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Calf Note #137 – Failure of passive transfer – effect of the calf

Introduction

Everyone who raises calves has a major stake in how well calves obtain passive immunity from the dam during the first 24 hours of life. Calves receive passive immunity via intake of colostrum during the first 24 hours when their intestines are able to absorb immune proteins without digestion. When this process fails, calves are more susceptible to disease, grow more slowly, and may make less milk as adults. Everyone in the dairy production chain lives with the ramifications of poor passive transfer when it occurs.

Dairy professionals generally use the term “failure of passive transfer” (**FPT**) to define the condition when calves (or certain other newborn animals such as foals and piglets) don’t receive enough colostral immunity from the cow. The specific condition is defined in various ways by various professionals within species. In the cattle industry, a common criterion to define FPT is when calves have a serum (or plasma) IgG concentration less than 10 grams per liter at 24 hours of age.

So, what causes FPT and why? The causes for FPT have generally focused on the cow – particularly in the quality of colostrum she produces. This makes sense, as the quality and quantity of colostrum have a big effect on IgG concentration in calves as can be seen in the following equation:

Plasma IgG (g/L) = colostrum quality (g/L) × colostrum intake (L) × apparent efficiency of absorption (**AEA**) / plasma volume (L)

The most common cause of FPT (and the most easily recognized) is a lack of sufficient IgG intake. The IgG intake is a function of the quality of colostrum (IgG in colostrum, grams/liter) × the quantity of colostrum fed (liters). So, if a calf is fed 4 L of poor quality colostrum (e.g., 25 g of IgG/L), then the calf will consume only 100 grams of IgG (4 × 25) in the first 24 hours.

However, what other factors affect FPT and what role does the calf have in all of this? We’ll use two different studies to look at this interesting question.

In the first study, Swan et al. (2007) fed 239 calves maternal colostrum either from the dam or fresh colostrum from another cow.

Calves were born on 12 dairy farms in Minnesota and Wisconsin during 2003. Most farms fed 3.8 L (1 gallon) of colostrum in the first feeding and one farm fed 1.9 L (2 quarts) in the first feeding. Seven of the farms also fed a second feeding of 1.9 L in a second feeding of colostrum approximately 12 hours after the first. After feeding colostrum, calves were fed commercial milk replacer. Note that this study was designed to evaluate a colostrum replacer product – for the purposes of this Calf Note, we’re only going to look at the control calves fed colostrum.

The average time of first feeding of the calves on these dairies was 1.0 hr, with a range of 0.2 hr to 9.0 hr. About half the farms fed calves by nipple bottles; the other half used esophageal feeders to administer colostrum. The colostrum quality (IgG content) ranged from 9 to 186 g of IgG/L with an average of 77 g/L. Intake averaged 292 grams of IgG in the first feeding (3.8 L), which is an impressive amount of immunoglobulins and should have resulted in very low rates of FPT.

The serum IgG content in calves at 24 hours averaged 14.6 g/L and ranged from 2 to 39 g/L. Interestingly, 67 of the 239 calves (28%) had FPT (serum IgG concentration < 10 g/L) which is much higher than would be expected, based on the early rate of feeding and large amount of colostrum (and IgG) administered. To summarize, though these farms were doing things very well (i.e., feeding early) and feeding large amounts of IgG, many calves STILL had FPT.

Well, one obvious possible explanation for the number of calves with FPT could be the variation in IgG intake. In the study by Swan, at least some of the calves would have consumed only a small amount of IgG if their dam's colostrum was poor quality.

That leads us to the next study, which was conducted at Virginia Tech. In this study, 39 Holstein and Jersey calves were fed pooled colostrum as soon as possible after birth. The colostrum had been previously collected, tested to be high quality (i.e., high IgG content) and pooled so that every calf received the same amount of IgG (and other proteins) from the colostrum.

Unlike the previous study, this study was conducted on one farm, so management factors (age at first feeding, etc.) could be removed. All calves were fed at 1.5 and 13.5 h of age to provide 250 (Holstein) or 180 (Jersey) grams of IgG in the first 24 hours of life. Calves were all fed by nipple bottle and a tube feeder was used only when calves refused to nurse all colostrum. Blood samples were taken precisely at 24 hours of age to minimize effects of time of blood collection on serum IgG concentration.

These researchers removed essentially all of the variation associated with colostrum quality, time of feeding and farm to farm management. So, we might expect that all calves fed the colostrum would have successful passive transfer, since they consumed a large amount of IgG. Let's assume that these calves had an AEA of 30% (typical of that reported in the scientific literature) and the Holsteins weighed 40 kg and the Jerseys weighed 30 kg. Then, for Holsteins:

$$\begin{aligned}\text{Plasma IgG} &= \text{g IgG} \times \text{AEA} / \text{volume}; \\ &= 250 \times 30\% / (40 \times 9\%) \\ &= 20.8\end{aligned}$$

For Jerseys, the same calculation leads to 20.0 g/L at 24 hours of age (assuming 9% PV).

The results showed remarkable variability. In this study, 21% of the calves fed colostrum had FPT, even though their plasma IgG concentrations should have been about 2× that amount. Interestingly, none of the Jersey calves fed colostrum had FPT; about 31% of Holsteins were reported to have FPT (in both treatments in the study).

So, what's going on? Why do some calves absorb so little IgG? Well, there are several possible factors that can affect the calf's ability to absorb IgG. The most obvious factor is the time after feeding. We assume that AEA declines as calves get older; this means that calves fed colostrum at 12 h of age will absorb less IgG from colostrum than calves fed the same colostrum at 1 h of age.

Of course, in the Jones study, all calves were fed at the same time, so age didn't affect the proportion of calves with FPT. There are other factors which can affect the calf's ability to absorb IgG – such as their metabolic state. Calves that have a difficult birth may be less able to absorb IgG from the gut than other calves. Another possible factor is the calving environment (dirty vs. clean) – if calves swallow feces or bedding contaminated with bacteria, this may inhibit IgG absorption also.

These two studies suggest that there will be a proportion of calves on dairies that – even if fed large amounts of high quality colostrum shortly after birth – will be unable to absorb enough IgG and will have FPT. It is still unclear (at least to me!) what this percentage is. The Swan study suggested 28% and the Jones study reported 21%. Other studies have reported higher or lower numbers. For example, a recent Danish study (Jacobsen et al., 2002) reported 0% FPT in calves fed 40 ml of colostrum/kg of body weight 3 times in 24 hours. Another recent Canadian study also reported low rates of FPT in calves in Ontario (Trotz-Williams et al., 2008). However, these studies had much less control compared to the above studies and/or measured other indices of passive transfer (e.g., total protein) which may impart added variability in the results.

The bottom line of these two studies is this – although we can certainly influence the percent of calves with FPT by feeding increasing amounts of colostrum, there may be a certain proportion of calves that absorb little IgG due to their physiological state at birth. These calves will require special care throughout their young lives.

References

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