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# Estimation of the basic reproduction number for *Streptococcus equi* spp. *equi* outbreaks by meta-analysis of strangles outbreak reports

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## Abstract

**Background:** *Streptococcus equi* spp. *equi* (*S. equi*), the cause of strangles in horses, is considered a highly contagious pathogen affecting equines and the equine industry worldwide. Fundamental epidemiological characteristics of outbreaks, such as the basic reproduction number ($R_0$), are not well described.

**Objectives:** Estimate $R_0$ for *S. equi* in equine populations from outbreak data.

**Study design:** Systematic review and meta-analysis of published and unpublished data.

**Methods:** A literature search for outbreak reports was carried out. Depending on data available in the reports, the early epidemic growth rate or final attack rate (AR) approach was used to estimate the basic reproduction number for that outbreak. Other recorded outbreak characteristics were the type of housing (group vs. individual). An overall estimate for $R_0$ was computed by meta-analysis.

**Results:** Data from eight outbreaks were extracted from peer-reviewed publications. Data from two additional, non-published outbreaks was also included in the meta-analysis. A conservative estimate for $R_0$ was 2.2 (95% confidence interval [CI] 1.9–2.5). A less conservative estimate, including outbreaks with a 100% AR for which a lower limit $R_0$ was estimated, was 2.7 (95% CI 2.1–3.3).

**Main limitations:** Few papers describing longitudinal incidence data were found so most estimates were based on the outbreaks' final size. Several outbreaks had a 100% attack rate and could therefore only be included as a lower limit estimate in the meta-analysis. The reported result therefore may be an underestimation.

**Conclusions:** This estimate for $R_0$ for *S. equi* informs parameters for future mathematical modelling, quantifies desired preventive vaccine coverage and helps evaluate the effect of prevention strategies through future modelling studies.

## 1 | INTRODUCTION

Strangles is a disease in equids caused by infection with *Streptococcus equi* spp. *equi* (*S. equi*) which is endemic nearly worldwide.[1] *S. equi*'s impact on the global equine industry is severe enough to warrant a Consensus Statement by the American College for Veterinary Internal Medicine[2] and incorporation in the International Codes of Practice on infectious diseases of the Horserace Betting Levy Board in the United Kingdom.[3] It is a notifiable disease in several countries, including the United States.

Despite recent advances in understanding the epidemiology of *S. equi* and widespread knowledge on possible measures for the prevention of introduction and transmission, no trend towards decreasing incidence of illness due to *S. equi* has been reported over the past decade.[2]

Mathematical models of *S. equi* epidemiology may help identify key interventions which are practical and effective for reducing the impact of *S. equi*. Control of epidemics caused by *S. equi* transmission is complicated by the presence of silent, post-clinical 'carriers' which can shed infectious material from their guttural pouches or paranasal sinuses without showing clinical signs.[2,4]

*S. equi* is usually considered to be a highly contagious pathogen[2]; however, no estimate of the basic reproduction number ($R_0$) for acute strangles was found after a review of the available literature. The basic reproduction number is defined as the average number of new infections of a pathogen caused by the introduction of one infectious individual into a completely susceptible population.[5]

Estimates of the value of $R_0$ are useful as they can provide information on key parameters of mathematical models[5] that are difficult to measure in the field, such as transmission rates. Such models can be used to study how interventions influence transmission dynamics, such as a change in husbandry practices or vaccination strategy. Estimates of $R_0$ can be used to provide a rough estimate of minimal vaccine coverage $(1 - 1/R_0)$[5] required to reach the herd immunity threshold for the prevention of outbreaks. One could also interpret the quantity in terms of the probability that an introduction of an infectious individual into a well-mixed naive population will result in a major outbreak. This probability is $1 - 1/R_0$ (for $R_0 > 1$ and when the length of the infectious period follows an exponential distribution).[5]

The aim of this study was to estimate the basic reproduction number of strangles by analysing data from naturally occurring outbreaks.

## 2 | METHODS

### 2.1 | Search strategy

The PUBMED/MEDLINE and CAB Abstracts databases were searched with the query:

(equine OR horse AND "*Streptococcus equi*") OR (equine OR horse AND strangles)

The resulting hits' titles and abstracts were screened for mentions of *S. equi* horse-to-horse transmission or an outbreak, and full-text manuscripts were retrieved where possible. As clinical signs of strangles are highly specific, no limitation of the year of publication was applied as the authors considered that descriptions of outbreaks occurring at times preceding molecular diagnostics might still carry relevant information.

Studies were considered for inclusion if they contained, in the English language and within the manuscript or abstract, a description of naturally occurring horse-to-horse transmission of *S. equi* in a herd of likely naive horses and/or ponies. Information on the final size of the epidemic (attack rate) and/or early outbreak longitudinal incidence data of unmitigated strangles outbreaks were collected. Reports were excluded if from the outbreak description it seemed likely that multiple infectious individuals were introduced to the susceptible group; reports were only considered eligible if one point source of the outbreak was detected or if the source of the outbreak was not determined but assumed to be from a single source. Reports describing experimental infection were not included. Information was recorded, where available, on the herd composition and husbandry; in particular, whether horses were housed in groups for at least a significant part of the day, or were kept in individual boxstalls. For horses kept in individual boxstalls, the assumption of random mixing within the population is likely violated; however, the information obtained by evaluating $R_0$ for this husbandry practice can be potentially useful in future studies. The type of premises and main use of the herd and this information was used to assess whether the herd was likely naive to *S. equi* at the time of the introduction of *S. equi*, and whether horses were group or individually housed, when this was not mentioned explicitly.

In addition to published outbreak reports obtained through the systematic review, unpublished data from the Animal Health Trust (HT) records on numerous outbreaks in the United Kingdom, collated prior to its closure in 2020, were checked for adherence to the inclusion criteria and outbreaks were added to the meta-analysis if the criteria were met.

### 2.2 | Data extraction and calculation of the basic reproduction number

Data extracted from the reports, when available, were: type of housing (housed in groups with unhindered mingling, such as grazing or paddock turnout, with horses in boxstalls but with 'daily turnout' classed as group housed) or housing in individual boxstalls; herd immune status prior to the outbreak (whether or not some animals in the herd were likely to have at least partial protective immunity, this

information was often assumed based on descriptions of the herd and its history); number of animals at risk; the number of animals infected by the end of the outbreak; and method for diagnosing infection with *S. equi*.

An estimate of the basic reproduction number was computed using two common estimators from early epidemic (exponential) growth rate data or based on the final attack rate (AR). The latter is the total fraction of the initial population that eventually becomes infected in the outbreak $(1 - AR)$. This is also referred to as the final size of the outbreak, $1 - s(\infty)$, the fraction of the original population that has escaped infection when the outbreak has run its course. For these estimates, there are a number of assumptions.[5] We assume that the herds are closed for the duration of the outbreak in the sense that there are no births or deaths or migration into or out of the herd in that time period. In addition, we assume that mixing inside the herd is homogeneous in the sense that a contact of the type that can potentially lead to transmission is equally likely for any pair of individuals in the herd (the herd is 'well-mixed'). We assume that immunity that arises from infection lasts at least for the duration of a typical outbreak. We assume that all individuals in the herd are equal in their susceptibility, infectivity and contact pattern. Finally, it is assumed that the outbreaks run their course without mitigation of control measures of any kind.

The estimator based on the final size/attack rate is given by

$$\widehat{R}_0 = \frac{\ln(s(0)) - \ln(s(\infty))}{s(0) - s(\infty)}, \tag{1}$$

where ln is the natural logarithm, $s(0)$ is the fraction of the herd that is susceptible at the start of the outbreak (so $s(0) = 1$ in a fully susceptible herd) and $1 - s(\infty)$ is the final size.[6]

The simplest estimator based on early outbreak exponential growth rate, denoted by $r$, is given by

$$\widehat{R}_0 = e^{rT}, \tag{2}$$

where $T$ is the generation interval of the epidemic (see Roberts and Heesterbeek[7] and Wallinga and Lipsitch[8] for this and related estimators). We assumed the mean generation interval, or the time interval between successive cases in a chain of transmission, to be 13 days (standard deviation of 5 days) as suggested by Sweeney et al.[9] For Estimator 2, a compartmental model of susceptible-infectious-recovered (and resistant) or SIR model is assumed,[6] which we believed was an appropriate assumption for the timespan of the early exponential growth period of a typical *S. equi* outbreak.

Both estimators, including 95% confidence intervals (CIs) were calculated using the $R_0$-package[10] in R[11] which incorporates both, following the methods as described by Dietz[6] and Wallinga and Lipsitch.[8] The meta-analysis was then performed using the meta-package.[12] A generic inverse variance meta-analysis was applied, and a random effects model was selected for the meta-analysis to address heterogeneity in herd composition between the included outbreaks.[13] As the CIs produced by the $R_0$-package were derived via a log

transformed variable, they were not always symmetrical around the mean (but usually very close). However, the meta-package assumes a symmetrical estimate and calculates the standard error as $(CI_{max} - CI_{min})/3.92$. As the deviations from this assumption were minimal, we did not consider this issue problematic.

Estimator 1 cannot not be applied to reports in which the AR is 100%, as the resulting $R_0 = \infty$. *S. equi* outbreaks with a 100% AR can occur, and longitudinal data are not always available for such outbreaks, meaning that a point estimate and confidence interval for these outbreaks cannot be obtained and they cannot be included in the meta-analysis. In order to circumvent under-estimation of the overall $R_0$ estimate due to the exclusion of these 100% AR outbreaks, a lower limit $R_0$ estimate was calculated by assuming one horse in the herd escaped infection. Applying this method, only a lower bound for the 95% CI could be calculated via the method described by Dietz[6] as a result of the upper bound on the 95% CI AR being >1. For a weighted inclusion in the meta-analysis, an upper bound is required, therefore a symmetrical CI was assumed $CI_{upper} = R_0 + (R_0 - CI_{lower})$ for these outbreaks.

## 2.3 | Sensitivity analyses

To evaluate the effect of an alternative assumption on the generation interval for *S. equi* infection, a 'worst case scenario' short interval of 4 ± 1 days was assumed for $R_0$ estimations of outbreaks for which longitudinal data were available. The interval of 4 days was chosen as the shortest possible interval between one infection to the next, assuming horses become febrile 3 days after a large challenge dose[14] and become infectious 1 day later. This has been observed following experimental challenge of naive ponies with a dose of $10^8$ CFU of *S. equi*. The meta-analysis was then repeated with these alternative $R_0$ estimates.

A second sensitivity analysis was performed where all outbreaks that had a 100% AR were included by applying Estimator 1 when assuming not $n$, but $n - 1$ animals were involved in the outbreak, that is, assuming that one animal escaped infection.

The meta-analysis was also repeated after excluding outbreak data from non-published sources, that is, the outbreaks marked HT.

## 2.4 | Individual housing

A meta-analysis was also performed combining reports on outbreaks which did not meet the criterium of group housing, but met the other inclusion criteria.

## 3 | RESULTS

## 3.1 | Outbreak reports retrieved

PUBMED/MEDLINE and CAB Abstracts (1910–present) databases were accessed between 26 December 2020 and

**TABLE 1** Overview of outbreaks included in the meta-analysis

| Reference | Manuscript availability | Group size | Attack rate | $R_0$ estimate | Herd description | Housing | Premises type | Country | Diagnosis of cases | Interventions |
|---|---|---|---|---|---|---|---|---|---|---|
| Tscheschlok et al.[24] | Full text | 112 | 0.83 | 2.2 | Yearlings with no history of strangles | Group | Group housing with direct contact between groups. | Germany | Clinical, culture, PCR, serology | None |
| Riihimaki et al.[23] | Full text | 41 | 1 | 3.81 $(n − 1)$ | Adult Icelandic horses | Group | Boxstalls with direct contact and turnout in small groups with indirect contact between groups until 10 months after the index case | Sweden | All animals clinical signs and culture positive | None until 10 months after index case |
| Bhardwaj et al.[22] | Full text | 43 | 0.88 | 2.43 | Working horses | Assumed group | Brick kiln | India | Clinical and culture | Penicillin for severe cases |
| Davidson et al.[21] | Full text | 30 | 0.93 | 2.9 | Naive pony weanlings | Group | Research herd | UK | Clinical and serology | None |
| Wilsher and Allen 2006[20] | Full text | 20 | 1 | 3.15 $(n − 1)$ | Out of training TB fillies 2–3 research herd | Group | Paddocks 24/7 | UK | Not mentioned, assumed clinical | None |
| Katayama et al.[19] | Abstract only | 58 | 0.43 | 1.31 | Riding school | Assumed individual | Riding school | Japan | Culture of index case, clinical | |
| Newton et al.[18] | Full text | 85 | 0.66 | 2.07 | Closed herd | Assumed group | Unknown | UK | Culture of index case, clinical | Splitting into risk groups, but final AR not used for this outbreak. |
| Dalgleish et al.[16] | Abstract only | 19 | 0.84 | 2.19 | Young ponies | Assumed group | Research herd | UK | NP swab culture of all horses; S. equi not identified in all, possible overgrowth of S. zooepidemicus? | Some animals subjected to euthanasia while still showing clinical signs |
| Zadeh et al.[17] | Full text | 16 | 1 | 2.96 $(n − 1)$ | Adults 8–16 years old | Individual (direct contact possible) | Not mentioned | Iran | Nasal swab culture of some horses | All horses with clinical signs treated with penicillin |
| Zadeh et al.[17] | Full text | 9 | 0.89 | 2.47 | Adult horses 6–18 years old | Assumed individual | Not mentioned | Iran | Nasal swab culture of some horses | All horses with clinical signs treated with penicillin |
| Piché[15] (youngster group) | Full text | 131 | 1 | 4.91 $(n − 1)$ | No strangles for 11 years; transient population, resident young stock (<4 yo) housed in separate (crowded) group; outbreak originated from mares | Group | Stud farm | Canada | Clinical | None except treatment of severely affected animals |
| HT22 2007 | N/A | 23 | 0.87 | 2.34 | | Mostly group | Charity yard | UK | | |

(Continues)

**TABLE 1** (Continued)

| Reference | Manuscript availability | Group size | Attack rate | $R_0$ estimate | Herd description | Housing | Premises type | Country | Diagnosis of cases | Interventions |
|---|---|---|---|---|---|---|---|---|---|---|
| HT27 2006 | N/A | 21 | 0.67 | 1.65 | Mostly adult horses. Yard had seen an outbreak ~15 years prior | Individual | Livery yard | UK | Clinical, serology, NP swab culture and/or PCR on all cases | |
| HT38 2006 | N/A | 26 | 0.62 | 1.55 | TB mares and young stock | Mostly group | Stud farm | UK | Clinical, serology, NP swab culture and/or PCR on all cases | |
| HT52 2007 | N/A | 8 | 0.63 | 1.57 | Adult mixed | Individual | Livery yard | UK | Clinical, serology, NP swab culture and/or PCR on all cases | |

*Note:* Outbreaks in individually housed herds were analysed separately.

30 December 2020. The queries resulted in 562 and 1574 hits, respectively. Eight of these titles described horse-to-horse transmission or an outbreak meeting the inclusion criteria and contained sufficient information for an $R_0$ estimate in their full-text (where available) or abstract,[15–24] of which[18] contained information on two outbreaks and only one of the outbreaks met the inclusion criteria. Two additional outbreak reports[17,19] were retrieved which met the inclusion criteria except for the assumption of random mixing. The CAB Abstracts search returned one report[17] which was not found by the PUBMED/MEDLINE search. The reverse did not occur. Attack rates were available for four additional outbreaks from HT records, two of which did not meet the criterium of random mixing (HT27 and HT52); three of the HT outbreaks (HT22, HT27 and HT52) were featured in Mitchell et al.,[1] as United Kingdom outbreak 44, 15 and 43. Two of the included outbreak reports contained suitable longitudinal incidence data for an $R_0$ estimate based on the outbreak exponential growth rate.[18,24] All other outbreak $R_0$ estimates were based on the reported AR.[6] A summary of herd, husbandry and numerical data for each included outbreak is given in Table 1.

## 3.2 | Basic reproduction number

A forest plot of per-outbreak $R_0$ estimates and 95% CI and overall $R_0$ is presented in Figure 1. The overall estimate was $R_0$ = 2.1 (95% CI 1.8–2.4).

## 3.3 | Sensitivity analyses

Adding the 100% AR outbreak reports, as described in Section 2, resulted in an overall estimate of $R_0$ = 2.7 (95% CI 2.1–3.3).

Removing the data from the two outbreaks that were obtained from non-published sources, but including the 100% AR outbreaks, resulted in an overall estimate of $R_0$ = 2.9 (95% CI 2.3–3.5).

An overview of the $R_0$ estimates produced by the different generation interval assumptions is given in Table 2. The resulting overall $R_0$ estimate in the meta-analysis, when including the lower per-outbreak estimates which resulted from applying the shorter generation interval to the two outbreaks for which an $R_0$ was obtained by Estimator 2, and including the 100% AR outbreaks was $R_0$ = 2.5 (95% CI 1.8–3.2).

## 3.4 | Individual housing

The overall estimate for the five outbreaks that occurred in herds which were housed in individual boxstalls was $R_0$ = 2.0 (95% CI 1.3–2.6). The corresponding forest plot is available in Figure S1.

**FIGURE 1** Overall estimate by meta-analysis of the basic reproduction number ($R_0$). An overall estimate, when outbreaks with 100% attack rate (AR) are included by assuming $n - 1$ animals became infected (as described in the text), is provided (lower diamond) as well as a more conservative estimate, where the outbreaks with a 100% AR (which required a workaround to produce a lower limit estimate), were not included (upper diamond).
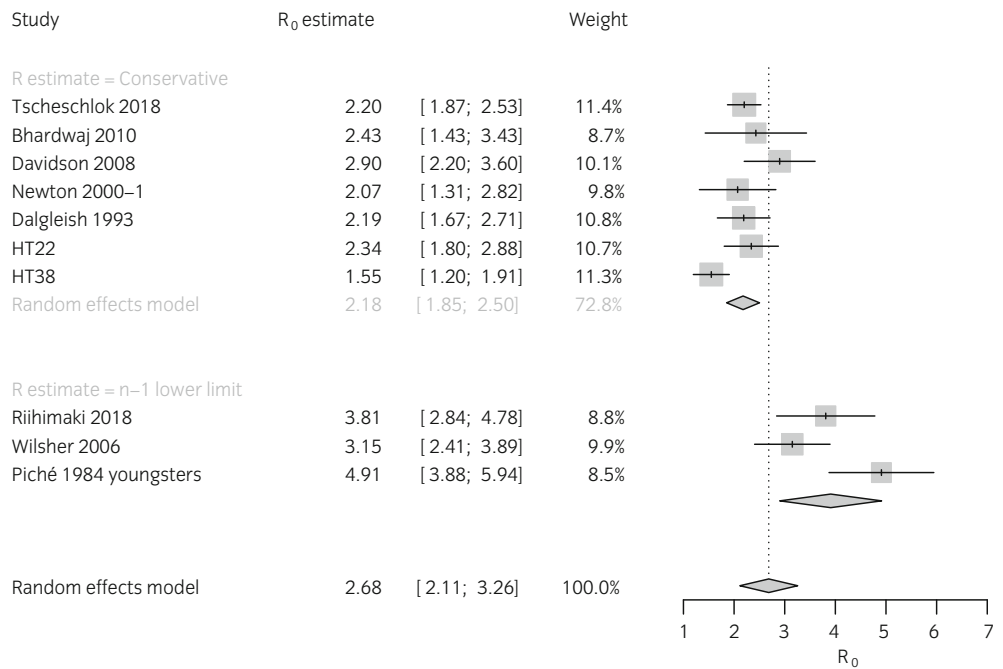


| Study | $R_0$ estimate | | Weight |
|---|---|---|---|
| R estimate = Conservative | | | |
| Tscheschlok 2018 | 2.20 | [1.87; 2.53] | 11.4% |
| Bhardwaj 2010 | 2.43 | [1.43; 3.43] | 8.7% |
| Davidson 2008 | 2.90 | [2.20; 3.60] | 10.1% |
| Newton 2000–1 | 2.07 | [1.31; 2.82] | 9.8% |
| Dalgleish 1993 | 2.19 | [1.67; 2.71] | 10.8% |
| HT22 | 2.34 | [1.80; 2.88] | 10.7% |
| HT38 | 1.55 | [1.20; 1.91] | 11.3% |
| Random effects model | 2.18 | [1.85; 2.50] | 72.8% |
| | | | |
| R estimate = n–1 lower limit | | | |
| Riihimaki 2018 | 3.81 | [2.84; 4.78] | 8.8% |
| Wilsher 2006 | 3.15 | [2.41; 3.89] | 9.9% |
| Piché 1984 youngsters | 4.91 | [3.88; 5.94] | 8.5% |
| | | | |
| Random effects model | 2.68 | [2.11; 3.26] | 100.0% |

**TABLE 2** A comparison of per-outbreak $R_0$ estimates based on epidemic growth rate, when assuming a realistic or a very short generation interval (see text) between successive cases

| | | 13 ± 5 days | 4 ± 1 days |
|---|---|---|---|
| | Generation interval | $R_0$ estimate | |
| Outbreak reference | [24] | 2.20 [1.90–2.56] | 1.32 [1.25–1.39] |
| | [18] | 2.07 [1.47–2.98] | 1.29 [1.14–1.47] |

## 4 | DISCUSSION

In this study, we provide the first estimate of the value of the basic reproduction number $R_0$ for *S. equi*, the causal agent of strangles, based on a range of published descriptions of outbreaks. The overall estimate for $R_0$ of 2.2 (or, less conservatively, 2.7) found in this study does not support the assumption that *S. equi* is a highly contagious pathogen. Notorious, highly contagious human diseases (such as rubella: $R_0 = 6$–7, measles: $R_0 = 12$–18, or pertussis: $R_0 = 12$–17)[25] have substantially higher basic reproduction numbers. Given that the infectious period for strangles is not particularly short, usually assumed to be 14–21 days,[2] the number of daily contacts sufficient for disease transmission of disease per day per infectious horse likely is small.

Few basic reproduction numbers for equine infectious diseases are available for comparison. Estimates for equine influenza have been computed by outbreak analysis and/or modelling and have resulted in estimates of 2–5 to 10.[26,27] The higher $R_0$ estimates for equine influenza, compared to *S. equi*, can probably to some extent be explained by the fact that equine influenza, unlike *S. equi*, can be transmitted via aerosols,[28] and is known to travel further distances and more easily spreads between premises without horse or fomite movement.

Some variation of estimates of $R_0$ for each of the individual outbreaks was found in the present study. Contributing to this variation, besides factors such as herd composition and husbandry, might be the previously observed reduction of virulence of the organism after prolonged persistence in a guttural pouch.[29–31] This was the proposed cause for the low morbidity in the outbreak described by Tscheschlok et al.,[24] but may also have been a factor in other outbreaks that reported AR at the lower end of the spectrum. It is also important to note that in smaller herds, the effects of chance play a more important role in determining the final size of the outbreak. The size of the major outbreak from simple stochastic models has been shown to have a normal distribution around the mean $N(1 - 1/R_0)$,[5] which can explain the chance occurrence of a relatively small (or indeed a relatively large) AR, given the value of $R_0$.

The overall estimate for $R_0$ found in this study was relatively low compared to other notorious veterinary and human diseases. This should be considered a favourable finding as a comparatively low $R_0$ suggests that measures such as enhanced biosecurity, pre-entry diagnostic screening and other prevention strategies could effectively minimise the probability of an outbreak occurring. For example, if vaccination of a herd is considered, the herd immunity threshold $(1 - 1/R_0)$ based on an $R_0$ of 2.7 is 62% which, considering the efficacy reports of some strangles vaccines[32,33] could be an achievable goal. Vaccines would only need to be >68% effective to be able to attain the herd immunity threshold, and for vaccines with substantially higher efficacies, vaccination coverage may not need to be 100%.

Care should be taken, however, to consider other, unexpected and/or unwanted effects of various levels of vaccine coverage, and cost/benefit ratios should be established to determine the optimum vaccination strategy, which may vary depending on age distribution and husbandry practices within the herd. An important example of a potential adverse effect is a shift in the age groups which carry the majority of the burden of the disease,[34,35] which might lead to strangles cases increasingly occurring at ages where the horse's economic potential is more seriously affected than it would have been had they been infected as a youngster. Whether or not, and under which circumstances, this or other unwanted effects are likely to occur should be the subject of future modelling studies.

It is worth pointing out that the estimates for $R_0$ reported in this paper are dependent on the assumptions underlying the estimators being met to a reasonable degree,[36] and deviations from these assumptions affect the accuracy of the $R_0$ estimate. In addition to this, the accuracy of the $R_0$ estimates is dependent on the accuracy of the data extracted from each of the reports; reports where the diagnosis was based on clinical signs only may underestimate the $R_0$ for that particular outbreak as silent infections may potentially have been missed. Silent infections with *S. equi* in naive horses are not common, but have been described.[24] The overall estimate for $R_0$ provided in this current work is intended to serve as a rough initial estimate, with the understanding that it may be an underestimation to some extent, but an estimate nevertheless which we hope will be of use in future studies evaluating possible interventions against transmission of *S. equi*. Alternative methods to calculate $R_0$ exist, in particular through mathematical models where all relevant parameters of disease transmission are assigned a certain value, transmission is described by equations and $R_0$ is calculated by solving these equations.[5,36] However, although much is currently known with some certainty about the natural history of *S. equi* infection, such as duration of the incubation period and average duration of infectiousness, key parameters such as most importantly the effective contact rate, were still absent. One way to find values for these missing parameters would be through infection experiments, where transmission can be followed in real-time and which would give the most accurate information. Hamlen et al.[37] in 1994 conducted an infection experiment in foals, with the aim of evaluating the effect of prior exposure to strangles illness upon re-exposure; however, in their setup, foals with prior exposure and naive foals co-mingled; also, no longitudinal data were available from this report. Setting up a suitable infection experiment, although a very reliable way to find $R_0$, will come at a significant financial and animal welfare cost. The current project was devised to find an estimate for $R_0$ from already available data.

Sparse data were available to evaluate the effect of housing on $R_0$ so it is not possible to draw a meaningful conclusion on the effect of this husbandry practice on $R_0$, other than that $R_0$ is likely lower in purely individually housed animals, which would be a plausible effect of a reduction of the opportunities for effective contact for transmission both direct (social behaviours, e.g., nosing) and indirect (e.g., shared water sources) contact.

The exclusion of non-published outbreaks had only a minor effect (increase from 2.1 to 2.3) on the overall estimate for $R_0$. Inclusion of the lower limit $R_0$ estimate for the 100% AR outbreaks did have a notable effect (increase from the conservative estimate of 2.2 to 2.7).

It is not immediately evident why these particular outbreaks had such a high AR; they did all occur in group-housed, juvenile or naive[23] horses, but so did other outbreaks with lower attack rates.[21,24] Herd sizes of the 100% AR outbreaks were mostly smaller (20–41 animals)[21,23] and as such were more susceptible to outcomes occurring by the effects of chance, as described earlier.

Using an alternative, very short estimate for the generation interval had a substantial impact on the $R_0$ estimates for those particular outbreaks (as summarised in Table 2) which, as could be expected, were lower than when a longer generation interval was assumed.[8] Due to the relatively few outbreaks for which the $R_0$ estimation was based on the growth rate, the effect on the overall estimate for $R_0$ was limited (decrease from 2.7 to 2.5). In the authors' clinical experience, the estimate of $13 \pm 5$ days[9] is probably closer to the real-life transmission dynamics than the assumption of a worst case scenario generation interval, where animals are assumed to become infectious in the shortest possible time and to always infect another horse within 1–2 days of becoming infectious. Going forward, the authors do not recommend using such a short generation interval for modelling the dynamics of *S. equi* transmission.

In the future, the estimate for $R_0$ calculated in this study should be corroborated by analysis of further unpublished outbreak data from sources such as diagnostic laboratories or national disease collating centres. To mitigate the drawbacks of analysing outbreaks with 100% AR where Estimator 1 cannot be applied, more longitudinal early outbreak incidence or peak incidence data of extensive outbreaks are required. Additional data will also be needed to better evaluate the effect of housing type on the reproduction number.

Many papers included in the study did not mention explicitly the immune status of the herd involved and therefore the decision to include outbreaks for the $R_0$ meta-analysis was open to classification error. In including papers, a decision was made to err on the side of caution as the accuracy of the $R_0$ estimate was prioritised. The first outbreak in Newton et al.[18] was included due to the description of the affected horses being in a 'closed herd'; the outbreak described by Piché.[15] was described to have occurred on a stud farm with a long strangles-free history, therefore the youngsters on the premises were considered to likely be naive. Whether assuming a fully naive population in these outbreaks was correct remains unknown. If the assumption was incorrect, the inclusion of these outbreaks could contribute to the underestimation of the overall $R_0$ estimate.

As the focus of the current paper was the reproduction number of outbreaks of strangles, the question of the epidemiological contribution of post-clinical persistent carriers of *S. equi* was not addressed as these carriers mainly become epidemiologically significant in instigating new outbreaks at time-spans exceeding those of the initial outbreaks analysed in this study.

## 5 | CONCLUSION

The overall estimate for $R_0$ produced by meta-analysis of outbreak reports was lower than anticipated for *S. equi*, which suggests that even small improvements in biosecurity, screening and disease prevention could have important benefits. The precision and accuracy of

the $R_0$ estimate found in this study may be improved through the analysis of more longitudinal outbreak data, and more outbreaks where housing type and herd immunity status are clearly stated. Nonetheless, the $R_0$ estimate produced in this study can be used to parameterise epidemiological models studying the possible effects of preventive or mitigating interventions, such as changes in husbandry practices, hygiene protocols and vaccination.

## CONFLICT OF INTEREST

Andrew S. Waller is Chief Scientific Officer for Intervacc AB. The remaining authors declare no conflict of interest.

## PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/evj.13865.

## DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed in this study.

## ETHICAL ANIMAL RESEARCH

Not applicable.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.