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with cattle, concurrent medications, and sharing a water source with other species of animals.

Discussion: Many of the findings in this study were consistent with previous reports. Foals less than 6 months of age were at highest risk of cryptosporidial cocyst shedding, and the presence of fecal oocysts was associated with diarrheal disease. Mares, however, did not seem to represent a source of infection for foals. In some cases, shedding was not associated with clinical disease, suggesting that sources of infection are not only limited to horses exhibiting clinical diarrhea.

There were strong associations between cryptosporidiosis and particular breeding farms, which confirms the role of *C. parvum* as a significant cause of foal diarrhea outbreaks. The specific sources of infection in these cases could not be identified, however; and the potential for *C. parvum* to cause outbreaks over consecutive foaling seasons on these farms could not be evaluated. The absence of *C. parvum* on other breeding farms suggests that *C. parvum* is not a ubiquitous pathogen, and is geographically localized to particular farms.

There were significant differences in the prevalence estimates depending upon the diagnostic test employed. The acid-fast stain exhibited good sensitivity, but suffered from poor specificity when compared to the IFA and flow cytometry.

Practitioners who care for foals need to consider *C. parvum* in all investigations of foal diarrhea. Although the overall mortality associated with outbreaks of cryptosporidial disease is usually low, it can result in significant financial losses associated with therapy and control. It is also zoonotic, and exposure to infected foals has resulted in human illness. Sources of infection may include water sources and other foals (even those not experiencing diarrhea), and these should be investigated. The acid-fast stain is simple to perform and is sensitive in detecting fecal oocysts. References available upon request.

Prevalence of β2-toxigenic *Clostridium perfringens* in horses with intestinal disorders

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The incidence of a new, yet unassigned toxin type of *C. perfringens* containing the α- and the recently described β2-toxin gene in horses with intestinal disorders is reported. Twenty samples of ingesta from the small and large intestine and five biopsies of the intestinal wall and 74 faeces samples were analysed bacteriologically of i) 18 horses suffering from typical typhlocolitis, 7 horses with atypical typhlocolitis and 16 horses with other intestinal disorders and ii) 58 horses without intestinal disease. *C. perfringens* isolates were typed for the presence of the α-, β1-, β2-, ε- and enterotoxin genes by PCR including a newly developed PCR for the detection of the β2-toxin gene *cpb2*. In samples from horses with typical or atypical typhlocolitis, β2-toxigenic *C. perfringens* were found in 9 of 12 (75%) ingesta specimen and biopsies and in 4 of 13 (31%) feces samples. From the 15 fatal cases of typical and atypical typhlocolitis 9 (60%) were positive for β2-toxigenic *C. perfringens* compared to only 4 (40%) of the 10 non fatal cases. Of the 9 animals with typhlocolitis which were treated with antibiotics in combination with non steroidal-antiinflammatory drug (NSAID) 8 had β2-toxigenic *C. perfringens* and died whereas of the remaining 16 cases not receiving this combination treatment only 7 died and 5 of them shed the organism ($p < 0.01$). No β2-toxigenic *C. perfringens* was found in the samples from the 58 control horses of which only one faeces sample contained *C. perfringens* type A. Surprisingly, no *C. perfringens* isolated in this study contained genes for the β1-, ε- and enterotoxin, indicating that *C. perfringens* type B, type C and type D and enterotoxigenic *C. perfringens* are not common in horses with intestinal disorders. The high incidence of β2-toxigenic *C. perfringens* in ingesta samples and biopsies of the intestinal wall of horses suffering or dying of typhlocolitis ($p < 0.001$) suggests that β2-toxigenic *C. perfringens* play an important role in the pathogenesis of typhlocolitis and may act as a lethal factor in toxic intestinal disorders in horses.

Muscle enzyme patterns and elimination of intravenous injected, homologous muscular enzymes in horses

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In myopathies of horses bloodchemistry shows often a rise in muscular enzyme activities. The purpose of this study was to get further information from bloodchemistry by determination of muscle enzyme patterns and elimination of intravenous injected, homologous muscular enzymes in horses.

Animals, material and methods: In liver, heart, diaphragm, M. masseter, M. glutaeus medius (superficial and deep part) and M. semitendinosus of 17 warm-blooded and small horses, the activities of CK, ASAT, ALD, LDH and HBDH were determined. Concentrations of haemoglobin and myoglobin were measured together. In addition, the kinetic behaviour of the cellular enzymes CK,

ASAT, ALD, LDH and HBDH were examined. The determination of the enzymes' elimination rates was performed on six horses (four adult and two young animals) after intravenous injection of a homologous extract from skeletal muscle and repeated blood sample collections.
Results: In homogenates of tissue significantly higher contents of glycolytic enzymes ALD and LDH were found in muscles with a higher percentage of type II B fibres (M. semitendinosus, M. glutaeus medius superficial part). The quotients of enzyme activities in muscles with a higher percentage of type I fibres (heart, diaphragm, M. masseter, M. glutaeus medius deep part) showed significant differences to muscles with predominant percentages of type II B fibres. Those with a higher content of type I fibres had significantly higher values of myoglobin and haemoglobin.
After intravenous injection of a muscle extract the elimination of the enzymes CK, ASAT, ALD, LDH and HBDH showed a two compartment kinetic. The half life periods of the enzymes were CK 9.07 ± 3.30 h, ASAT 58.28 ± 17.10 h, ALD 17.40 ± 2.67 h, LDH 7.65 ± 2.98 hand HBDH 12.76 ± 8.23 h. There were no significant differences between adult and young horses. Conclusions: The determined half life of CK in these studies is slower than those given in others (~ 2 h). Half life periods of ASAT, ALD, LDH and HBDH were measured for the first time in the present study.
The clinical use of the given results is discussed: While CK is useful for determination of the extent of muscular damage, measurement of ASAT and ALD enables the clinician to differentiate the fibre types that are mainly concerned. Enzyme quotients of ALD/ASAT > 0.2 show a predominant participation of type II B fibres while a quotient of ≤ 0.2 indicates damage of type I fibres. In this way exertional myopathies can be distinguished from nutritional and poisonous myopathies.