



Australian Government
Rural Industries Research and
Development Corporation

Risk Factors Associated with Racetrack Casualties in Thoroughbreds

Victoria, Australia 1989–2005

08/007



RIRDC Innovation for rural Australia



Australian Government

**Rural Industries Research and
Development Corporation**

Risk Factors Associated with Racetrack Casualties in Thoroughbreds

Victoria, Australia 1989-2005

by Dr Lisa Boden

June 2008

RIRDC Publication No 08/007
RIRDC Project No UM-58A

© 2008 Rural Industries Research and Development Corporation.
All rights reserved.

ISBN 1 74151 596 3
ISSN 1440-6845

Risk Factors Associated with Racetrack Casualties in Thoroughbreds: Victoria, Australia 1989 - 2005

Publication No. 08/007
Project No. UM-58A

The information contained in this publication is intended for general use to assist public knowledge and discussion and to help improve the development of sustainable regions. You must not rely on any information contained in this publication without taking specialist advice relevant to your particular circumstances.

While reasonable care has been taken in preparing this publication to ensure that information is true and correct, the Commonwealth of Australia gives no assurance as to the accuracy of any information in this publication.

The Commonwealth of Australia, the Rural Industries Research and Development Corporation (RIRDC), the authors or contributors expressly disclaim, to the maximum extent permitted by law, all responsibility and liability to any person, arising directly or indirectly from any act or omission, or for any consequences of any such act or omission, made in reliance on the contents of this publication, whether or not caused by any negligence on the part of the Commonwealth of Australia, RIRDC, the authors or contributors.

The Commonwealth of Australia does not necessarily endorse the views in this publication.

This publication is copyright. Apart from any use as permitted under the *Copyright Act 1968*, all other rights are reserved. However, wide dissemination is encouraged. Requests and inquiries concerning reproduction and rights should be addressed to the RIRDC Publications Manager on phone 02 6271 4165.

Researcher Contact Details

Dr Lisa A. Boden
CID, Ashworth Laboratories, Kings Buildings
University of Edinburgh
West Mains Road
Edinburgh EH9 3JT

Professor Ron Slocombe
School of Veterinary Science
University of Melbourne.
250 Princes Highway
Werribee VIC 3030.

Phone: +44 0131 650 7923
Fax: +44 0131 650 6564
Email: lboden@staffmail.ed.ac.uk

Phone: +61 03 8344 9565
Fax: +61 03 9731 2366
Email: r.slocombe@unimelb.edu.au

In submitting this report, the researcher has agreed to RIRDC publishing this material in its edited form.

RIRDC Contact Details

Rural Industries Research and Development Corporation
Level 2, 15 National Circuit
BARTON ACT 2600
PO Box 4776
KINGSTON ACT 2604

Phone: 02 6271 4100
Fax: 02 6271 4199
Email: rirdc@rirdc.gov.au.
Web: <http://www.rirdc.gov.au>

Published in June 2008

Contents

Foreword	v
Acknowledgments	vii
Executive Summary.....	ix
Objectives	xii
1. Background	1
2. Methodology	9
3. Post mortem study of Thoroughbred racing fatalities in Victoria (2001 – 2004).....	17
4. Risk of fatality and causes of death of Thoroughbred horses associated with racing in Victoria (1989 – 2004)	32
5. Risk factors for Thoroughbred racehorse fatality in flat starts in Victoria (1989-2004).....	43
6. Risk factors for Thoroughbred racehorse fatality in jump starts in Victoria (1989-2004).....	49
7. General Discussion.....	55
8. Specific recommendations for intervention strategies and further research.....	65
9. Bibliography.....	67

Table of tables

Table 1	Track classification system introduced by Racing Victoria Ltd. in 2005	2
Table 2	Comparison of track condition classifications in Australia and the United Kingdom	2
Table 3	Protocol for grading pulmonary lesions post mortem in racehorses that died or were euthanased on Victorian racecourses between February 1, 2001 and October 31, 2004.....	10
Table 4	Summary of categorical causes of fatality in Thoroughbred horses that died or were euthanised on Victorian racecourses between February 1 2001 and October 31 2004 as determined by post mortem examination	18
Table 5	Summary of anatomical location of catastrophic limb injuries sustained by racehorses on Victorian racecourses between February 1, 2001 and October 31, 2004 as determined by post mortem examination.....	20
Table 6	Summary of post mortem findings in 17 horses that died suddenly with acute pulmonary lesions whilst racing or training on Victorian racecourses between February 1, 2001 and October 31, 2004 (pulmonary lesions are classified as mild (+), moderate (++) , severe (+++) or absent (-)).....	21
Table 7	Numbers of Thoroughbred horses submitted for post mortem examination from Victorian racetracks between February 1, 2001 and October 31, 2004, by cause of death and location of racetrack	22
Table 8	Sensitivity and specificity of diagnoses made by racecourse veterinarians for various conditions in horses that were submitted for post mortem examination between February 1, 2001 and October 31, 2004.....	24
Table 9	Sensitivity and specificity of diagnoses made by racecourse veterinarians regarding the specific anatomical location of catastrophic limb injuries at single anatomical sites (n = 29) in Thoroughbreds racing in Victoria between February 1, 2001 and October 31, 2004.....	25
Table 10	Number of Thoroughbred racehorse fatalities and risk of fatality per 1000 starts in flat races in Victoria between 1989 and 2004 by year; the subtotals according to racetrack location and the risk ratio (RR) of fatality for each racing year compared with the racing year of 1989-1990.....	34
Table 11	Number of Thoroughbred racehorse fatalities and risk of fatality per 1000 starts in jump races in Victoria between 1989 and 2004 by year; the subtotals according to racetrack location and the risk ratio (RR) of fatality for each racing year compared with the racing year of 1989-1990.....	35
Table 12	Number of fatalities of Thoroughbred horses on racecourses in Victoria between 1989 and 2004, according to racetrack location, race type and reported cause of fatality	37
Table 13	Number of catastrophic limb injuries by anatomical location and race type	37
Table 14	Final Model - Risk factors for fatality in 3452 flat starts in Victoria between the 1989-1990 and 2003-2004 racing years with racecourse as a random effect	45
Table 15	Final multivariable model for risk factors for fatality in 2297 jump starts in Victoria between the 1989-1990 and 2003-2004 racing years with racecourse as a random effect	51

Foreword

The success of the Thoroughbred horse industry is vital to the economic health of Victoria and Australia. The industry contributes \$6.3 billion per annum to the national Gross Domestic Product annually, with racing and associated activities (such as breeding and wagering) generating just over half of this (Gordon 2001). In 2001 in Victoria, the Thoroughbred racing industry contributed \$2.2 billion to the national Gross Domestic Product and provided full-time employment for over 22,000 personnel (Australian Racing Board 2001). Approximately 23% of Australian races are held in Victoria (Australian Racing Board 2003).

Key research issues for the Victorian racing industry revolve around animal and jockey welfare. This focus is dictated by community concerns over wastage rates for racehorses, jockey injuries and fatalities sustained in racing. Although there will inevitably be injury in athletic competition, risk of injury could be reduced if risk factors were identified and modified.

In this context, the main role of epidemiological studies is to identify risk factors that can be mitigated to decrease the risk of injury. Dr. John Bourke, who was first appointed as a veterinary Stipendiary Steward at the Victoria Racing Club in 1963, was the first to take an epidemiological approach to document racehorse wastage in Victoria (Bourke 1994 and 1995). His pioneering work highlighted the need for further research into racehorse injury and fatality in Victoria. Since 2001, Racing Victoria Ltd and the Rural Industries Research and Development Corporation (RIRDC) have been responsible for funding all of the University of Melbourne research projects investigating Thoroughbred wastage in Victoria (Racing Victoria Ltd 2005b).

The aims of this multi-faceted epidemiological study were to:

- identify causes of death of horses during or after racing through post mortem examinations
- describe the risk of fatality in flat and jump racing for the previous 15 years
- identify risk factors for fatality in flat and jump racing so that intervention strategies to reduce the risk of fatality can be developed by the industry in the future.

This study confirmed that catastrophic limb injury was the most important cause of death and also demonstrated that sudden deaths formed a larger proportion of all deaths in Victoria than elsewhere in the world. In Victoria, the risk of fatality in flat starts was lower (0.44 per 1000 starts) than that in jump starts (8.3 per 1000 starts). The risk of fatality in flat starts in Victoria was lower than reported in the UK and the USA but the risk in jump starts was higher. The discrepancy between Victoria and overseas countries in the risk in jump starts suggested that it should be possible to decrease the risk of fatality if important causal factors were identified and modified. As there was a large difference in the risk of fatality between race types within Victoria, separate studies were undertaken to identify risk factors for fatality in flat and jump starts.

Multivariable logistic regression models for flat and jump racing identified several risk factors. In flat racing, stallions had greater odds of fatality than geldings. Horses that had a start in the 31-60 day period prior to the study start were observed to have increased odds of fatality whereas horses that had accumulated distance in jump races prior to the study start had decreased odds of fatality. The findings were consistent with the hypothesis that horses accumulating high speed exercise without respite are predisposed to catastrophic injury. The study also highlighted the need to investigate further the adverse effects of different track ratings on the incidence of injury and subsequent fatality.

In jump racing, increasing racing career length and increasing numbers of flat, hurdle and steeple starts accumulated in the 60 days prior to the study start were associated with decreased odds of fatality. Steeplechase races, increasing numbers of flat and jump starts accumulated in the racing career prior to the study start and, if there had been at least one start in the 14 days prior to the study start, were associated with increased odds of fatality. In the flat and jump models, racecourse location and the year of the start were identified as important risk factors.

The findings of this study illustrate the importance of collecting more information on the training and racing schedules in both flat and jump racehorse populations. Other risk factors identified in the flat and jump studies highlight areas for future research by the Victorian racing industry. In particular, further investigation is required to identify reasons for the effect of track condition and the difference between entire males and geldings relative to mares with respect to fatality. Identification of such track and horse-level risk factors may facilitate the development of methods to detect those horses at higher risk of fatality.

Sustaining the successful partnership between industry and academia requires that epidemiological researchers of racehorse injury and fatality maintain strong links with trainers, owners, racecourse veterinarians, racecourse officials and data recorders. Ideally, these links are maintained through prompt and thorough communication of research results and recommendations to the racing industry so that successful strategies can be implemented to improve the welfare of racehorses and jockeys. This report will be a useful basis for those contemplating investment or formulating policy and will help to inform RIRDC as it plans its research and development priorities into the future.

This project was funded from industry revenue which is matched by funds provided by the Australian Government.

This report, an addition to RIRDC's diverse range of over 1800 research publications, forms part of our Horse R&D Program, which aims to assist in developing the Australian horse industry and enhancing its export potential.

Most of our publications are available for viewing, downloading or purchasing online through our website:

- downloads at www.rirdc.gov.au
- purchases at www.rirdc.gov.au

Peter O'Brien

Managing Director

Rural Industries Research and Development Corporation

Acknowledgments

This project developed from a concept hatched by some of us after considering input from Racing Victoria Ltd, and the project led at its inception by Professor Andrew Clark, Chair of Equine Studies (2000-2005) at The University of Melbourne after extensive collaboration with the veterinary pathology section. This project report contains a substantial body of new information, and the integration of various components of the research arising from very different academic discipline areas would not have been possible without the oversight provided by one individual, Dr Lisa Boden. This research formed the major part of her dissertation and she was awarded a PhD from the university in 2006 for her research. The acknowledgement below relates to those who assisted Lisa for the duration of her studies at the University of Melbourne. This acknowledgement and the report that follows were authored by Lisa, with some minor editing by others.

Ron Slocombe
Chair, Veterinary Pathology

This study would not have been possible without contributions from many people.

Racehorse fatalities have been identified as a major research challenge facing racing industries around the world but regional differences between jurisdictions have made it difficult to extrapolate previous study results with confidence. Racing Victoria Ltd. has recognised the importance of understanding the causes of and risk factors associated with racehorse fatalities. This project is a direct result of their foresight and desire to improve the overall health and welfare of racehorses in Victoria.

I am grateful for the generous funding provided by Racing Victoria Ltd., the Rural Industries Research and Development Corporation (RIRDC) and The Equine Centre at The University of Melbourne. In particular, the considerable financial support of Racing Victoria Ltd. for the post mortem study at The University of Melbourne has been essential to the success of this project. This partnership has highlighted the benefit of collaboration between industry and academia. Professor Andrew Clarke, Director of the Equine Centre at The University of Melbourne, Professor Ronald Slocombe, Chair of Veterinary Pathology in the Department of Veterinary Science at The University of Melbourne and Dr. John McCaffrey, Chair of Veterinary Services at Racing Victoria Ltd. were key people in the development of this collaborative effort. Dr. John Morton was the consultant epidemiologist for this project. I would like to acknowledge his input in every chapter of this thesis and his advice and support throughout the entire duration of the project. Garry Anderson provided all of the statistical support for the studies in this thesis. His considerable experience in biometry and epidemiology has been invaluable in this project.

In 2004, Dr. Chris Riggs from the Hong Kong Jockey Club introduced me to The Epidemiology Group at The University of Liverpool. In that Group, Professor Kenton Morgan and Dr. Tim Parkin (now of the Animal Health Trust) were subsequently recruited to assist me with this project in 2005. I spent four weeks at The University of Liverpool and benefited enormously from their collective experience in epidemiology and the racing industry. I was incredibly fortunate to have had their support and guidance throughout this last year.

The post mortem study would not have been possible without the time and energy of the pathologists, Professor Ronald Slocombe, Dr. Jenny Charles, Dr. Peter Finnin and Dr. Jeanine Sandy, the insight of Professor Kenneth Jubb and the support of technical staff, Josie Wilson, Paul Benham and Dennis Miller in the Veterinary Pathology section at The University of Melbourne.

There are many other people who have been directly and indirectly involved in this project. I am grateful to Dr. John McCaffrey, Dr. Paul O'Callaghan, Heidi Lester and Rob Hall at Racing Victoria Ltd. for answering my many enquiries about the Victorian racing industry. I would like to thank Tony de Keiser for his help in building my database, David Urquhart and Melissa Brown at the Australian Associated Press Pty. Ltd. for their help in securing the racehorse performance data and Chris Skorsis and Phi-Ta Hung at Racing Victoria Ltd. for their assistance and access to the *i*-RIS database at Racing Victoria Ltd.

Lisa Boden

Executive Summary

What the report is about

This report describes investigation of the risk factors that lead to injuries and fatalities as a retrospective study of Thoroughbred racing in Victoria.

Background

The success of the Thoroughbred horse industry is vital to the economic health of Victoria and Australia. The industry contributes \$6.3 billion per annum to the national Gross Domestic Product annually, with racing and associated activities (such as breeding and wagering) generating just over half of this (Gordon 2001). In 2001 in Victoria, the Thoroughbred racing industry contributed \$2.2 billion to the national Gross Domestic Product and provided full-time employment for over 22,000 personnel (Australian Racing Board 2001). Approximately 23% of Australian races are held in Victoria (Australian Racing Board 2003).

Key research issues for the Victorian racing industry revolve around animal and jockey health and welfare. This focus is dictated by community concerns over wastage rates for racehorses and jockey injuries and fatalities sustained in racing. Although there will inevitably be injury in athletic competition, risk of injury could be reduced if risk factors are identified and mitigated.

In this context, the main role of epidemiological studies is to identify risk factors that can be modified to decrease the risk of injury. Dr. John Bourke, who was first appointed as a Veterinary Stipendiary Steward at the Victoria Racing Club in 1963, was the first to take an epidemiological approach to document racehorse wastage in Victoria (Bourke 1994 and 1995). His pioneering work highlighted the need for further research into racehorse injury and fatality in Victoria. Since 2001, Racing Victoria Ltd and the Rural Industries Research and Development Corporation have been responsible for funding all of The University of Melbourne research projects investigating Thoroughbred wastage in Victoria (Racing Victoria Ltd 2005b).

Aims/Objectives

This RIRDC-funded epidemiological research aimed to investigate Thoroughbred racing fatalities by:

- identifying causes of death of horses during or after racing through post mortem examinations over a three year period from February 1, 2001 to October 31, 2004
- describing the risk of fatality in flat and jump racing for the previous 15 years
- identifying risk factors for fatality in flat and jump racing so that intervention strategies to reduce the risk of fatality can be developed by the industry in the future.

Methods used

The causes and risk of fatality of Thoroughbred racehorses in flat and jump races in Victoria between 1989 and 2004 were investigated using archived records of Thoroughbred racehorse fatalities, recorded data on racehorse performance and, for a subset of fatalities, results from post mortem examinations.

Results/Key Findings

A three year prospective post mortem study of all racehorses that died or were euthanised on city tracks in Victoria was utilised to accurately determine specific causes of death. This study confirmed that catastrophic limb injury was the most important cause of death and also demonstrated that sudden deaths formed a larger proportion of all deaths in Victoria than

elsewhere in the world. The post mortem study was used to validate the provisional diagnoses of cause of death made by racecourse veterinarians. The results of this study suggested that it was reasonable to rely on data collated from veterinary reports provided that general case definitions were used, and where a track-side diagnosis of sudden death was sufficient. However, trackside diagnoses frequently failed to identify the organ system involved in causing sudden death associated with strenuous exercise.

Archived veterinary reports were used to measure the risk of fatality in flat and jump races. There were 514 fatalities and 743,552 starts over the 15 year period. There were 316 fatalities in flat starts and 198 fatalities in jump starts. In Victoria, the risk of fatality was lower in flat starts (0.44 per 1000 starts) than in jump starts (8.3 per 1000 starts). The risk of fatality in flat starts in Victoria was lower than that reported in the UK and the USA but the risk in jump starts was higher. The discrepancy between Victoria and overseas countries in the risk in jump starts suggested that it should be possible to decrease the risk of fatality if important causal factors were identified and modified. Because there was a large difference in the risk of fatality between race types within Victoria, separate studies were undertaken to identify risk factors for fatality in flat and jump starts.

Multivariable logistic regression models identified several risk factors. In flat racing, stallions had greater odds of fatality than geldings. Horses that had a start in the 31-60 day period prior to the study start were observed to have increased odds of fatality whereas horses that had accumulated distance in jump races prior to the study start had decreased odds of fatality. Additionally, increasing race length increased the odds of fatality in flat starts. Starts on fast or good tracks compared with heavy, slow or dead tracks increased the odds of fatality in flat starts in the early years of the study. However, by the final year of the study no effect of track condition was evident. In jump racing, increasing racing career length and increasing numbers of flat, hurdle and steeple starts accumulated in the 60 days prior to the study start were associated with decreased odds of fatality. Steeplechase races, increasing numbers of flat and jump starts accumulated in the racing career prior to the study commencing, and if there had been at least one start in the 14 days prior to the study, were associated with increased odds of fatality. In the flat and jump models, racecourse location and the year of the start were identified as important risk factors.

Implications

The findings relating to prior form history illustrate the importance of collecting more information on the training and racing schedules in both flat and jump racehorse populations. The number of starts and the distance accumulated in both official and unofficial trials need to be recorded accurately to determine monthly averages of racing intensity. Accurate measurement of speeds attained during racing and training using global positioning systems (GPS) technology would also improve the understanding of racing intensity thresholds that may predispose to catastrophic injury (Perkins 2005). Reliance on proxy measures of racing form history (such as the number of starts and the distance accumulated in different race types) in the absence of training data remains a significant weakness of this and other studies of racehorse fatality and injury.

Other risk factors identified in the flat and jump studies highlight areas for future research by the Victorian racing industry.

Recommendations

Recommendations which were suggested as a result of this study include:

- Further investigation is required to identify reasons for the increased odds of fatality amongst entire males and geldings relative to mares. Bodyweight, conformation, behaviour and sex hormone concentrations may mediate this association. Identification of such horse-level risk factors may facilitate development of methods to detect those horses at high risk of fatality.
- Collection of blood samples from horses at the point of death on the racecourse to facilitate the investigation of sudden deaths. Assays of markers of peracute myocardial injury (such as cardiac troponin I) may help to confirm the presence of myocardial degeneration or necrosis (Cornelisse *et al.* 2000; Phillips *et al.* 2003; Schwartzwald *et al.* 2003) and measurement of electrolytes (especially potassium, calcium and magnesium) may identify potential triggers of such injury. Coagulation studies on point-of-death blood samples may also be beneficial in investigating cases of sudden death from massive haemorrhage.
- Mandatory post mortem examinations for all horses that die or are euthanised on country racecourses in Victoria, in accordance with the current policy for fatalities sustained on city racecourses in Victoria or alternatively, a random sample of all country fatalities should be subjected to post mortem examination each year.
- Improvement of the design of the racehorse fatality report form currently in use by Racing Victoria Ltd.
- Systematic and standardised application of interventions throughout the Victorian racing industry. The effects of these interventions should be assessed using a well-designed monitoring system.
- Assessment of the repeatability of track condition measurements and subsequent standardization of the methods of measurement and reference ranges for each track classification across all Australian racecourses.
- The Victorian racing industry should continue to investigate reasons for differences in the risk of fatality between steeplechase and hurdle races. Any consequent interventions such as modifications to fence design and positioning should be implemented across all racecourses simultaneously so that the effect of intervention measures can be rigorously and scientifically assessed. Fatalities, injuries and falls should be recorded systematically so that the effect of the intervention can be measured accurately. The application of process control charts (Cusum charts) may be a useful method for the racing industry to monitor fatalities.
- Limitations on the number of flat starts that horses are allowed to accumulate before competing in jump races.
- Prioritisation of research into early detection of horses at high risk of fatality in flat and jump starts. Further investigation of appropriate rates of accumulation of high speed exercise in racing and training are necessary to identify periods when horses are at the most risk and to establish appropriate racing schedules. Future research should also incorporate prior medical histories of horses to ascertain the importance of prior subclinical and clinical injuries as risk factors for catastrophic injury and death.
- In light of differences in the odds of fatality on different racecourses, maintenance of the Victorian racetrack database developed by Stubbs (2004) is important for future studies investigating the effects of differences in track design and surface and the effects of modifications to race tracks on the risk of fatality in flat and jump starts.
- It is recommended that Racing Victoria Ltd initiate studies comparable to those undertaken in the UK on fatal fractures (Parkin *et al.* 2004a and b) and falls (Pinchbeck *et al.* 2002 and 2003) to identify risk factors associated with specific types of injury.

- There is a need for formal racehorse fatality monitoring programs to be developed in the other States in Australia. Ultimately, development of national and international fatality databases may be possible to enable direct comparisons of the risk of racehorse fatality in different countries.

Summary:

This study was the first in Australia to utilise the results of post mortem examinations to describe accurately the causes of death of racing Thoroughbreds and to validate the diagnoses of racecourse veterinarians. It was also the first in Australia to report the risk of fatality in jump and flat races over an extensive time period and to identify risk factors specific to fatality in flat and jump starts in Victoria.

Objectives

The objectives of this study were to:

- identify causes of death of horses during or after racing through post mortem examinations over a three year period from February 1, 2001 to October 31, 2004
- describe the risk of fatality in flat and jump racing for the previous 15 years
- identify risk factors for fatality in flat and jump racing so that intervention strategies to reduce the risk of fatality can be developed by the industry in the future.

1. Background

The Australian and Victorian racing industries

The success of the Thoroughbred horse industry is vital to the economic health of Victoria and Australia. The industry contributes \$6.3 billion per annum to the national Gross Domestic Product annually, with racing and associated activities (such as breeding and wagering) generating just over half of this (Gordon 2001). In 2001 in Victoria, the Thoroughbred racing industry contributed \$2.2 billion to the national Gross Domestic Product and provided full-time employment for over 22,000 personnel (Australian Racing Board 2001). Approximately 23% of Australian races are held in Victoria (Australian Racing Board 2003).

The Australian Racing Board is the national administrative body of the Australian Thoroughbred Racing Industry and is responsible for formulation and administration of the Australian Rules of Racing. There are also racing organisations which administer racing in each of the six Australian States and two Territories. The individual state and territory racing administrations may make local rules for specific local conditions as long as they are not inconsistent with the principles established by the Australian Rules as administered by the Australian Racing Board (Australian Racing Board 2003). In the state of Victoria, the racing authority is Racing Victoria Ltd. Racing Victoria Ltd. is responsible for registration and supervision of individual race clubs, licensing of relevant industry participants, handicapping, administration of industry funding, appointment of stewards' panels to apply the Rules of Racing, strategic planning, publicity and marketing (Australian Racing Board 2003).

Horses in Australia tend to race more frequently than horses in the United Kingdom (UK) (Bailey 1998a and 1998b) and there is greater emphasis on the racing of two year old horses. In contrast to the restricted racing season in the UK and the system of race-meets in the United States of America (USA) where races are conducted intensively over a limited number of days at the same venue, flat racing in Australia is conducted at different tracks on a daily basis throughout the year (Bailey *et al.* 1998a). Horses starting in flat races form the largest proportion of horses starting in all races in Australia (Australian Racing Board 2003). Horses starting in jump (hurdle and steeplechase) events in Australia are usually older than those starting in flat races and have previously had a career in flat racing (Bailey *et al.* 1998a).

The racing year in Australia begins on August 1 and ends on July 31. Within Australia, the direction of racing differs between States. In Victoria, racing is in an anti-clockwise direction. There are four metropolitan racecourses (Caulfield, Flemington, Moonee Valley and Sandown) and 53 country racing clubs. The design of racecourses in Australia vary considerably and generally speaking have tighter turns than courses in the UK (Bailey 1998a).

In Victoria, training tracks are located within racecourses and involve straight and curved sections. In contrast, training in the UK is usually conducted away from the racecourse on long straight expanses of turf, often on a gradual incline (Bailey 1998a).

While racing takes place on turf tracks in Victoria, training is predominantly on non-turf (artificial) tracks to minimise track maintenance requirements (Stubbs 2004). Most of the Victorian racecourse turf is composed of cool weather grasses such as ryegrass and Kentucky blue on sand with peat moss (Stubbs 2004). Each raceday, racecourse stewards assess the condition of the track (also called the track rating or the going of the track) subjectively and objectively (using penetrometer readings). Racing Victoria Ltd. regulates that races be held on good tracks with some give. Trainers are permitted to scratch horses after the official scratching time if there has been a subsequent downgrade of the track conditions (from fast or

good to heavy, slow or dead). A new numbers-based system of rating tracks was introduced in December 2005 to provide a more detailed description of track conditions. Table 1 describes the ten track rating classifications under the new system and also provides a reference for the five now out-moded categories of track rating (fast, good, dead, slow, heavy).

Table 1 Track classification system introduced by Racing Victoria Ltd. in 2005 (Racing Victoria Ltd. website 2005)

Category	Scale	Description
Fast	1	A dry hard track
Good	2	A firm track
Good	3	Ideal track with some give
Dead	4	Track with give, better side of dead
Dead	5	Significant amount of give, worse side of dead
Slow	6	A mildly rain affected track, better side of slow
Slow	7	Rain affected, worse side of slow
Heavy	8	Soft track, just into heavy range
Heavy	9	Very soft, genuinely heavy
Heavy	10	Very soft and wet, heaviest category

Correlation of the Australian categories of the track going with those used in the UK is presented in Table 2 (TDH Parkin, *personal communication*, 2005).

Table 2 Comparison of track condition classifications in Australia and the United Kingdom

Australia	United Kingdom
Fast	Hard, firm
Good	Good to firm
Dead	Good
Slow	Good to soft
Heavy	Soft, heavy

Most races in Australia and Victoria are flat races. In Victoria in 2003, 97% of Thoroughbred races were flat races (Australian Racing Board 2003). Some of Australia's most prestigious flat races are convened in Victoria (e.g. the Melbourne Cup, the Cox Plate and the Caulfield Cup). In Australia, jump racing is only held in Victoria, South Australia and Tasmania, with the majority of Australian jump races being held in Victoria. Hurdle and steeplechase races comprise 3% of all Victorian races (Australian Racing Board 2003).

Although the economic contribution of jump racing is difficult to assess (Racing Victoria Ltd. 2005a), the Country Racing Victoria Association considers jump racing to be a significant contributor to the economy of regional Australia, particularly in Victoria and South Australia (Racing Victoria Ltd. 2005a).

With increased public lobbying for improved horse and jockey welfare, there has been considerable pressure on the Victorian racing industry to justify the continuation of jump racing. In response, the Victorian racing industry has undertaken four reviews of jump racing over the last two decades (1994, 1998, 2003 and 2005) (Benton 1994; Racing Victoria Ltd. 1998, 2002 and 2005a). The Jump Review Panels have made a number of recommendations to improve the safety of jump racing. The most notable change has been made to the design of hurdles and steeplechase fences with a new modular fence design being introduced on selected Victorian racecourses from 2002 onwards.

Other changes recommended by the Jump Review Panels (Benton 1994, Racing Victoria Ltd. 1998 and 2002) and implemented by industry have included restrictions on the number of horses starting in jump races, improvements in prize money offered for jump races, tightening of pre-existing qualifying conditions for horses participating in jump events, specific educational programmes for jockeys and trainers, and changes to the scheduling of jump races so that the majority occurs in the autumn and winter. Further details on specific recommendations made by the Jump Review Panels are available in the Jump Review Panel reports (Benton 1994, Racing Victoria Ltd. 1998, 2002 and 2005a) and in Chapter 4.

Key research issues for the Victorian racing industry revolve around animal and jockey welfare. This focus has and is being dictated by community concerns over the wastage rates for racehorses as well as jockey injuries and fatalities sustained in racing. Although there will inevitably be injury in any athletic competition, chances of injury could be reduced if risk factors are identified and modified. In this context, the main role of epidemiological studies is to identify risk factors that can be modified to decrease the risk of injury. Dr. John Bourke, who was first appointed as a Veterinary Stipendiary Steward at the Victoria Racing Club in 1963, was the first to take an epidemiological approach to document racehorse wastage in Victoria (Bourke 1994 and 1995). His pioneering work highlighted the need for further research into racehorse injury and fatality in Victoria. Since 2001, Racing Victoria Ltd. and the Rural Industries Research and Development Corporation have been responsible for funding all of The University of Melbourne's research projects investigating wastage of Thoroughbred racehorses in Victoria (Racing Victoria Ltd. 2005b).

Sustaining the successful partnership between industry and academia requires that epidemiological researchers of racehorse injury and fatality maintain strong links with trainers, owners, racecourse veterinarians, racecourse officials and data recorders. Ideally, these links are maintained through prompt and thorough communication of research results and recommendations to the racing industry so that successful strategies can be implemented to improve the welfare of racehorses and jockeys.

Discussion of Australian and overseas studies of racehorse injury and fatality

The first studies to investigate racehorse fatalities in Australia were those of Bourke who described the frequency of injuries and fatalities in the Thoroughbred industry (Bourke 1995). He specifically described the risk of fatalities in different race types (Bourke 1994). Bourke (1994) also provided proportional mortality rates for specific causes of fatality such as catastrophic limb injury and sudden deaths. These data were used as a basis for comparison in later investigations of severe musculoskeletal injuries sustained by Thoroughbred horses in flat and jump races on Sydney and Victorian tracks (Bailey *et al.* 1997a and 1998). The studies of Bailey *et al.* (1997a and 1998) were the first in Australia to describe associations between potential risk factors and severe musculoskeletal injury sustained during racing. The major limitation of both of these studies was the potential for misclassification of cases and controls due to the case definition that was used. Cases were defined as horses that were recorded by attending veterinarians as having a musculoskeletal injury and then failing to race or trial for six months post-injury. This case definition was chosen so that the study would be comparable to that of Mohammed *et al.* (1991). However, the assumption that a non-return to racing over six months was directly a result of injury meant that researchers could not take into account other reasons for a non-return that may have operated following injury. For example, mares and stallions could have been sent to stud or horses could have been sold and moved overseas or retired or spelled for reasons apart from injury.

Bailey *et al.* (1998) demonstrated increased odds of severe musculoskeletal injury in hurdles and steeplechase events compared with flat races, a finding consistent with that of Bourke (1994). However, because risk factors were examined for serious injury in all races (flat and jump combined), Bailey *et al.* (1998) were not able to determine risk factors specific to these different race types. Moreover, because the odds of severe injury were so much greater in jump starts than in flat starts, this approach may have made it more difficult to identify other risk factors with weaker associations to severe injury and, to identify risk factors specific to these different race types. As the populations of horses that participate in flat and jump races in Victoria differ (Bailey *et al.* 1998), it is reasonable to assume that there may be different reasons for severe musculoskeletal injuries in each race type and thus, it is likely that the risk factors for severe musculoskeletal injury will differ between flat and jump races.

Studies of racehorse fatalities originated in the USA with a series of case-control and cohort studies to investigate injury in Thoroughbreds in racing in Minnesota (Haynes and Robinson 1988; Robinson *et al.* 1988; Kobluk *et al.* 1990 and 1992). The purpose of these studies was to assess whether it was feasible to implement a national system of monitoring racecourse fatalities in the USA. In the cohort study on injuries (Haynes and Robinson 1988; Robinson *et al.* 1988; Kobluk *et al.* 1990 and 1992), data were collected based on training data and clinical examinations pre- and post-racing. However, over 50% of the horses were lost to follow-up, potentially introducing selection bias and reducing the validity of the study. In the case-control study (Haynes and Robinson 1988; Robinson *et al.* 1989; Kobluk *et al.* 1990 and 1992), risk factors were examined at the univariable level for fatality, serious injury and non-return to racing of greater than six months. The approaches in these studies (Haynes and Robinson 1988; Robinson *et al.* 1988; Kobluk *et al.* 1990 and 1992) were not developed further and a multi-site national monitoring program for injuries or fatalities did not eventuate.

Other studies in different regions of the USA were undertaken to identify specific risk factors for injury and fatality. These studies highlighted regional differences in the types of risk factors identified. However, analytical methods differed between studies, ranging from simple univariable analyses (for example, Rooney *et al.* 1982a, 1982b, 1983a, 1983b, 1983c and 1983d) to multivariable logistic regression. Critical differences in study designs and case

definitions make it impossible to draw definitive conclusions about the reasons for differences in findings between studies. Furthermore, these differences in design make extraction of data from the different studies difficult for meta-analysis.

Other early studies in the USA focused on describing the risk of injury and fatality. Many of these descriptive studies relied entirely on retrospective records of racecourse veterinarian or stewards' reports (Palmer 1986; Suann 1992; Bathe 1994; Bourke 1994; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995; Mizuno 1996; Wilson *et al.* 1996b; Cohen *et al.* 1999b; Williams *et al.* 2001; Wilson *et al.* 1993; Verheyen *et al.* 2004) and may inadvertently have underestimated or overestimated the risk of particular conditions due to errors in classification of injuries/fatalities. A recent study in the UK reported that, of 100 fatalities with distal limb fractures, Jockey Club fracture reports were 'accurate' (i.e. the fractured bone was correctly identified as fractured in the report) for only 71%. For reports from individual Jockey Club veterinary officers, the accuracy varied between 33% and 100% (Parkin 2002). Therefore, the results of studies that rely solely on veterinary reports or databases for their case definitions should be interpreted cautiously.

Full post mortem examinations by specialist pathologists would minimise such misclassification errors. However, few studies of racehorse injury and fatality have incorporated full post mortem examination because of costs and practical difficulties of transporting cadavers from racecourses or training yards to facilities where post mortem examinations can take place and, because of the expense of the post mortem examination itself. Furthermore, a rigorous post mortem examination of a horse may take three to five hours (J.A. Charles and R.F. Slocombe, *personal communication* 2005). A sufficient number of qualified staff must be available if large numbers of post mortem examinations are to be performed. However, post mortem examinations are an essential component of racehorse fatality research as they can permit detection of additional concurrent injuries or conditions that would have otherwise not been recorded, in addition to validation of cause-of-death diagnoses made by racecourse veterinarians or stewards.

The most extensive investigation of racehorse fatality has been underway at the University of California at Davis since 1990 (Stover *et al.* 1992, 1993 and 1994; Estberg *et al.* 1993, 1994, 1995a, 1995b, 1996a, 1996b, 1998a and 1998b; Johnson *et al.* 1994a and 1994b; Carrier *et al.* 1998; Hill *et al.* 2001 and 2003). This program was established in conjunction with the California Horse Racing Board and the California Veterinary Diagnostic Laboratory System. Mandatory post mortem examinations are conducted following every death during racing or training in California on racetracks under the jurisdiction of the California Horse Racing Board. This was the first large-scale prospective study of racehorse fatalities in the world. Because of the post mortem examinations, researchers were able to describe specific causes of fatality with absolute confidence and determine proportional mortality rates and specific causes of sudden death from the data collected (Johnson *et al.* 1994a and 1994b). Previous studies that lacked post mortem examinations could only speculate on the number of sudden deaths attributable to pulmonary oedema, congestion or haemorrhage or cardiovascular disease (Bourke 1994).

The California post mortem research (Johnson *et al.* 1994a and 1994b) confirmed that distal catastrophic forelimb fracture was the major cause of racehorse fatality. That the most common fractures sustained involved the metacarpus III and proximal sesamoids in racing whereas tibial and pelvic fractures predominated in training raised questions about the potential differences in the pathogenesis of different types of injury (Stover *et al.* 1992 and 1993; Carrier *et al.* 1998; Hill *et al.* 2001). The demonstration of an association between stress fractures and tibial and pelvic fractures (Stover *et al.* 1992 and 1993) was another important finding that illuminated the need for scintigraphy and emphasised that research should focus on early diagnosis of these injuries or on identification of animals at risk before catastrophic injury occurs (Stover *et al.* 1992 and 1993).

Only a small number of studies have refined the case definition of fatality or musculoskeletal injury to investigate risk factors specific for injuries of the distal limb (Hill *et al.* 2001; Parkin *et al.* 2004b, 2004c and 2005b; Takahashi *et al.* 2004; Perkins *et al.* 2005a and 2005b). The studies by Parkin *et al.* (2004b, 2004c and 2005b) are particularly meaningful as they captured 99% (346/349) of all distal limb fractures and 100% of the lateral condylar fractures that occurred during racing over the study period. An interesting finding in some of these studies was the association of prior exercise with the risk of distal limb injury. Parkin *et al.* (2005b) showed that there was a strong association between horses doing no gallop work during training and the risk of fracture during racing. The risk reduced for horses doing more work, reaching a plateau for horses doing at least 1000 metres (5 furlongs) of gallop work per week. This prompted the authors to suggest that relatively short distances of gallop work during training are protective against fracture in racing. Perkins *et al.* (2005a) showed that the risk of musculoskeletal injury was lower for starts with intermediate cumulative distance raced in the previous 30 days but starts with either shorter or longer accumulated distances were at greater risk of injury.

Other authors have also observed associations between high-speed exercise intensity and musculoskeletal injury (Estberg *et al.* 1995a, 1995b, 1996b, 1998a and 1998b; Cohen *et al.* 2000a and 2000b). However, findings have varied because of regional differences or differences in study design, with Cohen *et al.* (2000a, 2000b) reporting a protective effect of exercise and Estberg *et al.* (1995a, 1995b, 1996b, 1998a and 1998b) observing increased odds of injury with increased exercise. Therefore, the true nature of the association between exercise and injury remains unclear. However, there is evidence to suggest that the most common limb injuries seen in racing (for example, fractures of the metacarpus III and metatarsus III) are due to an ongoing process of microdamage to bone as a result of long-term accumulation of exercise (Pool *et al.* 1990; Stover *et al.* 1992; Riggs *et al.* 1999a, 1999b and 1999c; Hill *et al.* 2001). Riggs *et al.* (1999a, 1999b and 1999c) demonstrated structural changes in the distal condyles of metacarpus III and metatarsus III that implied an adaptive response of bone in horses in race training (Parkin *et al.* 2005b).

Future studies will inevitably need to focus on training and racing exercise regimens in more detail. Studies that rely on retrospective records of racecourse performance are not optimal for investigating effects of high-speed exercise, as exercise that is accumulated in training cannot be evaluated (Perkins 2005). Furthermore, the strength of association between intensity of high-speed exercise and injury is also likely to be sensitive to the period of exposure chosen by the investigator (Perkins 2005). This is a key area for future research as modifiable risk factors such as exercise intensity are likely to be of practical use for racing industries selecting strategies to reduce the risk of racing fatalities.

Due to the nature of injury or fatality data, the epidemiological models described in this chapter have been predominantly hypothesis-generating models. In other words, the models described have identified risk factors that are important markers or flags for future areas of research. For example, research showing that gender (Rooney *et al.* 1983b; Estberg *et al.* 1996a and 1998a; Cohen *et al.* 1997 and 1999b; Hernandez *et al.* 2001; Takahishi *et al.* 2004) and age (Hill *et al.* 1986; Haynes and Robinson *et al.* 1988; Mohammed *et al.* 1991; Estberg *et al.* 1996a and 1998a; Cohen *et al.* 1997, 2000a and 2000b; Wood *et al.* 2000; Pinchbeck *et al.* 2002; Perkins *et al.* 2005a) are risk factors for injury or fatality may indicate that future research should focus on horse-related rather than race-related risk factors. Using multi-level models, it is possible to estimate the variance for each level (for example, horse, race, meet, trainer) to determine the importance of the contribution of factors at that hierarchical level. For example, Pinchbeck *et al.* (2002) reported that most of the variation in models of risk factors for equine falls was at the level of the individual start but that important proportions of the variance were also associated with the horse, race and sire levels. In contrast to the opinions of trainers, jockeys and racecourse clerks, minimal variation was associated with the

level of the trainer. As information regarding trainer-related training and schooling practices would have been difficult to obtain, targeting intervention strategies at this level would have required a major investment of time and money, but would have had only a minimal effect on the rates of falling (Pinchbeck *et al.* 2002). Quantification of variance is therefore another useful means to identify specific target areas of research.

A model that could predict injury or fatality would be most useful for the racing industry as it would enable early detection of starts at high risk and potentially would allow interventions to reduce this risk. To the author's knowledge, only one study of racehorse fatality or injury has assessed the predictive ability of an existing logistic regression model in another racing population (Wood *et al.* 2002). In the latter study, multivariable models for fatality in flat races, hurdle races and steeplechase races were validated on an independent source of British racing data collected the year after the completion of the study. While the flat and hurdle multivariable models fit the new data reasonably well, the steeplechase model failed to fit the independent data source well (Wood *et al.* 2002). The authors speculated that this may have been partially due to the lack of inclusion of detailed variables that represented what happened within the race. Difficulties in model-building may also arise because of the nature of the binary outcome (with a lack of transition states) or because causal factors differ between populations (i.e. risk factors do not necessarily have a causal relationship with the outcome).

As an alternative to a multivariable logistic regression model, Hill *et al.* (2003) developed a stochastic model to predict racehorse injury. Hill *et al.* (2003) categorised horses into subpopulations based on specified states of injury with transition probabilities (between states) that were based on levels of exercise intensity. The model was used to predict distributions of metacarpal condylar fracture and severe suspensory apparatus injury and was validated by using the occurrence of metacarpal condylar fractures reported by racetrack practitioners (Hill *et al.* 2003). Predictions of the development of metacarpal condylar fracture and severe suspensory apparatus injury from this model were relatively accurate for the study population, but overestimated the incidence risk for development of injuries in the southern Californian racehorse population (Hill *et al.* 2003). Models, such as the latter, that can predict injury or fatality may be useful for predicting the effectiveness of intervention strategies and may warrant further attention in future studies of racehorse injury or racing fitness. However, such models are unlikely to be as useful for accurate prediction of racehorse fatality because of the binary nature of the outcome.

Conclusions

Previous international studies have highlighted sufficient regional differences in the risk factors for racehorse fatality to warrant investigation of risk factors specific to fatality in racing in Victoria. Studies in the USA and the UK evolved from early investigations of single risk factors to complex multifactorial approaches that have generated plausible hypotheses on the pathogenesis of different types of racing injuries and fatalities. In comparison, Australian research on racehorse injury and fatality is still in its early stages. The descriptive studies by Bourke (1994 and 1995) and the sophisticated multifactorial studies by Bailey *et al.* (1997a and 1998) generated hypotheses about important factors for severe racing injury in Australia. A specific post mortem survey of racing fatalities in combination with identification of risk factors for specific race types in Victoria are the next logical steps in this area of research before further investigations of narrower focus can take place. A thorough understanding of the complex relationship between factors associated with fatality in flat and jump racing is essential before useful predictive models can be designed and validated.

The present study is the first in Australia to utilise the results of post mortem examinations to describe accurately the causes of death of Thoroughbreds in racing in Victoria and to validate the diagnoses of racecourse veterinarians (Chapter 3). It is also the first in Australia to report the risk of fatality in jump and flat races over an extensive time period.

2. Methodology

The following is a summary of the methodology for each study within this report.

A. Post mortem study

A prospective study was undertaken over a three year period from February 1, 2001 to October 31, 2004 inclusive. Post mortem examinations were performed on all Thoroughbred horses that died during racing or training or were euthanased within 24 hours of a racing or training event because of injuries sustained during that event on any of the four city racecourses in Victoria (Flemington, Moonee Valley, Sandown and Caulfield) over this period. In addition, owners, trainers and/or racetrack veterinarians could elect to submit for post mortem examination horses that died or were euthanased due to injury whilst racing or training on any of the 53 country tracks in Victoria during the study period.

For each horse, a fatality report form was completed at the time of death by the racecourse veterinarian and submitted to Racing Victoria Ltd. for inclusion of data in a national database (*i*-RIS). A copy of the fatality report also accompanied each horse submitted for necropsy. The report included details of the identification of the horse, the race location, track conditions, race type and length, a description of the incident, the date and time of death and a provisional diagnosis. For horses with catastrophic limb injuries, the anatomical location of the injury (i.e. distal/proximal, forelimb/hindlimb and left/right) was also described. Distal limb injuries were defined as those involving the carpus or tarsus or more distal structures. If the veterinarian destroyed the horse, details of the method of euthanasia were also provided.

Horses were transported to the Veterinary Clinical Centre of The University of Melbourne where necropsies were performed by one of four veterinary pathologists within 4-6 hours of death using a standardised protocol. Samples of all viscera, including cardiac conduction tissue and brain, were preserved in 10% neutral buffered formalin. Tissue sections were dehydrated in a graded series of 70-100% alcohol solutions, cleared in xylene and embedded in paraffin. Sections were cut from the paraffin blocks at a thickness of 2 μ m using a microtome, transferred to glass slides and stained with haematoxylin and eosin for light microscopic examination.

Table 3 Protocol for grading pulmonary lesions post mortem in racehorses that died or were euthanased on Victorian racecourses between February 1, 2001 and October 31, 2004

Acute pulmonary oedema	
Grade	Description
+	Patchy dependent areas of wet lung that fail to collapse and exude fluid from minor airways
++	Diffusely wet lungs that do not collapse, stream fluid from the cut surface and have stable foam in minor airways
+++	Diffusely wet and heavy lungs that fail to collapse, stream fluid from the cut surface and have stable foam in both minor and major airways including the primary bronchi and trachea
Acute pulmonary congestion	
Grade	Description
+	Lungs discoloured uniformly or patchily dark pink and heavier than normal
++	Lungs diffusely deep red and heavy
+++	Lungs diffusely plum-coloured, heavy and wet
Acute pulmonary haemorrhage	
Grade	Description
+	Discrete red petechial or ecchymotic parenchymal haemorrhages visible beneath the pleura but no obvious blood staining of any oedema fluid
++	Small confluent areas of red to blue black parenchymal haemorrhage and pink to red fluid in airways
+++	Large confluent areas of deep red to black parenchyma with obvious haemorrhage into airways and blood staining of any oedema fluid
Chronic pulmonary haemorrhage	
Grade	Description
+	Small areas of fibrosis and yellow brown discolouration of the subpleural parenchyma at the dorsocaudal extremity of the diaphragmatic lobes
++	Broad zones of fibrosis, yellow brown discolouration and/or mineralisation involving up to 50% of the subpleural parenchyma of the dorsal region of the diaphragmatic lobes
+++	Fibrosis, yellow brown discolouration and/or mineralisation involving more than 50% of the subpleural parenchyma of the dorsal region of the diaphragmatic lobes

The severity of pulmonary oedema, congestion and haemorrhage was graded as – (absent), + (mild), ++ (moderately severe) or +++ (severe) on the basis of the recorded description of the gross appearance of the lungs at necropsy.

To ensure consistency, one specialist veterinary pathologist was responsible for grading all pulmonary lesions identified in horses submitted over the study period, with the grading based on the recorded description of the gross appearance of the lungs at necropsy and on detailed review of the histological sections of the lungs. The protocol for grading pulmonary lesions is presented in Table 3.

If no mention was made of acute pulmonary oedema, congestion or haemorrhage in the gross description recorded by the pathologist who performed the necropsy examination but lesions of acute oedema, acute congestion or acute haemorrhage were detectable histologically in the lung sections, the relevant grade assigned was modified from absent (-) to mild (+). In horses that had no gross evidence of chronic pulmonary haemorrhage at the time of necropsy, the grade assigned for chronic pulmonary haemorrhage was modified from normal (-) to mild (+) if significant numbers of siderophages (haemosiderin-containing macrophages) were identifiable in histological sections of the lungs within the lumina of alveoli and/or bronchioles and/or within alveolar or lobular septa and/or pleural or perivascular connective tissues.

The total number of horses that died suddenly or were euthanased on Victorian city and country racetracks was obtained from Racing Victoria Ltd. records. Proportional mortality rates were calculated by dividing the number of fatalities referable to a specific cause of death by the total number of fatalities (Dohoo *et al.* 2003). Proportions were compared with two-tailed *p*-values calculated using Fisher's exact test using WINPEPI (<http://www.brixtonhealth.com/>). WINPEPI was also utilised to calculate 95% confidence intervals (Wilson's method) and to compare each proportion with a hypothetical value of 0.5.

Relative risks for submission of sudden death cases versus euthanasia cases from both city and country tracks and the associated heterogeneity χ^2 values were calculated using WINPEPI (Compare 2, version 1.31; <http://www.brixtonhealth.com/>). Horse age was tested for normality using the *sktest* command in Stata 9. If the *p*-value was > 0.05, there was insufficient evidence to conclude that the data were not normally distributed and the mean was reported.

The sensitivity and specificity of the racecourse veterinarians' reports for diagnosis of various conditions were determined using the primary diagnoses made by pathologists as the 'gold standard'. Sensitivity was defined as the proportion of horses with a specific condition that was correctly diagnosed by racecourse veterinarians. Sensitivity was calculated by dividing the number of horses diagnosed with a specific condition by racecourse veterinarians by the total number of horses confirmed by post mortem examination to have that condition. Specificity was defined as the proportion of horses without a specific condition that was correctly identified by racecourse veterinarians to not have that condition. Specificity was calculated by dividing the number of horses identified by racecourse veterinarians as not having the condition of interest by the total number of horses confirmed by post mortem examination not to have that condition.

Comparisons between the cause of fatality recorded in the fatality report and that determined by the post mortem examination were made on the basis of:

- 1 The broad category and subcategory of the cause of death (catastrophic (severe and necessitating euthanasia) musculoskeletal injury: appendicular (pertaining to the limbs or pelvis), axial (pertaining to the vertebral column, skull and/or ribs) or appendicular and axial, versus sudden death: cardiovascular collapse, respiratory failure or other cause).
- 2 The general anatomical location of a catastrophic musculoskeletal injury: distal versus proximal limb, forelimb versus hindlimb, left versus right limb.
- 3 The specific anatomical location of a catastrophic musculoskeletal limb injury (e.g. metacarpus III, metatarsus III).

The calculation of sensitivity and specificity in this study assumed that racecourse veterinarians specifically ruled out all other injuries or primary causes of death when the diagnosis was recorded on the fatality report. When a veterinarian's designation of the cause of death did not concur with that of the pathologist, the records were classified as misdiagnoses. Misdiagnoses included errors of omission (e.g. the veterinarian failed to report whether a musculoskeletal injury involved a left versus right limb or fore- versus hindlimb) and errors of diagnosis (e.g. metacarpus III versus sesamoid bone fracture, cardiovascular failure versus exsanguination). Additionally, if a veterinarian recorded a perceived primary cause of death in the fatality report but neglected to report other injuries considered to be part of the primary cause of death by the pathologist, the record was classified as a misdiagnosis. Wilson's 95% confidence intervals were calculated for proportions (Abramson 2004).

B. Risk of fatality and causes of death of Thoroughbred horses associated with racing in Victoria, Australia between 1989 and 2004

Study population

The study population comprised all horses participating in flat and jump races (hurdle and steeple races combined) on all four city racecourses and all 53 country racecourses in Victoria, Australia between August 1, 1989 and July 31, 2004. Each racing year in Australia begins on August 1 and ends on July 31.

Case definition and identification

A racing fatality was considered to have occurred whenever a horse died during or immediately after a race or was euthanased immediately after or within 24 hours of a race as a consequence of an injury sustained during that race. Horses that did not enter the starting gate were not included as cases in this study.

All fatalities were identified from an industry database (Racing Victoria Ltd.) compiled from fatality reports submitted by stewards and racecourse veterinarians. Prior to the 2001-2002 racing year, there was an informal policy of reporting of fatalities on city and country racecourses by veterinarians and stewards to Racing Victoria Ltd. The senior veterinary steward at Racing Victoria Ltd. confirmed the details of diagnoses made in fatality cases before they were recorded in the database. Compliance with reporting of fatalities on city racecourses was consistently high over the 15 year study period as an official Racing Victoria Ltd. veterinarian attended every city race meeting (Racing Victoria Ltd., J. McCaffrey, *personal communication*). In contrast, while country race meetings were attended by one or sometimes two veterinarians, official Racing Victoria Ltd. veterinarians were not in attendance at all of these meetings and as a consequence there may not have been 100% compliance in completing and/or submitting fatality reports. Moreover, although every country TAB (on- and off-course totalisator betting) meeting was attended by Racing Victoria stewards, prior to 2001-2002 non-TAB (on-course totalisator betting only) country meetings and picnic races were controlled usually by regionally based stewards.

Since the racing year 2001-2002, Racing Victoria Ltd. mandated that a formal fatality report be completed for all horses that died or were euthanased as a consequence of catastrophic injuries sustained during racing in Victoria and that two veterinarians attend all country TAB meetings, one of these being an official RVL veterinarian (Racing Victoria Ltd., J. McCaffrey, *personal communication*). Details in the fatality report included the name, sex and age of the horse, the racecourse, race-distance, track rating, type of race (flat, hurdle or steeplechase) and a brief description of the cause of death or the reason for euthanasia. After February 1, 2001, Racing Victoria Ltd. also made it compulsory for post mortem examinations to be performed on horses that died or were euthanised while racing on any of the four city racecourses in Victoria.

The identity of each fatality was considered to be unique and valid for inclusion in the study if the information relating to the date and racetrack location of the case (fatality) start, the race number and the horse name were identical in a commercial racing database (Australian Associated Press Pty. Ltd. database).

Start identification

A start occurred whenever a horse entered the starting gate for a race. Starts included those in which horses sustained injuries at the starting gate and those in which horses were pulled up or lost riders during the race. An individual horse could contribute more than one start over the study period. The numbers of starts were obtained from a commercial racing database (Australian Associated Press Pty. Ltd.). Starts in barrier trials and 52 jump-out race starts for which the location of the racecourse was not recorded were excluded from analyses. A barrier trial or jump out trial is a test of the ability of a horse to enter, stand and jump out of the barriers or starting gates and is officially observed by racing stewards.

Statistical analysis

The risk of fatality was expressed as the number of fatalities per 1000 starts and was calculated for flat and jump races on all tracks in Victoria between August 1, 1989 and July 31, 2004 for each racing year and over the entire 15 year period. Risks of fatality were compared between groups of starts using risk ratios and associated 95% confidence intervals. Risk ratios can vary from zero to high positive values. When the risks in two groups are identical, the risk ratio is one and no association exists. When the risk is greater in the exposed group relative to the referent group, the risk ratio is greater than one; when the risk is lower in the exposed group than in the referent group, the risk ratio is less than one. Where the 95% confidence interval for the risk ratio does not include one, the risk ratio estimate differs significantly ($p < 0.05$) from one (i.e. from no association).

Because of our particular interest in jump racing, the contribution of jump racing to the overall risk was estimated in two ways. The overall risk of fatality in the population (i.e. the risk across starts of all types) that could be attributed to jump racing (the population attributable risk or PAR for jump starts) was calculated by subtracting the baseline risk (the risk of fatality in flat starts) from the overall risk in the population (the risk of fatality in flat and jump starts combined) (Dohoo *et al.* 2003). The proportion of overall risk that was attributable to jump racing (the population attributable fraction or PAF for jump starts) was calculated by dividing the PAR for jump starts by the proportion of all starts (jump and flat races combined) that ended in fatality (Dohoo *et al.* 2003). These PAR and PAF estimates quantify the effects on population risk should one or a combination of unspecified strategies be successful in reducing the risk in jump starts to that in flat starts.

The risk of fatality (the number of fatalities per 1000 starts), 95% confidence intervals (CI) using Wilson's method (Wilson 1927), risk ratios (RR) and their 95% CI were calculated using Stata 9.1 statistical software (StataCorp, College Station, TX.). Using the same software, Poisson regression with robust standard errors (Zou 2004) was performed to estimate the risk ratio (McNutt *et al.* 2003) per extra year of the study from 1989 to 2004. The *aflogit* command using the *cc* option and Stata 9.1 were used to calculate the PAF and its 95% confidence interval via the method described in Greenland and Drescher (1993).

C Risk factors for Thoroughbred racehorse fatality in flat and jump starts in Victoria (1989-2004)

Potential risk factors for fatality in flat and jump starts in Victoria between August 1, 1989 and July 31, 2004 were assessed using a retrospective case-control study (with 283 cases and 3307 controls in the flat study and 191 cases and 2324 controls in the jump study). The studies were conducted at the start level (where a start represents a horse entering the starting gate for a race) rather than at the level of the horse (starter) or race because this allowed inclusion of start-level risk factors along with horse-, race- and track-level risk factors.

Selection of cases and controls

A case start was defined as a start in a flat or jump race or official flat or jump trial that ended in fatality. A racing fatality was deemed to have occurred if a horse died suddenly (i.e. without veterinary intervention) during or immediately after the event or was euthanased within 24 hours of the event as a consequence of injury sustained in that event.

Data on flat racing starts between August 1, 1989 and July 31, 2004 were obtained from three major sources: a Racing Victoria Ltd. (RVL) database, a Racing Information Services Australia Pty. Ltd. web-based database known as *i*-RIS and an Australian Associated Press Pty. Ltd. (AAP) database. The RVL database was compiled from fatality reports submitted by racecourse veterinarians and stewards, and provided information regarding the number and specific details of all racehorse fatalities sustained on city or country racecourses in Victoria during the study period. The database included the identity of the horse, the race type, race distance, date and location of the race and the reason provided by the racecourse veterinarian or steward for euthanasia or sudden death.

Prior to enrolment in the study, all identified fatality cases were verified using the *i*-RIS and AAP databases. The *i*-RIS database contained data on all horses that had raced at least once in Victoria. Horse performance histories recorded in the AAP database were used to validate the identity of each horse recorded as a fatality in the *i*-RIS and RVL databases. Racing fatalities reported to RVL were considered to be unique and valid for inclusion in the study if the information relating to the date and racetrack location of the case start, race type, race number and horse name were identical in the databases. Racing fatalities for which discrepancies in any of these details were identified were excluded from the study.

The racing year in Australia begins on August 1 and ends on July 31. Between August 1, 1989 and July 31, 2004, study starts in flat and jump races were recorded at four city racecourses and 62 country racecourses in Victoria. Over the 15 year period, 316 flat racing fatalities were reported to RVL. Of these, 33 were subsequently excluded (32 due to discrepancies in horse identity, race date, race location, race number and/or performance history and one due to a discrepancy in race type). Therefore, 283 flat cases were included in the study. In the jump study, 198 racing fatalities were reported to RVL. Of these, seven were excluded due to discrepancies in horse identity, race date, race location and/or race number. Therefore, 191 cases were enrolled in the jump study.

A control start could be a case horse in a start prior to its injury, a start for a non-case horse, or multiple starts for the same non-case horse or case horse prior to injury. Control starts were selected from the AAP database by simple random sampling of all flat starts in the study period that were not case starts. Starts were selected randomly without replacement (i.e. an individual start could be selected only once) from all starts that met the start selection criteria and all eligible starts had an equal probability of selection.

This study design meant that multiple starts by any one horse were eligible for selection as separate control starts (including previous starts by any horse that had ultimately become a case) and that more than one control start could be selected from the same race. If a horse was scratched prior to the commencement of a race, the start was deemed ineligible for selection. Starts in which a horse started but failed to complete the race were still eligible for selection.

As a rule of thumb, useful increases in statistical power are obtained by selecting three to four controls per case; higher ratios yield only marginal increases in power (Dohoo *et al.* 2003). In this study, approximately ten controls were selected for each case because the data for each control start were easy to obtain and the costs of enrolling more controls were small. After exclusion of ineligible cases and controls, the ratio of controls to cases was approximately 12:1 in both studies.

Risk factors

Potential risk factors were identified from the literature and from *a priori* hypotheses. A causal web diagram was also developed based on biologically plausible hypotheses (supported by the literature) for racing fatalities. Risk factors associated with the horse, its prior racing history, the race, the jockey and the track were considered. The AAP database was used as the major source of performance histories for all horses with starts selected as cases or control starts.

Gender of the horse was selected at the level of the start and was categorised as entire male, entire female or gelding (castrated male). Track rating (going of the track) was used to describe the racing surface (fast: very firm surface, good: firm surface, dead: track with give in the ground, slow: rain-affected, heavy: very rain-affected) (Racing Victoria Ltd. 2006). Speed of the race was calculated by dividing the winner's time (in seconds) by the race length (in metres). Calendar age was calculated by subtracting each horse's actual birth date from the date of the case or control start. Racing age, based on the convention in the southern hemisphere that August 1 is the official birthday of the horse, was not used. Racing career length (expressed in years) was determined by subtracting the date of the first official start from the date of the case or control start. Information about the number of prior starts (in flat and jump races) and the distance raced (in metres) was also refined to quantify prior racing history during different time periods of interest (14, 30, 60 and 90 days prior to the case or control start and various combinations of these periods).

A total of 115 variables for each start (four horse-related variables, 87 prior racing history-related variables, 16 race-related variables, five jockey-related variables and three track-related variables) were available for analysis. Details of the study starts and associated risk factor data were downloaded into an Access database (Microsoft® Access 2003).

Descriptive statistics

Descriptive statistics (mean, median, minimum and maximum values) were generated for each continuous variable. The 'best fit' of the variable (continuous or categorical) was determined by graphical assessment of the relationship between the log odds of the outcome by categories of an independent variable. A second assessment of linearity was made of the continuous variables in the final model by plotting fractional polynomials (Royston *et al.* 1999). If the relationship was non-linear, binary, polytomous categorical (quartiles or quintiles) or quadratic and cubic terms were considered at the univariable and multivariable levels (Dohoo *et al.* 2003). Continuous variables were centred by subtracting the mean value from each value of the variable and included with quadratic and cubic terms (Dohoo *et al.* 2003). Continuous variables were not centred if they were not included with a quadratic or cubic term. Nominal and ordinal categorical variables were numerically coded sequentially, with a zero being assigned to the reference group. Racing year was coded from zero (1989-1990) to 14 (2003-2004).

Model building

Potential risk factors were screened using univariable logistic regression. A likelihood ratio test p -value of ≤ 0.25 was used as a criterion for entry of a variable into subsequent single level, multivariable logistic regression model building. Variables were selected for inclusion in the model building process based on whether they were biologically plausible or supported by the literature. Parameters were estimated by a backward stepwise model building process. Variables were retained in the multivariable model if likelihood ratio test p -values were < 0.05 when establishing the model involving main effects (Hosmer and Lemeshow 2000). The Wald test p -value was used when comparing categories with the reference category.

Potential confounders were selected from those variables with a p -value ≤ 0.25 in the univariable analyses that were not included in the final model after the backward stepwise process of model building. Effects of each potential confounder on the estimates for variables in the final model were assessed by fitting each one at a time into the final model. If addition of the potentially confounding variable altered odds ratios for variables in the final model by more than 20% (Dohoo *et al.* 2003), confounding was considered to be present, the confounder was retained in the final model and adjusted odds ratios were reported for variables in the final model. Variables that were not significant in the univariable analysis were not routinely assessed for confounding in the final model unless on the basis of an *a priori* hypothesis. In addition, biologically plausible interactions between variables in the final model were identified and interaction terms were created and assessed. The small numbers of fatalities that occurred each racing year in flat races in Victoria meant that a long study period was desirable in order to achieve adequate study power to detect important risk factors if they were present. The prolonged time frame meant that it was likely that the association between the risk factors (exposures) and the outcome may have varied over the course of the study (for example, with changes in training methods, track design or racing regulations). An assumption of the analytical techniques employed in the study is that the associations between the risk factors and the outcome of interest are constant. The effect of time on the exposures of interest was therefore investigated by creating interaction terms between various exposures and racing year.

Co linearity of continuous variables was assessed on the basis of *a priori* hypotheses and confirmed using Spearman's rank correlation coefficients. Clustering of starts was investigated within the horse, trainer, jockey, meet and track. Residual intra-class correlation coefficients (ρ) and variance inflation factors (VIF) were estimated from random effects logistic regression models for each level of clustering using a latent variable approach (Snijders and Bosker 1999) and the variables in the final model.

Fit of the model and regression diagnostics

The fit of the final multivariable model was assessed using the Hosmer-Lemeshow goodness-of-fit test (Hosmer and Lemeshow 2000). Regression diagnostics were performed on the model. Covariate patterns with the greatest leverage, delta betas, delta χ^2 and delta deviance values were identified. The individuals within these covariate patterns were then removed from the model and the change in the value of the coefficients was reassessed (Hosmer and Lemeshow 2000). Sensitivity and specificity were calculated to measure the ability of the model to correctly classify individual starts. The predictive ability of the model was determined by generating a receiver operating characteristic (ROC) curve.

Statistical analysis

All statistical analyses in these chapters were performed using Stata 9.1 and 9.2 statistical software (Stata Statistical Software: 2005, StataCorp, College Station, TX, USA). WINPEPI (PEPI-for-Windows, COMPARE 2, version 1.45) (Abramson 2004) was used to calculate statistical power.

3. Post mortem study of Thoroughbred racing fatalities in Victoria (2001 – 2004)

Introduction

Death or catastrophic injury of horses during training or racing is an important cause of economic loss to the Australian racing industry. A fatality can be categorised as either a euthanasia (if a horse is destroyed after incurring a catastrophic injury whilst racing or training) or as a sudden death (a death occurring without veterinary intervention during or within minutes of finishing a race or training event).

Many studies have described the proportions of racehorse deaths attributable to different causes (for example, Vaughan and Mason 1976; Mohammed *et al.* 1991; Wilson *et al.* 1993; Bourke 1994; Peloso *et al.* 1994; Johnson *et al.* 1994a and 1994b; Macdonald and Toms 1995; McKee 1995; Mizuno 1996; Parkin *et al.* 2004a). However, in the majority of such studies, the cause of fatality used for analysis was that recorded by racecourse veterinarians or stewards, notwithstanding that such provisional diagnoses may be inaccurate (Vaughan and Mason 1976; Palmer 1986; Suann 1992; Wilson *et al.* 1993, 1996a and 1996b; Bathe 1994; Bourke 1994; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995; Mizuno 1996; Cohen *et al.* 1999b; Williams *et al.* 2001; Verheyen *et al.* 2004). Other studies have utilised post mortem examinations to determine the cause of fatality (Platt 1982; Gelberg *et al.* 1985; Gunson *et al.* 1988; Krook and Maylin 1988; Pool and Meagher 1990; Johnson *et al.* 1994a and 1994b). The most comprehensive post mortem study of racehorse fatalities has been underway at the University of California at Davis since 1990 (Stover *et al.* 1992, 1993 and 1994; Johnson *et al.* 1994a and 1994b). This program was established in conjunction with the California Horse Racing Board and the California Veterinary Diagnostic Laboratory System to ensure compulsory post mortem examination of every horse that dies during racing or training in California on racetracks under the jurisdiction of the California Horse Racing Board. This was the first large-scale prospective study of racehorse fatalities in the world, and it enabled collection of reliable data about the causes of death of racehorses during racing and training.

In February 2001, the Victorian principal racing authority, Racing Victoria Ltd., introduced mandatory post mortem examinations for all racing and training fatalities on racetracks in the metropolitan area of Melbourne. This ruling allowed the first systematic Australian study of racetrack fatalities through comprehensive pathological examination of the horses involved. Previous Australian studies of racecourse fatalities have relied upon the unverified observations of racecourse veterinarians to categorise the injuries responsible for death or euthanasia. The primary objectives of the study were to thoroughly document the range of catastrophic musculoskeletal injuries sustained in racing and training and to determine the causes of sudden death of horses on racecourses in Victoria. A secondary objective was to determine the proportion of racetrack fatalities that was due to sudden death and to establish whether sampling from a subset of fatalities can affect the estimated proportional mortality due to sudden death. A final aim was to compare the principal diagnoses made by pathologists and racecourse veterinarians for all horses submitted for post mortem examination over the study period to determine the reliability of Racing Victoria Ltd. records that are based on fatality reports submitted by racecourse veterinarians. The latter component of the study was considered important to provide a measure of the accuracy and limitations of archived racing reports for researchers who will utilise such data in the future.

Results

Over the study period, 180 deaths occurred during racing or training on racecourses throughout Victoria. Of these, 63 deaths occurred on city tracks and 117 deaths occurred on country tracks. Euthanasia due to catastrophic musculoskeletal or other injury accounted for 74% (134/180, 95% confidence interval (CI) 68-80%) of all fatalities over the study period. Of the fatalities resulting from euthanasia, 36% (48/134 CI 28-44%) occurred on city tracks and 64.2% (86/134, CI 56-72%) occurred on country tracks. Sudden death accounted for 26% (46/180, CI 2-32%) of all deaths during the study period. Of the sudden deaths reported, 33% (15/46, CI 21-47%) occurred on city tracks and 67% (31/46, CI 53-79%) on country tracks. Over the three years of the study, post mortem examinations were performed on 77/180 horses (43%, CI 36-50%), with 61 (79%, CI 69-87%) of all cadavers being submitted from city racecourses and 16 (21%, CI 13-31%) being submitted from country racecourses. The horses submitted for necropsy represented 97% (61/63, CI 89-99%) of all city fatalities and 14% (16/117, CI 9-21%) of all country fatalities.

Of the 77 fatality cases submitted for post mortem examination, 40% (31/77, CI 30-51%) were sustained in flat races, 25% (19/77, CI 16-35%) in jump races (10 hurdle and 9 steeplechase races), 3% (2/77, CI 1-9%) in hurdle qualification trials and 32% (25/77, CI 23-44%) in training or non-raceday events. Of the 77 horses, 52 were euthanased (68%, CI 57-77%) and the remaining 25 horses died suddenly without veterinary intervention (Table 4). Females accounted for 21% (16/77, CI 13-31%) of the horses. Most of the affected males were geldings (56/61, 92%, CI 82-96%).

The overall distribution of horse age and horse age stratified by gender and race type were normal (p -values > 0.05). The age of affected horses ranged from two to ten years with a mean of 5.2 years. The mean age of females was four years (range two to eight years) and the mean age of males was 5.5 years (range two to ten years). The mean age of horses dying in flat races was 4.8 years whereas those dying in jump races had a mean age of 7.1 years, in track events 4.4 years, and in barrier trials four years.

Table 4 Summary of categorical causes of fatality in Thoroughbred horses that died or were euthanised on Victorian racecourses between February 1 2001 and October 31 2004 as determined by post mortem examination

Category of cause of death	Euthanasia	Sudden death
Musculoskeletal		
Appendicular skeleton/soft tissue	44	1
Axial skeleton	7	2
Appendicular/axial skeleton	1	0
Muscle injury	0	2
Non-musculoskeletal		
Acute pulmonary lesions	0	17
Exsanguination	0	2
Unknown	0	1
Total	52	25

Catastrophic musculoskeletal injury

Catastrophic musculoskeletal injury was the cause of death or euthanasia of 57/77 of the horses (74%, CI 63-83%). Most of these horses (52/57, 91%, CI 81-96%) were euthanased. Of the 57 horses with musculoskeletal injuries, 45 (79%, 67-88%) sustained catastrophic injury to the appendicular skeleton or associated soft tissues, nine (16%, CI 9-25%) sustained cranial or vertebral fractures, one horse (2%, CI 0.3-9%) sustained multiple rib and proximal forelimb fractures and a brain laceration during a steeplechase, one horse (2%, CI 0.3-9%) died suddenly during a flat race due to massive rupture of the paravertebral musculature and one horse (2%, CI 0.3-9%) died suddenly during a flat race due to severe haemorrhage into multiple skeletal muscles. Of the 45 musculoskeletal limb injuries, 20 (44%, CI 31-59%) occurred in flat races, 11 (24%, CI 14-39%) in jump races, 12 (27%, CI 16-41%) in training sessions and two (4%, CI 1-15%) in hurdles qualifying trials. Of nine cranial or vertebral fractures, six (67%, CI 35-88%) were sustained in jump races, two (22%, CI 6-55%) in flat races and one (11%, CI 2-44%) in a training session. Cranial or vertebral fracture accounted for a greater proportion of fatalities in jump races (6/19, 32%, CI 15-54%) than in flat races (2/31, 6%, CI 2-21%) (p -value = 0.04) or training sessions (1/25, 4%, CI 1-20%) (p -value = 0.03).

Of the 45 horses with appendicular skeletal or soft tissue injuries, 40 (89%, CI 77-95%) sustained injuries to a single limb and five (11%, CI 5-24%) had concurrent injuries to two limbs. One horse sustained injuries to both the right fore- and right hindlimb. Two horses sustained catastrophic injuries to both forelimbs and two horses sustained injuries to both hindlimbs. In the 40 horses with catastrophic injury to a single limb, the most common injury involved the distal limb (30/40, 75%, CI 60-86%) and a forelimb (32/40, 80%, CI 65-90%) was more frequently affected than a hindlimb. In the same subgroup of horses, 23 animals sustained an injury to a left limb (23/40, 58%, CI 42-72%) and 17 horses (17/40, 43%, CI 29-58%) sustained an injury to a right limb. The proportion of horses with a single catastrophic left limb injury was not significantly different from the proportion of horses with a single catastrophic right limb injury (p -value = 0.43).

The anatomical site of the 45 catastrophic limb injuries is described in Table 5. Fracture of metacarpus III (MC III) or metatarsus III (MT III) was the most frequently observed distal limb injury (12/45, 27%, CI 16-41%), followed by fractures of the proximal sesamoid bones (6/45, 13%, CI 6-26%) and multiple fractures of the metacarpus III or metatarsus III and proximal sesamoids (5/45, 11%, 5-24%). Of proximal limb injuries, humeral fractures (6/45, 13%, CI 6-26%) were the most common.

Table 5 Summary of anatomical location of catastrophic limb injuries sustained by racehorses on Victorian racecourses between February 1, 2001 and October 31, 2004 as determined by post mortem examination

Catastrophic limb injuries (n = 45)	Number of horses
Distal limb injuries	
Metacarpus III/metatarsus III	12
Metacarpus III/metatarsus III + sesamoids	5
Sesamoids	6
Metacarpus III/metatarsus III + sesamoids + phalanx I	4
Carpus	2
Tendon/ligament rupture	2
Proximal limb injuries	
Humerus	3
Humerus + skull	1
Humerus + scapula	2
Radius	1
Radius + ulna	1
Tibia	1
Pelvis	2
Pelvis + femurs	1
Distal and proximal injuries	
Humerus + radius + carpus	1
Humerus + scapula + phalanx I	1

Non-musculoskeletal sudden deaths

Twenty horses in the study (20/77, 26%, CI 18-37%) died suddenly for reasons other than musculoskeletal injury. Seventeen horses died (six in flat races, one in a jump race and 10 during training sessions) with acute pulmonary oedema, congestion and/or haemorrhage, two horses (one in a flat race and one in a track session) exsanguinated into the peritoneal cavity and one horse died (in a steeplechase) with no significant lesions detectable.

In the 17 horses with acute pulmonary lesions, the severity of the lesions varied (Table 6). Fifteen horses had severe acute pulmonary haemorrhage and 15 had moderate to severe acute pulmonary oedema. Of the six horses with severe acute pulmonary oedema, four also had severe acute pulmonary haemorrhage. In two horses, severe acute pulmonary oedema and congestion were the predominant findings.

In all horses that died suddenly with acute pulmonary oedema, congestion and/or haemorrhage, the distribution of pulmonary oedema and congestion was diffuse. In most of the horses with severe acute pulmonary haemorrhage (13/15), the haemorrhage was most severe in the dorsal/dorsocaudal aspects of both the right and left diaphragmatic lung lobes. In eleven horses, there was evidence of previous episodes of pulmonary haemorrhage. Gross evidence of chronic haemorrhage included fibrosis and yellow-brown discolouration of parenchyma in the dorsocaudal or dorsal aspects of the diaphragmatic lung lobes. Affected areas of lung were palpably firmer than normal and did not collapse to the same degree as unaffected areas. Frequently, these parenchymal lesions were associated with plaques of fibrosis with or without haemosiderosis involving the visceral pleura of the dorsal aspect of the diaphragmatic lobes. A histological diagnosis of chronic pulmonary haemorrhage was made if siderophages were identifiable within the lumina of alveoli and/or bronchioles and/or within alveolar or lobular septa and/or pleural or perivascular connective tissues. Pulmonary haemosiderosis was associated with a variable degree of interstitial and peribronchiolar

fibrosis and a variable increase in the number of peribronchiolar arterial branches. In a minority of horses, there was mild multifocal chronic active lymphocytic or eosinophilic peribronchiolitis in the affected areas of lung.

Table 6 Summary of post mortem findings in 17 horses that died suddenly with acute pulmonary lesions whilst racing or training on Victorian racecourses between February 1, 2001 and October 31, 2004 (pulmonary lesions are classified as mild (+), moderate (++), severe (+++) or absent (-))

Horse	Sex	Age (years)	Acute pulmonary congestion	Acute pulmonary oedema	Acute pulmonary haemorrhage	Chronic pulmonary haemorrhage	Myocardial lesions	Central nervous system lesions
1	Male	3	+++	+++	+	+	+	+++
2	Male	2	+++	+++	+	-	+	-
3	Female	5	+++	+++	+++	++	-	-
4	Male	4	+++	+++	+++	-	-	-
5	Male	3	+++	+++	+++	+	-	-
6	Female	4	+++	+++	+++	+	+	-
7	Female	3	+++	++	+++	-	-	-
8	Male	2	+++	++	+++	-	+	-
9	Male	7	+++	++	+++	++	-	-
10	Male	5	+++	++	+++	+	-	-
11	Male	5	+++	++	+++	+	-	-
12	Male	4	+++	++	+++	-	-	-
13	Male	11	+++	++	+++	+	-	-
14	Male	7	++	++	+++	+	-	-
15	Male	6	++	++	+++	++	-	-
16	Female	4	+++	+	+++	-	-	-
17	Male	5	++	+	+++	++	-	-

Only one of the horses (#1) with acute pulmonary lesions had a grossly detectable cardiac lesion: a small right ventricular myocardial scar that was considered incidental. Three additional horses had microscopic cardiac lesions. One of these (#2) had minimal foci of peracute subendocardial myocardial necrosis in the left ventricle. Another (#6) had mild lymphocytic inflammation involving the atrial myocardium, the base of the aorta and the bundle of His and right bundle branch. In the remaining horse (#8), there was severe, multifocal to locally extensive, acute, subacute and chronic myocardial necrosis. Horse (#1) also had a deep laceration of the parietal cerebral cortex caused by a cranial depression fracture sustained terminally as it fell. Although acute brain injury may have triggered neurogenic pulmonary oedema in this animal, the severity of the pulmonary oedema suggested that the latter preceded the terminal head injury.

The mean age of the 17 horses that died suddenly with pulmonary lesions was 4.7 years (range 2-11 years).

Assessment of sampling bias

In the target racing population between February 1, 2001 and October 31, 2004, the proportional mortality rates due to sudden death were comparable on city and country tracks (24%, CI 15-36%, 15/63 and 26%, CI 19-35%, 31/117, respectively). However, amongst horses submitted for post mortem examination (Table 7), the proportional mortality rate due to sudden death was substantially higher for horses submitted from country tracks (63%, CI 39-82%, 10/16) than for those submitted from city tracks (25%, CI 16-37%, 15/61) (p -value = 0.007). The relative risk of sudden death cases from city tracks being submitted for post mortem examination compared with euthanasia cases was 1.04 (95% CI 0.98-1.11). The corresponding relative risk in submissions from country tracks was 4.62 (95% CI 1.83-11.66). There was a significant difference between these two relative risks (heterogeneity $\chi^2 = 9.9$, one degree of freedom, p -value = 0.002).

Table 7 Numbers of Thoroughbred horses submitted for post mortem examination from Victorian racetracks between February 1, 2001 and October 31, 2004, by cause of death and location of racetrack

Cause of death	Total deaths (n = 77)	City deaths (n = 61)	Country deaths (n = 16)
Euthanasia			
Musculoskeletal	52 (68%)	46 (75%)	6 (38%)
Sudden death			
Pulmonary oedema, congestion and haemorrhage	17	11	6
Other	8	4	4

Validation of fatality reports

Sensitivity and specificity estimates for diagnoses made by racecourse veterinarians and recorded in fatality reports for the 77 horses that were submitted for post mortem examination between February 1, 2001 and October 31, 2004 are reported in Table 8. All but one of the 45 catastrophic limb injuries was correctly diagnosed as such (sensitivity = 44/45 or 97.8%, CI 88.4-99.6%). The incorrect diagnosis resulted from misclassification of a horse that sustained a pelvic fracture and secondary fatal haemoperitoneum (pathology diagnosis) as a sudden death (veterinary diagnosis). For the 45 catastrophic limb injuries, veterinarians correctly recorded the distal and/or proximal location in 91.1% (41/45, CI 79.3-96.5%), the fore- and/or hindlimb location in 82.2% (37/45, CI 68.7-90.7%) and left and/or right location in 86.7% (39/45, CI 73.8-93.7%) of horses. The specificity of diagnoses of appendicular skeletal or soft tissue injury was 100% (CI 89.3-100).

Sensitivity of diagnosis of cranial or vertebral injury was low (5/9 or 55.6%, CI 26.7-81.1%) (Table 8). Two of the nine horses that died because of cranial or vertebral injury were misclassified as sudden deaths. Of the fatality reports for the other seven horses, five recorded a skull or vertebral injury and two only referred to neurological signs and did not specify the location of injury. The specificity of diagnoses of cranial or vertebral injury was 100% (CI 94.7-100%).

Although all 25 cases of sudden death (i.e. all fatalities not requiring euthanasia) were correctly identified as sudden deaths in the fatality reports (sensitivity 100%, CI 86.7-100%), only one report correctly identified the specific cause of death. One other report was deemed to be accurate in *not* specifying the cause of sudden death because no cause of death could be determined by pathologists on post mortem examination. For the 19 horses that died suddenly with acute pulmonary lesions or acute haemoperitoneum, only three fatality reports specified the cause of sudden death. Two veterinarians incorrectly diagnosed aneurysms and one veterinarian correctly diagnosed exercise-induced pulmonary haemorrhage on the basis of blood appearing at the nostrils of the horse. In 16 of these 19 cases (84.2%, CI 62.4-94.5%), racecourse veterinarians provided only a vague diagnosis of 'sudden death syndrome' and/or gave details such as 'collapsed and died' or 'blanched mucous membranes'.

The specificity of the diagnosis of sudden death due to cardiovascular or respiratory failure was high (57/58 or 98.3%, CI 90.9-99.7%). One horse that was misdiagnosed as dying suddenly due to cardiovascular collapse died as a result of acute muscle injury. The specificity of the diagnosis of sudden death due to an unknown cause was 56/76 or 73.7% (CI 62.8-82.3%). Twenty horses were diagnosed by racecourse veterinarians as having died suddenly due to unknown causes (sudden death syndrome). Of these, post mortem examination showed that one horse had died suddenly due to laceration of a blood vessel as a result of a pelvic fracture, two horses had died suddenly as a result of cranial or vertebral injury, two horses had exsanguinated, one horse had died suddenly as a result of extensive muscle haemorrhage and the remaining fourteen horses died as a result of cardiovascular or respiratory failure.

The sensitivity and specificity of veterinary diagnoses of catastrophic limb injuries involving a single anatomical site (n = 29) are reported in Table 9. Of the distal limb injuries, the sensitivity of diagnoses of metacarpus III and metatarsus III fractures was 66.7% (CI 39.1-86.2) and that of proximal sesamoid fractures was 50.0% (CI 18.8-81.2). The sensitivity estimates for diagnoses of fractures of the carpus (100%), humerus (66.7%), radius (100%), tibia (0%) and pelvis (50%) and of tendon/ligament rupture (50%) were imprecise due to the small numbers of cases in each group.

Failure to achieve 100% sensitivity in the diagnosis of catastrophic limb injuries was because veterinarians did not indicate the site of the injury, reported only a general location (for example, fetlock, leg or elbow) or reported the incorrect location. If a more specific location had been provided instead of 'fetlock' or 'leg', the sensitivity of diagnoses for single site distal limb injuries may have improved from 63.6% (14/22, CI 43.0-80.3%) to 90.9% (20/22, CI 72.2-97.5%) and the sensitivity of diagnoses for single site proximal limb injuries may have improved from 57.1% (4/7, CI 25.1-84.2%) to 85.7% (6/7, CI 48.7-97.4%). There was no significant difference between the proportions of diagnoses that were incorrect for proximal and distal limbs (p -value = 1.0).

The specificity of veterinarians' diagnoses of catastrophic limb injuries was 100% for all injury types except fracture of the proximal sesamoid bones. A fracture of the metacarpus III was incorrectly diagnosed as a fracture of the proximal sesamoids.

Table 8 Sensitivity and specificity of diagnoses made by racecourse veterinarians for various conditions in horses that were submitted for post mortem examination between February 1, 2001 and October 31, 2004

Diagnosis from post mortem examination	Sensitivity (% and 95% confidence interval)	Specificity (% and 95% confidence interval)
Musculoskeletal injury		
Appendicular skeleton/soft tissue	44/45 (97.8%, 88.4-99.6)	32/32 (100%, 89.3-100)
Axial skeleton	5/9 (55.6%, 26.7-81.1)	68/68 (100%, 94.7-100)
Appendicular and axial skeleton	1/1 (100%)	76/76 (100%, 95.2-100)
Non-musculoskeletal injury		
Sudden death due to acute extensive muscle injury	0/2 (0%, 0.0-65.7)	75/75 (100%, 95.1-100)
Sudden death due to cardiovascular or respiratory pathology	1/19 (5.3%, 1.0-25.8)	57/58 (98.3%, 90.9-99.7)
Sudden death (unknown cause)	1/1 (100%)	56/76 (73.7%, 62.8-82.3)

For proportions with small sample sizes ($n < 2$), 95% confidence intervals could not be generated

For catastrophic limb injuries involving multiple sites ($n = 16$), two reports used general terms such as 'fetlock' or 'leg'. In horses with concurrent fractures of the metacarpus III or metatarsus III and the proximal sesamoids ($n = 5$), and concurrent fractures of metacarpus III or metatarsus III, the proximal sesamoids and phalanx I ($n = 4$), racecourse veterinarians reported at least one bone correctly but none correctly identified all fractured bones. All of the multi-site limb injuries involving a proximal site ($n = 7$) were incorrectly diagnosed. Concurrent fractures of the radius and ulna were reported as elbow and shoulder fractures, concurrent fractures of the humerus and skull were reported as a scapular fracture, concurrent fractures of the humerus and scapula were broadly referred to as a shoulder fracture, concurrent fractures of the humerus, radius and carpus were described as an elbow fracture, concurrent fractures of the humerus, scapula and phalanx I were reported as humerus and fetlock fractures and concurrent fractures of the femurs and pelvis were reported as a stifle fracture.

Table 9 Sensitivity and specificity of diagnoses made by racecourse veterinarians regarding the specific anatomical location of catastrophic limb injuries at single anatomical sites (n = 29) in Thoroughbreds racing in Victoria between February 1, 2001 and October 31, 2004

Anatomical location of catastrophic limb injury	Sensitivity (% and 95% confidence interval)	Specificity (% and 95% confidence interval)
Distal limb injuries		
Carpus	2/2 (100%, 34.2-100)	27/27 (100%, 87.5-100)
Metacarpus III or metatarsus III	8/12 (66.7, 39.1-86.2)	17/17 (100%, 81.6-100)
Proximal sesamoid bones (medial or lateral)	3/6 (50%, 18.8-81.2)	22/23 (95.6%, 79.0-99.2)
Tendon/ligament rupture	1/2 (50%, 9.5-90.5)	27/27 (100%, 87.5-100)
Proximal limb injuries		
Humerus	2/3 (66.7%, 20.8-93.9)	26/26 (100%, 87.1-100)
Radius	1/1 (100%)	28/28 (100%, 87.9-100)
Tibia	0/1 (0%)	28/28 (100%, 87.9-100)
Pelvis	1/2 (50%, 9.5-90.5)	27/27 (100%, 87.5-100)

Discussion

Catastrophic musculoskeletal injuries

Most fatalities of Thoroughbred racehorses in racing and training in Victoria between February 1, 2001 and October 31, 2004 were the result of euthanasia due to catastrophic musculoskeletal injury to a forelimb.

This is consistent with previous studies in Australia and overseas (Vaughan and Mason 1976; Mohammed *et al.* 1991; Bourke 1994; Johnson *et al.* 1994a and 1994b; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995; Parkin *et al.* 2004a). A previous Victorian study that utilised veterinary records demonstrated that catastrophic injury of the forelimb was responsible for the majority of all limb injuries (75%, 77/102) (Bourke 1994).

No significant difference was demonstrable between the proportion of all limb injuries that involved a single left limb and the proportion that involved a single right limb, despite the unidirectional anticlockwise mode of racing in Victoria. This finding contrasts with the reported predisposition of the right limb to injury in the UK (Vaughan and Mason 1976; Parkin 2002; Parkin *et al.* 2004a) and the predisposition in the USA of the left limb to injury during racing and of the right limb during training (Rick *et al.* 1983; Rooney 1983a; Johnson *et al.* 1994a). In a previous Victorian study of racing fatalities, the number of carpal and metacarpal fractures involving right limbs was double that involving left limbs (Bourke 1994). Bourke (1994) suggested that the preponderance of right forelimb injuries was consistent with recorded observations of increased strain on the outside (right) cannon as horses negotiate racecourse bends (Davies *et al.* 1993).

In the post mortem study, fracture of metacarpus III or metatarsus III was the most common injury followed by fracture of one or both proximal sesamoid bones and concurrent fractures of metacarpus III or metatarsus III and one or both proximal sesamoids. These findings are similar to those of a previous Victorian study in which 68% of all forelimb injuries involved the fetlock and associated structures including flexor tendons, suspensory ligament and proximal sesamoid bones (Bourke 1994). The proportion of all limb injuries that was due to

fracture of metacarpus III or metatarsus III in isolation in the current study (26.7%) was comparable to the proportion (24%) reported in a Californian study (Johnson *et al.* 1994b). However, in the Californian study (Johnson *et al.* 1994b) and in other North American studies (Mohammed *et al.* 1991, Peloso *et al.* 1994), fractures of the proximal sesamoids were the most common injuries, followed by injuries to the metacarpus III and carpal bones. In contrast, in the UK, fractures of the metacarpus III with concurrent fractures of the medial proximal sesamoid bone and phalanx I were found to be the most common injuries sustained during racing (Vaughan and Mason 1976; McKee 1995; Parkin *et al.* 2004a), whereas fractures of phalanx I followed by fractures of the metacarpus III or metatarsus III were the most common injuries sustained in training (Verheyen *et al.* 2004). These differences in findings between countries may reflect international differences in race type, racing surfaces and racing practices such as shoeing and use of medications (Parkin *et al.* 2004a). In North America and in South Africa, where proximal sesamoid fractures were the most common catastrophic limb injury reported, most racing takes place on dirt tracks (Peloso *et al.* 1993; Johnson *et al.* 1994b; Macdonald and Toms 1994). In the UK, proximal sesamoid fractures were sustained most frequently on all-weather surfaces whereas metacarpus III and phalangeal fractures arose more commonly on turf surfaces (Parkin 2002). The number of catastrophic limb fractures recorded in the post mortem study in Victoria was insufficient to establish any meaningful associations between fracture types and racing surfaces. Continued collation of post mortem data will be necessary to permit future identification of any differences in the types of fracture sustained by horses whilst racing in Victoria on turf surfaces and those sustained whilst training on predominantly non-turf surfaces and to investigate any differences in the fractures sustained on tracks with different track ratings (i.e. fast or good compared with dead, slow or heavy).

In the UK, carpal fractures were reported in race training (Verheyen *et al.* 2004), flat racing (Johnson *et al.* 1994b; McKee 1995; Mizuno 1996) and most commonly in hurdle racing (Vaughan and Mason 1976; Parkin 2002). The high frequency of carpal fractures in jump races was thought to be a consequence of the carpus striking the top bar of hurdles (Vaughan and Mason 1976). Parkin (2002) speculated that the introduction of padded top rails in the UK might lead to a decrease in the prevalence of carpal fractures. The relatively small number of carpal fractures sustained by horses in jump races in Victoria over the study period may in part reflect the introduction of modular jumps to Victorian city racecourses in 2002. Modular jumps have foam rather than log take-offs and the uppermost part of the obstacle is a live or synthetic hedge of a consistency that horses can brush through (Racing Victoria Ltd. 2002).

Sudden death

Sudden deaths of racehorses have been attributed to respiratory and cardiovascular disease (Platt 1982; Gelberg *et al.* 1985; Gunson *et al.* 1988; Johnson *et al.* 1994a and 1994b). Respiratory causes of sudden death include exercise-induced pulmonary haemorrhage and pre-existent airway disease (Platt 1982; Gelberg *et al.* 1985; Johnson *et al.* 1994b). Cardiovascular causes of sudden death include myocarditis, rupture of chordae tendineae, aorta or other large arteries (Platt 1982; Gelberg *et al.* 1985; Gunson *et al.* 1988; Johnson *et al.* 1994b), aneurysm (Gelberg *et al.* 1985), atrial dysrhythmia (Platt 1982), valvular lesions (Platt 1982; Johnson *et al.* 1994b), cardiomyopathy (Johnson *et al.* 1994b), myocardial necrosis (Johnson *et al.* 1994b), sclerosing coronary arteropathy (Johnson *et al.* 1994b) and massive disseminated haemorrhage (Johnson *et al.* 1994b). Sudden deaths without detectable lesions have also been reported (Vaughan and Mason 1976; Platt 1982; Gelberg *et al.* 1985; Johnson *et al.* 1994a and 1994b).

The proportions of all racing or training sudden deaths that were attributed to particular causes have varied substantially in the literature (Gelberg *et al.* 1985; Gunson *et al.* 1988; Suann 1992; Bourke 1994; Johnson *et al.* 1994a and 1994b). For example, 17 of 25 (68%)

horses died from acute exercise-induced cardiovascular failure in Illinois (Gelberg *et al.* 1985) whereas exercise-induced pulmonary haemorrhage was the cause of sudden death in nine of 11 (82%) cases in another study (Gunson *et al.* 1988). In California, 50% of 64 horses had no abnormalities that could account for sudden death (Johnson *et al.* 1994b). In three of 15 (20%) horses in the UK, post mortem examination failed to reveal the cause of sudden death (Vaughan and Mason 1976). One possible explanation for this variability may be differences in the criteria used for case selection.

The current study suggests that sudden death is a more important contributor to racing fatalities than has been reported previously in Victoria and overseas. Sudden death accounted for 26% of all fatalities during racing and training on Victorian racetracks over the study period, compared with more than '10% of deaths' in a previous Victorian study (Bourke 1994), 9% (58/659) of Thoroughbred racing fatalities in a Californian study (Johnson *et al.* 1994a) and 12% (15/127) of racing fatalities in a British study (Vaughan and Mason 1976).

The study has also demonstrated that acute pulmonary oedema, congestion and haemorrhage are common findings in horses dying suddenly whilst racing or training in Victoria. Severe acute pulmonary haemorrhage was present in a large proportion (60%) of the sudden death cases. These findings parallel those of North American studies (Gelberg *et al.* 1985, Gunson *et al.* 1988).

In the 17 sudden death cases with acute pulmonary oedema, there was acute pulmonary congestion of equal or greater severity than that of the oedema, with engorgement of pulmonary veins and alveolar capillaries demonstrable histologically. The intra-alveolar oedema was always associated with interstitial oedema of equal or greater severity and with pulmonary lymphatic distension. In oedematous but non-haemorrhagic areas of lung, there was no histological evidence of damage to capillary endothelial or alveolar type I epithelial cells or of coagulated fibrin strands or highly proteinaceous fluid within alveoli. Therefore, the acute pulmonary oedema in all 17 cases was interpreted to be a consequence of increased hydrostatic pressure within pulmonary capillaries rather than increased vascular permeability arising from injury to alveolar type I epithelium and/or capillary endothelium (Dungworth 1993; Bachofen and Weibel 1996).

Pulmonary hypertension developing in horses during strenuous exercise (Erickson *et al.* 1990; Manohar *et al.* 1993; Langsetmo *et al.* 2000) is believed to cause an increase in the pulmonary capillary hydrostatic pressure (Sinha *et al.* 1996; Ducharme *et al.* 1999) and to contribute to the development of exercise-induced pulmonary haemorrhage. In exercise-induced pulmonary haemorrhage, haemorrhage results from rupture of alveolar capillaries, with extravasations of erythrocytes into alveolar spaces and interstitial connective tissues (West *et al.* 1993 and 1994; Couëtil and Hinchcliff 2004). Numerous mechanisms have been proposed to explain the pathogenesis of alveolar capillary rupture in exercise-induced pulmonary haemorrhage in racehorses (Couëtil and Hinchcliff 2004). The most widely accepted mechanism involves a high transmural pressure (the difference between the positive intravascular pressure and the negative intra-alveolar pressure) that exceeds the tensile strength of the capillaries (Couëtil and Hinchcliff 2004; Poole and Erickson 2004). During high-speed exercise, there is a substantial increase in the mean pulmonary arterial pressure and a substantial decrease in the inspiratory intra-alveolar pressure (West *et al.* 1994; Ducharme *et al.* 1999). As pulmonary capillaries in horses are of narrow diameter and mural thickness, they are prone to rupture even at relatively low positive transmural pressures (Poole and Erickson 2004).

Partial asphyxia due to obstruction of upper airways has also been proposed as a cause of exercise-induced pulmonary haemorrhage and acute pulmonary oedema and congestion in racehorses (Cook *et al.* 1988). Airway obstruction results in increased inspiratory effort, a decrease in the intra-alveolar pressure during inspiration and hence an increase in the alveolar

transmural pressure; the effects are exacerbated by exercise (Cook *et al.* 1988; Ducharme *et al.* 1999; Hackett *et al.* 1999). Upper airway obstruction may be referable to recurrent laryngeal neuropathy, laryngeal chondritis, epiglottic entrapment or simply reduction of the patency of the nasopharynx due to ventroflexion of the atlanto-occipital joint during racing (Cook *et al.* 1988). No structural upper airway obstruction was identified on post mortem examination of the 17 horses that died suddenly with acute pulmonary lesions.

Acute left-sided heart failure was also considered as a cause of increased pulmonary capillary hydrostatic pressure, especially in the sudden death horses #1-6 in which acute pulmonary oedema was the major finding or was comparable in severity to the pulmonary haemorrhage. Other authors have suggested that cases of sudden death with acute pulmonary oedema, congestion and haemorrhage may be due to acute left-sided heart failure caused by focal myocardial lesions or disorders of the conduction system (Gelberg *et al.* 1985, Kiryu 1999). In the current study, only four horses had microscopic myocardial lesions. However, a minimum survival interval of 4 hours post-injury is necessary for characteristic microscopic lesions of myocardial necrosis to emerge (Schoen 2005). Therefore peracute myocardial injury cannot be excluded in cases of sudden death with acute hydrostatic pulmonary oedema if only light microscopic examination of cardiac tissue is performed.

As sudden death horse #1 had a deep laceration of the parietal cerebral cortex due to a cranial depression fracture sustained terminally as it fell, the possibility of acute neurogenic pulmonary oedema had to be considered. Pulmonary oedema referable to acute brain injury develops initially due to increased hydrostatic pressure within the pulmonary microvasculature (mediated by catecholamine release) but is later augmented by increased capillary permeability (Dungworth 1993; Bachofen and Weibel 1996). The severity of the pulmonary oedema in this animal suggested, however, that the onset of pulmonary oedema preceded terminal cranial injury.

Future investigation of causes of sudden death may be facilitated by analysis of blood samples obtained immediately after death. Assays of markers of peracute myocardial injury (such as cardiac troponin I) may help to confirm the presence of myocardial degeneration or necrosis. Assays of cardiac-specific muscle enzymes and proteins such as myoglobin, creatine kinase-myocardial band isoforms, cardiac troponin T and cardiac troponin I are used in humans to diagnose myocardial damage. Of these, cardiac troponin is considered to be the gold standard marker due to its high sensitivity (> 90%) (Schwarzwalder *et al.* 2003). Moreover, in humans, cardiac troponin I persists in the circulation for 4-14 days after myocardial injury whereas other markers disappear from circulation more rapidly (Phillips *et al.* 2003). Studies have already demonstrated that immunoassays for human cardiac troponin I cross-react with equine cardiac troponin I (O'Brien *et al.* 1997) and there is preliminary evidence that cardiac troponin I may be useful as a diagnostic marker in horses with myocardial disease. Further research is still necessary to define the prognostic use and limitations of this clinical test in horses (Cornelisse *et al.* 2000; Schwarzwalder *et al.* 2003).

Collection of point-of-death blood samples would also permit detection of abnormalities in electrolytes (especially potassium, calcium and magnesium) that might afford an explanation for peracute myocardial injury. Coagulation studies performed on point-of-death blood samples may also be beneficial in investigating cases of sudden death from massive haemorrhage.

Validation of fatality reports

The majority of previous studies describing causes of racehorse injury or fatality have been based on unconfirmed diagnoses made by racecourse veterinarians (Vaughan and Mason 1976; Rosedale *et al.* 1985; Palmer *et al.* 1986; Suann 1992; Wilson *et al.* 1993, 1996a and 1996b; Bathe 1994; Bourke 1994; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995; Mizuno 1996; Cohen *et al.* 1999b; Williams *et al.* 2001; Verheyen *et al.* 2004). Studies that utilise findings from post mortem examinations are deemed more accurate, as post mortem examination is widely accepted as the gold standard for diagnosis (Dohoo *et al.* 2003). A recent study in the UK that utilised post mortem examinations of limbs to investigate distal catastrophic limb fractures demonstrated that only 71% of Jockey Club reports correctly identified the bone(s) involved (Parkin 2002).

Racing Victoria Ltd. has collated data over more than two decades from veterinary and steward fatality reports to monitor the numbers of fatalities sustained during racing and to record the causes. Data have been collated consistently since 1989. The horses submitted for post mortem examination were accompanied by a copy of the fatality report that was completed by the attending racecourse veterinarian at the time of death. The post mortem study was a unique opportunity to compare the provisional diagnosis recorded in the fatality report with the principal diagnosis made by pathologists after conducting a post mortem examination.

With few exceptions, racecourse veterinarians reliably classified fatality cases within the broad categories of catastrophic limb injury, cranial or vertebral injury and sudden death (due to any cause).

The sensitivity of diagnosis of fatalities due to injury to the axial skeleton (cranium, vertebrae or ribs) was 55.6% but specificity was high (100%). The sensitivity was low because of the misclassification of horses that died suddenly as a result of a cranial or vertebral injury. If the horse was dead by the time that the racecourse veterinarian arrived, the case was reported as a sudden death rather than as a cranial or vertebral injury. Also, veterinarians frequently described neurological signs (such as flaccid tail, ataxia or inability to move) in fatality reports but did not venture a diagnosis of cranial or spinal injury.

The sensitivity and specificity of diagnosis for specific causes of sudden death were understandably low as post mortem examination is essential to confirm suspicions of cardiovascular or respiratory failure or severe acute muscular damage. Specificity for the diagnosis of a sudden death due to unknown cause was low as racecourse veterinarians commonly used the terminology 'sudden death syndrome' to encompass all horses that died without veterinary intervention on the racecourse.

Diagnosis of catastrophic limb injuries by racecourse veterinarians was very reliable. The sensitivity and specificity of diagnosis for these cases were 97.8% (CI 88.4-99.6) and 100% (CI 89.3-100) respectively. Only one catastrophic limb injury was misclassified as a sudden death; post mortem examination revealed that acute haemoperitoneum was secondary to a pelvic fracture and severance of an artery. Fatality reports were also very reliable with respect to the broad anatomical location of a catastrophic limb injury (i.e. distal, proximal, fore, hind, left or right). When errors occurred in these diagnostic categories, they were predominantly due to omissions rather than incorrect descriptions. However, the ability of veterinarians to correctly identify the specific location of a catastrophic limb injury tended to be weak. The poor sensitivity of reporting of fractures to the distal limb no doubt reflected the difficulty in externally assessing the integrity of all fetlock components. Diagnoses of multiple bone fractures were also unreliable. Such unreliability may reflect the stressful circumstances in which decisions are made to euthanise horses on racecourses. Catastrophic fractures that necessitate immediate euthanasia are unlikely to be radiographed and often the veterinarian is under pressure to complete the euthanasia quickly. This may mean that fatality

reports are filled in quickly. It may also be that the introduction of post mortem examinations in February 2001 has meant that fatality reports are not filled in with as much attention to detail because racecourse veterinarians are aware that a post mortem examination will be performed.

Sensitivity and specificity estimates presented in this study should be interpreted cautiously as the numbers of fatalities were limited. Larger numbers of post mortem examinations and corresponding fatality reports will be necessary to allow precise estimates of the sensitivity and specificity of these reports. Nevertheless, the results of the study demonstrate the need for improved documentation in fatality reports. In the current format, the fatality reports (Appendix 1) have open-ended questions. Such a format encourages the use of ambiguous terminology such as 'fetlock' or 'leg'. A report form with checklists for different types of fractures may be preferable to an open-ended format in order to increase the diagnostic sensitivity and minimise the time required to complete the form.

The results of this study suggest that it is reasonable to rely on data collated from veterinary reports provided that general case definitions are used (e.g. sudden death versus catastrophic limb injury versus cranial or vertebral injury). Fatality report data can also be used to define catastrophic limb injuries according to their broad anatomical location (distal or proximal, fore- or hind, left or right). However, the study has shown that veterinary reports have low sensitivity for diagnosing specific conditions such as exercise-induced pulmonary haemorrhage or lateral condylar fracture of metacarpus III. The risk of specific conditions would be underestimated if industry monitoring systems were based on the frequency of diagnoses made in these reports. Because of the low sensitivity in diagnosis of specific conditions, veterinary reports should not be used to identify control fatalities in case-control studies for risk factors for specific conditions in which fatalities due to other causes are selected as controls, particularly in populations in which the condition of interest has a high proportional mortality rate. However, the high specificity of the diagnoses suggests that fatality reports may be useful for identifying cases in case-control studies.

Limitations of the study

As only 43% of all racehorse deaths were sampled over the study period, the potential for sampling bias had to be considered. Sampling bias can occur when a small proportion of eligible cases is sampled in a non-random fashion. Whereas city fatalities studied at necropsy were highly representative of all city cases, country fatalities submitted for post mortem were not representative of all country fatalities because sudden deaths were over-represented. The large difference between the relative risks of submission for necropsy from the city and country tracks and the very low *p*-value for heterogeneity demonstrate that sampling bias was present in submissions of horses from country tracks. This type of bias could be avoided by making post mortem examinations mandatory on all tracks in Victoria. Alternatively, random sampling of fatalities would be preferable to studying only those fatalities that horse owners, trainers and/or racetrack veterinarians choose to submit.

Apart from sampling bias, a limitation of this study was the relatively small number of post mortem examinations that were performed. Fatalities are relatively rare events in flat and jump racing. Therefore, studies that investigate racing fatalities must be conducted over long periods of time in order to identify genuine trends and significant associations. If mandatory submission of country fatalities were possible, this would expand the study size dramatically over a short time frame. However, the logistics of long transport distances, high costs and the increased workload of pathology staff at The University of Melbourne may mean that this is not feasible in the short term. The post mortem study was modelled on the California post mortem program (Johnson *et al.* 1994a and 1994b) and Racing Victoria Ltd. is committed to funding it for another ten years. A racing fatality database has been established at The University of Melbourne to ensure the continuity and consistency of data collection. In the

future, data from the post mortem study will be able to be used for more specific risk factor studies to investigate the risk of fatality due to exercise-induced pulmonary haemorrhage and other causes of sudden death.

Conclusions

This study has demonstrated that post mortem examinations are an essential component of any study of racehorse fatalities to ensure accurate assessment of musculoskeletal injuries warranting euthanasia and, to accurately identify the causes of sudden death, particularly cardiovascular or respiratory failure. The results from this study have given rise to recommendations to improve the investigation of sudden deaths and increase the diagnostic information contained in fatality report forms.

4. Risk of fatality and causes of death of Thoroughbred horses associated with racing in Victoria (1989 – 2004)

Introduction

Determining the risk of fatality of Thoroughbred horses whilst racing is essential to assess the impact of intervention measures designed to minimise such fatalities. Additionally, increased public cognisance of animal welfare issues has necessitated that the racing industry accurately measures the risk of injury or death of Thoroughbred horses during racing.

The risk of fatality of Thoroughbred horses whilst racing varies within and between countries and also with race type. Several North American and British studies have examined the risk in flat starts (Peloso *et al.* 1994; McKee 1995; Estberg *et al.* 1996a; Wood *et al.* 2002) and in jump starts (McKee 1995; Wood *et al.* 2002; Stephen *et al.* 2003). The risk of fatal injury in flat starts was 1.4 per 1000 starts in Kentucky (Peloso *et al.* 1994), 1.7 per 1000 starts in California (Estberg *et al.* 1996a) and 0.8-0.9 per 1000 starts in the UK (McKee 1995; Wood *et al.* 2002). In Virginia, the risk of fatal injury in jump starts was 3.4 per 1000 starts (Stephen *et al.* 2003). In the UK, the risk of fatality in jump starts was 4.9 per 1000 starts in hurdle races, 6.7 per 1000 starts in steeplechases and 5.6 per 1000 starts in jump races combined (McKee 1995; Wood *et al.* 2002). These studies demonstrated that the risk of fatality was greater in jump than in flat starts.

In Victoria, Australia, routine collection of fatality data by the racing industry over recent decades has facilitated measuring the risk of fatality of Thoroughbreds in racing and evaluating short-term fluctuations and long-term trends in the risk. There have been three previous studies of the risk in Victoria (Bourke 1994 and 1995; Bailey *et al.* 1998). The risk of Thoroughbred fatality in Melbourne, Victoria between 1986 and 1993 was 0.3 per 1000 starts in flat races, 6.0 per 1000 starts in hurdle races and 11.0 per 1000 starts in steeplechases (Bourke 1994 and 1995). Between 1988 and 1995, the risk of serious musculoskeletal injury (resulting in euthanasia or a failure to return to racing for at least six months) on city tracks in Victoria was 0.6 per 1000 starts in flat races, 6.3 per 1000 starts in hurdle races and 14.3 per 1000 starts in steeplechases (Bailey *et al.* 1998).

The major objective of this study was to retrospectively measure the risk of racehorse fatality in flat and jump starts on all Victorian racecourses over a 15 year period commencing at the start of the 1989 racing season. A long study period was deliberately chosen so that any important long-term trends in risk could be identified (Wood *et al.* 2002). The availability of 15 years of retrospective data enabled the inclusion of those periods previously reported by Bourke (1994 and 1995) and Bailey *et al.* (1998). The chosen period also included three major industry reviews of Victorian jump racing (Benton 1994; Racing Victoria Ltd. 1998 and 2002) that led to the introduction of measures to minimise the risks of injury and fatality (Benton 1994; Racing Victoria Ltd. 2002).

A second objective of the study was to determine proportions of fatalities according to specific causes in all race types combined, in flat versus jump races and on city versus country racetracks. This information is essential for a thorough analysis of the risks involved in Thoroughbred racing in Victoria and provides benchmarks for the industry to evaluate intervention strategies designed to minimise fatalities.

Results

Overall risk of fatality and risk by race type

Over the 15 year study period, there were 743,552 starts (719,695 flat and 23,857 jump starts) and 514 fatalities on Victorian racecourses. The risk of fatality over the entire period was 0.69 per 1000 starts (CI 0.63-0.75). Of the 514 fatalities, 316 occurred in flat races and 198 in jump races (101 in hurdle and 97 in steeplechase races). The risk of fatality in flat starts was 0.44 per 1000 starts (316/719,695, CI 0.39-0.49) (Table 10) whereas that in jump starts was 8.3 per 1000 starts (198/23,857, CI 7.2-9.5) (Table 11). The risk of fatality in jump starts was 18.9 (CI 15.8-22.6) times that in flat starts.

Risk of fatality by race location

There were 162,715 city starts and 580,837 country starts between 1989 and 2004. Of the total of 514 fatalities, 184 occurred on city tracks and 330 on country tracks. The risk of fatality in all race types was 1.1 per 1000 starts (CI 1.0-1.3) on city tracks and 0.57 per 1000 starts (CI 0.51-0.63) on country tracks. The risk of fatality on city racecourses was 2.0 (CI 1.7-2.4) times that on country racecourses.

Within each race type, the risk of fatality was significantly higher on city than on country racecourses. Within flat starts, the risk of fatality was 0.60 per 1000 starts (92/154,420, CI 0.49-0.73) on city racecourses whereas that on country racecourses was 0.40 per 1000 starts (224/565,275, CI 0.35-0.45) (Table 10). The risk of fatality in flat starts on city racecourses was 1.5 (CI 1.2-1.9) times that on country tracks. Within jump starts, the risk of fatality was 11.1 per 1000 starts (92/8295, CI 9.0-14.0) on city racecourses whereas that on country racecourses was 6.8 per 1000 starts (106/15,562, CI 5.6-8.2) (Table 11). The risk of fatality in jump starts on city tracks was 1.6 (CI 1.2-2.2) times that on country tracks.

Table 10 Number of Thoroughbred racehorse fatalities and risk of fatality per 1000 starts in flat races in Victoria between 1989 and 2004 by year; the subtotals according to racetrack location and the risk ratio (RR) of fatality for each racing year compared with the racing year of 1989-1990

Racing year	COUNTRY			CITY			CITY AND COUNTRY			
	Deaths	Starts	Deaths per 1000 starts (95% CI)	Deaths	Starts	Deaths per 1000 starts (95% CI)	Total deaths	Total starts	Deaths per 1000 starts (95% CI)	Risk ratio (95% CI)
1989-90	7	40 333	0.17 (0.08-0.36)	5	10 150	0.49(0.21-1.2)	12	50 483	0.24 (0.14-0.42)	1.00
1990-91	11	40 713	0.27 (0.15-0.48)	6	10 077	0.60 (0.27-1.3)	17	50 790	0.33 (0.21-0.54)	1.4 (0.67-3.0)
1991-92	16	39 545	0.40 (0.25-0.66)	6	9753	0.62 (0.28-1.3)	22	49 298	0.45 (0.29-0.68)	1.9 (0.93-3.8)
1992-93	14	39 079	0.36 (0.21-0.60)	10	9701	1.0 (0.60-1.9)	24	48 780	0.49 (0.33-0.73)	2.1 (1.0-4.1)
1993-94	11	38 168	0.29 (0.16-0.52)	5	9172	0.55 (0.23-1.3)	16	47 340	0.34 (0.21-0.55)	1.4 (0.67-3.0)
1994-95	14	36 167	0.39 (0.23-0.65)	9	9195	0.98 (0.52-1.9)	23	45 362	0.51 (0.34-0.76)	2.1 (1.1-4.3)
1995-96	13	37 225	0.35 (0.20-0.60)	2	9582	0.21 (0.06-0.76)	15	46 807	0.32 (0.19-0.53)	1.4 (0.63-2.9)
1996-97	11	38 067	0.29 (0.16-0.52)	7	10 093	0.69 (0.34-1.4)	18	48 160	0.37 (0.24-0.59)	1.6 (0.76-3.3)
1997-98	14	36 757	0.38 (0.23-0.64)	6	11 134	0.54 (0.25-1.2)	20	47 891	0.42 (0.27-0.64)	1.8 (0.86-3.6)
1998-99	14	36 295	0.39 (0.23-0.65)	7	11 136	0.63 (0.30-1.3)	21	47 431	0.44 (0.29-0.68)	1.9 (0.92-3.8)
1999-00	13	36 558	0.36 (0.21-0.61)	5	10 736	0.47 (0.20-1.1)	18	47 294	0.38 (0.24-0.60)	1.6 (0.77-3.3)
2000-01	24	37 340	0.64 (0.43-0.96)	6	11 030	0.54 (0.25-1.2)	30	48 370	0.62 (0.43-0.89)	2.6 (1.3-5.1)
2001-02	27	36 506	0.74 (0.51-1.1)	5	10 890	0.46 (0.20-1.1)	32	47 396	0.68 (0.48-1.0)	2.8 (1.5-5.5)
2002-03	14	36 962	0.38 (0.23-0.64)	8	10 986	0.73 (0.37-1.4)	22	47 948	0.46 (0.30-0.69)	1.9 (1.0-3.9)
2003-04	21	35 560	0.59 (0.39-0.90)	5	10 785	0.46 (0.20-1.1)	26	46 345	0.56 (0.38-0.82)	2.4 (1.2-4.7)
TOTAL	224	565 275	0.40 (0.35-0.45)	92	154 420	0.60 (0.49-0.73)	316	719 695	0.44 (0.39-0.49)	

Table 11 Number of Thoroughbred racehorse fatalities and risk of fatality per 1000 starts in jump races in Victoria between 1989 and 2004 by year; the subtotals according to racetrack location and the risk ratio (RR) of fatality for each racing year compared with the racing year of 1989-1990

Racing year	COUNTRY			CITY			CITY AND COUNTRY			
	Deaths	Starts	Deaths per 1000 starts (95% CI)	Deaths	Starts	Deaths per 1000 starts (95% CI)	Total deaths	Total starts	Deaths per 1000 starts (95% CI)	Risk ratio (95% CI)
1989-90	6	1291	4.7 (2.1-10.1)	9	751	12.0 (6.3-22.6)	15	2042	7.4 (4.5-12.1)	1.00
1990-91	10	1271	7.9 (4.3-14.4)	6	779	7.7 (3.5-16.7)	16	2050	7.8 (4.8-12.6)	1.1 (0.53-2.1)
1991-92	5	1183	4.2 (1.8-9.9)	9	709	12.7 (7.0-24.0)	14	1892	7.4 (4.4-12.4)	1.0 (0.49-2.1)
1992-93	13	1212	10.7 (6.3-18.3)	7	709	9.9 (4.8-20.2)	20	1921	10.4 (6.7-16.0)	1.4 (0.73-2.8)
1993-94	13	1109	11.7 (6.9-20.0)	9	655	13.7 (7.2-25.9)	22	1764	12.5 (8.3-18.8)	1.7 (0.88-3.3)
1994-95	9	1010	8.9 (4.7-16.8)	12	523	22.9 (13.2-39.7)	21	1533	13.7 (9.0-20.9)	1.9 (0.96-3.6)
1995-96	7	1069	6.6 (3.2-13.5)	2	464	4.3 (1.2-15.6)	9	1533	5.9 (3.1-11.1)	0.80 (0.35-1.8)
1996-97	6	964	6.2 (2.9-13.5)	6	537	11.2 (5.1-24.2)	12	1501	8.0 (4.6-13.9)	1.1 (0.51-2.3)
1997-98	5	961	5.2 (2.2-12.1)	2	558	3.6 (1.0-13.0)	7	1519	4.6 (2.2-9.5)	0.63 (0.26-1.5)
1998-99	8	1022	7.8 (4.0-15.4)	9	492	18.3 (9.7-34.4)	17	1514	11.2 (7.0-17.9)	1.5 (0.77-3.1)
1999-00	3	989	3.0 (1.0-8.9)	3	466	6.4 (2.2-18.8)	6	1455	4.1 (1.9-9.0)	0.56 (0.22-1.4)
2000-01	6	1055	5.7 (2.6-12.4)	2	423	4.7 (1.3-17.1)	8	1478	5.4 (2.7-10.6)	0.70 (0.31-1.7)
2001-02	5	962	5.2 (2.2-12.1)	8	444	18.0 (9.2-35.2)	13	1406	9.3 (5.4-15.8)	1.3 (0.60-2.6)
2002-03	6	788	7.6 (3.5-16.5)	5	408	12.3 (5.3-28.4)	11	1196	9.2 (5.1-16.4)	1.3 (0.58-2.7)
2003-04	4	676	5.9 (2.3-15.1)	3	377	8.0 (2.7-23.1)	7	1053	6.6 (3.2-13.7)	0.91 (0.37-2.2)
TOTAL	106	15 562	6.8 (5.6-8.2)	92	8295	11.1 (9.0-13.6)	198	23 857	8.3 (7.2-9.5)	

Proportional mortality rates

Catastrophic limb injury was the predominant cause of death, being responsible for 71.6% (368/514, CI 67.5-75.3%) of all racing fatalities (Table 12). Cranial or vertebral injury accounted for 7.8% (40/514, CI 5.8-10.4%) of the fatalities. Sudden death accounted for 13.0% (67/514, CI 10.4-16.2%) of all fatalities. Four of the 514 horses (0.78%, 4/514, CI 0.30-2.0%) collapsed during or immediately after racing and were euthanased due to suspected cardiovascular failure. The cause of death was not recorded for 6.6% of fatalities (34/514, CI 4.8-9.1%).

Of the 316 fatalities in flat races, 73.4% (232/316, CI 68.3-78.0%) were due to catastrophic limb injury, 2.5% (8/316, CI 1.3-4.9%) were due to cranial or vertebral injury and 19.0% (60/316, CI 15.0-23.7%) were sudden deaths. Of the 232 catastrophic limb injuries sustained in flat races (Table 13), distal or proximal location was described for 215 horses (92.7%). Of these, 80.9% (174/215, CI 75.2-85.6%) were classified as distal limb injuries and 18.6% (40/215, CI 14.0-24.3%) as proximal. One horse sustained both distal and proximal limb injuries. Forelimb or hindlimb location was described in 208 horses (89.7%). Forelimbs accounted for 75.5% (157/208, CI 69.2-80.8%) and hindlimbs 24.5% (51/208, CI 19.2-30.8%) of the catastrophic limb injuries in flat starts. Left or right limb location was described in 199 horses (85.8%). Most catastrophic limb injuries were unilateral, with only four horses sustaining injury to both a left and a right limb (Table 13). Left limb injuries accounted for 54.8% (109/199, CI 47.8-61.5%) and right limb injuries 43.2% (86/199, CI 36.5-50.2%) of catastrophic limb injuries in flat starts.

Of the 198 fatalities in jump races, 68.7% (136/198, CI 61.9-74.7%) were due to catastrophic limb injury, 16.2% (32/198, CI 11.7-21.9%) resulted from cranial or vertebral injury and 3.5% (7/198, CI 1.7-7.1%) were sudden deaths. Of the 136 catastrophic limb injuries (Table 12), distal or proximal location was described for 121 horses (89.0%). Of these horses, 50.4%

(61/121, CI 41.6-59.2%) were distal limb injuries and 46.3% (56/121, CI 37.6-55.1%) proximal limb injuries. A further four horses sustained injuries to both distal and proximal limbs. Forelimb or hindlimb location was reported in 120 horses (88.2%). Forelimb injury accounted for 75.8% (91/120, CI 67.4-82.6%) and hindlimb injury 23.3% (28/120, CI 16.7-31.7%) of the catastrophic limb injuries in jump starts. One horse sustained both a fore- and a hindlimb injury in a jump start. Left or right limb location was described for 109 horses (80.1%). Left limb injuries accounted for 52.3% (57/109, CI 43.0-61.4%) and right limb injuries 45.9% (50/109, CI 36.8-55.2%) of the catastrophic limb injuries in jump starts. Two horses sustained injuries to both a left and a right limb (Table 13).

Excluding multiple injuries to both distal and proximal limbs, the proportion of all limb injuries that involved the distal limb was significantly larger in flat starts (174/214) than in jump starts (61/117) (p -value < 0.001). Excluding multiple injuries to both fore and hindlimbs, the proportion of all limb injuries that involved the forelimb was not significantly greater in jump starts (91/119) than in flat starts (157/208) (p -value = 0.89). The proportion of all unilateral limb injuries that were on the left side did not differ significantly between flat starts (109/195) and jump starts (57/107) (p -value = 0.72) and did not differ from 0.5 within flat (95% CI 0.49-0.63) or jump (95% CI 0.44-0.62) race types.

Cause-specific risks of fatality

For each cause, the cause-specific risk of fatality was greater in jump than in flat starts. For catastrophic limb injury, the risk was 17.7 times greater (CI 14.3-21.8) in jump starts (5.7 per 1000 starts, 136/23 857) than in flat starts (0.32 per 1000 starts, 232/719 695) (Table 12). The risk of fatal cranial or vertebral injury was 120.7 times greater (CI 55.6-261.8) in jump starts (1.3 per 1000 starts, 32/23 857) than in flat starts (0.01 per 1000 starts, 8/719 695). The risk of sudden death was 3.5 times greater (CI 1.6-7.7) in jump racing (0.29 per 1000 starts, 7/23 857) than in flat racing (0.08 per 1000 starts, 60/719 695).

The risk of catastrophic limb injury in flat starts was 1.5 times (CI 1.2-2.1) greater on city than on country tracks. The risk was 0.45 per 1000 starts (69/154 420, CI 0.45-0.57) on city racecourses and 0.29 per 1000 starts (163/565 275, CI 0.25-0.34) on country racecourses. Catastrophic limb injury in jump starts was 1.6 times (CI 1.2-2.3) more likely to occur on city than on country tracks. The risk was 7.6 per 1000 starts (63/8295, CI 5.9-9.7) on city racecourses and 4.7 per 1000 starts (73/15 562, CI 3.7-5.9) on country racecourses.

Table 12 Number of fatalities of Thoroughbred horses on racecourses in Victoria between 1989 and 2004, according to racetrack location, race type and reported cause of fatality

Reported cause of fatality	CITY TRACKS			COUNTRY TRACKS		
	Number of fatalities in all city races (n = 162 715 starts)	Number of fatalities in flat races (n = 154420 starts)	Number of fatalities in jump races (n = 8295 starts)	Number of fatalities in all country races (n = 580 837 starts)	Number of fatalities in flat races (n = 565 275 starts)	Number of fatalities in jump races (n = 15 562 starts)
Catastrophic limb injury	132	69	63	236	163	73
Sudden death	18	15	3	49	45	4
Collapse and euthanasia	1	0	1	3	2	1
Cranial or vertebral injury	21	1	20	19	7	12
Axial and appendicular skeletal injury	1	0	1	0	0	0
Unknown	11	7	4	23	7	16
TOTAL	184	92	92	330	224	106

Table 13 Number of catastrophic limb injuries by anatomical location and race type

Location of catastrophic limb injury	Total number sustained in flat starts (% of total excluding missing and multiple limb injury) (n = 232)	Total number sustained in jump starts (% of total excluding missing and multiple limb injury) (n = 136)	Total sustained in both race types (n = 368)
	Distal	174 (81)	61 (50)
Proximal	40 (19)	56 (46)	96 (29)
Distal and proximal	1 (0)	4 (3)	5 (1)
Unknown	17	15	32
Total (excluding missing)	215	121	336
Forelimb	157 (75)	91 (76)	248 (76)
Hindlimb	51 (25)	28 (23)	79 (24)
Forelimb and hindlimb	0 (0)	1 (1)	1 (0)
Unknown	24	16	40
Total (excluding missing)	208	120	328
Left	109 (55)	57 (52)	166 (54)
Right	86 (43)	50 (46)	136 (44)
Left and right	4 (2)	2 (2)	6 (2)
Unknown	33	27	60
Total (excluding missing)	199	109	308

In jump starts, fatality due to cranial or vertebral injury was more likely to occur on city than on country tracks (RR 3.1, CI 1.5-6.4). The risk was 2.4 per 1000 starts (20/8295, CI 1.6-3.7) on city tracks and 0.77 per 1000 starts (12/15 562, CI 0.44-1.35) on country tracks. The risk of fatal cranial or vertebral injury in flat starts was low and not significantly different between city and country tracks.

Sudden death was just as likely to occur on city tracks as on country tracks in both flat and jump races, with relative risks of 1.2 (CI 0.7-2.2) and 1.4 (CI 0.3-6.3) respectively. The risk in flat starts was 0.10 per 1000 starts (15/154 420, CI 0.06-0.16) on city tracks and 0.08 per

1000 starts (45/565 275, CI 0.06-0.11) on country tracks. The risk in jump starts was 0.36 per 1000 starts on city tracks (3/8295, CI 0.12-1.06) and 0.26 per 1000 starts (4/15 562, CI 0.10-0.66) on country tracks.

Population attributable risk and population attributable fraction for jump racing

For jump starts, the population attributable risk of fatality was 0.25 per 1000 starts (514/743 552-316/719 695) and the population attributable fraction was 36.5% (0.25/0.69, CI 32.0-40.7%). Assuming that the association of fatality with jump starts is causal and unbiased, jump racing increased the overall risk of fatality in Victoria during the study period by 0.25 per 1000 starts (one death in 4000 starts) or 36.5%. In other words, had one or a combination of unspecified strategies been successful in reducing the risk in jump starts to that in flat starts during the study period, the overall risk of fatality in Victoria during that period would have been reduced by 0.25 per 1000 starts or 36.5%.

Risk of fatality over time

The risk of fatality in flat starts appeared to increase (RR per year 1.04, CI 1.01-1.07) over the entire study period (Table 9). However, comparison of the individual risk ratios for each racing year of the study did not suggest a trend over time. The apparent increase in risk over time was strongly influenced by the low risk of fatality in 1989-1990 and the high risk in 2000-2001 and 2001-2002 and appeared to be associated with an increase in risk over time on country racecourses for which the RR per year was 1.06 (CI 1.03-1.09). There was no change over time in the risk of fatality on city racecourses (RR per year 0.98, CI 0.94-1.03).

Between 1989 and 2004, the risk of fatality in jump starts fluctuated from year to year but there was no trend in risk over time (RR per year 0.99, CI 0.95-1.02) (Table 10). There was no change in the risk over time on either city racecourses (RR per year 0.99, CI 0.95-1.04) or country racecourses (RR per year 0.98, CI 0.94-1.02).

Discussion

The overall risk of fatality in horse racing calculated in this study was similar to that reported in three previous Australian studies (Bourke 1994 and 1995; Bailey *et al.* 1998). The risk of fatality in flat racing in Victoria (0.44 per 1000 starts) compared favourably with that in the USA (1.4-1.7 per 1000 starts) (Peloso *et al.* 1994; Estberg *et al.* 1996a) and the UK (0.9 per 1000 starts) (McKee 1995; Wood *et al.* 2002). However, the risk of fatality in Victorian jump racing (8.3 per 1000 starts) was twice that reported in the USA (3.9 per 1000 starts) (Stephen *et al.* 2003) and 50% greater than that reported in the UK (5.6 per 1000 starts) (Wood *et al.* 2002).

Bailey *et al.* (1998) speculated that differences in horse populations and racing cultures could explain why horses in jump races have a greater risk of fatality in Australia than in the UK. Most jump horses (75%) in the UK are trained exclusively for jumping (Ely *et al.* 2004) and have long careers in jump racing. Racehorses that jump in Victoria are older than those involved in flat racing and may have had a large number of starts in flat racing prior to commencing jump racing (Bailey *et al.* 1998). Only 1% of jump starts in Victoria are by horses that have never started in a flat race (Chapter 6). In a case-control study of fatalities in jump starts in Victoria, horses with selected study starts had on average 33 prior flat starts (Chapter 6). In contrast, in a case-control study of fatalities in flat starts, horses with selected study starts had on average only 17 prior flat starts (Chapter 5). It may be that long-term repetitive trauma to bones, ligaments and tendons sustained during flat racing and relative inexperience in jump racing contribute to the greater risk in jump starts in Victoria than in the

UK. However, such a hypothesis does not explain the discrepancy in risk between jump racing in Victoria and in the USA as racehorses that jump in steeplechase events in both locations have usually had prior flat racing careers.

Differences in fence size and construction may contribute to differences in risk of fatality. However, the size of obstacles in jump races was comparable in the UK and Victoria during the study period. Hurdles stood at approximately one metre high in both locations. Prior to 2002, steeplechase fences in Victoria, although not standardised, were at least 1.15 metres in height and on two city tracks were up to 1.4 metres high. In the UK, steeple fences were 1.37 metres in height. Rather than focussing on obstacle size, it may be more important to explore international differences in the number and placement of obstacles within races and in obstacle materials and design when investigating the risk factors for fatality. A comparison of Australian, North American and British jump racing, including factors such as training regimens, fence construction and track condition, would be particularly useful in understanding how to reduce the risks in all three racing populations.

The risk of fatality was found to be greater on city than on country racecourses. Differences in the risk of fatality on city and country racecourses in flat starts may be attributable to possible underreporting of fatalities by country racecourse officials prior to 2001. Alternatively, in both flat and jump starts, this finding may reflect differences in the calibre of horses, speed of race, track surface and experience of jockeys on city versus country tracks in Victoria. Races held on Melbourne racetracks are regarded as more prestigious and offer greater prize money than those convened on Victorian country tracks. Consequentially, there may be differences in race speeds, career lengths, career earnings and training regimens between horses racing in different locations that may be associated with the likelihood of death. Measurement of the risk on city and country racecourses may be a proxy measure of horse quality or competitiveness. A previous study found that horses starting in more competitive events in Sydney, Australia had a greater risk of severe injury than did horses in less competitive events (Bailey *et al.* 1997a). This contrasts with the finding of Wood *et al.* (2002) that, in flat racing in the UK, higher rated horses were less likely to suffer catastrophic injury than lower rated horses. Differences in track surface and design at the different locations may also influence the risk. Bailey *et al.* (1998) reported differences in the risk of fatality amongst the four city tracks in Melbourne over a period of six years. Because the data utilised in the current study only indicated whether the racecourse location was in the country or the city, potential differences in risk amongst the various racecourses in Victoria could not be explored. Future studies should aim to identify the particular features of individual racecourses that increase the likelihood of fatality so that these can be targeted by intervention strategies.

Irrespective of race type, catastrophic limb injury was the most common reason for Thoroughbred racehorse fatalities on city and country racetracks in Victoria between 1989 and 2004. This is consistent with studies in the USA (Johnson *et al.* 1994b), South Africa (Macdonald and Toms 1994) and the UK (McKee 1995; Wood *et al.* 2002). The proportion of all fatalities in flat races that was referable to catastrophic limb injury (73%) was comparable to that reported in the UK (74%) (Wood *et al.* 2002) and smaller than that reported in the USA (89%) (Johnson *et al.* 1994b). Catastrophic limb injury was responsible for a larger proportion of fatalities in jump races in Victoria (69%) than in the UK (48%) (Wood *et al.* 2002).

Of the catastrophic limb injuries observed, the majority involved a distal limb and a forelimb. This finding is not surprising as the distal limbs and forelimbs of horses sustain a greater force than do proximal limbs or hindlimbs during high-speed racing (Pratt and O'Connor 1976; Stephen *et al.* 2003). The proportion of limb injuries that involved the distal limb was significantly larger in flat racing than in jump racing. This observation points to differences between race types in the pathogenesis of catastrophic limb injuries. There is speculation that

certain types of catastrophic distal and proximal limb fractures may be the result of pre-existing stress fractures (Stover *et al.* 1992; Riggs *et al.* 1999; Riggs 2002). Stress fractures have been reported frequently in athletes and soldiers who undergo extreme repetitive weight-bearing activities such as running or marching (Jones *et al.* 2002). Repetitive high-speed exercise sustained in racing and training may predispose horses to stress fractures. The location of these stress fractures (and ultimately the catastrophic fractures) is likely to be influenced by differences in bone loading between flat and jump races.

There was no difference in the proportion of all limb injuries that were forelimb injuries in flat and jump races.

No difference was observed between the overall proportions of right and left catastrophic limb injuries in flat and jump racing despite the fact that all races in Victoria are run in an anticlockwise direction. This contrasts with the findings of studies in other countries where differences between the distribution of left and right limb injuries have been reported (Vaughan and Mason 1976; Johnson *et al.* 1994b; Parkin *et al.* 2004a). Although there is evidence that strains are increased on the outside limb during racing (Davies 1993), the results from this study did not confirm the suggestion of Bourke (1994) that the outside limb (i.e. the right limb in horses running anticlockwise) was at greater risk than the inside (left) limb during racing in Victoria.

In this study, the risk of fatality due to cranial or vertebral injury varied according to race type. The proportion of all fatalities due to such injury was larger in jump starts than in flat starts. In jump racing, this proportion (17%) was similar to that reported in the UK (19-23%) (Vaughan and Mason 1976; Wood *et al.* 2002). In flat races, the proportion (3%) was also similar to that reported in California (1.9%) (Johnson *et al.* 1994b) and in the UK (1%) (Wood *et al.* 2002). The greater risk of fatal cranial or vertebral injury in jump starts on city tracks than on country tracks may be attributable to the fast speeds of city races but intra-race studies are still required to investigate the precise sequence of events responsible for these injuries in jump races.

The proportions of fatalities attributable to catastrophic limb injury, cranial or vertebral injury and sudden death in this study were similar to those reported previously in Australia (Bourke 1995). Proportional mortality rates for specific causes of sudden death could not be described because diagnoses of fatality were based on observations by racecourse veterinarians or stewards without corroboration by necropsy. The post mortem study (Chapter 3) demonstrated both the need for thorough post mortem examinations to investigate sudden death cases and that acute pulmonary oedema, congestion and haemorrhage were common findings in Thoroughbreds dying suddenly whilst racing or training in Victoria (Chapter 3, Boden *et al.* 2005).

The risk of fatality in flat starts remained stable for city racecourses over the 15 year study period whereas those held on country racecourses appeared to increase over time. The latter increase may simply reflect greater stringency of reporting of fatalities by country racecourse officials to Racing Victoria Ltd. following the introduction of formalised reporting methods for fatalities and increased numbers of Racing Victoria Ltd. veterinarians attending country race meets after 2001-2002. The possibility that the overall increase over time was influenced by the observations in the later years of the study must be considered as a comparison of the individual risk ratios per year do not support a linear trend.

Although there were fluctuations in the risk of fatality in jump racing from year to year, there was no significant change in the risk over the 15 year period. During the study period, three Victorian industry reviews of fatalities in jump racing were conducted to determine strategies to decrease the risk of fatality and injury of horses and riders in jump races (Racing Victoria Ltd. 2002). These reviews were prompted by a Senate Inquiry into animal welfare in

Australia and by lobbying from Victorian animal welfare organisations. The risks associated with jump racing were formally reviewed in 1994, 1998 and in 2002 (Benton 1994; Racing Victoria Ltd. 1998 and 2002), with submissions from racing industry personnel, veterinarians and animal welfare groups being considered at each review. The initiatives arising from these reviews included a reduction of maximum hurdle height to no more than one metre, standardisation of the design of the log takeoffs in steeplechase events, imposition of a maximum field limit of 14 horses, introduction of artificial brush borders on steeple fences at Flemington racecourse, the addition of white “sighter” lines on all obstacles, repositioning of fences that were found to have high fall rates and improvement of qualifying conditions for horses in jump races. In the 2002 review, 38 recommendations were made to improve the overall safety of jump racing in Victoria. By 2005, 33 of these had been fully or partially implemented. The most noteworthy recommendations were the introduction of new hurdle and fence designs and the seasonal restriction of jump races between March and November.

It is difficult to accurately assess the impact on the risk of racehorse fatality of the interventions recommended by the Victorian jump racing reviews because the changes were implemented gradually post-review at several time points and not simultaneously on all racecourses. Also, only two years had elapsed between completion of the third review and the end of the study period. Because the implementation of the multiple interventions was not systematic, it is impossible to determine which of them may have been beneficial, deleterious or completely ineffective. However, the results of this study suggest that the recommendations of at least the first two of these reviews in 1994 and 1998 have had little long-term effect on the risk of fatality in jump races. This finding highlights the need for systematic application of interventions, for cost-benefit analyses and for well-designed and scientifically rigorous observational studies to assess the effect of specific intervention measures to reduce the risk of jump racing fatalities in Victoria. There is also a need for studies such as those undertaken in the UK on fatal fractures (Parkin 2002; Parkin *et al.* 2000, 2004a, 2004b and 2004c) and falls (Pinchbeck *et al.* 2002 and 2003) to identify risk factors associated with specific types of injury.

It is important to note that, although the risk of fatality for an individual racehorse competing in Victoria was much greater in jump starts than in flat starts, only 36% of the overall risk of fatality was attributable to jump racing and that most fatalities in the population occurred in flat racing (61% or 316/514). The impact of flat racing fatalities on the entire racing population warrants targeting of research nationally into fatalities in flat races. However, further reductions in the risk in flat starts may be difficult to achieve given that the risk in Victoria is already low and compares more than favourably with that in other countries (Peloso *et al.* 1994; Estberg *et al.* 1996b; Wood *et al.* 2002). In contrast, the risk in jump starts in Victoria is much greater than that overseas and investigation of the associated risk factors should also be prioritised by the Victorian industry, especially as 72% of all Australian jump starts occur in Victoria (Australian Racing Board Ltd. 2003).

A limitation of the study was that any clustering of outcome could not be accounted for in the analyses. Clustering of outcome was a possibility because racehorses could have more than one start over the study period and multiple starts by the same horse cannot be considered to be statistically independent of each other. Clustering could also have occurred at the level of the race, the racetrack and the trainer. Data could only be retrieved from the Australian Associated Press Pty. Ltd. database in an aggregated form as total numbers of starts by year; details of individual starts were not available. As a result, clustering of data could not be accounted for in the analysis and so the confidence intervals reported in this study may be artificially narrower than if clustering had been taken into account.

Conclusions

Considering that most jump races and a significant proportion of flat races in Australia are conducted in Victoria, the results of this study are in all likelihood relevant at a national level and provide benchmarks for both future monitoring of racetrack fatalities and assessing the effects of strategies introduced to minimise fatalities. Future research should focus on making accurate comparisons of the risk of fatality between countries. Furthermore, the emphasis for further research should be on development of monitoring programs that continually assess risk over shorter periods of time so that intervention strategies can be introduced in a more timely manner if the risk measured is at an unacceptable level.

5. Risk factors for Thoroughbred racehorse fatality in flat starts in Victoria (1989-2004)

Introduction

International studies have identified important risk factors for injury or fatality in flat racing. These include: horse- and prior racing history-related risk factors such as age (Estberg *et al.* 1996b; Williams *et al.* 2001), gender (Estberg *et al.* 1996a and 1998; Cohen *et al.* 1999; Hernandez *et al.* 2001), exercise intensity (Estberg *et al.* 1995 and 1996b; Cohen *et al.* 1997; Hernandez *et al.* 2001; Parkin *et al.* 2004a) and shoeing (Kane *et al.* 1996; Hill *et al.* 2001); race-related risk factors such as race type (chase, hurdle, National Hunt and flat) (Williams *et al.* 2001), race number (Cohen *et al.* 1999) and race distance (Peloso *et al.* 1994; Parkin *et al.* 2004b), and track-related factors such as track rating (Rooney 1982a; Williams *et al.* 2001).

These studies have identified different risk factors for severe injury or fatality of racehorses during flat racing. The disparity suggests that risk factors may differ amongst countries and even regions within countries and that research into risk factors should be focused at a regional level.

In Australia, only two studies have examined risk factors for catastrophic musculoskeletal racing injury, one in flat races on a selection of metropolitan courses in Sydney (Bailey *et al.* 1997) and the other in flat and jump (hurdle or steeplechase) races combined on all four metropolitan racecourses in Melbourne (Bailey *et al.* 1998). Because the latter study utilised data encompassing all race types, risk factors specific to different race types could not be determined. Differences between the risk of fatality in flat and jump races (for example, the absence or presence of obstacles) and between the populations of horses that participate in flat and jump races in Victoria (for example, horse age and prior racing experience) (Bailey *et al.* 1998) make it likely that the risk factors for fatality will differ according to race type. The objective of the current study was to identify and generate hypotheses about the risk factors that are important for fatality in flat starts on city and country racecourses in Victoria.

Results

Potential risk factors for fatality in flat starts in Victoria between August 1, 1989 and July 31, 2004 were assessed using a retrospective case-control study with 283 cases and 3307 controls.

The racing year in Australia begins on August 1 and ends on July 31. Between August 1, 1989 and July 31, 2004, study starts in flat races were recorded at four city racecourses and 62 country racecourses in Victoria. Over the 15 year period, 316 flat racing fatalities were reported to RVL. Of these, 33 were subsequently excluded (32 due to discrepancies in horse identity, race date, race location, race number and/or performance history and one due to a discrepancy in race type). There were 283 cases and 3307 controls included in the study. The 3590 study starts were represented in the study population by 3429 horses, 591 jockeys, 1218 trainers and 68 racecourses. The study starts occurred in 3551 races at 2915 race meets and on 2310 race dates.

Risk factors

Gender of the horse was selected at the level of the start and was categorised as entire male, entire female or gelding (castrated male). Track rating (going of the track) was used to describe the racing surface (fast: very firm surface, good: firm surface, dead: track with give in the ground, slow: rain-affected, heavy: very rain-affected) (Racing Victoria Ltd. 2006). Speed of the race was calculated by dividing the winner's time (in seconds) by the race length (in metres). Calendar age was calculated by subtracting each horse's actual birth date from the date of the case or control start. Racing age, based on the convention in the southern hemisphere that 1 August is the official birthday of the horse, was not used. Racing career length (expressed in years) was determined by subtracting the date of the first official start from the date of the case or control start. Information about the number of prior starts (in flat and jump races) and the distance raced (in metres) was also refined to quantify prior racing history during different time periods of interest (14, 30, 60 and 90 days prior to the case or control start and various combinations of these periods).

The study had statistical power of 82% to detect an association as significant at the 0.05 level if the odds ratio in the population was ≥ 1.7 and the prevalence of exposure in the control group was 0.1.

Further details of the descriptive statistics and the univariable analyses are available in Boden *et al.* (2007a). In the final multivariable model (Table 14), fatality was associated with gender, prior racing history, race length, racing year, racecourse location and track rating. Compared with geldings, stallions had greater odds of fatality (OR 2.2, 95% CI 1.4-3.5) and mares had lower odds of fatality (OR 0.6, 95% CI 0.5-0.8). Horses that had at least one start in the previous 31-60 days had 1.3 times (95% CI 1.0-1.8) the odds of fatality, compared with horses that had no starts during the same period. Increasing prior (career) jump distance was associated with decreased odds of fatality (OR per extra 1000 metres 0.96, 95% CI 0.9-1.0). Increasing race length was associated with increased odds of fatality (OR per extra 1000 metres 1.5, 95% CI 1.1-2.0).

Two interaction terms created between racing year and racecourse location and between racing year and track rating were significant in the final model. The odds of fatality were greater on city tracks than on country tracks in 1989-1990 (OR 3.1, 95% CI 1.7-5.4) but this decreased over time so that there was no effect of racecourse location in 2003-2004 (OR 0.9, 95% CI 0.5-1.5). The odds of fatality were greater on fast or good tracks than on heavy slow or dead tracks in 1989-1990 (OR 3.0, 95% CI 1.6-5.7). However, this difference decreased with every extra year of the study period until 2003-2004 when the odds ratio was 1.1 (95% CI 0.7-1.8). The odds ratio per racing year was 1.14 (95% CI 1.07-1.21, p -value < 0.001) on heavy, slow or dead country tracks and 1.06 (95% CI 1.02-1.10, p -value = 0.007) on fast or good country tracks. The odds ratio per racing year was 1.04 (95% CI 0.97-1.12, p -value = 0.25) on heavy, slow or dead city tracks and 0.97 (95% CI 0.92-1.03, p -value = 0.29) on fast or good city tracks. Other interaction terms created between racing year and other variables in the final model were not significant.

The multivariable model was adjusted for clustering using racecourse as a random effect. There were minimal changes in coefficients and standard errors compared with a single level model.

The final multivariable model was not affected by influential covariate patterns. There were sufficient cases per coefficient to justify the complexity of the model (Bagley *et al.* 2001). The Hosmer-Lemeshow goodness-of-fit statistic was 6.4 (8 degrees of freedom, p -value = 0.60). At a prevalence cut-off of 7.9%, the sensitivity and specificity of the model were 0.63 and 0.60 respectively, indicating modest predictive value. The area under the ROC curve was 0.66.

Table 14 Final Model - Risk factors for fatality in 3452 flat starts in Victoria between the 1989-1990 and 2003-2004 racing years with racecourse as a random effect

Risk factors for fatality	Coefficient	Standard error	<i>p</i> -value	Odds ratio (OR)	95% confidence interval
Gender			< 0.001		
Geldings				1 (REF)	
Entire males	0.78	0.243	0.001	2.18	1.36-3.52
Females	-0.48	0.151	0.002	0.62	0.46-0.83
Starts 31-60 days					
0 starts days 31-60				1 (REF)	
≥ 1 start days 31-60	0.28	0.149	0.057	1.32	0.99-1.77
Total prior (career) jump distance (per 1000 metres)^a	-0.044	0.027	0.023	0.96	0.91-1.01
Race length (per 1000 metres)^a	0.37	0.165	0.025	1.45	1.05-2.01
Racing year^b	0.13	0.031	< 0.001	1.14	1.07-1.21
Racecourse location					
Country racecourse				1 (REF)	
City racecourse	1.12	0.294	< 0.001	3.05	1.72-5.43
Interaction between racecourse location and racing year	-0.087	0.033	0.008	0.92	0.86-0.98
Track rating					
Heavy, slow or dead				1 (REF)	
Fast or good	1.11	0.319	<0.001	3.04	1.63-5.67
Interaction between track rating and racing year	-0.072	0.034	0.032	0.93	0.87-0.99
Intercept	-4.59	0.388			

Bolded *p*-values are likelihood ratio test *p*-values and unbolded *p*-values are Wald test *p*-values.
^aOne unit change equals 1000 metres. ^bOne unit change equals one race year.

Discussion

This paper reports the results of the first Australian study to identify risk factors specific for fatality in flat races. In the multivariable model, one horse, two prior performance, one temporal and three course/race level variables were associated with the risk of a start resulting in fatality.

Starts by male horses were at greater risk of ending in fatality than those by female horses. It has been suggested that the difference in the odds of fatality between males and females may reflect the willingness of owners to remove injured female horses from racing earlier than injured males, with the females being salvaged for breeding purposes (Perkins 2005). Castrated males with no breeding value may be encouraged by owners to continue to race despite subclinical injury. Cumulative musculoskeletal injury during a more protracted racing career may place males at greater risk of fatality than females (Bailey *et al.* 1999). The effect of sex hormones on bone density and body weight and differences in behaviour amongst stallions, geldings and mares may also influence the risk of fatality and should be considered in future investigations.

The prior racing and training history of racehorses has been shown to be an important factor that predisposes some horses to increased risk of fatality and career-ending injury in racing (Estberg *et al.* 1995, 1996b and 1998). In the current study, prior racing history was investigated over different time periods (14, 30, 60 and 90 days preceding the case or control start). The current study demonstrated that the 31-60 day period prior to the study start was found to be the most important in terms of the association between accumulation of high speed exercise and fatality. This result implies that there may be a one to two month hazard period for catastrophic injury and death following accumulation of one or more racing starts in the 31-60 day period. This finding is consistent with Californian studies that have shown that accumulation of large amounts of high-speed exercise prior to a racing start predisposes Thoroughbreds to injury; however, the hazard period identified in the Californian studies was up to 30 days post-exposure (Estberg *et al.* 1995, 1996b and 1998).

Our findings are consistent with the hypothesis that horses accumulating high speed exercise also accumulate subclinical or clinical bone damage that then needs to be repaired (Poole and Meagher 1990; Stover *et al.* 1992; Hill *et al.* 2001). Bone remodelling involves a sequence of initial osteoclastic resorption followed by osteoblastic synthesis of bone matrix. As a result, there may be a high risk period during which bone contains microdamage and is temporarily osteoporotic and susceptible to injury (Riggs 2002). If a horse undertakes high speed exercise before remodelling is complete, the risk of catastrophic injury may be increased.

In contrast, two studies have identified that increased distance accumulated in the previous 30 days is associated with a *reduced* likelihood of musculoskeletal injury (Cohen *et al.* 2000; Perkins *et al.* 2005). The most plausible explanation for this finding is the 'healthy horse effect' or survival bias, in which fit horses are the ones more likely to be racing and therefore less likely to suffer a catastrophic injury. However, there is evidence to suggest that the association between cumulative exercise and risk of musculoskeletal injury is complex and non-linear (Verheyen *et al.* 2003; Parkin *et al.* 2004a; Perkins 2005). It may be that horses have different exposures to various risk factors at different stages in their training. Some horses may break down early in training, for example, due to poor conformation, osteochondrosis, poor adaptation to training or from being pushed too hard too early. Others survive early training and enter a period of low risk in which they are fit and tolerant of higher exercise loads. As exercise loads increase further, horses may enter another period of increased risk due to accumulation of significant exercise-induced microdamage.

The distance accumulated in jump races over the prior racing career was protective (OR 0.96 per 1000 metres) with respect to fatality. Although this risk factor was included in the final model (due to a significant likelihood ratio statistic), the Wald *p*-value was not significant and thus the confidence interval spanned 1.0 (95% CI 0.91-1.01). However, this does not mean that the association was unimportant. This protective effect may reflect the fact that, if horses compete in both flat and jump starts, they are more likely to be injured or killed in jump starts than in flat starts (Bourke 1994 and 1995; Bailey *et al.* 1998; Boden *et al.* 2006). In other words, those horses that can survive multiple jump starts may also be more likely to survive subsequent less 'risky' flat starts. Nevertheless, the possibility that accumulated jump distance genuinely affords a degree of protection against fatality in a subsequent flat race needs to be explored further.

In the current study, the odds of fatality increased 1.5-fold with every extra thousand metres of race length. Increasing race length has been identified as a risk factor in other studies (Rooney 1982b; Bailey *et al.* 1997; Wood *et al.* 2000 and 2002; Parkin *et al.* 2004b; Pinchbeck *et al.* 2004). Increasing race length may increase the chance of catastrophic injury and subsequent fatality by increasing the exposure time at risk. Additionally, increased race length may be associated with increased fatigue of horses.

The effect of modifying race length should be investigated further. However, targeting race length as a modifiable risk factor may be impractical for the Victorian racing industry as most high prize money flat races held during the prestigious Spring Carnival are of medium to long distance (between 1600 and 3200 metres).

In the final model, increased odds of fatality were observed on city racecourses compared with country racecourses in the first year of the study (OR 3.1, 95% CI 1.7-5.4). However, by racing year 2003-2004, there was no effect of racecourse location (OR 0.9, 95% CI 0.5-1.5). This finding may well be attributable to an apparent increase in numbers of fatalities on country racecourses due to more consistent reporting of fatalities by country racecourse officials to RVL after 2001 (McCaffrey *pers. Comm.*). Alternatively, it may reflect a greater success of improvements made to city racecourses than those made to country racecourses over the study period. Redevelopment was undertaken at one of the four city racecourses, but only at a few of the 62 country racecourses, during the study period. The changes included modifications to turf structure (to improve drainage) and track shape (including track camber), addition of chutes, removal of crossings, and improvement in the durability of track surfaces to achieve a narrower range of track rating (ideally good) throughout the calendar year (Stubbs 2004). The impact of these specific changes could not be assessed in this study as improvements were introduced at different time points and not on all racecourses.

In the current study, there were increased odds of fatality for racing starts on fast or good tracks compared with heavy, slow or dead tracks in the racing year 1989-1990 (OR 3.0, 95% CI 1.6 – 5.7). However, this odds ratio for fatality decreased with every extra year of the study period until 2003-2004 when no effect of track condition was evident (OR 1.1, 95% CI 0.7-1.8). In an investigation of racing injuries at two racetracks in Sydney, track condition was not identified as a risk factor (Bailey *et al.* 1997) and an earlier study in Victoria also found no association between track condition and catastrophic injury (Bourke 1994). However track condition has been shown to be an important risk factor in most studies of fatality or injury of racehorses during races (Rooney *et al.* 1982a; Clanton *et al.* 1991; Wilson *et al.* 1996; Bailey *et al.* 1997; Williams *et al.* 2001; Parkin *et al.* 2004b; Parkin *et al.* 2005) with increased risk associated with firmer tracks. These findings are consistent with research in Victoria that showed increased likelihood of musculoskeletal injury on tracks with lower water content (Bailey *et al.* 1998). The greater risk on a fast or good track is most likely to be due to greater ground resistance, with increased stress being placed on limb bones during repetitive loading (Clanton *et al.* 1991).

The decrease in the odds of fatality on fast or good tracks compared to heavy, slow or dead tracks over the period of the study is difficult to explain. There was increased odds of fatality per racing year on *country* tracks when the track rating was heavy, slow or dead (OR 1.14, 95% CI 1.07-1.21) and fast or good (OR 1.06, 95% CI 1.02-1.10). There was no association with fatality per racing year on *city* tracks when the track rating was heavy slow or dead (OR 1.04 95% CI 0.97-1.12) or fast or good (OR 0.97, 95% CI 0.92-1.03). The greatest relative increase over the study period was on country tracks where the going was heavy, slow or dead (OR 1.14). The racing industry recognises that the preparation of race track conditions is not always precise due to unforeseen fluctuations in weather conditions on race-days and characteristics of track composition (Racing Victoria Limited 2005). Possible differences in track maintenance between country and city racecourses may have occurred as a result of limited financial and material resources available to cope with increasingly severe drought conditions experienced in Victoria from 2002 onwards (Botterill and Fisher 2003). This may have resulted in poorer turf coverage and/or firmer going on country racecourses in the later years of the study period. Tracks with poorer turf cover may have some of the properties of dirt tracks when wet. On dirt tracks (in distinct contrast to well-turfed tracks), an *increase* in water content is associated with greater risk of musculoskeletal injury (Rooney *et al.* 1983). Therefore attempts in later years to improve track safety by watering the track surface may have inadvertently resulted in an increase in risk on country tracks.

In any event, the study findings indicate that ongoing investigation and review of track design, construction and maintenance should continue to be industry priorities. This relates especially to maintaining the moisture content of track surfaces during very dry periods. Further investigation of changes in track assessment and maintenance on city tracks over the 15 year study period will help in the formulation of guidelines for maintenance of country tracks in order to reduce the risks of injury or death to horses racing on them.

The Hosmer-Lemeshow goodness-of-fit statistic indicated that there was no evidence that the model did not fit the data well and therefore the model was considered to be reasonably calibrated. At a prevalence cut-off of 7.9%, the sensitivity and specificity indicated modest predictive ability. Based on the rating system described by Hosmer and Lemeshow (2000), the discriminatory ability of the model was just below an acceptable level (ROC curve < 0.7). This may be because of the limited nature of the data utilised in the analysis. A proportion of racing fatalities may be caused by specific events that were neither measured nor measurable in this study. Additionally, as the data were obtained from race form histories, only proxy measures of training schedules could be derived. It is likely that potential risk factors other than those identified in this study (for example, in training patterns) may be important. If so, inclusion of these factors would improve the discriminatory ability of the model.

This study has identified specific risk factors for fatality in flat racing in Victoria. The results should be utilised to direct further research into starts, horses and races at high risk so that appropriate intervention strategies can be implemented to improve the welfare of horses and jockeys racing in Victoria.

6. Risk factors for Thoroughbred racehorse fatality in jump starts in Victoria (1989-2004)

Introduction

In Australia, there are two forms of jump racing: hurdling and steeplechasing. Hurdle races include jumps over brush fences that stand at approximately one metre high whereas steeplechase fences are higher (at least 1.15 metres in height and, on two city tracks in Victoria, up to 1.4 metres high). Overall, jump races account for less than one percent of all races held in Australia but 82% of all Australian jump races are held in Victoria (Australian Racing Board Ltd. 2003).

Jump races have been shown to be associated with a higher risk of fatality than flat races in the UK (Wood *et al.* 2002). However, there have been remarkably few studies in the UK (Wood *et al.* 2000 and 2002; Pinchbeck *et al.* 2002, 2003 and 2004) that have specifically addressed risk factors for fatality or injury in jump races. In the USA, where fewer horses compete in jump races, Stephen *et al.* (2003) investigated various types of injury sustained by horses and the associated risk factors in official races of the Virginia Steeplechase Association between 1996 and 2000. Most bone fractures were found to be caused by falls and most were sustained in hurdle races. The latter study suggested that age, previous injuries, quality of the horse, experience of the rider and design of the track could account for differences in the prevalence of various injury types in steeplechase and flat races (Stephen *et al.* 2003).

Three studies have described the risk of fatality in jump races in Victoria (Bourke 1995; Bailey *et al.* 1998 and Boden *et al.* 2006). All three studies demonstrated an increased risk associated with fatality in jump starts compared with flat starts. Whereas the risk of fatality in flat starts was 0.44 per 1000 starts, the risk in jump starts was 8.3 per 1000 starts or 18.9 times that in flat starts (Boden *et al.* 2006). The risk of fatality in Victorian jump racing was twice that reported in the USA (3.9 per 1000 starts) (Stephen *et al.* 2003) and 50% greater than that reported in the UK (5.6 per 1000 starts) (Wood *et al.* 2002).

In recent years in Victoria, jump racing has become a controversial issue because of the high risk of fatality relative to flat racing (Bailey *et al.* 1998; Boden *et al.* 2006) and the merits of its continuing existence have been debated by both the public and the racing industry. Since 1994, there have been three Victorian jump racing reviews that led to recommendations designed to improve the safety of jump racing (Benton 1994; Racing Victoria Ltd. 1998 and 2002). Despite these recommendations, the continued high risk of fatality in jump races has drawn increasing criticism from Australian animal welfare groups. In response, the Victorian racing industry has demonstrated a commitment to improve the welfare of horses in jump racing by investigating and attempting to reduce the risk. This study is the first to investigate the risk factors that are important for fatality in jump starts in Victoria.

Results

Over the 15 year period, 198 jump racing fatalities were reported to RVL. Of these, seven were excluded due to discrepancies in horse identity, race date, race location and/or race number. There were 191 cases and 2324 controls enrolled in the study. The 2515 jump starts in the study represented 1551 horses, 218 jockeys, 552 trainers, 1659 races, 1478 race meets and 1387 race dates. The starts were recorded at four city racecourses and 37 country racecourses. Within the study period, there were six races in which there were multiple fatalities. Five races had two fatalities and one race had three fatalities.

The study had statistical power of 90% to detect an association as significant at the 0.05 level if the odds ratio (OR) in the population was ≥ 2.0 and the prevalence of exposure in the control group was 0.1.

Further details of the descriptive statistics and the univariable analyses are available in Boden *et al.* (2007b). In the multivariable model (Table 15), the duration of the racing career, the numbers of flat, hurdle and steeple starts accumulated in the 60 day period prior to the case or control start, the numbers of flat and jump starts the horse accumulated in the prior racing career, if the horse had a start between one and 14 days prior to the case or control start, the type of jump race (hurdle or steeplechase), the calendar year of the start and the location of the racecourse emerged as significant. Of these variables, the numbers of prior racing career flat and jump starts, starts between one and 14 days prior to the case or control start, steeplechase races and city racecourses were associated with increased odds of fatality. Racing career duration and the numbers of flat, hurdle and steeple starts in the 60 day period prior to the case or control start were associated with decreased odds of fatality.

An interaction term was created to evaluate the interaction between the number of flat and jump starts accumulated throughout the prior career. This was not significant in the final model (p -value = 0.083).

Interaction terms between the risk factors included in the multivariable model and calendar year categories were created to investigate the possibility of an effect of time on the exposures of interest. No interactions of calendar year category with any of the risk factors were identified, with all nine possible interactions having a likelihood ratio test p -value > 0.60 . There were sufficient cases per coefficient to justify the complexity of the model (Bagley *et al.* 2001). The Hosmer-Lemeshow goodness-of-fit statistic for the fixed effects model was 7.16 (8 degrees of freedom, p -value = 0.52). This indicated that there was no evidence that the model did not fit the data well. At a predicted probability cut-off of 7.8%, the sensitivity and specificity of the model were 0.62 and 0.71 respectively. The area under the ROC curve was 0.72.

Table 15 Final multivariable model for risk factors for fatality in 2297 jump starts in Victoria between the 1989-1990 and 2003-2004 racing years with racecourse as a random effect

Risk factors for fatality	Coefficient	Standard error	<i>p</i> -value	Odds ratio (OR)	95% Confidence interval
Racing career duration (years) ^a	-0.41	0.070	< 0.001	0.66	0.58-0.76
Number of flat starts 1-60 days prior to start ^b	-0.30	0.069	< 0.001	0.74	0.64-0.85
Number of hurdle starts 1-60 days prior to start ^b	-0.28	0.068	< 0.001	0.76	0.66-0.87
Number of steeple starts 1-60 days prior to start ^b	-0.42	0.090	< 0.001	0.65	0.55-0.78
Total prior flat starts in career (per 10 starts) ^c	0.34	0.042	< 0.001	1.40	1.29-1.53
Total prior jump starts in career (per 10 starts) ^c	0.44	0.082	< 0.001	1.56	1.32-1.83
Total number of starts 1-14 days prior to start					
0 starts				1(REF)	
≥ 1 starts	0.43	0.189	0.019	1.54	1.07-2.24
Jump type					
Hurdle race				1(REF)	
Steeplechase race	0.93	0.226	< 0.001	2.53	1.63-3.94
Calendar year of start					
1989-1992				1(REF)	
1993-1996	0.11	0.230	0.64	1.11	0.71-1.74
1997-2000	-0.59	0.261	0.02	0.55	0.33-0.93
2001-2004	-0.27	0.267	0.31	0.76	0.45-1.29
Racecourse location					
Country racecourse				1(REF)	
City racecourse	0.44	0.194	0.024	1.56	1.06-2.28
Intercept	-2.14	0.334			

Bolded *p*-values are likelihood ratio test *p*-values and unbolded *p*-values are Wald test *p*-values.
^aOne unit change equals one year. ^bOne unit change equals one start. ^cOne unit change equals 10 starts.

Discussion

This paper reports the results of the first Australian study to identify risk factors for fatality in jump races. In the final multivariable model, seven prior race performance, one temporal and two course/race level variables were associated with the risk of a start resulting in fatality.

Prior racing history was associated with the odds of fatality in jump starts. Jump starts by horses with longer racing careers were less likely to result in fatality than those by horses with shorter racing careers but horses that had started in more flat starts prior to the case or control start were more likely to die in the next jump start than horses that had accumulated less flat racing experience. Additionally, increased numbers of jump starts prior to the study start were also associated with increased odds of fatality. These findings were not unexpected as accumulated racing distance has been associated with catastrophic injury (Johnson *et al.* 1994a and 1994b; Estberg *et al.* 1995, 1996 and 1998) and catastrophic injury is the leading cause of death in racing Thoroughbreds (Vaughan and Mason 1976; Mohammed *et al.* 1991; Bourke 1994; Johnson *et al.* 1994a and 1994b; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995; Parkin *et al.* 2004; Boden *et al.* 2006).

After adjusting for numbers of prior career starts, odds of fatality in jump starts were lower for horses with more years of racing experience than for horses with fewer years of racing experience. Assuming that pre-existing injury is associated with risk of fatality due to catastrophic injury in a start, this finding may be due partially to the removal of horses that sustain injuries early on in their racing careers (Mohammed *et al.* 1991), leaving a population

of horses more resilient to the rigors of racing. Persisting with suspect animals early in their careers might also contribute to the association of risk with less experienced race horses, if older horses were more rapidly removed from racing.

The numbers of flat and jump starts accumulated in the racing career of a horse were both associated with increased odds of fatality in jump starts. The results of the current study are consistent with the hypothesis that jump horses in Victoria accumulate bone, ligament and/or tendon damage because of repetitive limb strains and loading incurred during high speed exercise in prior flat and jump starts, and that this damage predisposes them to catastrophic limb injury.

The associations between the numbers of prior racing career flat and jump starts and increased odds of fatality were adjusted for racing career length in the final model. Therefore the results of the study suggest that accumulation of large numbers of flat and jump starts over a short racing career length (i.e. increased intensity of racing exercise) is an important risk factor for fatality. Other studies have also demonstrated that the odds of fatality increased for horses accumulating increased amounts of high-speed exercise, albeit over short periods of time (Estberg *et al.* 1995, 1996 and 1998; Cogger *et al.* 2003).

In light of the findings of the current study, it may be appropriate for the Victorian racing industry to set limits on the number of flat starts a horse may have before it commences jump racing. Ideally, horses should be trained specifically as jump horses, as is the case for the majority (75%) of horses in the UK (Ely *et al.* 2004). Such an intervention would be expected to have an important impact on the odds of fatality in jump starts even though the odds would still increase for each additional jump race in the career of a horse.

The odds of fatality in jump starts were lower for horses with more starts in flat, hurdle and/or steeplechase events in the 1-60 days prior to the study start than for horses with fewer starts in the same event categories. This does not necessarily mean that there was no adverse effect of accumulating more starts 1-60 days prior to the race start especially given the increased odds of fatality associated with accumulation of prior starts in jump and flat racing. Indeed, exposures in the period close to the next race start might be expected to contribute more than earlier exposures to the risk of catastrophic musculoskeletal injury at the next start because any recently sustained bone or soft tissue microdamage may be incompletely repaired by the time of the next start. That an adverse association of accumulating more starts in the 1-60 day period was not observed in the study may be due to survival bias ('healthy horse effect') in which uninjured horses are the ones most likely to be fit for racing in this time period. Other studies have identified that increased distance accumulated in the 30 days prior to a start is associated with a reduced likelihood of musculoskeletal injury at that start (Cohen *et al.* 2000; Perkins *et al.* 2005). In both studies, the researchers attributed this finding to the 'healthy horse effect'.

The risk factors associated with prior racing history in the 60 day period highlight potential pitfalls in the interpretation of risk factors in observational epidemiology. If the association between the numbers of starts in the prior 60 days and fatality was interpreted as causal, the racing industry could enforce an increase in the numbers of flat, hurdle and steeple starts in order to decrease the odds of fatality. However, assuming the 'healthy horse effect' explains this observed association, such an intervention would be ineffectual and might even result in an increase rather than a decrease in fatality in racing.

The finding of decreased odds of fatality for increased numbers of starts in the 60 day period takes into account the increased odds of fatality for horses with one or more starts within 14 days of the study start. Thus, the findings suggest that jump starts by horses with more starts in flat, hurdle and/or steeplechase events in the 60 days prior to the study start were less likely to result in fatality as long as the latter starts were not accumulated in the 14 days prior to the

case or control start. This association is consistent with previous studies in flat racing that have demonstrated that accumulation of high-speed exercise predisposes Thoroughbred horses to injury for a hazard period of approximately 30 days following exposure (Estberg *et al.* 1995, 1996 and 1998).

The tight scheduling of jump events may have contributed to the increased prevalence of exposure in horses starting within 14 days of a previous start. In Victoria, the jump racing year is condensed into seven months (Australian Jumping Racing Association 2003). The interval between jump events ranges from two to 28 days, with many events being scheduled within seven days of each other. The schedule is even more condensed for steeplechase events than for hurdle events, although horses seldom compete in every scheduled event. Insuring that the interval between jump starts exceeds 14 days could reduce the risk of serious injury. Further investigation of inter-race intervals could be useful future research in order to further reduce the risk of injury in jumps events.

The odds of fatality were increased (OR 2.53, 95% CI 1.63-3.94) for steeplechase starts relative to hurdle starts. This is consistent with the finding of Bailey *et al.* (1998). Obstacles in steeplechase races are higher and more solid structures than brush hurdles. In Victoria prior to 2002, hurdles were approximately one metre in height and steeplechase fences were at least 1.15 metres in height and on two city tracks were 1.4 metres high. Differences in the range of race lengths (interquartile range for hurdle race length 2700 to 3700 metres, interquartile range for steeplechase race length 3200 to 3500 metres) and in the size and positioning of obstacles may be major contributors to the greater risk in steeplechases than in hurdles, although other factors such as horse age, ability and pre-existing injury may also explain the increased odds of fatality in steeplechases (Perkins 2005).

Increased odds of fatality were observed for starts on city racecourses compared with starts on country racecourses (OR 1.56, 95% CI 1.06-2.28). This is likely to be attributable to underreporting of fatalities by country racecourse officials prior to 2001 or perhaps differences in race speeds, racing career lengths, racing career earnings and training regimens between horses racing in different locations. In addition, changes to fence and course design may alter the risk associated with individual tracks over time. Additional studies are needed to supplement what is already known with respect to track design and conditions, to enable the particular features of individual racecourses that have high rates of injury or fatality to be assessed. These racecourses can then be targeted for interventionist strategies.

In this study, the calendar year was significant in the final model. The period between calendar years 1997-2000 was associated with decreased odds of fatality (OR 0.55, 95% CI 0.33-0.93) but there was no significant difference in the odds of fatality in the period between calendar years 1993-1996 or 2001-2004 when compared with the period between calendar years 1989-1992. These findings are consistent with the previous descriptive study of Boden *et al.* (2006) that demonstrated that, even though the risk of fatality fluctuated from year to year between 1989 and 2004, the overall risk of fatality in jump racing in Victoria remained unchanged over the same time period (relative risk per year 0.99, CI 0.95-1.02).

The risks associated with jump racing were formally reviewed by the Victorian racing industry in 1994, 1998 and in 2002 (Benton 1994; Racing Victoria Ltd. 1998 and 2002). These reviews led to recommendations to improve the safety of jump racing (Boden *et al.* 2006). It is difficult to accurately assess the impact of these interventions because the changes were implemented gradually post-review at several time points and not simultaneously on all racecourses, and only two years had elapsed between completion of the third review and the end of the study period (Boden *et al.* 2006). It is possible that the risk of fatality in jump races declined between the years 1997 and 2000 as a delayed effect of some

of the interventions recommended by the Jump Review Panel in 1994. However, it is impossible from this study to distinguish the impact of the recommendations of the review panels on the risk of fatality in jump starts from other confounding factors such as changes in weather or track conditions over the same period. Moreover, if the recommendations of the Jump Review Panel in 1994 did have a significant impact on the risk of fatality in jump starts, the effect was short-lived. There is no evidence to suggest that the risk of fatality in 2003-2004 is significantly different from that in 1989-1990 (Boden *et al.* 2006). This finding highlights the need for systematic application of interventions and for well-designed and scientifically rigorous observational studies in the future to assess the effect of specific intervention measures aimed at reducing the risk of jump racing fatalities in Victoria (Boden *et al.* 2006).

The Hosmer-Lemeshow goodness-of-fit statistic for the fixed effects model indicated that there was no evidence that the final model did not fit the data well. Based on the ROC curve rating system (Hosmer and Lemeshow 2000), the model had reasonable discriminatory ability. The model only assessed factors associated with the horse, the prior racing history of horses that started in jump races, the race, the jockey and the track. The discriminatory ability of the model might have been improved had it been possible to consider factors that occurred within the race (such as the numbers of obstacles and obstacle placement), as previous studies have already demonstrated that intra-race factors are associated with falling and injury (Pinchbeck *et al.* 2002; Parkin *et al.* 2006).

This study is the first in Victoria to examine risk factors associated with fatality in jump starts. The results will inform the development of interventions to reduce the risk in jump starts in Victoria in the future and to generate hypotheses on which future prospective studies can be based. The study was timely as it corresponded with the release of the 2005 Jumping Race Review Panel Report (Racing Victoria Ltd. 2005). The latter has recommended that jump racing should continue in Victoria but that further interventions to improve safety are necessary. The success of this review will largely depend on the commitment of the Victorian racing industry to continue to monitor fatalities in jump racing and on detailed scientific analysis of the effect of the recommended interventions.

7. General Discussion

Introduction

This thesis was the first in Australia to combine a prospective post mortem case series and large-scale retrospective observational studies to investigate the causes of and risks associated with racehorse fatality in flat and jump starts. The study was encouraged by the Victorian racing industry in response to industry and community concerns regarding the wastage rates of racehorses as well as jockey injuries and fatalities in racing. The purpose of this study was to identify risk factors that could be modified to reduce the risk of fatality in Thoroughbred racing in Victoria.

Strengths and limitations of the study

A strength of the risk factor studies (Chapter 5 and 6, Boden *et al.* 2007a and b) was the large numbers of cases enrolled in each study (due to the extensive time frame) and subsequent excellent statistical power to identify important associations if associations existed. These studies were also the first studies in Australia to investigate the association between prior racing form history and fatality.

The challenges in this thesis primarily revolved around the retrospective nature of the studies and subsequently the access to and validity of the racehorse performance data.

In the studies presented in the thesis, the likelihood of selection bias was small because 90% of all fatality cases reported to Racing Victoria Ltd. were included in the study. There were no obvious indications of bias in the horses that were excluded with respect to the distribution of exposures. There was a possibility that small numbers of horses that died in racing (for example, on remote country racecourses in the earlier years of the study) may not have been reported to Racing Victoria Ltd, particularly prior to 1998. However, failure to report a fatality sustained on a race day was unlikely due to the attendance of racecourse veterinarians at every race meet. The likelihood of reporting error would be higher for those horses that were severely injured in racing and referred to off-site veterinary clinics and euthanised at a later date (more than 24 hours after a racing start). These losses from the study population were minimised by a strict case definition that excluded horses that were not euthanised within a 24 hour period post-race.

The possibility of bias due to selection of control starts was relatively small given that a random sampling method was used to select a large number of controls (approximately 12 control starts for each case start) from the entire database of starts between August 1, 1989 and July 31, 2004.

There was evidence to suggest that there was sampling bias in the horses submitted for post mortem examination. Although the fatalities submitted from the city racecourses were highly representative of all city fatalities due to the mandatory submission protocol, elective submissions from the country were skewed towards sudden death cases. Further discussion of this limitation and methods to reduce the impact of bias in future studies occurs in this chapter and Chapter 3.

Collection of standardised data from the horses that were submitted for post mortem examinations was excellent due to a pre-determined post mortem protocol. To ensure consistency for histological specimens, one specialist veterinary pathologist was responsible

for grading all pulmonary lesions identified in horses submitted over the study period, with the grading based on the recorded description of the gross appearance of the lungs at necropsy and on detailed review of the histological sections of the lungs. The post mortem study is continuing at The University of Melbourne. If, in future, the post mortem examination protocol were to be expanded (for example, by analysis of point-of-death blood samples, or computed tomography of bone samples), it may not be possible to compare diagnoses with those made in the study presented in Chapter 3. Lack of consistent analysis may become an important issue for future studies based on the post mortem examination records. In anticipation of such changes, a relational database containing comprehensive information on every post mortem examination has been developed. It will be important to maintain consistency of record keeping and compliance with standardised protocols for the success of future studies that utilise the data.

A limitation of this research was the lack of access to the training histories and prior medical histories of the racehorses in the study population. The retrospective nature of the descriptive and observational studies over a long time period meant that bias due to misclassification of prior training data would have been likely, even if trainers had been contactable. Variables identified from training and medical history data may improve the sensitivity and specificity of the models described in Chapters 5 and 6 for risk factors in flat and jump starts respectively (Boden *et al.* 2007a and b). Additionally, more refined variables describing within-race factors such as position of the horse within the field or the number and placement of obstacles would also improve the discriminatory ability of these models.

Important findings of the study

The post mortem study (Chapter 3, Boden *et al.* (2005)) was the first comprehensive post mortem study of racehorse fatalities to be conducted in Australia. Together with the analysis of fatality reports submitted by veterinarians between 1989 and 2004 (Chapter 4), it enabled accurate estimation of proportional mortality rates and of the risk of fatality for Thoroughbreds in racing according to the specific cause of death.

The post mortem study confirmed that most fatalities of Thoroughbred racehorses in racing and training in Victoria between 2001 and 2004 were the result of euthanasia due to catastrophic limb injury. The proportion of all fatalities due to catastrophic limb injury in the post mortem study (74%, CI 63.3-82.5%) was similar to the proportion reported by racecourse veterinarians and racing stewards to Racing Victoria Ltd. over the 15 year study period (72%, CI 67.5-75.3%).

Of single limb injuries, the most common injury involved the distal limb (30/40, 75%) and a forelimb (32/40, 80%) was more frequently affected than a hindlimb (Chapter 3). This was consistent with previous studies in Australia and overseas (Vaughan and Mason 1976; Mohammed *et al.* 1991; Bourke 1994; Johnson *et al.* 1994a and 1994b; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995; Parkin *et al.* 2004a). The proportion of horses with a single catastrophic left limb injury was not significantly different from the proportion of horses with a single catastrophic right limb injury (p -value = 0.43). This was surprising, given the unidirectional anticlockwise mode of racing in Victoria, and contrasts with the reported predisposition of the right limb to injury in the UK (Vaughan and Mason 1976; Parkin 2002; Parkin *et al.* 2004a) and the predisposition in the USA of the left limb to injury during racing and of the right limb during training (Rick *et al.* 1983; Rooney 1983a; Johnson *et al.* 1994a).

The most common catastrophic musculoskeletal injury in horses submitted for post mortem examination was fracture of metacarpus III or metatarsus III. The second most common injury

was concurrent fracture of metacarpus III or metatarsus III and the proximal sesamoid bones and phalanx I. In veterinary reports between 1989 and 2004, metacarpus III or metatarsus III fractures were the most commonly reported injury, followed by fractures of the proximal sesamoid bones. These findings contrast with those of overseas studies (Vaughan and Mason 1976; Mohammed *et al.* 1991; Peloso *et al.* 1994; McKee 1995; Parkin *et al.* 2004a). In North American studies, fractures of the proximal sesamoids were the most common injuries, followed by fractures of metacarpus III and carpal bones (Mohammed *et al.* 1991; Johnson *et al.* 1992 and 1994; Peloso *et al.* 1994). In the UK, lateral condylar fractures of metacarpus III were the most common racing injuries (Vaughan and Mason 1976; McKee 1995; Parkin *et al.* 2004a). Differences between countries in the anatomic location of fractures may point to differences in the pathogenesis of different fracture types. At present, the numbers of cases of catastrophic limb fractures in Victoria are insufficient to support a study of risk factors for specific types of fracture such as those conducted by Parkin (2002) and Parkin *et al.* (2005b). A specific post mortem investigation targeting catastrophic fractures of the distal limb in all fatality cases from city and country racecourses would enable larger numbers of cases to be collected for precise fracture diagnosis and thus would be useful to investigate the pathogenesis and specific risk factors for different types of limb fractures.

The post mortem study also allowed accurate identification of the cause of sudden death of horses during racing and training. Pathologists attributed most sudden deaths to cardiovascular or respiratory failure. Acute pulmonary oedema, congestion and haemorrhage were common findings in horses dying suddenly whilst racing or training in Victoria. The acute pulmonary oedema was attributed to increased pulmonary capillary hydrostatic pressure. Although pulmonary hypertension is known to develop in racehorses during high-speed exercise, upper airway obstruction and left-sided cardiac failure were considered as alternative causes of the oedema. Further investigation is required to conclusively determine the causes of sudden death of racehorses. Specifically, future investigations may be facilitated by collection of point-of-death blood samples for electrolyte analysis and assay of markers of peracute myocardial injury (e.g. cardiac troponin I and cardiac-specific muscle enzymes).

In the three year post mortem study, sudden death accounted for 26% (CI 17.5-36.7%) of all fatalities on Victorian racecourses and 32% of all fatality cases submitted for post mortem examination. In contrast, only 9% (58/659) of Thoroughbred racing fatalities in a Californian study (Johnson *et al.* 1994a) and 12% (15/127) in a British study (Vaughan and Mason 1976) were sudden deaths. However, in veterinary reports to Racing Victoria Ltd. covering the entire study period between 1989 and 2004, sudden deaths comprised only 13% (95% CI 10.4-16.2%) of all racecourse fatalities, a proportion that is comparable to those of international studies. The higher proportion of sudden deaths in the post mortem study than in the racing population was due most likely to sampling bias. Horses submitted for necropsy were highly representative of all city fatality cases because of the mandatory submission protocol. In contrast, the horses submitted from country racecourses were not representative of all country fatalities, with sudden deaths being over-represented. The non-random sampling strategy for submission of horses from country racecourses was a limitation of the post mortem study.

Post mortem examination allowed accurate classification of causes of fatality. Results of that study showed that, in the absence of a post mortem examination, there was potential for misclassification of the cause of fatality by racetrack veterinarians. The post mortem study was used to validate the diagnoses recorded in fatality reports by veterinarians and confirmed that the latter diagnoses were accurate for broadly categorising fatalities (catastrophic limb injury, cranial or vertebral injury or sudden death). As a result, additional studies employing these broad case definitions based on the fatality reports (Chapters 4, 5 and 6) could be undertaken with confidence. However, the validation process did highlight inaccuracies of veterinary reports in identifying the specific anatomic location of catastrophic limb injuries

and in determining the specific cause of sudden death in the absence of a post mortem examination. Redesign of the fatality report forms may improve the quality of data recorded in the future. Changing from open questions to option boxes may ensure more systematic examinations and recording. Similar changes to fatality report forms have been introduced in the UK in response to work by Parkin (2002).

A second limitation of the post mortem study was the relatively small number of cases ($n = 77$). This was unavoidable because Racing Victoria Ltd. implemented the post mortem policy only in February 2001. If future risk factor studies are to be based on diagnoses derived from post mortem examinations (in order to avoid bias due to misclassification of outcome), sample sizes must be large to ensure sufficient power to detect major risk factors. Due to the relatively low frequency of racing fatality in Victoria, long-term post mortem studies will be essential if these types of risk factor studies are to be feasible in the future.

In Chapter 4 (Boden *et al.* 2006), the risks of fatality were reported for both flat and jump starts over a 15 year period. This was the first study in Australia to assess the risk of fatality in racing over such a long period. The results provide important benchmarks for future monitoring of fatalities and evaluation of intervention strategies in Victoria.

The risk of fatality in flat starts was lower in Victoria (0.44 fatalities per 1000 starts) than that reported in North America (1.4-1.7 fatalities per 1000 starts) (Peloso *et al.* 1994; Estberg *et al.* 1996a) and the UK (0.8-0.9 fatalities per 1000 starts) (McKee 1995; Wood *et al.* 2002) but the risk of fatality in jump starts was greater in Victoria (8.3 fatalities per 1000 starts) than that reported overseas (3.9 fatalities per 1000 starts) in North America (Stephen *et al.* 2003) and 5.6 fatalities per 1000 starts in the UK (Wood *et al.* 2002). Differences in the risk of fatality in flat starts between countries may be due to differences in track surfaces (dirt racetracks in the USA or all weather in the UK versus turf racecourses in Victoria), shoeing practices (such as the use of toe grabs in the USA) and the timing and use of selected medications (such as phenylbutazone). The difference in risk in jump starts between Victoria and the UK may be due to differences in training regimens and previous racing career experience of jump horses and differences in the number and placement of obstacles, obstacle materials and design and track conditions. Ideally future studies of the risk of fatality in jump racing will compare the same risk factors in each of the racehorse populations in Australia, the UK and the USA. Although jump starts comprise only a small proportion of all racing starts in Australia, the majority (72%) of all Australian jump starts is convened in Victoria. If the risk of fatality in Australian jump starts is to be reduced, Racing Victoria Ltd. should prioritise research into the high risk of fatality in jump racing.

Trends in risk were also examined in Chapter 4. The risk of fatality in flat starts appeared to increase over the 15 year study period (Chapter 4 and Chapter 5) but it remains unclear whether this was due to a genuine increase in risk (resulting from changes in such factors as climate or racing practices over time) or to increased stringency of reporting of fatalities by country racecourse officials to Racing Victoria Ltd. after the introduction of a policy of increased attendance by veterinarians at country race meets and formalised reporting methods for fatalities post-2001-2002 (Boden *et al.* (2006 and 2007a). Additionally, the racing industry may have gradually changed its practices or attitude towards data recording, given the increase in public concern for and industry awareness of animal welfare issues following the publication of studies of racing fatalities in Australia (Suann 1992; Bourke 1994 and 1995) and overseas (Bathe 1994; Estberg *et al.* 1994, 1995a and 1995b; Johnson *et al.* 1994a and 1994b; Macdonald and Toms 1994; Peloso *et al.* 1994; McKee 1995).

The apparent increase in risk was strongly influenced by the low risk of fatality in 1989-1990 and high risks in 2000-2001 and 2001-2002, and appeared to be due to an increase in risk over time on country racecourses. If the increase in the odds of fatality over time were due

entirely to improved reporting, the true risk of fatality between 1989 and 2004 would be 0.58 fatalities per 1000 starts rather than 0.44 fatalities per thousand starts (Chapters 4 and 5).

The risk of fatality in jump starts remained constant over the study period despite jump racing reviews that recommended changes to hurdle and steeple races to improve safety (Boden *et al.* 2006). This suggests that the changes made following recommendations of at least the first two reviews (in 1994 and 1998) have had little effect. In 2005, Racing Victoria Ltd. initiated another review of jump races to examine the impact of the 2002 review (Racing Victoria Ltd. 2005). The 2005 review suggested that there had been an improvement in the risk of fatality and the risk of falling in jump races since 2002 and ascribed this decrease to the introduction of modular fences. However, the numeric decrease in the risk of fatality since 2001 was not statistically significant (p -value = 0.52) (Parkin and Boden 2005). Lack of statistical significance may have reflected the imprecision of effect estimates due to the limited period of time between the 2002 and 2005 reviews. To determine if a real decrease has occurred, the Victorian racing industry must continue to monitor racing fatalities in jump events over a longer time frame.

The risk of fatality in all race types was 1.1 per 1000 starts (CI 1.0-1.3) on city tracks and 0.57 per 1000 starts (CI 0.51-0.63) on country tracks. The risk of fatality on city racecourses was 2.0 (CI 1.7-2.4) times that on country racecourses. Within each race type, the risk of fatality was significantly higher on city than on country racecourses. This finding contradicted a preliminary hypothesis that poorer quality horses starting on country racecourses would be more likely to suffer catastrophic injury resulting in fatality than better quality horses starting on city racecourses. Races held on city racetracks are regarded as more prestigious and offer greater prize money than those on Victorian country tracks. Consequentially, there may be differences in race speeds, career lengths, career earnings and training regimens between horses that race in the different locations and these factors may be associated with the likelihood of death. It is possible that the increased likelihood of fatality on city racecourses reflects differences in the quality of horses racing on city versus country racecourses. It may be that better quality horses starting on city racecourses are raced more frequently and accumulate more training and racing distance prior to racing than those that start on country racecourses. Differences in track surface and design at the different locations may also influence the risk of fatality. Additionally, differences in the risk of fatality on city compared with country racecourses may be attributable to possible underreporting of fatalities by country racecourse officials before 2001 (especially in flat races).

In the descriptive study (Chapter 4, Boden *et al.* 2006), further investigation of factors such as speed, accumulation of starts and prize money was not possible. Thus, racecourse location was also considered as a risk factor in the case-control studies in Chapters 5 and 6. In jump racing, the odds of fatality were greater for starts on city racecourses than for starts on country racecourses. In flat racing, the increased odds of fatality were observed on city racecourses compared with country racecourses was apparent in the first year of the study but by racing year 2003-2004, there was no effect of racecourse location (Boden *et al.* 2007a). This finding may be due to an apparent increase in numbers of fatalities on country racecourses due to more consistent reporting of fatalities by country racecourse officials to RVL after 2001. Alternatively it may reflect a greater success of improvements made to city racecourses than those made to country racecourses over the study period. Redevelopment was undertaken at one of the four city racecourses, but only at a few of the 62 country racecourses, during the study period. The changes included modifications to turf structure (to improve drainage) and track shape (including track camber), addition of chutes, removal of crossings, and improvement in the durability of track surfaces to achieve a narrower range of track rating (ideally good) throughout the calendar year (Stubbs 2004). The impact of these specific changes could not be assessed in this study as improvements were introduced at different time points and not on all racecourses. (Boden *et al.* 2007a).

This association with location was still significant when speed (in the flat and jump models) and race career length (in the jump model) were taken into account. In the case-control studies (Chapters 5 and 6), differences in the odds of fatality between each individual racecourse were not assessed because of the large number of racecourses represented in the study. However, of the four city racecourses, the Moonee Valley racecourse appeared at a univariable level to be associated with a numerically increased odds of fatality in flat starts (when compared with the Flemington racecourse) and a significantly increased odds of fatality in jump starts (when compared with the Caulfield, Flemington and Sandown racecourses). Further investigation of racecourse differences, particularly in track design, is therefore warranted to identify features of high risk racecourses and to explore the difference in risk between racecourses in the city and the country.

The risk factor studies reported in Chapters 5 and 6 are the first to identify risk factors for racehorse fatalities in Victorian flat and jump starts separately (Boden *et al.* 2007a and b). These studies permitted identification of different risk factors from those reported in previous Australian studies (Bailey *et al.* 1997a and 1998) by analysing potential risk factors for fatality in flat and jump starts separately, by including fatalities from country racetracks and by assessing variables describing prior racing history of horses. In the final multivariable model for fatalities in flat starts, horse gender, prior racing history, race length, racing year, racecourse location and track rating were associated with fatality (Chapter 5, (Boden *et al.* 2007a). Risk factors for jump starts included the number of flat and jump starts accumulated in the racing career prior to the study start, one or more starts in the 1-14 days prior to the case or control start, race type (steeplechase compared with hurdle races), track location (city compared with country tracks) and calendar year (Chapter 6, (Boden *et al.* 2007b). In jump starts, there was also a protective effect associated with increased prior racing career length and the number of flat, hurdle and steeple starts in the 1-60 day period prior to the case or control start (Boden *et al.* 2007b).

Only two studies of risk factors for catastrophic injury of racehorses in Australia have been reported previously (Bailey *et al.* 1997a and 1998). On two city racecourses in Sydney, increasing age of the horse, barrier position (barriers 13-18) and Stakes races (i.e. races of high quality, categorised into Group 1, 2 and 3 and Listed races in order of importance) were associated with increased odds of serious injury and that a change (a decrease or an increase) in the race distance from the previous race was associated with a decrease in the odds of serious injury (Bailey *et al.* 1997a). On city racecourses in Melbourne, Bailey *et al.* (1998) identified increasing age of the horse, fast or good tracks, race type and the Flemington racecourse as significant risk factors for serious injury resulting in failure to return to racing. In contrast, in the current studies, age of the horse, barrier position and quality of race (Group and Listed races) were investigated but were not significantly associated with fatality in flat starts.

It is not surprising that different risk factors were identified in the current studies. Regional differences in risk factors have been demonstrated in the USA (for example, Estberg *et al.* 1994, 1995a, 1995b, 1996b and 1998b; Cohen *et al.* 2000a, 2000b and 2003) and may reflect regional differences in racing and training practices (Cohen *et al.* 1999a). This emphasises the need for separate studies to identify the risk factors specific to racing populations in different geographical areas.

Previous studies of racehorse injury and fatality have led to a range of recommended intervention strategies. These recommendations have included increasing moisture in the track surface (Bailey *et al.* 1998; Williams *et al.* 2001; Parkin *et al.* 2005b; Newton *et al.* 2005), modifications of track surface, structure and design (Clanton *et al.* 1991; Oikawa *et al.* 1994), modifications of the number, position and design of fences in jump races (Singer *et al.* 2003; Parkin 2005a), utilisation of pre-race inspections (Dibbern 1996; Cohen *et al.* 1999b), early detection of subclinical injuries through veterinary examinations (Hill *et al.* 2003) or use

of technologies such as scintigraphy (Stover *et al.* 1992 and 1993), training of horses at an early age (Wood *et al.* 2000), modifications of exercise speed and intensity (Estberg *et al.* 1995a and 1996b; Parkin *et al.* 2003, 2004c and 2005b; Verheyen *et al.* 2005), increased schooling of horses (Pinchbeck *et al.* 2003) and modifications of the use of whips in racing (Pinchbeck *et al.* 2004a). One study investigating horse falls in steeplechase races (Pinchbeck *et al.* 2002) demonstrated that most variation in outcome was associated with the level of the run (start) rather than the level of the trainer, suggesting that interventions in steeplechase racing in the UK should focus on start-level factors rather than trainer-level factors. This was a very useful finding to report to the racing industry, as quantifying the proportion of variance in the risk of falling specifically prevented the wastage of resources on preventative strategies that would have had little impact on the overall risk of falls because they targeted factors that made little contribution to the variation.

In flat racing, increased odds of fatality were observed with fast or good tracks compared with slow, heavy or dead tracks in the early years of the study. However, the risk of fatality decreased with every extra year of the study period until 2003-2004 when no effect of track condition was evident (Boden *et al.* 2007a). In an investigation of racing injuries at two racetracks in Sydney, track condition was not identified as a risk factor (Bailey *et al.* 1997) and an earlier study in Victoria also found no association between track condition and catastrophic injury (Bourke 1994). The association between firmer tracks and increased risk of fatality has been demonstrated in studies overseas (Wilson *et al.* 1996; Parkin *et al.* 2004b, 2004c and 2005b). In the UK, the Jockey Club has ruled that racing must not occur on hard tracks and permits jump races in the summer only if the track is no firmer than good to firm (Parkin *et al.* 2004a). In Victoria, Racing Victoria Ltd. regulates that races should be held on tracks that are good with some give (J McCaffrey, Racing Victoria Ltd., *personal communication* 2005). At present, trainers in Victoria are permitted to scratch a horse after the official scratching time if there has been a late downgrade of the classification of the track from fast or good to heavy, slow or dead. However, the latter concession suggests that a firmer track is actually preferred by trainers, possibly because of greater predictability of racing performance. Scratching a horse from the race after the official scratching time is also permitted if there has been an upgrade of the classification of the track (i.e. from heavy to slow, slow to dead, dead to good or good to fast) but this is discouraged by the racing industry.

The decrease in the odds of fatality on fast or good tracks compared to heavy, slow or dead tracks over the period of the study is difficult to explain. There were increased odds of fatality per racing year on *country* tracks when the track rating was heavy, slow or dead and fast or good. There was no association with fatality per racing year on *city* tracks when the track rating was heavy slow or dead or fast or good. The greatest relative increase over the study period was on country tracks where the going was heavy, slow or dead. The racing industry recognises that the preparation of race track conditions is not always precise due to unforeseen fluctuations in weather conditions on race-days and characteristics of track composition (Racing Victoria Limited 2005). Poorer track maintenance on country racecourses relative to city racecourses may have occurred as a result of limited financial resources available to cope with increasingly severe drought conditions experienced in Victoria from 2002 onwards (Botterill and Fisher 2003). This may have resulted in poorer turf coverage on country racecourses in the later years of the study period. Tracks with poorer turf cover may have some of the properties of dirt tracks when wet. On dirt tracks (in distinct contrast to well-turfed tracks), an *increase* in water content is associated with greater risk of musculoskeletal injury (Rooney *et al.* 1983). Therefore attempts in later years to improve track safety by watering the track surface may have inadvertently resulted in an increase in risk on country tracks (Boden *et al.* 2007a).

A difficulty in making recommendations based on track condition is the inherent subjectiveness of track ratings. In the UK, current research is focused on using a ‘going stick’

that measures the degree of penetration and the degree of shear (to simulate resistance to the hoof). The penetration and degree of shear are combined to produce a measure of going index. On each course where it is in use, repeated measures have been taken at up to 100 locations since 2003 (mostly but not exclusively on race days). A current study is assessing the degree of variation in penetration, shear and index on each day of measurement at each course and over time at each location to determine whether there are particular sites that should be targeted for future ground maintenance (TDH Parkin, *personal communication* 2005.). In Victoria, the track rating is based on penetrometer readings and the opinions of racecourse managers and stewards. Methods for rating tracks are unlikely to be consistent amongst assessors (racecourse managers and stewards) throughout Victoria and the repeatability of ratings within assessors may also not be high. Racing industries in Victoria and throughout Australia are conducting comparable research to that in the UK to standardise measurements of track condition if intervention strategies aimed at modifying track condition are to be implemented.

The results of this study suggest that these current policies are not appropriately targeted to reduce the risk of fatality on racecourses in Victoria. Given the association of fatality with fast or good tracks, the classification of a 'good' track needs to be recalibrated so that the racing industry calls a 'good' track one on which the risk of fatality is low. Thus the current classification of a 'dead' track may in future be known as a 'good' track. Furthermore, the paradigms held by some trainers regarding the benefits of a fast or good track must be altered so that they are aware of the risks associated with firmer track surfaces and encouraged to scratch horses from races when the track is considered unsuitable for a particular horse. The study findings indicate that track design, construction and maintenance should continue to be industry priorities. This relates especially to maintaining the moisture content of track surfaces during very dry periods or on tracks with poorer turf coverage. Further investigation of changes in track assessment and maintenance on city tracks over the 15 year study period will help in the formulation of guidelines for maintenance of country tracks in order to reduce the risks of injury or death of horses racing on them (Boden *et al.* 2007a).

The results of the study of risk factors for fatality in jump starts (Chapter 6, Boden *et al.* 2007b) suggest that decreasing the risk of fatality associated with steeplechase events would be an effective strategy to reduce the overall risk of fatality in jump starts. If steeplechase events were stopped altogether in Victoria, the risk of fatality in jump races would be reduced by 30%, sparing four horses per year. However, this recommendation is unlikely to be supported by Racing Victoria Ltd. Steeplechase racing is an important part of Victoria's racing history. The first Grand National Steeplechase was run in Ballarat in 1863 and the Victoria Racing Club's first Grand National was held in 1866. The Australian Steeple at Sandown, the Grand National Steeple at Flemington and the Dominant Hiskens Steeple at Moonee Valley are regarded as the flagship jump races in Melbourne (Racing Victoria Ltd. 2005a). The Grand Annual Steeple in Warrnambool is an iconic jump event that reportedly contributes \$15 million to the local economy (Racing Victoria Ltd. 2005a).

Several risk factors describing the prior racing history were included in the final multivariable models. In jump and flat races, there was evidence of risk factors associated with the long-term effects (racing career duration; number of flat and jump starts accumulated in prior racing career; total distance in jump races accumulated in prior racing career), short-term effects (total starts in the 31-60 day period prior to the study start; flat, hurdle and steeple starts in the 60 days prior to the case or control start) and immediate effects of racing experience (at least one start in the 14 days prior to the case or control start).

In summary, jump starts by horses with shorter racing careers were more likely to result in fatality. Over the course of a racing career, horses that started in more flat starts prior to the case or control jump start were more likely to suffer a fatality in the next jump start than

horses that had accumulated less flat racing experience. Additionally, increased numbers of jump starts prior to the study start were also associated with increased odds of fatality.

The odds of fatality in jump starts increased with each additional flat start accumulated in the racing career. Bailey *et al.* (1998) suggested that the prior flat racing career of jump horses in Australia may have contributed significantly to the greater risk of fatality or career-ending injury observed in jump starts but this is the first confirmation that this association exists in Australia. The average number of career flat starts for horses starting in flat races was 17.2 whereas horses starting in jump races had already accumulated an average of 33 career flat starts. If horses were allowed to start in up to 32 flat starts (and no more) before beginning a jump racing career, the risk of fatality in jump starts may decrease by 50% (approximately eight fatalities per year). This finding strengthens the hypothesis that horses are sustaining long term chronic injuries in flat racing (as a result of repetitive strains to bones, ligaments and tendons) and that these injuries predispose them to catastrophic injury when they eventually start in jump races. Identification of this variable is particularly important because the prior number of career flat starts could be modified readily by intervention strategies. By imposing a limit on the number of flat starts that horses may accumulate before commencing a jump career, the Victorian racing industry could reduce the average risk of fatality to a level comparable to that in the UK. Such an intervention would mean that trainers would need to make early strategic decisions about racing careers of their horses.

There was no apparent long-term association of racing career duration or increased numbers of prior flat starts with the odds of fatality in flat starts. This may be because of the differences in racing experience in the two populations of flat and jump horses. Horses in jump events had already had a long racing career (mean of 33 flat starts prior to the study start) whereas horses in flat starts had on average only 17.2 flat starts prior to the study start (Chapter 6). Horses in flat starts also had decreased odds of fatality if they had accumulated distance in jump races prior to the study start. It is doubtful that this association reflects a cause and effect and it would be erroneous for the racing industry to encourage horses to start in more jump races prior to a flat start. Horses that might benefit from this protective effect made up less than 1% of horses starting in flat races in the case-control study (Chapter 5). It is more plausible that this variable was in the model because of the inclusion in the data of a special class of flat races that was designed specifically for jump horses. In comparison with regular flat starts, these flat starts may be slower and less competitive and used as experience rather than as competition for horses that are about to begin a jump racing season.

The odds of fatality in jump starts was increased for horses that did not start in flat, hurdle or steeplechase events in the 1-60 days prior to the study start compared with horses that did start in that time period. This finding takes into account the increased odds of fatality for horses starting in jump races if they started in a race within 14 days of the study start. Thus, it may be that jump starts by horses that started in flat, hurdle or steeplechase events in the 60 days prior to the study start were less likely to result in fatality as long as those starts were not accumulated in the 14 days prior to the case or control start. Horses that switched from hurdle races to steeplechase events or started in multiple steeplechase events in the 14 days prior to the case or control start were more likely to die in the index start than those that were either switching from flat to jump events or jumping in hurdle events only in the same time period. This suggests that racing intensity is an important consideration for future research.

The short-term prior racing history also appears important for horses starting in flat races. In contrast to the 1-14 day hazard window for jump starts, in flat starts, the number of starts and distance accumulated in the 31-60 day period prior to the index flat race start were associated with increased odds of fatality. This discrepancy may point to differences between race types in the causes of fatality. Identification of a hazard window of 31-60 days prior to the index start in flat starts indicates a need for further research to develop methods to identify subclinically injured horses and to determine what are the most appropriate recovery periods

after injury and the most appropriate training patterns to accumulate exercise. The 1-14 day hazard period identified for jump horses suggests the need for further investigation of the rate of accumulation of exercise to establish what constitutes an appropriate racing schedule. As the odds of fatality in a jump start were shown to increase with every extra start within the 1-14 day period, horses that accumulate large numbers of starts within this time frame could be identified and closely monitored for evidence of injury.

It is assumed that the variables relating to prior form history (both long and short term) in flat and jump starts influence the risk of fatality by predisposing horses to catastrophic limb injury. There remains a need for Australian studies to emulate those undertaken in the UK on fatal fractures (Parkin 2002; Parkin *et al.* 2000, 2004a, 2004b, 2004c and 2005b) to identify any additional risk factors associated with specific types of bone fracture that may not have been identified in the current study.

The findings relating to prior form history illustrate the importance of collecting more information on the training and racing schedules in both flat and jump racehorse populations. The number of starts and the distance accumulated in both official and unofficial trials need to be recorded accurately to determine monthly averages of racing intensity. Accurate measurement by means of global positioning systems (GPS) technology of speeds attained during racing and training would also improve the understanding of racing intensity thresholds that may predispose to catastrophic injury (Perkins 2005). Reliance on proxy measures of racing form history (such as the number of starts and the distance accumulated in different race types) in the absence of training data remains a significant weakness of this and other studies (Chapter 1). A New Zealand study (Perkins 2005) has demonstrated the benefit of capturing racing and training data to investigate risk factors associated with different types of injury.

Other risk factors identified in the flat and jump studies highlight areas for future research by the Victorian racing industry. Further investigation is required to identify reasons for the increased odds of fatality amongst entire males and geldings relative to mares. Bodyweight, behaviour and sex hormone concentrations may mediate this association. Identification of such horse-level risk factors may facilitate development of methods to detect those horses at high risk of fatality.

8. Specific recommendations for intervention strategies and further research

Recommendations arising from the post mortem study

- A recommendation for the continuation of the post mortem study has been made to Racing Victoria Ltd. in order to continue to monitor and document the causes of fatalities occurring on racecourses in Victoria.
- It is recommended that veterinarians collect blood samples from horses at the point of death on the racecourse to facilitate the investigation of sudden deaths. Assays of markers of peracute myocardial injury (such as cardiac troponin I) may help to confirm the presence of myocardial degeneration or necrosis (Cornelisse *et al.* 2000; Phillips *et al.* 2003; Schwartzwald *et al.* 2003) and measurement of electrolytes (especially potassium, calcium and magnesium) may identify potential triggers of such injury. Coagulation studies on point-of-death blood samples may also be beneficial in investigating cases of sudden death from massive haemorrhage.
- Racing Victoria Ltd. should consider making post mortem examinations mandatory for all horses that die or are euthanised on country racecourses in Victoria, in accordance with the current policy for fatalities sustained on city racecourses in Victoria. Alternatively, a random sample of all country fatalities should be subjected to post mortem examination each year. Random sampling would be preferable to examining only those fatality cases on country racecourses that owners, trainers and/or racetrack veterinarians choose to submit. However, transporting horses over long distances to The University of Melbourne for post mortem examinations would require a major financial commitment by the racing industry. Prolonged transit times may also limit the value of post mortem examinations, especially in sudden death cases, by permitting advanced autolysis. Racing Victoria Ltd. could consider sending cadavers to regional pathology laboratories as the California post mortem program has done (Johnson *et al.* 1994a and 1994b). To enhance the investigation of risk factors for catastrophic appendicular musculoskeletal injury, Racing Victoria Ltd. could make mandatory submission to The University of Melbourne of affected limbs from horses killed or euthanised on country racecourses, especially as such injuries are less likely to be obscured by autolysis than visceral lesions.
- It is recommended that the design of the racehorse fatality report form be improved. The current form has an open-ended question format (Dohoo *et al.* 2003). As a result, there is considerable variation in the amount of detail provided by racecourse veterinarians on the cause of death or euthanasia. A closed question format, such as a checklist, would be faster and easier to complete and could minimise the ambiguities that currently arise when vague terms (such as leg or fetlock rather than lateral condylar fracture) are used to describe the location of catastrophic limb injuries. This approach may also stimulate more systematic examination of fatality cases by racecourse veterinarians.

Recommendations arising from the case-control studies of risk factors for fatality in flat and jump starts

- It is recommended that there be systematic and standardised application of interventions throughout the Victorian racing industry. The effects of these interventions should be assessed using a well-designed monitoring system.
- It is recommended that the method used to measure track condition be assessed for repeatability and standardised across all Australian racecourses. If penetrometer readings are part of this measuring system, standardised reference ranges for specific classification of different track conditions should be described and regulated. Further investigation into methods of maintaining an ideal track condition for both country and city racecourses remain important.
- The Victorian racing industry should continue to research reasons for differences in the risk of fatality between steeplechase and hurdle races. Any consequent interventions such as modifications to fence design and positioning should be implemented across all racecourses simultaneously so that the effect of intervention measures can be rigorously and scientifically assessed. Fatalities, injuries and falls should be recorded systematically so that the effect of the intervention can be measured accurately. The application of process control charts (such as Cusum charts) may be a useful method for the racing industry to monitor fatalities.
- It is recommended that Racing Victoria Ltd. impose limits on the number of flat starts that horses are allowed to accumulate before competing in jump races.
- It is recommended that the Victorian racing industry prioritise research into early detection of horses at high risk of fatality in flat and jump starts. Further investigation of appropriate rates of accumulation of high speed exercise in racing and training are necessary to identify periods when horses are at the most risk and to establish appropriate racing schedules. Future research should also incorporate prior medical histories of horses to ascertain the importance of prior subclinical and clinical injuries as risk factors for catastrophic injury and death.
- In light of differences in the odds of fatality on different racecourses, maintenance of the Victorian racetrack database developed by Stubbs (2004) is important for future studies investigating the effects of differences in track design and surface and the effects of modifications to race tracks on the risk of fatality in flat and jump starts.
- It is recommended that Racing Victoria Ltd. initiate studies comparable to those undertaken in the UK on fatal fractures (Parkin *et al.* 2004a and b) and falls (Pinchbeck *et al.* 2002 and 2003) to identify risk factors associated with specific types of injury.
- There is a need for formal racehorse fatality monitoring programs to be developed in the other States in Australia. Ultimately, development of national and international fatality databases may be possible to enable direct comparisons of the risk of racehorse fatality in different countries.

9. Bibliography

- Abramson, J.H. (2004) WINPEPI (PEPI-for-Windows): computer programs for epidemiologists. *Epidemiologic Perspectives & Innovations* **1**, 1-15. (Accessible at <http://www.epiperspectives.com/content/1/1/6>) (Accessed December 2005).
- Akaike, H. (1973) Information theory and an extension of the maximum likelihood principle. In: *Second International Symposium On Information Theory*. Eds. Petrov, B.N. and Csaki, F. Akademiai Kiado, Budapest, Hungary. pp 267–281. Reprinted (1992) in: *Breakthroughs In Statistics*, Volume I. Eds. Kotz, S. and Johnson, N.L. Springer, New York, USA. pp 599–624.
- Anonymous (2001) Constructing and using process control charts. In: *Harvard Business School Class Discussion Notes*. Harvard University, Massachusetts, USA. pp 2-17.
- Australian Jumping Racing Association (2003) *Victorian Jumping Race Schedule 2003*. <http://home.primus.com.au/jumps/JumpingInformerFeb.htm> (Accessed December 2005).
- Australian Racing Board Ltd. (2001) *Size and Scope Of The Australian Thoroughbred Racing Industry*. <http://www.australian-racing.net.au/> pp 1-18. (Accessed December 2005).
- Australian Racing Board Ltd. (2003) *Australian Racing Fact Book. A Guide to the Racing Industry in Australia*. RVL Publishing, Kensington, NSW. p 14.
- Bachofen, H. and Weibel, E.R. (1996) Pulmonary edema. In: *Spencer's Pathology of the Lung*, 5th edition. Ed. Hasleton, P.S. McGraw-Hill, New York. pp 707-722.
- Bagley, S.C., White, H. and Golomb, B.A. (2001) Logistic regression in the medical literature: Standards for use and reporting, with particular attention to one medical domain. *Journal of Clinical Epidemiology* **54**, 979-985.
- Bailey, C. (1998a) *Identification And Characterization Of Causes Of Wastage In The Australian Thoroughbred Racing Industry*. PhD thesis, University of Sydney, Australia
- Bailey, C.J. (1998b) *Wastage In The Australian Thoroughbred Racing Industry*. Rural Industries Research and Development Corporation, Kingston, ACT. pp 33-36.
- Bailey, C.J., Reid, S.W.J., Hodgson, D.R., Suann, C.J. and Rose, R.J. (1997a) Risk factors associated with musculoskeletal injuries in Australian Thoroughbred racehorses. *Preventive Veterinary Medicine* **32**, 47-55.
- Bailey, C.J., Rose, R.J., Reid, S.W.J. and Hodgson, D.R. (1997b) Wastage in the Australian Thoroughbred racing industry: a survey of Sydney trainers. *Australian Veterinary Journal* **75**, 64-67.
- Bailey, C.J., Reid, S.W.J., Hodgson, D.R., Bourke, J.M. and Rose, R.J. (1998) Flat, hurdle and steeple racing: risk factors for musculoskeletal injury. *Equine Veterinary Journal* **30**, 498-503.
- Bailey, C.J., Reid, S.W.J., Hodgson, D.R. and Rose, R.J. (1999a) Factors associated with time until first race and career duration for Thoroughbred racehorses. *American Journal of Veterinary Research* **60**, 1196-1200.

- Bailey, C.J., Reid, S.W.J., Hodgson, D.R. and Rose, R.J. (1999b) Impact of injuries and disease on a cohort of two-and three-year old Thoroughbreds in training. *The Veterinary Record* **145**, 487-493.
- Balch, O.K., Helman, R.G. and Collier, M.A. (2001) Underrun heels and toe-grab length as possible risk factors for catastrophic musculoskeletal injuries in Oklahoma racehorses. In: *Proceedings of the American Association of Equine Practitioners 47*. San Diego, California. pp 334-338.
- Bathe, A.P. (1994) 245 Fractures in Thoroughbred racehorses: results of a 2 year prospective study in Newmarket. *Proceedings of the American Association of Equine Practitioners 40*. Vancouver, British Columbia. pp 175-176.
- Benichou, J., Chow, W.-H., McLaughlin, J.K., Mandel, J.S. and Fraumeni Jr., J.F. (1998). Population attributable risk of renal cell cancer in Minnesota. *American Journal of Epidemiology* **148**, 424-429.
- Benton, L.D. (1994) *Victoria Racing Club Victorian Jump Racing Report*. Melbourne, Australia. pp 1-20.
- Berry, G. (1986) Statistical significance and confidence intervals. *Medical Journal of Australia* **144**, 618-619.
- Boden, L.A., Charles, J.A., Slocombe, R.F., Sandy, J.R., Finnin, P.J., Morton, J.M. and Clarke, A.F. (2005) Sudden death in racing Thoroughbreds in Victoria, Australia. *Equine Veterinary Journal* **37**, 269-271.
- Boden, L.A., Anderson, G.A., Charles, J.A., Morgan, K.L., Morton, J.M., Parkin T.D.H., Slocombe, R.F. and Clarke, A.F. (2006) Risk of fatality and causes of death of Thoroughbred horses associated with racing in Victoria, Australia between 1989 and 2004. *Equine Veterinary Journal*, **38**, 312-318.
- Boden, L.A., Anderson, G.A., Charles, J.A., Morgan, K.L., Morton, J.M., Parkin T.D.H., Slocombe, R.F. and Clarke, A.F. (2007a) Risk Factors for Thoroughbred Racehorse Fatality in Jump Starts in Victoria (1989-2004) *Equine Veterinary Journal*, *accepted for publication*.
- Boden, L.A., Anderson, G.A., Charles, J.A., Morgan, K.L., Morton, J.M., Parkin T.D.H., Slocombe, R.F. and Clarke, A.F. (2007b) Risk Factors for Thoroughbred Racehorse Fatality in Jump Starts in Victoria (1989-2004) *Equine Veterinary Journal*, *accepted for publication*.
- Bourke, J.M. (1994) Fatalities on racecourses in Victoria. A seven year study. In: *Proceedings of the 10th International Conference of Racing Analysts and Veterinarians*. Stockholm, Sweden. pp 265-268.
- Bourke, J. (1995) Wastage in Thoroughbreds. In: *Proceedings of the Annual Seminar of the Equine Branch of the New Zealand Veterinary Association*. New Zealand. pp 107-119.
- Box, G.E.P., Jenkins, G.M. and Reinsel, G.C. (1994) *Time Series Analysis: Forecasting and Control*, 3rd ed. Prentice Hall, Englewood Cliffs, New Jersey, USA. pp 200-201.
- Boyde, A. (2003) The real response of bone to exercise. *Journal of Anatomical Society of Great Britain and Ireland* **203**, 173-189.
- Bruzzi, P., Greed, S.B., Byar, D.P., Brinton, L.A. and Schairer, C. (1985) Estimating the population attributable risk for multiple risk factors using case-control data. *American Journal of Epidemiology* **122**, 904-914.

- Burr, D.B., Forwood, M.R., Fyhrie, D.P., Martin, R.B., Schaffler, M.B. and Turner, C.H. (1997) Bone microdamage and skeletal fragility in osteoporotic and stress fractures. *Journal of Bone and Mineral Research* **12**, 6-15.
- Caldwell, G.G., Kelley, D.B. and Health, C.W. Jr. (1980) Leukemia among participants in military maneuvers at a nuclear bomb test: a preliminary report. *Journal of the American Medical Association* **244**, 1575-1578.
- Carrier, T.K., Estberg, L., Stover, S.M., Gardner, I.A., Johnson, B.J., Read, D.H. and Ardans, AA. (1998) Association between long periods without high-speed workouts and risk of complete humeral or pelvic fracture in Thoroughbred racehorses: 54 cases (1991-1994). *Journal of the American Veterinary Medical Association* **212**, 1582-1587.
- Christley, R.M. and Reid, S.W.J. (2003) No significant difference: use of statistical methods for testing equivalence in clinical veterinary literature. *Journal of the American Veterinary Medical Association* **222**, 433-437.
- Clanton, C., Kobluk, C., Robinson, R.A. and Gordon, B. (1991) Monitoring surface conditions of a Thoroughbred racetrack. *Journal of the American Veterinary Medical Association* **198**, 613-619.
- Cogger, N., Evans, D., Hodgson, D., Perkins, N. and Reid, S.W.J. (2003) Risk factors for musculoskeletal injuries in two-year old Thoroughbred racehorses. *Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine*. Warwick, United Kingdom. pp 109-118.
- Cohen, N.D., Peloso, J.G., Mundy, G.D., Fisher, M., Holland, R.E., Little, T.V., Misheff, M.M., Watkins, J.P., Honnas, C.M. and Moyer, W. (1997) Racing-related factors and results of prerace physical inspection and their association with musculoskeletal injuries incurred in Thoroughbreds during races. *Journal of the American Veterinary Medical Association* **211**, 454-463.
- Cohen, N.D., Mundy, G.D., Peloso, J.G., Carey, V.J. and Amend, N.K. (1999a) Results of physical inspection before races and race-related characteristics and their association with musculoskeletal injuries in Thoroughbreds during races. *Journal of the American Veterinary Medical Association* **215**, 654-661.
- Cohen, N.D., Dresser, B.T., Peloso, J.G., Mundy, G.D. and Woods, A.M. (1999b) Frequency of musculoskeletal injuries and risk factors associated with injuries incurred in Quarter Horses during races. *Journal of the American Veterinary Medical Association* **215**, 662-669.
- Cohen, N.D., Berry, S.M., Peloso, J.G., Mundy, G.D. and Howard, I.C. (2000a) Thoroughbred racehorses that sustain injury accumulate less high speed exercise compared to horses without injury in Kentucky. In: *Proceedings of the American Association of Equine Practitioners* 46. San Antonio, Texas. pp 51-53.
- Cohen, N.D., Berry, S.M., Peloso, J.G., Mundy, G.D. and Howard, I.C. (2000b) Association of high-speed exercise with racing injury in Thoroughbreds. *Journal of the American Veterinary Medical Association* **216**, 1273-1278.
- Cook, D.A., Steiner, S.H., Cook, R., Farewell, V.T. and Morton, A.P. (2003) Monitoring the evolutionary process of quality: Risk-adjusted charting to track outcomes in intensive care. *Critical Care Medicine* **31**, 1676-1682.

- Cook, W.R., Williams, R.M., Kirker-Head, C.A. and Verbridge, D.J. (1988) Upper airway obstruction (partial asphyxia) as the possible cause of exercise-induced pulmonary hemorrhage in the horse: an hypothesis. *Equine Veterinary Science* **8**, 11-26.
- Cornelisse, C.J., Schlott, H.C., Olivier, N.B., Mullaney, T.P., Koller, A., Wilson, D.V. and Derksen, F. J. (2000) Concentration of cardiac troponin I in a horse with a ruptured aortic regurgitation jet lesion and ventricular tachycardia. *Journal of the American Veterinary Medical Association* **217**, 231-235.
- Couëtil, L.L. and Hinchcliff, K.W. (2004) Non-infectious diseases of the lower respiratory tract. In: *Equine Sports Medicine and Surgery*. Eds. Hinchcliff, K.W., Kaneps, A.J. and Geor, R.J. Saunders, Elsevier Limited, London, United Kingdom. pp 632-656.
- Davies, H.M., McCarthy, R.N. and Jeffcott, L.B. (1993) Surface strain on the dorsal metacarpus of Thoroughbreds at different speeds and gaits. *Acta anatomica (Basel)* **146**, 148-153.
- Dibbern, D.K. (1996) Use of the post parade examination as the primary method of minimizing catastrophic injury. In: *Proceedings of the 11th International Conference of Racing Analysts and Veterinarians*. Queensland, Australia. pp 275-277.
- Dixon, K.E. (1997) A comparison of case-crossover and case-control designs in a study of risk factors for hemorrhagic fever with renal syndrome. *Epidemiology* **8**, 243-246.
- Dohoo, I., Martin, W. and Stryhn, H. (2003) *Veterinary Epidemiologic Research*. AVC Inc., Charlottetown, Prince Edward Island. pp 18, 30, 72, 126-127, 140-141, 200, 220, 228, 236, 242, 274-275, 340, 349, 368, 546.
- Ducharme, N.G., Hackett, R.P., Gleed, R.D., Ainsworth, D.M., Erb, H.N., Mitchell, L.M. and Soderholm, L.V. (1999) Pulmonary capillary pressure in horses undergoing alteration of pleural pressure by imposition of various upper airway resistive loads. *Equine Veterinary Journal* **30**, Suppl. 27-33.
- Dungworth, D.L. (1993) The respiratory system. In: *Pathology of Domestic Animals*, Volume 2, 4th edition. Eds. Jubb, K.V.F., Kennedy, P.C. and Palmer, N.C. Academic Press, Inc, San Diego. pp 539-699.
- Edmonds, L.D. and James, L.M. (1990) Temporal trends in the prevalence of congenital malformations at birth based on the birth defects monitoring program, United States 1979-1987. *Morbidity and Mortality Weekly Report. CDC Surveillance Summaries* **39**, 19-23.
- Efron, B. and Tibishirani, R. (1993) *An Introduction To The Bootstrap*. Chapman and Hall, New York.
- Ely, E.R., Verheyen, K.H. and Wood, J.L. (2004) Fractures and tendon injuries in National Hunt horses in training in the UK: a pilot study. *Equine Veterinary Journal* **36**, 365-7.
- Erickson, B.K., Erickson, H.H. and Coffman, J.R. (1990) Pulmonary artery, aortic and oesophageal pressure changes during high intensity treadmill exercise in the horse: a possible relation to exercise-induced pulmonary haemorrhage. *Equine Veterinary Journal* **9**, Suppl. 47-52.
- Estberg, L., Stover, S., Case, J.T., Johnson B.J., Gardner, I.A., Ardans, A., Read, D.H., Anderson, M.L., Barr, B.C., Daft, B.M., Kinde, H., Moore, J., Stoltz, J. and Woods, L.W. (1993) Case-control study of racing related risk factors for catastrophic injuries of the

thoroughbred racehorse. In: *Proceedings of the American Association of Equine Practitioners* 39. San Antonio, Texas. pp 129-130.

Estberg, L., Stover, S., Case, J.T., Johnson B.J., Gardner, I.A., Ardans, A., Read, D.H., Anderson, M.L., Barr, B.C., Daft, B.M., Kinde, H., Moore, J., Stoltz, J. and Woods, L.W. (1994) Case-control study of a cluster estimate of cumulative exercise distance as a risk factor for fatal musculoskeletal injury in thoroughbred racehorses. *Proceedings of the American Association of Equine Practitioners* **40**,171-172.

Estberg, L., Stover, S., Gardner, I.A., Drake, C.M., Johnson B.J., Case, J.T., Ardans, A., Read, D.H., Anderson, M.L., Barr, B.C., Daft, B.M., Kinde, H., Moore, J., Stoltz, J. and Woods, L.W. (1995a) Several racing-career intensity characteristics are associated with racing-related fatal skeletal injury in California Thoroughbred racehorses. In: *Proceedings of the American Association of Equine Practitioners* 41. Lexington, Kentucky. pp 82-83.

Estberg, L., Gardner, I.A., Stover, S.M., Johnson, B.J., Case, J.T. and Ardans, A. (1995b) Cumulative racing-speed exercise distance cluster as a risk factor for fatal musculoskeletal injury in Thoroughbred racehorses in California. *Preventive Veterinary Medicine* **24**, 253-263.

Estberg, L., Stover, S.M., Gardner, I.A., Johnson, B.J., Case, J.T., Ardans, A., Read, D.H., Anderson, M.L., Barr, B.C., Daft, B.M., Kinde, H., Moore, J., Stoltz, J. and Woods, L.W. (1996a) Fatal musculoskeletal injuries incurred during racing and training in Thoroughbreds. *Journal of the American Veterinary Medical Association* **208**, 92-96.

Estberg, L., Stover, S.M., Gardner, I.A., Drake, C.M., Johnson, B.J. and Ardans, A. (1996b) High-speed exercise history and catastrophic racing fracture in Thoroughbreds. *American Journal of Veterinary Research* **57**, 1549-1555.

Estberg, L., Stover, S.M., Gardner, I.A., Johnson, B.J., Jack, R.A., Case, J.T., Ardans, A., Read, D.H., Anderson, M.L., Barr, B.C., Daft, B.M., Kinde, H., Moore, J., Stoltz, J. and Woods, L.W. (1998a) Relationship between race start characteristics and risk of catastrophic injury in Thoroughbreds: 78 cases (1992). *Journal of the American Veterinary Medical Association* **212**, 544-549.

Estberg, L., Gardner, I.A., Stover, S.M. and Johnson, B.J. (1998b) A case-crossover study of intensive racing and training schedules and risk of catastrophic musculoskeletal injury and lay-up in California Thoroughbred racehorses. *Preventive Veterinary Medicine* **33**, 159-170.

Falahati-Nini, A., Riggs, B.L., Atkinson, E.J., O'Fallon, W.M., Eastell, R. and Khosla, S. (2000) Relative contributions of testosterone and oestrogen in regulating bone resorption and formation in normal elderly men. *Journal of Clinical Investigation* **106**, 1553-1560.

Fredericson, I., Dalin, G., Drevmo, S. and Hjerten, G. (1975) A biotechnical approach to the geometric design of racetracks. *Equine Veterinary Journal* **7**, 91-95.

Gelberg, H.B., Zachary, J.F., Everitt, J.I., Jensen, R.C. and Smetzer, D.L. (1985) Sudden death in training and racing Thoroughbred horses. *Journal of the American Veterinary Medical Association* **187**, 1354-1356.

Gordon, J. (2001) The horse industry. In: *Contributing to the Australian Economy - A report for the Rural Industries Research and Development Corporation*. Rural Industries Research and Development Corporation, Kingston, Australian Capital Territory. pp 1-7.

Greenland, S. (1998a) Applications of stratified analysis methods. In: *Modern Epidemiology*, 2nd edition. Eds. Rothman, K.J. and Greenland, S. Lippincott-Raven, Philadelphia. pp 281-300.

Greenland, S. (1998b) Introduction to regression models. In: *Modern Epidemiology*, 2nd edition. Eds. Rothman, K.J. and Greenland, S. Lippincott-Raven, Philadelphia. pp 359-399.

Greenland S. and K. Drescher (1993) Maximum likelihood estimation of the attributable fraction from logistic models. *Biometrics* **49**, 865-872.

Greenland, S. and Rothman, K.J. (1998) Measures of effect and measures of association. In: *Modern Epidemiology*, 2nd edition. Eds. Rothman, K.J. and Greenland, S. Lippincott-Raven, Philadelphia. pp 47-64.

Gunson, D.E., Sweeney, C.R. and Soma, L.R. (1988) Sudden death attributable to exercise-induced pulmonary haemorrhage in racehorses: Nine cases (1981-1983). *Journal of the American Veterinary Medical Association* **193**, 102-105.

Hackett, R.P., Ducharme, N.G., Ainsworth, D.M., Erickson, B.K., Erb, H.N., Soderholm, L.V. and Thorson, L.M. (1999) Effects of extrathoracic airway obstruction on intrathoracic pressure and pulmonary artery pressure in exercising horses. *American Journal of Veterinary Research* **60**, 485-494.

Hahn, J.A. and Schulman, J. (1992) Cost efficient sampling design for quality control of a birth defects registry. *Journal of Clinical Epidemiology* **45**, 1371-1375.

Harkins, J.D. and Kamerling, S.G. (1993) Effect of race distance and competition on performance of Thoroughbred racehorses. *Journal of Equine Research* **13**, 41-45.

Haynes, P.F. and Robinson, R.A. (1988) Racetrack breakdown pilot study summary. In: *Proceedings of the American Association of Equine Practitioners 34*. San Diego, California. pp 673-676.

Henley, W.E., Rogers, K., Harkins, L. and Wood, J.L.M. (2003) Survival analysis of fatal injuries during racing: a comparison of different approaches. In: *Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine*. Warwick, United Kingdom. pp 147-159.

Hernandez, J., Hawkins, D.L. and Scollay, M.C. (2001) Race-start characteristics and risk of catastrophic musculoskeletal injury in Thoroughbred racehorses. *Journal of the American Veterinary Medical Association* **218**, 83-86.

Hernandez, J., Scollay, M.C., Hawkins, D.L., Corda, J.A. and Krueger, T.M. (2005) Evaluation of horseshoe characteristics and high-speed exercise history as possible risk factors for catastrophic musculoskeletal injury in Thoroughbred racehorses. *American Journal of Veterinary Research* **66**, 1314-1320.

Hill, T., Carmichael, D., Maylin, G. and Krook, L. (1986) Track condition and racing injuries in Thoroughbred horses. *Cornell Veterinary Journal* **76**, 361-379.

Hill, A.E., Stover, S.M., Gardner, I.A., Kane, A.J., Whitcomb, M.B. and Emerson, A.G. (2001) Risk factors for and outcomes of noncatastrophic suspensory apparatus injury in Thoroughbred horses. *Journal of the American Veterinary Medical Association* **218**, 1137-1143.

Hill, A.E., Carpenter, T.E., Gardner, I.A. and Stover, S.M. (2003) Evaluation of a stochastic Markov-chain model for the development of forelimb injuries in Thoroughbred racehorses. *American Journal of Veterinary Research* **64**, 328-336.

Hintze J. (2004) *NCSS and PASS. Number Cruncher Statistical Systems*.
<http://www.ncsss.com> (Accessed December 2005).

Hosmer, D.W. and Lemeshow, S. (2000) *Applied Logistic Regression*, 2nd Edition. John Wiley and Sons Inc., New York. pp. 116-117, 167-183, 346.

Johnson, B.J., Stover, S.M., Daft, B.M., Kinde, H., Read, D.H., Barr, B.C., Anderson, M., Moore, J., Woods, S.L. and Stoltz J. (1994a) Causes of death in racehorses over a 2 year period. *Equine Veterinary Journal* **26**, 327-330.

Johnson, B.J., Ardans, A., Stover, S.M., Daft, B.M., Kinde, H., Read, D.H., Barr, B.C., Moore, J., Woods, L., Anderson, M., Stolz, J. and Blanchard, P. (1994b) California racehorse post mortem program: A 4-year overview. In: *Proceedings of the 40th Annual Convention of the American Association of Equine Practitioners*. Vancouver, British Columbia. pp 167-169.

Jones, B.H., Thacker, S. B., Gilchrist, J., Kimsey, C.D. and Sosin, D.M. (2002) Prevention of lower extremity stress fractures in athletes and soldiers: a systematic review. *Epidemiological Review* **24**, 228-247.

Kane, A.J., Stover, S.M., Gardner, I.A., Case, J.T., Johnson, B.J., Read, D.H. and Ardans, A.A. (1996) Horseshoe characteristics as possible risk factors for fatal musculoskeletal injury of Thoroughbred racehorses. *American Journal of Veterinary Research* **57**, 1147-52.

Kaneene, J.B., Ross, W.A. and Miller, R. (1997) The Michigan equine monitoring system. II. Frequencies and impact of selected health problems. *Preventive Veterinary Medicine* **29**, 277-292.

Khoury, M.J. and Holtzman, N.A. (1987) On the ability of the birth defects monitoring to detect new teratogens. *American Journal of Epidemiology* **126**, 136-143.

Kiryu, K., Machida, N., Kashida, Y., Yoshihara, T., Amada, A. and Yamamoto, T. (1999) Pathologic and electrocardiographic findings in sudden cardiac death in racehorses. *Journal of Veterinary Medical Science* **61**, 921-928.

Kobluk, C.N., Robinson, R.A., Gordon, B.J., Clanton, C.J., Trent, A.M. and Ames, T.R. (1989) The effect of conformation and shoeing: A cohort study of 95 Thoroughbred racehorses. In: *Proceedings of the American Association of Equine Practitioners 35*. Boston, Massachusetts. pp 259-274.

Kobluk, C.N., Robinson, R.A., Clanton, C.J., Trent, A.M., Ames, T.R. and Gordon, B.J. (1990) Comparison of the exercise level and problem rate of 95 Thoroughbred horses: a cohort study. In: *Proceedings of the American Association of Equine Practitioners 36*. Lexington, Kentucky. pp 471-475.

Kobluk, C.N., Robinson, R.A. and Clanton, C. (1992) Technical summary 1987 racetrack breakdowns: Pilot studies. In: *Proceedings of the 9 International Conference of Racing Analysts and Veterinarians*. New Orleans, Louisiana. pp 7-17.

Kouha J. (2004) IC and BIC comparisons of assumption and performance. *Sociological Methods & Research* **33**, 188-229.

- Krook, L. and Maylin, G.A. (1988) Fractures in Thoroughbred race horses. *Cornell Veterinary Journal* **78**, Suppl. 1-133.
- Langsetmo, I., Meyer, M.R. and Erickson, H.H. (2000) Relationship of pulmonary arterial pressure to pulmonary haemorrhage in exercising horses. *Equine Veterinary Journal* **32**, 379-384.
- Last, J.M. (2001) *A Dictionary Of Epidemiology*. Oxford University Press, New York. pp 113, 129.
- Lee, T.C., Staines, A. and Taylor, D. (2002) Bone adaptation to load: microdamage as a stimulus for bone remodeling. *Journal of Anatomical Society of Great Britain and Ireland* **201**, 437-446.
- Loeffler, S.H., de Vries, M.J. and Schukken, Y.K. (1999) The effects of time of disease occurrence, milk yield and body condition on fertility of dairy cows. *Journal of Dairy Science* **71**, 2735-2743.
- Long, J.S. and Freese, J. (2006) *Regression Models for Categorical Dependent Variables Using Stata*, 2nd Edition. Stata Press, College Station, Texas. pp 1-9, 12.
- Macdonald, D.M. and Toms, T.S. (1994) Survey into the incidence of racetrack injuries in the Transvaal racing district of South Africa. In: *Proceedings of the 10th International Conference of Racing Analysts and Veterinarians*. Stockholm, Sweden. pp 262-264.
- Manohar, M., Hutchens, E. and Coney, E. (1993) Pulmonary haemodynamics in the exercising horse and their relationship to exercise-induced pulmonary haemorrhage. *British Veterinary Journal* **149**, 419-428.
- Martin, S.W., Meek, A.H. and Willeberg, P. (1987) *Veterinary Epidemiology Principles and Methods*. Iowa State University Press, Ames, Iowa. pp 53-58, 130, 181-182.
- McKee, S.L. (1995) An update on racing fatalities in the UK. *Equine Veterinary Education* **7**, 202-204.
- McNutt, L.A., Wu, C., Xue, X. and Hafner, J.P. (2003) Estimating the relative risk in cohort studies and clinical trials of common outcomes. *American Journal of Epidemiology* **157**, 940-943.
- Meagher, D.M. (1976) Lateral condylar fractures of the metacarpus and metatarsus in horses. In: *Proceedings of the American Association of Equine Practitioners* 22. Dallas Texas. pp 147-154.
- Melbourne Racing Club website. <http://www.melbournracingclub.net.au> (Accessed December 2005).
- Miettinen OS (1974) Proportion of disease caused or prevented by a given exposure, trait or intervention. *American Journal of Epidemiology* **99**, 325-332.
- Mizuno, Y. (1996) Fractures of the carpus in racing Thoroughbreds of the Japan Racing Association: prevalence, location, and current modes of surgical therapy. *Journal of Equine Veterinary Science* **16**, 25-31.

- Mohammed, H.O., Hill, T. and Lowe, J. (1991) Risk factors associated with injuries in Thoroughbred horses. *Equine Veterinary Journal* **23**, 445-448.
- Mohammed, H.O., Hill, T. and Lowe, J. (1992) The risk of severity of limb injuries in racing Thoroughbred horses. *Cornell Veterinary Journal* **82**, 331-341.
- Moore, J., Stoltz, J. and Woods, L.W. (1996) Fatal musculoskeletal injuries incurred during racing and training in Thoroughbreds. *Journal of the American Veterinary Medical Association* **206**, 93-96.
- More, S. (1999) A longitudinal study of racing Thoroughbreds: performance during the first years of racing. *Australian Veterinary Journal* **77**, 105-110.
- Morton, J.M. (2004) *Determinants Of Reproductive Performance Of Dairy Cows In Commercial Herds In Australia*. PhD thesis, The University of Melbourne, Australia.
- Mundy, G.D. (1997) Review of risk factors associated with racing injuries. In: *Proceedings of the American Association of Equine Practitioners 43*. Phoenix, Arizona. pp 204-209.
- Murray, J.K., Singer, E.R., Saxby, F. and French, N.P. (2004) Factors influencing risk of injury to horses falling during eventing. *The Veterinary Record* **154**, 207-208.
- Newton, J.R., Rogers, K., Marlin, D.J., Wood, J.L.N. and Williams, R.B. (2005) Risk factors for epistaxis on British racecourses: evidence for locomotory impact-induced trauma contributing to the aetiology of exercise-induced pulmonary haemorrhage. *Equine Veterinary Journal* **37**, 402-411.
- O'Brien, P.J., Landt, Y. and Landenson, J.H. (1997) Differential reactivity of cardiac and skeletal muscle from various species in a cardiac troponin I immunoassay. *Clinical Chemistry* **43**, 2333-2338.
- Oikawa, M., Ueda, Y., Inada, S., Tsuchikawa, T., Kusano, H. and Takeda, A. (1994) Effect of restructuring a racetrack on the occurrence of racing injuries in Thoroughbred horses. *Journal of Equine Veterinary Science* **14**, 262-268.
- Oki, H., Sasaki, Y., Lin, C.Y. and Willham, R.L. (1994) Influence of jockeys on racing time in Thoroughbred horses. *Journal of Animal Breeding Genetics* **112**, 171-175.
- Palmer, S. E. (1986) Prevalence of carpal fractures in Thoroughbred and Standardbred racehorses. *Journal of the American Veterinary Medical Association* **188**, 1171-1173.
- Parkin, T.D.H. (2002) *Identification Of Risk Factors For Fatal Distal Limb Fracture In Racing Thoroughbreds*. PhD thesis, The University of Liverpool, United Kingdom.
- Parkin, T. (2005) Video surveillance study. In: *Jumping Racing Review Panel Report*. Racing Victoria Ltd. Melbourne, Australia. pp 78-119.
- Parkin, T.D.H. and Boden, L.A. (2005) Univariate analysis of falling and fatality data in jump racing in Victoria (2001-2005). In: *Jumping Racing Review Panel Report*. Racing Victoria Ltd. Melbourne, Australia. pp 40-41.
- Parkin, T.D.H., Clegg, P.D., French, N.P., Proudman, C.J., Riggs, C.M., Singer, E.R., Webbon, P.M. and Morgan, K.L. (2000) Descriptive epidemiology of fatal fractures of the distal limb in racing Thoroughbreds. In: *Proceedings of the 13th International Conference of Racing Analysts and Veterinarians*. Cambridge, United Kingdom. pp 278-283.

Parkin, T.D.H., Clegg, P.D., French, N.P., Proudman, C.J., Riggs, C.M., Singer, E.R., Webbon, P.M. and Morgan, K.L. (2003) Analysis of horse race videos to identify risk factors for fatal distal limb fracture. In: *Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine*. Warwick, United Kingdom. pp 132-143.

Parkin, T.D.H., Clegg, P.D., French, N.P., Proudman, C.J., Riggs, C.M., Singer, E.R., Webbon, P.M. and Morgan, K.L. (2004a) Risk of fatal distal limb fractures among Thoroughbreds involved in the five types of racing in the United Kingdom. *The Veterinary Record* **154**, 493-497.

Parkin, T.D.H., Clegg, P.D., French, N.P., Proudman, C.J., Riggs, C.M., Singer, E.R., Webbon, P.M. and Morgan, K.L. (2004b). Horse level risk factors for fatal distal limb fracture in racing Thoroughbreds in the UK. *Equine Veterinary Journal* **36**, 513-519.

Parkin, T.D.H., Clegg P.D., French N.P., Proudman C.J., Riggs C.M., Singer E.R., Webbon P.M. and Morgan K.L. (2004c). Race and course level risk factors for fatal distal limb fracture in racing Thoroughbreds. *Equine Veterinary Journal* **36**, 521-526.

Parkin, T.D.H., Brown, P.E., French, N.P. and Morgan, K.L. (2005a) Cooking the books, or simply getting the best out of the data? – Assessing the nature of the relationship between variables. *Equine Veterinary Journal* **37**, 189-191.

Parkin, T.D.H., Clegg, P.D., French, N.P., Proudman, C.J., Riggs, C.M., Singer, E.R., Webbon, P.M. and Morgan, K.L. (2005b). Risk factors for fatal lateral condylar fracture of the third metacarpus/metatarsus in UK racing. *Equine Veterinary Journal* **37**, 192-199.

Parkin, T.D.H., Clegg, P.D., French, N.P., Proudman, C.J., Riggs, C.M., Singer, E.R., Webbon, P.M. and Morgan, K.L. (2006) Analysis of horse race videos to identify intra-race risk factors for fatal distal limb fracture. *Preventive Veterinary Medicine*, in press.

Peduzzi PN, Concato J, Kemper E, Holford TR and Feinstein A (1996) A simulation study of the number of events per variable in logistic regression analysis. *Journal of Clinical Epidemiology* **99**:1373-1379

Peloso, J.G., Mundy, G.D. and Cohen, N.D. (1994) Prevalence of and factors associated with musculoskeletal racing injuries of Thoroughbreds. *Journal of the American Veterinary Medical Association* **204**, 620-626.

Peloso, J.G., Cohen, N.D., Mundy, G.D., Watkins, J.P., Honnas, C.M. and Moyer, W. (1996) Epidemiologic study of musculoskeletal injuries in racing Thoroughbred horses in Kentucky. In: *Proceedings of the American Association of Equine Practitioners* **42**. pp 284-285.

Perkins, N.R. (2005) *Epidemiology Of Health And Performance In New Zealand Racehorses*. PhD thesis, Massey University, New Zealand.

Perkins, N.R., Reid, S.W. J. and Morris, R.S. (2005a) Risk factors for musculoskeletal injuries of the lower limbs in Thoroughbred racehorses in New Zealand. *New Zealand Veterinary Journal* **53**, 171-183.

Perkins, N.R., Reid, S.W. J. and Morris, R.S. (2005b) Risk factors for injury to the superficial digital flexor tendon and suspensory apparatus in Thoroughbred racehorses. *New Zealand Veterinary Journal* **53**, 184-192.

- Phillips, W., Giguere, S., Franklin, R.P., Hernandez, J., Adin, D. and Peloso, J. G. (2003) Cardiac troponin I in pastured and race-training Thoroughbred horses. *Journal of Veterinary Internal Medicine* **17**, 597-599.
- Pinchbeck, G.L., Clegg, P.D., Proudman, C.J., Morgan, K.L., Wood, J.L.N. and French, N.P. (2002) Risk factors and sources of variation in horse falls in steeplechase racing in the UK. *Preventive Veterinary Medicine* **55**, 179-192.
- Pinchbeck, G.L., Clegg, P.D., Proudman, C.J., Morgan, K.L. and French, N.P. (2003) Case-control study to investigate risk factors for horse falls in hurdle racing in England and Wales. *The Veterinary Record* **152**, 583-587.
- Pinchbeck, G.L., Clegg, P.D., Proudman, C.J., Morgan, K.L. and French, N.P. (2004a) Whip use and race progress are associated with horse falls in hurdle and steeplechase racing in the UK. *Equine Veterinary Journal* **36**, 384-389.
- Pinchbeck, G.L., Clegg, P.D., Proudman, C.J., Stirk, A., Morgan, K.L. and French, N.P. (2004b) Horse injuries and racing practices in National Hunt racehorses in the UK: the results of a prospective cohort study. *The Veterinary Journal* **167**, 45-52.
- Platt, H. (1982) Sudden and unexpected death in horses: a review of 69 cases. *British Veterinary Journal* **138**, 417-429.
- Poole, D.C. and Erickson, H.H. (2004) Heart and vessels: function during exercise and response to training. In: *Equine Sports Medicine and Surgery*. Eds. Hinchcliff, K.W., Kaneps, A.J. and Geor, R.J. Elsevier Limited, London, United Kingdom. pp 714-715.
- Poole, R.R. and Meagher, D.M. (1990) Pathogenic findings and pathogenesis of racetrack injuries. *Veterinary Clinics of North America: Equine Practice* **6**, 1-29.
- Pratt, G.W. Jr. and O'Connor, J.T. Jr. (1976) Force plate studies of equine biomechanics. *American Journal of Veterinary Research*. **37**, 1251-1255.
- Racing Victoria Ltd. (1998) *Report Of The Independent Panel Appointed By Racing Victoria Ltd. To Review Jump Racing In The State Of Victoria*. Melbourne, Australia. p 13.
- Racing Victoria Ltd. (2002) *Report Of The Independent Panel Appointed By Racing Victoria Ltd. To Review Jump Racing In the State Of Victoria*. Melbourne, Australia. pp 11-40.
- Racing Victoria Ltd. (2005a) *Jumping Racing Review Panel Report*. Melbourne, Australia. pp 2-37.
- Racing Victoria Ltd. (2005b) *2005 Annual Report*. Melbourne, Australia. pp 2-37.
- Racing Victoria Ltd. website 2005. <http://www.racingvictoria.com.au> (Accessed December 2005).
- Reimer, J.M., Reef, V.B. and Sweeney, R.W. (1992) Ventricular arrhythmias in horses: 21 cases (1984-1989). *Journal of the American Veterinary Medical Association* **201**, 1237-1243.
- Rick, M.C., O'Brien, T.R., Pool, R.R. and Meagher, D. (1983) Condylar fractures of the third metacarpal bone and third metatarsal bone in 75 horses: radiographic features, treatments and outcome. *Journal of the American Veterinary Medical Association* **183**, 287-296.

- Riggs, C.M. (1999) Aetiopathogenesis of parasagittal fractures of the distal condyles of the third metacarpal and third metatarsal bones – review of the literature. *Equine Veterinary Journal* **31**, 116-120.
- Riggs, C.M. (2002) Fractures - a preventable hazard of racing Thoroughbreds? *Veterinary Journal* **163**, 19-29.
- Riggs, C.M., Whitehouse, G.H. and Boyde, A. (1999a) Pathology of the distal condyles of the third metacarpal and third metatarsal bones of the horse. *Equine Veterinary Journal* **31**, 140-148.
- Riggs, C.M., Whitehouse, G.H. and Boyde, A. (1999b) Structural variation of the distal condyles of the third metacarpal and third metatarsal bones in the horse. *Equine Veterinary Journal* **31**, 130-139.
- Robinson, R.A and Gordon, B. (1990) American Association of Equine Practitioners track breakdown studies – horse results. In: *Proceedings of the 7th International Conference of Racing Analysts and Veterinarians*. Kentucky. pp 385-393.
- Robinson, R.A., Kobluk, C., Clanton, C., Martin, F., Gordon, B., Ames, T., Trent, M. and Ruth, G. (1988) Epidemiological studies of musculoskeletal racing and training injuries in Thoroughbred horses, Minnesota, U.S.A. *Acta Veterinaria Scandinavica* **84**, Suppl. 340-343.
- Rockhill B., Weinberg, C.R. and Newman, B. (1998) Population attributable fraction estimation for established breast cancer risk factors: considering the issues of high prevalence and unmodifiability. *American Journal of Epidemiology* **147**, 826-833.
- Rodríguez, G. and Elo, I. (2003) Intra-class correlation in random-effects models for binary data. *The Stata Journal* **3**, 32-46.
- Rooney, J.R. (1982a) The relationship of length of race to fatigue and lameness in Thoroughbred racehorses. *Journal of Equine Veterinary Science* May/June, 98-101.
- Rooney, J.R. (1982b) The relationship of season of the year to lameness and breakdown in Thoroughbred racehorses. *Journal of Equine Veterinary Science* September/October, 174-176.
- Rooney, J.R. (1983a) Impulse and breakdown on straights and turns in racehorses. *Journal of Equine Veterinary Science* **3**, 137-139.
- Rooney, J.R. (1983b) The relationship of sex to breakdown in Thoroughbred racehorses. *Equine Veterinary Data* **4**, 32.
- Rooney, J.R. (1983c) Further studies on breakdown in Thoroughbred racehorses. *Equine Veterinary Data* **4**, 133.
- Rooney, J.R. (1983d) Track condition in relationship to fatigue and lameness in Thoroughbred racehorses. *Equine Veterinary Data* **4**, 134.
- Rossdale, P.D., Hopes, R., Wingfield Digby, N.J. and Offord, K. (1985) Epidemiological study of wastage among racehorses 1982 and 1983. *The Veterinary Record* **116**, 66-69.
- Rothman, K.J. and Greenland, S. (1998a) Case-control studies. In: *Modern Epidemiology*, 2nd edition. Eds. Rothman, K.J. and Greenland, S. Lippincott-Raven, Philadelphia. pp 93-114.

- Rothman, K.J. and Greenland, S. (1998b) Precision and validity in epidemiologic studies. In: *Modern Epidemiology*, 2nd edition. Eds. Rothman, K.J. and Greenland, S. Lippincott-Raven, Philadelphia. pp 115-134.
- Rufenacht, J., Schaller, P., Audige, L., Knutti, B., Kupfer, U. and Peterhans, E. (2001) The effect of infection with bovine viral diarrhoea virus on the fertility of Swiss dairy cattle. *Theriogenology*. **56**, 199-210.
- Schoen, F. (2005) The heart. In: *Robbins and Cotran Pathologic Basis of Disease*, 7th edition. Eds. Kumar V., Abbas, A. and Fausto, N. W.B. Saunders Company, Philadelphia. pp 578-581.
- Sinha, A.K., Gleed, R.D., Hakim, T.S., Dobson, A. and Shannon, K.J. (1996) Pulmonary capillary pressure during exercise in horses. *Journal of Applied Physiology* **80**, 1792-1798.
- Schwarzwald, C.C., Hardy, J. and Buccellato, M. (2003) High cardiac troponin I serum concentration in a horse with multifocal ventricular tachycardia and myocardial necrosis. *Journal of Veterinary Internal Medicine* **17**, 364-368.
- Seeman, E. (2002) Pathogenesis of bone fragility in women and men. *The Lancet* **359**, 1841-1850.
- Sherman, K.M., Miller, G.J., Wronski, T.J., Colahan, P.T., Brown, M. and Wilson, W. (1995) The effect of training on equine metacarpal bone breaking strength. *Equine Veterinary Journal* **27**, 135-139.
- Singer, E.R., Saxby, F. and French, N.P. (2003) A retrospective case-control study of horse falls in the sport of horse trials and three-day eventing. *Equine Veterinary Journal* **35**, 139-145.
- Snijders, T.A.B. and Bosker, R.J. (1999) *Multilevel Analysis. An Introduction To Basic And Advanced Multilevel Modeling*. Sage Publications Ltd., London. pp 223-224.
- Stephen, J.O., White, N.A., McCormick, W.H., Cowles, R.R. and Corley, K.T.T. (2003) Risk factors and prevalence of injuries in horses during various types of steeplechase races. *Journal of the American Veterinary Medical Association* **12**, 1788-1790.
- Stover, S.M., Johnson, B.J., Daft, B.M., Read, D.H., Anderson, M., Barr, B.C., Kinde, H., Moore, J., Stoltz, J., Ardans, A.A. and Pool, R.R. (1992) An association between complete and incomplete stress fractures of the humerus in racehorses. *Equine Veterinary Journal* **24**, 260-263.
- Stover, S.M., Ardans, A.A., Read, D.H., Johnson, B.J., Barr, B.C., Daft, B.M., Kinde, H., Anderson, M., Woods, L.W., Moore, J., Stoltz, J. and Pool, R.R. (1993) Patterns of stress fractures associated with complete bone fractures in racehorses. In: *Proceedings of the American Association of Equine Practitioners 39*. San Antonio, Texas. pp 131-132.
- Stover, S.M., Read, D.H., Johnson, B.J., Harrington, T., Ardans, A.A., Anderson, M., Barr, B.C., Daft, B.M., Kinde, H., Moore, J., Stoltz, J. and Woods, L.W. (1994) Lateral condylar fracture histomorphology in racehorses. In: *Proceedings of the American Association of Equine Practitioners 40*. Vancouver, British Columbia. pp 173.
- Stubbs, A.K. (2004) *Racetrack Design And Performance. Database Of Current Knowledge - A Report For The Rural Industries Research And Development Corporation*. Rural Industries Research and Development Corporation, Kingston, Australia. pp 1-48.

Suann, C. (1992) Racecourse casualties. In: *Proceedings of the 9th International Conference of Racing Analysts and Veterinarians*. New Orleans, Louisiana. pp 25-26.

Takahashi, T., Kasashima, Y. and Ueno, Y. (2004) Association between race history and risk of superficial digital flexor tendon injury in Thoroughbred racehorses. *Journal of the American Veterinary Medical Association* **225**, 90-93.

Thrusfield M. (2005) *Veterinary Epidemiology*. 2nd Edition. Blackwell Science Ltd., Oxford. pp 39-64, 227-228.

Ueda, Y., Yoshida, K. and Oikawa, M. (1993) Analyses of race accident conditions through use of patrol video. *Journal of Equine Veterinary Science* **13**, 707-710.

Vaughan, L.C. and Mason, B.J.E. (1976) *A Clinico-pathological Study Of Racing Accidents In Horses. A Report Of A Study On Equine Fatal Accidents On Racecourses*. Horserace Betting Levy Board, London, United Kingdom. pp 3-88.

Verheyen, K.L.P., Newton, J.R. and Wood, J.L.N. (2003) A case-control study investigating factors associated with pelvic and tibial stress fractures in Thoroughbred racehorses in training. In: *Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine*. Warwick, United Kingdom. pp 119-131.

Verheyen, K.L.P. and Wood, J.L.N. (2004) Descriptive epidemiology of fractures occurring in British Thoroughbred racehorses in training. *Equine Veterinary Journal* **36**, 167-173.

West, J.B. and Mathieu-Costello, O. (1994) Stress failure of pulmonary capillaries as a mechanism for exercise induced pulmonary haemorrhage in the horse. *Equine Veterinary Journal* **26**, 441-447.

West, J.B., Mathieu-Costello, O., Jones, J.H., Birks, E.K., Logemann, R.B., Pascoe, J.R. and Tyler, W.S. (1993) Stress failure of pulmonary capillaries in racehorses with exercise-induced pulmonary hemorrhage. *Journal of Applied Physiology* **75**, 1097-1109.

Williams, R.B., Harkins, L.S., Hammond, C.J. and Wood, J.L. (2001) Racehorse injuries, clinical problems and fatalities recorded on British racecourses from flat racing and National Hunt racing during 1996, 1997 and 1998. *Equine Veterinary Journal* **33**, 478-486.

Wilson, E.B. (1927) Probable inference, the law of succession, and statistical inference. *Journal of the American Statistical Association* **22**, 209-212.

Wilson, J.H. and Robinson, R.A. (1996). Risk factors for equine racing injuries. *The Compendium of Continuing Education for the Practising Veterinarian* **18**, 682-690.

Wilson, J.H., Howe, S., Jensen, R. and Robinson, R.A. (1993) Injuries sustained during racing at racetracks in the U.S. in 1992. In: *Proceedings of the American Association of Equine Practitioners* 39. San Antonio, Texas. pp 267-268.

Wilson, J.H., Jensen, R. and Robinson, R.A. (1996a) Racing injuries of two year old Thoroughbreds and Quarter horses. *Pferdeheilkunde* **12**, 582-587.

Wilson, J.H., Robinson, R.A., Jensen, R.C. and McArdle, C. (1996b) Equine soft tissue injuries associated with racing descriptive statistics from American racetracks. In: *Proceedings of the 1st Dubai International Equine Symposium*. Dubai, United Arab Emirates. pp 1-21.

Wilson, J.H., Shaw, K.K. and King, V. (1997) Thoroughbred racing injury rates are inversely associated with trainer success. In: *Proceedings of the American Association of Equine Practitioners 43*. Phoenix Arizona. pp 229-230.

Wilson, P.D., Loffredo, C.A., Correa-Villasenor, A. and Ferencz, C. (1998) Attributable fraction for cardiac malformations. *American Journal of Epidemiology* **148**, 414-423.

Wood, J.L.N., Harkins, L. and Rogers, K. (2000) A retrospective study of factors associated with racehorse fatality on British racecourses from 1990-1999. In: *Proceedings of the 13th International Conference of Racing Analysts and Veterinarians*. Cambridge, United Kingdom. pp 274-277.

Wood, J.L.N., Eastment, J., Harkins, L. and Rogers, K. (2002) *Towards Better Welfare Of Racehorses. Final Report*. Animal Health Trust, Newmarket, United Kingdom. pp 1-15.

Woodward, R.H. and Goldsmith, P.L. (1964) *Cumulative Sum Techniques. Imperial Chemical Industries Mathematical and Statistical Techniques For Industry Monograph No. 3*. Oliver and Boyd, Edinburgh.

Zou G.Y. (2004) A modified Poisson regression approach to prospective studies with binary data. *American Journal of Epidemiology* **159**, 702-706.

Risk Factors Associated with Racetrack Casualties in Thoroughbreds

RIRDC Publication No. 08/007

This Report describes the investigation of the risk factors that lead to injuries and fatalities as a retrospective study of Thoroughbred racing in Victoria.

The success of the Thoroughbred horse industry is vital to the economic health of Victoria and Australia. Key research issues for the Victorian racing industry revolve around animal and jockey welfare. This focus is dictated by community concerns over high wastage rates for racehorses and jockey injuries and fatalities sustained in racing. Although there will inevitably be injury in athletic competition, risk of injury could be reduced if risk factors are identified and modified.

This RIRDC-funded epidemiological research aimed to investigate Thoroughbred racing fatalities by

- identifying causes of death of horses during or after racing through post mortem examinations

- describing the risk of fatality in flat and jump racing for the previous 15 years
- identifying risk factors for fatality in flat and jump racing so that intervention strategies to reduce the risk of fatality can be developed by the industry in the future.

The Rural Industries Research and Development Corporation (RIRDC) manages and funds priority research and translates results into practical outcomes for industry.

Our business is about new products and services and better ways of producing them.

Most of the information we produce can be downloaded for free from our website: www.rirdc.gov.au.

RIRDC books can be purchased by phoning 02 6271 4100 or online at: www.rirdc.gov.au/eshop.

Photo: Bruno Cammatelli, Ultimate Racing Photos



This publication can be viewed at our website—www.rirdc.gov.au. All RIRDC books can be purchased from:

www.rirdc.gov.au

Contact RIRDC:
Level 2
15 National Circuit
Barton ACT 2600

PO Box 4776
Kingston ACT 2604

Ph: 02 6271 4100
Fax: 02 6271 4199
Email: rirdc@rirdc.gov.au
web: www.rirdc.gov.au

RIRDC Innovation for rural Australia