Failure of passive transfer in foals: incidence and outcome on four studs in New South Wales

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Objective To determine the regional incidence and effectiveness of treatment of failure of passive transfer (FPT) in foals.

Design A study of disease incidence.

Animals Eighty-eight foals and 57 mares from four studs in the practice area of the Rural Veterinary Centre were tested.

Procedure Foals were tested for their serum IgG and total serum protein (TSP) concentration within the first 72 hours of life. Colostrum was collected from mares and specific gravity determined. FPT and partial failure of passive transfer (PFPT) of immunoglobulins was diagnosed when serum IgG concentrations were < 4 g/L and 4 to 8 g/L respectively. Owners of foals diagnosed with FPT were offered treatment with 1 to 2 L plasma (TSP > 70 g/L); 9 (64%) of the affected foals were treated.

Results Fourteen foals (16%) had FPT whereas 15 (17%) had PFPT. There were significant differences between the mean TSP concentration in foals with FPT (42.6 ± 4.2 g/L), PFPT (48.1 ± 3.9 g/L) and those acquiring adequate passive immunity (58.9 ± 5.5 g/L) ($P < 0.01$). Sixteen (29%) mares had pre-suck colostral specific gravity < 1.060 and 12 (71%) foals raised by these mares had FPT or PFPT. The incidence of severe disease (categorised by a sepsis score > 11, positive culture of bacteria from blood or disease requiring hospitalisation) in all foals in the first 2 months of life was 10%. However, none of the nine foals with FPT that received plasma experienced severe disease. In contrast, foals with PFPT had an increased susceptibility to severe disease ($P < 0.001$) when compared with normal foals.

Conclusion Treatment of foals with FPT may reduce the subsequent incidence of severe disease. Pre-suck colostral specific gravity and foal TSP may be used to predict the likelihood of FPT and PFPT. Even though the number of foals studied is small the results highlight the importance of optimal management practices in reducing the incidence of FPT and disease associated with this process.

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Foals are born immunocompetent but immunologically naive due to the lack of placental transfer of immuno-globulins.[1] Although foals begin to produce immuno-globulins soon after exposure to antigens, protective concentrations of immunoglobulins may not be reached until two months of age.[1] Foals are therefore dependent on the ingestion and absorption of maternal antibodies, via colostrum, for the acquisition of immunity to equine pathogens. The majority of immunoglobulin in equine colostrum is IgG, which provides protection against infection for the first one to two months of life.[1] Thus, under normal circumstances, immunoglobulins derived from colostrum provide protection for the foal while its own immunoglobulins are being developed.

FPT of colostral immunoglobulins can occur due to a variety of maternal and foal factors including premature lactation, inadequate production of colostrum by the mare, delayed onset of sucking by the foal or inability of the foal to absorb colostrum.[2] Serum IgG concentrations of < 2 g/L have been used to diagnose FPT in foals at 24 to 48 hours after birth and a serum IgG concentration < 4 g/L considered indicative of PFPT.[3] However, it has been found that serum IgG concentrations of > 8 g/L may be required for adequate immune protection.[4] FPT has been shown to be the most important contributory cause of serious disease, particularly septicaemia, in foals.[5,6] Thus, methods of early detection and treatment of FPT have been used in an attempt to reduce the risk of disease in foals.[7]

There are reports of the incidence of FPT in Australia and other countries, however the incidence varies considerably between studies. A factor contributing to this variation may be the range of management practices observed in different regions due to financial and practical constraints. These include methods of prediction, early detection and treatment of FPT in foals. At the time of this study, management practices on studs in the area serviced by the RVC often differed from those recommended in the literature. In general, there was no testing of the mare's colostrum for prediction of the foal's IgG status nor storage of colostrum in colostrum banks for early treatment. In addition, routine testing of foals to determine immunoglobulin status within 10 hours of birth was not practiced. Thus, there was no prophylactic treatment with colostrum of those foals at increased risk for neonatal disease.

This study was therefore undertaken to determine the incidence of FPT on stud farms within the Camden region. The effect of treating FPT in foals with plasma for reducing the incidence of serious disease was also examined.

Materials and methods

Three large Thoroughbred studs and one Arabian stud partici-pated in the study. Studs were chosen because of similar management practices during foaling season and the accuracy of records kept for mares and foals. Studs provided information about the mare and foal in the perinatal period and about the health of foals during the first 2 months of life.

To determine the incidence of FPT, the IgG status of all foals born on each stud in the 1992 stud season was determined. Blood was collected from the foals between 12 and 72 hours of age (most between 18 to 36 hours). This time range was chosen as IgG concentrations peak about 18 hours of age.[8] A sample of pre-suck colostrum was collected from the mare at parturition by the farm manager and stored at 4°C at the stud until blood samples from the foals were collected by the veterinarian. All blood and colostrum samples were analysed on the day of collection.

Colostrum was collected from 57 mares and specific gravity was measured at room temperature using a colostrometer (Jorvet, Jorgensen Laboratories Inc, Loveland, CO, USA). Any samples which were suspected or known to be collected after the foal had sucked from the mare were excluded. Serum IgG was measured in foal serum using the GC test. The test involves the addition of 50µL of 10% glutaraldehyde to 0.5ml of serum and the serum is observed for clotting.[9] The GC test was used because of its low cost compared to the CITE Foal Test Kit (Agritech Systems Inc, Portland, ME, USA) and, its high specificity and sensitivity at critical IgG concentrations.[9,10] Failure of passive transfer was diagnosed in foals whose serum took over 60 minutes to clot (equivalent to an IgG concentration < 4 g/L). Partial failure of passive transfer was diagnosed if serum took between 10 and 60 minutes to clot (equivalent to an IgG concentration of 4 to 8 g/L). Normal transfer of colostral immunoglobulins was diagnosed in foals if their serum clotted before 10 minutes (equivalent

to an IgG concentration > 8g/L). TSP concentration was measured using a refractometer (American Optical Co, Buffalo, NY, USA).

To determine the effectiveness of treatment in reducing the incidence of severe disease in foals diagnosed with FPT, owners or stud managers were given the option to treat affected foals with infusions of plasma. Treatment consisted of a slow transfusion of 1 to 2 L of thawed plasma. Plasma was collected from Standardbred geldings (universal blood donors) with a TSP concentration greater than 70 g/L. Blood was collected from the geldings into 3 L bags with acid citrate dextrose as the anticoagulant. Red cells were left to settle by gravity over 12 to 24 hours then plasma was harvested and stored at -20°C until required.

The health status of foals from birth to approximately 2 months of age was examined retrospectively by review of stud and RVC hospital records. Foals were diagnosed as having mild disease if they were considered ill and successfully treated at the stud by the manager or if they had a sepsis score less than 11.[11] Of the mild diseases, gastrointestinal disease was most commonly observed, particularly diarrhoea. Other disorders included colic or non-specific illness which resolved quickly with conservative management. Foals were diagnosed as having severe disease if they had a sepsis score greater than or equal to 11 or confirmed sepsis on the basis of blood culture results.[11]

The disease incidence in foals considered to have FPT, PFPT or normal transfer of immunity was compared using chi-squared analysis or, where there were small numbers involved, Fisher's exact probability. The disease incidence in treated and untreated foals with FPT and the incidence of FPT and PFPT in foals raised by mares with colostral specific gravities above and below 1.060 were similarly compared. Mean TSP was compared between groups of foals using one way analysis of variance. Where F values were significant, a post hoc least significant difference test was performed to obtain P values. Results were considered significant when $P < 0.05$.

Results

Serum IgG and protein concentration was measured in 88 foals born between August 4 and December 20, 1992. Fourteen foals (16%) had FPT and 15 (17%) had PFPT. The mean TSP in foals with FPT was 42.6 ± 4.2 g/L (range 35 to 49 g/L), and 48.1 ± 3.9 (range 41 to 56 g/L) in foals with PFPT. The mean TSP of foals with normal transfer of immunity (n = 59) was 58.9 ± 5.5 g/L (range 47 to 70 g/L). Values for TSP in foals with FPT and PFPT were lower than those in normal foals ($P < 0.01$).

Foals with PFPT had a significantly higher ($P < 0.01$) incidence of severe disease than normal foals (Table 1). In contrast, foals with FPT did not have a statistically higher incidence of severe disease than normal foals ($P = 0.06$) (Table 1). The incidence of mild disease was not significantly different between normal foals and foals with FPT or PFPT.

Nine foals with FPT and one with PFPT received a plasma transfusion. None of the treated foals developed severe disease in the first 2 months of life, but this was not statistically different from untreated foals ($P = 0.17$) because of the low number of foals involved (Table 2). There was also no significant difference between the incidence of mild disease in the treated versus untreated foals ($P = 0.14$).

The mean specific gravity of colostrum samples was 1.064 ± 0.016 (range 1.030 to 1.100). Foals raised by mares with pre-suck colostrum < 1.060 were more likely to have serum IgG < 8 g/L ($P < 0.001$) than foals raised by mares with pre-suck colostrum greater than or equal to

1.060. Sixteen mares had pre-suck colostral specific gravity < 1.060 while 31 mares had colostral specific gravity greater than or equal to 1.060. Twelve foals raised by mares with pre-suck colostrum < 1.060 had serum IgG < 8 g/L, while only seven foals raised by mares with pre-suck colostrum greater than or equal to 1.060 had serum IgG < 8 g/L.

Table 1. Immunoglobulin status of foals and susceptibility to mild and severe disease.

	No disease	Mild disease	Severe disease	Total
IgG > 8	40	17	2	59
IgG 4 to 8	8	2	5	15
IgG < 4	7	5	2	14
Total	55	24	9	88

Table 2. Effect of treatment of failure of passive transfer of immunity on the susceptibility to mild and severe disease.

	No disease	Mild disease	Severe disease	Total
Treated	5	4	0	9
Untreated	2	1	2	5
Total	7	5	2	14

Discussion

The reported incidence of FPT (IgG < 4 g/L) in foals is variable and ranges from 2.9%[12] to 24%.[3] More recently the incidence was estimated to be about 13% in the USA [13] and 15% in the UK.[14] Australian studies have described an incidence of FPT of approximately 10%,[15,16] however, one study did not include any foals that were abnormal at birth[15] and the other employed early, speculative treatment of foals at risk of developing FPT.[16] In the present study an incidence of FPT of 16% and PFPT of 17% was found. Several factors could cause this disparity with the reported incidence of FPT including different tests used to measure IgG concentrations, different reference ranges, age of foal when tested and management procedures used.

Although most of the commonly used methods for screening serum for the concentration of IgG are comparable,[10] interpretation of results vary depending on the reference ranges used for serum IgG concentrations. Earlier studies diagnosed FPT when serum IgG was < 2 g/L and PFPT when serum IgG was < 4 g/L3 while later studies used < 4 and < 8 g/L respectively.[12-14] This difference in interpretation would explain some of the apparent variation in reported incidence of FPT and PFPT. The age of foals at the time of testing will also affect incidence. A common recommendation is to test foals at 18 to 24 hours of age as peak IgG concentrations occur at this time7 and testing foals before this time could result in a greater apparent incidence of FPT. However, this suggestion has not been confirmed.

More importantly, specific management practices may influence the incidence of FPT on studs, especially the administration of colostrum to foals at increased risk for developing FPT. For example, Morris and colleagues[12] report an incidence of FPT of only 2.9% when colostrum was administered by bottle or tube within 3 hours of birth if there were any difficulties or delays in drinking by the foal. Similarly, two well-managed studs in the Hunter Valley region of Australia have an incidence of FPT of 1.5% and 3% respectively in foals 24 hours of age (AP Begg personal communication). All foals from these studs had received

adequate amounts of good quality colostrum within the first 12 hours after birth. It has been shown that when intake of colostrum is maximised by similar management practices, there was a statistically significant reduction in the incidence of FPT.[14]

Failure of passive transfer of immunity has been suggested as one of the leading contributory causes of neonatal infection and disease.[4,5] Increased risk of bacterial infection is associated with serum IgG < 4 g/L, while IgG > 8 g/L is considered satisfactory to ensure protection against most environmental pathogens.[7] The significance of serum IgG concentrations between 4 and 8 g/L is dependant on other factors including management practices at the stud and perinatal stresses such as asphyxiation.[7,16] In the present study, IgG < 8 g/L (PFPT) was associated with a higher incidence of severe neonatal disease, but IgG < 4g/L was not. The reason for this apparent contradiction in results may be the treatment with plasma transfusion of foals with FPT and not those with PFPT. No severe disease occurred in any treated foals but of the five untreated foals, two developed severe disease. Though the results were not statistically significant (presumably due to the low numbers of foals involved), the data suggests that plasma transfusion is effective in reducing the incidence of severe disease in foals by improving serum IgG concentrations and therefore immunity.

Management practices were probably contributory to the high incidence of FPT and severe disease in foals reported in this study in comparison to other studies. It should be noted that none of the studs were routinely administering colostrum to foals known to have delays or difficulties in sucking. Routine hygiene and care of foals was similar on all four studs in most aspects except that one stud (stud 1) gave prophylactic antibiotics (gentamicin sulphate) to all foals born. This stud had 5 out of 19 foals with severe disease (26%) compared with 2 out of 39 (stud 2), 2 out of 25 (stud 3) and none out of 5 foals (stud 4) (average of 4.3%) for the other three studs (Table 3). Prophylactic antibiotics have been associated with a higher percentage of foals developing diarrhoea[17] and septicaemia.[18]

TSP concentration has been found to be an inaccurate measure of IgG status in foals due to the wide variation of serum protein concentration observed in foals with normal IgG concentrations and changes in serum protein associated with hydration status.[1,15] However, in this study protein concentration was significantly different between FPT, PFPT and normal foals. While TSP is not able to accurately differentiate between FPT and PFPT, we suggest that any foal after 18 hours of age with TSP < 47 g/L is highly likely to have FPT or PFPT. Therefore, total serum protein may a useful preliminary guide to IgG concentration for veterinarians who do not have any other means of assessing IgG status.

Colostral specific gravity values have been correlated with Ig status of the foal and assessment of pre-suckle colostrum is useful for prediction of FPT in foals.[13,18] A colostral specific gravity value < 1.060 has been shown to increase the likelihood of FPT in foals.[12,18] In the present study, 16 mares had colostral specific gravity < 1.060 and 12 of these mares raised foals with FPT or PFPT. Thus, our results are similar to those reported by LeBlanc and colleagues[18] and help confirm the relationship between colostral specific gravity and FPT.

The findings of this study highlight the importance of optimal management practices to reduce both the incidence of FPT and PFPT and associated severe disease. The study showed that foals with PFPT were at increased risk of developing severe disease and that foals with FPT were also likely to be at risk. We believe that treatment of foals diagnosed with FPT reduced the incidence of severe disease, however further studies using larger numbers of foals are required to statistically prove the benefits of treatment of FPT.

Table 3. Incidence of failure of passive transfer and partial failure of passive transfer of immunity and the incidence of sever disease foals: inter-stud variation.

	Stud 1	Stud 2	Stud 3	Stud 4
IgG > 8	11	26	19	3
IgG 4 to 8	6	6	2	1
IgG < 4	2	7	4	1
Severe disease	5	2	2	0
Total	19	39	25	5

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