ACTUS NUTRITION

Animal Health

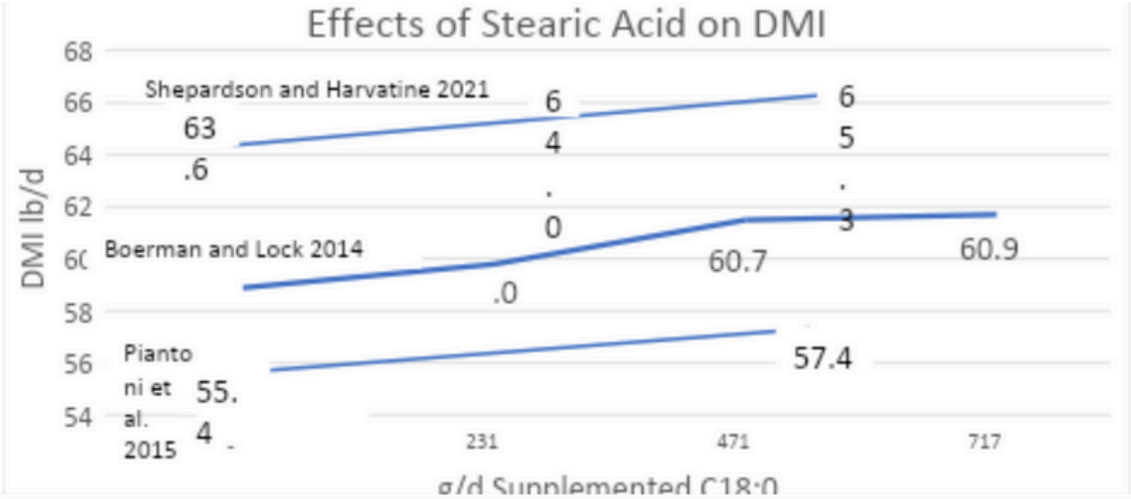
Hepatic Oxidation Theory

How It May Explain the Intake Increasing Effects of Stearic Acid

FROM THE MAKERS OF ENERGY BOOSTER AND RPMET™

The purpose of examining the HOT and the stearic acid (C18:0) connection to DMI is centered on three studies in the past few years illustrated in Figure 1.

Figure 1. The effects of highly purified C18:0 on DMI in lactating cows.



The HOT theory is based on the liver as the primary sensing organ integrating the long- and short-term mechanisms affecting DMI (Allen 2000). In simple terms, DMI is reduced when fuels taken up by the liver are oxidized or stimulate hepatic oxidation (Albornoz et al. 2023). When the oxidation of fuels in the liver is reduced, DMI is increased. The HOT proposes that when hepatic energy charge increases via fuel oxidation, the firing rate of hepatic vagal afferents decreases, subsequently signaling satiety, whereas a decrease in energy charge after a meal increases the firing rate, subsequently resulting in hunger and meal initiation (Allen et al. 2009). White et al. (2011) suggested that the circulating FA that are characteristically increased in transition cows may contribute to increased expression of pyruvate carboxylase (PC) mRNA to stimulate gluconeogenesis and maintain oxaloacetate for the tricarboxylic acid cycle. The data demonstrate a specific effect of PPARα agonist on promoter 1, and an effect of stearic acid to decrease promoter 1 activity. P1 activity is elevated during feed restriction and results in increased hepatic oxidation of NEFA (Velez and Donkin 2005). This may be one connection that C18:0 has to reducing hepatic oxidation of fuels such as NEFA. Shepardson and Harvatine (2021) fed 90% purified C16:0 and C18:0 and found a linear relationship between C18:0 and NEFA as shown in Table 1. Table 1. The effect of C16:0 and C18:0 on DMI and NEFA.

***Adapted from Shepardson and Harvatine 2021*

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Item	Control	2% C16:0	1% C16:0 and 1% C18:0	2% C18:0	P Value
DMI lb/d	61.2a	59.0b	61.6a	62.9a	<0.001
NEFA uEq/l	95.2c	126.6a	106.0b	95.6c	<0.001

Another study (Wang et al 2010) feeding EB 100 to heat stressed cows resulted in similar results on NEFA while significantly improving MY and SCM. These results are shown in Table 2.

Table 2. The effect of feeding 1.5% or 3% EB 100 to heat stressed cows on MY and NEFA.

**Adapted from Wang et al 2010

Item	Control	1.5% EB 100	3.0% EB 100	P Value
DMI lb/d	44.4	44.2	44.4	0.87
MY lb/d	58.1	62.9	62.7	0.02
SCM lb/d	55.9	62.5	64.0	0.01
NEFA uEq/l	376	359	330	0.03

Cows fed SFA had decreased (P < 0.03) NEFA levels, and there was a tendency for NEFA to decrease further with the level of fat fed (P < 0.10). In addition, cows fed EB 100 produced more milk, SCM, MFY, and MPY than control cows.

Another trial utilizing fresh cows (Piantoni et al. 2015b) illustrated yet again the relationship of EB 100 and NEFA. The results are shown in Table 3.

Table 3. The effect of different fNDF levels and SFA inclusion in the diet on intake and NEFA.

**Adapted from Piantoni et al 2015b

	20% fNDF		26% fNDF		SFFA vs Control
Item	No Fat	2% EB 100	No Fat	2% EB 100	
DMI lb/d	51.9a	53.2b	45.8a	50.6b	
NEFA uEq/l	689	522	965	868	0.04
					0.06

In general, C18:0 supplementation improves DMI while reducing circulating NEFA, which in turn, reduces the supply of NEFA for oxidation in hepatic tissue. DMI is still the key to more NE intake.