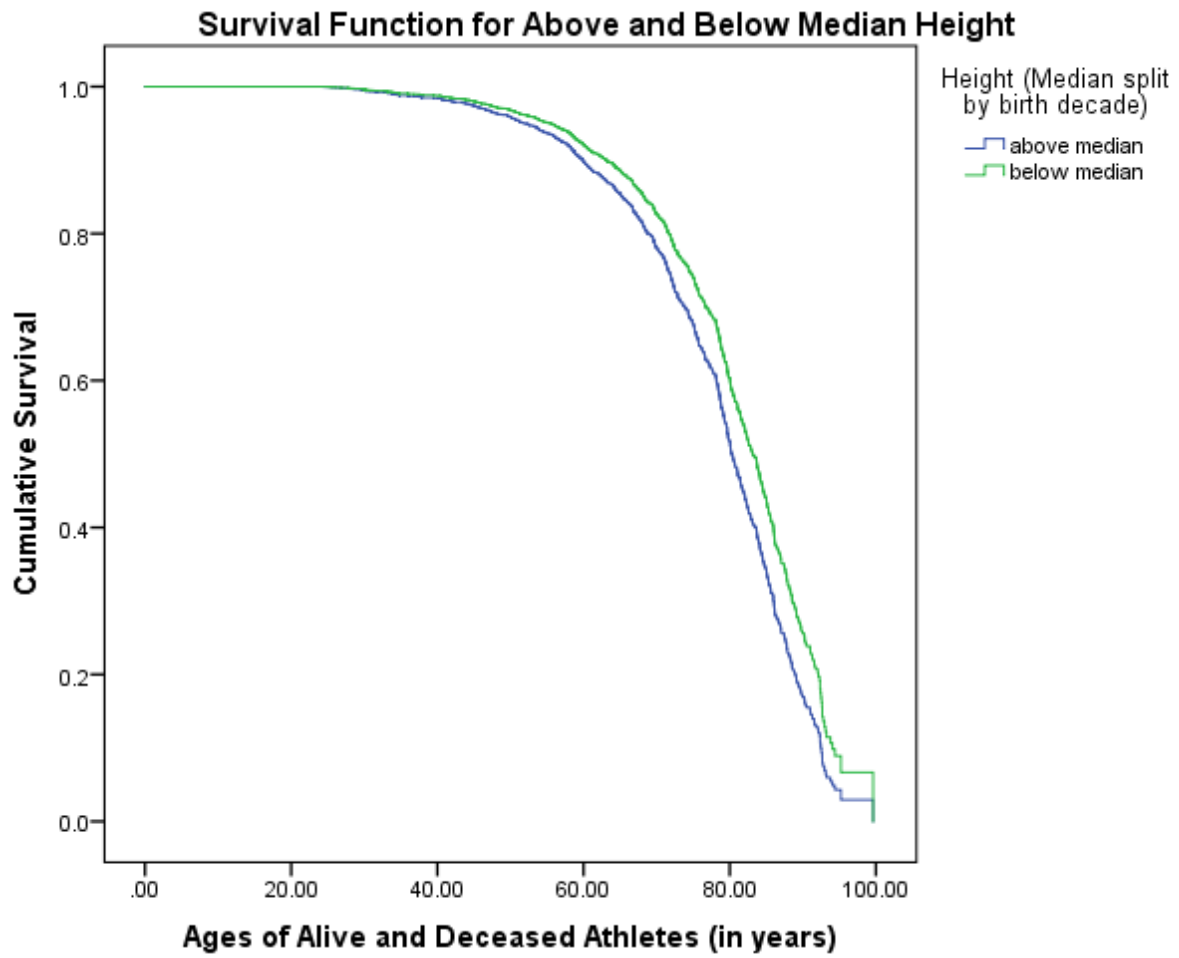


Table 4.1: Mean (*M*) ages of death per birth decade, by percentile height

Birth Decade	<i>M</i> (<i>M</i> height in cm)	Height percentiles, calculated for each birth decade separately					% censored (n of deaths)
		5 th	10 th	50 th	90 th	95 th	
≤ 1920*	76.71 ± 12.41 (188.65)	80.66 ± 10.88	77.12 ± 12.25	76.82 ± 12.80	74.41 ± 12.02	75.96 ± 16.25	1.8 (109)
1921 – 1930	75.29 ± 11.92 (191.49)	76.57 ± 12.64	76.78 ± 11.69	75.71 ± 10.86	73.49 ± 11.37	72.46 ± 11.05	18.6 (359)
1931 – 1940	66.19 ± 13.78 (196.88)	66.01 ± 8.46	66.42 ± 15.30	66.48 ± 13.24	65.73 ± 13.34	62.00 ± 14.28	59.3 (111)
1941 – 1950	56.69 ± 12.47 (197.21)	54.81 ± 14.84	56.34 ± 13.57	57.49 ± 12.43	54.67 ± 14.56	56.79 ± 14.37	82.3 (118)
1951 – 1960	51.24 ± 9.60 (198.93)	56.71 ± 3.81	56.70 ± 5.19	48.92 ± 10.60	54.17 ± 5.51	52.36 ± 5.72	91.6 (53)
≥ 1961*	39.61 ± 9.16 (205.67)	41.43 ± 11.87	36.98 ± 9.19	39.17 ± 9.05	39.28 ± 9.21	39.70 ± 8.97	97.9 (37)
Total	68.11 ± 16.03 (193.88)	75.11 ± 13.07	74.58 ± 13.21	72.32 ± 13.97	59.01 ± 16.50	56.66 ± 16.48	79.8 (787)

*The earliest birthdate was in 1902, and the most recent was latest was in 1982

CHAPTER SIX: CAUSES OF DEATH

Vital statistics and early death predictors of North American professional basketball players: A historical examination*

*A consolidated version of this article will be submitted for publication to *Journal of Science and Medicine in Sport*

Chapter Six Overview

Background: Health information disseminated through social media can be susceptible to creating misrepresentations and reductive generalizations, particularly in how we perceive visible issues such as a premature death of an elite athlete. While empirical evidence suggests elite athletes have superior lifespan outcomes relative to the general population, less is known regarding their causes of death. As such, the overarching purpose of this study was to critically examine the mortality outcomes of the entire deceased National Basketball Association (NBA) and American Basketball Association (ABA) population.

Methods: Death data were collected from publicly available sources until December 11, 2015, and causes of death were categorized using the *International Classification of Diseases, Tenth Revision* (ICD). Four separate measures of mortality were utilized: i) cause-specific crude death rates (CDR), ii) estimates of death rates per athlete-year (AY), iii) years of potential life lost (YPLL), and iv) binary and multinomial regression analyses with the inclusion of players' biological and occupational variables that were collected.

Results: Our search identified 514 causes of death out of a total of 787 deceased NBA/ABA players ($M = 68.1 \text{ y} \pm 16.0$) from 16 different ICD groups, 432 of which were from natural causes and 82 from unnatural or premature causes. Findings showed i) similar leading causes of death and CDRs to the sex- and race-matched controls from the US population, ii) higher death rate differences per AY within time-dependent variables such as birth decade, iii) a high premature death rate (59.4%) if life expectancy was set at 75 years, but much lower if life expectancy at birth for each player was considered (28.7%), and iv) a higher likelihood of dying below the median age of death for black and taller players, although regression analyses results were highly confounded by birth decade.

Conclusion: More knowledge of mortality outcomes from participating in high performance sport would advance sport medicine, provide broad public health applications, and disarm potentially harmful stereotypes of health in elite athletes. In accordance with the growth in social media usage, researchers and practitioners alike should negotiate best avenues to improve health and the transparency between different mediums of information concerning health in athletes in order to reconcile otherwise limited empirical evidence.

Vital statistics and early death predictors of North American professional basketball players: A historical examination

In recent years, our awareness of when an athlete retires from sport has grown, particularly with more notable athletes. The propagation of readily available information through various media formats and outlets (e.g., social media) has made an elite athlete's transition into retirement a compelling storyline. Incidentally, they are often only in the young adulthood period of human life (~20 – 40 years), which generally leaves them with considerable years of life left post-retirement. While relatively few are able to sustain their level of admiration and popularity (e.g., being visible through working in media and coaching), barring a rare event, most are typically brought to mind again once they die, either through local or national media coverage.

Similar to the differences in status while they were active athletes, the high-profile former athletes receive more coverage in death relative to the lesser known athletes. While this reporting is common throughout sports, it can create misrepresentations and empower biases in how we perceive the lifespans of certain groups of athletes, and in some cases the causes that contribute to their deaths. For example, it was widely reported when three relatively young and well-known former African-American National Basketball Association (NBA) players died from heart disease in 2015 (Anthony Mason, 48 years of age; Darryl Dawkins, 58 years of age; Moses Malone, 60 years of age). As such, the aggregation of similar causes of death in an alike demographic group in a short period of time can perpetuate reductive generalizations of death pertaining to certain subgroups of athletes.

Lemez and Baker's (2015) systematic review of mortality and longevity in elite athletes highlighted that almost all empirical evidence to date points to greater longevity for high performance athletes compared to age- and sex-matched controls from the general population. Researchers have investigated several risk factors that may impact longevity in athletes, such as

the type of sport participated in, which energy system was activated to meet the demands of that sport (i.e., aerobic, anaerobic, or mixed), playing position, race, weight, and handedness. While some risk factors appear to have more influence on lifespan than others, such as Body Mass Index (BMI), race, and player position (e.g., in National Football League (NFL) players; Baron, Hein, Lehman, & Gersic, 2012), it is difficult to itemize the importance of these characteristics on lifespan without knowing the cause of death. Unfortunately, cause of death in former athletes is reported far less often than data such as date of death.

Sudden Cardiac Death in Athletes

A majority of studies that have investigated causes of death in sport have established sudden cardiac death (SCD) as one of the leading causes of non-violent death in young athletes (see for example; Harmon et al., 2015; Harmon, Drezner, Wilson, & Sharma, 2014). Harmon and colleagues (2015) reviewed all National Collegiate Athletic Association (NCAA) deaths between 2003 and 2013 (N = 514) and reported that the most common medical cause of death was SCD (15%), with the highest rate of disease occurring in male NCAA basketball players (n = 19; Harmon et al., 2015). In addition to basketball players, males and African-American athletes were also found to be at a higher risk of SCD (Harmon et al., 2015), which was supported in the cumulative finding of Harmon and colleagues' (2014) 13-study review that investigated the rate of SCD in athletes.

While it appears that SCD accounts for a significant portion of cardiac-related deaths, certain caveats must be considered when describing its prevalence in competitive athletes. A review of SCD performed by Chandra, Bastiaenen, Papadakis, and Sharma (2013) described the challenges and diagnostic dilemmas inherent to evaluating athletes for underlying medical conditions. For example, several factors have been implicated as confounders of the

manifestation of SCD in young athletes, such as sex, race, and type of sport; however, identifying higher SCD risk in certain subgroups of athletes without considering the disproportionate participation rates may be misleading. Chandra and colleagues (2013) highlighted participation inequalities and the changing demographic of participants at the elite level in sport, where female involvement is generally lower compared to males, and black male involvement has dramatically increased in sports such as basketball and football over the past three decades (Chandra, Papadakis, & Sharma, 2012). They further suggested that potential biases such as the lack of sample size equivalency across sports (e.g., higher participation rates in basketball and football in the United States (US); Maron, Shirani, Poliac, Mathenge, Roberts, & Mueller, 1996), may confound the findings of higher SCD and perceived risk in these sports.

Cardiovascular diseases often advance health risks, and an athlete dying from a heart-related disease has become a highly visible tragedy (Maron, Haas, Murphy, Ahluwalia, & Rutten-Ramos, 2014). However, Maron and colleagues (2014) reported a participation of 4,052,369 student-athletes from 30 different intercollegiate sports between 2002 and 2011, which resulted in a low risk of SCD relative to the general population in their sample of athletes (1.2/100,000). As well, Chandra and colleagues (2013) described the etiology of SCD in athletes younger than 35 years of age and suggested that the common causes can be divided into three broad categories of cardiac abnormalities: (i) structural (e.g., SCD in young athletes is most commonly caused by an underlying genetic disorder, such hypertrophic cardiomyopathy; Semsarian, Sweeting, & Ackerman, 2015), (ii) electrical (e.g., Wolff Parkinson White syndrome), and (iii) acquired (e.g., infection such as myocarditis). Recently, Engel, Schwartz, and Homma (2016) examined cardiac structure (i.e., cardiac remodelling) in 526 active NBA players, the first large-scale study to provide comparative data for athletes with greater

anthropometry, and indicated that the characterizations of their hearts (e.g., left ventricular cavity sizes), although larger, were generally proportional to body size. Their findings highlighted the importance of standardized testing to account for abnormal physical dimensions of athletes, and argued that full medical context can prevent unnecessary exclusions of athletes from competition and improve clinical acumen (Engel et al., 2016). Altogether, while recent literature has emphasized the heterogeneity of cardiovascular disease and the clinical importance and complexity in accurately portraying risk of SCD in athletes, it has been argued that physical activity can nevertheless precipitate sudden death (Maron et al., 1996).

Other Known Leading Causes of Death in Athletes

Overall, epidemiological studies involving athlete mortality have found the leading causes of death to be similar to those identified in the general population in the developed countries. For example, Lemez, Wattie, and Baker (2015) examined incidence and causes of death of active professional athletes from the NBA, National Football League (NFL), National Hockey League (NHL), and Major League Baseball (MLB) and reported accidents (33.1%), homicides (9.3%), cardiovascular diseases (8.8%), and suicides (6.8%) as the leading causes of death in 205 athletes who were registered as active competitors. Similarly, Harmon and colleagues' (2015) review of 514 NCAA student-athlete deaths reported that accidents were the most common cause of death (50%) followed by medical causes (29%), homicides (8%), and suicides (8%). These are analogous to the leading causes of death in males between 15 and 44 years of age from the general population in the US and Canada (Centers for Disease Control and Prevention, 2014; Statistics Canada, 2014). Moreover, researchers have examined single causes of death and associated risk factors in athletes. For example, Rao and colleagues' investigations of NCAA athletes found a higher rate of suicide in males relative to females and in football

players relative to several other NCAA sports (Rao, Asif, Drezner, Toresdahl, & Harmon, 2015a), as well as a higher incidence of homicide-related deaths in males, black athletes, and football players (Rao, Poon, Drezner, Zigman, Asif, & Harmon, 2015b). Importantly, the cumulative conclusion of Rao and colleagues' findings highlighted that NCAA athletes had significantly lower relative risk of dying of homicide and suicide compared to the general and collegiate population of similar age (2015a; 2015b).

Decades of epidemiologic studies highlight many robust determinants of health and well-being, such as causal relationships between physical activity and reductions in certain chronic diseases. For example, in athletes, evidence has shown that former elite competitors have less incidence of cancer compared to the general population, which can be attributed to lifestyle factors such as abstaining from smoking (e.g., Robsahm et al., 2010; Sormunen et al., 2014). More recently, researchers have begun to focus more on the interplay between health and biological, behavioral, psychological, and social factors (Institute of Medicine (US) Committee on Health and Behavior, 2001). Consequently, as we begin to view health from a more multidimensional perspective, it may become more difficult for an individual to reconcile and comprehend how these various health factors interact with one another and impact their lifespan.

From a broader perspective, as the connection between physical activity and health continues to evolve, it is important for researchers to continue to investigate sport participants, which represent a considerable proportion of the post-industrial and developed population. Larson and Verma's (1999) cross-cultural examination of how children and adolescents spend time has been widely regarded as a seminal and timely consideration of the relationship between participation in sport and other leisure-time activities. They found that among post-industrial populations (particularly in North America and Europe), sport was one of the most common

leisure-time activities youth participated and spent time in (Larson & Verma, 1999). While work comparing participation rates of voluntary activities in youth has been relatively rare (e.g., sport was also identified as the most frequent voluntary activity by Kirshnit, Ham, & Richards, 1989), more contemporary reports still show a general pattern of high participation in sport among youth (i.e., >50%; e.g., Australian Sports Commission Annual Report, 2015; Canadian Heritage, 2013; The United Kingdom Sports Council Annual Report, 2015).

Two predominant goals suggested to improve population health have been to increase the *overall* population health and to reduce purported disparities, such as racial differences in lifespan (Kindig, 2012). However, is the divide in lifespans between certain subgroups of the population (e.g., high disparity in life expectancy at birth between the highest educated white and the lowest educated black Americans; Olshansky et al., 2012) generalizable to high performance athletes, or do common traits within their lifestyles that are valuable to lifespan (e.g., favourable genetic profiles and superior diet and physical activity) neutralize the disparities? Lemez and Baker (2015) highlighted several risk factors and characteristics that researchers have identified as potential precursors of early mortality in athletes; however, although there are a growing number of studies examining mortality in elite athletes, our knowledge on their *causes* of death remains incomplete. Thus, this study examined incidence and causes of death in professional basketball players, a sport where the historical composition of athletes allowed us to evaluate some of the previously suggested robust predictors of longevity in elite athletes.

The NBA (1946-current) and the American Basketball Association (ABA; 1967-1976) contain a historical population of athletes whereby a large quantity may be deceased. Equally pertinent to this study, the popularity of this athletic cohort accompanied with a rich history of high participation rates and earlier inaugural seasons relative to other sports and sporting leagues

(e.g., the Women's National Basketball Association's inaugural season was in 1997) creates a greater opportunity to account for a higher proportion of authenticated deaths in the overall population. The current empirical understanding of mortality in professional basketball players is limited by relatively few studies, which have identified favourable lifespans (Fafian Jr., 1997), no relationship between handedness and lifespan (Lawler & Lawler, 2011a), and a racial disparity through which white players lived on average 1.5 years longer than the black players, although both races lived significantly longer relative to the general population (Lawler, Lawler, Gibson, & Murray, 2012). As such, the overarching purpose of our study was to obtain a more comprehensive understanding of the relationship between high performance sport and mortality through examining causes of death and early and later death predictors in former NBA and ABA players. More specifically, we aimed to initiate a more critical consideration of the interplay between previously identified risk factors and mortality outcomes by gathering data on the entire deceased NBA/ABA population.

Methods

Population and Variables

The population of this study was comprised of all players who played in the NBA and/or ABA from the inception (1946) until the 2009-10 season, and were deceased as of December 11, 2015. The variables collected were related to their biological (i.e., age, birth decade, race, playing BMI, and handedness) and occupational (i.e., career length, playing position, and Hall of Fame (HOF) induction) characteristics. Birth decades were categorized as ≤ 1920 , 1921-1930, 1931-1940, 1941-1950, 1951-1960, and ≥ 1961 . Race was categorized as "black" (deceased biracial players of Caucasian and African-American descent were categorized in this group for the purposes of this study; $n = <5$) or "white" (a single deceased player who was born outside of North America (i.e., Europe) was categorized in this group). BMI was calculated based on the

players' height in inches and weight in pounds, and categorized as either "Normal" (18.5-24.9) or "Overweight" (25-29.9). One player who was considered "Underweight" (<18.5) and one player who was considered "Obese" (>30) were categorized as "Normal" and "Overweight," respectively. Playing positions were categorized as "Guard," "Guard/Forward," "Forward," "Forward/Center" and "Center." As the study did not involve living human participants and data were collected from publicly available sources, ethics review was not required.

Data Collection

Dates of death

As with previous work in this area (Lawler & Lawler, 2011a, 2011b; Lawler et al., 2012), four main online sources were queried to compile our data; two primary online basketball sources, basketball-reference.com and apbr.org (Association of Professional Basketball Research), a general Google search of the player's name followed by "NBA" or "basketball" (typically redirected to wikipedia.org), and/or the Social Security Death Index (SSDI). The SSDI is a freely available database of death records that was accessed through www.ancestry.com to search for deceased players. The collation of data included on the two basketball websites was performed through a collective effort of researchers, employees and volunteers, and the information helped to define the study population and ascertain the dates of birth and death, race, height, weight, handedness, playing position, and the duration of the professional basketball career of each participant. In addition, to examine high achievers, HOF data were gathered from the official Naismith Memorial Basketball HOF website (hoophall.com/hall-of-famers-index), and were fully cross-referenced in basketball-reference.com. Only those individuals who were inducted as *players* (i.e., not inducted as a coach or contributor) were categorized as HOF inductees. Credibility of the retrieved data was assessed by comparing information from the four

web-based sources for agreement. If disparities were apparent, the official encyclopedia of the NBA *Total Basketball* was referenced where applicable (Koppett, Shouler, Ryan, Smith, & Bellotti, 2003). As well, the reported death date on wikipedia.org was used in preference to basketball-reference.com for seven cases (a majority were <10 day difference), and we used “15” as the day of death for nine cases who were missing complete date of death information. All players born <1945 (70+ years of age) were cross-referenced in a minimum of two sources to ensure accurate reporting of lifespan.

Causes of death

Causes of death were collected through publically available web-based sources. In all cases, multiple data sources were searched to verify cause of death information of our sample. Cause of death records were gathered from the following websites: Google.com (player name, followed by a combination of “NBA,” “basketball,” “death” and “cause of death,” or “former NBA death” through Google News), wikipedia.org, apbr.org, nytimes.com, legacy.com (e.g., obituaries; <http://www.legacy.com/memorial-sites/nba/>; <http://www.legacy.com/memorial-sites/nba/profile-search.aspx?beginswith=All>), basketball-reference.com, nba.com, gobulldogs.truman.edu, old.post-gazette.com, articles.latimes.com, sabr.org, highbeam.com, collegehoopedia.com, calbears.com, vindy.com, grfx.cstv.com, exnba.com/tag/dead-nba-players/, sportingnews.com (e.g., <http://www.sportingnews.com/photos/4631498-notable-sports-deaths-of-2015/>), <http://hoopshype.com/2015/12/27/the-members-of-the-nba-family-gone-in-2015/>, espn.go.com (Associated Press), bostonglobe.com, and other local newspapers (e.g., <http://www.baltimoresun.com/news/obituaries/bs-md-ob-walter-budko-20130604-story.html>).

Classifications of Causes of Death

We used the *International Classification of Diseases, Tenth Revision* (ICD) to categorize the different causes of death in our sample, through the online application provided by the World Health Organization, Version: 2016 (World Health Organization, 2016). In addition, listed causes such as “died at home after a long illness” were generally considered to be affected by chronic, natural and/or age-related conditions or diseases that may have been initiated by metabolic syndrome risk factors such as elevated blood pressure and cholesterol levels. Therefore, we subsequently categorized these types of causes as “natural/old age,” as it was highly unlikely that an unnatural cause of death would be reported in that manner. The two overarching classifications of death periodically used in general discussion were “natural” and “unnatural” causes, which encompassed the corresponding cause-specific deaths (natural/old age was considered a cause-specific death that was under the broad classification of “natural causes,” much like other diseases such as cardiac- and cancer-related deaths; i.e., they were not used interchangeably).

Comparisons to the General Population

The National Vital Statistics Reports released by the Centers for Disease Control (CDC) and Prevention in February 2016 were used to compare mortality statistics of our sample to the sex- and race-matched controls from the general US population. The reports included data on the rates of death (“*Deaths: Final data for 2013*,” 2016) and the leading causes (Heron, 2016) for the US general population in 2013. Cause of death data were compared by ICD classification and all data were disseminated using a common epidemiological reporting method of expressing death rates per 100,000 individuals per year, by dividing the number of reported causes of death by the total population (3,901), and multiplying the result by 100,000. This method is also known as the *crude death rate* (CDR), which measures the number of deaths in a population, relative to the

size of the population over a given period of time, and can provide insights into the health status of a population over time (World Health Organization, 2014). CDRs were also calculated separately for white and black players, and both races combined.

Comparisons to Living NBA and ABA Players

Estimates of death rates were reported in athlete-years (AY) by calculating ages of the entire deceased and living NBA/ABA player population who played between 1946 and 2010 (N = 3,901) as of December 11, 2015 (i.e., until the cut-off date for death data collection). Data for living players were collected using the same aforementioned sources.

Calculating Premature Death: Years of Potential Life Lost (YPLL)

It has been suggested that alternative measures to reflect mortality trends of younger age groups may be of added value, particularly to traditional mortality analyses such as CDRs which can provide incomplete understanding of temporal changes in mortality as there is often an inherent susceptibility to emphasize the underlying disease processes of the elderly (Centers for Disease Control, 1986). Thus, YPLL was included as an added measure of young death to estimate how many potential years were lost if a player died prematurely. YPLL were calculated by subtracting the age at death from two separate upper reference ages: from i) a set age of 75 years (YPLL₇₅), which is a common predetermined upper reference age used when calculating YPLL in the developed world (e.g., University of Wisconsin Population Health Institute, 2015; Vila, Booske, & Remington, 2006), and from ii) the life expectancy at birth for each player (YPLL_{LE}). Life expectancies by birth year for the male general US population was accessed through Noymer and Garenne's (2000) study on mortality differences in the United States and were cross-referenced in the latest CDC report ("*Deaths: Final data for 2013*," 2016), although single year life expectancies were not provided until 1975 in the report (i.e., grouped by decade

prior to 1975). Birth year life expectancies were used to calculate YPLL at the time of death as opposed to life expectancies in year that they died to remove the birth cohort effect caused by changing lifespan factors, such as shifting causes of death and social conditions. Players who lived longer than their respective upper reference age were not included in this sub-analysis (i.e., did not die prematurely).

Statistical Analyses

Mortality outcomes were analyzed using i) descriptive and ii) binary and multinomial logistic regression analyses, with the aforementioned biological and occupational variables serving as covariates (i.e., predictors). A t-test median split of age of death dichotomized the outcome variable into “below” or “above” categories. Analyses were performed using SPSS version 23, and statistical significance was defined as $p < 0.05$, at the 95% Confidence Interval (CI). Data were predominantly analyzed descriptively as the entire NBA/ABA player population was accessible; thus, inferential statistics were not needed to predict likelihood estimates. However, regression analyses were utilized to illustrate statistical limitations and potential artifacts of using historical data for likelihood estimates with relevance in the present.

Summary of Measures of Mortality

Vila and colleagues (2006) concede that there is no one best measure that captures all aspects of mortality, and that various methods exist to quantify death rates, including CDRs, cause-specific mortality, age-specific mortality, age-adjusted mortality, YPLL, and life expectancy. Conceptually, all of these methods were either directly or indirectly incorporated in the analyses, although some were limited by the relatively small nature of the population studied (i.e., differentiating causes of death by age group would have drastically lowered the death rate

frequencies, further limiting generalizations to the general population). Below, we summarize the main measures of mortality used for this population.

i) Cause-specific CDRs: compares death rates of the NBA/ABA player population to the general population and by race, reported as per 100,000 individuals.

ii) AY: accounts of living players as well to estimate death rates per AY for causes and early predictors of mortality.

iii) YPLL: measures premature death by calculating YPLL using two upper reference ages ($YPLL_{LE}$ and $YPLL_{75}$). This method differs from the CDR calculation by providing a more accurate portrayal of premature deaths by ranking the leading causes of early death rather than overall numbers of deaths.

iv) Regression Analyses: provides odds ratios of dying at younger and older ages, based on predictors of early mortality.

Results

Descriptives

As of December 11, 2015, 787 former NBA/ABA players were identified as deceased out of a total of 3,901 players who competed between 1946 and 2010 (20.1%). The mean (M) age of death was 68.1 years (y) with a standard deviation (\pm) of 16.0 (range (R) = 24.3 – 99.6 y). Of those 787 former players with a confirmed death date, our search identified 514 causes of death (65.3%) from 16 different ICD groups, 432 of which were from natural causes (i.e., not directly caused by external forces; $M = 68.9 y \pm 14.6$) and 82 from unnatural or premature causes (e.g., accident, suicide and homicide; $M = 50.8 y \pm 19.5$). Further, the top two leading causes of death were from diseases of the circulatory system (i.e., cardiac-related) and malignant neoplasms (i.e., cancer), both natural causes, and accounted for 54.6% (281/514) of all-known deaths in the

population. The youngest ages of death on average occurred from certain infectious and parasitic diseases ($n = 2$; $M = 39.1 \text{ y} \pm 4.5$) and intentional self-harm ($n = 6$; $M = 43.0 \text{ y} \pm 15.5$) and the oldest from diseases of the digestive ($n = 4$; $M = 80.8 \text{ y} \pm 3.5$) and genitourinary system ($n = 5$; $M = 79.0 \text{ y} \pm 17.6$). A high majority of the located causes of death were cross-verified in another data source ($>80\%$). See Table 5.1 for a complete list of causes of death and CDRs by ICD classification and race, and Figures 5.1 to 5.3 for illustrations of the leading causes of known death of this population and age of death distributions.

Of the biological variables collected, a majority of the deceased NBA/ABA player population were white ($n = 591$; 75.1%), right-handed ($n = 758$; 96.3%), classified as “Normal” on the BMI scale during their playing career ($n = 621$; 78.9%) and were born in 1940 or earlier ($n = 579$; 73.6%). Of the occupational variables collected, a majority were Guards ($n = 216$; 27.4%), non-inductees into the HOF as a player ($n = 751$; 95.4%) and played on average for 3.61 years (± 3.4 ; Median = 2 years; one player played a high of 21 years). Table 5.3 shows average ages of death for all of the predictors collected.

Comparisons to the general population

All known causes of death in NBA/ABA players who competed between 1946 and 2010 were compared to data from the 2013 US general population by i) CDRs and race, where cause-specific CDRs were reported by the average annual instance per separate calendar year calculated between 2010 and 2015 (Table 5.1), and by ii) CDRs for each year between 2010 and 2015 for the two leading causes of natural and unnatural death (Table 5.2). With regard to Table 5.1, since our comparative data were from a single year (2013), standardized CDRs were provided by calculating athlete causes of death over the past six years and producing an average. Due to the historical nature of this examination, investigating CDRs across multiple decades and

providing a rate for each year in which a death occurred in our population would have lacked usefulness and been largely redundant. For example, although 61.7% of our population died within the last 15 years (486/787), the first reported death dates back to 1957 (theoretically, a death could have occurred anywhere from the inaugural professional basketball season in 1946 until December 11, 2015 in which the athlete would have entered our deceased sample). Further, Table 5.2 reported the leading causes of death by individual year, and only one instance of the second leading cause of unnatural death occurred since 2010 (i.e., homicide in 2010). This illustrates how fluctuation of cause-specific deaths in a relatively short time period can easily alter CDRs, particularly in a relatively smaller population.

Nevertheless, considering the differences in population size, our findings depict moderately similar CDRs across causes of death of NBA/ABA players compared to the sex- and race-matched controls from US general population (see Tables 5.2 and 5.3). For example, the average CDR of malignant neoplasm across the last six years for the NBA/ABA player sample was 187.9 relative to 197.6 for the general population. CDR for circulatory disease was nearly identical between players in 2015 (256.3) and controls in 2013 (257.4). Race-matched comparisons were also similar for causes such as accidents, where white players had a CDR of 55.2 compared to 56.8 for the general population, whereas other causes such as diseases of the nervous system (e.g., Alzheimer's disease) for white players (77.3) were less similar relative to the controls (19.7). Interestingly, CDR discrepancies were also evident in the absence of certain causes of death for the player sample since 2010, such as no reported deaths from endocrine, nutritional and metabolic disease (e.g., diabetes mellitus) for white players (25.7 for race-matched controls), no intentional self-harm (i.e., suicide) for white players (23.4 for race-matched controls), one external cause of morbidity and mortality (i.e., homicide) for black

players (7.5 vs. 33.1 for race-matched controls), and no incidences of digestive system or infectious and parasitic diseases for either race.

Athlete-Year

There were a total of 219,561 AY, with the highest representation of players who were non-inductees into the HOF (211,921 AY), right-handers (208,678 AY) and players with normal BMIs (173,009 AY). Overall, incidences of natural deaths per AY were more common across all predictors of mortality compared to unnatural deaths. Understandably, the most common rates of death were for players born in 1920 or earlier from natural causes (occurring once every 174 AY), particularly from cardiac diseases (1:450 AY), and the least from cancer in those players born in 1961 or later (1:12,136 AY). Other predictors of mortality that were less time-dependent (i.e., were more likely to stay constant over time) such as career length, handedness and playing position did not appear to have considerable differences in rates of death per AY (e.g., death from cancer for right-handers occurred at a rate of 1:1,592 AY compared to 1:1,554 AY for left-handers). Table 5.3 provides the estimates of the rates of natural, unnatural, cardiac and cancer deaths in athletes across all variables collected.

Years of Potential Life Lost

Table 5.4 shows cause-specific YPLL by both life expectancy and race. Out of the total of 787 deceased players, 59.4% (468/787; 7,951.75 YPLL using an upper reference age of 75 years) and 28.7% (226/787; 3,608.09 YPLL using life expectancy at birth as the upper reference age) died prematurely. Interestingly, there was a 30.7% decrease in NBA/ABA players whose deaths were classified as premature when using the more specific measure of individual life expectancy at birth, a reduction of 4,343.66 YPLL. Notably, the ranking of the overall leading causes of premature death in the deceased player population was the same as the leading causes

of all death, with the exception of diseases of the nervous system, where only four out of the 16 incidences were considered premature when using 75 years as the upper reference age for life expectancy. Premature deaths were primarily due to diseases of the circulatory system (YPLL₇₅, 1,813.15; YPLL_{LE}, 840.62), malignant neoplasms (YPLL₇₅, 1,358.56; YPLL_{LE}, 566.54) and accidents (YPLL₇₅, 1,097.09; YPLL_{LE}, 761.21). Predictably, a higher overall percentage of premature deaths were from unnatural (YPLL₇₅, 84.1%, 69/82; YPLL_{LE}, 67.0%, 55/82) than natural (YPLL₇₅, 59.7%, 258/432; YPLL_{LE}, 29.8%, 129/432) causes. The highest percentages of cause-specific premature deaths with more than 10 cases were from homicides (YPLL₇₅, 100%, 21/21; YPLL_{LE}, 85.7%, 18/21) and accidents (YPLL₇₅, 76.7%, 33/43; YPLL_{LE}, 60.4%, 26/43).

As it pertains to race (calculated using YPLL_{LE}), black players had more overall YPLL compared to white players (2,242, n = 136 vs. 1,365.68, n = 90, respectively), and a higher mean of YPLL per player (16.4 vs. 15.1, respectively). Notably, all five premature incidences of death from respiratory system diseases and three incidences of endocrine, nutritional and metabolic diseases occurred in black players, in addition to having a far greater mean of YPLL from homicide compared to white players (26.2 vs. 14.8, respectively). Below, the influence of racial disparities in the earlier decades of the NBA on mortality outcomes is discussed.

Statistical Biases from Regression Analyses

As highlighted previously, we deemed descriptive statistics to be the most appropriate methodological approach to analyze our population data, considering there was little to infer. On the other hand, analyzing likelihood estimates may also be useful in this circumstance since the NBA player population continues to grow each year as new cohorts of players get drafted into the league. In addition, inferential statistics in this regard also help to highlight the potential for

misinterpretations of early mortality risk and causes of death for certain predictor variables due to the nature of the population analyzed.

Lifespan predictors

Race. Table 5.5 reports unadjusted and adjusted odds ratios (OR) for all of the biological and occupational variables collected. Notably, a significantly (unadjusted) higher likelihood of dying below the median age of death (71.0 years) was found in black players (OR 10.32, 95% CI: 6.65 – 16.02); however, it is important to consider cohort effects resulting from racial disparities in the earlier decades of professional basketball in North America. In the NBA/ABA population, we found low participation of black players relative to white players born in 1930 or earlier (12 vs. 539) or between 1931 – 1940 (89 vs. 184), and in contrast, higher participation for black players relative to white players born between 1941 – 1950 (373 vs. 307), 1951 – 1960 (451 vs. 180) and 1961 – 1990 (1,296 vs. 298). These disproportions create statistical artifacts when using race as a predictor of lifespan and cause of death. For a player to enter the population analyzed in this study they had to be deceased. Consequently, black players become artificially predisposed to having a relatively younger age of death and to die from unnatural causes; 73.5% of deceased black players were born in 1941 or later ($n = 144$), which allow them to live a maximum of 74 years when using December 11, 2015 as the cutoff. In contrast, the higher proportion of white players who were born earlier, and thus debuted in the league in earlier decades, had a higher likelihood to live a full life and die from a natural cause. As a result, the mean age of death for white players was 72.2 years (± 14.3) compared to 55.5 years (± 14.1) for black players. This statistical bias was supported in YPLL outcomes and also illustrates multicollinearity, which exists when there is a strong correlation between two or more predictors (Field, 2013); Pearson bivariate correlations revealed that race and birth decade were

significantly correlated with one another ($r = .648, p < .05$). When race was adjusted for birth decade, the previously inflated significant OR of 10.32 for black players dying below the median age of death became insignificant (OR 1.67, 95% CI: .915 – 3.05).

Height. Players who were 77 inches (i.e., 6'5") or taller were also found to have a significantly higher likelihood of dying below the median age of death (OR 2.70, 95% CI: 2.02 – 3.61). Similarly, birth decade should be taken into account; 77.2% of deceased players who were below the median height (≤ 76 inches) were born in 1930 or earlier ($n = 325$). Thus, when height was adjusted for birth decade (i.e., multicollinear predictors; $r = .460, p < .05$), height became an insignificant predictor of lifespan (OR 1.27, 95% CI: .88 – 1.84). Therefore, having been born in earlier decades and playing in the NBA at a time when the average height was relatively shorter allowed the deceased players in this study a greater opportunity to live a full life.

Cause of death predictors

Similar caution should be given when interpreting cause of death predictors. Logistic regressions revealed that white players had a significantly higher incidence of death from natural/old age causes compared to black players (OR 2.46, 95% CI: 1.38 – 4.37), and black players had a significantly higher incidence of death from homicide compared to white players (OR 2.53, 95% CI: 1.05 – 6.10). Further, natural/old age was used as the referent group in a multinomial logistic regression which revealed that white players had a significantly lower likelihood of dying from cardiac disease (OR 0.36, 95% CI: .195 – .697), cancer (OR 0.45, 95% CI: .235 – .859), homicide (OR 0.19, 95% CI: .070 – .526), suicide (OR 0.10, 95% CI: .018 – .625) and respiratory, endocrine, genitourinary, digestive and mental diseases (OR 0.34, 95% CI: .141 – .817) than they are of natural/old age causes relative to black players. This further corroborates previous trends where decade of play influences lifespan outcomes; in this case, the

nature of the population analyzed biases the influence of cause of death predictors such as race by allowing those who were born earlier (i.e., predominantly white players) to have a greater likelihood of dying from natural/old age causes (e.g., metabolic syndrome risk factors) relative to black players.

Discussion

The purpose of this investigation was to expand the limited empirical understanding of mortality in professional basketball players by a) ascertaining cause of death knowledge in former NBA/ABA players, and b) examining how exposure to certain biological and occupational variables influences mortality. Our overarching findings indicated that i) NBA/ABA players have similar leading causes of death and CDRs to sex- and race-matched controls from the US general population, ii) differences in death rates per AY exist within time-dependent variables (e.g., birth decade) but less in more stable variables (e.g., handedness), and overall incidences of unnatural deaths per AY were less common across all predictors of mortality relative to natural deaths, and iii) a majority of the deceased player population died prematurely if the life expectancy was set at 75 years for each player, but less than 30% were considered young deaths if life expectancy at birth for each player was individually calculated. In addition, this investigation highlighted the potential dangers of disseminating likelihood estimates for certain predictors of early mortality in a population where the demographics have considerably evolved since its inception.

Gillett, Ross and Switzer (2015) highlighted two predominant storylines surrounding sport and the malleability of chronological age that are perpetuated through conversations and different media outlets; i) warning of the dangers and destructive potential of competitive sport, and ii) the transformative capacity of sports that allows individuals to overcome personal and

social challenges and constraints. While this ‘power of sport’ narrative is diverse (Gillett et al., 2015), the vast dissemination of information through social media can dominate how we perceive certain issues. Thus, chronological age (i.e., early mortality risk) may actually be perceived less ‘malleable’ and more rigid in elite athletes, augmented through media’s predisposition to report a premature death rather than a death in old age (particularly for the earlier unheralded players). This supports the comprehensive undertaking of mortality measurements used in this study, which allowed us to examine the nuances and portrayals of death in more depth concerning the deceased NBA/ABA player population.

The absence of rigorous methodology when examining a historically evolving population such as NBA participants can promote common oversights of death risks and propagate negative stereotypes. For example, parameters of time-dependent predictors of mortality were highly impacted in both the AY and regression analyses. More specifically, white and relatively shorter players were artificially predisposed to living longer than black and taller players since they made up a majority of the participants in the earlier decades; the unique shift in North American professional basketball participant demographics (even though nearly all deceased players were US-born) resulted in black and taller players entering the NBA in later decades, giving them less opportunity to live a full life since our analyses only included deceased players. In turn, while there was a noticeable difference between races for a cause of death such as homicide, where there were more deaths in black players ($n = 11$) relative to white players ($n = 10$) despite the significantly higher proportion of white deceased players (591/787), an odds ratio of 2.53 estimating high risk of homicide for black players may be misleading.

The CDR comparison to the general population provided a more standardized method of informing us how deaths in high performance athletes may relate to a less active population.

While there was a considerable discrepancy between the population sizes in our comparison, alongside elite athletes having superior longevities (Lemez & Baker, 2015), little variability was found in the leading causes of death and CDRs between the players and the general population. Interestingly, it appears that there were no apparent protective effects of exercise for the athlete group in this mortality measure. Another interesting finding was that there appeared to be a relatively lower number of premature deaths (YPLL) when each player's life expectancy at birth was considered (28.7%). This notion was supported through a 2014 CDC press release which stated that approximately 900,000 Americans die prematurely from the five leading causes of death alone each year ("*CDC Newsroom*," 2014), and accounting for the 2,596,993 reported deaths in the US in 2013 ("*Deaths: Final data for 2013*," 2016), that equates to at least 34.6% of the general US population deaths being classified as premature. However, it is also important to recognize that players born earlier in the 20th century had lower life expectancies at birth, thus potentially reducing the number of premature death classifications which would otherwise be considered a young death today.

Limitations

A key limitation of this study in reporting vital statistics of former NBA/ABA players was that it was a non-forensic-based investigation; therefore, we were limited to how the causes of death were reported publically. The World Health Organization (2014) emphasizes that when assigning causes of death that it is critical that deaths are classified by the *underlying* cause (i.e., the cause that initiated the events leading to the death) rather than the *immediate* cause of death. This approach is more helpful in generating information that is useful for public health purposes (World Health Organization, 2014). For example, professional wrestler "Rowdy" Roddy Piper's death in 2015 at the age of 61 years was due to cardiac arrest; however, hypertension caused his

chronic blood pressure, which created blood clots resulting in a pulmonary embolism that triggered his heart attack. As it relates to the ICD classification adopted in this study, another example includes endocrine, nutritional and metabolic disease being a precursor to disease of the circulatory system. Unfortunately, cause of death can be complicated by a number of contributing known and unknown underlying factors that are generally not reported publically, such as genetics and inheritance of diseases, along with a number of behavioural/preventable risk factors such as smoking and unhealthy diet. In the NBA, for example, anecdotal evidence of rampant drug use amongst players in the 1970s and early 1980s may have detrimentally impacted the cardiac longevity of players of this era, which would be considered an unnatural contributor to a 'natural cause' of death. While this limitation particularly applies to our incomplete knowledge of the underlying causes that contributed to *natural* deaths such as heart disease and stroke, which may have been precipitated by other factors such as prior drug use, reporting of unnatural deaths could have also confounded our results (e.g., suicide vs. alcohol-related death). In addition, our inclusion of natural/old age as a cause of death category is a broad classification that may include more precise illnesses and diseases that led to players' deaths that went unreported. Therefore, our findings may underestimate the importance of diseases that contribute to the underlying causes of deaths (Centers for Disease Control, 1986).

A limitation of using CDRs of our athletic population to compare mortality trends to the general population was that they are physically dissimilar populations. Further, Vila and colleagues (2006) suggested that a problem with this mortality measure is that the age distributions between the two populations may be different, which can confound the results. Consequently, the CDRs for the US general population in 2013 were for all age groups, whereas the athlete population had to have reached a certain age in order to be included in this analysis

(i.e., age in which they debuted in the NBA or ABA). Another common concern of this measure is comparing across time periods since mortality outcomes can vary considerably; however, we adjusted for this by calculating annual CDRs between 2010 and 2015 and reporting the average.

Last, while CDRs treat each death equally, using YPLL as a measure of mortality has been criticized for valuing premature death more than death at later ages, and the chronic and other conditions that contribute to it (Dranger & Remington, 2004; Vila et al., 2006). In addition, the year-to-year changes in the total and cause-specific rates of YPLL can alter how we rank the leading causes of YPLL among population subgroups (Centers for Disease Control, 1986). For example, we found black players to have 288.74 YPLL_{LE} from homicide (Table 5.4); however, we also revealed that only one incident of homicide in the NBA/ABA player population has occurred in the last six years (Table 5.2). While rates of premature mortality have been found to differ among racial groups (Centers for Disease Control, 1986), these year-to-year changes can create cause for concern to intervene and target groups that are unnecessarily perceived to be at high risk.

Data Retrieval Limitations

In Link and Estes' (2012) study on SCD, they drew attention to the tendency of athletes' deaths to be nationally noted, relative to lesser media speculation given to non-athletes, which often skews the public's perception of risk. Comparatively, we were unable to locate cause of death for 273 persons in our study (34.7%). While ultimately it is the family's decision of whether to disclose a cause of death, arguably, athletes who were more identifiable generally have a cause that accompanies the news of their death. In further examining this phenomenon in our population, two indicators of high achievement and recognizability attained were HOF and career longevity; only three were inducted into the HOF (1.1% vs. 4.6% who were inducted in

the overall deceased population of 787 players) and a majority played only two seasons or less in the NBA or ABA (69.5% vs. 54.5% overall) of the 273 former players whose cause of death data were undisclosed. Interestingly, while these outcomes appear to support common anecdotal assumptions of disproportional media coverage in favour of the better recognized athletes after they die, it is important to note that birth cohort effects may also play a factor. More specifically, 205 of the 273 (75.1%) former athletes who were missing a cause of death were born in 1930 or earlier, compared to 59.5% of former players in the overall deceased sample (468/787). In addition, only 15.3% (42/273; $YPLL_{LE}$) of players with unknown causes of death died prematurely, which suggests that it is less likely that a cause of death is publically reported if the player died later in life. Thus, cause of death data retrieval limitations are likely a result of a combination of inaccessible accurate death data of players from earlier generations and disparities between achievement/recognizability and relative obscurity amongst the players (i.e., media subjectivity in reporting).

Future Directions

Although there appears to be obvious caveats and inherent biases to disseminating mortality outcomes in a historically evolving population such as the one studied, the mortality measures utilized also offer several strengths and complement one another. For example, by knowing the (immediate) reported cause of death, it may inadvertently illuminate the morbidity caused by the disease (Vila et al., 2006), thus providing researchers an avenue for secondary examinations into the underlying causes of death and potential risky behaviours that preceded it. As well, $YPLL$ measures of mortality can be used for prevention of future premature deaths in athletes. More specifically, death at younger ages has been suggested to be more attributable to preventable causes, which is desirable from an economic and social standpoint (Dranger &

Remington, 2004). By determining the relative importance of premature causes of death in a subpopulation, effective interventions and resources can be targeted towards the high risk groups in an attempt to extend lifespans and modify negative lifestyle and behavioural factors.

Conclusion

From a broader perspective, despite the relative eminence of professional athletes, comprehensive knowledge on their causes of death is limited. While it is important to respect that families of the deceased may not want to publically release cause of death knowledge, a media/content repository that collates and publicizes the causes of death if they are released, regardless of the level of popularity of the athlete, could advance sport medicine, provide broad public health applications, and help to disarm potentially harmful stereotypes of health in elite sport. This study found that deceased NBA/ABA players had similar causes of death to the general population, along with lower premature death if life expectancy at birth was considered, yet questions still remain, such as to what extent these results are generalizable to other high performance athletes and whether superior athletic ability transcends general life expectancies. It will also be worthwhile for future research to examine physical activity and other health patterns of high performance athletes after they retire, and to compare their health outcomes to similarly active controls. Therefore, in accordance with the growth in social media usage, researchers and practitioners alike should negotiate best avenues to improve health and the transparency between different mediums of information concerning health in athletes in order to reconcile otherwise limited empirical evidence.

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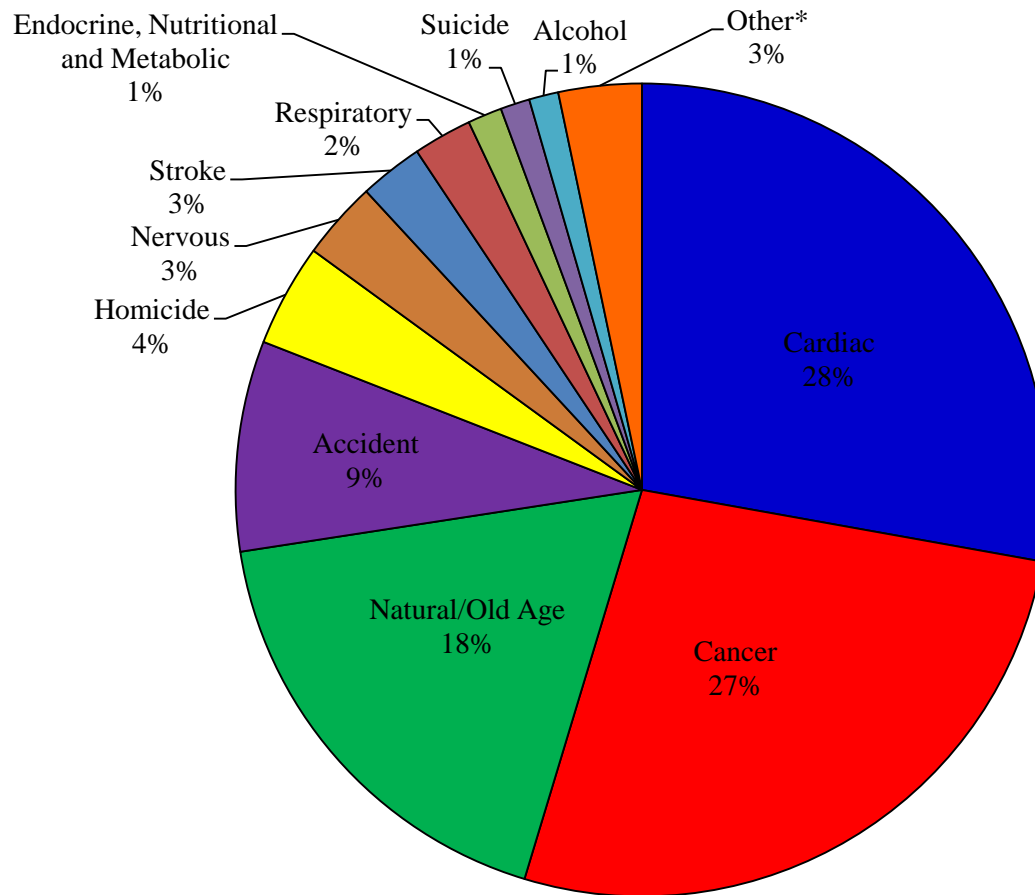
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Figure 5.1: Causes of known death in former NBA and ABA players, by percentage (N = 514)



*Other: Disease of the Genitourinary System (n = 5), Disease of the Digestive System (n = 4), Complication of Surgical and Medical Care (n = 4), Poisoning by Drugs, Medicaments and Biological Substances (n = 2), Certain Infectious and Parasitic Disease (n = 2)

Figure 5.2: Leading causes of known death in former NBA and ABA players, by age of death (in years)

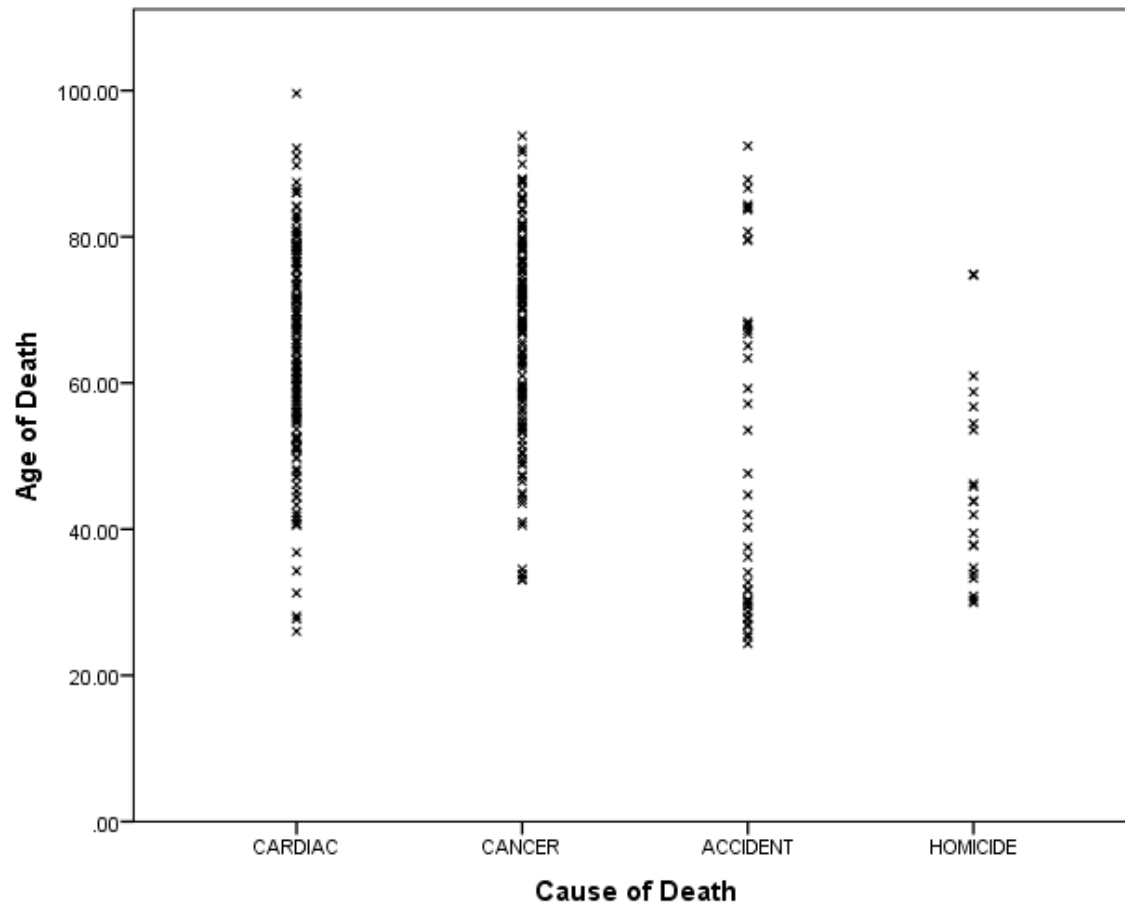


Figure 5.3: Age distributions of death in former NBA and ABA players, by frequency

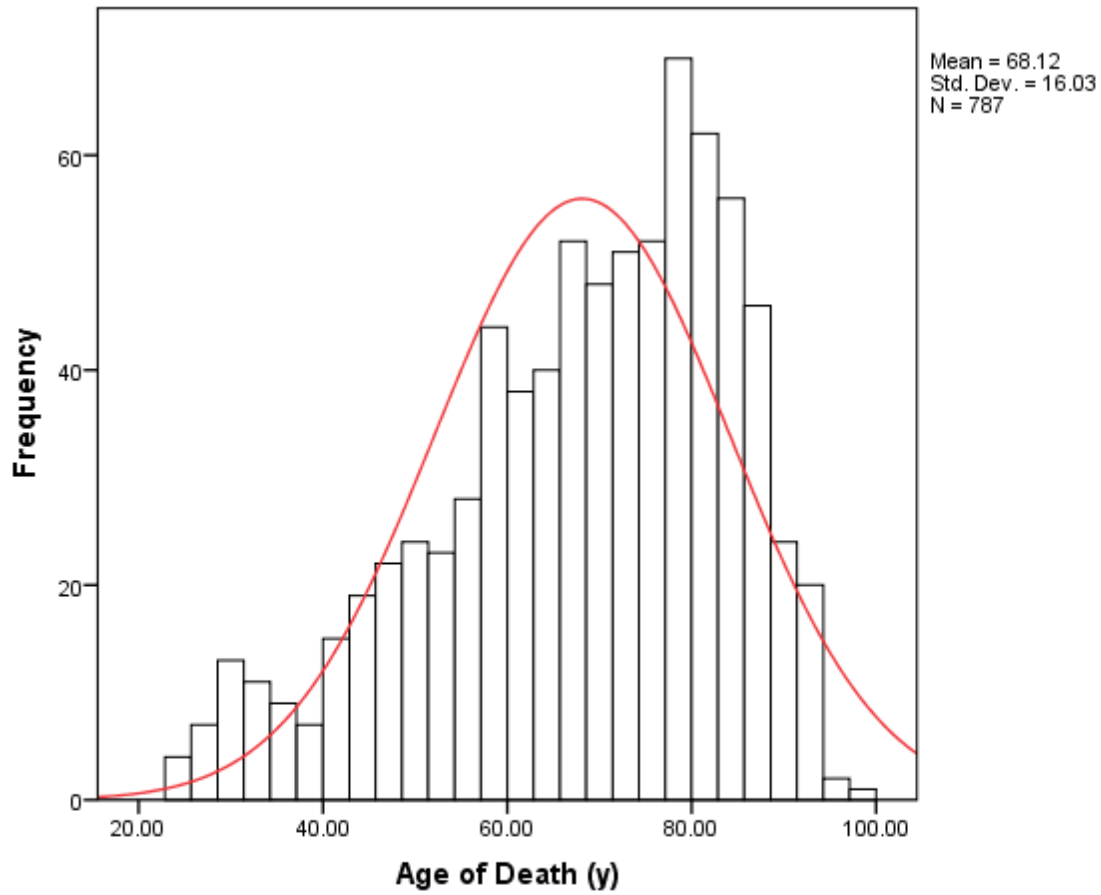


Table 5.1: Cause-specific CDRs, by race

	ICD Classification	n	Age (yrs)	Race (n)		NBA/ABA Players 1946-2010: Deaths/100,000 ¹		Male US General Population 2013: Deaths/100,000 ²			
				W	B	All Races	W (n)*	B (n)*	All Races	W	B
Natural Causes	Disease of the Circulatory System ^a	143	63.9 ± 14.4	91	52	145.2	154.7 (14)	150.0 (20)	257.4	275.3	230.8
	Malignant Neoplasm ^b	138	67.2 ± 13.7	94	44	187.9	309.4 (28)	120.0 (16)	197.6	213.0	165.6
	Natural/Old Age ^c	92	77.1 ± 10.8	76	16	158.0	320.5 (29)	60.0 (8)	³ 83.2	90.7	66.7
	Disease of the Nervous System ^d	16	73.8 ± 16.9	14	2	34.1	77.3 (7)	7.5 (1)	⁴ 16.6	19.1	7.9
	Cerebrovascular Disease ^e	13	70.8 ± 10.7	10	3	12.8	11.0 (1)	15.0 (2)	34.5	35.8	35.1
	Disease of the Respiratory System ^f	12	72.3 ± 19.9	7	5	34.1	66.3 (6)	15.0 (2)	45.2	51.6	24.2
	Endocrine, Nutritional and Metabolic Disease ^g	7	67.5 ± 14.2	2	5	8.5	-- (0)	15.0 (2)	25.6	25.7	30.5
	Disease of the Genitourinary System ^h	5	79.0 ± 17.6	4	1	12.8	22.1 (2)	7.5 (1)	0.1	0.1	0.1
	Disease of the Digestive System ⁱ	4	80.8 ± 3.5	4	0	--	-- (0)	-- (0)	⁵ 15.2	16.9	9.3
	Certain Infectious and Parasitic Disease ^j	2	39.1 ± 4.5	1	1	--	-- (0)	-- (0)	2.0	2.1	2.0
Unnatural Causes	Accident ^k	43	51.6 ± 22.9	31	12	29.9	55.2 (5)	15.0 (2)	52.6	56.8	43.1
	External Cause of Morbidity and Mortality ^l	21	45.8 ± 13.5	10	11	4.2	-- (0)	7.5 (1)	8.2	4.4	33.1
	Intentional Self-Harm ^m	6	43.0 ± 15.5	2	4	4.2	-- (0)	7.5 (1)	20.6	23.4	9.0
	Mental and Behavioral Disorder ⁿ	6	53.7 ± 12.6	4	2	8.5	-- (0)	15.0 (2)	⁶ 13.7	15.0	9.1
	Complication of Surgical and Medical Care ^o	4	68.6 ± 16.1	4	0	--	-- (0)	-- (0)	0.9	0.9	0.9
	Poisoning by Drugs, Medicaments and	2	63.74 ± 2.2	0	2	8.5	-- (0)	15.0 (2)	⁷ 18.2	20.2	13.5

Biological Substances^p

Unknown	273	72.0 ± 13.4	237	36	--	--	--	--	--	--
TOTAL	787	68.1 ± 16.0	591	196	649.4	1,016.7	450.2	839.1	899.1	739.3

^aIschaemic heart disease (n = 139), diseases of the arteries, arterioles and capillaries (n = 3), pulmonary embolism (n = 1); ^bUnspecified cancer (n = 74), leukemia (n = 11), prostate (n = 11), brain (n = 9), lung (n = 9), liver (n = 4), colon (n = 3), bladder, bone, esophagus, multiple myeloma, pancreas (n = 2), bone marrow, colon and liver, kidney, melanoma, myelodysplastic syndrome, rectum, throat (n = 1); ^cReported causes included: natural causes, died peacefully, died at home, died after a brief illness, died after a long illness, and died from various illnesses (e.g., metabolic syndrome); ^dAlzheimer’s disease (n = 7), Parkinson’s disease (n = 4), epilepsy (n = 2), Amyotrophic Lateral Sclerosis (ALS), brain aneurysm, brain disease (n = 1); ^eStroke (N = 13); ^fPneumonia (n = 7), cardiorespiratory failure (n = 2), influenza, lung disease, pulmonary fibrosis (n = 1); ^gDiabetes mellitus (n = 6), amyloidosis (n = 1); ^hKidney failure (N = 5); ⁱLiver disease, peritonitis, portal hypertension, ulcer (n = 1); ^jAcquired Immune Deficiency Syndrome (AIDS), complications from staphylococcal infection (n = 1); ^kCar accident (n = 27), fall (n = 6), drowning (n = 4), plane crash (n = 2), All-Terrain Vehicle (ATV) accident, falling tree, injury, motorcycle crash (n = 1); ^lHomicide (N = 21); ^mSuicide (N = 6); ⁿAlcohol abuse (N = 6); ^oSurgery complication (n = 3), pacemaker infection (n = 1); ^pDrug overdose (N = 2)

Standard Deviation = ±; ¹Total population = 3,901 (white = 1,508; black = 2,221); Cause-specific CDRs for the NBA/ABA player population were reported by the average annual incidence between the years of 2010 and 2015; For consistently in general population comparisons, only white players of North American nationality were included in this statistic (i.e., does not include European, Australian, or New Zealand players); ²Data from the Centers for Disease Control and Prevention (CD) national vital statistics reports (“Deaths: Final data for 2013,” 2016; Heron, 2016); ^{*}Total frequencies between 2010 and 2015; ³Data for “All other diseases”; ⁴Data for “Alzheimer’s disease”; ⁵Data for “Chronic liver disease”; ⁶Data for “Alcohol induced deaths”; ⁷Data for “Drug induced deaths”

Table 5.2: Cause-specific CDRs for leading causes of natural and unnatural death, by individual year (2010 – 2015)

ICD Classification		NBA/ABA Players 1946-2010: Deaths/100,000 ¹										Male US General Population 2013: Deaths/100,000 ²		
		2010		2011		2012		2013		2014			2015 ³	
		n	Athletes	n	Athletes	n	Athletes	n	Athletes	n	Athletes	n	Athletes	
Natural Causes	Disease of the Circulatory System	6	153.8	7	179.4	6	153.8	2	51.2	3	76.9	10	256.3	257.4
	Malignant Neoplasm	5	128.1	9	230.7	11	281.9	9	230.7	3	76.9	7	179.4	197.6
Unnatural Causes	Accident	--		2	51.2	3	76.9	1	25.6	--		1	25.6	52.6
	External Cause of Morbidity and Mortality	1	25.6	--		--		--		--		--		8.2

¹Total population = 3,901; ²Data from the Centers for Disease Control and Prevention (CD) national vital statistics reports (“Deaths: Final data for 2013,” 2016; Heron, 2016); ³Causes of death were collected until December 11, 2015

Table 5.3: Predictors of mortality (N = 787) and incidence of known death (N= 514) per Athlete-Year (N = 3,901^a)

Predictors	Deceased Players			AYs ^a	NAT	INC	UNN	INC	Cardiac	INC	Cancer	INC
	(n)	%	Age (yrs)		Death	/ AY	Death	/ AY	Death	/ AY	Death	/ AY
Overall	787	100.0	68.1 ± 16.0	219,561 ^a	432	1 in 508	82	1 in 2,677	143	1 in 1,535	138	1 in 1,591
Race												
White	591	75.1	72.2 ± 14.3	100,202 ^b	303	1 in 330	51	1 in 1,964	91	1 in 1,101	94	1 in 1,065
Black	196	24.9	55.5 ± 14.1	112,542 ^c	129	1 in 872	31	1 in 3,630	52	1 in 2,164	44	1 in 2,557
Career Length ^d												
≤ 1-y	315	40.0	70.9 ± 15.1	68,155	146	1 in 466	28	1 in 2,434	36	1 in 1,893	48	1 in 1,419
2-y	114	14.5	67.6 ± 16.2	25,369	52	1 in 487	13	1 in 1,951	18	1 in 1,409	16	1 in 1,585
3-y	74	9.4	68.6 ± 16.2	17,663	45	1 in 392	5	1 in 3,532	16	1 in 1,103	9	1 in 1,962
4+-y	284	36.1	65.0 ± 16.3	95,271	189	1 in 504	36	1 in 2,646	73	1 in 1,305	65	1 in 1,465
Handedness												
Right	758	96.3	68.3 ± 15.8	208,678	412	1 in 506	77	1 in 2,710	135	1 in 1,545	131	1 in 1,592
Left	29	3.7	61.2 ± 19.2	10,883	20	1 in 544	5	1 in 2,176	8	1 in 1,360	7	1 in 1,554
BMI												
Normal	621	78.9	68.2 ± 15.8	173,009	350	1 in 494	67	1 in 2,582	104	1 in 1,663	112	1 in 1,544
Overweight	166	21.1	67.6 ± 16.5	46,552	82	1 in 567	15	1 in 3,103	39	1 in 1,193	26	1 in 1,790
Birth Decade												
≤ 1920	109	13.9	76.7 ± 12.4	8,557	49	1 in 174	7	1 in 1,222	19	1 in 450	9	1 in 950
1921-1930	359	45.6	75.2 ± 11.9	34,356	183	1 in 187	24	1 in 1,431	48	1 in 715	53	1 in 648
1931-1940	111	14.1	66.1 ± 13.7	20,226	69	1 in 293	9	1 in 2,247	21	1 in 963	32	1 in 632
1941-1950	118	15.0	56.6 ± 12.4	46,232	71	1 in 651	16	1 in 2,889	23	1 in 2,010	30	1 in 1,541
1951-1960	53	6.7	51.2 ± 9.6	37,370	35	1 in 1,067	14	1 in 2,669	17	1 in 2,198	8	1 in 4,671
≥ 1961	37	4.7	39.6 ± 9.1	72,819	25	1 in 2,912	12	1 in 6,068	15	1 in 4,854	6	1 in 12,136
Position												
Guard	216	27.4	69.1 ± 15.1	77,592	117	1 in 663	23	1 in 3,373	31	1 in 2,502	42	1 in 1,847
Guard/Forward	145	18.4	70.5 ± 15.1	22,172	85	1 in 260	9	1 in 2,463	27	1 in 821	25	1 in 886
Forward	190	24.1	65.8 ± 16.9	69,531	100	1 in 695	24	1 in 2,897	38	1 in 1,829	27	1 in 2,575
Forward/Center	155	19.7	68.3 ± 16.3	24,105	79	1 in 305	20	1 in 1,205	26	1 in 927	27	1 in 892
Center	81	10.3	65.8 ± 16.4	26,159	51	1 in 512	6	1 in 4,359	21	1 in 1,245	17	1 in 1,538
Height												
≤ 76 in	421	53.5	72.3 ± 13.9	88,203	221	1 in 399	34	1 in 2,594	70	1 in 1,260	65	1 in 1,356
≥ 77 in	366	46.5	63.2 ± 16.8	131,358	211	1 in 622	48	1 in 2,736	73	1 in 1,799	73	1 in 1,799
HOF												

Inductee	36	4.6	71.1 ± 16.2	7,640	31	1 in 246	2	1 in 3,820	13	1 in 587	8	1 in 955
Non-inductee	751	95.4	67.9 ± 16.0	211,921	401	1 in 528	80	1 in 2,649	130	1 in 1,630	130	1 in 1,630

AY = Athlete-Year, INC / = Incidence per, NAT = Natural, UNN = Unnatural; ^aIncludes both living and deceased players who played in the NBA or ABA between 1946 and 2010; Ages for living players were calculated as of December 11, 2015 (i.e., until the cut-off date for death data collection); ^bNorth American nationality (i.e., does not include European, Australian, or New Zealand players); ^c27 players were identified to be of African nationality, and were included in this statistic; ^dLiving players who were active past the 2009-2010 season were excluded (n = 398) for consistency (i.e., *full* career length only)

Table 5.4: Cause-specific YPLL, by life expectancy and race

Cause of premature death	YPLL Sum (Mean per player; Number of premature deaths)			Race		
	YPLL ₇₅	YPLL _{LE}	Rate Change*	W _{LE}	B _{LE}	
Natural Causes	Disease of the Circulatory System	1,813.15 (17.1; 106)	840.62 (14.4; 58)	-972.53	262.82 (13.1; 20)	577.80 (15.2; 38)
	Malignant Neoplasm	1,358.56 (14.4; 94)	566.54 (12.5; 45)	-792.02	230.79 (14.4; 16)	335.75 (11.5; 29)
	Natural/Old Age	325.38 (9.8; 33)	82.56 (8.2; 10)	-242.82	22.39 (7.4; 3)	60.17 (8.5; 7)
	Disease of the Nervous System	94.30 (23.5; 4)	62.16 (31.0; 2)	-32.14	35.81 (n = 1)	26.35 (n = 1)
	Cerebrovascular Disease	90.67 (11.3; 8)	15.88 (5.2; 3)	-74.79	3.42 (n = 1)	12.46 (6.2; 2)
	Disease of the Respiratory System	115.48 (23.0; 5)	70.88 (14.1; 5)	-44.6	--	70.88 (14.1; 5)
	Endocrine, Nutritional and Metabolic Disease	69.80 (13.9; 5)	31.97 (10.6; 3)	-37.83	--	31.97 (10.6; 3)
	Disease of the Genitourinary System	27.29 (n = 1)	19.09 (n = 1)	-8.2	--	19.09 (n = 1)
	Disease of the Digestive System	--	--			
	Certain Infectious and Parasitic Disease	71.75 (35.8; 2)	55.35 (27.6; 2)	-16.4	24.64 (n = 1)	30.71 (n = 1)
Unnatural Causes	Accident	1,097.09 (33.2; 33)	761.21 (29.2; 26)	-335.88	403.53 (28.8; 14)	357.68 (29.8; 12)
	External Cause of Morbidity and Mortality	611.16 (29.1; 21)	392.61 (21.8; 18)	-218.55	103.87 (14.8; 7)	288.74 (26.2; 11)
	Intentional Self-Harm	191.93 (31.9; 6)	129.01 (25.8; 5)	-62.92	13.37 (n = 1)	115.65 (28.9; 4)
	Mental and Behavioral Disorder	127.47 (25.4; 5)	59.86 (14.9; 4)	-67.61	32.67 (16.3; 2)	27.19 (13.5; 2)
	Complication of Surgical and Medical Care	34.96 (17.4; 2)	12.55 (n = 1)	-22.41	12.55 (n = 1)	--
	Poisoning by Drugs, Medicaments and Biological Substances	22.52 (11.2; 2)	3.64 (n = 1)	-18.88	--	3.64 (n = 1)

Unknown	1,900.24 (13.4; 141)	504.15 (12.0; 42)	-1,396.09	219.81 (9.5; 23)	284.34 (14.9; 19)
TOTAL	7,951.75 (16.9; 468)	3,608.09 (15.9; 226)	-4,343.66	1,365.68 (15.1; 90)	2,242 (16.4; 136)

*Calculated difference between $YPLL_{75}$ and $YPLL_{LE}$ (at birth)

Table 5.5: Mortality odds ratios, by earlier (≤ 71.0 years) or later (≥ 71.01 years) age of death predictors (N = 787); Predicted probabilities reported in reference to below median age of death

Predictors	Age of Death Predictors	Adjusted for	Adjusted for	Adjusted for Career
	Unadjusted	Race	Birth Decade	Length, Handedness, BMI, Position and HOF
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Race				
White (ref)				
Black	10.32 (6.65–16.02)	--	1.67 (.915–3.05)	8.96 (5.67–14.16)
Career Length	1.12 (1.07–1.17)	1.03 (.98–1.08)	1.00 (.95–1.06)	--
Handedness				
Right (ref)				
Left	1.96 (.90–4.27)	1.94 (.83–4.50)	1.28 (.46–3.56)	--
BMI				
Normal (ref)				
Overweight	1.14 (.81–1.60)	1.15 (.79–1.68)	1.16 (.76–1.76)	--
Birth Decade*				
≤ 1920 (ref)				
1921-1930	1.08 (.67–1.75)	1.07 (.66–1.72)	--	1.10 (.67–1.79)
1931-1940	2.78 (1.58–4.87)	2.29 (1.25–4.20)	--	2.75 (1.52–4.97)
1941-1950	36.20 (15.76–83.18)	27.20 (11.21–66.00)	--	35.97 (15.22–84.97)
1951-1960	<i>M</i> age of death = 51.2 y		--	--
≥ 1961	<i>M</i> age of death = 39.6 y		--	--
Position				
Guard (ref)				
Guard/Forward	.83 (.54–1.27)	.88 (.55–1.41)	1.17 (.70–1.95)	--
Forward	1.29 (.87–1.91)	1.28 (.83–1.97)	1.18 (.72–1.92)	--
Forward/Center	1.13 (.74–1.71)	1.11 (.70–1.76)	1.26 (.76–2.10)	--
Center	1.62 (.96–2.72)	1.58 (.89–2.79)	1.33 (.69–2.56)	--
Height (median split)				
≤ 76 in (ref)				
≥ 77 in	2.70 (2.02–3.61)	2.02 (1.48–2.77)	1.27 (.88–1.84)	4.53 (2.84–7.20)
HOF				
Inductee (ref)				

Non-inductee	1.41 (.71–2.78)	1.80 (.82–3.94)	1.27 (.57–2.83)	--
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Significant ORs bolded ($p < .05$, 95% CI); *When analyzed into broader categories, players born between 1931 and 1950 had a significantly higher likelihood of dying below the median age of death relative to those born in 1930 or earlier (OR 6.64, 95% CI: 4.66 – 9.46)

**CHAPTER SEVEN: MORTALITY IN PROFESSIONAL ATHLETES FROM THE NBA,
NFL, NHL AND MLB**

Mortality in professional athletes from the NBA, NFL, NHL and MLB*

*A consolidated version of this article will be submitted for publication to *The BMJ*

Chapter Seven Overview

Background: Critical and comprehensive investigations into the mortality outcomes of elite athletes can assist in decoding risk factors of premature mortality along with providing fruitful avenues to explore issues of human health through engagement in sport. As such, the purpose of this study was to comprehensively examine the lifespans and mortality trends of athletes from the four major sports in North America: Major League Baseball (MLB), National Basketball Association (NBA), National Football League (NFL) and National Hockey League (NHL).

Methods: Sports-reference.com and wikipedia.org were used as the primary sources of information for all data collected, in addition to sport-specific publically available sources. All data were rigorously cross-referenced, and were analyzed descriptively and through Kaplan-Meier and Cox regression survival analyses.

Results: Overall, 17,523 out of 50,515 (34.7%) athletes were deceased as of the respective data collection cut-off date for their sport, with MLB players having the highest risk of imminent mortality ($\chi^2(3) = 541.38, p < .05$). Professional basketball players generally had the highest relative proportion of death when standardizing data by debut year, although NHL and NFL players who debuted after 2005 had the highest proportion of death. In addition, a one year increase in career length significantly decreased the risk of death (HR .982, 95% CI: .978 – .985), even after adjusting for sport type (HR .977, 95% CI: .974 – .980).

Conclusions: Meaningful significance should be considered given the historical and unique nature of the dataset analyzed, which can influence our interpretation and dissemination of the results. Nevertheless, investigating risk of death differences through different occupational and biological variables can help highlight aversive trends to lifespan that permeate throughout high performance athlete populations.

Mortality in Professional Athletes from the NBA, NFL, NHL and MLB

There has been substantial evidence suggesting a clear inverse dose-response relationship between physical activity and all-cause mortality, the ultimate health outcome (e.g., Gebel et al., 2015). Aggregations of epidemiologic studies normally show a 30% risk reduction of all-cause mortality among active individuals relative to the least active individuals, although the shape of the dose-response curve appears curvilinear where the lifespan benefits from being active become smaller as the amount of physical activity increases (Physical Activity Guidelines Advisory Committee, 2008). Interestingly, Moore and colleagues (2012) highlighted that few studies have quantified the actual *years gained* from engaging in regular physical activity (e.g., Franco et al., 2005; Paffenbarger, Hyde, Wing, & Hsieh, 1986; Wen et al., 2011). Accordingly, Arem and colleagues (2015) and Moore and colleagues (2012) conducted large pooled cohort analyses on over 650,000 participants each and demonstrated consistent years of life gained from physical activity involvement, particularly for those participating at moderate to vigorous intensities. These lifespan benefits extend to elite athletes as well (Garatachea et al., 2014; Lemez & Baker, 2015); however, although some evidence suggests that they are more physically active in later life compared to the general population (e.g., Fogelholm, Kaprio, & Sarna, 1994), much less is known about elite athletes' physical activity patterns post retirement and whether their superior activity levels during youth and young adulthood translate into middle and late adulthood. Importantly, critical and comprehensive investigations into the mortality outcomes of elite athletes can assist in decoding risk factors of premature mortality (i.e., dying earlier than life expectancy at birth) along with providing fruitful avenues to explore issues of human health through engagement in sport.

There are four major professional team sports leagues in North America that are universally considered to be the most prominent in their sport (generally defined by revenue generated and professional competition level): Major League Baseball (MLB), the National Basketball Association (NBA), the National Football League (NFL), and the National Hockey League (NHL). Lemez and Baker's (2015) systematic review of mortality and longevity in elite athletes identified 16 studies exclusively conducted on MLB players, four on NFL players, three on NBA players and none on NHL players at the time of their review. However, a majority of these studies used restrictive sampling strategies and inclusion criteria that did not capture all of the living and deceased players at the time of the study, while others had different foci, in addition to the same research teams conducting multiple studies. For example, Abel and Kruger performed six studies (2004, 2005a, 2005b, 2006, 2007a, 2007b) that exclusively examined lifespans of MLB players, in addition to one study on NFL players and one study on a mixed athletic sample. Two of their studies examined the relationship between the initials of players' names and longevity (Abel & Kruger, 2007b; Abel & Kruger, 2010); however, Smith (2011a) criticized their 2007 study on the basis of a small sample, selective initials, and a weak statistical test. Moreover, Smith (2011b) reported that the Sean Lahman Baseball Archive, which was used as the primary data retrieval method for nearly all of Abel and Kruger's studies, had incomplete death data for a number of cases (particularly for the lesser known players who played in the earlier decades of the league), and highlighted the difficulty in determining whether missing values in the database was a result of the player still being alive or whether the death date was unknown. This is obviously problematic if researchers falsely assume that missing death data for each athlete implies that they are still alive. In addition, four of the 16 studies on baseball players were responses to previously published literature and one study investigated 28 players who died

from a poisoning-related cause (i.e., Boren & Erickson, 1998). While there were no studies exclusively examining the lifespans of NHL players, two mixed-sample studies included ice-hockey players, although the inclusion criteria were restricted to players being born between 1875 and 1930 and were at least 25 years of age at the time of death (Abel & Kruger, 2010) and Hall of Fame (HOF) players born between 1860 and 1930 (Bianco et al., 2007). Although the birthdate restriction method may have intuitive appeal that enables authors to capture the ‘full’ lifespans of former players, it also excludes those that died prematurely.

Interestingly, Lemez and Baker (2015) located only one study that compared survival curves across the four major sports (among five other sports that were included in the study; Bianco et al., 2007); however, the sample sizes were largely limited due to their focus on HOF players born prior to 1931 (baseball, $n = 154$; basketball, $n = 58$; football, $n = 81$; ice-hockey, $n = 130$). Nevertheless, Bianco and colleagues (2007) concluded that life expectancies appear to be unaffected by the type of sport discipline after reporting no significant differences between the median life expectancies, which ranged from 74.5 years (ice-hockey players) to 78.0 years (basketball players) in the four major sports. More recently, Lemez, Wattie, and Baker (2015) addressed the relative lack of research comparing mortality outcomes of athletes from the four major sports and were able to gather cause of death data on 205 players who were still active competitors when they died. Similarities were found between leading causes of death of their sample to the Canadian and United States (US) general population, and the rare event (0.38%) of a professional athlete dying while active (i.e., premature) was highlighted. As a majority of the studies examining MLB, the NBA, the NFL and the NHL players were published in 2007 or earlier and/or included restrictive inclusion criteria, the purpose of this study was to provide a more up-to-date and comprehensive comparison of lifespans and mortality trends of athletes

from the four major sports. Investigating these large populations of athletes can provide broader and deeper awareness of the health status of elite athletes from sports with different physiological demands and challenge commonplace assumptions regarding health risks of participating in high performance sport. Our hypotheses were twofold. First, due to the inaugural season of MLB being much earlier than the other three major sports, we hypothesized that i) MLB players would have a significantly higher imminent mortality risk given the overall earlier birthdates of players compared to players from the other three leagues. Second, due to a curvilinear dose-response relationship between physical activity and lifespan, along with socio-medical and economic similarities from playing in high revenue leagues, it was hypothesized that ii) proportional death rates would be similar across the four sports, when standardizing the data by debut years.

Methods

Data Collection

With the exception of the MLB dataset, sports-reference.com and wikipedia.org were used as the primary sources of information for all data collected. Sports-reference.com is a recognized sports archive of aggregated publically available athlete records, which includes statistics on professional baseball, basketball, football, and hockey players. Wikipedia.org has been argued to be a similar or superior method of data retrieval to traditional sources due to the accessibility from its open access structure (e.g., Coate & Sun, 2013). A random sample from each player population was also cross-referenced in their respective official sport encyclopedia, among other sources (including all centenarians and players who died at very young ages). If a discrepancy between variables was identified during cross-referencing, the statistic that was most commonly reported in a majority of the data retrieval sources was used. In addition, a general web search was performed for all players who were missing a death date and were 65 years or

older in order to locate potential death dates that were not captured through the primary sources of data retrieval. A player was removed from the dataset if a death date was not located and they were 100 years old or older. Since life expectancies in all countries in the world are less than 100 years old, and becoming a centenarian remains a relatively rare achievement, we were confident that this cut-off age would remove a vast majority of players who were already deceased. As well, while some studies examining lifespan in the four major sports excluded players who played for a short period of time (e.g., Lawler, Lawler, Gibson, and Murray (2012) excluded NBA players who appeared in less than five games), the aim of this study was to capture *all* players who played in any of the four leagues at any point in time. We argue that debuting in any of these professional leagues alone indicates a relatively superior level of cardiovascular fitness and athletic ability for most cases compared to the general population. Career length was reported in years and was calculated by subtracting the players' last year in the league from their debut year. As some players, particularly in the earlier eras, took years off between their debut year and year in which they retired (e.g., Charley O'Leary played from 1904 to 1913, but returned to bat in one game in 1934), actual years played were searched (and adjusted accordingly) in a minimum of 25% of the players for each sport through their biographical pages on sports-reference.com as a more precise measure of active involvement in the league. In particular, a secondary search for precise career lengths was performed for all NBA/American Basketball Association (ABA) players since these athletes were more likely to go play in other professional leagues (e.g., particularly in Europe for the modern-era players) and come back to the NBA relative to athletes from the other three sports, as well as for all players who had a career length of 25 years or greater after subtracting their last year from their debut year. All

height and weight variables were converted to centimetres (cm) and kilograms (kg), respectively. Ethics review was not required as all data were collected from publicly available sources.

Samples and Variables

MLB

Data on living and deceased MLB players were retrieved from two primary online sources; baseball-reference.com and through the 2014 version of the Sean Lahman baseball database (www.baseball1.com). Despite Smith's (2011b) reporting of incomplete data for some players, this otherwise well-respected database, compiled by recognized statisticians and a team of researchers, was used as the initial method of data collection as it provided a current and comprehensive list of all players who played professional baseball starting in the National Association of Professional Base Ball Players (1871 – 1875; the first professional baseball league) to the 2014 MLB season. Nevertheless, our method of removing players from the datasets who were 100 years or older without a death date, along with a general web search for all players who were missing a death date and were 65 years or older, was rigorously instituted for this athlete population.

The variables collected were: player name, career length, handedness (for both batting and throwing), height, weight, birthdate and death date. The Master table from the Lahman database used to collect these data did not include player position; therefore, these data were collected through mlb.com's player index (mlb.mlb.com/stats/sortable.jsp). Death data were collected until January 25, 2016. We used “15” as the day of birth and/or “6” as the month of birth (less frequently) for 279 cases that were missing complete birthdate information, and for six cases that involved incomplete death date information. Since there was a time gap between the 2014 version of the Lahman database and our data collection cut-off date, recent deaths (i.e., January 1, 2014 – January 25, 2016) were added through the online Baseball Almanac, which

provides a historical breakdown by year of death (http://www.baseball-almanac.com/players/baseball_deaths.php) and each death was cross-referenced with baseball-reference.com. All but one of the deaths that occurred in 2014 identified through the online Baseball Almanac was already included in the Lahman database. Players who were active past the 2013 season (i.e., were part of a MLB roster in 2014 or beyond) were censored for the career length analysis to ensure that a full career was captured. All data were cross-referenced in both of the primary sources, along with a random sample of 10% in *Total Baseball* (Thorn et al., 2004) and through thedeadballera.com.

Total size of sample examined

Overall, 18,588 players were identified and extracted through our search. We removed 127 players whose birthdate was publicly inaccessible. We removed an additional 42 players who were 100 years or older and were missing a death date ($M = 150.26 \text{ y} \pm 12.76$). Therefore, a total of 18,419 players were included in the analyses. In addition, 187 cases that were missing “career debut” and “career end” data in the Lahman database and were unable to be located through our search were censored for the career length/debut analyses, but were considered for other analyses (i.e., they were part of the total 18,419 player sample).

NBA/ABA

Data were collected on living and deceased NBA and ABA players who played from the 1946–1947 inaugural season to the 2009–2010 season from two primary online sources; basketball-reference.com and wikipedia.org. The variables collected were: player name, player position, career length, induction into the HOF (collected from the official Naismith Memorial Basketball HOF website, hoophall.com/hall-of-famers-index), handedness, height, weight, birthdate and death date. Death data were collected until December 11, 2015. We used “15” as

the day of death for nine cases that were missing complete death date information. Players who were active past the 2009–2010 season were censored for the career length analysis. All data were cross-referenced in both of the primary sources, along with a random sample of 10% in *Total Basketball* (Koppett, Shouler, Ryan, Smith, & Bellotti, 2003), nba.com’s historic player index and apbr.org’s biographical database compiled by John Grasso.

Total size of sample examined

Birthdates for all players were publicly accessible and no players were at least 100 years old and missing a death date, as the oldest living player as of December 11, 2015 was 98 years of age. Therefore, a total of 3,901 players were included in the analyses.

NFL

Data were collected on living and deceased NFL players who played from the 1920 inaugural season (originally named the American Professional Football Association from 1920–1921) to the 2012 season (ended on December 30, 2012) from two primary online sources; pro-football-reference.com and wikipedia.org. The variables collected were: player name, player position, career length, height, weight, birthdate and death date. Death data were collected until September 10, 2015. We used “15” as the day of birth for 31 cases and “6” as the month of birth for 26 cases that were missing complete birthdate information, and “15” as the day of death for 158 cases and “6” as the month of death for eight cases that involved incomplete death date information. Players who were active past the 2011 season were censored for the career length analysis. All data were cross-referenced in both of the primary sources, along with a random sample of 10% in *Total Football* (Carroll, Gershman, Neft, & Thorn, 1999) and nfl.com’s historic player index.

Total size of sample examined

Overall, 22,797 players were identified and extracted through our search. We removed 113 players whose birthdate was publicly inaccessible. We removed an additional 69 players who were 100 years or older and were missing a death date ($M = 113.65 \text{ y} \pm 6.34$). Therefore, a total of 22,615 players were included in the analyses.

NHL

Data were collected on living and deceased Canadian- and US-born NHL players who played from the 1917–1918 inaugural season to the 2010–2011 and 2012–2013 season, respectively, from four primary online sources; hockey-reference.com, quanthockey.com, wikipedia.org and sihrhockey.org. Variables were collected from hockey-reference.com and quanthockey.com and included player name, nationality, player position, career length and birthdate. Height and weight were only collected for US-born players during the time of data collection. Death data were collected until December 31, 2015 for both the Canadian- and US-born players from hockey-reference.com, wikipedia.org and sihrhockey.org. We used “15” as the day of death for two cases that were missing complete death date information. During the time period in which data for the Canadian-born players were collected, we elected to use “1” as the day of birth for this sample of players, which likely minimally altered the age of death findings. Canadian-born players who were active past the 2009–2010 season and US-born players who were active past the 2011–2012 season in the NHL were censored for the career length analysis. All data were cross-referenced in each of the primary sources, along with a random sample of 10% in *Total Hockey* (Duplacey, Diamond, Dinger, Kuperman, & Zweig, 1998) and hhof.com (official website of the hockey HOF).

Total size of sample examined

Birthdates for all players were publicly accessible and no players were at least 100 years old and missing a death date. The oldest living Canadian-born player was 97 years of age, and the oldest living US-born player was 96 years of age as of December 31, 2015. Therefore, a total of 5,580 (4,601 Canadian-born and 979 US-born) players were included in the analyses.

Overall, 50,515 athletes from the four major sports in North American were examined in this study.

Statistical Analyses

Data were analyzed descriptively and through Kaplan-Meier and Cox regression survival analyses. All data were analyzed using SPSS version 23, and statistical significance was defined as $p < 0.05$ at the 95% Confidence Interval (CI). Given the large sample sizes where p values indicated statistical significance, Hazard Ratios (HRs) were only marked as significant if CI excluded 1.00. Separate analyses for each sport were also performed because of the unique histories and constraints to each sport. For example, professional baseball started in 1871 while professional basketball started in 1946, and each sport has unique playing positions. Of the variables collected, career length was the only variable included as a covariate in the combined sport survival analysis, as it was collected for all players and did not appear to be highly confounded by type of sport.

Results

Combined Sport Analyses

Descriptives. Overall, 17,523 out of 50,515 (34.7%) athletes were deceased as of the respective data collection cut-off date for their sport. More specifically, 9,358 of 18,419 (50.8%) MLB players were deceased as of January 25, 2016, 787 of 3,901 (20.2%) NBA/ABA players were deceased as of December 11, 2015, 6,129 of 22,615 (27.1%) NFL players were deceased as

of September 10, 2015, and 1,250 of 5,580 (22.4%) Canadian- and US-born NHL players were deceased as of December 31, 2015. Descriptive data on mortality outcomes by sport are shown in Table 6.1.

Standardized Comparisons. Descriptive data standardized by debut years are shown in Table 6.2. Given the inaugural season differences between the leagues, this allowed proportional comparisons of athlete death rates across the four sports, standardized by the time period in which they debuted in their respective league. NBA/ABA players who debuted between the inaugural season and 1975 and between 1986 and 1995 had the highest proportion of death relative to the other three sports during the same time period. In contrast, MLB players who debuted between 1976 and 1985 and between 1996 and 2005 had the highest proportion of death relative to the other three sports during the same time period. Interestingly, NHL and NFL players who debuted after 2005 had a higher proportion of death relative to MLB players, while no deaths have been reported in NBA/ABA players. Overall, there was a moderate amount of variability in the death rates between the four sports in each of the debut year time periods analyzed. Much of this variability was created by NHL players having consistently lower proportions of death between the debut years of 1946 and 2005 relative to the other three sports. The largest death rate disparity was observed for athletes who debuted between 1946 and 1955, where 63.0% of the NHL sample was deceased at the time of data collection, which was a considerably lower proportion of deceased athletes compared to those who played in the NBA/ABA (82.2%), NFL (81.5%) or MLB (78.0%) during the same time period.

Survival and Cox Regression Analyses. Kaplan-Meier survival analysis found the median survival times to be 75.0 years for MLB players, 81.4 years for NBA/ABA players, 78.7 years for NFL players, and 80.0 years for NHL players (77.5 years overall; $\chi^2(3) = 541.38, p < .05$;

see Figure 6.1). Compared to MLB players, Cox regression analysis found a significantly lower risk of death for NBA/ABA (HR) 0.56, 95% CI: .53 – .61), NFL (HR 0.75, 95% CI: .73 – .78), and NHL (HR 0.66, 95% CI: .63 – .71) players (see Figure 6.2). After excluding those players who were still active (i.e., measuring only full career lengths), we found that a one year increase in career length significantly decreased the risk of death (HR .982, 95% CI: .978 – .985), even after adjusting for sport type (HR .977, 95% CI: .974 – .980). Interestingly, while this effect was small and appeared to be influenced by the large sample size, unadjusted Cox regression analyses found a similar pattern across each of the four sports (NBA/ABA athletes narrowly missed statistical significance). Survival and Cox regression data on mortality outcomes by sport are shown in Table 6.3.

League-Level Analyses

MLB

Descriptives. The median age of death was 70.4 years and the mean age of death was 68.0 years (SD = 16.2) for the entire MLB sample. A total of 17 centenarians were identified with the oldest player living to 107.4 years, and the youngest death occurred at 19.6 years. Of the occupational variables collected, we found players to average 5.8 years (SD = 5.1) in career length, with a majority being right-handed batters (65.7%) compared to batting left-handed (27.6%) or ambidextrously (6.7%), and a majority were right-handed throwers (79.9%). We identified 42.9% pitchers, 9.6% catchers, and 47.5% who played other positions (i.e., first, second and third baseman, shortstop, and left, center and right fielder). Of the biological variables collected, players averaged 84.2 kg (SD = 9.5) in weight and 183.5 cm (SD = 6.6) in height, with the highest participation in those who were born between 1960 and 1979 (20.3%) and the least in those born between 1820 and 1839 (0.1%) and 1840 and 1859 (4.0%).

Survival and Cox Regression Analyses. MLB players were found to have a significantly lower risk of death for every one year increase in career length (HR .977, 95% CI: .973 – .981), one kg increase in weight (HR .985, 95% CI: .982 – .988) and one cm increase in height (HR .975, 95% CI: .972 – .978). In addition, pitchers had a lower risk of death compared to players in other positions (HR .946, 95% CI: .906 – .988). After adjusting for birth decade, only career length remained as a significant predictor of early mortality risk. In contrast, no statistically significant differences in mortality risk were found between laterality preferences of players.

NBA/ABA

Descriptives. The median age of death was 71.0 years and the mean age of death was 68.1 years (SD = 16.0) for the entire NBA/ABA sample. No centenarians were identified with the oldest player living to 99.6 years, and the youngest death occurred at 24.4 years. Of the occupational variables collected, we found players to average 4.9 years (SD = 4.4) in career length, with a high majority being right-handed (94.5%). We identified 35.1% guards, 10.0% guards/forwards, 31.4% forwards, 11.1% forwards/centers and 12.4% centers. In addition, 2.9% of the living and deceased player population were inducted into the basketball HOF. Of the biological variables collected, players averaged 93.9 kg (SD = 11.7) in weight and 197.8 cm (SD = 9.3) in height, with the highest participation in those who were born between 1960 and 1979 (33.4%) and the least in those born between 1900 and 1919 (1.9%).

Survival and Cox Regression Analyses. NBA/ABA players who played as both a guard and forward during their career had a significantly lower risk of death compared to players who just played as a guard (HR .738, 95% CI: .571 – .953), while players who played exclusively at the center position had a significantly higher risk of death compared to guards (HR 1.50, 95% CI: 1.14 – 1.97). Each of these positions remained significant predictors of risk of early mortality

after adjusting for birth decade. No statistically significant differences in early mortality risk were found between player career longevities, anthropometrics, laterality preferences or inductees and non-inductees into the HOF.

NFL

Descriptives. The median age of death was 71.8 years and the mean age of death was 69.1 years (SD = 15.6) for the entire NFL sample. Five centenarians were identified with the oldest player living to 104.2 years, and the youngest death occurred at 21.9 years. Of the occupational variables collected, we found players to average 4.4 years (SD = 3.7) in career length, and a majority playing the offensive line position (18.6%) and the least playing as a punter (1.1%) or kicker (1.3%). Of the biological variables collected, players averaged 101.4 kg (SD = 17.3) in weight and 186.3 cm (SD = 6.4) in height, with the highest participation in those who were born between 1960 and 1979 (32.7%) and the least in those born between 1880 and 1899 (4.2%; two players were born prior to 1880).

Survival and Cox Regression Analyses. NFL players were found to have a significantly lower risk of death for every one year increase in career length (HR .971, 95% CI: .964 – .979) and one cm increase in height (HR .982, 95% CI: .978 – .986), although each of these effects became insignificant when adjusting for birth decade. In addition, player position appeared to have a moderate influence on early mortality risk. For example, relative to wide receivers, offensive linemen (HR 1.77, 95% CI: 1.61 – 1.95) and running backs (HR 1.62, 95% CI: 1.47 – 1.79) had a significantly higher risk of death, whereas defensive backs (HR .713, 95% CI: .615 – .827) had a significantly lower risk of death. Each of these positions remained significant predictors of early mortality risk after adjusting for birth decade. Interestingly, weight was not found to be a significant precursor to premature mortality.

NHL

Descriptives. The median age of death was 72.7 years and the mean age of death was 69.7 years (SD = 15.6) for the entire NHL sample. One centenarian was identified, living to 100.9 years, and the youngest death occurred at 20.4 years. Of the occupational variables collected, we found players to average 5.8 years (SD = 5.0) in career length, and a majority playing defense (28.1%), followed by center (21.7%), right wing (21.2%), left wing (19.2%) and goalie (9.8%). Of the biological variables collected with the US-born sample, players averaged 88.7 kg (SD = 7.6) in weight and 184.1 cm (SD = 5.7) in height, with the highest participation in those who were born between 1960 and 1979 (36.1%) and the least in those born between 1880 and 1899 (3.0%; one player was born prior to 1880).

Survival and Cox Regression Analyses. NHL players were found to have a significantly lower risk of death for every one year increase in career length (HR .972, 95% CI: .961 – .984). When accounting for country of birth, Canadian-born players showed a similar pattern of lower risk of death for every one year increase in career length (HR .971, 95% CI: .959 – .983); however, this relationship was not statistically significant for the US-born players (HR .971, 95% CI: .900 – 1.05). Shorter US-born players were found to have a lower risk of death (HR .956, 95% CI: .915 – .999), although this relationship became statistically insignificant after adjusting for birth decade, while no statistically significant differences in early mortality risk were found between player weight. In addition, combined and separate Cox regression analyses on Canadian- and US-born players did not reveal playing position to significantly influence the risk of death, although lower risk of death for US-born left wingers relative to goalies narrowly missed statistical significance ($p = .07$). Nevertheless, career length remained a significant predictor of mortality risk for the overall NHL player sample after adjusting for birth decade.

Discussion

The purpose of this study was to provide a current and comprehensive examination of mortality outcomes of athletes from the four major sports in North America. We hypothesized that i) MLB players would have a significantly higher imminent mortality risk relative to the other three sports and that ii) proportional death rates would be similar across the four sports. Our first hypothesis was supported; the combined sport Kaplan-Meier survival analysis indicated that MLB players had a significantly higher imminent mortality risk relative to the other three major sports. In addition, the overarching influence of career length on mortality suggests that longer participation in professional sport lowers risk of death, regardless of physiological and other context-specific demands. When adjusting for birth decade, the key finding from the league-level analyses indicated that player position has a low to moderate influence on early mortality risk for NBA/ABA and NFL players, and no influence in MLB and NHL players. The other variables analyzed (i.e., anthropometric data, handedness and HOF induction) were not found to significantly impact mortality risks of the athletes.

Our second hypothesis was partially supported; similarities between the proportional death rates of athletes who played during the same time periods were mostly seen between MLB, NBA/ABA and NFL players, although NBA/ABA generally had higher rates of death. Alternatively, former NHL players debuting between 1946 and 2005 consistently had the lowest proportionate rate of death. Further, the finding that NHL and NFL players who debuted after 2005 had a relatively *higher* proportion of death supports the recent media attention garnered towards the issue of concussions and their relationship to suicide in these two sports. A visible tragedy such as young player committing suicide as a result of head injuries sustained from competition threatens to ignore the complete picture of mortality risk that preceded the event.

Interestingly, the overall mean ages of death, including the variability in ages of death, were very similar in athletes across the four sports examined. Therefore, while the ages of death are generally similar between the four sports, there appears to be proportionate death disparities between NHL players and athletes from the other three sports, where Canadian- and US-born NHL players die at a lower rate.

Statistical vs. Meaningful Significance

Importantly, caveats exist to the practical use of some of the key findings of this study. In contextualizing these findings, it is important to recognize how the characteristics of the historical and unique nature of the dataset analyzed can influence our interpretation and dissemination of the results. MLB players were found to have a higher risk of death relative to the other three sports; however, statistical significance was most likely driven by the MLB inaugural season (1871) occurring much earlier than the other three sports (≥ 1917), resulting in 15.1% ($n = 2,783$) of the players being born in the 19th century. Thus, while MLB had the relatively highest proportion of deceased players (50.8%), it also contained a large cohort of players whose life expectancies at birth were lower compared to athletes born after 1900, predisposing the Kaplan-Meier estimate to produce a statistically significant hazard risk. The intuitiveness of this finding arguably limits the meaningfulness of the result, which likely would have been more useful if each of the four major sports started on the same date. As such, the death rate comparisons standardized by debut year across the four sports may have more meaningful significance than mortality risk estimates.

The characteristics of the dataset also indicate that the practical use of the statistically significant results from the Cox regression analyses was likely limited by the large sample sizes of athletes driving the effect. A majority of the unadjusted and adjusted variables measured had

small effect sizes, and the CI ranges were small and close to 1.00 (i.e., equivalence in the hazard rate). The largest effect sizes of early mortality risk were found in the player position variable. For example, centers in basketball were found to have the highest early mortality risk of any other player position examined after adjusting for birth decade. In addition, we found NFL defensive linemen to have the second-highest adjusted mortality risk, which supports Baron, Hein, Lehmen and Gersic's (2012) finding of NFL defensive linemen having the highest mortality risk from cardiovascular disease. Since centers and linemen are usually the largest players on their respective field of play, the largest effect sizes of the variables analyzed suggest that relatively larger body size increases risk of early mortality. In contrast, anthropometric predictors (i.e., height and weight) did not have a robust impact on early mortality risk for athletes from any of the four sports when adjusted for birth decade, although the unadjusted trends for MLB, NFL and US-born NHL players suggested that lower height and weight was conducive to longevity. While the reasoning behind this counterintuitive finding is unclear, we speculate that given the positional demands, defensive linemen have a higher percentage of body fat relative to players in other positions where muscularity is more favourable to performance (e.g., running backs).

Overall, this study suggests that i) MLB players have the highest imminent mortality risk, ii) NHL players generally die at the lowest proportional rate, and iii) longer career lengths and smaller body sizes generally reduce early mortality risk. While the overall magnitude of the effect sizes of the variables analyzed was small, the aforementioned trends provide interesting avenues for future research to explore in more depth. For example, previous work in this area has suggested that the energy systems required for participation in certain sports (i.e., aerobic/endurance, mixed, and anaerobic/power) may influence mortality outcomes; athletes

from sports with aerobic demands generally have superior longevities to athletes participating in anaerobic sports (Lemez & Baker, 2015; Teramoto & Bungum, 2010). However, this trend was only partially supported in this study through the proportional death rate analysis; NHL players died at the lowest proportional rate, but NBA/ABA players died at the highest proportional rate (both sports are more aerobically demanding than MLB and NFL). Unfortunately, it is outside the scope of this study to determine the causes behind the mortality trend disparities between these sports.

Limitations

The main limitation of this type of analysis is the inability to account for the lifestyle factors of these athletes prior to their death. Due to the historical nature of our examination and the lack of publicly accessible information regarding their physical, mental, and social well-being, our knowledge of their health outcomes is largely limited to how long they lived. In other words, exposure to a certain type of sport (cause) does not necessarily explain an athletes' mortality outcome (effect). Our data are also unable to delineate the difference between lifespan and health span, where a number of athletes may have lived a long life but were unhealthy from a disease and/or disability standpoint. In addition, the inclusion of causes of death would likely help explain certain mortality trends seen in these athletes (e.g., deaths during World War II). Another limitation of this study is not having a reference control group from the general population; however, the main purpose of this study was to compare the mortality outcomes of athletes *between* the four major sports. A final limitation of this study relates to data collection; there were possible data entry errors and potential omissions of death dates due to the large quantity of data that were collected, despite adopting a rigorous cross-referencing approach. In addition, a function of longitudinal data collection resulted in having different ending points for

the death data between the four sports, which may have minimally biased some of the results (i.e., death data cut-offs were five months apart between the four sports, September 2015 to January 2016).

Conclusion

More broadly, the association between sport type and lifespan is buffered by several determinants of health; however, investigating risk of death differences through different occupational and biological variables can help highlight aversive trends to lifespan that permeate throughout high performance athlete populations. For example, identifying robust predictors of premature death and other worrisome mortality trends in groups of athletes may allow for practical feedback to health and sport practitioners that may initiate modifications to current healthcare models for active and retired athletes. In turn, continued empirical examinations into the health outcomes of high performance athletes can help dispel common assumptions regarding health risks from participating in sport.

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Figure 6.1: Kaplan-Meier survival probability for living and deceased MLB, NBA, NFL and NHL players [$\chi^2(3) = 541.38, p < .05$]

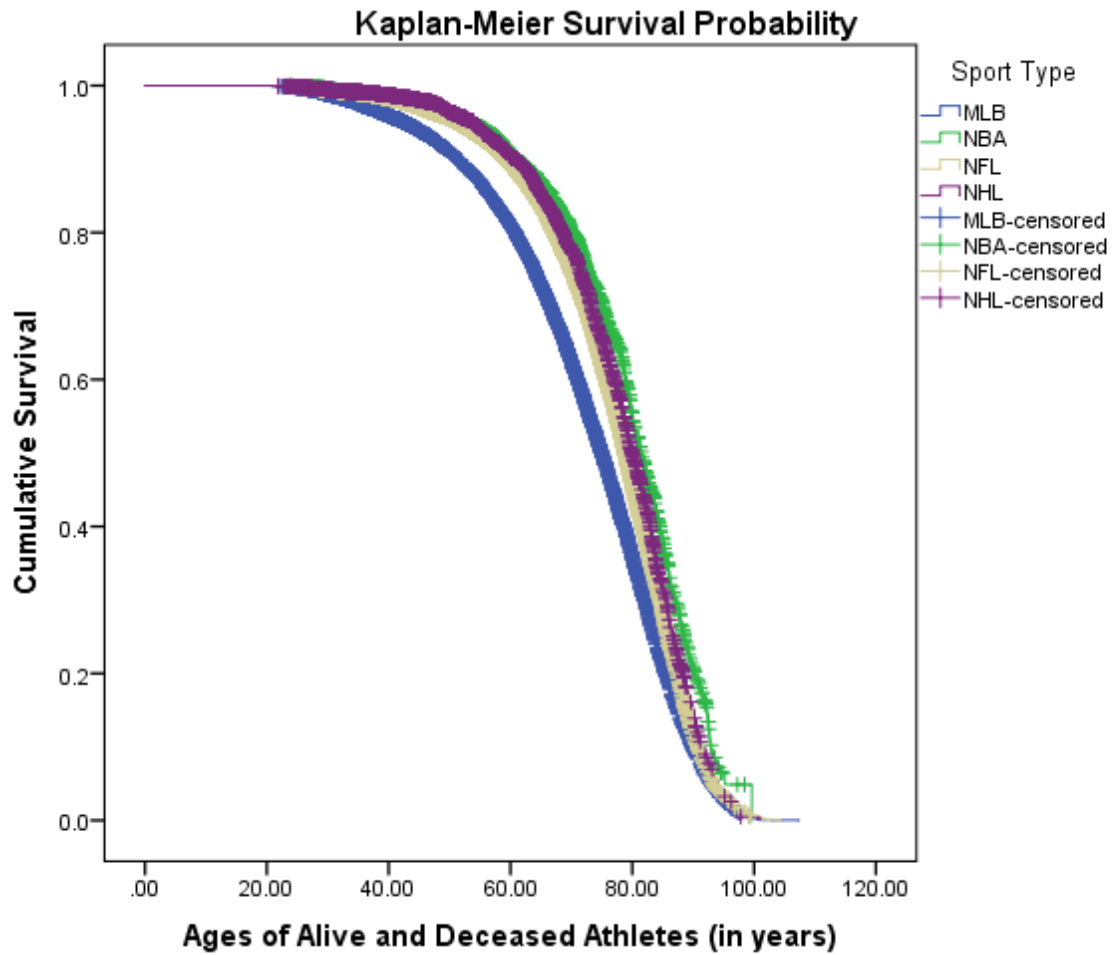


Figure 6.2: Cox regression hazard model for living and deceased MLB, NBA, NFL and NHL players

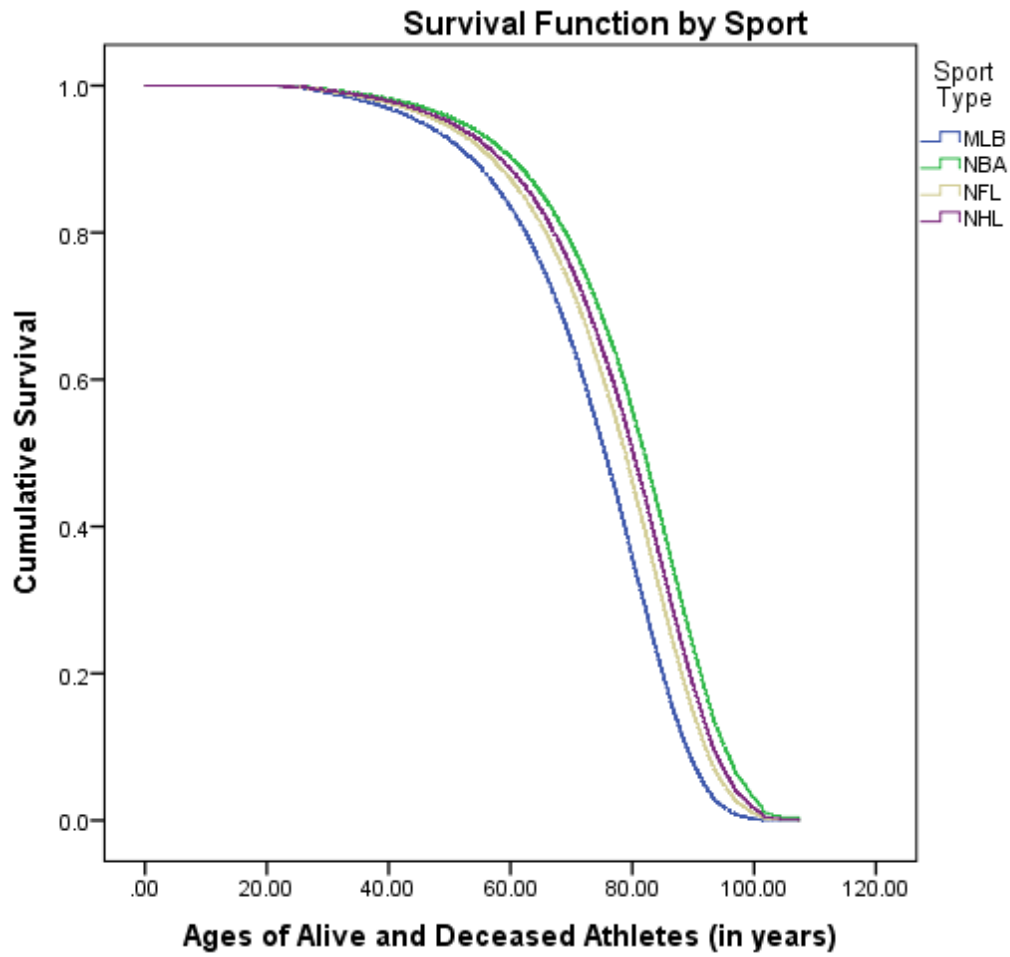


Table 6.1: Descriptive mortality data of professional athletes from the four major sports in North America

Sport (Debut Years Analyzed)	n	% Dead by Cut-off Date*	Median Death Age in Years	Mean Death Age in Years	Minimum	Maximum
MLB (1871 – 2014)	18,419	50.8	70.4	68.0 ± 16.2	19.6	107.4
NBA/ABA (1946 – 2009)	3,901	20.2	71.0	68.1 ± 16.0	24.4	99.6
NFL (1920 – 2012)	22,615	27.1	71.8	69.1 ± 15.6	21.9	104.2
NHL – CA (1917 – 2010)	4,601	25.5	72.7	69.7 ± 15.5	20.4	97.2
NHL – US (1917 – 2012)	979	7.9	73.4	70.1 ± 16.8	25.7	100.9
Overall	50,515	34.7	71.1	68.5 ± 16.0	19.5	107.4

*Uncensored cases

Table 6.2: Standardized mortality data of professional athletes from the four major sports in North America, by debut years

Debut Years	MLB			NHL			NFL			NBA/ABA		
	n	<i>M</i> Death Age (y)	% Dead by Cut-off	n	<i>M</i> Death Age (y)	% Dead by Cut-off	n	<i>M</i> Death Age (y)	% Dead by Cut-off	n	<i>M</i> Death Age (y)	% Dead by Cut-off
1871 – 1900	2,058	61.3 ± 17.6	100.0		--			--			--	
1901 – 1916	2,333	68.3 ± 15.6	100.0		--			--			--	
1917 – 1919	268	69.1 ± 15.5	100.0	65	67.1 ± 15.0	100.0		--			--	
1920 – 1930	1,304	71.0 ± 14.8	100.0	321	70.3 ± 14.7	100.0	1,821	70.6 ± 14.5	100.0		--	
1931 – 1945	1,696	73.8 ± 14.3	98.4	457	74.0 ± 13.8	98.5	1,763	72.7 ± 14.8	98.6		--	
1946 – 1955	1,098	73.8 ± 12.8	78.0	335	73.5 ± 12.9	63.0	1,680	73.2 ± 12.8	81.5	578	75.6 ± 12.1	82.2
1956 – 1965	1,125	66.5 ± 12.7	35.5	280	64.6 ± 14.1	28.2	1,771	64.8 ± 12.3	33.3	260	65.8 ± 13.6	41.5
1966 – 1975	1,288	56.5 ± 12.8	14.0	736	56.8 ± 13.4	10.6	2,570	56.3 ± 11.4	12.6	782	56.0 ± 11.9	17.1
1976 – 1985	1,384	51.4 ± 9.3	6.1	1,068	47.9 ± 7.7	2.2	2,937	44.9 ± 9.1	5.0	596	49.6 ± 10.0	6.0
1986 – 1995	1,697	40.8 ± 8.0	1.9	962	36.3 ± 9.0	1.4	3,979	37.9 ± 7.7	2.4	674	40.4 ± 9.4	4.0
1996 – 2005	1,995	33.3 ± 5.7	1.2	827	29.9 ± 3.8	0.8	3,546	31.6 ± 5.5	1.0	674	34.0 ± 5.9	1.0
2006 – 2014*	1,986	24.6 ± 3.2	0.2	529	25.1 ± 5.3	0.4	2,548	26.6 ± 2.1	0.4	337	--	--

*Debut was calculated until 2009 for NBA/ABA players, 2010 for Canadian-born NHL players and 2012 for US-born NHL players and NFL players

Table 6.3: Cox proportional hazard ratios for sport-specific variables

Variables	Age of Death Predictors	Adjusted for
	Unadjusted	Birth Decade
	HR (95% CI)	HR (95% CI)
MLB		
Career Length	.977 (.973 – .981)	.986 (.983 – .990)
Position		
Other (ref)		
Pitcher	.946 (.906 – .988)	1.02 (.984 – 1.07)
Catcher	1.07 (1.00 – 1.15)	1.05 (.986 – 1.13)
Weight	.985 (.982 – .988)	1.01 (1.00 – 1.01)
Height	.975 (.972 – .978)	1.00 (1.00 – 1.01)
Bats		
Both (ref)		
Right	1.02 (.928 – 1.12)	1.02 (.927 – 1.12)
Left	1.01 (.916 – 1.12)	1.02 (.927 – 1.13)
Throws		
Left (ref)		
Right	1.01 (.959 – 1.07)	.985 (.958 – 1.01)
NBA		
Career Length	.985 (.966 – 1.00)	.989 (.969 – 1.00)
Position		
Guard (ref)		
Guard/Forward	.738 (.571 – .953)	.734 (.569 – .949)
Forward	1.23 (.945 – 1.63)	1.22 (.927 – 1.60)
Forward/Center	.813 (.627 – 1.05)	.813 (.627 – 1.05)
Center	1.50 (1.14 – 1.97)	1.48 (1.13 – 1.94)
Weight	1.01 (1.00 – 1.02)	1.01 (1.00 – 1.02)
Height	1.01 (1.00 – 1.02)	1.01 (1.00 – 1.02)
Handedness		
Left (ref)		
Right	.938 (.647 – 1.36)	.925 (.638 – 1.34)
HOF		
Non-inductee (ref)		
Inductee	.842 (.602 – 1.17)	.835 (.597 – 1.16)
NFL		
Career Length	.971 (.964 – .979)	.996 (.988 – 1.00)
Position		
Wide receiver (ref)		
Defensive back	.713 (.615 – .827)	.794 (.684 – .921)
Defensive line	1.29 (1.13 – 1.48)	1.44 (1.26 – 1.65)
Kicker	.611 (.394 – .947)	.725 (.467 – 1.12)
Linebacker	.829 (.791 – .980)	1.01 (.854 – 1.19)
Multiple	1.54 (1.39 – 1.70)	1.20 (1.08 – 1.33)
Offensive line	1.77 (1.61 – 1.95)	1.29 (1.17 – 1.42)
Punter	.619 (.340 – 1.12)	.862 (.473 – 1.56)

Quarterback	.996 (.835 – 1.18)	.972 (.815 – 1.16)
Running back	1.62 (1.47 – 1.79)	1.19 (1.08 – 1.32)
Tight end	.548 (.387 – .774)	.719 (.508 – 1.01)
Weight	.998 (.996 – 1.00)	1.01 (1.00 – 1.01)
Height	.982 (.978 – .986)	1.00 (1.00 – 1.01)
NHL		
Career Length	.973 (.961 – .984)	.983 (.971 – .995)
Position		
Goalie (ref)		
Center	.874 (.699 – 1.09)	.889 (.711 – 1.11)
Left wing	.843 (.675 – .994)	.825 (.661 – 1.03)
Right wing	.876 (.703 – 1.09)	.858 (.688 – 1.07)
Defense	1.03 (.833 – 1.27)	1.01 (.820 – 1.26)
Weight*	.990 (.963 – 1.02)	1.00 (.976 – 1.03)
Height*	.956 (.915 – .999)	.989 (.943 – 1.04)

Significant HRs bolded ($p < .05$, 95% CI exclude 1.00); *US-born players only

CHAPTER EIGHT: GENERAL DISCUSSION

The overarching purpose of this research was to provide an evidence-based portrayal of i) incidence, ii) predictors and iii) causes of death in athletes from Major League Baseball (MLB), the National Basketball Association (NBA)/American Basketball Association (ABA), the National Football League (NFL) and the National Hockey League (NHL). Mortality differences of athletes between and within sport(s) were explored throughout this dissertation beginning in Chapter Two, which systematically reviewed mortality and longevity literature in elite athletes from a broad range of sports, to Chapter Seven, which examined survival trends of players from the four major sports in North America. In addition, biological and occupational risk factors and characteristics of early mortality were also consistently investigated, and attention was given to statistical artifacts that may distort the risk of death from certain predictors, particularly in Chapters Three (precocity) and Six (birth decade artificially inflating risk of premature death for black and taller players). The seven studies in this dissertation provide a more complete understanding of whether participation in high performance sport is a healthy pursuit when viewed from a lifespan perspective.

Key Findings

Chapter Two

The initial investigation into the mortality and longevity of elite athletes identified 54 peer-reviewed studies and three online articles, where an overwhelming majority reported favourable lifespans for athletes compared to their age- and sex-matched controls from the general population. This review substantiated previous research in this area that suggested aerobic (i.e., endurance athletes such as cyclists) and mixed-sport (e.g., soccer players) athletes have superior longevity outcomes relative to more anaerobic (i.e., power athletes such as

weightlifters) sport athletes. Further, player position, weight, education and race were identified as robust predictors of longevity, whereas other risk factors such as precocity appeared to be less examined in sport.

Chapter Three

In exploring the generalizability of the precocity-longevity hypothesis in sport, the overarching finding of the studies on former Canadian-born NHL players and NBA/ABA players did not find precocity to be a significant predictor of lifespan in athletes, even after using three different indicators of achievement to define precocity. The results present a more complicated narrative of the precocity-longevity effect, suggesting that contextual factors, such as the unique physical demands of sport, and measurement methodologies, may influence support for the proposed hypothesis in sport.

Chapter Four

A low incidence of death was found in athletes who were still active when they died, which is a cohort of the athlete population that is particularly susceptible to having their death widely disseminated by popular media. As well, the leading causes of death in players from the four major sports reflected the leading causes of death in the age- and sex-matched controls from the Canadian and United States (US) general population.

Chapter Five

Addressing the recent heightened anecdotal belief that ‘bigger’ athletes die younger, taller former NBA/ABA players were found to die younger and have significantly higher risk of early mortality compared to shorter players, supporting recent claims in the media by former players.

Chapter Six

Investigations into the causes of death of former NBA/ABA players indicated that i) they have similar leading causes of death and Crude Death Rates to sex- and race-matched controls from the US general population, ii) differences in death rates per Athlete-Year (AY) exist within time-dependent variables (e.g., birth decade) but less in more stable variables (e.g., handedness), and overall incidences of unnatural deaths per AY were less common across all predictors of mortality studied relative to natural deaths, and iii) a majority of the deceased player population died prematurely if the life expectancy was set at 75 years for each player, but less than 30% were considered young deaths if life expectancy at birth for each player was individually calculated. In addition, birth decade was a robust confounder of race and height's influence on lifespan, as black and taller players entered the NBA in later decades, giving them less opportunity to live a full life since our analyses only included deceased players.

Chapter Seven

A comprehensive examination of mortality outcomes of athletes from the four major sports found former MLB players to have a significantly higher imminent mortality risk relative to the other three major sports, although this was anticipated given MLB's early inaugural season resulting in more players whose life expectancies at birth were relatively lower. Further, NBA/ABA players generally had the highest relative proportion of death when standardizing data by debut year, although NHL and NFL players have recently (debut > 2005) had the highest proportion of death. Career length also appeared to play an overall factor on lifespan, where each one year increase in career length lowered the risk of death.

Type I vs. Type II Error: Limitations of Previous Work

This dissertation highlighted caveats to the type of statistical measures used in previous literature, along with potential ramifications of disseminating risks of death without considering

the unique characteristics of the population studied. In particular, Chapters Three and Five drew attention to the importance of independent scientific replication, using the precocity- and height-longevity hypotheses as examples of areas of research that are dominated by one researcher or team of researchers. Consequently, if extensive work is being performed by a small number of researchers, a larger onus on performing rigorous and appropriate methodological procedures may be needed.

From a hypothesis testing standpoint, a Type I error (i.e., a false positive), often thought to be the more problematic error outside of medical research, occurs when the null hypothesis is incorrectly rejected in favour of the alternative. In contrast, a Type II error (i.e., false negative) occurs when the null hypothesis is falsely accepted. As it relates to the studies conducted on precocity-longevity, both types of errors may be present. First, a Type I error in this context erroneously suggests that early career achievement reduces lifespan. As noted in the historical review of precocity-longevity literature, the life expectancy bias/artifact describes the potential that younger death age among high achievers may occur because having a younger age is associated with shorter life expectancy (McCann, 2001). There is also the possibility that early death (and hence early achievement) simply permits a person to enter a study sample. This ‘selection artifact’ (Simonton, 1994) means that the absence of still-living early achievers can skew effects to artificially support a precocity-longevity effect. Both of these artifacts describe the inherent potential for bias when studying a sample of *only* deceased eminent achievers (which all previous research has done) which can exaggerate the effect of the relationship, thus increasing the likelihood of a Type I error (i.e., identifying a precocity-longevity effect when one may not exist).

A related issue with Type I error is confusing statistical significance with practical significance (addressed in Chapter Seven). While MLB players were found to have a higher risk of death relative to the other three sports, statistical significance was most likely driven by the MLB inaugural season (1871) occurring much earlier than the other three sports (≥ 1917), predisposing the Kaplan-Meier estimate to produce a statistically significant hazard risk. As a result, the meaningfulness of this result is diminished, as the finding would have likely been more useful if each of the four major sports started on the same date. In addition, large sample sizes likely caused statistically significant Cox regression results, as the variables measured had predominantly small effect sizes with confidence interval ranges close to 1.00. The extremes of this Type I error issue were demonstrated in Chapter Six, where birth decade largely confounded the unadjusted predictors of race and height which portrayed black and relatively taller players as having significantly higher risks of earlier death.

A Type II error in the context of precocity-longevity suggests early career achievement does not reduce lifespan when in fact it does. In sport, both Abel and Kruger (2007) and Lemez, Wattie, Ardern, and Baker (2014; Study 1 in Chapter Three) defined precocious achievement in athlete samples as the age at which athletes entered professional sport. However, the manner in which ‘precocity’ was operationalized may have reduced the likelihood of finding a significant precocity-longevity effect considering the age range for professional sport debut is typically quite narrow. This makes Abel and Kruger’s (2007) finding all the more interesting; however, they excluded living players in their sample.

In any case, this dissertation addressed the Type I and Type II error likelihoods through rigorous methodological approaches which allowed for more ecologically valid analyses to be performed. In Chapter Three, while the first study on Canadian-born NHL players used ‘debut

age' as a measure of precocity, living players were included in the analysis, accounting for the aforementioned statistical artifacts. In the second study on NBA/ABA players, precocity was defined through *three* different indicators of achievement, in addition to using both 'deceased only' and 'deceased and living' subsample strategies. It should be noted, however, that examining subgroups (e.g., players who were all-stars) may inflate Type I error, as more heterogeneities are likely to be found in smaller samples. In Chapter Six, an entire section was devoted to highlighting statistical biases from regression analyses in populations such as NBA/ABA players. More specifically, cohort effects were accounted for through combinations of odds ratio analyses that adjusted for different predictor variables, which illustrated the robust impact of birth decade on statistical significance. This bias was further corroborated and discussed in the *cause* of death interpretation, where those who were born earlier (i.e., predominantly white players) had a greater likelihood of dying from natural/old age causes relative to black players.

(More) Practical Implications

The key findings of this dissertation suggest that athletes from the four major sports in North America have favourable mortality outcomes, although numerous factors need to be taken into consideration. The value of focusing on professional athletes rather than recreational or amateur athletes is that more biological and occupational factors are documented and publicly accessible at the elite levels of sport compared to lower levels of competition, thus allowing this dissertation to control for a greater number of variables that help increase the explained variance of mortality. The findings of this dissertation may also have applications for semi-professional and collegiate athletes, where similar biological and behavioural characteristics of athletes at lower levels of sport may illuminate health risk factors earlier, helping to increase overall athlete

well-being and reduce health outcome disparities within the athlete population. In turn, a comprehensive understanding of the implications of involvement in professional sport is necessary for the formation of evidence-based models of athlete development and care.

The systematic review examining mortality and longevity of elite athletes in Chapter Two detailed the differences and nuances of health outcomes between and within sport(s). Investigating these large populations of athletes can provide broader and deeper awareness of the health status of elite athletes from sports with different physiological demands and behavioural expectations and challenge common assumptions regarding health risks of participating in certain high performance sports and/or positions. For example, the energy system demands of the four primary sports examined in this dissertation differ, where NBA and NHL players are generally exposed to more aerobic training and performance demands compared to MLB and NFL players. Training and performance demands differ between positions as well, such as a wide receiver needing to have higher aerobic capacity relative to a kicker in the NFL. Positional demands are also correlated with certain biological mechanisms that are conducive to performance but can be less favourable to health, such as NFL linemen being generally bigger in size relative to the other positions, and centers in the NBA whose exceptional heights assist in getting rebounds. Further, different playing eras between these four sports contained different socially-constructed factors that may have impeded long-term health, such as the widely known ‘steroid-era’ in MLB and anecdotal evidence of widespread drug use amongst NBA players in the 1970s and early 1980s.

In particular, this research informs our broader understanding of general athlete health, which can necessitate assessments of programs sporting organizations currently have in place for active and retired athletes. For example, the NBA, in conjunction with the Players Association

and the Retired Players Association, has recently sponsored a cardiovascular screening program that allows retired NBA players to receive free baseline tests and familiarize themselves with local cardiology groups (Smith, 2016). This is also an example of media and research attention working in a positive capacity through raising awareness of premature death in athletes (technology can also play a critical role in health education through ‘digital epidemiology,’ a timely way to receive health and disease information through digital data sources such as electronic devices; Salathé et al., 2012). While attention given to the recent string of former NBA player deaths from a cardiovascular-related cause likely produced the initial impetus to implement screenings, a by-product of this program may be more valuable to long-term health, helping former athletes build proactive rather than reactive health behaviours (e.g., regular doctor visits).

While this is a positive and proactive first step, more empirical evidence on elite athlete health risk factors is needed to bolster health insurance policies post-retirement. In particular, health issues that manifest several years after retirement need to be considered in future discussions on health insurance restructurings, particularly from contact- and combat-based sports. For example, the repercussions of repetitive head trauma have been at the forefront of recent media coverage of NFL players, with chronic traumatic encephalopathy (CTE), a neurodegenerative brain disease generally manifesting in later life, being identified as a primary by-product of long-term concussive and subconcussive injury (e.g., Huber, Alosco, Stein, & McKee, 2016).

From an alternative perspective, while investigating risk of death differences through different occupational and biological variables can help highlight aversive trends to lifespan that permeate throughout high performance athlete populations, different measures of lifespan can

provide interesting insight into the impact of death outside of the health domain. An example of this is the measure of years of potential life lost (YPLL) in Chapter Six, which is used to help quantify social and economic loss as a result of premature death (Gardner & Sanborn, 1990). For professional athletes, the determinants of health created from societal-level conditions such as opportunity, social connections and resources are generally superior relative to the general population; however, a high rate of premature death can impact economic productivity. Premature deaths of athletes from the four major sports in particular, which generate a substantial amount of revenue, can create societal burden and produce indirect economic costs from productivity losses. In contrast, premature death of an exceptional achiever can actually produce a short-term economic *gain*, such as a spike in album sales of a recently deceased artist. Nevertheless, the occasional economic boost from a high achiever's premature death is often short lived, and public health policies should aim to effectively target risk factors of early mortality to reduce economic costs to society. In sport, while YPLL may not have as large an economic impact as a chronic condition such as cancer in the general population (since the general population contains a very low proportion of elite athletes), this measurement is a microcosm of well-being that can nevertheless stimulate action to improve health and health education by illuminating precursors to specific causes of death, which in turn can cut healthcare costs through more health conscious behavioural change (e.g., altering diet to lower risk of diabetes, and by association, heart disease).

Future Directions

As the connection between physical activity and health continues to evolve, it is important for researchers to continue to investigate sport participants, which represent a considerable proportion of the post-industrial and developed population. Although typical

measures of mortality provide information about death and lifespan, we have a limited understanding of the contribution of specific diseases, injuries and underlying conditions (e.g., diet) to death (Parrish, 2010). In particular, studying elite athletes longitudinally (i.e., prior to death) will elicit a more complete understanding of their health outcomes and the causes that contribute to their death. For example, metrics relating to i) mortality and life expectancy, such as condition-specific mortality rates (e.g., diseases, injuries, occupational mortality, smoking-attributable mortality, etc.) and ii) self-perceived health states, such as reporting psychological distress, would provide more targeted and efficient outreach efforts to investigate the underlying causes of death as opposed to the *immediate* causes of death.

Parrish (2010) suggested that the relative value placed on long life compared to well-being during life varies. While measuring mortality and life expectancy has been the traditional approach to indicate the health status of a population, information regarding the quality of their physical, mental and social domains of life is limited (“Healthy People 2020,” 2010). Going forward, the quality of the years lived will be an important avenue for researchers to explore in more depth considering the slow but steady increases in life expectancies globally. For example, future work in this area could look to utilize the multidimensional concept of health-related quality of life (HRQoL) through Ferrans, Zerwic, Wilbur, and Larson’s (2005) model that encompasses various domains and characteristics to better explain HRQoL over time (as suggested in Bakas et al.’s 2012 systematic review). More specifically, *The Short Form (36) Health Survey* (Ware & Sherbourne, 1992) is an example of a questionnaire that could be administered to athlete samples to broadly capture their quality of life post-retirement from elite sport by assessing their physical and mental health. In turn, this can highlight important HRQoL

differences between former elite athletes and the general population to help determine whether participation in elite sport is indeed a healthy pursuit.

In addition, more research attention should be given to the career transition process and post-career experience of elite athletes, and the coping challenges that accompany retiring from sport, such as adjusting their (athletic) self-identity to meet their new day-to-day needs. Initial transitional challenges are typically influenced by whether discontinuation was normative (i.e., predictable and anticipated) or non-normative (i.e., involuntary); thus, using a lifespan model such as Wylleman and Reints' (2010) "whole career/whole person" conceptualization of career transitions of elite athletes could be valuable. Examining longitudinal health-related behaviour patterns in athletes after they retire can highlight the importance of adopting a holistic perspective when considering the entirety of a lifespan. In particular, a strong and exclusive athletic identity is associated with negative psychological outcomes such as inadequate coping, negative affect and disturbed mood (Stambulova, 2003). As physical health can moderate psychosocial health (and vice versa), future work examining former athletes' adaption to their post-athletic career life may assist in identifying factors leading to longer lifespans, such as illuminating sociobehavioural patterns of former athletes who lived exceptionally long.

Conclusion

The aggregation of different health measures and outcomes creates better assessment of public health research, practice and policy. This dissertation demonstrates that numerous biological and socially-constructed factors operating throughout the lifespan can have complex interactions and important implications on lifespan. Gavrilov and Gavrilova (2015) emphasized that knowledge of robust predictors of mortality and longevity is critical for actuarial science and practice. Key findings of this dissertation have practical implications for sport federations and

governing bodies (i.e., policies and programs underpinning sport organizations), as well as healthcare practitioners and clinicians. Therefore, as life expectancies continue to increase (Oeppen & Vaupel, 2002), public dialogue on health disparities between sub-populations and risk factors for premature death will be important, and continued research on athletes will assist sport stakeholders in implementing strategies to circumvent early incidence, predictors and causes of death.

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