## FAT FEEDING FACTS 13

# Critical Questions About Feeding High Palmitic Fat Supplements

Previously, issues about feeding fat supplements were addressed in *Fat Feeding Facts 1 – Should We Feed Palmitic, Stearic, or both in Fat Supplements?;* and in *Fat Feeding Facts 10 – High Palmitic Fed in Early Lactation.* A September 9, 2019 Hoard's Dairyman Webinar on Supplemental Fatty Acids raised a number of questions on some key issues about feeding high palmitic fatty acid supplements which are addressed below.

**References and Studies.** Scientists should acknowledge other research, especially when it differs from their own findings. All references cited in this webinar were from the author's studies. A listing of cited reference distribution is: 5 from the Journal of Dairy Science (includes 2 which the author indicated were published recently although cited on his slides as In Press – 7 slides cited these articles); 9 from ADSA abstracts over a period of 3 years with 5 slides from 1 abstract alone (abstracts are not peer-reviewed, contain limited description and data, and the abstract numerical designations were not listed for reference so it is uncertain if some of these slides were from different studies in the same abstract year; 1 regional nutrition conference—not peer-reviewed, and 2 cited as from unpublished sources. Thus, the preponderance of studies shown were from non-peer reviewed sources—and all from the author's studies.

**Milk Fat % increase.** Most studies done with high palmitic (>80%) supplements show an increase of about 0.1 to 0.3% milk fat. But a regression of palmitic intake (range of 0.2 to 2 lb/cow/day) only had an R2 of 0.34. That means the regression only represented about one-third of variation. What were the sources of the other two-thirds variation?

**NDF and total fatty acid digestibility.** This area of fatty acid impact on NDF digestibility is at best debatable and not corroborated by any other research group. There are questions about NDF and fatty acid assay methodology, possible mode (s) of action, and only about 50% of the variation (R2 = 0.54) was accounted for by palmitic intake and NDF digestibility. What accounts for the other 50% variation? What would this regression look like with other C16 and C18 fatty acids?

**Ruminal Biohydrogenation of Oleic.** The author did not acknowledge the extent of ruminal biohydrogenation of oleic to stearic, and how much oleic survives the rumen. Several studies (Harvatine and Allen 2006, Jenkins and Bridges 2007) indicate only about 15 to 25% of dietary oleic survives ruminal biohydrogenation to stearic. If 20 to 60 g oleic post-ruminal is optimal, feeding 100 to 300 g oleic would be needed to provide that amount post-ruminal. How would you do that? Calcium salts have been shown to not protect against ruminal biohydrogenation.

**Length of Studies and Stage of Lactation.** Many of the author's studies have been 21-day periods with md-lactation cows. One study was headlined as a Long Term study. Five weeks is not long term. It is longer term than the various 21-day studies. Another study was billed as a longer-term study of 10 weeks, but not of 15 or 16 consecutive weeks which would cover the first third of a 305-day lactation. Typical of many of the author's studies, why does he not include another fat supplement treatment so the study is not simply no-fat vs C16:0? That would have provided more comparative fatty acid supplement data. The complementary effects of palmitic and stearic fatty acids have been reviewed by Loften et al., (2014).

**Fat Supplement Treatments in Studies.** In a key study addressed in 9 slides, why was there not another fat supplement treatment so the study was not simply no-fat vs palmitic? That would have provided more comparative fatty acid supplement data.

**Oleic Fatty Acid Supplementation.** In one key study, why was not DMI shown? Reviewing the published paper revealed the palmitic plus oleic treatment had lower DMI than the other two fat supplements. Why? Seven slides covered a 2018 ADSA abstract study which used decreasing palmitic/increasing oleic blends. Data were interpreted that increasing oleic was more beneficial. But since ~80% of oleic is biohydrogenated in the rumen to stearic, it can also be interpreted that the benefits were due to stearic—not to oleic per se. Furthermore, another interpretation is that decreasing palmitic was the reason for beneficial responses. Why would that be?

**Author "Recommendation was: consider use of FA supplements containing C16:0 and C18:1."** After an end-of-webinar question from an attendee, the author reinforced that recommendation. When all of these studies were done by the author's research group, and that recommendation reflects some of the sources of funding, that is a scientific red flag.

**Why do high palmitic fatty acid supplements consistently decrease dry matter intake?** The author contended that only in maybe one of their studies did they see this DMI decrease. However, a review of palmitic treatments vs non-fat controls in various of the author's studies yielded negative DMI from high palmitic supplementation 9 times, positive DMI 4 times, and no effect on DMI 6 times. There are also 2 Penn State studies which showed negative effects on DMI with high palmitic supplementation. In addition, a 2017 meta-analysis (granted it is in an abstract by Sellers et al.) found an average DMI decrease of 1.2 lb per cow daily for palmitic vs non-fat control. At the May 2019 ADSA DISCOVER Conference, a question was raised as to whether decreased DMI with calcium salts of fatty acids—CSFA (which most commonly use palm fatty acid distillate—PFAD) might be due to linoleic, palmitoleic, or palmitic levels? In a review (Allen 2000), which was then incorporated into the 2001 Dairy NRC, CSFA decreased DMI by 2.5% for each percentage unit in the diet above the control. As high palmitic supplements also decrease DMI, maybe the decreased DMI with CSFA is also due to the higher palmitic fatty acid content in PFAD? A study (Davis et al., 2017) by West Virginia and Michigan State Universities (including this webinar author) had found that higher palmitic may increase ceramide formation and affect insulin; and these may be related to mechanisms for high palmitic decreasing DMI.

**What About NEFAs?** When cows maintain milk production in the face of decreased DMI due to fat supplements which decrease DMI, they typically do that by mobilizing body condition which increases blood NEFAs. That occurred with a study using CSFA (Harvatine and Allen 2005). And in a few of the author's studies where NEFAs were measured, that also occurred to varying degrees, including in earlier lactation (Fat Feeding Facts 12). Cows need to replenish lost body condition at some point.

**Nature of TMR and basal fat levels.** A question was raised at the end of the webinar by an attendee about basal fat levels in the author's studies? A similar question about TMR formulation was raised at the May 2019 ADSA DISCOVER Conference as to why this author's studies often used atypical diets for mid-lactation cows? For instance, 22% corn silage, 15% alfalfa silage, 4.6 % wheat straw, and 7% whole cottonseed. That calculates only about 37% forage, and even with the added wheat straw that is slightly over 40% roughage. Baseline fatty acids with the 7% whole cottonseed was about 3.4% (de Souza and Lock 2019). Why was 7% whole cottonseed used?

**References**

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