


# Racing-associated fatalities in Norwegian and Swedish harness racehorses: Incidence rates, risk factors, and principal *postmortem* findings

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## Abstract

**Background:** There are no reports on the number of fatalities or causes of death in the Norwegian and Swedish harness racehorses.

**Objectives:** The incidence rates (IRs), risk factors, and *postmortem* findings in horses that died or were euthanized associated with racing between 2014 and 2019 were investigated.

**Animals:** Thirty-eight Standardbreds and 10 Norwegian-Swedish Coldblooded Trotters died or were euthanized associated with racing. A total of 816 085 race-starts were recorded.

**Methods:** Incidence rates were calculated for both countries and horse breeds. Risk factors for sudden death were identified using a case-control logistic model. *Postmortem* examinations were performed in 43 horses.

**Results:** The overall fatality IR was 0.059/1000 race-starts. Traumatic injuries accounted for 14.5%, while sudden death for 85.5% of fatalities. Only minor differences between countries and breeds were recorded. The number of starts within the last 30 days increased the risk of sudden death (5 starts odds ratio (OR) 228.80, confidence interval (CI) 10.9-4793). An opposite non-linear effect was observed in number of starts the last 180 days (>10 starts OR 0.12, CI 0.02-0.68). Seven horses were euthanized because of catastrophic injury. Acute circulatory collapse because of suspected cardiac or pulmonary failure or both was recorded in 30 horses, while major hemorrhage after vessel rupture was the primary cause of death in 10 cases. One horse collapsed and died but was not submitted for autopsy.

**Conclusions and Clinical Importance:** Comparatively low rates of catastrophic orthopedic fatalities were reported, while causes and IR of sudden death were similar to previous studies.

**Abbreviations:** CI, confidence interval; IR, incidence rate; NSCT, Norwegian-Swedish Coldblooded Trotter; OR, odds ratio; PME, *postmortem* examination; RASD, racing-associated sudden death; SAD, sudden athletic death; STB, Standardbred.

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## KEYWORDS

horse, sudden athletic death, sudden cardiac death, sudden death

## 1 | INTRODUCTION

Horses that die or are euthanized in association with racing challenge the sport concerning horse welfare and the public perception of racing. Therefore, it is essential to investigate all racing-related fatalities to better understand their etiology to help prevent these from occurring.

Previous studies have described the incidence rates (IRs) and *postmortem* findings and identified possible risk factors for horses that die in association with exercise and racing.<sup>1-7</sup> The IR has been reported to vary and appear to be influenced by a number of factors, including race discipline and distance and track surface.<sup>2,4,6,8-10</sup> The identification and interpretation of risk factors have proven challenging concerning separating those horses euthanized because of a catastrophic orthopedic injury and those that collapse and die of a suspected cardiopulmonary cause.<sup>4,5</sup> A similar challenge occurs when evaluating *postmortem* findings, as similar cardiopulmonary findings can be present in both groups of horses making interpretation of their relevance difficult.<sup>3,5</sup> Autopsy protocols and tissue sampling sites can also vary between investigators making comparison between studies difficult.<sup>3,5</sup>

Although harness racing is a popular and economically important sport in Norway and Sweden, there are no published reports on IR, risk factors, or *postmortem* findings of racing-associated fatalities in these countries. The principal aims of this study were to describe the IR of equine fatalities associated with harness racing in Norway and Sweden between 2014 and 2019 and to investigate possible risk factors for those horses that collapsed and died using a case-control approach. Additionally, the *postmortem* findings and proposed causes of death in these horses are described.

## 2 | METHODS

### 2.1 | Study design

The retrospective case-control study recorded the number of fatalities on Norwegian and Swedish harness racetracks during 6 years from 1 January 2014 to 31 December 2019. Information on the total number of horses that died and the total number of race-starts was obtained from the Norwegian and Swedish Trotting Associations.

In order to identify possible risk factors, breed, age, sex, the total number of starts, the number of starts within the previous 30, 60, 90, 180, and 360 days, respectively, the racing distance at the time of death and the time of year in which the horse died were all evaluated. The time of year was divided into 4 groups: winter (December-February), spring (March-May), summer (June-August), and fall (September-November). Whether the horse competed in a trotting race under saddle or a regular harness race was also recorded. All individual horse and racing data were obtained from the respective racing

associations' official web pages (<https://www.travsport.no>; <https://www.travsport.se>).

### 2.2 | Inclusion criteria

The inclusion criteria were defined as apparently healthy horses that died suddenly or were euthanized after injury immediately before the start, during, or within 1 hour after racing. These 2 groups of horses combined were defined as a racing-associated deaths (RAD). Horses within the RAD group that died spontaneously or were euthanized while recumbent because of extreme cardiac, respiratory distress, or both in the absence of a catastrophic orthopedic or traumatic injury were defined as sudden athletic deaths (SAD). This latter term was proposed by Physick-Sheard and Slack<sup>11</sup> to describe any horse that dies suddenly associated with exercise. Proportional fatality rates were used to describe the proportion of all fatalities attributed to either fatal orthopedic/traumatic injury or SAD.

Standardbreds (STBs) and Norwegian-Swedish Coldblooded Trotters (NSCTs) of any sex or age were included in the study. Both breeds are used in commercial harness racing in Norway and Sweden. They compete in separate breed-specific races but over the same racing distances and for similar prize money, making racing equally competitive between the 2 breeds.

### 2.3 | Risk factors

A case-start was defined as a start resulting in death. For each case-start, 3 control horses that participated in the same race resulting in a case were randomly selected using Excel software (Excel Random Generator, RAND, Microsoft).

To identify individual risk factors for SAD, breed, age, sex, the total number of starts, and the number of starts within the previous 30, 60, 90, 180, and 360 days were evaluated. Individual horse and racing data were obtained from the respective racing associations' official web pages (<https://www.travsport.no>; <https://www.travsport.se>). With this matched design, breed, season, year, country, race type (harness race or under saddle), and distance were not included in the statistical risk analysis.

### 2.4 | Statistical analyses

#### 2.4.1 | Incidence rates

Incidence rates are defined as the number of new cases of disease in a population per unit of animal-time during a given time period.<sup>12</sup> The

IRs were therefore calculated based on the number of animals that died compared to the total number of race-starts within the same 6-year investigation period. Incidence rates for all deaths combined (RAD) and SAD were calculated and expressed as IR/1000 race-starts. The same rates were also calculated for the 2 different countries and NSCTs and STBs separately.

#### 2.4.2 | Risk factors

To identify individual risk factors for SAD, data were analyzed using a conditional logistic model in Stata (16/ MP for Windows, StataCorp, College Station, Texas). To assess model fit, the `fitstat` command in Stata was used to assess the improvement linked to choosing a conditional model. The fit was clearly improved. After checking for correlations between explanatory variables, a backward elimination method was used to identify the model using the Likelihood Ratio test at a cut-point of  $P < .05$ . Results were presented as odds ratios (ORs) with a 95% confidence interval (CI). To give a more meaningful interpretation of results, separate ORs were presented for the number of starts the last 30 days (1-5 starts respectively) and the last 180 days (1-4, 5-7, 8-10, and >10 starts, respectively).

#### 2.4.3 | Postmortem examination

Horses that die or are euthanized during official race meetings in Norway and Sweden are subjected to a *postmortem* examination (PME) at the nearest state-run veterinary investigation center (The Norwegian Veterinary Institute, Norway and The National Veterinary Institute, Sweden). Details on all horses that died and their *postmortem* reports were made available through the respective racing bodies with the owners' informed consent. All reports were handled confidentially.

As there were several pathologists involved using different terminologies not only between the 2 countries but also between the different pathologists in the same country, the *postmortem* findings were evaluated and summarized by 2 experienced pathologists, 1 from each of the respective countries (EK and MV).

The presence of pulmonary edema, congestion, and hemorrhage was recorded and graded as none present, mild, moderate, or marked. Any macro- or microscopic cardiac findings were summarized and described, as were other major relevant findings. The stated primary cause of death and the *postmortem* findings related to this were based on the pathologist's conclusion performing the individual PME.

### 3 | RESULTS

#### 3.1 | Horses

A total of 48 horses died during the study period, which included 38 STBs and 10 NSCTs. There were 12 stallions (2 STBs, 10 NSCTs),

22 geldings (18 STBs, 4 NSCTs), 13 mares (10 STBs, 3 NSCTs) in total, and 1 horse where this information was missing. The overall age range was 2 to 11 years (median 5.0 years), 2 to 11 years for STBs (median 5.0 years), and 3 to 9 years for NSCTs (median 6.0 years).

#### 3.2 | Racing data

The race distances in the current study varied from 1609 to 2680 m. Six races were run over 1609 m, 7 races over 2500 to 2680 m, and 34 races were run over 2100 to 2200 m. In 1 race, the racing distance was not recorded. The track surfaces for all tracks were sand-based standard all-weather tracks.

#### 3.3 | Incidence rate

Of the 48 RAD recorded, 33 occurred in Sweden and 15 in Norway. The total number of race-starts was 816 085, of which 576 162 were in Sweden and 239 923 in Norway.

This represented an overall RAD IR of 0.059/1000 race-starts for the 2 countries combined (Table 1). The overall RAD IR in Sweden was 0.057 while in Norway it was 0.063/1000 race-starts.

The total number of STB race-starts during the study period was 645 613 (where 38 RAD were recorded), while for the NSCTs, the number of race-starts was 170 472 (10 RAD recorded), reflecting the racing population of the 2 breeds.

Forty-one horses were classified as SAD, representing an overall IR of 0.050/1000 race-starts for the 2 countries combined (Table 2). Sweden recorded 30 SADs, while in Norway there were 11 such cases representing an overall IR of 0.052 and 0.046/1000 race-starts, respectively.

Seven horses (5 STBs and 2 NSCTs) were euthanized because of catastrophic orthopedic or traumatic injury. This represents an overall IR of 0.009/1000 race-starts. Three cases (all STBs) were recorded in Sweden and 4 in Norway (2 STBs and 2 NSCTs), representing an IR of 0.005 and 0.017/1000 race-starts, respectively, for the 2 countries.

The IR for SAD varied between seasons, being highest in the spring (0.064/1000 race-starts; Figure 1). Both spring and summer

**TABLE 1** Incidence rates for racing-associated death (RAD) per 1000 race-starts for Standardbred racehorses (STB) and Norwegian-Swedish Coldblooded Trotters (NSCT) in Norway and Sweden combined between 2014 and 2019

Year	STB	NSCT	STB and NSCT
2014	0.035	0.069	0.043
2015	0.043	0.139	0.062
2016	0.082	0	0.065
2017	0.095	0.070	0.090
2018	0.030	0.035	0.031
2019	0.072	0.037	0.064
2014 to 2019 overall	0.059	0.059	0.059

had similarly high rates in Sweden (0.063 and 0.066/1000 race-start respectively; Figure S1). In Norway, the highest IR occurred during spring (0.065/1000 race starts; Figure S2).

### 3.4 | Proportional fatality rates

The overall proportional fatality rates were 14.5% for catastrophic orthopedic/traumatic injuries and 85.5% for SAD. For STBs, the proportional fatality rates were 13.2% and 86.8%, and for the NSCTs, 20.0% and 80.0% for fatal orthopedic/traumatic injuries and SAD, respectively.

Seven of the 41 SAD horses died before the start of the race (5 immediately before the start and 2 during warm-up approximately 30 minutes prior to the race), 14 during the race, and the remaining 20 immediately after the race.

### 3.5 | Risk factors

A total of 41 sets of 1 case and 3 randomly selected control horses from the same race were used in the analyses, with the case as the outcome. Individual factors in the risk analysis included sex (mares [n = 46]; geldings [n = 94]; stallions [n = 25]), age groups (<4 years [n = 32]; 4-5 years [n = 59]; 6-8 years [n = 57]; >8 years [n = 18]), and number of starts in the last 30, 60, 90, 180, and 360 days. The final logistic model included sex and starts in the last 30 and 180 days as the remaining explanatory variables. The results from this model are shown in Table 3.

As shown in Table 3, the number of starts within the last 30 days increased the risk of SAD (5 starts OR 228.80, CI 10.9-4793). An opposite non-linear effect was observed in number of starts the last 180 days (>10 starts OR 0.12, CI 0.02-0.68).

### 3.6 | Postmortem examinations

Of the 48 RAD recorded, 43 horses received a full PME. These included 40 SAD cases and 3 horses euthanized because of catastrophic trauma.

**TABLE 2** Incidence rates for sudden athletic death (SAD) per 1000 race-starts for Standardbred racehorses (STB) and Norwegian-Swedish Coldblooded Trotters (NSCT) in Norway and Sweden combined between 2014 and 2019

Year	STB	NSCT	STB and NSCT
2014	0.030	0.034	0.028
2015	0.043	0.139	0.063
2016	0.073	0.000	0.058
2017	0.095	0.035	0.082
2018	0.030	0.035	0.031
2019	0.041	0.037	0.040
2014 to 2019 overall	0.051	0.047	0.050

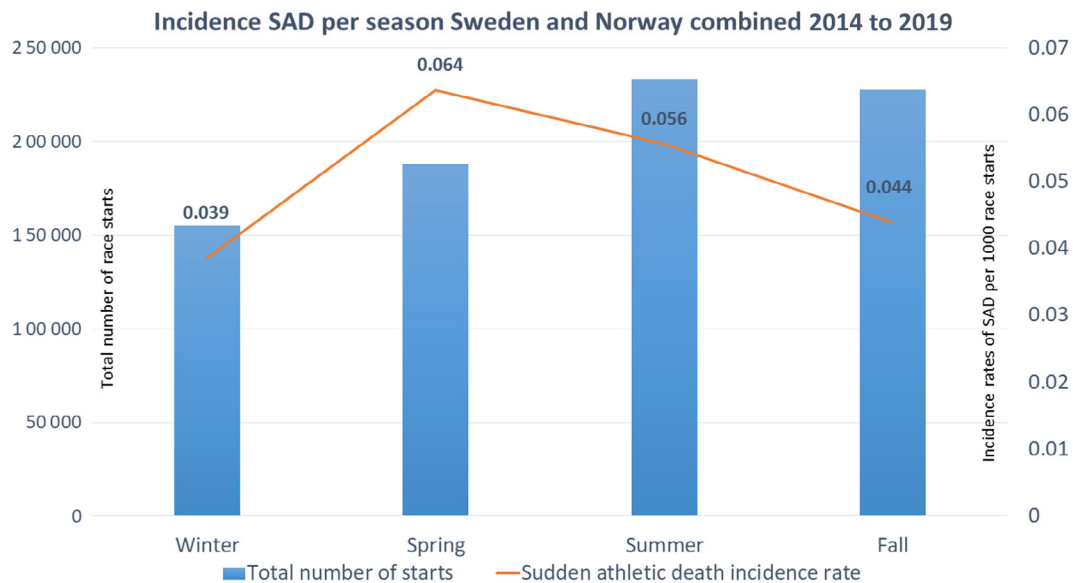
The 4 Norwegian horses that did not have a full PME were all horses that died or were euthanized because of traumatic injuries. However, on-course clinical, radiographic examinations or both were performed on all cases. Only 1 horse in Sweden did not have a PME performed. As this horse collapsed and died immediately after the race, it was classified as a SAD case but with an unknown primary cause.

### 3.7 | Primary cause of death and principal postmortem findings

Seven of the 48 RADs were euthanized because of traumatic injuries, 4 of which were in the Norwegian horses, while the remaining 3 occurred in Sweden. Three horses were euthanized because of catastrophic proximal phalanx (P1) fractures (2 in Norway and 1 in Sweden), 1 horse because of a tibial fracture (Sweden), 1 horse suffered a cervical dislocation because of a fall (Sweden), 1 horse sustained a soft tissue trauma to a hindlimb, and finally, 1 horse suffered a thoracic impalement trauma (both in Norway).

The primary cause of death in the remaining 40 SAD horses (with 1 SAD case in Sweden that did not have a PME performed excluded) was concluded by the respective pathologists to be caused by acute circulatory collapse because of suspected cardiac, pulmonary failure, or both in 30 horses. Major hemorrhage because of vessel rupture was reported as the cause of death in 10 cases. Of the 30 horses with acute circulatory failure, 27 were of an unknown primary cause. Although none of these horses had gross cardiac findings consistent with being the primary cause of death, 3 horses were reported to have a dilated right ventricle with additional congestion of the coronary arteries reported in 1 horse. Another 8 horses had focal areas of myocardial hemorrhage or petechiae while hydropericardium was reported in 4 further horses. Microscopic changes in 5 horses included focal and diffuse areas of myocardial hemorrhage, congestion, and inflammation. The latter included local areas of mild lymphocytic inflammatory infiltrates. Moderate multifocal fibrosis and necrosis were separate findings in 3 animals. In 3 horses, the autolytic changes were too advanced to enable microscopic evaluation of the myocardium. In 3 of 30 horses, exercise-induced pulmonary hemorrhage, pulmonary edema, or both were determined to be the cause of death. However, all 30 horses had varying degrees of pulmonary hemorrhage, edema, and congestion.

Ten horses had rupture of major vessels, of which 7 were intrathoracic while 3 were intraabdominal mesenteric artery ruptures. The 3 intrathoracic vessel ruptures were either complete or incomplete aortic wall ruptures, 1 *truncus brachiocephalicus* rupture, and 2 unidentified vessel ruptures. All 3 horses with abdominal vessel ruptures died because of hemorrhagic shock, as did 4 horses with intrathoracic bleeds. In 1 of the remaining 3 horses, cardiac tamponade associated with an incomplete aorta rupture was thought to be the principal consequence and attributing cause of the sudden death. Similarly, 2 horses had dissecting myocardial hemorrhages, 1 caused by aortic rupture and the other of unknown origin, which were proposed to have affected the electrical conduction system resulting in fatal arrhythmias.



**FIGURE 1** Incidence rates for sudden athletic death (SAD) per 1000 race-starts during 4 seasons of the year for Standardbred racehorses and Norwegian-Swedish Coldblooded Trotters combined in Sweden and Norway between 2014 and 2019. The seasons were divided into 4 groups: winter (December-February), spring (March-May), summer (June-August), and fall (September-November)

**TABLE 3** Final conditional logistic model showing odds ratios (ORs) for the risk of sudden athletic death (SAD) occurring with 95% confidence intervals (CI) and corresponding *P*-values for sex and number of starts in the last 30 and 180 days

Variable	OR	95% CI	<i>P</i> -value
Sex			
Mare	1.00	-	-
Gelding	1.09	0.38 to 3.07	.88
Stallion	4.20	0.90 to 19.60	.07
Number of starts last 30 days			
1	1.00	-	-
2	4.07	0.77 to 21.6	.10
3	8.42	1.53 to 46.3	.01
4	14.71	2.12 to 102.1	.007
5	228.80	10.9 to 4793	<.001
Number of starts last 180 days			
1 to 4	1.00	-	-
5 to 7	0.16	0.03 to 0.92	.04
8 to 10	0.25	0.05 to 1.15	.08
>10	0.12	0.02 to 0.68	.02

Note: Significance levels were set at  $P < .05$ .

The 3 horses that underwent a full PME after orthopedic trauma included 1 horse with a tibial fracture, 1 horse with cervical dislocation, and 1 with a P1 fracture. Only the horse with a tibial fracture had marked pulmonary edema, congestion, and hemorrhage. Neither of the other 2 horses had any recorded pulmonary or cardiac changes.

A summary of the *postmortem* findings is described in Table S1.

## 4 | DISCUSSION

This study is the first report on racing fatalities in harness racehorses in Norway and Sweden and demonstrated the overall RAD IR to be lower than previously reported.<sup>2,4,6,9,10,13-16</sup> The number of starts in the last 30 and 180 days was identified as risk factors for SAD, although the low number of cases meant that the statistical power of these findings was weak. The *postmortem* findings, as well as the challenges in interpreting these, were similar to previous reports.<sup>3,9,17,18</sup>

Varying RAD IR has been reported to be dependent on several factors, including horse breed, race discipline, and track surface. Thoroughbred racing typically has a higher number of fatalities ranging from 0.44 to 8.3/1000 race-starts with steeplechase racing the highest rate and flat racing on turf the lowest.<sup>2,6,10,14,16,19</sup> Only limited information is available on STBs, although Physick-Sheard et al.<sup>6</sup> reported an RAD rate of 0.63/1000 race-starts, which is still higher than the overall IR of 0.059/1000 race-starts reported in the current study. However, Physick-Sheard et al.<sup>6</sup> also included horses euthanized up to 60 days after a race start. In the current study, only horses that died or were euthanized on the track within 1 hour after the race were included. A similar death registry does not exist in Norway and Sweden. Therefore, it is likely that if horses euthanized within 60 days of racing were included in the current study, the number of fatalities would be higher. Orthopedic injuries commonly seen in the current racehorse population involve suspensory ligament and tendon injuries (unpublished observations). The severity and extent of these may not be immediately apparent but may later result in the horse's euthanasia.

Additional factors that could contribute to racing fatalities include track surface.<sup>10</sup> In the current study, all races were run on sand-based standard all-weather tracks, which make racing conditions very similar

all year round and identical between the 2 breeds examined. This may help explain the similar RAD IR in the current study, despite including these 2 different breeds.

Gait and lower race speeds have also been proposed as reasons for the lower number of fatalities in STBs compared to Thoroughbreds.<sup>20</sup> The lower rate of catastrophic orthopedic injury observed in 1 such study was attributed to earlier management and higher training intensity in the latter breed.<sup>6</sup> The risk of injury in human athletes has been associated with training load,<sup>21</sup> and an increased acute to chronic workload and rate of change in workload appears to increase injury risk.<sup>22</sup> In horses, higher intensity of recent exercise increased the risk for suspensory apparatus breakdown<sup>23</sup> Also, racehorses that covered large total high-speed distances or increased their total amount of high-speed work over a short period of time had an increased risk of catastrophic musculoskeletal injury.<sup>13,24</sup> Similarly, acute increases in workload increased the risk of injury in international 3-day event horses while chronic workload had no such effect.<sup>25</sup> Training regimens are likely to differ widely between different racing and equine sport disciplines as well as geographical regions. Inclusion of such data may contribute to shed light on how this could influence fatalities during competitive equestrian activities.

No data on either RAD or SAD have previously been published for NSCTs, making comparison for this breed difficult. However, the overall RAD and SAD were remarkably similar between STBs and NSCT in the current study, with both breeds experiencing comparatively few catastrophic orthopedic injuries. The NSCT has since the 1950's been an established breed originating as a rural farm and carriage horse in Norway and Sweden. However, since then it has been bred primarily for racing and is now used widely for competitive harness racing. The NSCT is a smaller but slightly heavier breed than the STB, and the maximum trotting speed and racing times are slower than for STBs. Typically, elite NSCTs may have average racing times approaching 1 minute 20 seconds/km (12.5 m/s) compared to the racing times of elite STBs of around 1 minute 10 seconds/km (14.5 m/s).<sup>26</sup> A typical racecard would have several races specifically for NSCTs and with similar prize money as for the STBs, making these races equally competitive.<sup>27,28</sup> There are few commercial 2-year-old STB races and none for NSCTs in Sweden and Norway, with the principal races being run for 4-year-old horses. Hence the NSCTs normally start their racing career at a slightly older age than the STBs but typically have a longer career.<sup>28,29</sup> Consequently, there is less pressure on having a horse race-ready at a young age compared to Thoroughbred racing. Also, both Norway and Sweden have strict regulations regarding medication and racing. This includes a conservative withdrawal time policy after systemic or local treatments and a ban on racing on any medication, including analgesics such as phenylbutazone. This may contribute to the low rate of catastrophic injuries reported for both these breeds by enforcing a period of rest for the horses and not disguising existing injuries. Similarly lower RADs have been reported in 3-day eventers, which also start training later in life and have to comply with strict regulations regarding medications. However, a larger proportion of fatalities were caused by catastrophic orthopedic injuries (62% of fatalities),<sup>30</sup> which may be attributed to the nature of competing over jumps at different surfaces as previously discussed.

In the current study, the proportion of fatalities attributed to catastrophic orthopedic/traumatic injuries was 14.5%, while the remaining were attributed to SAD (85.5% of all fatalities). This demonstrates that the vast majority of Swedish and Norwegian racetrack fatalities were due to SAD. This differs markedly from previous reports in Thoroughbred racehorses where the majority of RAD are due to catastrophic orthopedic injuries and where SAD proportional rates vary from 9% to 19%.<sup>1-6</sup>

The overall SAD IR of 0.05/1000 race-starts was closer to previously reported values. In Thoroughbreds, the lowest SAD rates were recorded for horses racing on the flat with reports of 0.07 to 0.08/1000 race-starts compared with 0.29 to 1/1000 race-starts for horses racing over jumps with the highest number of fatalities being in horses participating in steeplechases.<sup>2,4</sup> Physick-Sheard et al.<sup>6</sup> reported SAD rates for Thoroughbreds in Ontario, Canada, to be 0.376/1000 race-starts while STBs had a rate of 0.102/1000 race-starts.

Risk factors for RAD were not evaluated in the present study, given that the vast majority of these were SAD cases, and only a very small proportion caused by catastrophic traumatic injuries. The final multivariable model demonstrated that horses frequently raced in the last month before death were at increased SAD risk. Only a limited number of studies have attempted to identify risk factors associated with sudden death alone. Lyle et al.<sup>4</sup> noted in a study in Thoroughbred racehorses in the United Kingdom that age, race distance, and type, season, and the number of starts within the last 60 days were all risk factors for sudden death. They found a reduced risk of sudden death with an increasing number of starts in the 60 days before the race and proposed that frequently raced horses were likely healthy and fit, which both would reduce the risk of fatal cardiac arrhythmia.<sup>4</sup>

In contrast, the current study found an increased risk of SAD with an increasing number of starts in the last 30 days. One can only assume that these horses were all race-fit considering the frequent race-starts in the given period. It is difficult to speculate on the reason for this. However, increased cardiac troponins concentrations have been described in STBs after racing<sup>7,31,32</sup> and have also been related to subclinical myocardial damage in athletes.<sup>33-35</sup> In human athletes, increased cardiac load and inadequate recovery times contribute to cardiac structural changes,<sup>36</sup> and these changes increase the risk of arrhythmic events in humans. Several studies have documented a common occurrence of exercise-associated arrhythmias in STBs and Thoroughbred racehorses<sup>37-39</sup> and recently also in NSCTs.<sup>40</sup> However, a consensus is lacking what can be considered normal variations and also if an increased risk of SAD exists for some of these commonly observed arrhythmias<sup>36</sup> and indeed whether cardiac remodeling contributes to development of fatal arrhythmias in horses. However, cardiac fibrosis and remodeling have been linked to fatal arrhythmias and SCD in humans.<sup>41</sup> Therefore, it is conceivable, although at this point speculative, that frequent race-starts within a short period could increase myocardial damage with little time for tissue repair. This again could, therefore, possibly also increase the risk of cardiac-related SAD.

In contrast, risk associated with an increased number of starts within the last 180 days did not reach statistical significance for the



8 to 10 starts group (OR 0.25, CI 0.05-1.15). Assuming that the horses have had time to recover sufficiently between races, allowing possible myocardial injuries to heal, this could reflect that horses were better conditioned. However, this remains a speculation. The number of horses included in this study was too few to draw any firm conclusions on the risk of SAD and association with frequency of race starts.

A recent meta-analysis by Hitchen et al.<sup>42</sup> found that male horses were at increased risk of catastrophic orthopedic injuries during flat racing. However, they found no difference between intact males and geldings. In contrast, Physick-Sheard et al.<sup>6</sup> did not find a strong association with sex for the group of horses that died suddenly during racing. In the current study, there was no statistical association being an intact male horse but the low number of horses included makes it difficult to make firm conclusions based on these results.

The SAD IR was highest in the spring and summer in Norway and Sweden respectively. This is consistent with the findings of Lyle et al. They proposed that this may be due to greater metabolic demands being placed on the horse, with increased sweating increasing the risk of fatal cardiac arrhythmias.<sup>4</sup> It is certainly possible that exertional heat illness could be a contributing factor to the SAD cases but once again, it is not easy to speculate on the cause of this, primarily as the race-day temperature was not recorded.

Although race distance is a risk factor for sudden death,<sup>4</sup> this was not the case in the current study when evaluated as a single variable. However, the range of distances for harness racing in Scandinavia is narrow, with most races being run over 2100 m, with sprint races held over 1600 m, and the longer races run over 2800 to 3200 m. It is possible that the range of distances in the current study was too narrow in order to establish if there is an increased risk of dying when racing over a particular distance.

The *postmortem* findings described in this study were similar to previous reports. They included pulmonary edema, congestion and hemorrhage, major vessel rupture, as well as macro- and microscopic cardiac changes.<sup>1,3,5,9,43-48</sup> In most cases, SAD was attributed to acute circulatory failure (30/40 cases), although it was not possible to determine if death was of primary cardiac or pulmonary origin. Other studies report similar findings highlighting the difficulty in interpreting *postmortem* findings, especially relating to the importance of pulmonary and cardiac changes.<sup>1,3,5,9,43,48</sup>

Although 28 (70%) of 40 horses in the current study had marked pulmonary hemorrhage on PME, in only 3 (11%) horses was exercise-induced pulmonary hemorrhage (EIPH) determined to be the definitive cause of death by the examining pathologist. These findings are consistent with previous studies.<sup>1,3,5,9,43,48</sup> There are contrasting opinions regarding EIPH as a direct cause of sudden death. However, a recent consensus statement from the American College of Veterinary Internal Medicine (ACVIM) stated that there is currently little evidence in the literature that confirms a link between sudden death and EIPH.<sup>49</sup> In this study, 2 of the horses determined to have died of EIPH were examined by the same pathologist. However, similar findings were made in other horses not given this diagnosis by other pathologists. Pulmonary changes including congestion and hemorrhage might be present in horses euthanized from catastrophic orthopedic injury

and those suspected to have died from acute fatal arrhythmias.<sup>3,6</sup> As already suggested by other investigators, the interpretation of PME findings involves some degree of subjectivity.<sup>3,5</sup>

Similarly, macroscopic and histological cardiac findings were interpreted differently among pathologists. In the current study, 3 horses that died from acute circulatory failure with unknown cause had a dilated right ventricle. In comparison, cardiac histological changes were found in 5 horses and included myocardial inflammation, necrosis, and fibrosis. Lyle et al.<sup>3</sup> reported similar macroscopic or histological cardiac lesions in 5% and 25% of sudden death horses, respectively. Cardiac fibrosis has previously been linked to exercising arrhythmias and sudden cardiac death in horses<sup>50,51</sup> and may occur as part of the exercise-induced hypertrophic process in human athletes. However, similar cardiac findings are commonly described in horses not experiencing sudden cardiac death<sup>17,36</sup> making interpretation of their significance challenging.<sup>3,18,36,48</sup> Additionally, the lack of a consistent *postmortem* sampling protocol of cardiac tissue makes the interpretation of microscopic findings even more challenging.

Major vessel rupture was reported to be the direct cause of death in 10/40 SAD cases (25%) of which 3 horses died as a result of cardiac tamponade or dissecting myocardial hemorrhage respectively rather than acute hemorrhagic shock after aortic wall ruptures. Although major vessel rupture as a cause of SAD varies, the current rate was higher than several of these<sup>1,5,9,48</sup> although still slightly lower than the reported rate by Lyle et al.<sup>3</sup> of 27% (39/143). There were no clear reasons as to explain the cause of the vessel ruptures.

There are several limitations in this study. The current study only covered a relatively short period (6 years), and in this period the IR for both RAD and SAD varied greatly. Given the relatively low number of fatalities, minor variations in the numbers made a substantial difference to the IR. A more extended period of study with more horses included is required in order to confirm these rates. Similarly, given the low number of cases, it is difficult to draw firm conclusions on the identified risk factors and possible causes.

In conclusion, this paper reports on the IRs of racing-associated fatalities in Norwegian and Swedish harness racehorses during a 6-year period. A relatively low IR of catastrophic orthopedic injury was reported while confirmed or suspected cardiopulmonary causes were similar to previous reports. Determining the underlying pathophysiological causes of these latter cases is still a major challenge, which needs further investigation.

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obtained from the official web pages of the respective racing associations (<https://www.travsport.no>; <https://www.travsport.se>).

#### CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

#### OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

#### INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Although the cases in this study were handled anonymously and hence should prevent identification, owners of all animals were contacted for their approval to participate in the study. This included access to the postmortem reports.

#### HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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### SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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