2002

Short communication: relationship between body growth and mammary development in dairy heifers

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Recommended Citation
L F. Silva, M J. VandeHaar, Brian K. Whitlock, R P. Radcliff, and H A. Tucker. "Short communication: relationship between body growth and mammary development in dairy heifers" Journal of Dairy Science 85 (2002): 2600-2602.

J. Dairy Sci. 85:2600–2602

Short Communication: Relationship Between Body Growth and Mammary Development in Dairy Heifers

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ABSTRACT

Our objective was to determine if prepubertal rate of body weight (BW) gain, independent of diet, was related to mammary development of dairy heifers. Data from two studies recently conducted at Michigan State University were used to identify factors, within a dietary treatment group, that would account for variation in first lactation milk production or amount of mammary parenchymal DNA at the time of puberty. Factors analyzed for variation in milk production during first lactation were: postpartum BW, prepubertal BW gain, gestational BW gain, postpartum BW gain, body condition score (BCS) at breeding, and BCS at calving. Factors analyzed for variation in mammary parenchymal DNA at puberty were: BW at slaughter, age at puberty, prepubertal BW gain and body protein and body fat content at slaughter. For both analyses, prepubertal BW gain did not account for any of the variation in mammary development. The only significant covariate for the milk production model ($r^2 = 0.44$) was BCS at breeding. Similarly, the only significant covariate in the parenchymal DNA model ($r^2 = 0.22$) was body fat content at slaughter. These results suggest that, within a dietary treatment, heifers that grow faster do not have impaired mammary development, and increased body fatness may be a better predictor of impaired mammary development than BW gain.
(**Key words:** bovine, mammary gland, milk yield, prepubertal)

Abbreviation key: ECM = energy-corrected milk production, **KBS** = Kellog Biological Station, **ME** = metabolizable energy, **MSU** = Michigan State University Teaching and Research Center.

High-energy diets promoting rapid BW gain during the prepubertal phase of mammary growth (3 to 10 mo of age) impair mammary development and subsequent milk production of dairy heifers (Sejrsen and Purup, 1997). Although the mechanism by which high-energy diets impair mammary development is not clear, the general concept that emerged was that rapid BW gain impairs mammary development (Capuco et al., 1995; Van Amburgh et al., 1998). However, in all published studies, differing amounts of energy intake were the treatments applied to the animals. Therefore, changes in BW gain were a result of differing dietary energy intake. Thus, it should not be necessarily assumed that changes in BW gain per se, rather than differing dietary energy intake, are the cause of impaired mammary development. Sejrsen et al. (2000) recognized this concept and stated that when heifers are fed the same diet, those with the highest growth rate throughout the rearing period are expected to be heavier at calving and have the highest milk yield.

Our objective was to determine if prepubertal rate of BW gain, independent of dietary treatment, was related to mammary development. Data from two studies recently completed at Michigan State University were used to investigate factors associated with variation in either milk production (Radcliff et al., 2000) or mammary development (Whitlock et al., 2002) independent of diet. Briefly, 60 heifers in the first study (Radcliff et al., 2000) were fed diets high (2.8 Mcal of metabolizable energy (**ME**)/kg and 19.3% CP) or low (2.3 Mcal of ME/kg and 17.5% CP) in energy and protein density and were bred at 365 kg. Heifers were housed in one of two locations, the Michigan State University Dairy Teaching and Research Center (**MSU**) or the Kellogg Biological Station (**KBS**). Treatment started when the animals were 4 mo of age and ended when the animals were confirmed pregnant. Thereafter, all heifers were fed the same diet as appropriate for gestation and first lactation requirements. In the second study (Whitlock et al., 2002), 46 heifers were fed one of three diets beginning at 14 wk of age. All diets were high in energy (2.8 Mcal of ME/kg) but varied in protein content (14, 16, or 19% CP). Heifers were slaughtered at the fourth estrous cycle after puberty.

Received April 9, 2002.
Accepted May 1, 2002.
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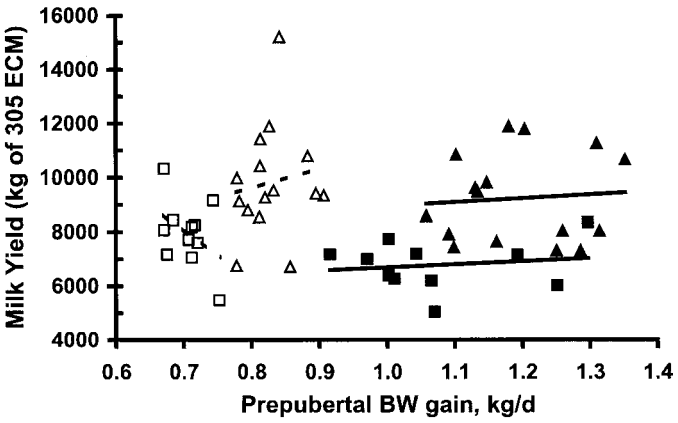

Figure 1. Relationship between prepubertal BW gain within a dietary group and 305-d energy-corrected milk production (305 ECM). Individual data points and regression lines are for heifers fed low (△, ----, r = 0.12, $P > 0.67$) and high (▲,—,r = 0.07, $P > 0.75$) energy diets at the Michigan State University Dairy Teaching and Research Center, and those fed low (□, ----, r = –0.46, $P > 0.15$) and high (■, r = 0.15, $P > 0.65$) energy diets at the Kellogg Biological Station.

Regression analysis was used to identify factors that might account for variation in energy-corrected, 305-d projected milk production (**ECM**, first study), or mammary parenchymal DNA at puberty (second study) using the general linear model procedure of SAS (2000). The following covariates were tested in the development of the model for ECM in the first study: postpartum BW, prepubertal BW gain, gestational BW gain, postpartum BW gain, BCS at calving and BCS at breeding, all within diet. For the mammary parenchymal DNA model (second study) the covariates tested were: BW at slaughter, age at puberty, prepubertal BW gain, body protein and body fat at slaughter, all within diet.

Selection of the best model was based on improvement of r^2 and number of covariates. The existence of multicorrelation among covariates was evaluated by correlation analysis and variance inflation factor (SAS, 2000), and correlated variables were tested separately when developing the model. The significance of two-way and three-way interaction terms between the covariates was also evaluated after elimination of nonsignificant covariates from the model. A covariate was deemed to be nonsignificant if the P value of the t-test from the Type III sum of squares was greater than 0.50.

Factors explaining milk production. Although heifers that received the high-energy diet before puberty had lower milk production during first lactation (Radcliff et al., 2000), within a dietary treatment, prepubertal BW gain was not related to milk production (Figure 1) and had no value as a predictor of milk production (Table 1). Heifers that grew faster within a treatment produced as much milk as those that grew slower. Also, prepubertal BW gain was not related ($P > 0.01$) to postpartum BW in any of the four treatment groups. This result suggests that although high dietary energy concentration before puberty increases BW gain and decreases milk production in a group of animals, rapid BW gain per se is not the cause of reduced milk production.

The BCS at breeding within a dietary treatment was the only factor that improved the prediction of milk production after considering the effects of diet and location (Table 1). This relationship between BCS at breeding and milk yield was negative ($P < 0.05$) for heifers fed the low-energy diet (Table 1), suggesting that when heifers were fed the same low-energy diet ad libitum, those that gained a higher proportion of fat might be expected to produce less milk as a cow. For the heifers fed a high-energy diet, the relationship between BCS at breeding and milk yield was not significant ($P = 0.98$, Table 1). Most heifers (82%) fed the high-energy diet were overconditioned at the time of breeding (BCS equal to or greater than 4), which could explain the lack of relationship between BCS and milk yield. The MSU dairy herd has a higher milk production than the KBS herd, which explains the significant effect of location ($P < 0.01$, Table 1).

Factors explaining mammary parenchymal DNA at puberty. There was no relationship between BW gain of an individual heifer and her mammary gland development (Figure 2) despite considerable variation in individual gains (range = 0.9 to 1.4 kg/d). Because dietary protein treatment had no effect on parenchymal DNA/ kg of BW and because all groups were fed the same forage and energy concentration, we combined

Table 1. Sources of variation in milk yield (305 energy-corrected milk production) determined by regression (model $r^2 = 0.44$)[1].

Source[2]	DF	Type III SS	P
Location	1	58438406	<0.01
Diet	1	12517858	0.03
BCS at 365 kg within diet	2	16195828	0.04
Error	45	109487629	
	Diet[3]	Partial regression coefficients[4]	P
BCS at 365 kg	Low	–2799	0.01
BCS at 365 kg	High	20	0.98

[1]From Radcliff et al. (2000).

[2]Factors that did not enter the model ($P > 0.50$): prepubertal BW gain, gestational BW gain, postpartum BW and BCS at calving, all within diet.

[3]Low = 2.3 Mcal of metabolizable energy/kg, 17.5% CP and high = 2.8 Mcal of ME/kg, 19.3% CP.

[4]Value is for total 305-d energy-corrected milk production (kg). Range of BCS = 3 to 4.25 for the low-energy diet and 3.25 to 5.0 for the high-energy diet.

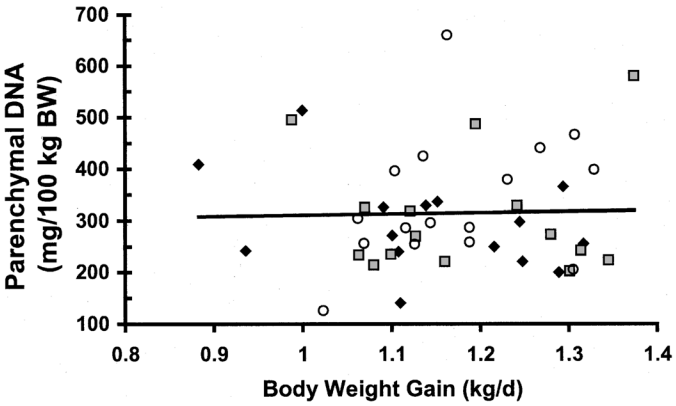

Figure 2. Relationship between prepubertal BW gain and mammary parenchymal DNA content (mg/100 kg BW, r = 0.03, $P > 0.85$). Individual data points are for heifers fed low (◆), standard (■), and high (○) protein diets.

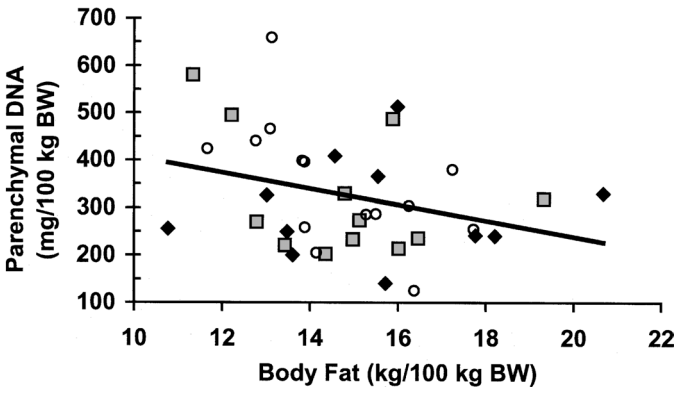

Figure 3. Relationship between prepubertal body fatness and mammary parenchymal DNA content (mg/100 kg BW, r = –0.30, $P < 0.07$). Individual data points are for heifers fed low (◆), standard (■), and high (○) protein diets.

the data from the three dietary treatments when developing the regression shown in Figure 2.

ANOVA in mammary development revealed that total body fat, irrespective of dietary treatment, was the only factor that explained some of the individual variation in mammary parenchymal DNA (Table 2). Heifers that were fatter tended to have less parenchymal DNA than those that were leaner ($P = 0.07$, Figure 3). Prepubertal BW gain was not related to mammary development. Similarly, age at puberty, body protein concentration, and BW at slaughter were not related to mammary development (Table 2).

Body fat concentration was negatively related to BW gain (r = –0.36, $P < 0.05$), suggesting that when heifers were fed a high-energy diet, heifers with the highest propensity for lean gain were those with fastest total BW gains. If faster rates of gain are a risk factor for impaired mammary development, then the leanest heifers in this data set should have had less mammary parenchymal DNA. Instead, we found that among heifers fed the same high-energy diet ad libitum, those that gained a higher proportion of fat grew slower and had less mammary parenchyma at puberty. Perhaps heifers with a higher genetic potential for milk also have a higher genetic propensity for lean rather than fat accretion.

In conclusion, feeding heifers high-energy diets to induce rapid BW gain before puberty decreases mammary development. However, when evaluated independent of dietary treatment, heifers that grew faster did not have impaired mammary development. Furthermore, increased body fatness was a better predictor of impaired mammary development than was rapid BW gain.

Table 2. Sources of variation in parenchymal DNA (mg/kg BW) determined by regression (model $r^2 = 0.22$)[1].

Source[2]	DF	Type III SS	*P*
Diet[3]	2	64409	0.11
Body fat within diet[4]	3	101374	0.08
Error	31	416536	

[1]From Whitlock et al., 2002.

[2]Factors that did not enter the model ($P > 0.50$): age at puberty, prepubertal BW gain, body protein and BW at slaughter, all within diet.

[3]Diets were 14% (low), 16% (standard), or 19% CP (high).

[4]Total body fat at the time of slaughter, 7 wk after detection of first corpus luteum.

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