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CONGRESS

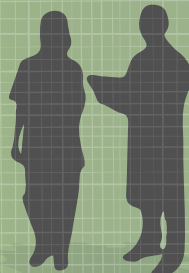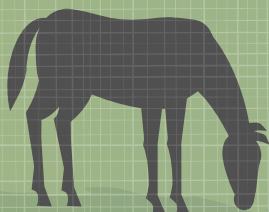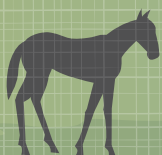### Championing the Equine Vet

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Is salmonellosis a problem in the UK? Real life experiences

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Salmonellosis is a recognised cause of infectious diarrhoea in the horse, usually resulting from enteric infection with *Salmonella enterica*, although various serovars have been implicated in the various disease outbreaks reported in the literature. Cases can occur sporadically or infection can become endemic, with extended hospital closures resulting from established nosocomial infections [1-6]. Consequently, consideration should be given not only to therapeutic interventions aimed at the clinical case, but also to interventions aimed at reducing the likelihood of disease transmission, particularly within a hospital environment. Such interventions require an understanding of the faecal–oral route of disease transmission, the risk factors for clinical disease and increased shedding and the recognised sources of infection (shedders, clinical cases and environmental contamination). One should also remain cognisant of the zoonotic potential of this enteric pathogen.

Three forms of salmonellosis are recognised: the subclinical carrier, mild clinical disease and acute diarrhoea. As the name suggests, the subclinical carrier is asymptomatic; however, it remains a potential source of direct transmission or environmental contamination. Recognition relies on the detection of the organism in the faeces, which, as shedding can be intermittent, will often necessitate the submission of multiple samples. Although the prevalence of shedding is low within the general horse population (~1%), this can significantly increase during periods of hospitalisation (up to 8%). Mild clinical disease is generally manifest as dullness, anorexia, fever and the passage of soft (cowpat) faeces. Although this mild clinical illness is generally self-limiting (4–5 days), bacterial shedding may continue for several days to months. Acute diarrhoea cases are characterised by severe dullness, anorexia, profound neutropenia, passage of watery diarrhoea and occasionally, abdominal pain. The severity of disease is usually reflected in the rapid development of hypovolaemia/dehydration, metabolic acidosis, electrolyte derangement and protein loss.

Recognised risk factors for salmonellosis include the following: presenting complaint of colic; nasogastric intubation; treatment with antibiotics; diagnosis of large colon impaction; withholding feed; number of days fed bran mash; treatment with potassium penicillin G; increase in mean daily ambient temperature; abdominal surgery; large intestinal evacuation and lavage; ileus [1,7-9]. Such associations are likely to reflect a change in gastrointestinal physiology and microflora and an alteration in host defence.

Although diagnostic *suspicion* is based on a combination of clinical signs (especially diarrhoea, fever) and blood work (severe neutropenia), *confirmation* relies on the detection of salmonellae in submitted samples; whole faecal samples are superior to rectal/faecal swabs. Detection can be achieved by culture or PCR, with PCR being considerably more sensitive and rapid. Irrespective of the detection method adopted, enrichment techniques are generally applied.

Prevention can be difficult for a variety of reasons; these include the environmental persistence of the bacteria, the unknown status of asymptomatic shedders and the inevitable periods of increased risk, especially within a hospital environment. Therefore, reliance is placed on adherence to general biosecurity practices (e.g. hand washing, glove wearing, boot cleaning, strategic use of foot baths), a continued awareness of the 'level of risk' for hospitalised patients, the timely recognition of cases/shedders and the strict isolation of such horses. Although isolation or barrier nursing should be applied to all horses confirmed as salmonellosis cases, such practices should also be applied to those unconfirmed cases with clinical signs consistent with this diagnosis (e.g. diarrhoea, fever, leucopenia), pending the availability of culture and/or PCR results. Furthermore, consideration should be given to the separate housing of horses considered as being at greater risk of being *Salmonella*-positive (e.g. those on long-term antibiotic treatment, gastrointestinal problems) with 'traffic control' and escalated biosecurity measures applied to these groups.

Although confirmation of nosocomial infection can be achieved through serotyping, antimicrobial susceptibility profiles, genotyping, plasmid profile analysis and phage typing, this is largely a retrospective exercise. More immediate detection, allowing the early implementation of infection control measures, relies on the application of a working case definition, pending serotyping data. Temporal and spatial overlap of cases is a strong indicator when the potential source of infection is another hospitalised horse; however, this has limited applicability when the potential source is environmental, with common sources including shared equipment, stall drains, rectal thermometers, vermin and mats in stalls and recovery rooms. Every effort should be made to minimise the opportunity for established environmental contamination as the organism can persist on porous/difficult to clean surfaces, withstand freezing temperatures, multiply in temperatures ranging from 7–45ºC and potentially survive for years under adverse environmental conditions. Factors which are likely to reduce the likelihood of nosocomial infections include the following: establishment and application of a 'suspect case' definition; day-to-day monitoring of potential nosocomial cases; routine environmental sampling; and the implementation of immediate 'enhanced infection control measures' upon case suspicion.

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