CLA's-A modern view of Milk fat test depression for the layman

The Term CLA is short for conjugated Linoleic acids. These are the "new buzz words" in milk fat depression. They produce milk fat depression by a different means than the old way that most of us are familiar with.

The old story with milk fat depression was centred on a lack of fibre digestion, which in turn produced less acetate from the rumen and thus less milk fat (acetate being a precursor of milk fat).
We were taught we could do this by feeding big licks of grain or starchy ingredients and dropping rumen pH, which killed off the fibre digesting bugs.
We could also do it by feeding too much fat into the rumen which it was supposed would also disrupt fibre digestion.

In Australia we saw the first incidence (starch) pretty regularly, but the second (fat) not very often.

That is the "old" thinking in a nutshell. The new thinking says that this is not the only way we depress fat test/yield. It still works to a degree, but there is more to it than that.

More recent research on low milk fat yield shows that it wasn't so much lack of acetate from fibre digestion that was giving lower fat test rather increased amounts of propionate from starch digestion that were driving extra volume of milk and extra protein test. IE we were just diluting the same total amount of milk fat per day over more litres.

Maybe we didn't have a milk fat production problem? Maybe it was just "dilution"?

So people said, "Well does diet really have an impact or is it all just dilution over more volume?"

The answer was yes, diet still had a big impact. The diets that produced depressed fat test in the past still did depress total fat yield… they just did it by a different way to how we thought it worked. It isn't based around fibre digestion and acetate as much as we thought. It is based around fats in the diet and how they are processed in the rumen.

Fats in the diet go into the rumen. Normally these fats are Poly-unsaturated-fatty – acids (PUFA) otherwise known as vegetable oils, and they are comprised of two main fatty acids, Linoleic acid (grains,oilseeds,maize silage) and alpha Linolenic acid (pastures).

These PUFA's are toxic to rumen bacteria and as such need to be processed in the rumen and converted from veg oils to animal fat via a process called Biohydrogenation. This process takes time in the rumen to be completed. If we are

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feeding cows high dry matter intakes, the dwell time of tucker in the rumen is greatly shortened, so the process is only partially completed.
Instead of being fully converted to saturated animal fat we get an intermediate product called Conjugated Linoleic Acids (CLAs).
If we put higher levels of veg fats into the rumen, we make it even harder for the rumen to process the veg fats, and we get even more CLAs.

These CLAs move out of the rumen to the intestine- and are absorbed into the blood.

If they were normal saturated animal fat then this would probably be a good thing for milk fat yield…. but some of these CLAs have a really big negative impact on the mammary gland where a large part of the milk fat is produced and deposited into milk.
Not all CLAs are going to impact on fat test negatively. Just some of them!
The type produced is important in depressing fat yield. Diet plays a major role on the type produced.

If we make lots of CLAs from lots of fat in the diet, and specifically if we do this at a lowered rumen pH, we produce a specific type of CLA that is called Trans-10 CLA.

This Trans-10 type of CLA is the one that has a massive impact on the mammary gland and will stop the mammary gland producing milk fat at normal levels.

This is why low rumen pH produces low milk fat test…it's the Terans-10 CLA's.

All this relates back to our new grass species. Our new grasses, especially when we use nitrogen generously, have fat contents of up to 6%. Our old idea of fat in grass was about 2-3%, so it's heaps more then we (or the cows!) are used to.

So we now have heaps of fat going into the rumen…one of the preconditions for CLA production.

At the same time these grasses are being eaten in bigger intakes per cow per day.
The grasses themselves have low effective fibre, low cud chewing and less saliva production.
So we also have high turn over of feed in the rumen and low rumen pH as well.

Then add a bit of fast fermenting winter cereal grain like wheat or triticale or barley producing VFA that pushes the pH down a bit more.

With these conditions we have plenty of CLAs produced, and sadly the conditions to produce the trans-10 CLAs which depress milk fat production.

Preconditions:

1/ Higher fat in diet into rumen >> Rumen struggles to completely biohydrogenate the fat

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2/ High rumen outflows>> rumen struggles to completely biohydrogenate the fat
3/ Low pH >> The type of CLAs produced favours more Trans-10 CLAs which bad for milk fat.

So there you have it! Why milk fat is probably depressed on modern diets of premium pasture plus grain.

The silly thing is that the control mechanism for this process is the same thing that we have done for years to keep fat test up when we thought it was all about fibre digestion i.e. DON'T LET YOUR RUMEN pH CRASH.

So, same preventative action but for a slightly different reason.

- use your buffers
- keep effective fibre present to aid salivation (not add fibre)
- be aware of how we offer and feed fast fermenting grain

Funny thing is that some of the CLAs from milk have been shown to have really amazing positive results on human health, maybe prevention for cancer! So CLAs in general maybe a good thing in milk in the future, and we may want our milk fat to contain CLAs.

The thing is we probably won't want the Trans-10 ones that depress overall milk fat. We will probably want a normal fat test, but with the other types of CLAs that bring the good health benefits.

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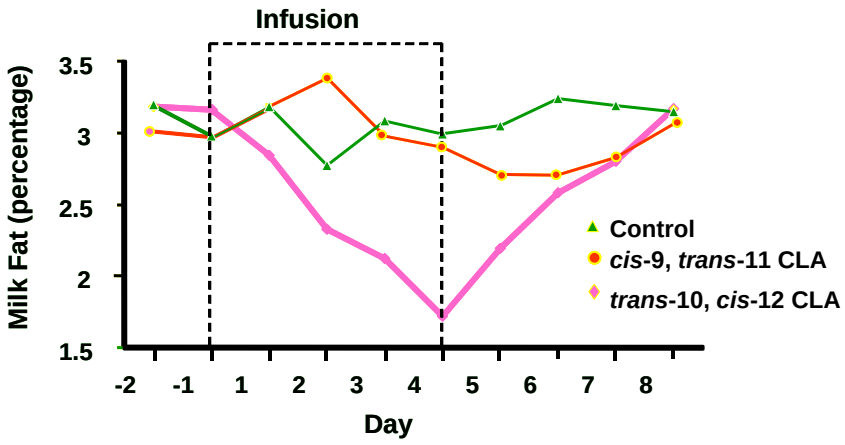
Effect of CLA Isomers on Milk Fat



Baumgard et al., 2000

Graph#1: The importance of the type of CLA we produce in the rumen. Cows normally make the cis 9:trans11 CLAs in a rumen doing normal things.
Note the very large drop in fat that occurs when trans 10 isomers of CLA are present

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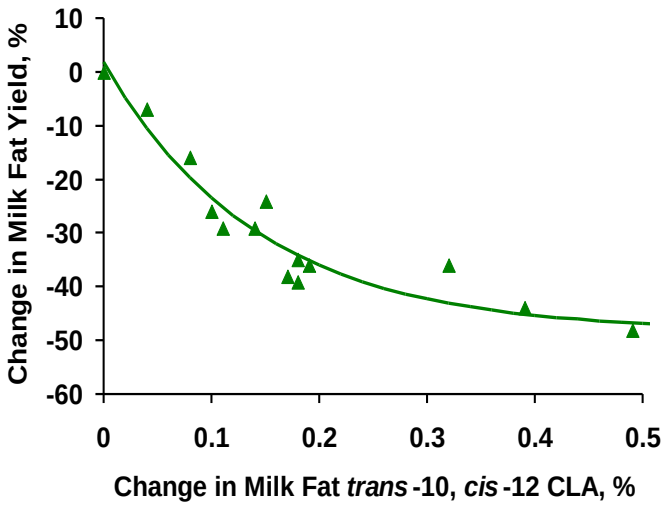
trans-10, *cis*-12 CLA in Milk Fat



Adapted from de Veth et al., 2004

Graph #2 The change in milk fat yield with increasing dose of trans 10 CLA. Note that fat test is depressed up to 30% by doses of CLA that may occur in the real world. This is what we see in practice as well

Further points around what impact incomplete biohydrogenation has on milk fat depression.

We have already discussed the impact of unsaturated fat in the diet, along with changed ruminal circumstances, leading to production of specific "trans 10 isomers of CLA" that in turn depress milk fat synthesis in the mammary gland.

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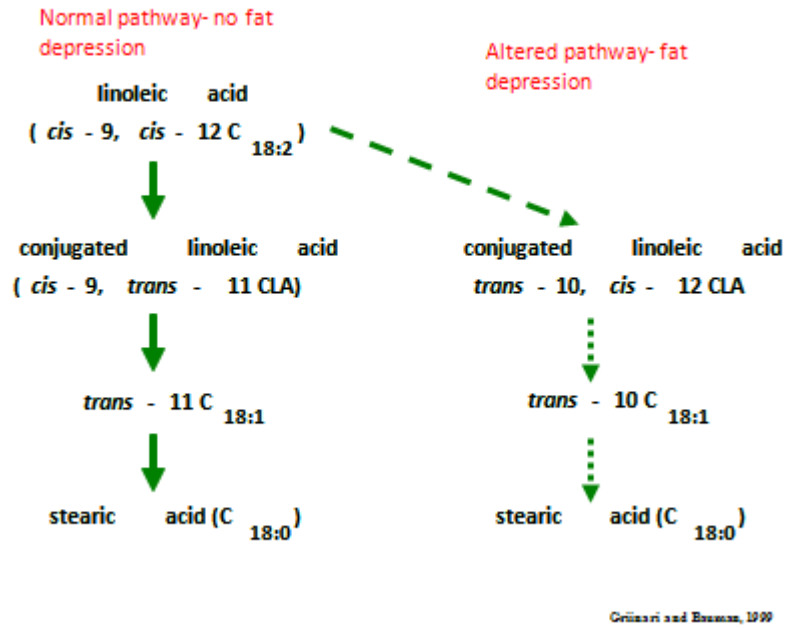

The two areas that we focussed on were

1- High PUFA diets as presented by the lipid content of modern rye grasses. This gives us the PUFA as the precursor of the CLAs. It also appears to be a direct impact on triggering the Trans10 CLA "flip" that depresses fat yield, as in itself it alters the bugs that grow in the rumen , in similar way to SARA impacting bug populations and changing biohydrogenation pathways
2- SARA as the key trigger to change the rumen conditions and change the pathway of biohydrogenation from normal (no milk fat depression) to Trans 10 pathway (milk fat depression)

More recent papers have highlighted the importance of ionaphores, especially monensin, in this process.

If we look at the process we see that we have three areas that we can possibly manipulate that will make the impact on fat yield worse or better. Ionaphores will impact two of the three

1- The amount of PUFA being consumed. Very hard to manage in our grazing systems. Largely beyond our control
2- The change in fermentation ie the flip to the problem trans 10 CLA
3- The new one , is the clearance of the problematic Tran s10 CLAs to fully saturated fat as 18:0

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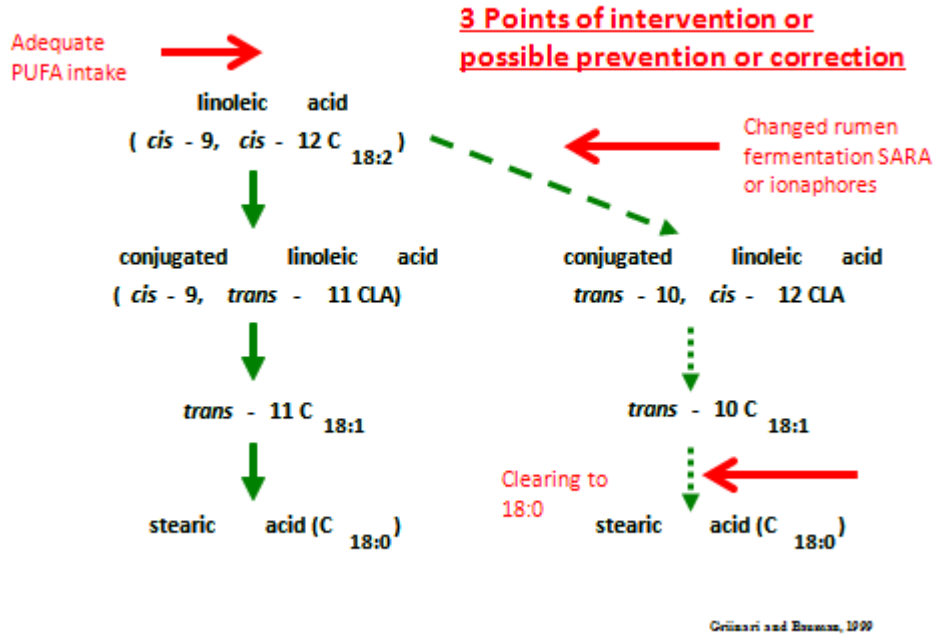

Ionaphores impact is on the two areas above ie changed rumen fermentation that leads to the initiation of the trans10 CLAs to begin with, and then also in preventing the clearance of the Trans10 CLAs to 18:0 saturated fat.

The work of Jenkins 2003 demonstrates four things from the table below

1- A control diet will have low trans10 CLA yield. Only about 1g/100g PUFA. NB it only takes about 3-5g of Trans10 CLA to cause milk fat depression
2- Addition of fat alone (as soyabean oil for PUFA) will promote more trans10 CLAs. It provides more precursors, and also promotes the trans10 Flip
3- Addition of monensin alone will also promote more trans10CLA. It alters rumen conditions , promotes the trans 10 Flip and then also impedes the full processing of vegetable fats to saturated 18:0 animal fats
4- Addition of both fat and monensin is even worse.

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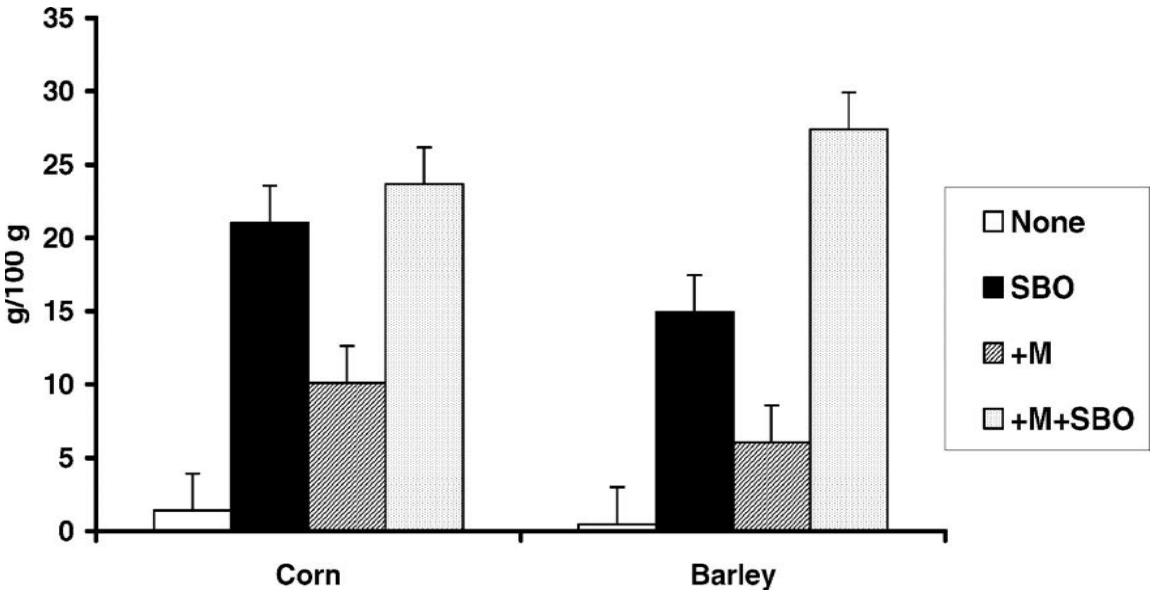

Monensin has negative impacts at the point of altering rumen fermentation to favour Trans10 CLAs, but also it will make it harder for the rumen to clear the trans 10 CLAs once formed.

The step of hydrogenating the 18:1 fatty acid is a rate limiting step wether the cow is in normal or altered Biohydrogenation pathways. Getting from 18:1 to 18:0 is the slow bit. It is carried out by a specific and small group of gram +ve bacteria. Without these guys we don't get to 18:0, and the trans10 CLAs build up

Monensin kills many of the small group of bugs that move 18:1 to 18:0. Less of these guys means less fat moved completely to 18:0, more building up as 18:1 or 18:2

It means we tend to accumulate the intermediate 18:1 and 18:2 fats in either pathway. If are on the left side accumulating them means very little. It wont depress fat yield.

Accumulating them on the right hand side pathway is bad though, they are the trans 10 isomers, the ones that depress fat synthesis and yield.

So , monensin means we are making more of them at the top of the chart , and making it harder to get rid of them at the bottom.

They accumulate as Trans 10 CLAs and we get fat depression

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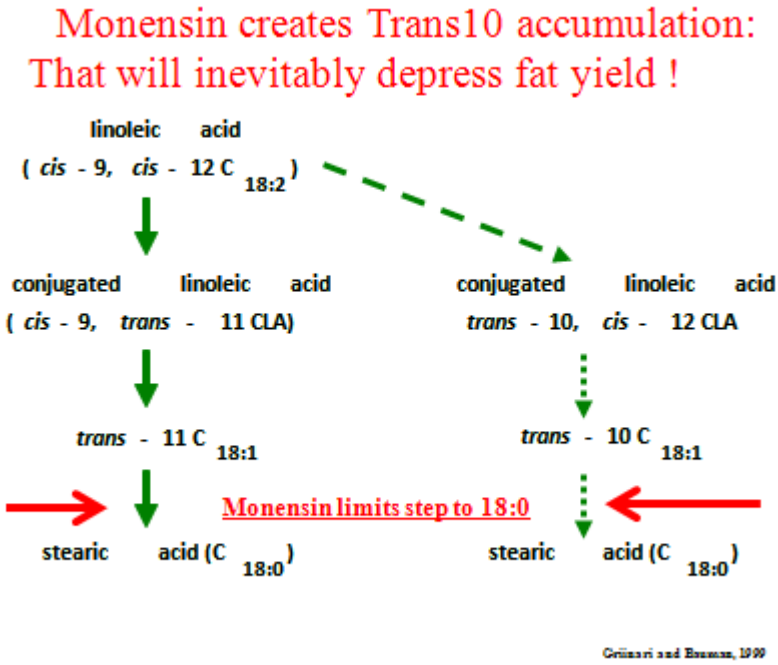

Implications of this:

It is possible to have monensin in the diet, and not have fat depression. This occurs when PUFA intake is lower-, the rumen is pretty stable and lower trans10 CLA yield occurs as we are not flipped from normal to altered pathways.

However, in times when the risk is higher-, monensin increases both the risk of the pathway flipping, and also the impact on fat yield when it does flip.

There seems to be some implications for “prevention vs cure” as well.

Prevention can focus on limiting PUFA intake if possible (though hard), and on maintaining a non-altered rumen setup so we don’t “flip” to altered pathway of fat processing on the right. That aspect of SARA has been the chief area of possible control to date.

This new info on monensin suggests that not using monensin will also assist prevention approaches by having:

1- less chance of changing rumen set up , and thus flipping pathways
2- also a better chance of clearing the Trans10 CLA faster if we do flip.

Cure is harder, as once the Trans10 CLA accumulate we must not only prevent future production (reset the rumen) but we must clear the existing production.

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Anecdotally, it is very hard to do this with ionaphores/monensin still in the diet. You may be better off removing the monensin for a period, as that is likely to assist the cure.

Monensin research (including the USA registration work) suggests clearly that milk yield outright is not promoted by monensin.

Rather, it is a FCE response (same solids yield from a bit less feed intake) that is the outcome. Given this we suggest that such a strategy to correct milk fat yield has limited down side beyond bloat management as you won't be losing milk.

Alternatively, consider products that promote milk yield, but do not promote Trans10 CLA production as monensin does.

Diamond V is a classic example. It promotes propionate yields above monensin, so provides the positives… without the down side of monensin and milk fat depression that is now clearly shown in independent university research.

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