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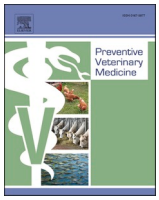

Small mammals as carriers of zoonotic bacteria on pig and cattle farms – Prevalence and risk of exposure in an integrative approach

Maria Simola [a], Saija Hallanvuo [a], Heikki Henttonen [b], Otso Huitu [b], Jukka Niemimaa [b], Heidi Rossow [a], Leena Seppä-Lassila [a], Jukka Ranta [a,*]

[a] Finnish Food Authority, Finland
[b] Natural Resources Institute Finland, Finland

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ABSTRACT

To prevent foodborne infections from pigs and cattle, the whole food chain must act to minimize the contamination of products, including biosecurity measures which prevent infections via feed and the environment in production farms. Rodents and other small mammals can be reservoirs of and key vectors for transmitting zoonotic bacteria and viruses to farm animals, through direct contact but more often through environmental contamination. In line with One Health concept, we integrated results from a sampling study of small mammals in farm environments and data from a capture-recapture experiment into a probabilistic model which quantifies the degree of environmental exposure of zoonotic bacteria by small mammals to farm premises. We investigated more than 1200 small mammals trapped in and around 38 swine and cattle farm premises in Finland in 2017/2018. Regardless of the farm type, the most common species caught were the yellow-necked mouse (*Apodemus flavicollis*), bank vole (*Clethrionomys glareolus*), and house mouse (*Mus musculus*). Of 554 intestine samples (each pooled from 1 to 10 individuals), 33% were positive for *Campylobacter jejuni*. *Yersinia enterocolitica* was detected in 8% of the pooled samples, on 21/38 farm premises. Findings of *Salmonella* and the Shiga-toxin producing *Escherichia coli* (STEC) were rare: the pathogens were detected in only single samples from four and six farm premises, respectively. The prevalence of *Campylobacter*, *Salmonella*, *Yersinia* and STEC in small mammal populations was estimated as 26%/13%, 1%/0%, 2%/3%, 1%/1%, respectively, in 2017/2018. The exposure probability within the experimental period of four weeks on farms was 17–60% for *Campylobacter* and 0–3% for *Salmonella*. The quantitative model is readily applicable to similar integrative studies. Our results indicate that small mammals increase the risk of exposure to zoonotic bacteria in animal production farms, thus increasing risks also for livestock and human health.

1. Introduction

Zoonotic bacteria form a considerable part of the disease burden caused by infectious diseases worldwide. Transmission of zoonotic bacteria can occur through direct contact between animals and their environment, via ingested water and food, and products (EFSA and ECDC, 2019). The most common zoonotic bacteria infecting humans are *Campylobacteria*, *Salmonella*, Shiga toxin-producing *Escherichia coli* (STEC) and *Yersinia* (EFSA and ECDC, 2019; Gould et al., 2013). In Finland, these pathogenic bacteria cause altogether thousands of infections per year in a population of 5.5 million people (THL infectious diseases register, 2023).

On farm premises, of special concern are animals carrying potential zoonotic bacteria having access to feed storages and animal housings. In particular, rodents are generally considered key species which greatly increase the risk of zoonotic bacteria exposure and transmission to production animals and eventually the food chain (Meerburg et al., 2009; Jahan et al., 2021). Several factors influence the level of this risk. Firstly, rodents are ubiquitous in farm environments, as they are attracted to the warmth, dryness, and protection from predation that these environments provide (Miño et al., 2007) Secondly, animal farms provide rodents with ample food resources, functionally akin to large scale supplementary feeding stations (Becker et al., 2018). While foraging in feed storage facilities, rodents excrete zoonotic bacteria in their faeces and urine, thus contaminating the feed of production animals, and facilitating the horizontal transmission of bacteria between

* Corresponding author.
E-mail address: jukka.ranta@foodauthority.fi (J. Ranta).

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rodents (Andrés-Barranco et al., 2014; Backhans et al., 2013; Healing and Greenwood, 1991; Meerburg et al., 2006).

Rodents may have a major amplification role of zoonotic bacteria in farm environments. For example, 15 *Salmonella* bacteria are required to establish infection in a rodent. After infection, it is estimated that ca. 230,000 *Salmonella* bacteria are shed in a single mouse faecal pellet, and such pellets are excreted at a rate of one hundred per day. Thereby, a minimum of 23 million *Salmonella* can be introduced into a farm environment by a single rodent pest every day (Trampel et al., 2014; Jahan et al., 2021).

Zoonotic pathogens can be part of the normal microbe flora of the host animals and thus asymptomatic, although systemic illness in the form of septicemia can sometimes occur, for example, in infections of *Salmonella* (Hatch, 1996; Hoelzer et al., 2011). Zoonoses can thus be chronically harbored by, and transmitted between, small mammals in farm environments even when livestock are not infected, thus greatly hindering permanent eradication of the zoonoses in these environments (Vaughan et al., 2016).

The study aims at estimating the probability of pathogen exposure to occur from small mammals to domestic animal population in farms. In other words, the probability that at least one small mammal carries pathogens and invades farm premises. We make no assumption on the pathogen prevalence in the domestic animals on the farms, which may be zero or non-zero. We do not estimate transmission of actual infections, nor prove the first origin of the pathogens. For estimating dynamic transmissions that can occur both ways, detailed surveillance sampling would be needed simultaneously from both domestic animals and small mammals over longer times, taking into consideration e.g. the effects of seasonality. The exposure estimation was based on combined mathematical models estimating (1) the prevalence of pathogens in small mammals around animal farms, and (2) the abundance of small mammals around farms and their movement into farm buildings using a capture-mark-recapture study. Hence, two studies are integrated into one exposure estimate.

2. Materials and methods

2.1. Sampling procedures

In 2017 and 2018, sampling was carried out on the premises of 20 pig and 18 cattle farms (i.e., prevalence study farms) in Western Finland, evenly distributed in an area of circa 40,000 km^2 of the most intensive agricultural area in Finland. The 38 farms were recruited with the help of two major slaughterhouses in Finland. A prerequisite for the farms to be part of the study was that animals were reared for slaughter and sent to slaughterhouses directly from the farm, creating a potential transmission pathway from small mammals via the production animals to meat products. The cattle farms were recruited by the slaughterhouse by contacting farms in random order until the minimum number of farms logistically feasible (approx. 20 farms) was attained. The pig farms were recruited by open invitation delivered by the slaughterhouse and included in the study in the order of their acceptance. Farm participation was voluntary, and one farm decided to withdraw from the study after the first year in 2018. The median number of animals per farm was 1630 pigs and 270 bovines per farm, respectively. To assess differences in the prevalence of zoonotic microbes in farm-inhabiting small mammals and those inhabiting natural habitats not influenced by human activities, sampling was carried out in 2018 in 12 forested sites located in the same regions as the farms, but each with a minimum distance of 5 kilometers to the nearest human settlement.

Sampling consisted of trapping small mammals in autumn, between October and November. Snap traps as well as Ugglan live traps (a total of 115 traps) were used during two consecutive nights per farm, such that 40 small mouse snap traps were placed in natural forest and field habitats 20–200 m from any farm buildings; 20 mouse snap traps along the immediate outer perimeter of farm buildings; and 40 snap traps, 10 large rat snap traps and 5 Ugglan live traps inside farm buildings (excluding feed storage and animal production facilities). In the more distant natural habitat sites, 100 mouse snap traps were set for two days. In the morning, the small mammals were collected and those caught in live traps euthanized by cervical dislocation.

The intestines, including stomach, of all caught animals, were collected as samples, frozen using dry ice, and transferred frozen to the laboratory. Due to logistic reasons, approximately 13% of the animals had to be frozen as whole animals. Samples from these animals were later harvested immediately upon thawing and transported directly to the analyzing laboratory in which analyses were started immediately. The intestines and stomachs (with content) of caught small mammals were cut to an even mass with scalpels on petri discs. Then, the samples were pooled. One pooled sample was formed of the intestines and stomachs of the animals belonging to the same species, caught from the same farm, either next to the farm buildings or far from the buildings. One pooled sample consisted of 1–10 individual samples, i.e., animals. Next, the pooled samples were weighed and the same mass of buffered pepton water (BPW 1%) was added to the sample by pipetting. The initial suspension was then mixed well with the tip of the pipette. In Fig. 1, the protocol for the microbiological analyses is shown.

2.2. Campylobacter analysis

For *Campylobacter* analysis, the standards ISO-10272–1 (2017) and ISO-6887/6 (2013) were applied with modifications. The cultivation of each pooled sample was done from the initial suspension with 10 µl loop on a mCCD-agar plate (modified charcoal cefoperazone deoxycholate) (OXOID Deutschland GmbH, Wessel, Germany). The plates were then incubated in a micro-aerophilic atmosphere (10% CO_2, 5% O_2, 85% N_2) at +41.5 ± 1.0 °C for 48–72 h. After incubation, 3 typical colonies from the mCCD plates were cultivated on a blood agar (bovine blood) and incubated in a micro-aerophilic atmosphere at +41.5 ± 1.0 °C for 24–72 h. The isolates were confirmed as *Campylobacter* at species level with Maldi Biotyper® (Bruker Daltonics GmbH, Saksa) following the manufacturer's instructions. In case of uncertain results, the isolates were further confirmed by microscopy. The isolates were also analyzed using whole genome sequencing technique, of which the results are published elsewhere (Olkkola et al., 2021). Finally, the isolates were stored in a BHI broth at −80 °C.

2.3. Salmonella analysis

The VIDAS SLM assay (AOAC International 020901; bioMérieux, Inc., Marcy l'Etoile, France) was used for screening all samples for the presence of *Salmonella*, with a modification. For pre-enrichment, BPW was added to the initial suspension to achieve a 1:10 mixing of the sample and PBW. The samples were then incubated at 37 °C for 20±4 h. For enrichment, 0.1 ml of the pre-enriched sample was added to a 10 ml pre-warmed RVS broth and 1.0 ml to a 10 ml pre-warmed MKTTn broth. Both enrichment broths were then incubated at 41.5 °C for 18–24 h. After the pre-enrichment and enrichment phases, the VIDAS SLM protocol was followed. *Salmonella* strains from Vidas positive samples were isolated following the ISO 6579 method replacing TSI and Urea tubes with Maldi-TOF confirmation. The isolates which were confirmed presumptive *Salmonella* using the ISO 6579 were further serotyped in the Finnish Food Authority in Kuopio.

2.4. STEC analysis

STEC analysis started from the same pre-enriched sample, from which *Salmonella* enrichment was started. The analysis followed the International Organization for Standardization technical specification (ISO/TS) 13136:2012. The protocol included a pre-screening phase by real-time PCR and a cultivation of the presumptively positive pre-enrichment broths.

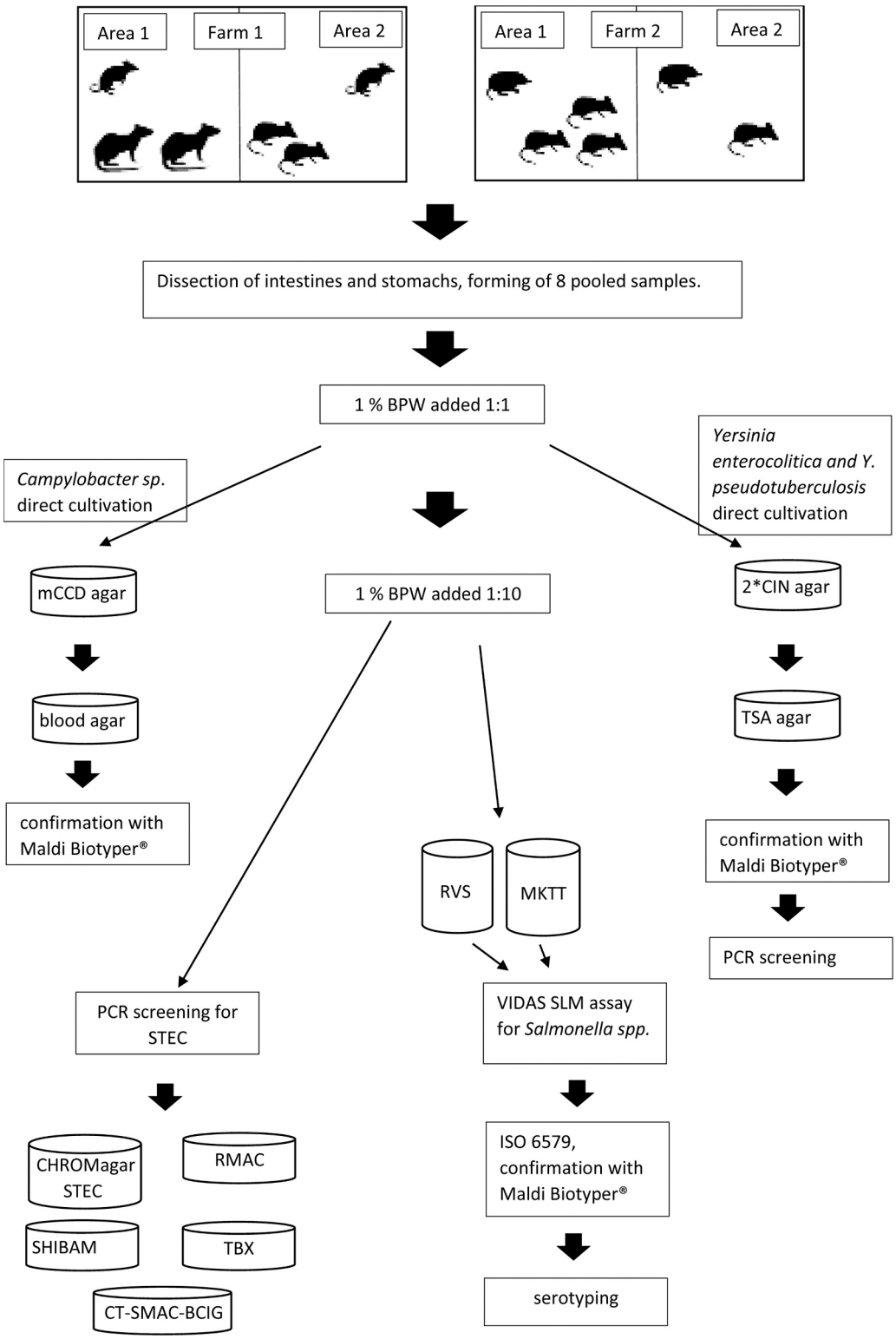

Fig. 1. Laboratory protocol.

DNA was extracted from 1 ml (MasterPure kit; Epicentre, Madison, WI, USA) or 100 µl (iQ-Check STEC kit; Bio-Rad, Marnes-la-Coquette, France) of an enriched sample broth and screened for the virulence genes stx and eae using real-time PCR (TaqMan ISO assay [Life Technologies/Thermo Fisher Scientific, Carlsbad, CA, USA] or iQ-Check STEC kit). PCR inhibition was monitored with an internal amplification control. The samples that had tested positive for stx and eae were further analyzed for serogroups O157:H7, O26, O103, O145, O111, O121, and O45 using real-time PCR (iQ-Check STEC kit).

The samples that had tested positive for stx -genes were further analyzed by plating onto selective agars: cefixime-tellurite sorbitol MacConkey agar with 5-bromo-4-chloro-3-indoxyl-β-d-glucuronide SMAC-BCIG (Harlequin; Lab M, Lancashire, UK) CHROMagar STEC (CHROMagar, Paris, France), Rhamnose-MacConcey agar with cefixime-tellurite (RMAC, Difco™ BD BioSciences, Le Pont de Claix, France), SHIBAM agar (Hardy Diagnostics, USA), and Tryptone Bile Glucuronide

agar (Harlequin™ TBGA/TBX, Lab M, Lancashire, UK).

2.5. Yersinia enterocolitica and Yersinia pseudotuberculosis analysis

The cultivation of *Y. enterocolitica* and *Y. pseudotuberculosis* for each pooled samples was performed from the initial suspension by direct plating with 10 µl loop on two CIN (Cefsulodin irgasan novobiocin agar, Oxoid, United Kingdom) plates. Typical colonies were cultured on TSA (Tryptone Soya Agar, Merck KGaA, Darmstadt, Germany) plates after 24 h and 48 h. The Isolates were confirmed with Maldi-Tof biotyper. The presence of *ail*-gene in *Y. enterocolitica* strains was confirmed by real-time PCR following ISO/TS 18867:2015; Annex B, method 2. *Y. enterocolitica* isolates were biotyped by following ISO 10273:2017.

2.6. Capture-mark-recapture study

The estimation of the exposure risk of livestock to small mammal-borne zoonotic pathogens was based on a capture-mark-recapture study on three additional farms (i.e., exposure study farms; not part of prevalence study) located in Southern Finland (Jokioinen, Forssa and Luumäki). In the present study, conducted on the farms in autumn 2019, small mammals were live trapped and tagged in their natural surroundings for seven days after which trapping was commenced inside farm buildings (not inside animal rearing or feed facilities, but in adjacent equipment storage facilities) to estimate the rates of movement from outside to inside. Live trapping was performed using Ugglan multiple capture live traps baited with oats and potato. Cotton wool was placed in the traps as bedding material.

Sixty live traps were placed randomly and evenly approx. 20–100 m away from a production animal building in the forested natural habitats of small mammals. Traps were set in the afternoon and checked and re-baited daily in the morning or midday. Each caught individual small mammal was identified to species and sex, weighed and individually marked with a subcutaneous PIT-tag (Trovan ID-100A, Trovan Ltd) before release at the point of capture. Following the first trapping round, each caught individual was scanned with a PIT-tag reader (LID-560, Trovan Ltd). Each previously tagged individual was recorded, along with the trap ID, and released immediately. Live trapping was carried out for seven days.

Immediately after the live trapping period, a snap trapping protocol was launched inside the farm building. Here, 20–40 metal snap traps were placed along potential small mammal runways and other sites, baited with cheese and thereafter checked and re-baited and re-set when necessary for four weeks. During this time, each trapped individual was collected and stored at −20℃ for further examination of PIT tag presence in the laboratory.

3. Statistical models

3.1. Estimation of small mammal pathogen prevalence on prevalence study farms

From the pooled samples, the prevalence of *Campylobacter*, *Salmonella*, *Yersinia* and STEC in small mammals from pig and cattle farms was estimated. The number of individual samples within the i=1,…,554 pools varied in the range 1–10 on k=1,…,38 farms. The probability (Eq. (1)) for the ith positive pooled sample X_i with n_i individual samples in the pool from farm z_i year t_i was thus

Eq. 1

$$P\left(X_i = 1 | p_{z_i,t_i}, n_i \right) = 1 - \left(1 - p_{z_i,t_i}\right)^{n_i}$$

where the farm specific prevalence p has a variation (Eq. (2)) over the 38 farms, described as for each year t (t=2017,2018) as

Eq. 2

$$Normal\left(logit\left(p_{k,t}\right) | logit\left(p_{0,t}\right), \sigma_t^2\right)$$

From this hierarchical model, the posterior distribution (Eq. (3)) of all unknown parameters (for each of the four pathogens separately) was derived from Bayes theorem as

Eq. 3

$$P(\ \{p_{1,t}, ..., p_{38,t}, p_{0,t}, \tau_t\ \} \mid X_1, ..., X_{554}\ ) \propto$$

$$\prod_{i=1}^{554} Bernoulli(X_i | 1 - \left(1 - p_{z_i,t_i}\right)^{n_i}\ )$$

$$\times \prod_{k=1}^{38} \prod_{t} Normal(logit\left(p_{k,t}\right) | logit\left(p_{0,t}\right), \sigma_t^2\ )\ P(p_{0,t}) P(\tau_t)$$

where the prior distributions were as $P(\tau_t)$=Gamma(0.1,0.1) for $\tau_t=\sigma_t^{-2}$ and $P(p_{0,t})$=Uniform(0,1), for both years. A model for each year is useful if there is a difference in the overall prevalence between years. If not, the model could be simplified by removing year-specific parameterization. This model was implemented in OpenBUGS and R using R2OpenBUGS package (Lunn et al., 2012). As a result, estimates for farm specific prevalences $p_{k,t}$ and overall prevalence $p_{0,t}$ were obtained for each pathogen, and 95% Bayesian credible Intervals (95% CI) calculated. The results were derived from 20,000 MCMC iterations after a burn-in of 8000 iterations and a visual check for convergence.

3.2. Estimation of small mammal population size on exposure study farms

Estimation of a small mammal population size N on the exposure study farms was done using a Bayesian capture-recapture model (Oldemeyer et al., 2018; Schwarz et al., 2009). The capture-recapture data were collected from three farms A, B and C, which were different than the 38 farms used for the prevalence studies. In a first sampling on a farm, s_1 animals were captured, tagged and released. This left $U = N - s_1$ untagged animals and $m_1 = s_1$ tagged animals. In a second sampling, s_2 were captured among which m_2 were already previously tagged and u_2 previously untagged. Then, assuming that independent of tagging, each animal always has the same probability p to be captured, we have $m_2 \sim Bin(p, s_1)$ and $u_2 \sim Bin(p, U)$. Hence, after observing m_2 and s_1, we could estimate $p = m_2/s_1$. Since $E(u_2|p,U) = pU$, and replacing the expected value by the observed u_2, and p by its estimate, we could obtain $u_2 = m_2U/s_1$. By definition of $U = N - s_1$, the estimate of N follows as s_1s_2/m_2, which is the Lincoln-Petersen estimate. However, instead of such a point estimate, we derived an uncertainty distribution for the two unknown parameters p & N using Bayesian modelling. For a single recapture experiment on a farm, the posterior distribution (Eq. (4)) is, according to Bayes theorem:

Eq. 4

$$P(N,p \mid s_1, m_2, u_2) = Bin(s_1 \mid p, N)\ Bin(m_2 \mid p, s_1)\ Bin(u_2 \mid p, N - s_1)\ P(N)P(p)\ I(N >= s_1+u_2) / constant$$

where the first Binomial probability corresponds to the very first capture count s_1, and prior distributions were chosen as P(p)=Uniform(0,1) and $P(N) = Poisson(\theta)$ where the expected value θ was eventually given a hyper prior when combining the data from all three farms as a meta-analysis. The prior for N implies that farm specific population sizes are as Poisson distributed variables with a common overall mean value. In addition, the lower bound for N on each farm should equal the sum of the first capture s_1 and the subsequent capture u_2 of unmarked animals. This is imposed by the indicator function $I(N >= s_1+u_2)$ which takes value one when this condition is met and zero otherwise.

New captures and taggings of previously unseen individuals and releases were repeated daily for a total of seven days. For example, after the second sample of size s_2 was captured, previously unseen individuals were tagged before release which then makes the total count of currently tagged individuals $m_2 = s_1 + u_2$. Then, a third sample of size s_3 was

captured among which m_3 were previously tagged and u_3 previously untagged. This provides probabilities for such observations as $m_3 \sim$ Bin (p, $s_1 + u_2$) and $u_3 \sim$ Bin(p, N - s_1 - u_2). Similar sampling was continued for up to seven times. The number of traps was sufficiently large so that it did not artificially restrict the number of captures since many traps were empty each week. The binomial distributions were simplified to Poisson-distributions which provided the same results. Finally, the assumption of constant capture probability p was relaxed by allowing variability between the capture weeks on each farm as logit(p_k) $\sim$ Normal(logit(p_0),σ^2) with prior distributions $p_0 \sim$ Uniform(0,1) and $1/\sigma^2 = \tau \sim$ Gamma(0.1,0.1). This model was applied as a hierarchical model using hyper prior $\theta \sim$ Gamma(0.1,0.1) to make a synthesis of population size estimation from the data of all of the three farms A, B and C.

By combining the total capture-recapture data of all three farms f=1,2,3, and defining $u_{1,f}$ =0 in each, the posterior (Eq. (5)) is given by Bayes theorem as

Eq. 5

$$P\left(\{N_f, p_{1,f}, \ldots, p_{K,f}, p_{0,f}, \tau_f\} \mid \{s_{1,f}, m_{2,f}, \ldots, m_{K,f}, u_{2,f}, \ldots, u_{K,f}\}\right) \propto$$

$$\prod_{f=1}^{3} \left[Poisson\left(s_{1,f} \mid p_{1,f} N_f\right) \times \right.$$

$$\prod_{k=2}^{K} Poisson\left(m_{k,f} \mid p_{k,f}\left(s_{1,f} + \sum_{i=1}^{k-1} u_{i,f}\right)\right) Poisson$$

$$\left(u_{k,f} \mid p_{k,f}\left(N_f - s_{1,f} - \sum_{i=1}^{k-1} u_{i,f}\right)\right) \times$$

$$I\left(N_f \geq s_{1,f} + \sum_{i=1}^{K} u_{i,f}\right) \times$$

$$\left. \prod_{k=1}^{K} Normal(logit\left(p_{k,f}\right) \mid logit\left(p_{0,f}\right), \sigma_f^2) Poisson\left(N_f, \mid, \theta\right) Uniform \right.$$

$$\left. \left(p_{0,f}\right) Gamma\left(\tau_f\right) \right] Gamma(\theta)$$

This model was implemented in OpenBUGS and R using R2OpenBUGS package (Lunn et al., 2012). As a result, estimates for small mammal population sizes on the three farms N_f and the expected population size θ were obtained and 95% Bayesian credible Intervals (95% CI) calculated. The results were derived from 20,000 MCMC iterations after a burn-in of 8000 iterations and visual check for convergence.

3.3. Estimation of exposure to pathogens from small mammals on exposure study farms

Exposure estimation is one of the four parts in a risk assessment consisting of (1) hazard identification, (2) exposure assessment, (3) hazard characterization (incl. dose-response), and (4) risk characterization. Often, the risk is defined as the probability and severity of illness for humans. Instead, here 'risk' refers to domestic animals becoming carriers of bacteria which are human pathogens. In this study, we focus solely on exposure assessment, i.e., the estimation of probability of exposure for domestic animals. We do not estimate actual transmissions, nor consider what the original source of zoonotic bacteria at the farms is.

In a given situation with already existing pathogens in a population of small mammals around a farm, the theoretical exposure of farm animals to pathogens from small mammals depends on both the (unknown) pathogen prevalence in small mammals, and the (unknown) small mammal population size. Additionally, the proportion (q) of small mammals entering farm premises is a key factor that can be affected by biosecurity measures. An estimate for q was drawn from the capture-recapture experiment where traps were placed inside the farm. From the total number of tagged animals, a fraction was captured in these traps providing an estimate of the invading proportion. This parameter was described by the posterior distribution q $\sim$ Beta(y+1,*m*-y+1) = Beta (7185) where y is the total number of animals captured inside and m is the total number of tagged animals (outside). By hypothesizing small mammal entrances as independent events, the exposure was defined as an event where at least one small mammal enters the premises, and – without other explanatory variables and parameters – the probability of exposure (Eq. (6)) was written as

Eq. 6

$$P(Exposure) = 1 - (1 - qp_0)^{\theta}$$

Since all the parameters q (proportion of small mammals entering the premises), p_0 (pathogen prevalence in small mammals) and θ (expected population size of small mammals) were estimated by their posterior distributions described in previous sections, an uncertainty distribution for this whole expression is implied. The exposure estimate is a synthetic combination from different studies: q and θ are overall means estimated from the capture-recapture experiments (farms A, B, C) and p_0 is an overall prevalence from the prevalence studies (38 farms) for each pathogen.

4. Results

4.1. Prevalence study: animals caught

During the prevalence study in autumn 2017 and 2018, altogether 1204 small mammals were caught from the premises of pig and cattle farms (Table 1). In 2017, animals were caught from 35/38 farm premises, whereas in 2018, animals were caught on all farms. In 2017, the total catch was 406 individuals, and in 2018, 798 animals. The catch per farm varied from 0 animals to 30 animals in 2017 and from 5 to 50 animals in 2018.

The yellow-necked mouse *Apodemus flavicollis* was the most common catch in the study both on pig and cattle premises (299 and 183 animals, respectively), followed by the second-most common species trapped in the study, the bank vole *Clethrionomys glareolus*. The house mouse *Mus musculus* was common within farm premises. The fourth most common catch was the common shrew *Sorex araneus*. *Rattus norvegicus* was trapped only sporadically (Table 1).

In total, 123 small mammals were trapped from the natural habitat locations away from farm premises in autumn 2018. On average, the number of individuals trapped per site was 11 (range 3–31 individ.). *C. glareolus* was the most common catch with 78 animals. Other caught species included: *S. araneus* (35 animals), the masked shrew *Sorex caecutiens* (4 animals), the sibling vole *Microtus rossiaemeridionalis* (3 animals) and *A. flavicollis* (3 animals).

4.2. Prevalence study: microbiological findings

The total number of pooled farm samples analyzed microbiologically from years 2017 and 2018 was 554. Almost half of the samples consisted of material from one small mammal individual only. At least one of the four zoonotic bacteria species was present in 40% of the pooled samples (Table 2).

The most common pathogen finding was *C. jejuni*, discovered in 181 pooled farm samples. Other *Campylobacter* species were not detected. *C. jejuni* was isolated from all caught animal species, except from the pygmy shrew *Sorex minutus* and water shrew *Neomys fodiens*. It was also identified on every farm premise in the study. The bacteria were detected somewhat more often in samples from cattle farms than in samples from pig farms. Although *C. jejuni* was most commonly detected from the harvest mouse *Micromys minutus* samples (58% of samples), it was also common in *A. flavicollis* (45%) and in *C. glareolus* (41%).

Table 1
Animals caught during the study.

	Piggeries			Bovine farms				Natural habitats
Animal species	2017	2018	Piggery total	2017	2018	Bovine total	In total	2018
Rattus norvegicus	2	14	16	9	1	10	26	0
Apodemus flavicollis	109	190	299	82	101	183	482	3
Mus musculus	34	66	100	28	35	63	163	0
Micromys minutus	2	25	27	8	25	33	60	0
Myodes glareolus	35	79	114	41	69	110	224	78
Microtus agrestis	2	11	13	0	3	3	16	0
Microtus levis	17	49	66	3	30	33	99	3
Sorex araneus	11	52	63	18	44	62	125	35
Sorex minutus	1	2	3	1	0	1	4	0
Sorex isodon	0	0	0	2	2	4	4	0
Sorex caecutiens	0	0	0	0	0	0	0	4
Neomys fodiens	0	0	0	1	0	1	1	0
In total	213	488	701	193	310	503	1204	123

Table 2
Number of positive samples related to small mammal species and type of location.

Bacterium	Rats, n=26	Mice*1 n=708	Voles*2, n=420	Shrews*3, n=173	Piggeries, n=20	Bovine farms, n=18	Natural habitats, n=12
Campylobacter jejuni (n=183)	3	126	52	1	19	17	1
Salmonella enterica (n=10)	0	0	0	7	1	3	3
Yersinia enterocolitica (n=41)	0	8	21	12	13	8	0
Pathogenic Yersinia enterocolitica (n=4)	0	2	2	0	4	0	0
Shiga toxin-producing E. coli (STEC) (n=7)	0	5	2	0	2	4	0

*1 *Apodemus flavicollis, Mus musculus, Micromys minutus*
*2 *Myodes glareolus, Microtus agrestis, Microtus rossiaemeridionalis*
*3 *Sorex araneus, Sorex minutus, Sorex isodon, Sorex caecutiens, Neomys fodiens*

Salmonella sp. was detected on 4/38 farms. The findings originated from *S. araneus* and *S. isodon* individuals, from three cattle farms.

STEC isolates were detected on 6/38 farms, in seven rodent samples. Five of the positive samples were from *A. flavicollis*, one from *M. rossiaemeridionalis,* and one from *M. agrestis*. The animals were caught on four cattle farms and two pig farms.

Yersinia enterocolitica was detected on the premises of 21 farms, in 41 samples. Though on most of the farms only one of the samples was positive, the number of positive samples per farm varied from 1 to 6. The bacterial strains were most commonly isolated from *M. rossiaemeridionalis* samples (13/41) and *S. araneus* samples (12/41). Of the 44 *Y. enterocolitica* strains isolated from the samples, 42 harbored *ail*-gene (95%), 26 belonged to biotype 1 A, and four strains belonged to classical pathogenic biotypes 2, 3 and 4. Of 14 strains that could not be associated to known biotypes, 9 typed close to biotype 2, and 5 were untypeable (Table 3, Supplementary material). Unexpectedly, 24 of 26 (92%) *Y. enterocolitica* biotype 1 A strains contained *ail*-gene. In summary, 9% (4/44) of the isolated strains had all the pathogenicity properties studied.

The number of pooled samples collected from the 11 natural habitat areas in 2018 was 43. Among them, one was positive for *C. jejuni*, three for *Salmonella* sp., and none for *Yersinia* and STEC.

4.3. Prevalence study: estimation of small mammal pathogen prevalence

Campylobacter was the most prevalent of the four pathogens, with an overall small mammal population prevalence (posterior mean & 95% CI) of 0.26 [0.19, 0.34] in 2017 and 0.13 [0.10, 0.17] in 2018. Likewise, the overall prevalence of *Salmonella*, *Yersinia* and STEC were 0.01 [0.00, 0.02], 0.02 [0.01, 0.04], 0.01 [0.00, 0.02] in 2017, and 0.00 [0.00, 0.01], 0.03 [0.02, 0.05], 0.01 [0.00, 0.01] in 2018. The difference between years was significant (i.e. posterior probability for "$p_{0,2017} > p_{0,2018}$" is higher than 95% or smaller than 5%) only for *Campylobacter*, $P(p_{0,2017} > p_{0,2018}) = 0.999$.

There was no clear difference in the prevalence of different pathogens between pig farms and cattle farms, but merely a variation between individual farms (Fig. 2).

4.4. Exposure study: estimation of small mammal population size

The lower bound for a small mammal population size N is naturally the total observed count of marked animals, i.e. the sum of the first catch s_1 and the subsequent unmarked catches to be marked $u_1,\ldots, u_K$. These totals were 67, 68 and 52 in the three farms where capture-recapture experiments were conducted. According to the estimates, probably more than 50% of the population became marked. Posterior means for N were 75, 88 and 69, and the 95% CIs [68,85], [76,102] and [59,83], respectively. The posterior distribution for p_0 and N shows a modestly negative correlation for farms A, B and C (Fig. 3). There was no trend in weekly capture probabilities p_k but merely random weekly variations. The distribution for the expected population size $E(N)=\theta$, mean 75, 95% CI [63,88] was finally used for an assessment of exposure probabilities, according to Eqn 6, which depends both on the population size and the prevalence of pathogens in the population.

4.5. Exposure study: estimation of exposure to pathogens from small mammals

The proportion of small mammals entering the farm buildings during the study was estimated as 0.037, 95% CI [0.015, 0.067], i.e., about 4%. Combining the previous posterior probability distributions on prevalence and expected population size, the estimated exposure probabilities (posterior mean & 95%CI) for *Campylobacter* were: 0.49 [0.24, 0.76], for *Salmonella*: 0.02 [0.00, 0.06], for *Yersinia*: 0.06 [0.01, 0.14], and for STEC: 0.01 [0.00, 0.04] in 2017. Corresponding estimates for 2018 were, for *Campylobacter*: 0.30 [0.13, 0.51], for *Salmonella*: 0.01 [0.00,

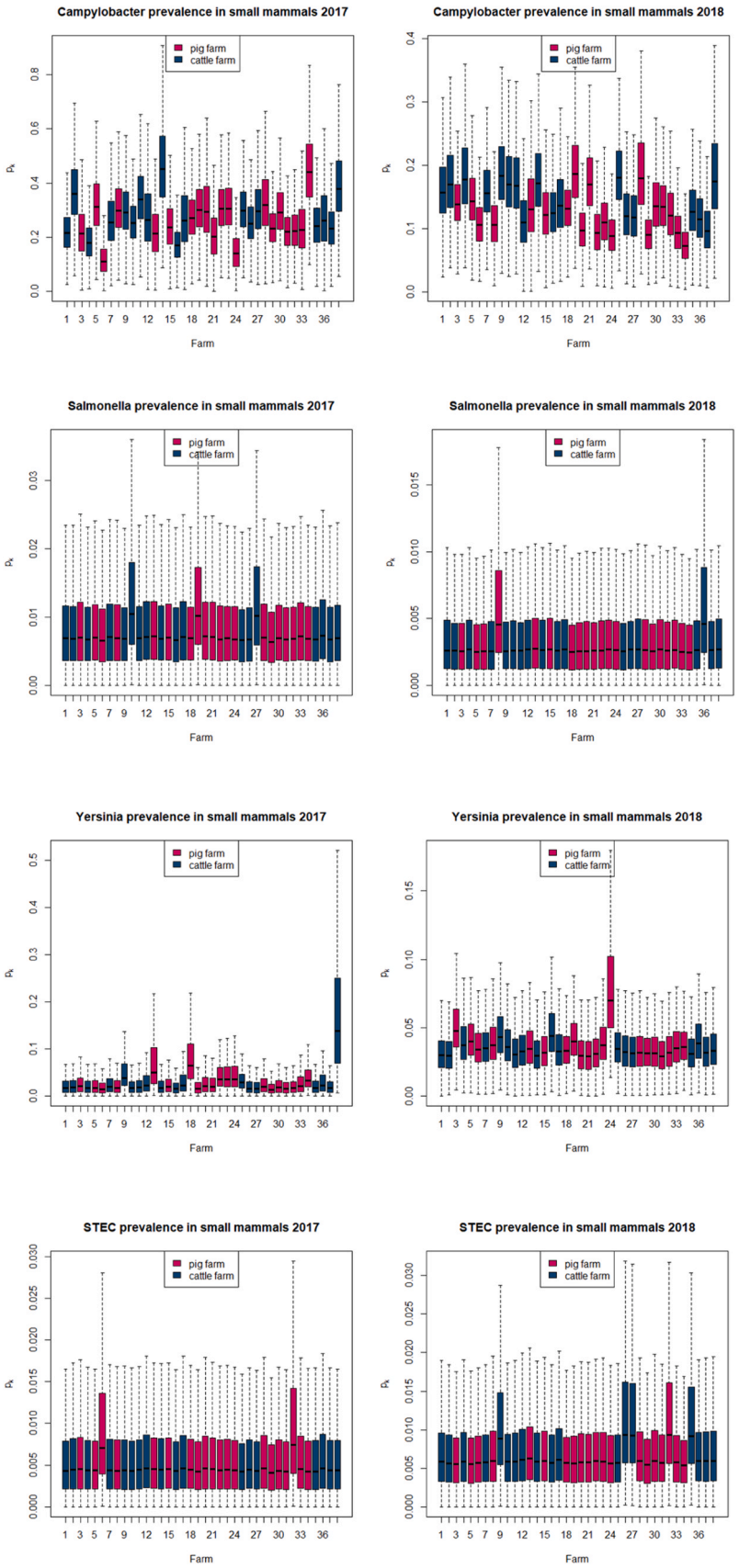

Fig. 2. Farm specific prevalence estimates (2017, 2018) in small mammals for *Campylobacter*, *Salmonella*, *Yersinia* and STEC.

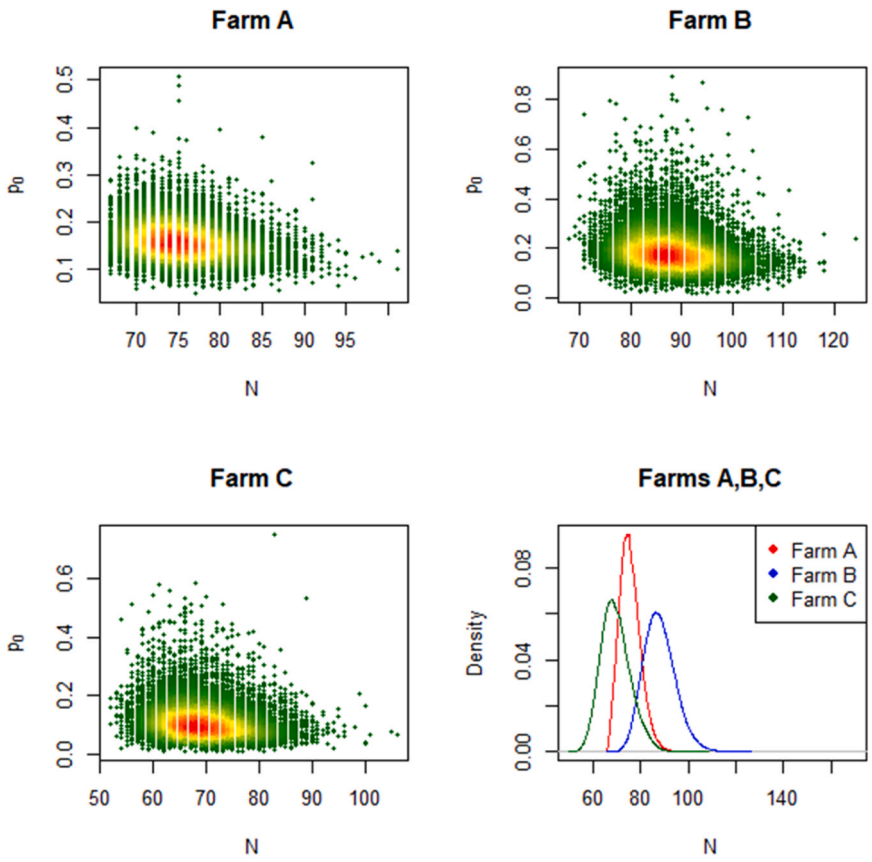

Fig.(3). Estimated overall capture probability p_0 and small mammal population size N for farms A,B,C.

0.03], for *Yersinia*: 0.09, [0.03, 0.18], and for STEC: 0.02 [0.00, 0.04]. The exposure probabilities can be interpreted as the proportion of farms that would be exposed under similar conditions in a similar time frame (four weeks), describing such one-directional causality only (according to Eqn 6).

5. Discussion

In the present study, small mammals were collected at swine and cattle farm premises as well as in natural habitats. As expected, the number of animals caught per site was on average greater on farm premises than in natural habitats, as there is more food and shelter available for animals near farm buildings. The number of small mammal species caught was also different, as house mice were very rarely trapped from locations in more natural habitats, whereas on farms house mice formed almost 60% of the total catch. Small mammals trapped at swine and cattle farm premises were shown to be carriers of pathogenic strains of zoonotic bacteria: *Salmonella*, *Campylobacter*, *Yersinia* and STEC, but comparisons between pathogen prevalence in small mammals in natural habitats and farm premises remains inconclusive due to small sample sizes from natural habitats, i.e. large uncertainty. Our study demonstrates not only that small mammals are hosts of these microbes, but their movements into and around animal housing facilities pose a tangible risk of exposure to livestock, increasing the likelihood of zoonotic disease emergence – or re-emergence. Such movements are typically seasonal in small rodents, especially *A. flavicollis*, which in autumn often migrate from natural surroundings to farm premises (Miño et al., 2007), thus increasing the risk of pathogen invasion.

Campylobacter was the most commonly found pathogen in small mammals in the present study, having been detected in roughly one third of all pooled samples. This was more than in earlier reports on small mammals regarded as pest species. In a study conducted in the Netherlands in nine swine farms and one broiler farm, the prevalence of *Campylobacter* spp. in small mammals was around 10% (Meerburg et al., 2006). The same prevalence was observed by Adhikari et al. (2002) in rodents on a dairy farm. In Sweden, the prevalence of *C. jejuni* in different species of small mammals was 2–28%, the highest portion of positive samples isolated from *A. flavicollis* (Backhans et al., 2013). In the present study, *C. jejuni* was more commonly found in small mammals than in earlier studies. As cultivation was done from the primary suspension without delay in this study, the reason behind the difference could partly be the sampling procedure. *C. jejuni* was isolated from almost every species of small mammals and on every farm in the study, suggesting that the incidence of the pathogen is not related to geographical location within the study area or production type of the farms.

In the present study, *Salmonella* was only rarely detected from small mammal samples. The four findings were from shrews, which as a species differ from other small mammals most pronouncedly by their diet, which consists of invertebrates compared to rodents which subsist on an herbivorous diet (Gliwicz and Taylor, 2002). Therefore, farm buildings contain a more attractive and plentiful source of food for rodents than for shrews, particularly during colder months. Thereby, shrews are likely to enter farm buildings less often than rodents, resulting most likely in less frequent contact rates between shrews and farm animals compared to other small mammals. It may thus be that the *Salmonella* circulates in shrew populations without posing a major threat to livestock.

Previous studies suggest that *Salmonella* findings in rodents near farm areas are closely related to the *Salmonella* findings in the production animals of the farm. For example, in Denmark, *Salmonella* was detected in rodents only during periods during which *Salmonella* was detected also in the production animals (Skov et al., 2008). In a study by Bachans et al. (2013) on swine and poultry farms, one rodent sample

tested positive for *S. enterica* serovar Typhimurium. The farm had recently experienced an outbreak of salmonellosis, which might explain why also the mice living on the farm premises carried the pathogen. To our knowledge, the farms that participated in our study did not experience an outbreak of salmonellosis during the study and had not done so in the recent past. In Finland, the prevalence of *Salmonella* is generally very low in production animals. In 2017, *Salmonella* was detected from 6 cattle farms and 11 swine farms out of 11,500 and 1200, respectively (Finnish Food Authority, 2018). Following this, in 2018, it was detected from 28 cattle farms and 7 swine farms out of 10,617 and 1200 (Finnish Food Authority, 2019). Therefore, it was not surprising that *Salmonella* was not detected from rodents in the present study.

It is well known that healthy cattle can be carriers of STEC, and that bovine carcasses and subsequent meat products can be contaminated. In Finland, the prevalence of STEC O157:H7 serotype in cattle was monitored in a national control program until 2021. The prevalence in slaughter animals and herds remained below 1.5% during 2006–2010 and increased to 1.5–3.2% during 2012–2020 (Finnish Food Authority, 2022), which corresponds with the small mammal findings in the present study. In a previous study from Denmark, the prevalence of STEC among 260 wild animal samples was generally low, but high in rodent fecal samples collected near cattle farms. Rodents nearby cattle farms were identified as STEC carriers – either transmitting the infection to farm animals or getting infected in the farm (Nielsen et al., 2004).

STEC are present also in pork products, but often in much lower numbers than in products derived from ruminant animals (World Health Organization and Food and Agriculture Organization of the United Nations, 2018). The prevalence of STEC in pig populations in European studies has varied from 0.1% to 0.7% (Tseng et al., 2014). The study by Nielsen et al. (2004) found no STEC positive rodents nearby two swine farms studied. In the present study, the prevalence of STEC in small mammals did not differ between cattle or swine farms. From a public health perspective, the occurrence of STEC in swine populations may be overlooked in Finland.

In the present study, the prevalence of pathogenic *Y. enterocolitica* was generally low and *Y. pseudotuberculosis* was not found in small mammals. Likewise, in a recent study of 244 samples collected near farms and fodder factories in Poland (Platt-Samoraj et al., 2020), *Y. enterocolitica* was isolated from 16.8% rodents and *Y. pseudotuberculosis* was detected in one sample. A low prevalence of *Y. pseudotuberculosis* and detection of pathogenic *Y. enterocolitica* in rodents in connection with swine farms was observed in Sweden by Backhans et al. (2011). However, *Y. pseudotuberculosis* has been found in *S. araneus* in Finland, associated with a foodborne outbreak, by Kangas et al. (2008). Similarly to this study, *Y. enterocolitica* strains representing the classical pathogenic biotypes 2, 3 and 4 were found in small mammal samples only in pig farms. Pigs are the main reservoir for *Y. enterocolitica* infections in humans and biotype 4 is the most reported pathogenic type in pigs, pork products and humans (Fredriksson-Ahomaa et al., 2006), (The European Union One Health 2021 Zoonoses Report | EFSA, 2022). In a recent study in Great Britain (Arden et al., 2022), the overall prevalence of *Y. enterocolitica* in the randomly sampled population was 3.73%, which is in line with our findings. As in earlier studies on small mammals (Arden et al., 2022; Joutsen et al., 2017; Platt-Samoraj et al., 2020), most of the *Y. enterocolitica* strains in this study belonged to biotype 1 A. Platt-Samoraj et al. (2020) and Arden et al. (2022) reported all their *Y. enterocolitica* findings belong to biotype 1 A and concluded that they do not pose a high epidemiological risk. Though the strains of this biotype are classically considered non-pathogenic, this highly heterogeneous group may harbor subgroups that can be opportunistic pathogens and cause disease (Batzilla et al., 2011; Huovinen et al., 2010).

In the present study, an exceptionally high proportion of *Y. enterocolitica* strains had the pathogenicity associated *ail*-gene but did not belong to classical pathogenic biotypes or have other biochemical virulence associated traits. Unexpectedly, most of the *Y. enterocolitica* biotype 1 A strains in our study contained the *ail*-gene. Joutsen et al. (2017) reported that small mammals can be carriers of *ail* positive *Y. enterocolitica* BT1A and speculated that these strains have an ability to colonize the animal host, which explains their frequent isolation. The proportion of *ail* positive BT 1 A strains in our study was, however, even higher (92%) than detected for biotype 1 A strains in the 7-year study of Joutsen et al. (23%) (2017). The role of the *ail*-gene as the sole pathogenicity marker in diagnostics can be regarded as controversial. However, the pathogenic properties of the isolated strains in the present study need further evaluation.

Our study did not investigate zoonotic bacteria prevalence or changes of such prevalence in domestic animals in the study farms. Thereby, estimation of pathogen transmissions, or comparisons of bacteria strains between domestic animals and small mammals is not possible, nor demonstrating the initial source of pathogens. Instead, taking the cross-sectional situation as a current state of affairs, the probability of exposure could be quantified as the probability of at least one small mammal carrying the pathogens into the farm premises.

The quantitative estimation of average exposure potential to pathogens from small mammals involves data sources from both the pathogen prevalence study and the capture-recapture experiment, and refers to a similar length of time in the autumn as in the capture-recapture experiment, i.e., four weeks, which represents the time of year with the most likely exposure due to small mammals. Significant farm-to-farm variation in exposure probabilities can occur due to both spatial and temporal variation in pathogen prevalence and small mammal population size. High rodent numbers after the reproductive period in autumn can be associated with higher numbers of infected individuals, even when prevalences are not dependent upon population density – however, this varies substantially from pathogen to pathogen (Davis and Calvet, 2005). Furthermore, rodents in northern boreal environments typically seek refuge from incremental environmental conditions in human settlements, e.g., farm houses, in late autumn (Khalil et al., 2014), which increases the likelihood of exposure.

Of note, the exposure probability in the present study refers to a single generic farm. When there are more farms, it is more likely that at least one of them is exposed. For example, there were 10,617 cattle farms in Finland in 2018 (Finnish Food Authority, 2019). Assuming the possible time of exposure is within the autumn months (as in the capture-recapture experiment) the expected number of *Salmonella* exposed farms would be about $10{,}617 \times 0.01 = 106$. In the same year, 2018, the number of detected positive cattle farms in Finland was 28. Considering that probably not all of the farms were infected by small mammals, 0.01 may be an overestimate for exposure, or there are undetected positive farms, as was suggested by Ranta et al. (2005). The catch ratio of infected herds can be significantly less than 100% when not all herds are tested in control programmes and due to animal sampling randomness and test sensitivity. Moreover, exposure from small mammals may not necessarily imply infection in farm animals. Consequently, a quantitative assessment of the probability of infection from exposure would require further study on the relationship of exposure and animal dose-response. With these unquantified uncertainties in mind, the exposure estimates have a realistic magnitude. Adding complementary data on remaining issues and extending the models for a more dynamic process could improve quantitative risk assessments on environmental sources of infection.

The present study concurs with the literature, in that wild small mammals carry zoonotic bacteria in their digestive tract and may thus constitute an important factor in the epidemiology of these pathogens (Jahan et al., 2021). At risk are especially all-in/all-out systems in pig and cattle production, where small mammals may maintain and spread bacteria within a farm, especially between different production batches. In areas where different farms are situated close to each other, populations of the most common small mammal species, e.g., bank voles and yellow-necked mice, may be contiguous and encompass numerous farms, enabling the transmission of pathogens not only within the small

mammal population but also from farm to farm. The probability of pathogen transfer between farms is low in areas where the prevalence of zoonotic bacteria in both production animals and wild small mammals is low. However, when the infection pressure increases in production animal herds, the role of small mammals as carriers of these diseases may also increase. Likewise, an increase in the abundance of infected wild small mammals in the surrounding matrix of the farms and their contact rates with production animals will likely elevate both the probability of exposure of production animals to pathogens and the probability of farm-to-farm transmission.

Although the role of small mammals as sources of environmental bacterial contamination on farms, and the link between rodent infestation and foodborne pathogen outbreaks has received increasing corroboration worldwide, reviewed by Jahan et al. (2021), detailed knowledge of transmission dynamics in such environments are still rare. More research is sorely needed to quantify the dynamics of zoonotic pathogen infections in wild small mammal – production animal systems with special emphasis on the elucidation on the role of wild small mammals as reservoir hosts. Our study provides a one-directional quantitative exposure assessment as one part on the way towards a more comprehensive risk assessment.

To prevent foodborne infections in humans, the whole food chain must act to minimize the contamination of the products. Production farms, such as pig and cattle farms, need to have such biosecurity measures in place so that infections in the animals via their feed and environment are prevented. On farms, one of the key elements of biosecurity measures is pest control (Collins and Wall, 2004).

CRediT authorship contribution statement

Heikki Henttonen: Writing – review & editing, Conceptualization. **Saija Hallanvuo:** Writing – review & editing, Resources, Methodology, Investigation, Data curation. **Jukka Niemimaa:** Resources, Investigation. **Otso Huitu:** Writing – review & editing, Supervision, Resources, Project administration, Investigation, Conceptualization. **Maria Simola:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Jukka Ranta:** Writing – review & editing, Visualization, Software, Methodology, Formal analysis, Conceptualization. **Leena Seppä-Lassila:** Writing – review & editing, Resources. **Heidi Rossow:** Writing – review & editing, Resources.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.prevetmed.2024.106228.

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