The characterisation of cardiac structure and function of registered Australian jockeys

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Glossary

ACE	Angiotensin-Converting Enzyme
AFL	Australian Football League
AN	Anorexia Nervosa
BP	Blood pressure
bpm	Beats per minute
BSA	Body Surface Area
CI	Confidence interval
cm	Centimetres
CO	Cardiac output
d	Cohen's d effect size
E:E'	Mitral inflow early diastolic velocity to tissue Doppler mitral annular early diastolic velocity ratio
EMG	Electromyography
FMD	Flow-mediated dilation
GLS	Global longitudinal strain
HR	Heart rate
ICC	Intraclass correlation coefficients
Kg	Kilograms
L	Litres
LA	Left atrial
LAV	Left atrial volume
LAVi	Left atrial volume index

LV	Left ventricular
LVEDV	Left ventricular end diastolic volume
LVEDVi	Left ventricular end diastolic index
LVEF	Left ventricular ejection fraction
LVESV	Left ventricular end systolic volume
min	Minutes
Mitral E:A ratio	Ratio of peak velocity blood flow from left ventricular relaxation in early diastole to peak velocity flow in late diastole
ml	Millilitres
PWV	Pulsed wave velocity
RA	Right atrial
RWT	Relative wall thickness
RV	Right ventricular
SD	Standard deviation
SPSS	Statistical Package for the Social Sciences
STE	Speckle tracking echocardiography
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
SV	Stroke volume
TBW (%)	Total Body Water %
TDI	Tissue Doppler imaging
TRIMP	Training impulse
V	Cramer's V effect size

VED	Volume at end diastole
VES	Volume at end systole
WHO	World Health Organisation
2D	Two-dimensional

Publications and Presentations

Journal Articles

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Summary

Jockeys are a highly specialized group of athletes who are subject to intense physical demands during thoroughbred racing. On top of this, they are also required to maintain consistently low body weights with many employing weight shedding practices in order to do this. Despite the considerable physical challenges associated with race riding, jockeys have not traditionally been considered as athletes in the same way as competitors in other sports. Given the well-established cardiac adaptations observed in athletes across various sports, coupled with evidence linking calorie deficits, dehydration, and intense physical activity to changes in cardiac structure and function, it is plausible that the demanding nature of thoroughbred racing may induce similar adaptations in jockeys. However, there is a significant gap in research specifically investigating how the demands of competitive horse racing impact the cardiac health of jockeys. Hence, the aim of study 1 is to examine whether differences exist in the cardiac structure and function of Australian jockeys compared to age-matched controls from the general population. There is also a paucity of research comparing the cardiac structure and function of jockeys at different stages of their careers. As such, study 2 aims to address this gap by assessing the differences in race riding, workload, and weight management practices, and the cardiac structure and function of jockeys at varying career stages.

Chapter 2 compares jockeys with aged-matched members of the general population; revealing larger left ventricular (LV) and left atrial (LA) volumes in the jockey group, as well as increased LV mass when indexed for body surface area (BSA). These findings are consistent with adaptations observed in other athlete populations such as rowers and hockey players. Notably, these structural changes did not occur with impaired systolic function, as left ventricular ejection fraction (LVEF) remained normal. Interestingly, it was demonstrated that 17% of jockeys participating in this study exhibited impaired global longitudinal strain (GLS), a sensitive marker of subclinical myocardial dysfunction. This reduction in GLS is an area requiring further research where jockey exercise, weight management practices and career longevity should be analysed while assessing cardiovascular structure and function of these unique athletes.

Chapter 3 examined the influence of career stage between established (≥ 5 years race riding experience) and early career jockeys (< 5 years of race riding experience). It was

found that established jockeys demonstrated greater LV mass and there was also a trend toward more frequent GLS impairment. The chapter highlights the significant physical workload of jockeys, with both early and established career groups far exceeding the World Health Organization's (WHO) recommended physical activity levels. Additionally, weight management practices, including food restriction and dehydration techniques, were found to be widely employed in both jockey groups, raising concerns about their potential long-term effects on myocardial stress and function. While non-race day total body water (TBW (%)) levels were within normal ranges, race day dehydration may still influence cardiac function and is something that requires further investigation.

These findings underscore the need for further research into the long-term cardiovascular health of jockeys, particularly focusing on the effects of weight management practices and the cumulative impact of race riding. Longitudinal studies with larger cohort numbers are essential to determine the trajectory of cardiac adaptations and their potential risks, while also providing valuable insights into safe practices for weight management and cardiovascular health in this unique athletic cohort.

Declaration

This thesis presents work carried out by myself and does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; to the best of my knowledge it does not contain any materials previously published or written by another person except where due reference is made in the text; and all substantive contributions by others to the work presented, including jointly authored publications, are clearly acknowledged.

Angela Farley



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1 Introduction

1.1 Background

1.1.1 Thoroughbred racing and the physiological demands on jockeys in Australia

Thoroughbred horse racing is one of the oldest and most lucrative sports in the world, renowned for its dynamic blend of speed, skill, and strategy (1). Other than the racehorses, central to this high-stakes competition are jockeys, whose performance directly influences race outcomes.

In Australia, thoroughbred racing encompasses over 19,000 races annually, across metropolitan, regional, and rural racing tracks and involves approximately 844 registered jockeys (2). Races typically range from 1000 to 3200 meters in distance and are completed within two to four minutes depending on the race distance. Jockeys frequently participate in multiple races per day and multiple race meetings per week (2). Unlike many other professional sports, thoroughbred racing operates year-round, with no formal off-season (1). This unrelenting schedule places considerable physiological and psychological demands on jockeys, as they must continuously manage their fitness, weight, and recovery (1).

Both male and female jockeys compete on equal terms against each other in Australian thoroughbred racing, with various licensing categories to distinguish their levels of experience and specialization (3). These categories include amateur, apprentice, professional and jumps jockeys (2). Apprentice jockeys, who are typically younger and at the beginning of their careers, are required to undertake structured training programs under the supervision of licensed trainers (3). These programs are designed to develop technical proficiency, with progress primarily evaluated through riding competency (4). However, this competency-based approach overlooks physiological adaptations that may develop with increased race-riding experience. Apprentice jockeys often face significant physical demands as they acclimate to the unique challenges of the

profession, including managing bodyweight and building and maintaining fitness levels to ensure they develop the endurance required for competition (3). Additionally, they must learn to perform under the intense pressure of race riding, which demands rapid decision making, situational awareness, and a deep understanding of horse behavior (3). In contrast, established professional jockeys, those who have completed their apprenticeship and accumulated extensive race-riding experience, tend to have more refined technical skills and a greater ability to anticipate race dynamics (5). Despite the vital role jockeys play in racing, much of the research in the sport has historically focused on the performance and fitness of the horses, with far less attention given to the health, physiology, and performance optimisation of jockeys themselves (1).

This athlete group does not align neatly with traditional sports classification frameworks. Mitchell et al. (2005) proposed a framework which categorises sports based on the type and intensity of exercise with references to their chances of bodily injury from collision and the consequences of syncope. When viewed through this framework, jockeys exhibit characteristics consistent with both moderate-to-high static and moderate dynamic loading athletes (6). This hybrid profile aligns them, in part, with sports such as rowing, combat sports and gymnastics (7). However, several critical physiological and occupational distinctions make jockeys difficult to match up with other athlete groups. This is especially the case when occupational hazard is considered within this framework (7). Jockeys operate in an environment with one of the highest injury rates in professional sport, with a high risk of falls and collision-based trauma (4). As such, jockeys are considered within the highest-risk category for serious consequences in the event of a cardiovascular event occurring during activity (7).

Physiological studies reveal that jockeys experience elevated cardiovascular and metabolic demands during races, with average heart rates ranging from 130–180 beats per minute (bpm), peak heart rates of 150-190 bpm, and peak oxygen uptake (VO_2) values between 42.7 (±5.6) and 57.5 (±4.7) ml/kg/min (8-10). These metrics underscore the need for high aerobic and anaerobic fitness to meet the sport's unique physical challenges. The ‘martini glass’ posture adopted by jockeys during races which involves standing in the stirrups, knees bent, and body parallel to the horse, also requires significant isometric muscle activation, particularly in the legs and abdominals in order to maintain position while absorbing vertical and horizontal forces from the horse (11).

These isometric requirements place jockeys closer to athletes in sports like alpine skiing and wrestling, where muscular control and static endurance are integral to success (7).

Jockeys' exercise loads during races, quantified using metrics such as Training impulse (TRIMP) scores, are comparable to those experienced by elite athletes in other high-intensity sports, including cyclists and rowers during competition (3). Comparatively, sports like soccer and hockey, which are classified as high dynamic and moderate static in the Mitchell et al. (2005) framework, require repeated bouts of sprinting, direction change, and aerobic endurance, but they do not impose the same sustained isometric postural demands (11).

It is well known that chronic and intense physical activity, as undertaken by many elite athletes, leads to cardiovascular adaptations, including chamber enlargement, increased left ventricular (LV) wall thickness, and enhanced diastolic filling, all of which augment cardiac output (CO) during exercise (12). While these changes are well-documented in athletes from other sporting disciplines that have both static and dynamic exertion, such as rowing and cycling, the cardiovascular adaptations specific to jockeys remains largely unexplored (13). Table 1 below, adapted from Mitchell et al. (2005), offers a comparative overview of jockeys relative to other sporting populations based on their physiological and clinical risk profiles (7).

Table 1. Comparative overview of jockeys relative to other sporting populations

Athlete Type	**Static Load**	**Dynamic Load**	**Risk of Syncope**	**Key Features**
Marathon Runners	Low	High	Low	Aerobic, high-volume load
Weightlifters	High	Low	Low	High afterload, short duration
Rowers	High	High	Low	Combined pressure and volume load
Soccer Players	Moderate	High	Low	Intermittent sprinting, aerobic base, agility
Hockey Players	Moderate	High	Low-Moderate	High-intensity bursts, frequent direction change
Jockeys	High	Moderate	High	Isometric exertion, extreme risk, weight restriction

As well as cardiac chamber remodelling, sustained or repeated elevations in afterload during intense exercise may influence myocardial wall thickness, particularly of the left ventricular posterior wall (PW), and overall cardiac morphology (14). Jockeys are subjected to frequent surges in blood pressure during races due to elevated sympathetic activity, isometric exertion, and mechanical constraints imposed by racing posture. These conditions increase left ventricular afterload, which has been associated with concentric remodelling in other endurance athletes, including cyclists and rowers (12). Over time, such afterload-induced stress may promote thickening of the PW and interventricular septum (IVS), particularly in older or more experienced athletes (15). Although this study does not include direct vascular assessments, the potential influence of blood pressure responses and mechanical loading on myocardial structure, especially in relation to PW thickness, merits further discussion and investigation.

In addition to the physical demands of race riding, jockeys face the unique challenge of adhering to strict weight requirements to ensure fair competition. In Australian horse racing, the assigned weight for a jockey in a given race is determined by the race type, the horse's assigned handicap, and specific race conditions (2). These weights typically range from 50 to 61 kilograms, which includes both the jockey's body mass and the combined weight of their riding equipment, such as the saddle and safety gear (10). The system is designed to create a balance in performance potential among competing horses, with heavier weights often allocated to horses perceived as having a competitive advantage based on prior performance or pedigree (16).

For many jockeys, maintaining a body mass within this range requires meticulous weight management, often involving restrictive dietary practices, dehydration techniques, and intensive exercise regimens which will be discussed in greater detail in the following sections. Despite these challenges, the ability to "make weight" is crucial for jockeys to secure rides, and failure to meet the weight limit can result in lost earnings or penalties (10). Efforts to address these challenges by industry leaders include the introduction of minimum weight limits, which have been gradually raised in some jurisdictions to promote jockey health (10). Nonetheless, the delicate balance between meeting industry weight requirements and safeguarding jockey health remains a pressing issue within Australian horse racing (17).

Given their unique blend of static and dynamic exertion, combined with occupational stress and weight cycling, it is possible that jockeys exhibit distinct cardiac structural adaptations not yet fully characterized in the literature.

1.1.2 Chronic energy deficits and the effect on cardiac function in athletes

Chronic energy deficits, a state in which energy expenditure consistently exceeds caloric intake, are common among athletes involved in weight-sensitive sports such as mixed martial arts and boxing (18). The cardiovascular consequences of such deficits have been well-documented in clinical populations, such as individuals with eating disorders, but remain underexplored in athletic populations like jockeys, who engage in rigorous weight management practices to meet competitive requirements (19).

Jockeys routinely employ extreme methods to achieve and maintain the strict weight requirements for competitive racing. A recent study indicates that two-thirds of Australian jockeys lose weight by fasting prior to race day, reducing an average of 2% of their total body mass each time they employ this method (16). This practice is often compounded by other weight-loss strategies such as sauna use and dietary restriction, leading to chronic energy deficits (20). Given that jockeys may race three to four times weekly with no designated off-season, these methods are often used persistently throughout the year (21).

While these practices enable jockeys to meet weight requirements, they may also have unintended consequences for cardiovascular health. Chronic calorie restriction has been linked to reduced left ventricular ejection fraction (LVEF), CO, and chamber volumes in athletes that cut weight for combat sports such as mixed martial arts (18). However, the specific implications of these practices on cardiac function in jockeys remain poorly understood, especially considering their unique physiological and occupational demands.

The persistent calorie deficits and frequent fasting required for jockeys' weight maintenance may predispose them to cardiac remodeling and functional changes similar to those observed in populations with eating disorders. Studies on anorexia nervosa (AN) patients highlight a range of cardiac abnormalities, including reduced chamber sizes, impaired myocardial contractility, and altered strain patterns, particularly in the

apical regions (22). These findings underscore the potential for weight-shedding practices to contribute to early subclinical myocardial dysfunction in jockeys, detectable through sensitive measures such as global longitudinal strain (GLS). Despite potential implications for cardiovascular health, these effects remain unexamined in the jockey population.

1.1.3 Dehydration and its effects on cardiac function in athletes

Dehydration, often employed as a strategy for rapid weight loss in weight-sensitive sports, poses significant risks to cardiovascular function (23). Jockeys routinely employ dehydration as part of their weight management regimen, particularly in the hours and days leading up to a race. Studies have shown that jockeys typically lose 2–3% of their body weight in the hours preceding a race meeting and up to 6–10% in the week prior to a race meeting (16, 24). This weight reduction is commonly achieved through prolonged sauna sessions, fluid restriction, and wearing sweat-inducing gear during exercise which leads to fluid loss from the plasma (16). While these methods are effective for rapid weight loss, they can induce acute dehydration, which may have the potential to increase core body temperature while having a detrimental impact on CO (16, 23).

As mentioned above, dehydration has been demonstrated to reduce fluid from the plasma and blood volume, which diminishes venous return to the heart and subsequently reduces LV stroke volume (SV) and LV end-diastolic volume (LVEDV) (25). As a result, CO declines, forcing the heart to work harder to maintain adequate perfusion (26). This reduction in venous return has been associated with impaired myocardial deformation, as evidenced by small but significant impairment in GLS (25). This decline in GLS has been demonstrated in weight-cutting athlete populations such as combat sports (18).These changes may be particularly concerning for jockeys who repeatedly subject themselves to acute dehydration (16).

The effects of dehydration extend beyond cardiac function. Reduced plasma volume increases blood viscosity, elevating peripheral arterial resistance and blood pressure (BP) (26). This heightened resistance not only impairs muscle blood flow and thermoregulation during exercise but also increases the risk of ischemic heart disease and stroke over time (26). This increase in workload on the heart could therefore

contribute to long-term cardiovascular complications, particularly if these practices are sustained over years (25).

While the acute cardiovascular effects of dehydration have been previously investigated, the long-term implications of repeated rapid weight-loss practices remain unclear, particularly in athlete populations such as jockeys. Unlike endurance athletes or individuals experiencing occasional dehydration, jockeys frequently engage in these practices year-round to meet weight requirements for competition (16, 18). On top of this, jockeys commonly compete without being allowed to rehydrate. To ensure fairness and credibility in racing, there are regulations which require jockeys to be weighed an hour prior to a race and then reweighed again directly after competing. If there is a discrepancy between these weights then this will result in disqualification from the race and jockeys may face a suspension from racing or be ordered to pay large fines (27). This is unlike other weight-cutting sports that allow athletes to rehydrate once they have been weighed prior to competition (18). This raises questions about whether chronic exposure to dehydration coupled with the physiological demands of racing, could exacerbate cardiac remodeling or impaired function over time.

1.1.4 The effects of physical activity on cardiac structure and function

It is well known that the heart readily adapts to the demands of regular physical activity, with specific changes occurring in cardiac structure and function depending on the intensity, type, and duration of exercise (12). Extreme physical activity, such as that undertaken by elite athletes, imposes significant physiological stress on the cardiovascular system, leading to both adaptive and potentially maladaptive remodeling (28). While these changes are generally beneficial and can help to enhance performance, there is evidence to suggest that prolonged exposure to extreme exercise may predispose some individuals to adverse cardiac outcomes (29).

Athletes engaged in regular high-intensity training often experience a phenomenon known as athlete's heart, characterized by physiological remodeling of cardiac chambers (30). The extent and nature of cardiac remodeling that occur with physical activity are influenced by several factors, including the type of sport, training intensity, and individual predisposition (12). Endurance athletes, such as marathon runners and rowers, where the activity effort is sustained, frequently exhibit increased LVEDV,

improved diastolic filling, and increased chamber sizes (31). These adaptations support increased SV and CO during exercise (12). In contrast, sports requiring static or isometric effort, such as weightlifters, demonstrate increased LV wall thickness in a concentric hypertrophy pattern, enabling the heart to cope with elevated afterload during isometric exercise (12). Additionally, age, sex, and genetic predispositions play significant roles in determining the degree and pattern of cardiac adaptations. For example, male athletes tend to develop greater LV mass than their female counterparts due to differences in hormonal and hemodynamic responses to exercise (30).

Despite the generally beneficial effects of exercise on the heart, extreme physical activity over extended periods has been associated with maladaptive remodeling in some athletes (29). Cases of myocardial fibrosis and arrhythmias have been reported in endurance athletes who engage in chronic, high-intensity training regimens such as marathon runners and long distance cyclists (32). These findings suggest that while the heart can adapt to high workloads, chronic exposure to extreme exercise may increase the risk of structural and functional abnormalities.

While most exercise-induced cardiac remodeling is benign and reversible upon detraining, distinguishing physiological adaptations from early maladaptive changes remains challenging (33). Subclinical myocardial dysfunction, as detected by GLS or other advanced imaging techniques, may serve as a critical indicator for early detection of abnormalities and therefore intervention (34).

1.1.5 Global longitudinal strain – an advanced measurement of cardiac structure and function

Echocardiography is the most clinically utilized diagnostic imaging modality for assessing cardiac function (35). Echocardiographic strain analysis is a non-invasive imaging modality that allows for the quantitative assessment of myocardial mechanics throughout the cardiac cycle (36). Among the various strain techniques available, speckle tracking echocardiography (STE) has become one of the most widely utilized due to its precision, reproducibility and versatility (35). This grayscale-based technique employs specialized software algorithms to identify and track unique ‘speckle’ patterns within the myocardial tissue produced by ultrasound beams (36). These speckles serve

as reference points, enabling the calculation of myocardial shortening and lengthening in different directions as a percentage of deformation, referred to as ‘strain’ (36).

One key parameter derived from strain analysis is GLS, which measures the longitudinal deformation of the entire LV myocardium (35). GLS is typically obtained from the apical four-chamber, two-chamber, and long-axis echocardiographic views. This parameter reflects the average strain across all myocardial segments, providing a comprehensive assessment of LV systolic function (36). GLS has gained prominence as a sensitive and reliable marker for detecting subclinical myocardial abnormalities, prior to the development of symptoms or manifestation of clinical changes (37). This makes GLS a valuable tool for identifying early stages of myocardial adaptation, damage, or dysfunction. Notably, GLS is considered a strong predictor of heart failure and mortality, outperforming traditional measures like LVEF (37). A comparative study reported an interobserver intraclass correlation coefficient (ICC) of 0.92 for GLS, significantly higher than the 0.80 reported for LVEF, underscoring GLS's superior reliability and reproducibility (37).

Another advantage of GLS is its broad applicability across a variety of populations and clinical scenarios (35). GLS has demonstrated utility in conditions ranging from heart failure with both preserved and reduced LVEF, to asymptomatic individuals with risk factors for cardiovascular disease (36). Its ability to detect subtle myocardial changes makes it a preferred method for monitoring early cardiac adaptations, particularly in athletic populations or individuals undergoing extreme physical stress, where traditional parameters like LVEF may remain within normal limits (33).

While a reduction in GLS is commonly associated with intrinsic myocardial dysfunction, it is important to acknowledge that GLS is also highly influenced by loading conditions, particularly preload and afterload (38). Therefore, a decrease in GLS does not necessarily imply pathological myocardial impairment (38). Flanagan et al. (2023) emphasise that GLS values in endurance-trained athletes, elite cyclists, and rugby players are often similar to those in sedentary controls, despite significant cardiac remodelling, indicating that small reductions in GLS may reflect physiological adaptation rather than disease (38). Additionally, athletes may experience reductions in stroke volume due to reduced LV filling, particularly in settings such as dehydration, energy restriction, or hypovolemia which have been demonstrated to be common among

professional jockeys (21). In these contexts, a larger LV may require less contractile shortening to maintain output, which can appear as a mildly reduced GLS even in the presence of normal or enhanced systolic function (38).

Given its high validity, ease of use, and ability to detect early myocardial changes, GLS has become the investigational tool of choice for advanced cardiac functional assessment (37). For this body of work, GLS was employed as a primary measure of cardiac structure and function, providing a robust and reproducible metric to assess potential myocardial adaptations and subclinical changes. That being said, while GLS is considered a highly sensitive parameter, its interpretation must be contextualised with broader cardiac haemodynamic assessment (38). A reduction in GLS, particularly in the athletic population, may not reflect subclinical myocardial disease but rather an adaptive or load-dependent response (38). This is particularly relevant in this research, where GLS was employed as a primary measure of cardiac structure and function in jockeys. Its use was combined with assessment of multiple conventional volumetric cardiac parameters to ensure that any observed changes in GLS were interpreted within a robust, multifactorial study accounting for the unique physiological stressors of thoroughbred horse racing.

1.2 Research aims and significance

Given the well-documented cardiac adaptations observed in athletes from various sports through echocardiography, and the evidence linking calorie deficits, dehydration, and extreme physical activity to cardiac adaptations, it stands to reason that the intense demands of thoroughbred racing may lead to changes in cardiac structure and function in jockeys. Jockeys are uniquely exposed to chronic stressors, including high training volumes, rapid weight fluctuations, frequent dehydration, and repetitive high-intensity physical exertion during races. Despite these extreme demands, there is a paucity of research examining how competitive horse racing influences the cardiac structure and function in this population.

Also, there is limited research that exists comparing the cardiovascular health of jockeys at varying career stages. Understanding how cumulative exposure to racing-related stressors affects the heart over time is essential, particularly given emerging concerns

regarding the long-term cardiovascular health of other retired athlete groups (38). The potential consequences of cardiac remodelling in jockeys may include increased risk of arrhythmias, myocardial fibrosis, and even heart failure later in life, as evidenced in retired athletes (39). Thus, the clinical relevance of this research lies in its ability to identify determine if there are cardiac adaptions that occur to jockey hearts and if there are early markers of adverse cardiac adaptation. It will also help to inform cardiovascular monitoring strategies during and after a jockey's career. Therefore, this study sought to explore the physiological effects of competitive racing on the cardiac structure and function of jockeys at different career stages.

Accordingly, this thesis was guided by two primary aims:

- To determine if there are differences in the structure and function of Australian jockey's hearts when compared to age matched controls from the Australian general population (Chapter 2).
- To investigate differences (if any) in heart structure between early career jockeys (<5 years competing) and established career jockeys (>5 years competing) while also examining differences in workload and weight shedding practices between the two cohorts (Chapter 3).

Multiple measures of cardiac structure and function, including GLS were assessed using echocardiography in both Chapter 2 and Chapter 3 to determine differences, if any, between groups.

As such our hypotheses for this thesis were:

- Cardiac remodeling such as increased cardiac chamber sizes and greater LV mass, would be demonstrated in jockeys when compared to the general population (Chapter 2).
- Established career jockeys would display more pronounced cardiac adaptations, including larger chamber sizes and increased LV mass, with these changes becoming more significant as their careers progress (Chapter 3).

The findings of this thesis may offer valuable insights for the racing industry regarding the impact of competitive racing on jockeys' cardiac health. Additionally, they shed light on the unique athletic adaptations of jockeys' hearts resulting from their careers,

providing a foundation for future research such as longitudinal studies aimed at identifying whether these adaptations align with those observed in other athlete populations and if they predispose retired jockeys to long-term cardiovascular complications.

2 Cardiac structure and function of elite Australian jockeys compared to the general population: An observational cross-sectional study

2.1 Chapter Overview

Chapter 1 introduced Australian thoroughbred racing and the involvement of jockeys in the sport. It also discussed the physiological demands of racing placed on jockeys, the effects of chronic energy deficits, dehydration, and the effects of extreme physical activity on cardiac structure and function in relation to athletes and other clinical populations. An overview of echocardiography and global longitudinal strain (GLS) was also provided to give an idea of its use in detecting myocardial subclinical changes.

The following chapter (Chapter 2) is an observational cross-sectional study that investigates the cardiac structure and function of Australian jockeys and compares them to the Australian general population.

A paper based off the manuscript presented in Chapter 2 has been published in *Sports Medicine Open*.

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To ensure consistency of numbering and to maintain consistency across the thesis the referencing format has been updated and is presented at the end of the thesis.

2.2 Abstract

Background

Research highlights the intense physiological demands of thoroughbred racing on jockeys, with elevated heart rates and substantial oxygen uptake, confirming the rigorous physical nature of the sport, however, the cardiovascular changes resulting from the physical demands of thoroughbred racing remain unexplored in Australian jockeys.

Therefore, the objective of this study was to compare measures of cardiac structure and function of professional Australian jockeys to that of the general population and to determine if there are differences in heart structure and function detected using echocardiography.

Methods

Forty-six jockeys and thirty-three participants from the general population underwent two-dimensional echocardiography, which included all standard views and measurements. Each measurement was compared between groups using a Mann-Whitney U test.

Results

Groups were matched for age (jockeys (35±12 years) and controls (36±13 years)). Jockeys were shorter (1.64±0.07 vs. 1.75±0.09m, $p<0.001$), lighter (56.5±6.0 vs. 74.2±12.9kg, $p<0.001$) and had a lower body surface area (BSA) (1.55±0.17m2 vs.1.9±0.2m2, $p<0.001$). Jockeys had a larger absolute left ventricular end diastolic volume (LVEDV) than the control group (120±18.2 ml vs.109.3±29.0 ml, $p=0.05$) which had a larger variation when indexed for BSA (78.0±12.2 ml/m2 vs. 57.5±13.3 ml/m2, $p<0.001$). Jockeys demonstrated a higher left ventricular (LV) mass index (79.4±18.1g/m2 vs 64.2±15.4g/m2, $p<0.001$). Left atrial (LA) volume index was larger in jockeys (33.4±6.5mL/m2 vs. 26.3±7.0mL/m2, $p<0.001$). There were no differences in GLS for either group overall (-19.3±3.0 vs. -19.8±1.6%, $p=0.52$), but 17% of the jockey group demonstrated an abnormal GLS.

Conclusions

Jockeys have adaptations to their cardiac structure and function compared to the general population. Differences could be attributed to chronic physiological demands of racing and should be considered in future research involving jockeys.

Keywords

Thoroughbred horse racing, jockey, athlete's heart, echocardiography, 2D echocardiography

Key Points

- Jockeys exhibit distinctive cardiac adaptations that are comparable to other athlete groups, including larger LV and LA volumes, and LV mass when compared to the general population.
- These adaptations are likely attributed to the chronic physiological and physical demands of horse racing.
- Further research should explore whether these changes increase with time spent participating in the sport.

2.3 Introduction

Thoroughbred horse racing is one of the oldest and most lucrative sports in the world (40). Jockeys are at the forefront of this dynamic and at times perilous competition with their performance having a direct impact on race outcomes (41). In Australia, there are approximately 844 registered jockeys participating in over 19,000 races annually (2). Race riding demands physical strength, stamina, and quick decision-making during races (11). Although there are many studies on the race fitness required by equine athletes, there is little data around jockey participants (11).

A literature review of the physiological demands of racing conducted by Ryan and Brodine (2021) identified only three studies that measured heart rate and mean oxygen uptake (VO_2) of jockeys during race conditions. It was demonstrated that jockeys experience elevated heart rates throughout each race (130-180 beats/min) with peak heart rates ranging from 150-190 beats/min (8, 9, 42). Substantial oxygen uptake was also demonstrated during races with oxygen uptake values ranging from 42.7 (±5.6) to 57.5 (±4.7) ml/kg/min (8, 9, 42). These findings indicate the rigorous physical nature of the sport and demonstrate the need for endurance (isotonic) fitness for this group of athletes (8, 9, 42).

On top of endurance (isotonic) fitness, the unique crouched posture that jockeys maintain during races requires strength (isometric) fitness (11). This posture, often referred to as the ‘martini glass’ position, involves jockeys standing in the stirrups while keeping their knees bent and their bodies forward and parallel to the horse’s body (2). They must also stay low and close to the horse's neck (2). This position ensures better aerodynamics and helps jockeys to absorb the horse's movements through their legs.

Drawing from studies utilizing skin surface electromyography (EMG) and Training Impulse (TRIMP) scores, (used to quantify the load or intensity of an athlete’s training over time and originally defined as the product of training volume, measured in minutes, and training intensity, normally measured as average heart rate) to quantify the muscle activity used by jockeys while maintaining this unique squat posture, it is clear that jockeys experience isometric exercise loads comparable to elite athletes in other sports such as soccer players (11). In a study by Legg et al. (2022) the TRIMP score for jockeys during 16 professional races was quantified at 292 ± 106 arbitrary units (au) (11). This is noteworthy considering the TRIMP experienced by a professional soccer

player during a 1.5-hour match approximates ~190 au, whereas that of a world-class marathon runner participating in a 2-hour race reaches approximately 275 au (11). These findings highlight the quasi-isometric muscle activation that occurs during racing and therefore the athletic requirements while maintaining this position, indicating that jockeys require a unique blend of both endurance and strength fitness (11), and suggest they may experience cardiovascular changes in line with other athletic populations.

Cardiovascular physiological adaptations can occur due to the unique demands of regular and intense physical activity in the hearts of athletes (28). Adaptations commonly seen due to chronic and high-intensity exercise include enlargement of all four cardiac chambers, an increase in LV wall thickness, and enhanced diastolic filling of the LV (31). This remodeling allows for increased stroke volume (SV) which in turn increases cardiac output when exercising (31). Whilst these changes have been well demonstrated in many elite athlete groups such as rowers and cyclists (12), the cardiovascular changes resulting from the physical demands of thoroughbred racing remain unexplored in this population of athletes.

The physiological demands of this sport on jockey participants extend beyond mere athleticism. Beyond the elite fitness requirements previously mentioned, jockeys face the additional challenge of maintaining a low body mass (50-61 kg) to meet race-related weight requirements (42). Often this is daily and, as there is no 'off season', many attempt to 'cut weight' year-round (43). Weight-loss techniques, including jogging in sweat gear, hot baths, and saunas, coupled with severe energy restriction and dehydration, are commonly used by jockeys to maintain a chronically low body mass (8). There is evidence to suggest that chronic food and fluid restriction can have detrimental effects on cardiac structure and function in the general population as seen in patients with anorexia nervosa (AN) (22). These practices can lead to decreased LV chamber size, LA enlargement, impaired myocardial contractility, altered diastolic function, reduced stroke volume, and decreased cardiac output (22). Additionally, changes in GLS may indicate impaired myocardial performance (22). The combination of these adaptations can potentially increase the risk of cardiac dysfunction, arrhythmias, and other long-term cardiovascular complications (22, 25, 26). However, to the authors' knowledge, this has yet to be investigated in jockeys.

Therefore, the aims of this study were to determine if there are differences in heart structure and function detected using echocardiography in registered Australian jockeys when compared to the general population. We hypothesised that cardiac remodeling related to athletic training, such as increased cardiac chamber sizes and greater LV mass, would be demonstrated in jockeys when compared to the general population.

2.4 Materials and Methods

2.4.1 Participant Selection

Professional Australian jockeys holding a current riding license in South Australia and Western Australia were invited to take part in the study. Data were collected from all jockeys on days of rest from race riding. Recruitment for this study occurred during 2020 which affected recruitment of the control group due to COVID-19 imposed restrictions. Therefore, the control group was comprised of participants who had previously participated in another ethically approved research project that collected all relevant cardiovascular data by one of the authors. Whilst there were no restrictions on physical activity for participants recruited to the study, they were excluded if they were <18 years of age or if there was a previously undocumented heart abnormality discovered during the echocardiogram. All participants gave written informed consent, and the study was approved by the University of South Australia Human Research Ethics Committee (project number: 204235).

2.4.2 Anthropometric Measurements

Immediately prior to their echocardiogram, anthropometric data were collected from all participants. Height (with shoes and socks removed) was measured to the nearest centimetre using a portable stadiometer (Seca 213, Hamburg, Germany). Body mass and Total Body Water % (TBW (%)) for the jockey group were measured in minimal light clothing using portable digital weighing scales (Tanita Innerscan, Body Composition Monitor, BC-541). As the control group was comprised of participants who had previously participated in another study that did not obtain measurements of TBW, this is not reported for the control group. Body Surface Area (BSA) was calculated according to the formula of Dubois and Dubois as a ratio of weight (kg) to height (m) (44).

2.4.3 Echocardiography

Two-dimensional (2D) echocardiography included all standard views and measurements. These were taken in accordance with American Society of Echocardiography guidelines (45). All images were acquired from parasternal and

apical windows with participants in the left lateral decubitus position and imaging was completed by one member of the research group, a qualified echocardiographer. Commercially available echocardiography systems with either a M55c XD clear Matrix or 4Vc phased array transducer were used for image acquisition (GE E95, Vivid IQ, General Electric, Horten, Norway). Images were recorded digitally in cine-loop format. Advanced image analysis was performed using commercially available analysis software (TOMTEC Image Arena and Autostrain analysis, Unterschleissheim, Germany). As part of a standard echocardiogram, specific images for diastolic function including mitral valve inflow and tissue Doppler imaging (TDI) were taken. LA volume was measured from both the apical 4 and 2 chamber views with care to open the LA to its major axis. LV volumes and ejection fraction were measured using the Simpson's rule of discs from the apical 4 and 2 chamber views where the major axis of the LV was found. Stoke volume was calculated from the LV outflow tract diameter and velocity time integral. LV GLS was calculated from LV focused images from the apical 4-chamber, 2-chamber, and long-axis views. Studies were deemed inadequate for GLS analysis if 2 or more LV segments could not be visualized. A region of interest was automatically applied by the software to the endocardial and epicardial borders, with tracking adjusted to cover the entire myocardium. GLS was calculated as the change in the length of line of the region of interest across all apical view from diastole to systole. To determine intra-rater reliability, the echocardiographic data from 20 jockeys and 20 normal controls was analysed twice with a minimum duration of one month between measurements. Intra-operator variability during the image collection process was determined using intraclass correlation coefficients (ICC) with an ICC < 0.5 indicating poor agreement, 0.5 to 0.7 moderate agreement and >0.8 good agreement (46).

2.4.4 Lifestyle Questionnaire

Jockey participants were also required to complete a lifestyle questionnaire modified from a previous study (20). The questionnaire collected information on years of jockeying, race riding statistics and weekly exercise activities completed in addition to race riding. Information on eating habits, previous medical history, medications and cardiovascular risk factors was also obtained as part of the questionnaire. Where applicable, participants were instructed to select the most appropriate descriptor, select as many options as deemed personally relevant or type extra information. The survey

tool Qualtrics (XM, Provo, UT) was used to design and distribute the questionnaire and to record responses. The complete lifestyle questionnaire is presented in Appendix 1.

2.4.5 Statistical Analysis

This study was reported in accordance with the Strengthening the Reporting of Observational Studies (STROBE) guidelines. A completed STROBE document for this study is presented in Appendix 2.

All statistical analyses were conducted using Statistical Package for the Social Sciences (SPSS) (v26, IBM, USA) with the level of statistical significance set at $p<0.05$. Any participants without full data sets were excluded from analysis. Parametric data were presented as mean ± standard deviation (SD) and nonparametric data as median (interquartile range) unless otherwise stated. A Mann-Witney U test to determine differences between the specific groups, jockeys, and general population, was used as the data were not normally distributed. Cohen's effect size [ES] was calculated to determine differences between the two groups with the difference between the means divided by the pooled standard deviation calculated. Effect sizes were calculated (= z /sqrt(N)) and interpreted as follows: <0.20 = *trivial*, 0.20 – 0.49 = *small*, 0.50 – 0.79 = *moderate*, and >0.79 = *large* (47).

Furthermore, as BSA has been suggested to be a potentially flawed indexing method for cardiac parameters in individuals at the extremes of body size (48), an exploratory sub-analysis was conducted to identify whether the observed differences in cardiac parameters might be influenced by differences in body size, rather than solely by training or occupational factors. To achieve this, we compared 19 echocardiographic and anthropometric variables between the eight smallest participants in the control group and eight largest participants in the jockey group using the same statistical methods outlined above. As this analysis was exploratory, it is not attached to any formal hypothesis testing or sample size estimates. Rather, it will be used as a sensitivity analysis to provide insight into whether the reported outcomes are being notably impacted by body size. Furthermore, to ensure our findings were robust to different approaches for controlling body size differences, we conducted an additional sensitivity analysis using multiple linear regression with BSA as a continuous covariate

on all absolute variables that were indexed for BSA (i.e., stroke volume, LVEDV, LA vol, and LV mass), rather than simple indexing.

In addition, as the groups were not sex matched, and this is a known area of variation in terms of cardiac remodeling (12), subgroup analysis was also performed comparing the females in the jockey group to the females in the control group. Males in the jockey group were also compared with the males in the control group.

Sample size was calculated using the normal value of GLS (-20 ± 3%) and the expectation of a 10% clinically relevant change in GLS between the normal population and jockeys. With reference to Diggle et al. (2002) this means that a sample size of 13 participants in both groups was required for 80% power to detect a clinically significant difference between jockeys and controls with a significance level (α) of 0.05 (49). To account for possible participant dropout, we aimed to recruit a minimum of 20 participants per group.

2.5 Results

Forty-six Australian jockeys (age range: 18-51 years of age) and thirty-three participants from the general population (control group) (age range:18-52 years of age) consented to take part in this study. Descriptive and mean anthropometric data for the jockey and control groups are presented in Table 2. The gender distribution for groups included 17 males and 16 females within the control group (N=33) and 32 males and 14 females in the jockey group (N=46). There were no significant differences in age ($p=0.54$) and sex ($p=0.11$) between the jockeys and the control group. Jockeys were shorter ($p<0.001$), weighed less ($p<0.001$) and therefore had a lower BSA than the control group ($p<0.001$). TBW (%) and blood pressure (BP) was recorded for the jockey group only. Minutes of physical activity per week which included aerobic activity that was not related to riding in races was recorded for the jockey group only as well. Also, there were no jockeys that listed any known previous cardiovascular issues as part of this study.

Table 2. Descriptive data for controls and jockeys

Value	**Control (N = 33)**	**Jockey (N = 46)**	**U =**	**p-Value**	**Effect Size**
Age (years)	36±13	35±12	697.5	0.54	0.07
Sex (males %)	17 (52%)	32 (70%)	622.0	0.11	0.19
Weight (kg)	74.2±12.9	56.5±6.0*	119.0	<0.001	0.73
Height (m)	1.75±0.09	1.64±0.07*	252.5	<0.001	0.58
BSA (m2)	1.9±0.2	1.55±0.17*	118.0	<0.001	0.73
TBW (%)	Not recorded	59.3±4.4	N/A	N/A	N/A
HR (bpm)	60±11	65±13	577.5	0.23	0.14
Systolic BP (mmHg)	Not recorded	131±16	N/A	N/A	N/A
Diastolic BP (mmHg)	Not recorded	82±10	N/A	N/A	N/A
Minutes physical activity per week (mins)	Not recorded	1182±414	N/A	N/A	N/A

*p<0.05 compared with control group: U - Mann-Witney U test value, BSA – Body surface area, TBW (%) – Total body water %, HR – heart rate, BP – blood pressure. Values are presented as mean ± standard deviation or number and (percentage). Bold text indicates significant p-values.

The results of the echocardiographic measurements for the jockey and control groups are presented in Table 3. Even though their height, weight and BSA were less than the control group, jockeys had a larger LVEDV ($p=0.05$), which remained when indexed for BSA ($p<0.001$) and higher LVEF ($p< 0.001$). Example images representing the comparison of group averages of these values can be seen in Figures 2A and B.

Absolute LV mass did not differ between the two groups ($p=0.92$); however, the jockey group demonstrated a higher LV mass index than the control group ($p<0.001$). The male control group demonstrated a higher LV mass and LV mass index than the female control group (100.5±25.3 vs 146.5±28.3 for LV mass, $p<0.001$, female vs male and 56.5±12.7 vs 73.3±13.4 for LV mass index $p=0.001$, female vs male). However, the difference was not as significant between the female and male jockeys: (107.2±25.3 vs 131.1±38.8 for LV mass $p=0.032$, female vs male and 74.9±18.0 vs 81.3±18.1 for LV mass index, $p=0.18$ female vs male. Figure 2 presents LV mass and LV mass index by participant group.

Furthermore, LA volume values were larger in jockeys, but only when indexed for BSA ($p<0.001$). Example images representing the comparison of group averages of the chamber sizes can be seen in Figures 3A and B.

A lower SV ($p= 0.021$) was also demonstrated by the jockey group. However, when SV was indexed for BSA there was no longer a difference between the groups ($p=0.32$).

Relative wall thickness (RWT) was statistically significant between controls and jockeys (0.32±0.05 vs 0.37±0.08, $p<0.001$ respectively). This held true between male controls (0.32±0.04) vs jockeys (0.37±0.08, $p=0.012$) and female controls (0.32±0.05 vs jockeys 0.37±0.07, $p=0.019$).

Table 3. Echocardiographic measurements for controls and jockeys

Value	**Control (N = 33)**	**Jockey (N = 46)**	**U =**	**p-Value**	**Effect Size**
GLS (%)	-19.8±1.6 (-17 to -23%)	-19.3±3.0 (-11 to -25%)	695.0	0.52	0.07
LVEDV (mL)	109.3±29.0	120.0±18.2	547.0	0.06	0.22
LVESV (mL)	47.4±13.6	48.7±12.1	710.5	0.80	0.03
LVEF (%)	57±3	60.8±5.2*	386.0	<0.001	0.41
Mitral E:A ratio	1.9±0.6	2.1±0.9	654.5	0.42	0.09
E:E' (avg)	5.6±1.2	6.2±1.5	556.0	0.09	0.19
Stroke Volume (mL)	75.7±20.7	64.1±12.6*	473.0	0.02	0.26
Stroke Volume index (mL/m2)	39.8±9.3	41.7±8.2	601.0	0.32	0.11

Cardiac Output (L/min)	4.5±1.4	4.1±0.9	592.0	0.30	0.12
LA vol (biplane, mL)	49.7±13.8	51.6±10.7	593.5	0.34	0.11
LA vol index (mL/m2)	26.3±7.0	33.4±6.5*	311.0	<0.001	0.46
LVEDV index (mL/m2)	57.5±13.3	78.0±12.2*	195.0	<0.001	0.63
LV mass (g)	124.2±35.3	123.8±36.7	748.5	0.92	0.01
LV mass index (g/m2)	64.2±15.4	79.4±18.1*	429.0	<0.001	0.34
RWT	0.3±0.1	0.4±0.8	969.0	<0.001	0.23

*p<0.05 compared with control group: U - Mann-Witney U test value, GLS – global longitudinal strain, LVEDV – left ventricular end diastolic volume, LVESV – left ventricular end systolic volume, LVEF – left ventricular ejection fraction, Mitral E:A ratio – ratio of peak velocity blood flow from left ventricular relaxation in early diastole to peak velocity flow in late diastole, E:E' – mitral inflow early diastolic velocity to tissue Doppler mitral annular early diastolic velocity ratio, LA vol – left atrial volume, LV – left ventricular, RWT – relative wall thickness. Values are presented as mean ± standard deviation or number and (percentage). Bold text indicates significant p-values.

There was no difference in GLS between controls and jockeys in both female (control = -20.0±1.9% vs jockeys = -20.1±3.2%, p=0.6) or male (control = -19.6±1.5% vs jockey -18.9±2.9%, p=0.42) groups. However, despite the GLS being the same in both the jockey and control groups, there were 8 (17%) jockeys who had a GLS more impaired than -16% (the widely regarded value used in echocardiography for GLS comparison), whereas all the control group demonstrated normal GLS (50). Of these 8 jockeys, 7 had been riding for more than 5 years and 1 had been riding for less than 5 years. 5 were male and 3 were female. 1 was between the ages of 20-30 years, 3 were between the ages of 30-40 years and 4 were between the ages of 40-50 years.

The eight largest jockeys and eight smallest of the control group were also compared. There was no difference in BSA between the 2 groups (p=0.28) suggesting that any cardiac changes observed are independent of body size. LVEDV, LVESV, LA volume, LVEDV indexed for BSA, LV mass and LV mass index were all larger in jockeys compared to the control group ($p<0.05$). Furthermore, the sensitivity analysis using BSA-adjusted regression confirmed our primary findings. Significant differences in LVEDV and LA vol indexed for BSA were replicated, albeit with slightly different p-values (LVEDV, $p<0.001$; LA vol, p=0.012). The differences in LV mass indexed for BSA remained highly significant ($p<0.001$) and differences in SV indexed for BSA remained non-significant, albeit with a larger p-value (p=0.99), reinforcing the notion that SV differences were attributable to body size rather than cardiac adaptation.

Females in the control group and the jockey group, along with males in each group were compared. The results remained significant for weight, height, BSA, left ventricular end diastolic volume index (LVEDVi), LVEF and left atrial volume index (LAVi) ($p<0.05$). However, only female jockeys demonstrated a higher LV mass index compared to the female control group 74.9±17.9 vs 56.5±12.7 g/m^2, p=0.004). Whereas male jockeys did not when compared to the male control group (81.3±18.1 vs 73.3±13.4 g/m^2, p=0.13). ICC of all measurements ranged from 0.85 to 0.95 (all p values <0.0001) indicating good intra-observer agreement. The results of the echocardiographic measurements separated for sex comparison of jockey vs control groups are presented in Table 4 and Figure 1.

Table 4. Echocardiographic measurements for comparison of males and females in each group

Value	**Female Control (N = 16)**	**Female Jockey (N = 14)**	**Male control (n=17)**	**Male jockey (n=32)**
HR (bpm)	61±11	68±12	60±11	64±14
GLS (%)	-20.0±1.9	-20.1±3.2	-19.6±1.5	-18.9±2.9
LVEDV (mL)	94.3±26.9	107.8±13.4	122.6±24.5	125.3±17.6
LVESV (mL)	39.4±12.8	43.4±13.0	54.5±10.1	51.0±11.1
LVEF (%)	58.7±3.3	62.5±5.1*	55.6±2.9	60.1±5.1#
Mitral E (m/s)	0.84±0.15	0.82±0.14	0.71±0.11	0.79±0.20
Mitral A (m/s)	0.44±0.13	0.40±0.11	0.41±0.11	0.44±0.13
Mitral E:A ratio	2.0±0.6	2.1±0.4	1.9±0.7	2.0±1.1
E:E’ (avg)	6.0±1.3	6.2±1.5	5.3±1.1	6.2±1.6#
Stroke Volume (mL)	62.6±12.9	57.1±9.7	87.2±19.7	67.2±12.6#

Stroke Volume index (mL/m2)	35.3±7.0	39.8±6.4	43.8±9.4	42.5±8.9
Cardiac Output (L/min)	3.8±1.0	3.8±0.9	5.2±1.5	4.2±0.9#
LA vol (biplane, mL)	42.1±11.3	44.5±8.2	56.8±12.2	54.3±10.3
LA vol index (mL/m2)	23.7±5.8	31.2±6.2*	28.8±7.3	34.2±6.4#
LVEDV index (mL/m2)	52.8±12.0	75.2±9.2*	61.8±13.2	79.3±13.3#
LV mass (g)	100.5±25.3	107.2±25.3	146.5±28.3	131.1±38.8#
LV mass index (g/m2)	56.5±12.7	74.9±18.0*	73.3±13.4	81.3±18.1

*p<0.05 between female control and female jockey, #p<0.05 between male control and male jockey: GLS – global longitudinal strain, LVEDV – left ventricular end diastolic volume, LVESV – left ventricular end systolic volume, LVEF – left ventricular ejection fraction, Mitral E:A ratio – ratio of peak velocity blood flow from left ventricular relaxation in early diastole to peak velocity flow in late diastole, E:E' – mitral inflow early diastolic velocity to tissue Doppler mitral annular early diastolic velocity ratio, LA vol – left atrial volume, LV – left ventricular. Values are presented as mean ± standard deviation or number and (percentage). Bold text indicates significant p-values.

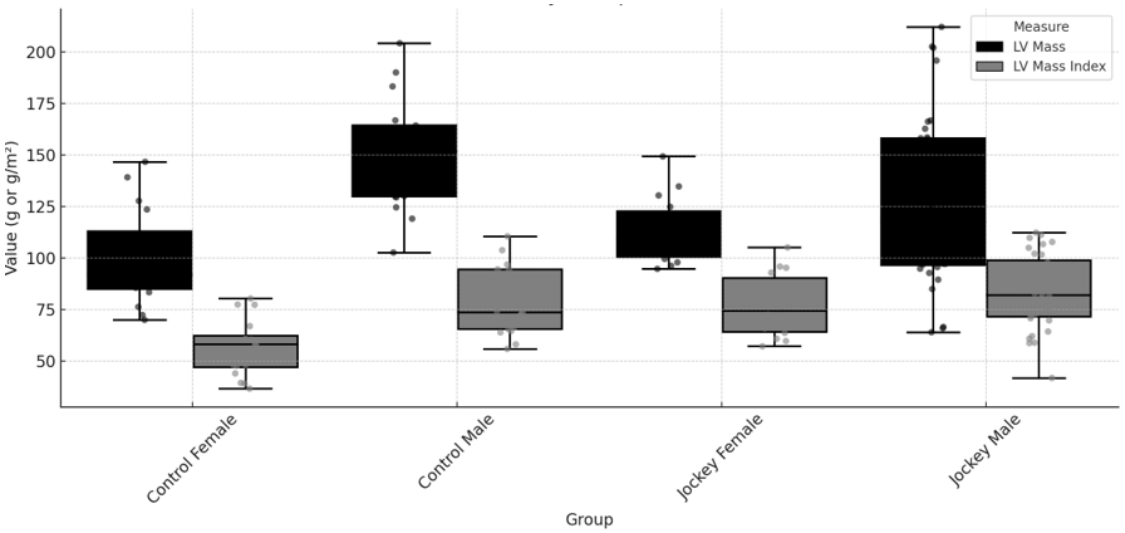
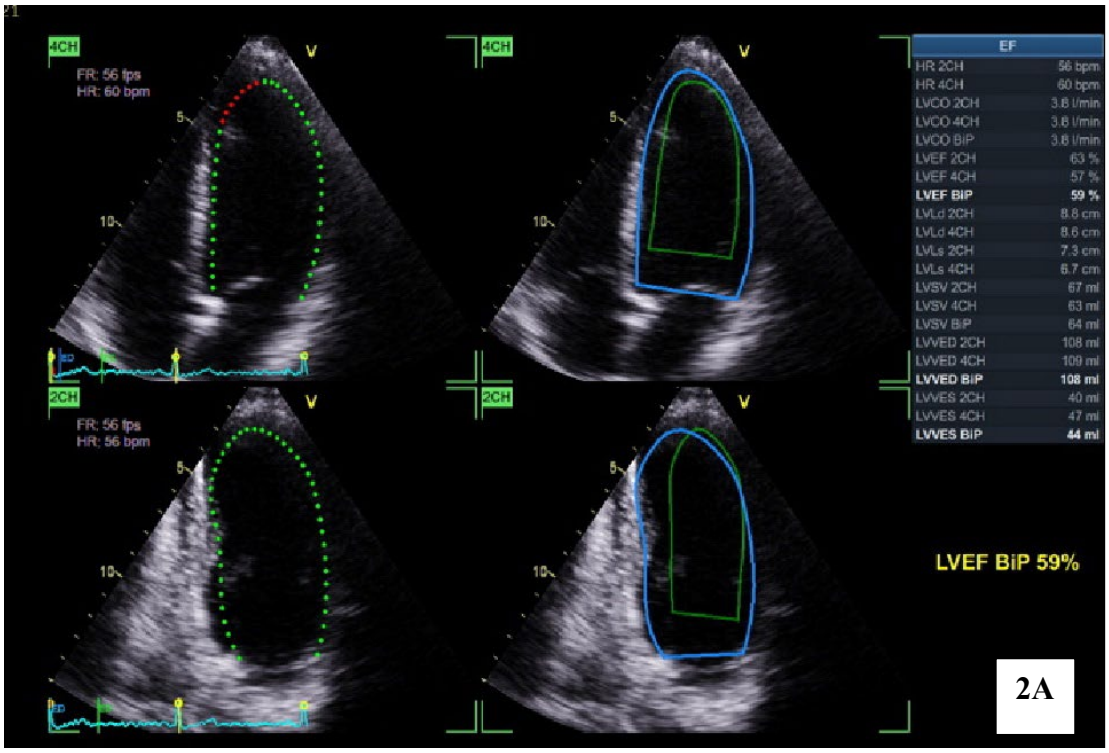
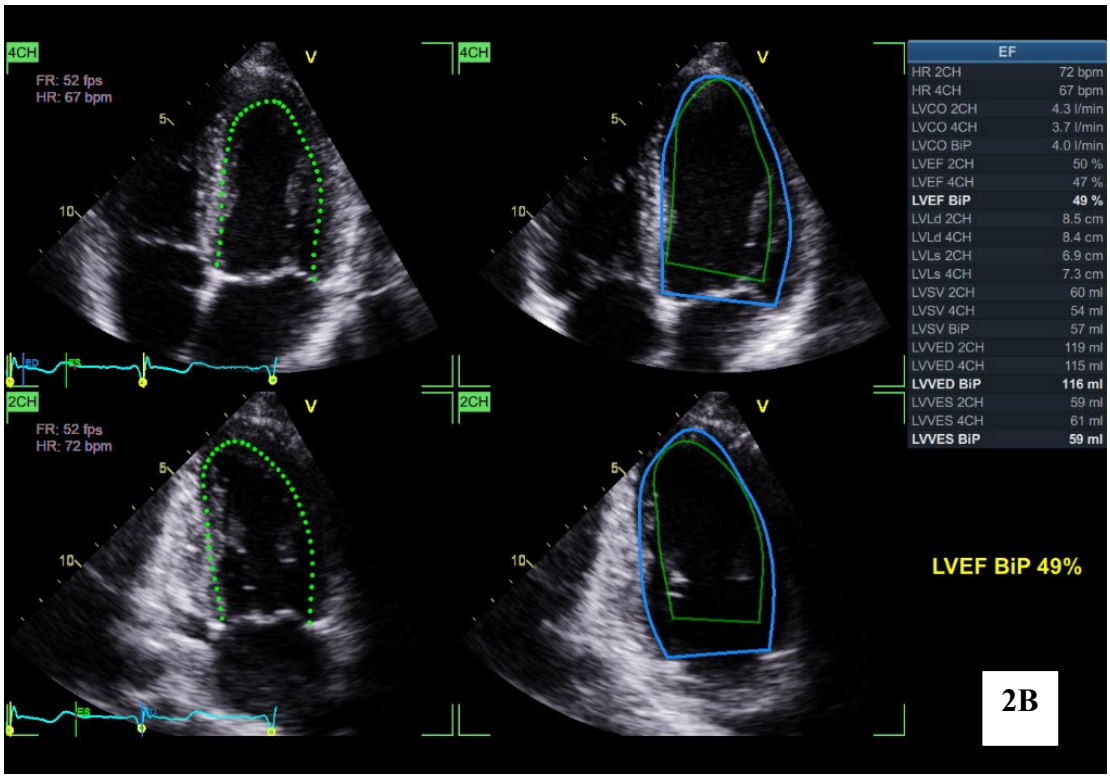
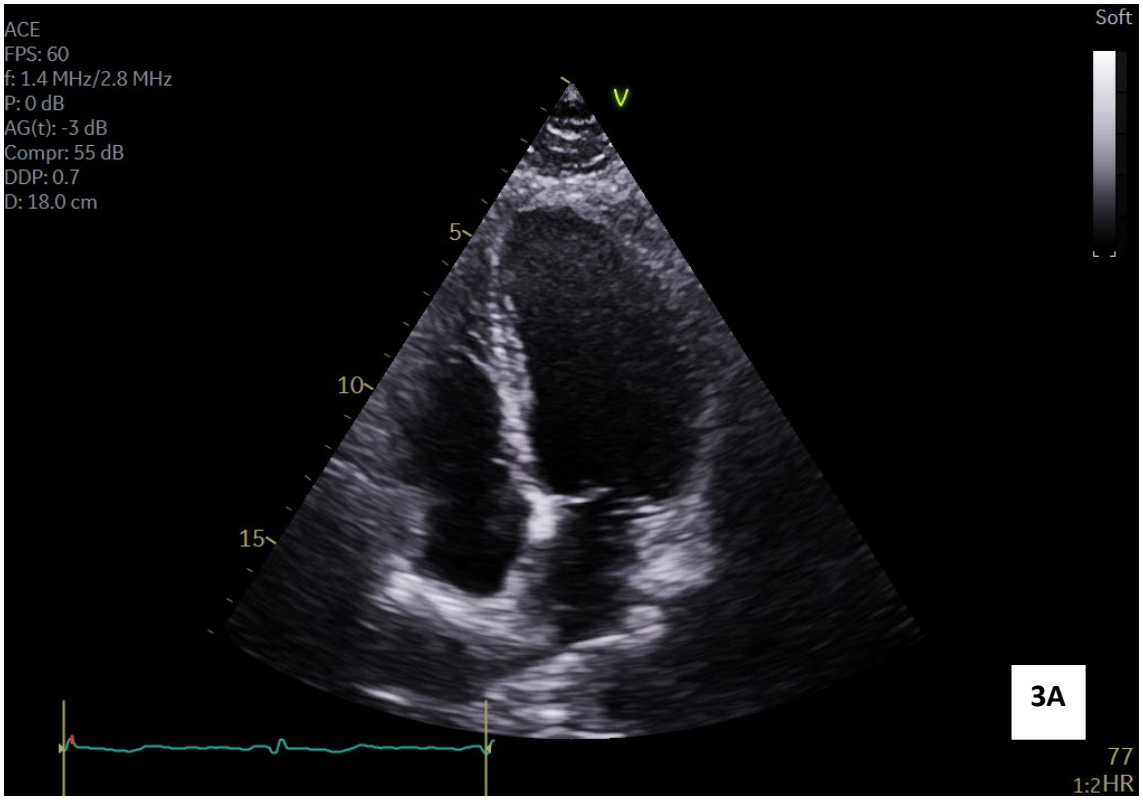


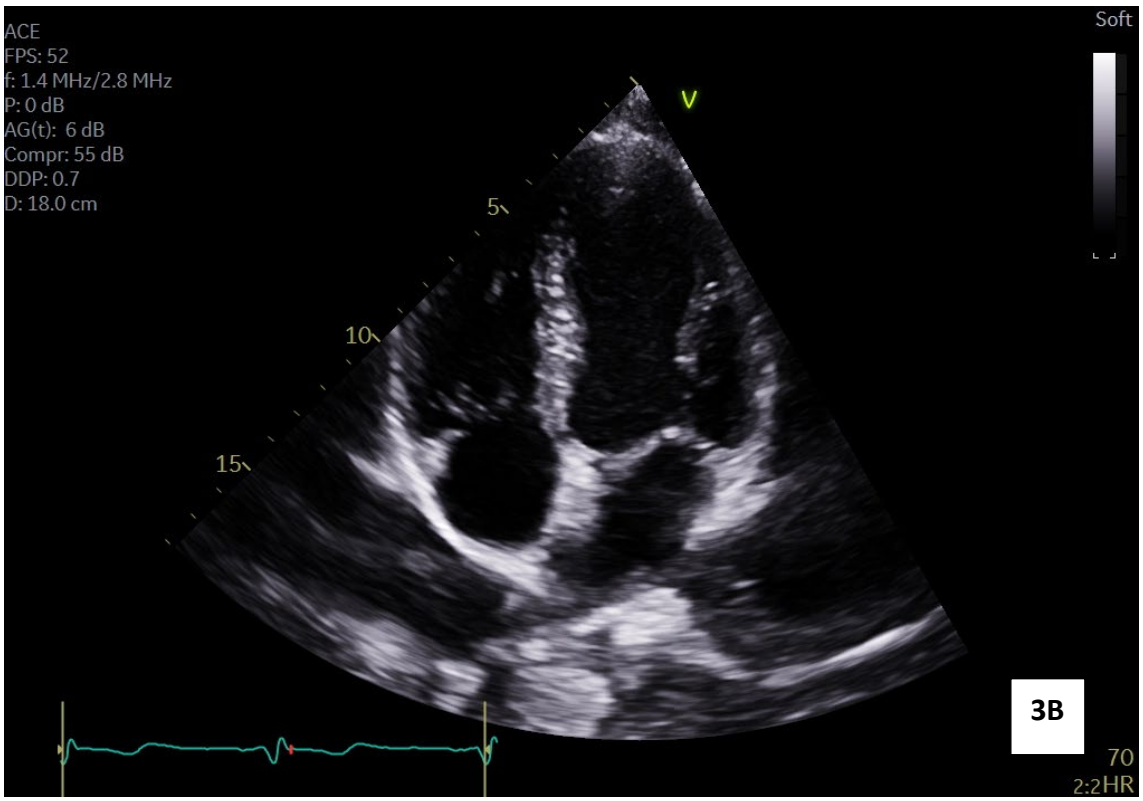
Figure 1: Box plots of LV mass (black boxes) and LV mass index (grey boxes) in male and female participants from the control and jockey groups. Boxes represent the interquartile range (IQR) with the median depicted by the central line, and whiskers extend to 1.5 x IQR. The plot illustrates the larger sex-based difference in LV mass and LV mass index within the control group and the comparatively attenuated difference between male and female jockeys.





Figures 2A & 2B: Echocardiographic images demonstrating the measurement of left ventricular volume and ejection fraction from a general population participant (A) demonstrating a left ventricular end diastolic volume of 109mL and ejection fraction of 59% and in a jockey (B) demonstrating a larger left ventricular end diastolic volume of 116mL and lower ejection fraction of 49%. The dotted green line represents the instantaneous volume, the solid blue line represents end diastolic volume, and the solid green line represents end systolic volume. Abbreviations: 2CH – two chamber view, 4CH – four chamber view, BiP – biplane, bpm – beats per minute, cm – centimetres, CO- cardiac output, EF – ejection fraction, HR – heart rate, L – litres, LV – left ventricular, ml – millilitres, VED – volume at end diastole, VES – volume at end systole.





Figures 3A and 3B: Apical 4-chamber echocardiogram images demonstrating the chambers sizes in a general population participant (A) and jockey (B). Chronic high intensity physical activity results in dilation of cardiac chambers, particularly the left ventricle (14).

2.6 Discussion

To the authors' knowledge, this is the first study to evaluate the cardiac structure and function of jockey athletes, revealing distinctive cardiac adaptations of considerable interest that may be comparable to other athlete groups. A critical finding is the presence of physiological remodeling of cardiac chambers in jockeys, particularly when indexed for BSA. Notably, jockeys exhibited larger LV and LA volumes as well as increased LV mass when indexed for BSA compared to the general population.

It is crucial to emphasize that these alterations in chamber dimensions were not associated with impaired cardiac function, as evidenced by the jockeys demonstrating normal LVEF (39). It has been demonstrated that athletes generally have LVEF values that are close to the general population (14). As this is a critical indicator of cardiac systolic function, the preservation of normal LVEF observed in the jockey group, would suggest that despite the increased LV and LA volumes, the contractile capacity of their hearts was not compromised (51).

This observed increase in LV and LA volumes as well as LV mass, when indexed for BSA, could be attributed to the chronic high-intensity physical regimens jockeys complete (42). According to the World Health Organisation's (WHO) 2020 physical activity guidelines, adults are advised to engage in 150-300 minutes of moderate intensity physical activity, 75-150 minutes of vigorous intensity physical activity or a combination of both, weekly (52). Jockey participants in this study, reported performing much more exercise than these recommendations, indicating exposure to considerably elevated levels of physical activity when compared to the general population (52). Therefore, this extensive physical activity may be a factor for the structural and functional changes in jockeys' hearts observed in this study. It has been well documented that an increase in cardiac chamber volumes, particularly LV, occurs in athletes exposed to high levels of physical activity, particularly those that train for endurance sports with both isometric and isotonic exercise, such as rowers and cyclists (12). The haemodynamic overload and increased cardiac output during exercise helps to better perfuse working muscles (14). Therefore, giving athletes a competitive edge, particularly those in endurance sports where an increase in the size of LA and right atrium (RA) volumes is seen (12). Given the increase in both LA and LV volumes observed in the jockey group, this suggests that this population, a group that has not

previously been included in athlete heart studies, fit within this demographic of endurance athletes (12, 30, 34). Whilst it was not a focus of this study, this finding necessitates further research comparing jockeys to other athlete groups to understand the full extent of these adaptations.

Additionally, cardiovascular adaptions seen in athletes do not occur as discrete, all-or-nothing changes, but instead as gradual progression that is proportional with the intensity and duration of training (14). Perry et al. (2018) examined the physiological changes in 145 Australian Football players over a 6 year period (31). They demonstrated typical cardiac remodeling that is generally seen in athletes such as increased LV mass and volume in proportion to exercise levels with RV remodeling becoming more apparent with longevity of training and increasing age (31). They also found that these changes occur over time and are dependent on individual fitness level, physical activity type and age (31). Therefore, it is plausible to suggest more time spent in the sport could lead to further cardiac adaptations (14). Thus, examining jockeys over career longevity is another possible avenue for future research.

Jockeys also displayed an increased LVEDV, although this was not statistically significant until it was indexed for BSA. This is a common observation in athlete hearts, similar to that seen in endurance athletes such as rowers and cyclists (12, 14, 30). Regular intensive training, especially endurance training, leads to increased preload due to a higher blood volume and enhanced venous return (53). This increased preload stretches the ventricular walls, promoting chamber enlargement and increased LVEDV (53). Physiological remodeling then allows the heart to pump a greater volume of blood per beat, contributing to improved overall cardiovascular efficiency during high-intensity physical activities, such as was observed in the jockey group (53).

Additionally, the sex-based comparison of LV mass and LV mass indexed to BSA among the control group, demonstrated that males had significantly higher LV mass and LV mass index compared to females, which is consistent with existing literature and likely reflects physiological differences in cardiac structure between sexes (54). This aligns with the well-established understanding that males generally have larger absolute and indexed cardiac dimensions than females, even after adjusting for body size, which is why separate reference ranges for cardiac parameters exist in clinical and athletic populations (54).

Interestingly, these same sex-based differences were not demonstrated in the jockey group. While male jockeys still exhibited higher absolute LV mass than female jockeys, the difference in LV mass index was not statistically significant. This relative convergence between sexes within the jockey group is an unusual finding for athletic populations as they follow the conforms of non-athletic groups and may be driven by a substantial increase in LV mass index in female jockeys compared to their non-athletic counterparts (54). Such findings may suggest that the physiological demands of professional horse racing may exert a more uniform effect on cardiac remodelling across sexes than typically seen in other sports (54). While this observation is not definitive, it raises a hypothesis that horse racing may illicit sex-independent cardiac adaptations. This could potentially be due to the unique combination of endurance, high-intensity effort, and occupational stressors involved in the sport (7). Further research is warranted to explore whether similar patterns are observed in larger populations to determine if this effect is unique to jockeys.

Jockeys exhibited significantly lower weight, height, and BSA compared to the control group. This may be attributed to the rigorous weight management requirements they face to meet racing standards, combined with the generally shorter stature of jockeys compared to the general population (24). These factors could potentially influence cardiac parameters as, whilst their LVEF and GLS were clinically normal as a group, it is important to note that 17% (8) of the jockeys in this study presented with reduced GLS. GLS is a sensitive marker of myocardial deformation and can reveal subclinical changes in cardiac function such as LV dysfunction and hypertrophic myocardiopathy (31, 55). It has been reported in previous studies, that no difference in GLS has been found in endurance trained rugby players, elite cyclists or rowers when compared to sedentary controls or recreational athletes (38). This was further supported in a meta-analysis in which no athlete-control differences existed for GLS (38). Although this study was not designed to delve deeply into specific causes, the finding that 7 of the 8 jockeys with reduced GLS had been riding for more than 5 years, may suggest that certain practices associated with this unique athlete group may lead to undesirable adaptations in cardiac function detected only by GLS. Especially as all age matched general population participants demonstrated GLS within a normal range. One avenue for future investigation may centre on the effects of weight-shedding practices commonly employed by jockeys to meet the rigorous weight requirements of their

profession. These practices, which include strategies such as saunas and extreme dietary restrictions, could conceivably exert detrimental effects on myocardial function, similar to those seen in patients with the early stages of AN where altered GLS while systolic function is preserved can indicate early myocardial deformation, thus warranting further exploration (19, 26).

With this in mind, chronic dehydration and calorie deficiencies are known to demonstrate reduced chamber volumes, decreased LVEF and CO (22). However, the jockey group demonstrated TBW (%) within a normal range for athletes (56-58). Matais et al (2016), examined the body composition of 200 athletes from 15 sports including basketball, track and field, soccer, wrestling, tennis, swimming and sailing, demonstrating a mean TBW (%) of ~50% (58). Similar values have been observed in marathon runners (57%) and rowers (54%) (56, 57, 59). This suggests that the jockey group in the present study were comparable to other athletes and, on the day of data collection, were not dehydrated. This could be due to the measurements being taken on a day of rest when jockeys had not been restricting their diet or sweating in preparation for race day. This also suggests that hydration did not affect preload and LVEDV values of jockeys during echocardiography data collection.

Also, given the findings for height, weight and BSA for the jockey group, it is consistent that jockeys demonstrated a lower absolute stroke volume; however, this was within normal limits and did not differ from the control group when indexed to BSA. This finding aligns with previous studies such as one conducted by De Simone et al., (1997) which observed a relationship between stroke volume and measures of body size (60). When compared to the general population it would be expected that they also have a lower absolute stroke volume (60).

To explore whether extreme differences in body size could have influenced cardiac parameters independent of athletic adaptation, an exploratory analysis was undertaken comparing the eight smallest participants from the control group to the eight largest participants in the jockey group. This comparison was not intended as a primary outcome measure but rather as a hypothesis-generating analysis to examine the potential confounding effect of body size on cardiac morphology and function. The decision to select eight participants from each group was informed by practical constraints in participant availability and the aim to contrast the most extreme phenotypes within the

sample. While we acknowledge that this subgroup comparison is underpowered for inferential conclusions, it was included to provide preliminary insight into whether extreme body size variation could partially explain the main findings. However, the combined sensitivity analysis demonstrates that the observed cardiac adaptations in jockeys are likely due to training-related changes rather than artifacts of body size differences.

2.6.1 Limitations

While the study's results are consistent with the hypothesis of cardiac remodeling in jockeys, it is important to acknowledge the limitations of the cross-sectional design. While the control group used in this study were free of cardiovascular disease, they were not required to meet any physical activity guidelines or report their exercise habits (52). Therefore, the results may not be replicated in highly active individuals from the general population, and it is unclear whether differences in activity levels were driving the differences seen in cardiac structure and function seen between groups. Also, the sample sizes for both groups were relatively small, which may have affected the statistical power to detect significant differences in some parameters. This may limit the generalisability of these results to other jockey populations. However, the small jockey sample size can be attributed to the low number of jockeys that compete Australia wide and therefore is a good representation of jockeys competing in Australia.

Additionally, while groups were matched for age, given the exploratory nature of the research and the small sample size, they were unable to be matched for sex. There is evidence that differences in sex can influence certain parameters such as chamber size (45), which may have influenced the results. However, as the data was normalised for BSA, it is unlikely to have had a notable impact.

Participants in the control group were not specifically screened for a history of anorexia nervosa (AN), and no individuals self-disclosed such a history during data collection. As the presence of AN was not a primary focus of this study, it was not included as part of the eligibility screening. However, given that AN can independently affect cardiac structure and function, this represents a potential limitation. If undetected, a history of AN in a control participant could have influenced cardiac parameters and thus impacted

group comparisons. Future research may benefit from more detailed clinical screening to ensure such factors are appropriately accounted for.

Finally, the exploratory comparison of the eight smallest controls and the eight largest jockeys was not powered to detect statistically significant differences and should be interpreted with caution. Nevertheless, it provided useful context in assessing the potential role of body size as a confounding factor and may help to inform the design of future investigations.

2.7 Conclusions

This study investigated the cardiovascular structure and function of a group that has not previously been examined. It provides insights into the cardiovascular characteristics of registered Australian jockeys compared to the general population. The observed differences in LA volume index, LVEDV index and LV mass index between the two groups may be due to the high level of exercise experienced by the jockey participants. However, as this study was cross-sectional, it cannot be determined if this relationship was causative. These findings highlight the need for further research on this specialised athlete population, especially against other elite athletes. Also, the identification of a subset of jockeys with reduced GLS raises questions about the effects of the physiological demands of racing and is a potential area for future investigation.

3 The effect of career duration on the cardiac structure and function of Australian jockeys: An observational cross-sectional study.

3.1 Chapter Overview

Chapter 2 presented the findings from comparisons of cardiac structure and function between Australian jockeys and the general population. The analysis revealed cardiac adaptations in jockeys, potentially comparable to those observed in other athlete groups. Notably, 17% of jockey participants exhibited a global longitudinal strain (GLS) value more impaired than -16% (50). This finding prompted further investigation into whether the observed cardiac changes were associated with career longevity and whether career length, weight-management practices and/or the amount of physical activity completed by jockeys contributed to the impaired GLS. These questions formed the basis for Chapter 3.

The following chapter (Chapter 3) is an observational cross-sectional study that investigates the cardiac structure and function of Australian jockeys divided into career duration (early career jockeys with < 5 years' experience and established career jockeys with ≥ 5 years' experience) while also examining differences in weight shedding practices between the two cohorts.

The manuscript presented in Chapter 3 'The effect of career duration on the cardiac structure and function of Australian jockeys: An observational cross-sectional study' has been submitted to the *Journal of Athletic Training* and is being considered for publication.

To ensure consistency of numbering and to maintain consistency across the thesis the referencing format has been updated and is presented at the end of the thesis.

3.2 Abstract

Background

To determine whether differences in cardiac structure and function, as detected by echocardiography, exist between early career jockeys (<5 years of competitive race riding experience) and established career jockeys (≥5 years) in Australia while also examining differences in weight shedding practices between the two cohorts.

Methods

Twenty early-career and twenty-two established career jockeys were recruited and underwent two-dimensional echocardiography to assess cardiac structure and function. Data pertaining to career, race riding, weekly exercise activities, and weight management practices, was also collected. All outcomes were compared using a Mann-Whitney U test.

Results

Of the forty-two jockeys, 28 (67%) were male and 14 (33%) were female. The experienced jockey group was older (p=<0.001), had more males (p=0.03), and demonstrated higher systolic (p=0.003) and diastolic (p=0.003) blood pressure. There was no difference between groups for race rides (p=0.47) and hours of physical activity per week (p=0.12), however early career jockeys spent more hours per week completing trackwork (p=<0.001).

Experienced jockeys had an increased Mitral E:A ratio (p=0.01), larger left ventricular (LV) mass (p=0.02) and LV mass index (p=0.01). Seven (17%) jockeys had impaired global longitudinal strain (GLS) >-16%, of which six were established career jockeys. All other echocardiographic parameters did not differ between the two groups.

Conclusions

Established jockeys show larger LV mass and mass index, suggesting career-related adaptations. They are also more likely to exhibit subclinical cardiac function. These findings underscore the need for further research into the impact of physical exercise and dietary weight-loss strategies on the cardiovascular health of jockeys across all career stages.

Key words

Thoroughbred horse racing, jockey, athlete's heart, echocardiography, 2D echocardiography

Key Points

- Early career jockeys complete more track work weekly than established jockeys, though both groups race equally often.
- Early career and established jockeys have similar athletic cardiac adaptations when assessed with echocardiography, but established jockeys exhibit greater LV mass, LV mass index and a lower Mitral E:A ratio.
- Both groups use similar weight management strategies.
- Jockeys far exceed the weekly recommended hours of physical activity recommended by World Health Organisation (WHO) 2020 physical activity guidelines.

3.3 Background

Thoroughbred racing is a highly demanding sport, requiring peak performance for success (61). In Australia, approximately 844 registered jockeys compete in over 19,000 races annually, across various licensing categories: amateur, apprentice, professional and jumps (2). Both male and female jockeys race together, with apprentice jockeys undergoing comprehensive training programs, where progress is primarily assessed through riding competency (4). While this approach evaluates technical skill, it overlooks physiological adaptations that may develop with increased race-riding experience. Although it is known that race riding requires a high fitness levels, its impact on cardiovascular structure and function across a jockey's career remains unclear (6).

To the authors knowledge, only one study to date has examined differences in fitness between apprentice and experienced jockeys. Legg et al. (2022) examined the riding fitness of New Zealand jockeys at different career stages (11), and found that experienced jockeys have more stable riding positions, faster reaction time, and a greater core strength than less experienced jockeys (11). While these results provide preliminary insight into the athletic performance and fitness development that occurs over a jockey's career, they do not consider the potential health-related consequences of a riding career.

Australian jockeys can ride in up to nine races a day, several days a week, year round (3). During races, they adopt a physically demanding crouched stance, by standing in the stirrups with bent knees and bodies parallel and low to the horse's (2). This result in elevated heart rates (150-190 beats/min) and oxygen uptake (42.7-57.5 ml/kg/min) (1, 8, 9, 42). Such intense exertion can lead to cardiovascular adaptations similar to those seen in other athletes, including larger cardiac chambers, and increased LV wall thickness, which impacts on both stroke volume (SV) and cardiac output (CO) (31, 45). We have previously demonstrated that jockeys have a higher LV mass index, indexed left ventricular end diastolic volume (LVEDV) and left atrial volume index (LAVi) than the general population (62). However, whether these adaptions change over a jockey's career, indicating potential cardiac adaptions related to race riding, have not previously been investigated.

In conjunction with their high fitness levels, jockeys must also maintain low body mass to meet race weight requirements (50-63 kg) (3). Common weight-loss techniques include jogging in sweat gear, hot baths, saunas, severe energy restriction and dehydration (8). This is noteworthy, as chronic food and fluid restriction, such as is seen in patients with anorexia nervosa (AN), can impair cardiac structure and function, causing reduced LV chamber size, left atrium (LA) enlargement, and decreased myocardial contractility (22). Additionally, changes in GLS may indicate impaired myocardial performance (22). The combination of these adaptations could potentially increase the risk of cardiac dysfunction, arrhythmias, and other long-term cardiovascular complications (22, 25, 26).

Whilst extensive research has focused on cardiovascular adaptions in elite athletes, the specific changes in jockeys observed over the course of their careers, remain largely unexamined. Therefore, this study aims to investigate whether early career jockeys (less than 5 years of competitive race riding experience) and established career jockeys (5 years or greater of competitive race riding experience) in Australia show differences in cardiac structure and function, as measured by echocardiography. It is hypothesised that established career jockeys will show greater cardiac adaptions, including larger cardiac chamber sizes and increased LV mass, compared to early career jockeys, with these adaptions becoming more pronounced with career longevity.

3.4 Materials and Methods

3.4.1 Participant selection

Professional Australian jockeys holding a current riding license in South Australia and Western Australia were invited to participate in the study. Recruitment focused on these states due to their comparable racing calendars and weight allocation systems. Data collection occurred on rest days to minimise the impact of acute race-related factors on study outcomes. Jockeys were included based on their availability and interest in participating in the study and if they were ≥18 years old. They were excluded if initial echocardiographic screening identified previously undocumented heart abnormalities. Participants were grouped based on race-riding experience into early career jockeys (<5 years of competitive race riding experience, n=20) and established career jockeys (>5 years of competitive race riding experience n=22). All participants provided written

informed consent. The study followed Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines and was approved by the University of South Australia Human Research Ethics Committee (project number: 204235). A completed STROBE document for this study is presented in Appendix 3.

3.4.2 Anthropometric Measurements

Immediately prior to their echocardiogram, anthropometric data were collected from all participants. Height (with shoes and socks removed) was assessed to the nearest centimetre using a portable stadiometer (Seca 213, Hamburg, Germany). Body mass, Total Body Water % (TBW (%)), fat mass, and muscle mass were measured in minimal light clothing using portable digital scales (Tanita Innerscan, Body Composition Monitor, BC-541). Body surface area (BSA) was calculated according to the formula of Dubois and Dubois as a ratio of weight (kg) to height (m) (44).

3.4.3 Echocardiography

Two-dimensional (2D) echocardiography included all standard views and measurements. These were taken in accordance with American Society of Echocardiography guidelines (45). All images were acquired from parasternal and apical windows with participants in the left lateral decubitus position and imaging was completed by one member of the research group, a qualified echocardiographer that was blinded to jockey group allocation. Commercially available echocardiography systems with either a M55c XD clear Matrix or 4Vc phased array transducer were used for image acquisition (GE E95, Vivid IQ, General Electric, Horten, Norway). Images were recorded digitally in cine-loop format. Advanced image analysis was performed using commercially available analysis software (TOMTEC Image Arena and Autostrain analysis, Unterschleissheim, Germany). As part of a standard echocardiogram, specific images for diastolic function including mitral valve inflow and tissue doppler imaging (TDI) were taken. LA volume was measured from both the apical 4 and 2 chamber views with care to open the LA to its major axis. LV volumes and ejection fraction were measured using the Simpson's rule of discs from the apical 4 and 2 chamber views where the major axis of the LV was found. SV was calculated from the LV outflow tract

diameter and velocity time integral. LV GLS was calculated from LV focused images from the apical 4-chamber, 2-chamber, and long-axis views. Studies were deemed inadequate for GLS analysis if 2 or more LV segments could not be visualized. A region of interest was automatically applied by the software to the endocardial and epicardial borders, with tracking adjusted to cover the entire myocardium. GLS was calculated as the change in the length of line of the region of interest across all apical view from diastole to systole. To determine intra-rater reliability, the echocardiographic data from 20 jockeys was analysed twice with a minimum duration of one month between measurements. Intra-operator variability was determined using intraclass correlation coefficients (ICC) with an ICC < 0.5 indicating poor agreement, 0.5 to 0.7 moderate agreement and >0.8 good agreement (46).

3.4.4 Lifestyle Questionnaire

Prior to their echocardiogram, all participants completed a lifestyle questionnaire, modified from a previous study (20). Completion was carried out independently under the supervision of the lead researcher to avoid responses of other athletes impacting the results. Information collected included years of jockeying, race riding statistics, eating habits, weekly exercise (in addition to race riding), smoking habits, alcohol consumption, medications and drug use, and weight management practices. Information on previous medical history such as known cardiovascular issues or anorexia was also obtained. Qualtrics software (XM, Provo, UT) was used to design and distribute the questionnaire and to record responses. The modified questionnaire was piloted by two people from the research group who were not involved with the design and two jockeys to ensure clarity and ease of use before full implementation. The lifestyle questionnaire is presented in Appendix 1.

3.4.5 Statistical Analysis

All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) (v26, IBM, USA), with statistical significance set at $p<0.05$. Any participants without full data sets were excluded from analysis. Parametric data are presented as

mean ± standard deviation (SD) and 95% confidence interval (CI), while nonparametric data are reported as median (interquartile range), unless otherwise stated.

To evaluate differences between jockeys with less than 5 years' experience and jockeys with 5 or more years of experience of riding in races, a Mann-Whitney U test was used, as the data did not follow a normal distribution. Cohen's *d* effect size [ES] was calculated for all reported parameters except for the dichotomous parameter of GLS more impaired then -16% (50, 63). The effect size was determined by dividing the difference between the means by the pooled SD. Effect sizes were calculated (= z /sqrt(N)) and interpreted as follows: <0.20 = *trivial*, 0.20 – 0.49 = *small*, 0.50 – 0.79 = *moderate*, and >0.79 = *large* (47).

For dichotomous data, specifically the GLS more impaired than -16% parameter, a chi-square test was used to assess differences between groups. The effect size for dichotomous data was calculated using Cramer's V, with interpretations as: 0.00 to 0.09 = negligible, 0.10 to 0.19 = weak, 0.20 to 0.39 = moderate, 0.40 to 0.59 = relatively strong, 0.60 to 0.79 = strong association and 0.80 and above = very strong association (64).

Sample size calculations were based on the normal value of GLS (-20 ± 3%) and an expected 10% clinically relevant change in GLS between the two groups. According to Diggle et al. (2013), a sample size of 13 participants per group was sufficient to achieve 80% power at a significance level (α) of 0.05 to detect a clinically significant difference (49). To account for potential dropouts, a target sample of at least 20 participants per group was set.

3.5 Results

3.5.1 Anthropometry measures

Forty-two Australian jockeys (age range: 19-51 years of age) consented to take part in this study (28 males [67%] and 14 [33%] females). All females participating in this study were below the age of 35. Jockeys were from two Australian states, South Australia (n=23), and Western Australia (n=19). Experienced jockeys were older (p=<0.001), more predominantly male (p=0.03) and demonstrated higher blood pressures, both systolic (p=0.003) and diastolic (p=0.003). Early career jockeys (n=20)

had considerably less years of race riding experience when compared to the experienced jockey group (n=22) (p=<0.001). There was no difference between jockey groups for race rides per week (p=0.47) and hours of physical activity per week which included aerobic activity not related to race riding or trackwork (p=0.12). However, early career jockeys spent more hours per week riding trackwork when compared to the established jockey group (p=<0.001). There were no jockeys that listed any known previous cardiovascular issues as part of this study. Descriptive data for the jockey groups are presented in Table 5.

Table 5. Descriptive, anthropometric, race riding experience workload and exercise metrics for early career jockeys and established career jockeys

Parameter	**Early Career Jockeys (n = 20)**	**Established Jockeys (n = 22)**	***U* =**	**p-Value**	**Effect Size**	**Effect Size Descriptor**
Age (years)	23±3.0 CI: 21.9 to 24.5	41±7.5 CI: 37.6 to 44.3	3.0	**<0.001***	d=0.84	*Large*
Sex (males %)	10.0 (50%)	18.0 (82%)	150.0	**0.03***	d=0.33	*Small*
Weight (kg)	54.3±4.0 CI: 52.6 to 56.0	55.8±3.2 CI: 54.4 to 57.3	156.5	0.11	d=0.25	*Small*
Height (m)	1.6±0.07 CI: 1.6 to 1.7	1.64±0.1 CI: 1.6 to 1.7	218.5	0.97	d=0.01	*Trivial*
BSA (m^2)	1.5±0.1 CI: 1.4 to 1.5	1.5±0.1 CI: 1.5 to 1.6	174.0	0.25	d=0.17	*Trivial*
TBW (%)	60.8±5.0 CI: 58.5 to 63.2	58.4±2.9 CI: 57.1 to 59.7	166.0	0.25	d=0.18	*Trivial*

Fat Mass	14.2±8.0 CI: 10.4 to 18.1	12.3±5.8 CI: 9.8 to 14.9	216.0	0.92	d=0.02	*Trivial*
Muscle Mass	44.3±5.9 CI: 41.5 to 47.0	46.7±4.2 CI: 44.8 to 48.6	164.0	0.24	d=0.18	*Trivial*
Systolic BP (mmHg)	122±9.6 CI: 118.4 to 127.4	135.0±15.8 CI: 127.7 to 141.7	101.5	**0.003***	d=0.46	*Small*
Diastolic BP (mmHg)	76±8.0 CI: 72.5 to 80.1	84.0±7.4 CI: 81.0 to 87.6	103.0	**0.003***	d=0.46	*Small*
HR (bpm)	66.3±14.0 CI: 60.0 to 72.6	62.0±11.0 CI: 56.7 to 66.4	180.0	0.31	d=0.16	*Trivial*
Race Riding Experience (years)	1.2±0.69 CI: 0.8 to 1.5	8.7±1.9 CI: 7.8 to 9.5	0.0	**<0.001***	d=0.90	*Large*
Race rides/week	10.0±5.0 CI: 7.6 to 12.5	11.2±4.5 CI:9.2 to 13.2	173.0	0.47	d=0.11	*Trivial*

Track work hours/week	14.6±1.2 CI: 14.0 to 15.2	10.5±3.6 CI: 8.9 to 12.0	71.0	**<0.001***	d=0.62	*Large*
Exercise hours/week^	21.4±6.2 CI: 18.4 to 24.4	18.3±7.5 CI: 15.0 to 21.6	149.5	0.12	d=0.24	*Small*

*p<0.05 compared with early career jockey group (bold text indicates significant p-values): U - Mann-Witney U test value, CI – Confidence Interval, BSA – body surface area, TBW (%) – Total Body Water %, BP – blood pressure, HR – heart rate, bpm – beats per minute, ^not including time spent race riding or completing trackwork, d=Cohen's d effect size.

3.5.2 Echocardiography measures

Experienced jockeys had increased Mitral E:A ratio (p=0.01) and Mitral Valve E (p=0.01) when compared to the early career group. They also demonstrated larger LV mass (p=0.02) and LV mass index (p=0.01). All other parameters were not statistically different. The mean (mean ± SD) echocardiographic data for jockey groups are presented in Table 6. While figure 4 presents box plots of the distribution of these key cardiac parameters in early career versus established career jockeys.

Despite similar GLS between both groups, there were seven (17%) jockeys who had an impaired GLS (>-16%) (50). Of these seven, six were established jockeys and one was an apprentice. However, this difference between groups was not significant (p=0.06).

Table 6. Echocardiographic data for jockeys for early career jockeys and established jockeys

Parameter	**Early Career Jockeys (n = 20)**	**Established Jockeys (n = 22)**	***U* =**	**p-Value**	**Effect Size**	**Effect Size Descriptor**
LVEDV (mL)	117.5±16.6 CI:109.7 to 125.3	122.9±20.9 CI: 113.6 to 132.1	174.5	0.25	d=0.18	*Trivial*
LVEDV index (mL/m^2)	78.9±10.1 CI: 74.2 to 83.7	80.4±12.5 CI: 74.9 to 86.0	192.0	0.48	d=0.11	*Trivial*
LVEF (%)	62.4±4.6 CI: 60.2 to 64.5	60.7±5.0 CI: 58.5 to 62.9	176.5	0.27	d=0.17	*Trivial*
Mitral E:A ratio	2.4±0.7 CI: 2.1 to 2.7	2.0±1.0 CI: 1.5 to 2.4	109.0	**0.005***	d=0.43	*Small*
Mitral E	0.9±0.2	0.8±0.1	122.5	**0.01***	d=0.38	Small
Mitral A	0.4±0.1	0.4±0.1	183.5	0.36	d=0.14	Trivial
E:E' (avg)	6.3±1.4 CI:5.6 to 6.9	6.2±1.7 CI: 5.4 to 7.0	197.5	0.74	d=0.05	*Trivial*

E' Velocity (cm/s)	13.1±2.3	11.3±2.4	124.5	0.02*	d=0.37	Small
Stroke Volume (mL)	64.7±13.7 CI: 58.3 to 71.1	64.0±10.8 CI: 59.2 to 68.8	200.0	0.61	d=0.08	*Trivial*
Stroke Volume index (mL/m^2)	43.3±8.0 CI: 39.6 to 47.0	42±7.2 CI: 38.8 to 45.2	192.0	0.48	d=0.11	*Trivial*
Cardiac Output (L/min)	4.2±1.0 CI: 3.8 to 4.7	3.9±0.9 CI: 3.5 to 4.3	184.0	0.37	d=0.14	*Trivial*
LA vol (biplane, mL)	48.1±8.9 CI: 43.8 to 52.4	54.1±11.4 CI: 49.0 o 59.3	141.0	0.11	d=0.26	*Small*
LA vol index (mL/m^2)	32.2±5.3 CI: 29.7 to 34.7	35.4±7.2 CI: 32.2 to 38.7	143.0	0.13	d=0.24	*Small*
LV mass (g)	106.6±26.0 CI: 94.5 to 118.8	126.4±24.7 CI: 115.5 to 137.3	124.0	**0.02***	d=0.37	*Small*

LV mass index (g/m^2)	71.6±16.9 CI: 63.7 to 79.5	82.1±14.2 CI: 76.3 to 88.9	118.0	**0.01***	d=0.40	*Small*
IVS Thickness (cm)	0.74±0.14	0.79±0.12	164.5	0.15	d=0.22	Small
PW thickness (cm)	0.78±0.14	0.84±0.07	175.5	0.24	d=0.17	Trivial
RWT	0.35±0.07	0.36±0.06	206.0	0.72	d=0.05	Trivial
GLS	-20.2±2.6 CI: -21.4 to -19.0	-19.1±2.9 CI: -20.3 to -17.8	192	0.48	d=0.18	*Trivial*
GLS >-16%	1.0(5%)	6.0 (27%)	Chi-squared = 2.7	0.06	V=0.25	*Moderate*
Diastolic Function	Normal = 19 (95%) Grade 1 = 1 (5%)	Normal = 17 (77%) Grade 1 = 5 (23%)	181.0	0.11	d=0.15	Trivial

*p<0.05 compared with early career group (bold text indicates significant p-values): U - Mann-Witney U test value, CI – Confidence Interval, LVEDV – left ventricular end diastolic volume, LVESV – left ventricular end systolic volume, LVEF – left ventricular ejection fraction, Mitral E:A ratio – ratio of peak velocity blood flow from left ventricular relaxation in early diastole to peak velocity flow in late diastole, E:E' – mitral inflow early diastolic velocity to tissue Doppler mitral annular early diastolic velocity ratio, LA vol – left atrial volume, LV – left ventricular, IVS – interventricular septum, PW – posterior wall, RWT – relative wall thickness. Values are presented as mean ± standard deviation or number and (percentage), GLS – Global Longitudinal Strain, GLS >-16% - GLS more impaired than -16%, d=Cohen's d effect size, V=Cramer's V effect size.

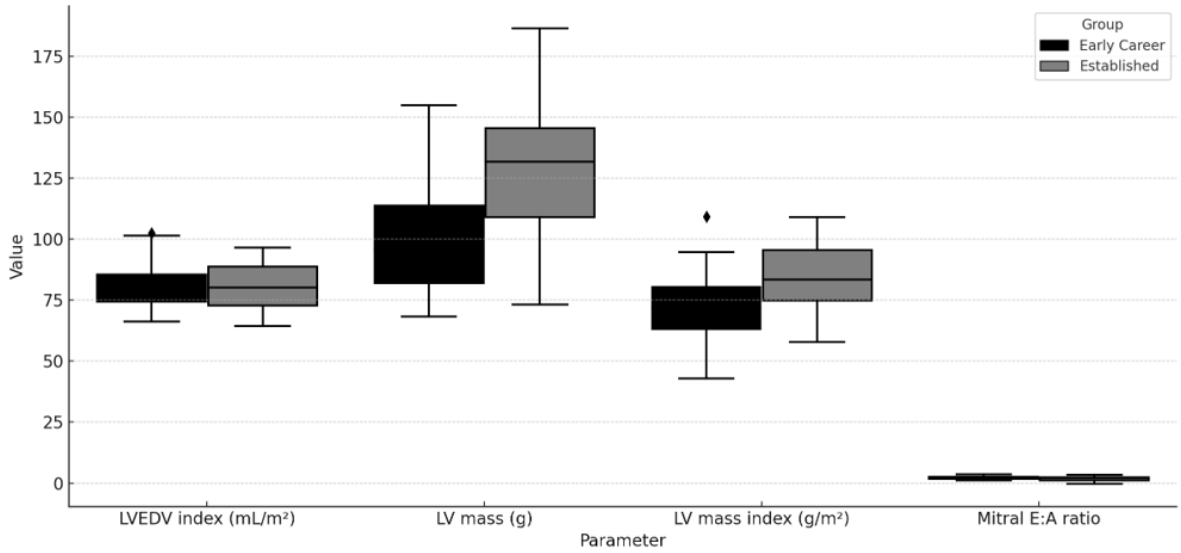


Figure 4: Box plots of LVEDV, LV mass, LV mass index and Mitral E:A ratio in early career (black boxes) and established career (grey boxes) participants. Boxes represent the interquartile range (IQR) with the median depicted by the central line, and whiskers extend to 1.5 x IQR. Overlaid dots represent individual observations. The plot illustrates established jockeys exhibits slightly greater LVEDV index and LV mass whereas mitral E:A ratio appears slightly lower.

3.5.3 Weight management strategies

As part of the lifestyle questionnaire, jockeys were asked to indicate strategies that they use to manage their weight to reach race riding weights. There were no statistically significant differences demonstrated between the two groups although a majority of all participants reported actively participating in food restriction (81%), and hot salt baths (83%) to cut weight before competition. The results are displayed in table 7 and beneath this, figure 5 demonstrates data distribution as bar charts.

Table 7. Weight management strategies used for early career jockeys and established jockeys

Practice	**Early Career Jockeys (n=20)**	**Established Career Jockeys (n=22)**	**p-Value**
Smoking	2.0 (10%)	3.0 (14%)	0.72
Sauna	9.0 (45%)	10.0 (46%)	0.9
Sweat suit	8.0 (40%)	12.0 (55%)	0.43
Hot salt bath	10.0 (50%)	15.0 (68%)	0.32
Water diet	6.0 (30%)	5.0 (23%)	0.53
Food restriction	14.0 (70%)	20.0 (91%)	0.15
Vomiting	1.0 (5%)	0.0 (0%)	0.28
Diuretics	1.0 (5%)	1.0 (5%)	0.35
Drug use	1.0 (5%)	0.0 (0%)	1.00
Laxatives	2.0 (10%)	1.0 (5%)	0.47

*$p<0.05$ compared with early career group (bold text indicates significant p-values).

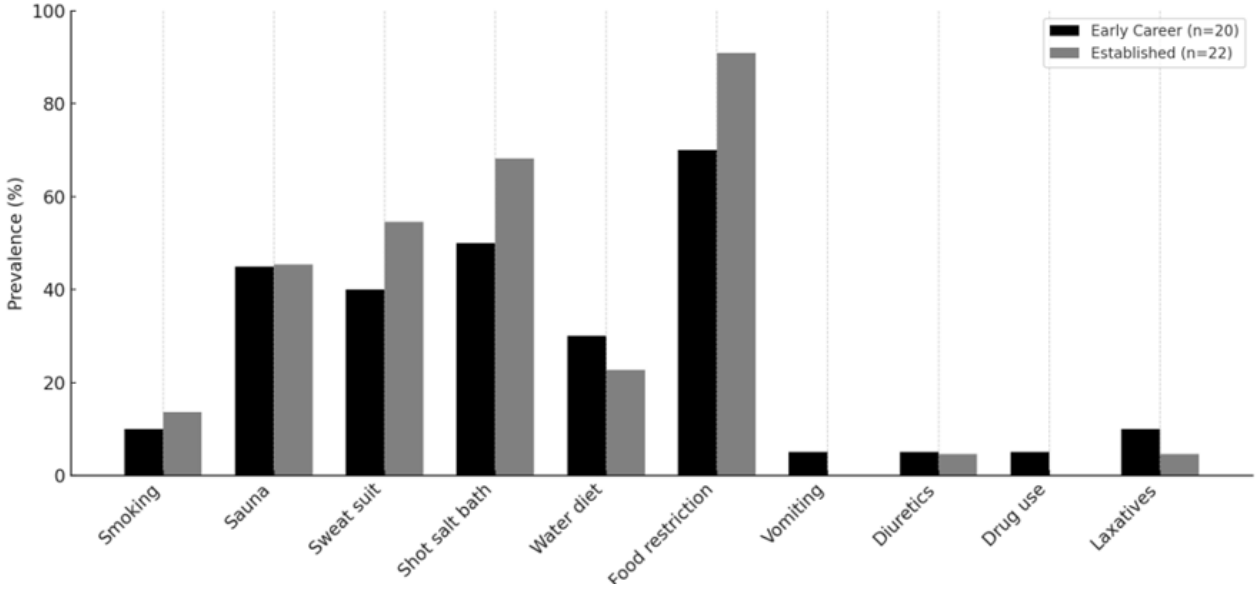


Figure 5: Prevalence of weight-management practices reported by early-career (black, n = 20) and established (grey, n = 22) jockeys. Bars represent the percentage of riders in each career stage who use each method. Food restriction, sauna use, sweat-suit training, and hot salt baths were the most commonly adopted strategies, with established jockeys generally reporting higher usage rates. Less frequent practices—including smoking, vomiting, diuretics, drug use, and laxatives—showed low prevalence in both groups.

Comparison was also made between jockeys with normal GLS and GLS more impaired than -16% values, again no statistically significant differences were demonstrated. The results are displayed in table 3.4 and beneath this, figure 3.3 demonstrates data distribution as bar charts.

Table 8. Weight management strategies used for jockeys with normal GLS and jockeys with abnormal GLS

Practice	**Jockeys With Normal GLS (n=35)**	**Jockeys With Abnormal GLS (n=7)**	**p-Value**
Smoking	4.0 (11%)	1.0 (14%)	0.40
Sauna	15.0 (44%)	4.0 (57%)	0.95
Sweat suit	17.0 (50%)	2.0 (29%)	0.35
Hot salt bath	22.0 (64%)	4.0 (57%)	0.11
Food restriction	29.0 (83%)	4.0 (57%)	0.42

*p<0.05 compared with early career group (bold text indicates significant p-values).

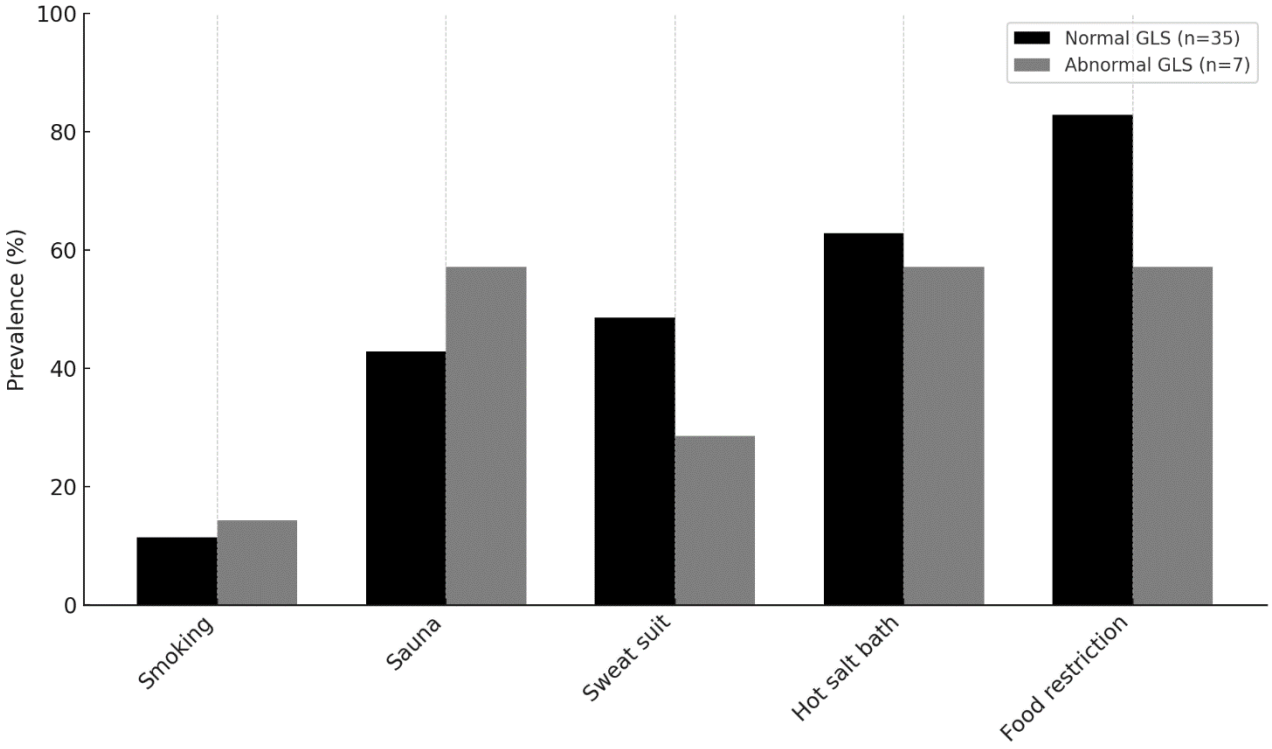


Figure 6: Prevalence of common weight-management practices in jockeys stratified by global longitudinal strain (GLS) status. Bars depict the proportion of riders with normal GLS (black, n = 35) and abnormal GLS (grey, n = 7) who report using each method. Hot salt baths and food restriction were the most frequently employed strategies in both groups, whereas practices such as smoking and sweat-suit training showed lower overall usage. No practice exhibited a statistically significant difference between GLS groups (all $p > 0.05$).

3.6 Discussion

This study explored the cardiac structure and function of early versus established career jockeys to examine the potential adaptions associated with professional race riding. Findings revealed that established jockeys exhibited significantly greater LV mass, LV mass index and mitral E:A ratio when compared to early career jockeys. Although there were no statistically significant differences in GLS or other echocardiographic parameters between the groups, established jockeys demonstrated a trend towards having a higher frequency of impaired GLS than early career jockeys.

3.6.1 Age, sex riding experience and anthropometric measures

Early career jockeys had significantly less race riding experience than their established counterparts. This disparity highlights the differing level of experience between career stages, which is not surprising, as it aligns with the expected career progression from apprentice to senior jockey (3). The sex distribution also differed between groups. While early career jockeys had equal male to female representation (50%), established jockeys were predominantly male (83%). This finding aligns with a study investigating New Zealand jockey careers, which also reported higher attrition rates among female jockeys transitioning from apprentice to senior ranks (3). The reasons for this trend remain unclear but may relate to the challenges of maintaining consistently low body weight and a higher incidence of race fall injuries in females (3).

Notably, established jockeys demonstrated significantly higher resting systolic and diastolic blood pressure than their early career counterparts ($p = 0.003$ for both). While regular endurance training is typically associated with lower resting blood pressure, even with increasing age (65), age-related vascular stiffening may contribute to a gradual increase in systolic blood pressure (SBP) despite preserved fitness (66). In veteran endurance athletes, systolic pressure can rise mildly with age due to progressive loss of arterial compliance, even when diastolic pressure remains stable or slightly declines (65, 66).

However, in the jockey cohort, the elevated blood pressure observed in established athletes may also reflect the cumulative effects of occupational stress, chronic weight cycling, and repeated dehydration, which have been associated with endothelial

dysfunction and increased arterial stiffness (67). Although vascular measures such as pulse wave velocity (PWV) and flow-mediated dilation (FMD) were not assessed in this study, the findings suggest that cardiovascular monitoring of jockeys should not be limited to cardiac structure and function alone but should also include peripheral and central vascular health in future studies.

3.6.2 Exercise and workload metrics

Although the number of race rides per week did not significantly differ between groups, early career jockeys spent significantly more time undertaking track work. This suggests that early career jockeys dedicate more time to track training, likely as part of their development and training process to improve riding skills and break into the industry. Similar findings have been seen in New Zealand, where higher trackwork workloads among early career jockeys was thought to enhance fitness and riding skill development (3).

It was also demonstrated that all jockeys within this study, regardless of career stage, significantly exceeded the WHO 2020 physical activity guidelines (52). This high level of physical activity is much higher than one would expect to see amongst the general population (52). Our study comparing the cardiac structure and function of Australian jockeys to the general population suggests that jockeys may experience unique cardiovascular adaptations likely due to the intense, specialized physical demands of race riding (62). This aligns with physiological changes seen in other endurance athletes such as rowers and cyclists (32).

3.6.3 Echocardiographic findings

Most echocardiographic parameters were similar between the two jockey groups, which may indicate that cardiac adaptations in the sport of thoroughbred racing occurs at an early stage in their career due to the high intensity of physical activity required. However, established jockeys exhibited significantly lower mitral E:A ratios compared to early career jockeys. The mitral E:A ratio, a key indicator of diastolic function, was significantly lower in established jockeys. The observed reduction in the mitral E:A ratio suggests a possible shift toward greater reliance on atrial contraction during

ventricular filling, which may reflect subclinical early diastolic dysfunction, or perhaps be a function of older age in the established jockey cohort (29). Changes in myocardial compliance and relaxation in relation to age are well-documented contributors to such shifts in diastolic function (68).

While a reduced mitral E:A ratio is commonly observed in endurance athletes, it is generally considered a physiological adaptation when seen alongside preserved systolic function (32). However, there is evidence to suggest long-term endurance training, such as that associated with jockeying, could possibly lead to mild myocardial fibrosis with increasing age, further reducing ventricular compliance and thus causing the reduction of mitral E:A ratio observed in established jockeys (29, 32). This remains a contentious topic and therefore warrants further investigation (68).

Established jockeys also demonstrated higher LV mass and LV mass index in comparison to their early career counterparts. This may reflect chronic cardiac adaptations to the intense physical demands of race riding (32). Increased LV mass is a hallmark of physiological hypertrophy in endurance athletes, facilitating enhanced SV and CO to meet elevated demands during exercise (34). For jockeys, this adaptation may potentially be driven by the repetitive cardiac volume loading associated with race riding, track work and additional physical activity. Comparable increases in LV mass have been documented in endurance athletes from other disciplines, such as cyclists and rowers, who engage in similarly strenuous endurance activities (31, 51). However, the combined impact of prolonged exertion, alongside potential confounding factors such as weight loss strategies, may increase the risk of maladaptive cardiac remodeling over time (38).

Although GLS data did not differ significantly between groups, impaired GLS (> -16%) was observed in seven jockeys, six of whom were in the established cohort. This represents 17% of the entire jockey sample. While a previous study reported a much lower 2% prevalence of impaired GLS among Olympic athletes in comparison tour study (69). This was across a broad range of sports, including including both skill-based and power-based disciplines such as equestrian, fencing, sailing, golfing, wrestling, weightlifting, soccer, basketball, and tennis (69). This figure may not be an appropriate comparator for a population engaged in year-round high-volume competition. More recent research has demonstrated that impaired or borderline GLS is not uncommon in

endurance-trained athletes, particularly those exposed to high cumulative training loads over time. For example, Claessen et al. (2024) reported a prevalence of mildly reduced GLS (more impaired 16%) in up to 10% of elite endurance athletes (cyclists and triathletes), despite normal ejection fraction and clinical health status (15).

Furthermore, several studies have failed to find significant GLS differences between athletes and matched controls, especially when accounting for the type of sport and training intensity (32). This variability underscores the complexity of interpreting GLS in highly trained populations and suggests that mildly reduced GLS may reflect either physiological adaptation, early maladaptation, or a combination of both depending on context, training load, and recovery.

The higher prevalence of impaired GLS among established jockeys may reflect the combined impact of rising age, workload, and weight management practices on myocardial function. Impaired GLS can correlate with myocardial fibrosis, raising the possibly that the physiological demands of race riding combined with chronic weight shedding strategies, could impact the cardiac structure and function of jockeys over time (68). While these findings suggest a potential trend toward greater subclinical myocardial impairment with career longevity, the cross-sectional design of the study and small number of cases in this study precludes definitive conclusions. Further research is warranted to explore long-term implications of these adaptions with a greater participant number.

3.6.4 Weight management practices

Both early career and established jockeys employed a variety of weight management strategies, with no significant differences between groups. The most common practice was food restriction, reported by 70% of early career and 91% of established jockeys. Other prevalent methods included salt baths (50% early career vs. 68% established), sauna use (45% early career vs. 46% established) and sweat suits (40% early career vs. 55% established). Extreme methods, such as vomiting, diuretics, and laxatives, were infrequent across both groups. This consistency suggests that weight management practices remain similar across career stages, regardless of the level of experience. These findings align with prior research on nutritional practices, where jockeys reported engaging in one or more weight loss strategies to ensure they met the strict weight

requirements of their profession (16, 21). It was found that the reason for this was reflective of the majority of jockeys obtaining weight management advice from peers or ex jockeys, perpetuating an entrenched ‘in-house’ cultural norm for this athlete group and is a possible reason why our study found similarities between career length groups (16, 21).

Notably, 57% (4 out of 7) of jockeys with impaired GLS reported using saunas as a rapid weight loss strategy. While all jockeys in this study had normal TBW (%) when tested, they were all on days of rest from competition and had not been sweating prior to their scan. Research indicates that dehydration from such practices can reduce plasma and blood volume (18). This diminishes the amount of blood returning to the heart, subsequently decreasing SV and CO (18). As a result, the heart must work harder to maintain adequate blood flow, which can lead to impaired cardiovascular function, muscle blood flow, and thermoregulation (23). Dehydration also increases blood viscosity, raising peripheral arterial resistance leading to increased blood pressure (BP) and in turn, potentially heightening the risk of ischemic heart disease or stroke (18). Previous studies investigating these effects did not evaluate the effect of long-term weight loss strategies used by athletes, and this represents an area for future research.

Chronic food restriction, another common strategy used by all jockeys in this study, may also contribute to subtle cardiovascular changes. Studies on AN have shown that prolonged energy deprivation can impair myocardial contractility, decrease LV mass, reduce SV, and alter systolic and diastolic function (22). These are all features typically attributed to severe malnutrition and catabolic states (22).

In contrast, jockeys in this study exhibited increased LV volumes and stroke volumes, more consistent with physiological adaptations seen in endurance athletes. This disparity highlights that, although jockeys may share some elements of chronic undernutrition with individuals affected by AN, their overall cardiac phenotype more closely resembles that of trained athletes than of individuals experiencing pathological cardiac atrophy (70). Indeed, endurance athletes demonstrate enlarged LV cavity dimensions, enhanced stroke volume, and increased LV mass as adaptive responses to sustained high cardiac workloads (70).

Nevertheless, the interplaybetween repetitive dehydration, acute weight cycling and chronic food restriction may introduce cardiovascular stressors in jockeys that are not

typically present in other athletic populations. While not directly mirroring the changes seen in AN, these factors could still influence cardiac performance over time. The observed prevalence of impaired GLS in this cohort raises the hypothesis that cumulative physiological and occupational stressors may eventually shift cardiac adaptations toward a maladaptive trajectory and therefore contribute to the higher incidence of abnormal GLS observed in established jockeys. These findings highlight the need for further investigation into the long-term cardiovascular impacts of acute on chronic weight management practices employed by jockeys.

3.6.5 Limitations

While a number of the study's results are consistent with the hypothesis of cardiac remodeling in jockey career longevity, it is important to acknowledge the limitations of the cross-sectional design. Forty-two jockeys participated in our study and given the relatively small sample sizes, and the dichotomous nature, comparisons across different career stages made it difficult to see more granular changes. Also, the small sample sizes may limit the generalisability of these results; however, the jockey sample size could be attributed to the low number of jockeys that compete Australia wide and therefore is a good representation of jockeys competing in Australia. Additionally, the cross-sectional design of the study limits the ability to draw causal relationships or determine the long-term effects of jockey training on cardiac health. Further research with larger sample sizes and longitudinal designs is warranted to validate and expand on these findings. Additionally, while groups were matched for age, given the exploratory nature of the research and the small sample size, they were unable to be matched for sex. There is evidence that differences in sex can influence certain parameters such as chamber size (45), which may have influenced the results. However, as the data was normalised for BSA, it is unlikely to have had a notable impact.

Due to equipment constraints and the limited resources available in this Masters study, lean muscle mass was measured using Tanita bioelectrical impedance analysis. While this method provided a general estimate, it was determined that indexing echocardiographic values to lean muscle mass would not yield scientifically reliable results in this context. As such, body surface area (BSA), a well-established and widely accepted indexing method in cardiac research, was used instead. Although this approach

strengthens the validity of the findings, future studies with access to more advanced methods of body composition assessment may consider indexing to lean muscle mass to provide additional insights into the relationship between cardiac structure and musculoskeletal profile in jockeys.

3.7 Conclusion

This study investigated the cardiovascular structure and function of early career and experienced jockeys. The findings from this study reveal some differences between early and established career jockeys.

These insights emphasize the importance of further investigation of dietary and weight-loss strategies for jockeys at different stages of their careers and the effect of the high amounts of physical exercise this athlete group completes each week. Future research should explore the long-term health implications of these findings on cardiac structure and function and develop strategies to optimize the development, performance, and well-being of jockeys both throughout and after their career.

4 General Discussion and Conclusions

4.1 Chapter Overview

Chapter 4 summarises the results of Chapter 2 and Chapter 3, contextualising the research outcomes within the themes of summary of key findings, future research recommendations, and the overarching strengths and limitations of the thesis.

4.2 Summary of key findings

This thesis provides the first comprehensive evaluation of the cardiac structure and function of jockey athletes. Several adaptations were revealed during Chapter 2 and Chapter 3 that are possibly shaped by the intense demands of competitive race riding.

Chapter 2 determined that jockeys exhibit larger indexed left ventricular (LV) and left atrial (LA) volumes when compared to the general population, as well as increased LV mass, when indexed for body surface area (BSA). These changes are consistent with physiological adaptations observed in other endurance athletes, suggesting that jockeys, despite not being traditionally classified as an athletic population, experience similar cardiac remodeling due to the high-intensity physical demands of their profession (12).

Importantly, while these structural changes were evident, they were not associated with impaired systolic function, as indicated by normal left ventricular ejection fraction (LVEF) values. However, 17% of the jockey cohort within this study demonstrated a global longitudinal strain (GLS) more impaired than -16%, which we know is a sensitive marker of subclinical myocardial dysfunction (50). While not statistically different from apprentice jockeys, this prevalence was particularly pronounced among established jockeys with more than five years of competitive race riding experience (Chapter 3), highlighting the potential cumulative impact of prolonged exposure to the physiological demands of race riding. This finding contrasts with results from Olympic endurance athletes such as rugby players, cyclists, and rowers, where GLS values are typically comparable to sedentary or recreationally active controls (69).

In Chapter 3, although several cardiac parameters were not statistically significant, career stage appeared to influence a few notable cardiac adaptations. Established jockeys demonstrated a greater LV mass, LV mass index, and a trend toward more frequent impairment in GLS compared to their early-career counterparts. Additionally, established jockeys exhibited a lower mitral E:A ratio, indicative of a shift toward greater reliance on atrial contraction during diastolic filling (28). This finding may reflect subclinical early diastolic dysfunction or age-related changes, compounded by the chronic extreme physical demands of the sport (29).

The chapter also highlighted the substantial physical workload undertaken by jockeys, with all participants significantly exceeding the World Health Organisation (WHO) recommended physical activity levels (52). Both jockey groups also rode in a similar number of races per week. This possibly accounts for the similarities in most echocardiography parameters, indicating that jockeys may experience changes to their cardiac structure and function early on in their career, most likely due to the high physiological workload and is similar to that seen in other athlete groups (31).

Furthermore, this chapter demonstrated that early-career jockeys spent more time completing track work riding (training horses to race) when compared to their established counterparts. This suggests that early career jockeys dedicate more time to training, likely as part of their development and training process to improve riding skills and break into the industry. Similar findings have been seen in New Zealand, where higher trackwork workloads among early career jockeys was thought to enhance fitness and riding skill development (3).

Chapter 3 also determined that weight management practices were widely used by jockeys across all career stages. The most common techniques used by both early and established career jockeys were food restriction and dehydration techniques, such as hot salt baths and sauna use. This is most likely to help jockeys meet strict weight requirements for racing and is potentially the reason for the similarity in anthropometric values such as weight and BSA across career length. While weight management practices are effective for maintaining competition weight, there is reason to believe that these practices may contribute to myocardial stress. Chronic strategies like sauna use and calorie restriction have been linked to impaired GLS (22). This finding raises concerns about the long-term cardiovascular effects of these practices on jockeys, especially given previous findings in individuals with Anorexia Nervosa (AN), who exhibit similar GLS impairments due to extreme chronic caloric restriction (22). The parallels between these groups warrant further investigation, as altered GLS in AN population has been associated with early myocardial deformation, despite preserved systolic function (22).

Although weight-loss practices in jockeys often involve dehydration, this thesis indicates that on non-race days, Total Body Water (TBW (%)) in jockeys remains within the normal range for athletes (58). For example, TBW values for jockeys in

Chapter 3 (~50%) are comparable to those of marathon runners (57%) and rowers (54%) (58). This suggests that hydration status on rest days may not significantly influence left ventricular end diastolic volume (LVEDV) or preload during echocardiographic assessments but may differ on days just prior to or on race days when jockeys are trying to lose weight immediately prior to competition.

Although the results for this thesis did not identify dehydration in jockeys on days of rest, the use of dehydration as a weight-loss strategy among jockeys is a critical area of concern, given its demonstrated effects on stroke volume (SV), LVEDV, and GLS (23). The potential for these practices to contribute to long-term cardiovascular dysfunction certainly warrants further investigation. Understanding the impact of chronic dehydration cycles on the cardiac structure and function of jockeys could inform safer weight management practices within the profession and may improve cardiovascular health in this unique athletic population.

In summary, the findings advance the understanding of cardiac adaptations in jockeys and underscore the complex interplay between physical demands, career longevity, and weight management in shaping their unique cardiovascular profile. They also highlight the importance of targeted research and health monitoring in this unique athlete group. It can also contribute to the development of informed practices for professions working with jockeys that can aim at maintaining both peak athletic performance and cardiovascular health over time.

4.3 Strengths and limitations

This study has several strengths, including its novel focus on a unique athletic population, the use of comprehensive echocardiographic analysis, and a robust methodological approach to indexing cardiac parameters for BSA. These strengths allow for a nuanced understanding of the structural and functional cardiac adaptations in jockeys, a group that has traditionally been underrepresented in athlete health research. Additionally, the inclusion of jockeys at different career stages provides valuable insights into how career longevity and physical demands of racing may influence cardiovascular adaptations. Despite a relatively small sample size, the representation of jockeys in this study aligns with the limited pool of professional jockeys in Australia, offering a reasonable reflection of this specific athletic cohort.

However, several limitations must be acknowledged. The cross-sectional design precludes the establishment of causal relationships between participant groups and limits the ability to assess longitudinal changes in cardiac structure and function over career span. Also, the control group in chapter 2 was not required to meet physical activity guidelines or report exercise habits, which introduces potential variability in baseline activity levels, which has a direct influence on cardiovascular structure and function (12). This limitation raises questions about whether differences observed between jockeys and the general population might be influenced by disparities in physical activity rather than the unique demands of race riding. The inability to match groups for sex, despite age-matching efforts, presents another limitation, as sex differences can influence parameters such as chamber size (50). While normalizing data for BSA mitigates some of these concerns, a more balanced sex representation would strengthen the conclusions made in chapter 2. Additionally, in chapter 3, comparisons across career stages were limited by the dichotomous nature of the analysis and relatively small subgroup sizes, making it difficult to detect more granular changes in cardiac adaptations over career longevity.

To address these limitations, future research would benefit from larger, longitudinal studies that explore cardiac remodeling in jockeys over time and compare them with highly active individuals in other athlete populations. Incorporating a matched control group with comparable physical activity levels would also help isolate the specific impact of race riding and help to determine if this is the main reason for cardiac adaptions seen in jockeys throughout this thesis. Expanding sample sizes and including a more diverse cohort in terms of sex and geographic representation, perhaps even internationally, would further enhance the robustness and generalizability of the findings. These steps will provide a more comprehensive understanding of the long-term cardiovascular adaptations and potential risks associated with professional race riding.

4.4 Future research

Future research should aim to build upon the findings of this thesis by addressing its limitations and exploring new avenues to deepen our understanding of cardiac adaptations in jockeys. A key area for future investigation is the longitudinal assessment

of cardiac remodeling in jockeys with larger sample sizes. While this thesis provided insights into structural and functional differences in jockeys' hearts compared to the general population and across career stages, the cross-sectional design limited the ability to assess causal relationships or track the progression of these changes over time. Longitudinal studies would enable researchers to identify the trajectory of cardiac adaptations, examine potential thresholds for physiological versus pathological changes, and better understand the long-term cardiovascular implications of professional race riding. Little research on cardiac adaptations over career length has been undertaken on other athlete populations in this area, but a study conducted on Australian Football League (AFL) players, demonstrated that remodeling of the heart in this population of athletes is a continuous spectrum of changes that occurs in response to increased physical activity where increases in both LV and right ventricular (RV) size and LV mass were observed (31).

In addition to understanding how cardiac remodelling evolves during a jockey's active career, it is critical to investigate the long-term cardiovascular outcomes in retired jockeys. To date, there is a paucity of research exploring whether the adaptations observed during competitive years persist, regress, or progress into maladaptive or pathological states after retirement. Future studies should incorporate longitudinal follow-up into retirement, ideally with serial imaging assessments and comprehensive clinical evaluation. Particular attention should be paid to the incidence of cardiovascular events (e.g., arrhythmias, myocardial infarction, or heart failure) and the use of cardiac medications such as beta-blockers, Angiotensin-Converting Enzyme (ACE) inhibitors, or anti-arrhythmic drugs in this population. These outcomes would help clarify whether impaired GLS or altered diastolic parameters observed in current jockeys have clinical significance over time, and whether early echocardiographic markers can predict future cardiovascular risk.

Another important direction is the inclusion of larger sample sizes and a more diverse participant pool. While this study's sample size was reflective of the relatively small population of professional jockeys in Australia, larger-scale studies would enhance statistical power and generalizability. Expanding the geographic scope to include jockeys from other states in Australia and even other countries could also provide a more comprehensive understanding of the global jockey population and where they sit in comparison to other athlete groups. It has been shown that professional jockeys in

New Zealand and Ireland have similar workloads to Australian jockeys, and ride in 1-8 races a day, in 2-4 race days a week, and would therefore be a good population to include in further research to increase sample sizes (5). Conversely, jockeys in the United Kingdom and America reported having a higher workload of up to 7 days of racing per week (5). Comparing these jockeys against Australian jockeys may also offer additional insight into how training load may impact cardiovascular function in this population.

Additionally, matching control groups for physical activity levels would allow researchers to isolate the specific effects of race riding and its associated demands from general exercise-related cardiac adaptations. Similarly, a comparison with other athlete groups with similar physical activity levels but with no required weight management practices such as rowers and cyclists may provide some insight as to what might be driving the adaptations seen in this thesis. As we know, changes in athletes hearts occurs but with 17% of jockey participants demonstrating an impaired GLS of <-16% which is not comparable to other elite athlete groups such as Olympians (2% of participants had an impaired GLS), these studies may help to further clarify whether observed differences in cardiac structure and function are unique to jockeys or are shared by other endurance athletes (69).

It is well known that sex influences cardiac parameters such as chamber size in elite athlete populations with male athletes demonstrating more pronounced cardiac adaptions to endurance training (71). For this reason, we must take this into account when considering the results of this thesis, and future studies should strive for more balanced representation across sexes or conduct sex-specific comparison analyses on jockeys to uncover potential differences in cardiac adaptations.

Also, examining the impact of weight management practices, particularly chronic dehydration, and calorie restriction, on myocardial structure and function is another critical area for exploration. This thesis's findings suggested a possible link between such practices and impaired GLS, particularly among established jockeys but as cohort numbers were low, this was not statistically significant. Similar findings have been seen in athletes competing in combat sports where weight cutting is common practice to meet competition weight restrictions such as mixed martial arts and boxing (18). There have however been no studies investigating the effects of weight cutting practices over time.

Longitudinal research by assessing early career jockeys prior to commencing racing and then following them over their career annually or biannually could help determine whether these strategies have cumulative effects on cardiac health, and whether they contribute to maladaptive remodeling or subclinical dysfunction over the duration of a career.

Further research exploring the role of specific training regimens, race-day stressors, and workload distribution across career stages could provide insights into how different aspects of jockeying influence cardiac remodeling. For example, the observed differences in mitral E:A ratios and LV mass between early and established jockeys suggest that career longevity may play a role in shaping certain cardiac adaptations. Longitudinal studies that track athletes from their early to late career stages could help clarify how these changes evolve and whether they predispose jockeys to certain cardiovascular risks.

The higher prevalence of impaired GLS noted in both Chapter 2 and Chapter 3 when compared to other athlete groups raises questions about its clinical significance and underlying causes. Future research should investigate whether impaired GLS in jockeys is associated with subtle myocardial fibrosis or other pathological changes that may not manifest in standard measures of systolic function. Advanced imaging techniques, such as cardiac magnetic resonance imaging, could provide more detailed assessments of myocardial structure and composition, offering valuable insights, when compared to echocardiography, into the potential risks associated with long-term exposure to the unique demands of professional race riding.

Finally, investigations into the cardiovascular structure and function of retired jockeys would help determine whether the changes noted while competing persist after the cessation of racing or lead to long-term pathology. By assessing retired jockeys, particularly those who rode for an extensive number of years, researchers could explore whether early subclinical finings like reduced GLS are linked to future cardiovascular disease, increase cardiac medication use, or hospitalisations. Along with whether the physiological stressors of a racing career predispose individuals to adverse cardiac events later in life. These insights would be invaluable for informing health surveillance strategies and targeted interventions aimed at safeguarding jockey's cardiovascular health across their lifespan.

4.5 Conclusion

In conclusion, this thesis has provided novel insights into the cardiac adaptations of jockeys, a unique athlete population that has been largely underrepresented in athlete cardiology research. The findings demonstrate potential physiological remodeling of cardiac structure and function in jockeys, with increased left ventricular (LV) and left atrial (LA) volumes and LV mass, when indexed for body surface area (BSA). These changes appear consistent with adaptations observed in other endurance athletes, such as rowers and cyclists where prolonged exposure to combined pressure and volume overload drives cardiac enlargement and eccentric hypertrophy (12). Suggesting that the high-intensity and specialized physical demands of race riding could drive these cardiovascular adaptations as is seen in other athlete groups as mentioned above. Importantly, despite these observed structural changes, the preservation of left ventricular ejection fraction (LVEF) across the jockey cohort indicates that systolic function remains intact, further supporting the view that these adaptations are predominantly physiological.

However, the findings also reveal important nuances. Differences in cardiac parameters between early and established career jockeys, including trends toward greater LV mass and reduced mitral E:A ratios in the established group, highlight the influence of career longevity and cumulative workload on cardiac remodelling. Such trends have been similarly reported in studies of elite endurance athletes, where career duration, age, and training intensity are associated with progressive myocardial remodelling, including increased wall thickness and altered diastolic function (38, 53). While these changes are often benign, distinguishing physiological from early maladaptive or pathological changes remains a complex and evolving area of athlete hearts (34).

The observation that 17% of jockeys in this cohort exhibited impaired GLS further complicates this picture. Though GLS was preserved on average within this cohort, the proportion with abnormal values was higher than some reported in mixed-sport athlete cohorts (15, 69). Yet, this must be interpreted with caution. Recent research has shown that mildly reduced GLS can occur in otherwise healthy endurance athletes, particularly those with high cumulative training loads, without necessarily indicating pathological

dysfunction (15). Factors such as chronic weight cycling, dehydration, and occupational stress contribute to unique myocardial loading conditions in jockeys which makes them unique when compared to more commonly studied athletic groups.

The complexity of interpreting myocardial strain and wall thickness in elite athlete populations is increasingly recognized, particularly in sports with combined isometric and dynamic demands, such as rowing, wrestling, or gymnastics (12). These sports demonstrate similar heterogeneity in cardiac responses, including disproportionate increases in wall thickness relative to chamber size, and emerging evidence of subclinical fibrosis in select subgroups (29). These findings underscore the importance of considering sport-specific physiological stressors, sex differences, and cumulative exposure when assessing cardiac health in athletes (12).

The strengths of this body of work include its focus on a highly specialized and understudied athlete group and its comprehensive echocardiographic assessment. However, several limitations must be considered. The cross-sectional design precludes casual inference and limits assessment of progression of the cohort over time. The relatively small sample size while reflective of the overall national jockey population, may reduce statistical power. Additionally, the control group was not matched for physical activity level, which could confound the interpretation of between-group differences.

Future research should aim to address these limitations through longitudinal designs, larger and more diverse participants, and multimodal imaging techniques such as cardiac MRI to assess for fibrosis, cardiac chamber volume, and relative wall thickness. The use of 24-hour ambulatory blood pressure monitoring and vascular measures such as arterial stiffness and central blood pressure would also provide valuable insight into afterload and vascular influences on cardiac remodeling. Further, comparisons with other athlete groups of similar physiological and occupational profiles, such as combat sport athletes, lightweight rowers, or wrestlers, may help contextualize these findings and refine our understanding of cardiac remodeling in high-risk athletic populations.

Ultimately, this thesis contributes to a growing body of evidence highlighting the complex interplay between sport-specific demands, cardiovascular adaptation, and potential long-term health implications in elite athletes. It establishes an important foundation for understanding the cardiac health of jockeys and opens critical avenues

for further investigation—particularly around the effects of weight management strategies, workload, and career duration. By advancing this research, the racing industry and clinical community can better inform evidence-based guidelines to optimize health, performance, and safety in this highly specialized athlete population.

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Appendix 1

Appendix 1: Lifestyle questionnaire distributed to each jockey participant.

Block 1

Project Title: The cardiac structure and function of registered Australian jockeys.
A Questionnaire relating to your general health, exercise, nutrition, and general weight control practices.

Ethics application: 204235
Primary researcher: Angela Farley
Supervisors: Dr Rebecca Perry, Dr Hunter Bennett & Prof Roger Eston

Thank you for participating in this study.

You have been invited to take part in this research project, because you are either a registered jockey or part of the general population participant group and you have provided your contact details to the investigators.

This questionnaire is anonymous. The record of your responses does not contain any specific identifying information about you. However, an identifying number has been assigned to you to allow us to link your responses with your ultrasound scan. This identification number will not be stored with your responses and the results from your ultrasound. It is managed in a separate database and will only be updated to indicate whether you did (or did not) complete this questionnaire.

This is an internet-based survey. Every effort will be made to ensure that responses are confidential, however the researcher cannot guarantee the confidentiality or anonymity of material transferred by email or the internet.

You may close the questionnaire down at any time if you wish to withdraw your participation at any stage. Your responses will not be used as part of the study if this occurs.

The primary researcher will have shown you the Participant Information Sheet which tells you about the research project. It explains the processes involved to help you decide if you want to take part in the research. Please read this information carefully. Ask questions about anything that you don't understand or want to know more about.

Participation in this research is voluntary. If you do not wish to take part, you don't have to. If you decide you want to take part in the research project, please click the blue

button below.

You have provided your consent to participate in the study already by signing the consent form, but by clicking this button, you are again confirming that you:
- Understand what you have read
- Consent to take part in the survey component of the research project
- Consent to the use of your information as described.

Please click on the blue button if you wish to commence the questionnaire.

Unique participant code

Please enter the unique code you have been given by the research team below:

Height in cms

Weight in kgs

Total body water percentage from scales

Fat mass percentage from scales

Muscle/fat free percentage from scales

Resting systolic blood pressure reading

Resting diastolic blood pressure reading

Gender

- ○ Male
- ○ Female
- ○ Non-binary / third gender
- ○ Prefer not to say

Age

Block 2

Which Australian state is your main place of residency? (Select one answer)

- ○ South Australia
- ○ Victoria
- ○ Western Australia
- ○ Other

Within your state of residency, where are you mainly based? (Select one answer)

- ○ City
- ○ Provincial
- ○ Country

Are you currently taking any medication? (Select one answer)

- ○ Yes
- ○ No
- ○ Unsure

Please list these medications. (Type your response below)

Have you previously had any test/s on your heart? (Select one answer)

- ○ Yes
- ○ No
- ○ Unsure

Please list these tests – if unsure of the name of the test, please describe what you had. (Type your response below)

Have you previously experienced any problems with your heart? (Select one answer)

- ○ Yes
- ○ No
- ○ Unsure

Please list what these problems with your heart are/were. (Type your response below)

Have you been diagnosed with any of the following illnesses by a doctor or health professional? Please select the appropriate response for each illness.

	Yes	No	Maybe	Not Applicable
Cardiovascular Disease	☐	☐	☐	☐
Diabetes	☐	☐	☐	☐
High Blood Pressure	☐	☐	☐	☐
Low Blood Pressure	☐	☐	☐	☐
Osteoporosis	☐	☐	☐	☐
Kidney problems	☐	☐	☐	☐
Dental problems	☐	☐	☐	☐
Gastric problems	☐	☐	☐	☐
Reproductive problems	☐	☐	☐	☐
Menstrual problems	☐	☐	☐	☐

Are you a smoker? (Select one answer)

- ○ Yes
- ○ Yes, but only occasionally
- ○ No, but I am an ex-smoker
- ○ No

How many cigarettes would you smoke per day (on average)? (Type your response below)

Do you drink alcohol? (Select one answer)

- Yes, 5-7 times per week
- Yes, 1-4 times per week
- Yes, but only occasionally
- No

Approximately how many standard drinks would you consume per week (on average)? (Type your response below)

On average how much exercise are you doing per week that is not considered part of your work? (Select one answer)

- None
- 1-3 hours
- 4-7 hours
- 8-12 hours
- 13-16 hours
- 17-20 hours
- 21+ hours

What type/s of exercise are you doing? (Type your response below)

Have you ever tried to lose weight? (Select one answer)

- Yes
- No
- Unsure
- Prefer not to say

Approximately how many kilograms did you lose? (Type your response below)

How long did it take you to lose the weight? (Type your response below)

What methods did you follow to lose the weight? (Type your response below)

Are you currently trying to lose weight? (Select one answer)

- Yes
- No
- Unsure

How long have you been doing this? (Type your response below)

How many kilograms have you lost? (Type your response below)

How many kilograms are you hoping to lose? (Type your response below)

List the practices that you are undertaking to do this. For example, calorie counting, exercising etc. (Type your response below)

Are you a jockey?

- Yes, I am in the jockey participant group.
- No, I am in the general population participant group

Which Australian state is your main place of competition? (Select one answer)

- South Australia
- Victoria

- ○ New South Wales
- ○ Other

Are you an apprentice jockey? (Select one answer)

- ○ Yes
- ○ No

What year of your apprenticeship are you in? (Select one answer)

- ○ 1st year
- ○ 2nd year
- ○ 3rd year
- ○ 4th year
- ○ 5th +

Have you had any rides in official races? (Select one answer)

- ○ Yes
- ○ No

How many years have you been competing as a registered jockey? (Select one answer)

- ○ 0-3 years
- ○ 4-6 years
- ○ 7-9 years
- ○ 10-12 years
- ○ 13-15 years
- ○ 16-18 years
- ○ 19-21 years
- ○ 22+ years

On average, approximately how often are you riding track work per week? (Select one answer)

- ○ 0 days

- ○ 1 day
- ○ 2-3 days
- ○ 4-5 days
- ○ 6 days
- ○ Every day of the week

On average, approximately how often are you riding in trials per week? (Select one answer)

- ○ 0 days
- ○ 1 day
- ○ 2-3 days
- ○ 4-5 days
- ○ 6 days
- ○ Every day of the week

On average, approximately how often are you riding at race meetings per week? (Select one answer)

- ○ 0 days
- ○ 1 day
- ○ 2-3 days
- ○ 4-5 days
- ○ 6 days
- ○ Every day of the week

On average, how many rides would you have at each individual race meeting? (Select one answer)

- ○ 0 races
- ○ 1 race
- ○ 2-3 races
- ○ 4-5 races
- ○ 6-7 races
- ○ 8+ races

On average, approximately how many race rides would you have per week? (Select one answer)

- ○ 0 races
- ○ 1 race
- ○ 2-5 races
- ○ 6-9 races
- ○ 10-14 races
- ○ 15+ races

Are you currently NOT riding in races? (Select one answer)

- ○ Yes
- ○ No

What is the reason that you are not currently riding? (Type your response below)

How long have you not ridden in races for? (Type your response below)

When do you plan to resume race riding? (Type your response below)

What is your usual race riding weight? (Type your response in kilograms below)

How do you feel about your current weight? (Select one answer)

- ○ Great
- ○ Good
- ○ Just all right
- ○ Frustrated
- ○ No feelings

Do you currently have difficulty managing your racing weight? (Select one answer)

- ○ Yes
- ○ No

At any time in the past, have you had any difficulty managing your racing weight? (Select one answer)

- ○ Yes
- ○ No

What is the lowest race riding weight you have ridden at? (Type your response below)

How often do you ride at this lowest riding weight? (Type your response below)

Do you have to lose weight to 'make weight' for your race rides? (Select one answer)

- ○ Always
- ○ Occasionally
- ○ Rarely
- ○ Never

Prior to a normal race day, what do you do in relation to eating and drinking? (Type your response below)

- ○ Eat and drink normally
- ○ Eat and drink more
- ○ Eat and drink less

On average how much weight do you cut for a race day? (Select one answer)

- ○ None
- ○ 0-0.5 kilograms
- ○ 0.6-1 kilograms
- ○ 1.5-2 kilograms
- ○ 2.5-3 kilograms
- ○ 3.5-4 kilograms

- 4.5+ kilograms

When do you start to cut weight for a race day? (Select one answer)

- The morning prior to weigh in
- 1 day prior to weigh in
- 2-3 days prior to weigh in
- 4-6 days prior to weigh in
- 1 week prior to weigh in
- 2 weeks prior to weigh in
- 3+ weeks prior to weigh in
- I don't cut weight

When would you consider you drop the most weight before your race? (Select one answer)

- 2 weeks prior
- 1 week prior
- 5 days prior
- 3 days prior
- 48 hours prior
- 24 hours prior
- 12 hours prior
- 6 hours prior
- 2 hours prior
- Just prior to weighing out before race

Approximately how often are you required to cut weight per week to compete? (Select one answer)

- 0 days
- 1 day
- 2-3 days
- 4-5 days
- 6 days
- Each day of the week

During a normal race meeting, what do you do in relation to eating and drinking? (Select one answer)

- Eat and drink normally
- Eat food between races
- Drink fluids between races
- Don't eat or drink during the meeting

Following race day, how much weight do you put back on before your next race day or next weight cut? (Select one answer)

- None
- 0-0.5 kilograms
- 1-3 kilograms
- 4-5 kilograms
- 6+ kilograms

Following race day, what do you do in relation to eating and drinking? (Type your response below)

- Eat and drink normally
- Eat and drink more
- Eat and drink less

Do you have a professional nutritionist/dietitian that advises you for your weight cutting? (Select one answer)

- Yes, I regularly work with a professional nutritionist/dietitian.
- I have worked with a professional nutritionist/dietitian a few times.
- I have worked with a professional nutritionist/dietitian once.
- No, I have never worked with a professional nutritionist/dietitian.

Where do you receive your nutrition advice? (Select all that apply)

- [ ] Registered dietitian/nutritionist
- [ ] Personal trainer/fitness instructor
- [ ] Fellow jockeys
- [ ] Family/friends

- [ ] Magazines/books
- [ ] Doctor
- [ ] Social Media
- [ ] Professional Organizations
- [ ] Other

Would you be interested in speaking to a nutritionist/dietitian that is a specialist in jockey nutrition programming? (Select one answer)

- ○ Yes
- ○ Maybe
- ○ No

What concerns do you have regarding your sport nutrition needs or weight cutting regimen as a registered jockey? (Type your response below)

Please consider the following weight management methods. Select the most appropriate responses(s) for when you use each weight-loss method. You can select more than one response. Also, indicate in the last two columns why you use the method by selecting the appropriate boxes. If you answer never to any of the method, then please leave the last two columns blank.

Example answer: if you use food restriction leading up to and on race day to lose weight rapidly, you would select 'day before race day' and 'one race day' and you would then select the 'lose weight rapidly' column.

	When do you use it?				Reason for use		
	Never	Between Race Days	Day Before Race Day Only	On Race Day	Maintain Constant Weight	Lose Weight Rapidly	Not Applicable
Food restriction	○	○	○	○	○	○	○
Fluid restriction	○	○	○	○	○	○	○
Sauna	○	○	○	○	○	○	○
Diuretics	○	○	○	○	○	○	○

	When do you use it?				Reason for use		
	Never	Between Race Days	Day Before Race Day Only	On Race Day	Maintain Constant Weight	Lose Weight Rapidly	Not Applicable
Other weight loss medication	○	○	○	○	○	○	○
Drugs	○	○	○	○	○	○	○
Plastic sweat suit wearing	○	○	○	○	○	○	○
Exercise as jockey for weight control (track work, stable work)	○	○	○	○	○	○	○
Other exercise for weight control (e.g., jogging, gym, or other sport)	○	○	○	○	○	○	○
Water-diet (water loading followed by urination)	○	○	○	○	○	○	○
Vomiting	○	○	○	○	○	○	○
Laxatives	○	○	○	○	○	○	○
Hot Salt Bath	○	○	○	○	○	○	○
Smoking cigarettes	○	○	○	○	○	○	○
Other	○	○	○	○	○	○	○

The following question refers to the physical effects, you may experience when rapidly loosing weight. Please indicate how often you experience the effects by circling the appropriate response. Also, indicate how often you experience the effects by selecting either the minor or serious columns. If you never experience any of the following, then leave the last two columns blank.

Example answer: if you often experience muscular cramps and feel that this is serious, you would select 'often', and also select the last column 'serious'.

	Frequency			Severity		
	Never	Sometimes	Always	Minor	Severe	Not Applicable
Muscular Cramps	○	○	○	○	○	○
Fever	○	○	○	○	○	○
Nausea	○	○	○	○	○	○
Dizziness	○	○	○	○	○	○
Upset Stomach	○	○	○	○	○	○
Stomach Cramps	○	○	○	○	○	○
Poor Circulation	○	○	○	○	○	○
Fatigue	○	○	○	○	○	○
Visual Distortion/Impairment	○	○	○	○	○	○
Joint Pain	○	○	○	○	○	○
Heart Palpitations	○	○	○	○	○	○
Chest Pain	○	○	○	○	○	○
Dehydration	○	○	○	○	○	○
Fainting	○	○	○	○	○	○
Other	○	○	○	○	○	○

The following table refers to physiological effects that you may experience when weight cutting. Please indicate how often you experience the effects by selecting the appropriate response. Also indicate the severity of your experience by selecting either minor or serious in the last columns. If you never experience any of the following the leave the last two columns.

Example answer: if you often have persistent thoughts about thirst, and feel that it is serious, you would select 'often' and also select the last column 'serious'.

	Frequency			Severity		
	Never	Sometimes	Always	Minor	Serious	Not Applicable
Persistent thoughts about thirst	○	○	○	○	○	○
Persistent thoughts about food	○	○	○	○	○	○

	Frequency			Severity		
	Never	Sometimes	Always	Minor	Serious	Not Applicable
Short term memory loss (forgetting things that have just happened)	○	○	○	○	○	○
Long term memory loss (forgetting things that you know well)	○	○	○	○	○	○
Mood swings	○	○	○	○	○	○
Depressed thoughts and feelings	○	○	○	○	○	○
Wandering thoughts	○	○	○	○	○	○
Irritability	○	○	○	○	○	○
Irrational thoughts	○	○	○	○	○	○
Sleep disturbance	○	○	○	○	○	○
Mental fatigue	○	○	○	○	○	○
Suicidal thoughts	○	○	○	○	○	○
Slow to make decisions	○	○	○	○	○	○
Making poor decisions (mistakes)	○	○	○	○	○	○
Loss of concentration	○	○	○	○	○	○
Slow reactions/response times	○	○	○	○	○	○
Other	○	○	○	○	○	○

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Appendix 2

Appendix 2: STROBE statement for chapter 2.

STROBE Statement—Checklist of items that should be included in reports of ***cross-sectional studies***

	Item No	Recommendation	Page No.	Relevant text from manuscript
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	Page 1	Study design is identified on lines 3-4 as a "observational cross-sectional study" study.
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	Page 3	Lines 68-72 provide study methods. Lines 73-87 provide key findings and conclusions.
Introduction				
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	Pages 4-6	Lines 92-133 highlight the physiological demands on jockeys over their career span and findings from previous studies completed on jockeys and other athlete groups, while also identifying relevant gaps.
Objectives	3	State specific objectives, including any prespecified hypotheses	Pages 5-6	Lines 126-133 provide research aims and hypothesis.
Methods				
Study design	4	Present key elements of study design early in the paper	Pages 6-9	Lines 135-216 describes the study design in detail.
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	Pages 6-8	Lines 135-193 describes periods of recruitment, exposure, and data collection.
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	Page 6	Lines 136-148 details participant recruitment, inclusion, and exclusion criteria.
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	Pages 6-9	Lines 135-216 describe statistical analysis in detail, including all relevant exposures, outcomes, and predictors.

1

Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	Pages 6-9	Lines 150-156 describe anthropometric data collection, Lines 158-181 describe echocardiography data collection, Lines 183-193 describe lifestyle questionnaire data collection. Lines 177-181 describe reproducibility of echocardiography measurements.
Bias	9	Describe any efforts to address potential sources of bias	Page 7	Lines 160-162 and 177-181 describe reproducibility of echocardiography measurements and steps taken to reduce bias.
Study size	10	Explain how the study size was arrived at	Pages 8-9	Lines 212-216 provide sample size calculation.
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	Pages 8-9	Lines 195-216 describe data analysis.
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	Pages 8-9	Lines 195-216 describe data analysis.
		(*b*) Describe any methods used to examine subgroups and interactions	Pages 8-9	Lines 195-216 describe data analysis.
		(*c*) Explain how missing data were addressed	N/A	N/A
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	Pages 8-9	Lines 195-216 describe data analysis.
		(*e*) Describe any sensitivity analyses	Pages 8-9	Lines 195-216 describe data analysis.
Results				
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	Page 9	Lines 220-221 identifies number of participants for study.
		(b) Give reasons for non-participation at each stage	N/A	N/A
		(c) Consider use of a flow diagram	N/A	N/A
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	Page 9	Lines 220-229 describe demographic information.
		(b) Indicate number of participants with missing data for each variable of interest	N/A	N/A
Outcome data	15*	Report numbers of outcome events or summary measures	Pages 9-14	Tables 1 & 2 highlight all primary outcome data. Table 3 & 4 highlight secondary outcome data.

Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	Pages 9-14	Lines 220-274 provide all results, including Cohen's *d* effect size & Cramer's V effect size for all outcomes.
		(*b*) Report category boundaries when continuous variables were categorized	N/A	Continuous variables were treated continuously.
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N/A	N/A
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	Pages 11-14	Lines 243-245 summarise GLS findings for both jockey groups. Lines 257-266 summarise subgroup analysis for weight management strategies used by both jockey groups. Lines 268-274 summarise subgroup analysis for comparison between weight management practices used by jockeys with normal and abnormal GLS.
Discussion				
Key results	18	Summarise key results with reference to study objectives	Page 14	Lines 276-282 summarize key findings.
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	Page 18-19	Lines 383-397 discuss limitations that should be acknowledged when considering the study findings.
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	Page 14-19	Lines 276-397 offer an interpretation of each key finding, considering prior research and potential limitations.
Generalisability	21	Discuss the generalisability (external validity) of the study results	Page 14-19	Lines 276-406 suggest generalisability of the findings and highlight the need to explore this research in other population groups.

Other information				
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N/A	N/A

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

Appendix 3

Appendix 3: STROBE statement for chapter 3.

STROBE Statement—Checklist of items that should be included in reports of ***cross-sectional studies***

	Item No	Recommendation	Page No.	Relevant text from manuscript
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	Page 1	Study design is identified on lines 3-4 as a "observational cross-sectional study" study.
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	Page 3	Lines 68-72 provide study methods. Lines 73-87 provide key findings and conclusions.
Introduction				
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	Pages 4-6	Lines 92-133 highlight the physiological demands on jockeys over their career span and findings from previous studies completed on jockeys and other athlete groups, while also identifying relevant gaps.
Objectives	3	State specific objectives, including any prespecified hypotheses	Pages 5-6	Lines 126-133 provide research aims and hypothesis.
Methods				
Study design	4	Present key elements of study design early in the paper	Pages 6-9	Lines 135-216 describes the study design in detail.
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	Pages 6-8	Lines 135-193 describes periods of recruitment, exposure, and data collection.
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	Page 6	Lines 136-148 details participant recruitment, inclusion, and exclusion criteria.
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	Pages 6-9	Lines 135-216 describe statistical analysis in detail, including all relevant exposures, outcomes, and predictors.

1

Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	Pages 6-9	Lines 150-156 describe anthropometric data collection, Lines 158-181 describe echocardiography data collection, Lines 183-193 describe lifestyle questionnaire data collection. Lines 177-181 describe reproducibility of echocardiography measurements.
Bias	9	Describe any efforts to address potential sources of bias	Page 7	Lines 160-162 and 177-181 describe reproducibility of echocardiography measurements and steps taken to reduce bias.
Study size	10	Explain how the study size was arrived at	Pages 8-9	Lines 212-216 provide sample size calculation.
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	Pages 8-9	Lines 195-216 describe data analysis.
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	Pages 8-9	Lines 195-216 describe data analysis.
		(*b*) Describe any methods used to examine subgroups and interactions	Pages 8-9	Lines 195-216 describe data analysis.
		(*c*) Explain how missing data were addressed	N/A	N/A
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	Pages 8-9	Lines 195-216 describe data analysis.
		(*g*) Describe any sensitivity analyses	Pages 8-9	Lines 195-216 describe data analysis.
Results				
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	Page 9	Lines 220-221 identifies number of participants for study.
		(b) Give reasons for non-participation at each stage	N/A	N/A
		(c) Consider use of a flow diagram	N/A	N/A
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	Page 9	Lines 220-229 describe demographic information.
		(b) Indicate number of participants with missing data for each variable of interest	N/A	N/A
Outcome data	15*	Report numbers of outcome events or summary measures	Pages 9-14	Tables 1 & 2 highlight all primary outcome data. Table 3 & 4 highlight secondary outcome data.

Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	Pages 9-14	Lines 220-274 provide all results, including Cohen's *d* effect size & Cramer's V effect size for all outcomes.
		(*b*) Report category boundaries when continuous variables were categorized	N/A	Continuous variables were treated continuously.
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N/A	N/A
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	Pages 11-14	Lines 243-245 summarise GLS findings for both jockey groups. Lines 257-266 summarise subgroup analysis for weight management strategies used by both jockey groups. Lines 268-274 summarise subgroup analysis for comparison between weight management practices used by jockeys with normal and abnormal GLS.
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Key results	18	Summarise key results with reference to study objectives	Page 14	Lines 276-282 summarize key findings.
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Generalisability	21	Discuss the generalisability (external validity) of the study results	Page 14-19	Lines 276-406 suggest generalisability of the findings and highlight the need to explore this research in other population groups.

Other information				
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N/A	N/A

*Give information separately for exposed and unexposed groups.

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Appendix 4

Appendix 4: Statement of Authorship for chapter 2 publication.

University of South Australia

Statement of Authorship

Title of Paper	Cardiac Structure and Function of Elite Australian Jockeys Compared to the General Population: An Observational Cross-Sectional Study
Publication Status	☒ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style
Publication Details Relevant reference details	An observational cross sectional study comparing the cardiovascular structure and function of Australian jockeys to the Australian general population. Citation: Farley, A., Bennett, H., Eston, R. et al. Cardiac Structure and Function of Elite Australian Jockeys Compared to the General Population: An Observational Cross-Sectional Study. Sports Med - Open 10, 119 (2024). https://doi.org/10.1186/s40798-024-00783-9
PRINCIPAL AUTHOR	
Name of Principal Author (Candidate)	Mrs Angela Farley
Contribution to the Paper Brief description of your work in this publication	Study conception and design, material preparation and coordination of participants or data collection, data collection and analysis Drafting of the manuscript then approval of the final version of manuscript for submission to journal.
Overall Percentage (%)	80
Certification	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature *Signatures will be redacted by the Library at the time of publication*	[redacted] Digitally signed by Angela Date: 2024.12.09 10:49:27 +10'30' — Date 9-Dec-24
CO-AUTHOR CONTRIBUTIONS	
By signing the Statement of Authorship, each author certifies that: I. the candidate's stated contribution to the publication is accurate (as detailed above); II. permission is granted for the candidate in include the publication in the thesis; and III. the sum of all co-author contribution is equal to 100% less the candidate's stated contribution.	
Name of Co-Author 1	Dr Rebecca Perry

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% Contribution to the Paper	10		
Signature *Signatures will be redacted by the Library at the time of publication*	Rebecca Perry — Digitally signed by Rebecca Perry Date: 2024.12.09 09:15:45 +10'30'	Date	9-Dec-24
Name of Co-Author 2	Dr Hunter Bennett		
% Contribution to the Paper	5		
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Name of Co-Author 3	Professor Roger Eston		
% Contribution to the Paper	5		
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