Summary

Enterobacteriaceae such as *Escherichia coli* are common commensals as well as opportunistic and obligate pathogens. They cause a broad spectrum of infectious diseases in various hosts, including hospital-associated infections. In recent years, the rise of extended spectrum beta-lactamase (ESBL)-producing *E. coli* in companion animals (dogs, cats and horses) has been striking. However, reports on nosocomial infections are mostly anecdotic. Here we report on the suspected nosocomial spread of both ESBL-producing and non-ESBL-producing multi-drug resistant *E. coli* isolates in three equine patients within an equine clinic. Unlike easy-to-clean hospitalization opportunities available for small animal settings like boxes and cages made of ceramic floor tiles or stainless steel, clinical settings for horses are challenging environments for infection control programs due to unavoidable extraneous material including at least hay and materials used for horse bedding. The development of practice-oriented recommendations is needed to improve the possibilities for infection control to prevent nosocomial infections with multi-drug resistant and other transmissible pathogens in equine clinical settings.

Keywords: nosocomial infection, horse clinic, equine infection, ESBL, *E. coli*, infection control, hygiene

Zusammenfassung


Schlüsselwörter: nosokomiale Infektionen, Pferde klinik, equine Infektion, ESBL, Infektionskontrolle, *E. coli*, Hygiene
**Introduction**

A nosocomial infection, also called “hospital-acquired” (ha-) infection, is defined as “an infection acquired in a hospital by a patient who was admitted for a reason other than that infection” (World Health Organization, 2001). Whether the patient develops an active disease in succession of an individual colonization present at admission (endogenous nosocomial infection) or consecutively after infection by an exogenous source (e.g. environment, staff, other patients) during hospital stay is irrelevant for the definition of a nosocomial infection. Since the beginning of the 21st century nosocomial transmission events or even outbreaks were reported for small animal clinics (Francéy et al., 2000; Boerlin et al., 2001; Ishihara et al., 2010). A recent study on veterinary teaching hospitals revealed that 31 (82%) out of 38 reported outbreaks of nosocomial infections during the five years prior to an interview, and nineteen (50%) hospitals reported that zoonotic infections had occurred during the last two years (Benedict et al., 2008). With respect to equine patients, nosocomial infections associated with hospitalized horses also gain increasing attention in the scientific community (Seguin et al., 1999; Boerlin et al., 2001; Dargatz and Traub-Dargatz, 2004; Cuny et al., 2006; Henninger et al., 2007; Weese, 2007; Cuny et al., 2008; Walther et al., 2008; Eki et al., 2009; Walther et al., 2009; Dallap Schaer et al., 2010; Bergstrom et al., 2012; Damborg et al., 2012; Van den Eede et al., 2012). Even failure of an infection control program (ICP) resulting in an outbreak of salmonellosis has been reported: A fatal series of infections of equine patients associated with an AmpC-beta-lactamase-producing *Salmonella enterica* subsp. *enterica* Serovar Newport (S. Newport) strain ended up in a temporary but complete hospital closure (Dallap Schaer et al., 2010). It is inevitable that a hospital closure creates important financial losses, adversely affects the reputation of hospitals, and limits provision of care for animals. In case of an university clinic, education of veterinary students, interns, and residents is at halt (Steneroden et al., 2010). In general, some bacterial species of the resident equine microbiota on skin and mucosa (host microbiota) are assigned as “opportunistic pathogens”, including *Staphylococcus aureus* and *Escherichia coli*. When these bacteria overcome the natural body barriers and invade the body, fulminant and severe infections may develop, depending on the individual’s immune status. The report by Dallap Schaer et al. (2010) highlights that pathogens commonly involved in nosocomial infections are antimicrobial-resistant bacteria like meticillin resistant *S. aureus* (MRSA), extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae or multi-drug resistant *Acinetobacter baumannii* (Walther et al., 2013; Müller et al. 2014). Infections with those or other (multi-drug-)resistant pathogens lead to intensive and prolonged medical care, higher treatment costs and risk of nosocomial spread. This spread includes other patients, the environment, and – very important – veterinary personnel (Panchaud et al., 2010).

Here we present the follow-up of *E. coli* infections (ESBL-producing and non-ESBL-producing isolates) associated with three different hospitalized equine patients after colic surgery with respect to molecular typing results and spatiotemporal association indicating a nosocomial relationship.

**Material and Methods**

**Equine patients, environmental specimens and bacterial isolates**

The present retrospective study includes three equine patients with clinical signs of surgical site infection (SSI) after initial colic surgery (Tab. 1). Hospital admission for horse A was 12 days prior to the start of the sampling period (total hospital stay: six months), for horse B six days prior to the sampling time (hospital stay: one month), whereas patient C was admitted to the equine clinic at the third day of the sampling period (hospital stay: 11 days). The horses were of middle age (11–18 years), none of them received continuin medical care. Chronic diseases were not reported in any of the patients. Initially, horse A had a colic surgery, followed by a paralytic ileus afterwards, was then re-operated, developed SSI and a generalized disease state including a septic joint infection together with osteomyelitis within 22 days (overview provided in Tab. 1). Finally the moribund horse had to be euthanized. Horse B underwent colic surgery as well as re-laparotomy because of complications (SSI, leakage at the anastomosis site and ileus IMT26117). Horse C underwent colic surgery, was then re-laparotomized because of complications (SSI, leakage at the anastomosis site and ileus IMT26117). In total, six horses were hospitalized for a period of 11–22 days, prior to the sampling period.

**TABLE 1: Clinical data on three equine patients**

<table>
<thead>
<tr>
<th>Horse</th>
<th>Admission</th>
<th>Demographic parameters</th>
<th>Disease</th>
<th>Day of sampling</th>
<th>Site (sample)</th>
<th>Isolate designation</th>
<th>Pulsotype</th>
<th>Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>12 days prior start of sampling period</td>
<td>17 years, Mecklenburger Warmblood, male (neutered)</td>
<td>ileus of the jejunum caused by pendunculated lipoma</td>
<td>1</td>
<td>SSI</td>
<td>IMT26112</td>
<td>B</td>
<td>after six months (euthanized)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>bronchitis, duodeno-jejunitis</td>
<td>7</td>
<td>tracheal wash</td>
<td>IMT26383</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>septic arthritis and osteoarthritis</td>
<td>22</td>
<td>infected joint</td>
<td>IMT26404</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>6 days prior start of sampling period</td>
<td>11 years, Brandenburg Warmblood, male (neutered)</td>
<td>colon descendens: obstructive intestinal polyp with luminal bleedings</td>
<td>5</td>
<td>SSI</td>
<td>IMT26115</td>
<td>A-1</td>
<td>after one month</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>peritoneal fluid</td>
<td>8</td>
<td></td>
<td>IMT26116</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>third day of the sampling period</td>
<td>18 years, Oldenburg, female</td>
<td>ileus of the jejunum caused by pendunculated lipoma</td>
<td>12</td>
<td>SSI</td>
<td>IMT26172</td>
<td>B</td>
<td>after 11 days (euthanized)</td>
</tr>
</tbody>
</table>

Admission: date of hospital admission of three equine patients with colic surgery, relative to the start of the 22-sampling period (Tab. 2); SSI: surgical site infection; Pulsotype according to assignment presented in figure 1.
peritonitis). Horse C developed SSI after initial colic surgery and was re-operated, whereby obstruction at the anastomosis site was diagnosed. The patient was euthanized due to animal welfare reasons 11 days after the first surgery (Tab. 1).

One to three samples from different infected sites were taken from these patients during the study period (Tab. 2). For room status evaluation, wetted cotton swabs were used on rough surfaces (19 locations) within five rooms (surgery ward, surgery preparation room, pharmacy room, surgery associated storage room, knock down box) on day 14 (at work end) prior to cleaning and disinfection measures. Microbiological examination was carried out as follows: the swabs were plated on Columbia Agar containing 5% sheep blood (Oxoid Wesel, Germany), Brilliance UTI Clarity-Agar plates (Oxoid Wesel, Germany) and Gassner Agar (Oxoid Wesel, Germany), at 37°C for 18 hours. Samples were investigated qualitatively for the presence of ESBL-producing Enterobacteriaceae, Acinetobacter baumannii and MRSA.

**FIGURE 1:** Dendrogram based on PFGE fragment pattern of XbaI digested total cellular DNA from 13 E. coli isolates. Two major pulsotypes (PT) A (including A-1; first two rows) and B were distinguished from singletons associated with PT C and D. Day of isolation: Sampling according to time scale provided in table 2. ST: sequence type; SSI: surgical site infection; ESBL-enzymes: CTX-M1: cefotaximase-munich-1; SHV-12: sulfhydryl-variable 12.

**TABLE 2:** Overview on E. coli isolates obtained from three patients and two environmental sources during the 22-days sampling period

<table>
<thead>
<tr>
<th>Origin</th>
<th>Sample site</th>
<th>Day of isolation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horse A</td>
<td>SSI (after median laparotomy)</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td></td>
<td>tracheal wash</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td></td>
<td>joint puncture (septic osteomyelitis)</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td>Horse B</td>
<td>SSI (after median laparotomy)</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td></td>
<td>peritoneal fluid</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td>Horse C</td>
<td>SSI (after median laparotomy)</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td>Environ</td>
<td>fixation rope</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
<tr>
<td></td>
<td>drop down box</td>
<td>1 A 2 B 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22</td>
</tr>
</tbody>
</table>

Pulsotypes (PT) of the E. coli isolates are indicated by squares (A, A-1, B, C and D); SSI: surgical site infection

**Phenotypic and genotypic characterization of E. coli**

Based on phenotypical morphology, each presumptive E. coli variant (up to two per sample) was sub-cultured and identified using the API 20 E identification system (BioMérieux Nürtingen, Germany). E. coli isolates were screened for ESBL-production using the phenotypic confirmatory test, performed and interpreted according to CLSI standard (Clinical and Laboratory Standards Institute, 2012). Using the VITEK®2 system (BioMérieux, Germany) minimal inhibitory concentrations (MIC) testing for antimicrobials was performed according to the standards given by the CLSI M31-A3 (CLSI, 2008) and M100-S21 (CLSI, 2011). Genotypic screening (PCR) for the resistance genes blaCTX-M,blaTEM,blaSHV,blaOXA,teta-C,qnrA/B/S,sul1–3,strA/B,aadA,aac(VI) and aac(3)-VI as well as for plasmid incompatibility and rep-licon types (inc/rep) was performed for ESBL-producing isolates and their transconjugants as described previously (Carattoli et al., 2006; Ewers et al., 2010). Multi-locus sequence typing (MLST) was carried out as described earlier (Wirth et al., 2006) using primers
specified on the E. coli MLST web site (http://mlst.ucc.ie/mlst/mlst/dbs/Ecoli). Sequences were analyzed with the software Ridom SeqSphere 0.9.39 (http://www3.ridom.de/seqsphere). Phylogenetic groups were determined using the software Structure 2.3.4 based on the concatenated sequences of the seven housekeeping genes used for MLST (http://pritch.bsd.uchicago.edu/structure.html) (Bednorz et al., 2013). To assess the relatedness of the E. coli isolates, macrorestriction analysis was performed as previously described using a CHEF DRIII System (BioRad, Munich, Germany) (Bednorz et al., 2013). PFGE profiles generated by restriction of chromosomal DNA with XbaI were compared digitally using BioNumerics software (Version 6.6, Applied Maths, Belgium). Cluster analysis of Dice similarity indices based on the unweighted pair group method with arithmetic mean (UPGMA) was applied to generate a dendrogram depicting the relationship of PFGE profiles. Isolates were considered to belong to a group of clonally related isolates if the Dice similarity index of the PFGE pattern was ≥85% (Ewers et al., 2010).

**Conjugation experiments**

Transfer of beta-lactam resistance was performed by the filter mating method using E. coli J53 Azis® as recipient (Martinez-Martinez et al., 1998). Mating experiments were performed at 37°C and 22°C in liquid and solid (filter) media. Transconjugants were selected on trypticase soy agar, containing 100 mg/ml sodium azide and 1 mg/ml ceftazidime. Co-transfer of resistance determinants was explored by amplifying the respective genes in the transconjugants as described above, followed by sequence analysis, if necessary (blaCTX-M-1, blaTEM-1, blaSHV).

**Results**

Here we report on the microbiological results of samples obtained from three hospitalized equine patients with clinical signs of surgical site infection (SSI) after colic surgery and from environmental sources. While neither A. baumannii nor MRSA were detected in the samples during microbiological examination, eleven E. coli were isolated from six samples received from the equine patients and two from environmental swabs (Fig. 1) within the sampling period of 22 days. These 13 isolates were assigned to two major pulsotypes, (PT) A (including A-1, one band difference) and B as well as to two singletons (PT C and D) (Fig. 1 and Tab. 2). Identical pulsotypes are judged as E. coli that belong to the same clone.

PT B included six E. coli isolates originating from different infection sites of each of the three horses and one environmental source (fixation rope). Due to the spatiotemporal relationship of these isolates, a nosocomial appearance is indicated (Tab. 2). In addition, the ESBL-producing E. coli associated with PT A was identified for three isolates obtained from horses A (SSI and tracheal wash) and C (SSI) at three distinct sampling times, and the closely related PT A-1 originated from horse A (septic arthritis joint puncture) and horse B (SSI) at different time points (Fig. 1, Tab. 1 and 2.). Interestingly, samples taken simultaneously (day 5) from the SSI (after median laparotomy) and the peritoneal fluid of horse B presented different microbiological results: the SSI was positive for both the ESBL-producing E. coli associated with PT A-1 and PT B (non-ESBL-producing E. coli), while the peritoneal fluid was associated with the singleton PT C (IMT26118), a further multi-drug resistant ESBL-producing E. coli (Tab. 2). One additional ESBL-producing E. coli was isolated solely from the floor of a knock down box (PT D; IMT26576). Resistance determinants detected in the ESBL-producing isolates were blaCTX-M-1, blatem-1, tetB, sul2, and strA/B for those belonging to PT A-1, blaCTX-M-1, blatem-1, tetB, sul1 and sul2, and strA/B for PT A isolates, blatem-1, blatem-1, and strA/B for PT C and blasm-12, strA/B for PT D. Irrespective of their pulsotype, all isolates showed the same mutation in the gyrA (83.S-1_87.D-N) and parC region (805-1). Further, transconjugation experiments with the ESBL-producing E. coli showed a general transferability of plasmids for all isolates, while plasmid incompatibility group testing detected group FIB for all CTX-M-1 isolates and FIA for the SHV-12 isolate IMT26576. Non-beta-lactam resistance genes that were co-transferred were sul1, strA and strB.

Multilocus sequence typing revealed that isolates assigned to PT A belonged to Sequence type 10 (ST10), those associated with PT B were ST354 and the singletons belonged to ST224 (IMT26576) and ST1011 (IMT26118), respectively (Fig. 1).

Minimal inhibitory concentration results obtained for the 13 E. coli match with their respective PT (Tab. 2). Regardless if ESBL-producing E. coli or not, all isolates exhibited phenotypic resistance towards ampicillin, tetracycline, piperacillin, gentamicin, enrofloxacin, marbofloxacin, and trimethoprim-sulfamethoxazole combinations.

**Discussion**

Here we report microbiological results of samples received from three horses which developed SSI after colic surgery, including re-laparotomy due to different complications. Molecular characteristics together with the spatiotemporal relationship (all E. coli isolates were obtained within 22 days, all horses were hospitalized in the same clinic and had colic surgery in the same ward) indicated nosocomial transmission of E. coli assigned to PT B, including the three horses and one environmental site. In addition, the ESBL-producing E. coli isolates associated with PT A and subtype PT A-1 were also associated with nosocomial transmission. Since the non-ESBL producing E. coli isolate (PT B; IMT26190) was obtained at the end of a working day (day 14) from a fixation rope within the surgery, spread of this particular pulsotype (PT B) within the clinic seems to be reasonable. However, the extremities of horses were not completely disinfected in cases of colic surgery, and a certain degree of environmental contamination due to the handling and fixation of the equine patient is possibly inevitable (Walther et al., 2013). Within a hospital environment, a baseline degree of selective pressure on residing microorganisms is generally reasonable, induced amongst others by the use of antibiotics in diseased hospitalized patients. In consequence, a (multi-drug) resistant phenotype provides a fundamental advantage for the survival of the individual organism within a certain facility. A study performed on fecal E. coli illustrates this mechanism: an unanticipated high frequency and genetic diversity of CTX-M-producing E. coli was observed in the fecal microbiota of hospitalized patients receiving...
TABLE 3: Results of minimum inhibitory concentration testing of E. coli isolates using VITEK®2

<table>
<thead>
<tr>
<th>Strain designation</th>
<th>ST Pulso-type</th>
<th>AMP</th>
<th>AMC</th>
<th>PIP</th>
<th>CPD</th>
<th>CEF</th>
<th>AN</th>
<th>GM</th>
<th>TOB</th>
<th>ENR</th>
<th>MAR</th>
<th>TET</th>
<th>FM</th>
<th>C</th>
<th>SXT</th>
</tr>
</thead>
<tbody>
<tr>
<td>IMT26383</td>
<td>10 A</td>
<td>≥ 32</td>
<td>≥ 32</td>
<td>≥ 128</td>
<td>≥ 8</td>
<td>≥ 8</td>
<td>&lt; 2</td>
<td>≥ 16</td>
<td>≥ 16</td>
<td>≥ 4</td>
<td>≥ 4</td>
<td>≥ 4</td>
<td>≥ 16</td>
<td>≥ 16</td>
<td>≥ 64</td>
</tr>
<tr>
<td>IMT26112</td>
<td>10 A</td>
<td>≥ 32</td>
<td>≥ 32</td>
<td>≥ 128</td>
<td>≥ 8</td>
<td>≥ 8</td>
<td>&lt; 2</td>
<td>≥ 16</td>
<td>≥ 16</td>
<td>≥ 4</td>
<td>≥ 4</td>
<td>≥ 4</td>
<td>≥ 16</td>
<td>≥ 16</td>
<td>≥ 64</td>
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<td>IMT26176</td>
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<td>≥ 32</td>
<td>≥ 128</td>
<td>≥ 8</td>
<td>≥ 8</td>
<td>&lt; 2</td>
<td>≥ 16</td>
<td>≥ 16</td>
<td>≥ 4</td>
<td>≥ 4</td>
<td>≥ 4</td>
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<td>≥ 16</td>
<td>≥ 64</td>
</tr>
<tr>
<td>IMT26115</td>
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<td>≥ 32</td>
<td>≥ 128</td>
<td>≥ 8</td>
<td>≥ 8</td>
<td>&lt; 2</td>
<td>≥ 16</td>
<td>8</td>
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<td>≥ 16</td>
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<tr>
<td>IMT26405</td>
<td>10 A-1</td>
<td>≥ 32</td>
<td>≥ 32</td>
<td>≥ 128</td>
<td>≥ 8</td>
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<td>&lt; 2</td>
<td>≥ 16</td>
<td>8</td>
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<td>≥ 16</td>
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<td>≥ 32</td>
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<td>&lt; 2</td>
<td>≥ 16</td>
<td>8</td>
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<td>≥ 128</td>
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<td>4</td>
<td>&lt; 2</td>
<td>≥ 16</td>
<td>8</td>
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<td>IMT26118</td>
<td>1011 C</td>
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<td>≥ 128</td>
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<td>≥ 4</td>
<td>≥ 16</td>
<td>≥ 16</td>
<td>4</td>
</tr>
</tbody>
</table>

Dark shaded strain designation (left side). E. coli isolated coincidently from horse B (PT A-1 and B: SSI; PT C: peritoneal fluid) at day 5 of the study period. Grey shaded boxes (right side) = resistant, light grey boxes = intermediate susceptibility, white boxes = susceptible, bold isolate designation: ESBL-producing E. coli (according to CLSI M1-A3; M100-S21)

AMC = amoxicillin-clavulanic acid; AMP = ampicillin; AN = amikacin; C = chloramphenicol; CEF = ceftiofur; CPD = cefpodoxime; ENR = enrofloxacin; FM = nitrofurantoin; GM = gentamicin; MAR = mafenoxacim; PIP = piperacillin; SXT = trimethoprim-sulfamethoxazole; TOB = tobramycin; TET = tetracycline

broad-spectrum antimicrobials (Damborg et al., 2012). Further, nosocomial transmission was suggested by finding five identical CTX-M-1-producing E. coli in the feces of multiple horses (Damborg et al., 2012). In this study, horse B suffered from SSI after median laparotomy and repeat celiotomy revealed leakage of the anastomosis with severe peritonitis. Coincidently, the “outer” SSI was positive for isolates assigned to PT A-1 (ESBL-producing) and PT B, while the peritonitis isolate (PT C; IMT26118) belonged to a further ESBL-producing E. coli. While incisional infection is a common complication after colic surgery, and incidence reported in the literature varies from 7.1% up to 37% for initial surgery, rates seem to raise up to 87.5% if re-laparotomy is necessary (reviewed in: Hassel, 2013). Thus, only specimens taken from all infected sites of the patient are able to reflect the complete microbiological picture (here: three different E. coli isolates). This information together with the distinct resistance profile recorded for each isolate is necessary to choose an adequate anti-infective therapy, although the chemotherapeutic possibilities are rather limited in this case (resistance profiles of isolates obtained from infected sites of horse B, indicated by dark shaded strain numbers in Table 3).

Since Damborg and colleagues (2012) assumed nosocomial transmission of distinct ESBL-producing E. coli within clinical environments before, the nosocomial relationship of isolates assigned as PT A and subtype A-1 from three equine patients seems to be more likely than plasmid spread of the ESBL-encoding resistance determinant among indistinguishable recipients. However, the possibility of plasmid transfer is likely, since one of the clonally unrelated ESBL-E. coli (PT C; IMT26118) carried both the same ESBL-encoding gene (blaCTX-M-1) as well as an identical incompatibility group plasmid (FIB). Since blaCTX-M-1 is amongst the most prominent ESBL-encoding genes associated with Enterobacteriaceae of companion animal origin in Europe (Ewers et al., 2012) including horses (Damborg et al., 2012), only deeper analyses (via sequencing) of the plasmids could prove a plasmidic transfer.

The sequence types obtained from the ESBL-producing E. coli like ST10 and ST224 have been commonly found in human and veterinary clinical isolates as well as in food and wild animals, underlining the capacity to adapt to completely different hosts and environments of the pathogens (Ewers et al., 2012).

Hospitalized equine patients reside in stables including stall bedding (e.g. straw, wood pellets, moss, shavings) and the naturally occurring waste is a potential infection hazard, challenging the distinction between endogenous and exogenous infection sources and mirrors the complexity for infection control measures within this clinical environment: a recent study detected 6.3% of 650 fecal samples from horses attended by veterinary surgeons in the United Kingdom positive for commensal ESBL-producing E. coli (Maddox et al., 2012). Incidental environmental sampling included in this study was limited to five rooms in the proximity of the surgery and provided only qualitative data on samples obtained before cleaning and disinfection measures on day 14 of the sampling period. However, two different multidrug resistant E. coli were obtained from the floor of the drop-down box (IMT26576) and the fixation rope (IMT26190).

Since ESBL-harboring plasmids are often easily transmissible between different members of Enterobacteriaceae, transmission to already multidrug resistant Salmonella strains as recipients is a kind of “worst case” scenario for equine hospitals. In 2007, Vo et al. showed that plasmids carrying bla-CTX-M-1 were transmissible in vitro from clinical E. coli (ESBL-producing) to clinical Salmonella enterica subsp. enterica Serovar Typhimurium isolates (Vo et al., 2007).

One further aspect should be mentioned: when long-time hospitalization of multi-morbid equine patients (here: patient A) in clinical settings is inevitable, these patients should be regarded as a potential, permanent source of multi-drug resistant pathogens threatening other clinic patients. Thus, transmission prevention including risk management of equine patients is necessary (Walther et al., 2014).
Conclusion

A remarkable multi-drug resistance was associated with all 13 E. coli reported here, not only with the ESBL-producing isolates. In veterinary medicine, beta-lactams are among the most important anti-infective drugs and are widely used for treating bacterial infections, including those caused by Enterobacteriaceae (Rubin and Pitout, 2014). Thus, the resistance profiles of the E. coli reported here are alarming, since the availability of licensed antibiotic drugs for a sufficient anti-infective chemotherapy in equine patients is very limited and alternatives in terms of useful agents are rare or even absent (Maddox et al., 2012). Consequently, further multi-disciplinary efforts to establish evidence-based hygiene improvements and patient-based antibiotic stewardship within the challenging clinical environment in equine clinics are indispensable to prevent nosocomial spread to an absolute inescapable baseline-limit.

Conflict of interest

The authors declare that no competing interests exist.

Reference


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