WHAT CAUSES HIGH PRODUCTION?

Story of the Role of the Lactogenic Hormone in Milk Secretion

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The inherited capacity of a dairy cow for high milk and fat production is primarily dependent upon the growth of the lobule-alveolar system in the udder. These cells secrete milk.

The growth of the milk secreting cells is stimulated during recurring estrous cycles, pregnancy and early lactation. The hormones which stimulate growth of the udder have been described (1). It was suggested that the greatest growth of the udder occurs in cows which secrete optimum amounts of the hormones which stimulate this growth.

To stimulate the cells of the udder to secrete milk requires the secretion by the anterior pituitary of another hormone called lactogen. This bulletin will tell the story of the role of this hormone in milk secretion.


In first calf heifers, during the first six months of pregnancy, growth of the lobule-alveolar system occurs. At this time, the milk secreting cells present begin to secrete milk under the stimulus of the lactogenic hormone.

At first, the cells lining the alveoli increase in length as milk is formed within the cell. Then the secretion within the cells is discharged into the lumen (cavity) of the alveoli causing the gradual expansion or ballooning out of the alveoli. As the first milk (called colostrum) continues to be secreted into the lumen of the alveoli, it slowly flows out through the ducts and gradually fills the entire duct system and the large cistern above each teat (Fig. 1).

Externally, the udder during the last three months

Fig. 1. THE INITIATION OF MILK SECRETION NEAR THE END OF PREGNANCY. During the first six months of pregnancy, the udders of first calf heifers show no external signs of growth (Left). During the last 3 months, while growth continues, the swelling of the udder is caused by the initiation of milk secretion (colostrum) in the secreting cells. By the time of calving, the udder is greatly enlarged (Right).
of pregnancy shows signs of enlargement as the colos­
trum fills the udder. Many dairymen have believed that
the swelling observed at this time indicated that the ud­
der was growing. It is true that the growth of the udder
takes place during the entire period of pregnancy, but the
enlargement of the udder during the last 1/3 of preg­
nancy is caused by the initiation of colostrum secretion,
not by more rapid growth of the udder.

Many heifers and some cows show excessive enlarge­
ment and swelling, not only of the udder, but of the
tissue in front of the udder. This swelling is caused by
the presence of increased amounts of blood and lymph in
the skin and the filling of the udder with colostrum. The
increased secretion of the estrogenic and lactogenic hor­
mones causes this swelling rather than the type of feed
consumed. It occurs when heifers are experimentally in­
duced to secrete milk with estrogen as well as normally
in late pregnancy.

Prepartum Milking

Normally, heifers and cows are not milked before
parturition (calving time). Some dairymen try to prevent
the congestion and swelling by milking the cows before
parturition. This practice may not only relieve the swel­
ling of the udder but may be of help in preventing milk­
fever (parturient paresis) by regular removal of the colos­
trum and milk prior to calving. However, this practice
introduces a problem for the calf.

The calf after birth needs to be fed the colostrum or
first milk because it contains gamma globulins which are
transferred intact from the mother’s blood into the colos­
trum. This blood protein contains immune bodies which
protect the calf from harmful bacteria for a period after
birth until the calf can gradually build up its own im­
munity.

If cows are milked before calving, the colostrum is
all removed and the milk becomes normal. It is neces­
sary, therefore, either to save the colostrum from the dam
or to obtain colostrum from another cow to feed to the
calf immediately after calving.

Lactation

At the time of calving, the cow is prepared to pro­
duce milk. By the regular removal of the milk, the yield
increases rapidly for as long as 30 to 60 days or more.
During this period additional growth of milk secreting
cells occurs. As these newly-grown cells develop, they
may account for part of the increase in milk yield as they
begin to secrete milk.

Maximum Milk Production

The maximum yield of milk is of great significance
since it is one of the best measures of the size of the ud­
der (the number of milk secreting cells present in the
udder). Cows capable of secreting 50 to 100 lbs of milk
per day must have udders containing increasing numbers
of milk secreting cells (Fig. 2). Thus, the average maxi­
num milk yield indicates the effectiveness of the hor­
mones which stimulate the growth of the udder during
pregnancy and early lactation. Bulls which sire daughters
with high average maximum milk yields are transmitting
to their progeny the inheritance of hormone secretion
rates necessary for optimal udder growth (1).

In addition to the size of the udder, other factors in­
fluence maximum milk production. These factors influence
the intensity of milk secretion in each cell. Thus, each
cell may secrete variable amounts of milk per hour or per
day. A number of hormones will be described which in­
fluence the secretion and the intensity of secretion in the
cells. However, the hormones are only stimulators of

\[1\] C. W. Turner, A comparison of Guernsey Sires, II. Based on the Average
Mature Equivalent Fat Production of Daughters During the Month of Maxi­

Fig. 2. THE NORMAL LACTATION CURVE. At the time of parturition,
the cow is prepared to lactate. Cows increase in milk yield very rapid­
ly reaching maximum production within a period of 30 to 90 days,
depending upon their condition at calving time, the frequency of milk­
ing, their feed consuming capacity and the yield of milk at the maxi­
um. Cows then begin to decline in milk yield at a regular rate (per­
sistency) varying with the individual. The persistency depends pri­
marily upon the rates of secretion of the lactogenic hormone, the
thyrotropic hormone (and thyroxine) the growth hormone and other
hormones. If any one of these hormones is secreted at less than their
optimal rates, then the cow’s persistency will be governed by the rate
of secretion of the most deficient hormone.

Cows should be bred to calve at yearly intervals. The cows should
be dried off in time to provide a dry period of about 40 days. It is
important to feed the dry cow in order to improve her condition and
restore elements depleted by the previous lactation.
milk secretion. Along with the hormones, the dairymen plays a vital role in his ability to feed and manage the lactating cow. The full potential capacity of the cow rests upon good nutrition, favorable environment and good milking practices.

More specifically, high maximum milk yield depends upon the condition of the cow at calving time. The store of fat is utilized for milk production while the cow is reaching her maximum feed consuming ability which may take 4 or 5 months (Fig. 3). Further, the cow must have the capacity for large feed intake and digestion to supply the nutrients to be converted into milk. Recent study has shown the importance of the rumen bacteria in the utilization of roughage (cellulose), in the utilization of non-protein-nitrogen (such as urea) and in the production of many B-complex vitamins. If the roughage is poor, the cows cannot obtain sufficient nutrients from the roughage to supply the energy required unless the grain mixture is of high energy content.

The frequency of milking also influences the maximum yield of milk. As milk accumulates in the lumen of the alveoli and holding spaces of the udder it tends to gradually inhibit the intensity of milk secretion (Fig. 1). The more frequent removal of milk permits maximum intensity of the milk manufacturing process made possible by the hormones. The completeness of milk removal at milking time is also of great importance. If part of the milk present in the udder at milking time is not removed, it tends to check the secretion of milk during the interval between milking.

**Fig. 3. RELATION BETWEEN MILK YIELD, FEED CONSUMPTION AND BODY WEIGHT.** In this group of cows, note that milk production reached a peak during the second month, then gradually declined during the remainder of the lactation period. The average persistency of the cows was 94%. These cows were provided with all the feed they would consume. Protein consumption increased for 5 months and carbohydrates (energy) consumption increased for 4 months. Increasing feed consumption did not prevent the decline in milk production. Thus high feeding will not increase milk yield above its hereditary limits (endocrine control). However, it is true that underfeeding will depress milk yield.

Body weight declined for a period of 3 months. The high condition (fat) is being used by the cows to make possible the high milk they attained when the feed intake was insufficient to supply the nutrients required for milk secretion. At the time that feed intake had reached its peak and milk yield had declined sufficiently, then equilibrium between nutrient intake and milk yield was attained. Following this period, feed intake exceeded the needs for milk production and the cows began to increase in body weight. It indicated that the condition of cows can be restored during the latter part of the lactation, if full fed, in preparation for the next lactation period (Turner, 1924).

**Fig. 4. CROSS SECTION OF THE UDDER.** When an udder filled with milk is sectioned, one sees many alveoli filled with milk. The individual cells lining each alveolus are too small to see at this magnification. The heavy band running from top to bottom at the left of the picture is composed of connective tissue which surrounds groups of alveoli. A group of such alveoli is called a lobule. Groups of lobules are bound together by connective tissue to form lobes.
Persistency of Milk Secretion

After cows reach the peak of milk yield, there usually follows a gradual decline in milk production. The rate of decline in milk yield or the persistency of milk secretion varies greatly in individual cows. Some cows are very persistent. That is, the rate of decline is slow, whereas other cows decline very rapidly or show poor persistency. The rate of decline in milk production when feed, environment, and milking practices are uniform follows a regular pattern from month to month in individual cows. Some cows decline at the rate of 2 to 4% of their previous month's production, whereas other cows decline in milk yield at a rate of 5 to 8% and a few decline as much as 9 to 12% per month (Fig. 5). Another way of indicating the persistency of a cow is to say that the yield of milk each month is a constant percentage of the previous month's production, thus, the cow produces 98%, 95% or 90% of the previous month's production. Cows of high persistency produce more milk in a lactation than cows of low persistency when their maximum production is the same.

The two factors in high total milk and fat production are large maximum yield with highly persistent production during the lactation period (Fig. 6).

The causes of the differences in persistency of milk secretion in individual cows are now understood. It is the purpose of this bulletin and those which will follow to explain the role of the several hormones in maintaining high persistency or intensity of milk secretion.

![Fig. 5. PERSISTENCY OF MILK SECRETION. After dairy cows reach their peak milk production, they usually begin to decline. The persistency of milk secretion has been defined as the degree with which the rate of secretion is maintained as lactation advances. A few cows maintain a uniform level of production for many months. In these animals the secretion of the lactogen, thyroxine, growth hormones and possibly other hormones are maintained at optimum levels. If cows have a high maximum production associated with great persistency, they become the outstanding record holders of their breed. Most cows, however, decline at varying rates from month to month as indicated above and the feeding and management is not able to increase their persistency. If one or more hormones is administered to cows showing a lack of persistency, then the decline can be arrested to the extent that the animals are deficient in the secretion rate of the given hormone. By this method (replacement therapy), the extent of the inherited deficiency in the secretion rate of the hormone can be estimated by the response observed. If no response is observed, then clearly, the decline in lactation is not due to a deficiency of the hormone administered.](image1)

![Fig. 6. PERSISTENCY AND VARIABLE MAXIMUM PRODUCTION. In this chart is presented the decline in milk production of cows varying in maximum milk production from 20 lbs/day to 100 lbs/day with a constant persistency of 95%. In other words, these cows produce each month 95% of their previous month's production. It can be clearly observed that while their persistency is constant, the absolute rate of decline in monthly milk production is much greater in high maximum milk producing cows than in low producers. It shows graphically the importance of optimal secretion of the hormones which influence the persistency of milk production. Increasing the persistency of low producing cows results in a few pounds of increased milk yield whereas increasing the persistency of high maximum producing cows results in many pounds increase in milk production.](image2)
The Lactogenic Hormone

The hormone responsible for initiating the secretion of colostrum during the last 3 months of pregnancy and for maintaining secretory activity in the cells of the udder during lactation is secreted by the anterior lobe of the pituitary gland. It is called the lactogenic hormone (Fig. 7). This hormone causes the individual cells of the udder to begin functional activity (milk secretion) as contrasted to multiplication or growth. After maximum lactation is reached the multiplication or replacement of cells is slow as contrasted to the period of rapid growth of the udder during pregnancy and early lactation.

Enzymes are present within each epithelial milk secreting cell of the mammary gland. The lactogenic hormone has the unique function of stimulating increased enzyme activity. When the enzymes in the cells are activated, the cells have the ability of converting the various constituents of blood into the various constituents of milk. While other hormones, to be described, may influence the intensity of milk secretion, the lactogenic hormone, is primarily responsible for activating the enzyme systems in the cells and for stimulating milk secretion. Thus, in the absence of the lactogenic hormone, the mammary gland cells will not secrete milk.

The discovery of the lactogenic hormone dates back to the years 1928-29. In the early experiments, the anterior pituitary or crude extracts were used. As time went on, methods of purification were developed and at present relatively pure preparations are available for experimental work. Since the pituitary is small and methods of purification rather difficult, this hormone is scarce and expensive. It cannot be used as a practical method of stimulating milk secretion. However, it has been shown in many experimental animals as well as in cattle, that when the mammary gland has been grown, the injection of the lactogenic hormone quickly initiates the secretion of milk in the cells of the mammary gland comparable to that observed in late pregnancy and after calving (Fig. 7). The question arises as to the normal mechanism in late pregnancy which stimulates the anterior pituitary to gradually secrete increased amounts of the lactogenic hormone and after parturition to further increase its secretion to cause milk secretion to increase up to the period of maximum production. Following the period of maximum production, what is the role of the lactogenic hormone in regulating the rate of decline of milk yield or the cow's persistency? During the past 15 years the answers to some of these questions have been discovered.

Study of the Anterior Pituitary

Examination of the anterior pituitary in thin sections by means of the microscope show the presence of two types of cells which take up dye. One group of cells takes up acid dye, whereas a second group of cells takes up basic dyes. The cells which stain with acid dyes are called acidophilic cells, whereas those which take up basic dyes are called basophilic cells. The tiny granules in these cells contain the hormone which the cells secrete. It has been shown that certain acidophilic cells secrete the lactogenic hormone.

The pituitaries of animals in late pregnancy and during lactation have been examined. It has been observed that there is a rise in the number of acidophilic cells in late pregnancy which continues for a time after parturition. The granules in these cells indicate that they are secreting lactogenic hormone intensely. In later lactation, the number and secretory activity of these cells may decline (Fig. 9).
A second type of study shows the secretory activity of the cells secreting the lactogenic hormone. The amount of lactogenic hormone in the pituitaries of animals during pregnancy and lactation has been determined. These studies show that the amount of lactogenic hormone in the pituitaries in early pregnancy is low. In late pregnancy the amount begins to increase and continues to rise for a time after parturition, then gradually declines.

These two types of observations concerning the role of the anterior pituitary in the secretion of the lactogenic hormone may be directly correlated with the beginning of milk secretion in the udder of heifers in late pregnancy and the rapid rise in milk secretion after calving. The decline in milk secretion after maximum production is reached is related in part to the decline in the secretion of lactogenic hormone at that time.

Discovery that Estrogen Stimulates Secretion of Lactogenic Hormone

When it was observed that the secretion of the lactogenic hormone increased in late pregnancy and early lactation, it was decided to determine whether the ovarian hormones, estrogen and progesterone, which are secreted during pregnancy, stimulated the increased secretion of the lactogenic hormone. Animals were injected with estrogen alone, progesterone alone, and combinations of the two hormones. It was discovered that when estrogen was injected into animals, the lactogenic hormone content of their pituitaries increased (Fig. 10). Progesterone, on the other hand, had little effect.

With this discovery, the explanation of the gradual rise in the secretion of the lactogenic hormone in late pregnancy was clarified. It has been shown that there was a rapid rise in the estrogen secretion by the dairy cow in late pregnancy (Fig. 8). It appeared that estrogen stimulated the secretion of the lactogenic hormone which in turn stimulated the secretion of milk. If this were true, then in animals whose mammary glands have been grown, instead of stimulating the secretion of milk with scarce lactogenic hormone, why not stimulate the pituitary of the animal with estrogen and cause it to secrete the lactogenic hormone just as it occurs in normal pregnancy?

Estrogen has been injected into virgin heifers whose udders had been stimulated to growth by the injection of estrogen and progesterone for variable periods. When these heifers were then injected with 3 mg estradiol benzoate, daily, for a period of 2 weeks, milk secretion was initiated and continued to increase for periods of weeks even after estrogen was discontinued (1).


Similarly, it has been observed that virgin heifers of increasing age (up to 3 years) when treated with estradiol benzoate could be induced to secrete considerable amounts of milk. These latter observations have suggested that considerable growth of the lobule-alveolar system is induced by recurring estrous cycles.

The observation that estrogen will induce the secretion of milk, even in virgin heifers, is believed to prove that the rise in estrogen secretion in late pregnancy stimulates the increased secretion of the lactogenic hormone. This explains the gradual increase in colostrum secretion in late pregnancy and the preparation of the cow for increasing milk production after calving.

Relation Between Estrogen and Lactogenic Secretion

Cows vary in the amount of the estrogenic hormone secreted in late pregnancy. Cows which secrete low levels of estrogen would be less able to stimulate their pituitaries to optimal lactogenic hormone secretion. It was shown many years ago that the estrogen secreted by beef cattle in late pregnancy was, on the average, considerably lower than the average secretion of dairy cattle. The low secretion of estrogen by beef cattle may be one of the causes of low lactogenic hormone secretion and poor milk secretion.

In some dairy cattle, which may secrete lower than normal levels of estrogen, the injection or feeding of estrogen for a few days before or after parturition might be of value in stimulating increased amounts of the lactogenic hormone. A further test of the effectiveness of estrogen in increasing the secretion of the lactogenic hormone could be made by administering estrogen for a few days at the time of maximum milk production (Turner et al 1957). Cows showing a further rise in milk yield would indicate that the estrogen secreted in late pregnancy was insufficient to produce optimal amounts of lactogenic hormone.

Some cows abort about the 7th month of pregnancy. At this time the growth of the mammary glands is not complete but extensive. However, the yield of milk of such animals is very low. The low milk yield is caused in part by the fact that the estrogen secretion rate is still low and the secretion of the lactogenic hormone will be low. If cows which abort at this time are injected or fed estrogenic hormone for a period of about 2 weeks, the yield of milk will be greatly increased (Fig. 8).
Fig. 8. ENDOCRINE CONTROL OF MILK SECRETION. The figure shows the endocrine control of the growth of the udder which is stimulated by progesterone and estrogen during pregnancy. Later, the increasing secretion of estrogen by the placenta stimulates the secretion of the lactogenic hormone which stimulates the initiation of lactation by the epithelial cells of the udder (see Fig. 7). If prepartum milking is practiced, increasing amounts of milk may be obtained. Otherwise, the udder swells up with the contained milk and the cow is prepared to milk at the time of parturition (Fig. 1).

The rapid rise in estrogen secretion in late pregnancy also overrides the physiological function of progesterone (which normally maintains the pregnant condition) and aids in the initiation of parturition.

In experimental animals, the withdrawal of progesterone and the injection of estrogen, similarly, stimulates the secretion of the lactogenic hormone and initiates the secretion of milk.

Following parturition and the removal of the placenta, the secretion of estrogen quickly drops and the maintenance of the secretion of the lactogenic hormone is caused by the regular discharge of the hormone stimulated by milking.

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Fig. 9. THE POSTPARTUM INCREASE in the acidophils and lactogen content of the pituitaries of rats. Since the acidophiles also secrete growth hormone, its rate of secretion may also increase.

Fig. 10. ESTROGEN STIMULATES SECRETION OF LACTOGEN. It was shown in our laboratory that the injection of estrogen into experimental animals would stimulate the secretion of increasing amounts of lactogen in the pituitary. In normal animals, the increasing amounts of estrogen secreted in late pregnancy has this same effect and thus initiates the gradual secretion of milk. In experimental heifers, after the mammary glands have been grown, the daily injection of 3 mg of estradiol benzoate for 2 weeks, similarly, stimulates the pituitary to secrete the lactogenic hormone and cause a rapid initiation of milk secretion which continues to increase for many weeks. It will be observed that if overdoses of estrogen are administered then the secretion of lactogenic hormone is inhibited.
Cause of the Rise in Milk Secretion

In the experimental heifers, estrogen was given for a period of 14 days, then stopped. Milk yield, however, continued to rise for a number of weeks. Similarly, in normal pregnancy, the secretion of estrogen stops at the time of parturition. Thus the initial stimulus of estrogen upon the pituitary to stimulate the secretion of the lactogenic hormone stops at parturition. To maintain the secretion of the lactogenic hormone during lactation some other mechanism must be involved. This mechanism has been discovered. It has been shown that once the secretion of lactogenic hormone is started by estrogen during late pregnancy, the stimulation of regular milking maintains the secretion of the lactogenic hormone after parturition. (Fig. 11).

The Relation of the Hypothalamus to the Anterior Pituitary

At each milking period, a nervous stimulation passes from the teats to the spinal column and to the lower part of the brain just above the pituitary to an area called the hypothalamus. However, the nervous stimulus cannot pass to the anterior pituitary because there are no nerves from the hypothalamus to the anterior pituitary. Instead, there is a hormone secreted in the hypothalamus which flows down blood vessels from the hypothalamus (called portal veins) to the anterior pituitary to prevent the release or discharge of the lactogenic hormone. This hormone has been called the lactogen inhibiting factor. So long as this factor flows from the hypothalamus to the pituitary it inhibits lactogen discharge. When the secretion of the hypothalamic factor is depressed, then lactogenic hormone is discharged and in turn milk secretion is stimulated.

At this time, the lactogen inhibiting factor has been shown to be depressed in two ways—by the estrogenic hormone and by the milking stimulus. Thus, the secretion of the estrogenic hormone in late pregnancy depresses the lactogen inhibiting factor and lactogen secretion proceeds. At milking time, the neural stimulus temporarily depresses the lactogen inhibiting factor and lactogen is released.

During the interval between milkings, the lactogen inhibiting factor prevents the discharge of lactogen and it then builds up again as indicated and would again be at the original level in 10 hours.

Oxytocic Hormone

The stimulus of milking also causes the release of a hormone from the posterior lobe of the pituitary, called oxytocin. The presence of this hormone in the blood stimulates the contraction of myoepithelial cells which surround each alveolus. This hormone is frequently called the milk "let-down" factor since it aids in the rapid removal of milk at milking time. Thus, the milk present in the udder is removed. The lactogenic hormone which is also discharged into the blood is picked up by the milk secreting cells of the udder and stimulates the secretion of milk during the interval between milkings.
Maintenance of Maximum Milk Secretion

The original stimulus to the anterior pituitary to secrete lactogenic hormone is imparted by the estrogenic hormone in late pregnancy. The continued stimulus to maintain a high level of secretion of the lactogenic hormone is imparted by the regular discharge of the lactogenic hormone by the stimulus of milking. If cows are not milked regularly and if the lactogenic hormone is not stimulated to be discharged, the secretion of lactogenic hormone gradually declines. Within a short time the level of hormone in the pituitary is back to the level of a non-lactating animal (Fig. 12).

The rise in the yield of milk up to the period of maximum production is believed to be caused by the continued rise in the rate of secretion of the lactogenic hormone and certain other hormones which will be described later as well as continued growth of the udder during early lactation. The pituitaries of some cows have the capacity to secrete optimal amounts of the lactogenic hormone and cause the continued increase of milk secretion for many weeks. Other cows’ pituitaries are deficient in their capacity to secrete the lactogenic hormone and peak lactation is reached within a few weeks.

The Decline in Milk Secretion

While a few cows are very persistent in lactation and continue milk secretion at levels close to their peak for many weeks and months of lactation, most cows begin a regular decline in milk yield even with super-feeding and frequent milking (Fig. 5, 6). This decline is believed to be caused, primarily, by the gradual decline in the secretion of the lactogenic and other hormones. If the rate of decline is more rapid, then the rate of decline in milk secretion is more rapid.

It is extremely important, regarding dairy cattle, to breed cows whose pituitary is capable of secreting optimal amounts of the lactogenic and other hormones and of having the capacity of maintaining a high level of hormone secretion during the entire lactation period. How can one select cows for breeding improvement which have the capacity for high and sustained secretion of these hormones? If cows show a persistency of lactation of 98% or more, it demonstrates clearly that the secretion of the lactogenic and other related hormones is very persistent. If high persistency is associated with high maximum production, then the cow has all the desirable endocrine factors sought. Deficiency in persistency of secretion of the lactogenic hormone will be found most commonly in cows with high maximum milk yield. Under such conditions, the injection of lactogenic hormone will stimulate a marked rise in milk production and the extent of the increase will measure the extent of the deficiency in lactogenic hormone secretion.

Cows with small udders require less hormone to stimulate maximum milk secretion intensity. Their persistency is usually higher due to adequate amounts of lactogenic hormone being secreted. Thus, 2 year old heifers are usually more persistent than they are at maturity when the udder has developed greater size with recurring pregnancy.

In addition to the endocrine factors influencing the persistency of milk secretion, there is a cellular reduction as well. It was indicated earlier that after maximum milk yield is reached the multiplication of the epithelial milk secreting cells is very slow. It is possible that one or more hormones may cause slow cell division during lactation. In most cows, however, there is a slow loss of milk secreting cells. In other words, after the cells secrete milk for variable periods, the cells die and are discharged into

![Fig. 12. REGULAR MILKING OR NURSING MAINTAINS LACTOGEN SECRETION. While estrogen stimulates the cells of the pituitary to secrete increased amounts of the lactogenic hormone in late pregnancy, the maintenance of lactogen secretion after parturition is caused by the stimulus of regular milking or nursing. This is shown in the above figure. If rabbits are not nursed after parturition, the lactogenic hormone content of the pituitaries is lower at the maximum and declines rapidly to the non-lactating level. The maintenance of lactogen secretion is due to the neuroendocrine discharge of the accumulated lactogen at each milking period transmitted to the anterior pituitary by the stimulus of milking.](image)
the milk. Thus, after several months of lactation the total number of secreting cells is appreciably reduced. The slow loss of cells continues until the end of the lactation period.

For this reason, the injection or feeding of one or more hormones may stimulate an increased intensity of milk secretion, yet, because of the loss of cells which has occurred, the yield of milk will not increase up to the level of maximum production. Thus, in late lactation, the apparent effectiveness of hormone therapy is reduced.

**Estrogen**

The fact that estrogen stimulates the secretion of the lactogenic hormone in preparation for calving, but drops off quickly with the removal of the placenta (after-birth) at the time of parturition suggested that the continued administration of estrogen (or diethylstilbestrol) would tend to maintain the secretion of the lactogenic hormone at a high level. It was shown that the only present stimulus to the maintenance of lactogen secretion after parturition is the regular discharge of the hormone at milking time.

Experiments have been conducted to determine whether the feeding of diethylstilbestrol (a synthetic estrogen) would have a beneficial effect upon the persistency of milk secretion. Such a beneficial effect has been observed in a number of individual cows. The feeding of diethylstilbestrol to cows showing a decline in milk yield has been followed by a cessation of the decline and, in some instances, a slight increase in milk yield which was sustained for many weeks (Turner et al. 1957). The beneficial effect of estrogen is believed to be partly caused by the maintenance of the level of lactogenic hormone secretion. Naturally, it would show little benefit in cows of high persistency where an optimal amount of lactogenic hormone was being secreted.

Studies have shown that the amount of hormone fed in no way interferes with reproduction. In pregnant animals, the fed hormone may contribute to the normal estrogen secretion and thus favorably influence the growth of the udder. In fact, it has been shown that the feeding of diethylstilbestrol at the same level (10 mg/day) with progesterone will stimulate the growth of the udder (Turner 1959).

**Effect of Pregnancy Upon Milk Secretion**

To calve within a year, cows must be rebred within 90 days after calving. Thus, after the first pregnancy, cows normally are lactating and pregnant at the same time. During the second and successive pregnancies, the ovarian hormones, estrogen and progesterone are again secreted and additional growth of the udder is stimulated with each pregnancy up to 7 or 8 years of age at which time maximum yearly milk yield is observed. The increase in yearly milk yield up to 8 years, is believed to be due to the additional growth of the udder, to the increase in body size and weight, and to the increased size of the endocrine glands and their capacity to secrete greater amounts of the various hormones involved in milk secretion.

The yield of milk secreted by pregnant and open cows during the first 5 months of pregnancy appears to be the same. After 5 months, the pregnant cows begin to decline more rapidly in milk yield. It has been suggested that this more rapid decline in milk yield is caused by the fact that the growth of the fetus requires increasing amounts of nutrients which are diverted from the mammary gland to the uterus (Fig. 13).

![Figure 13. EFFECT OF PREGNANCY UPON MILK YIELD. During the first 5 months of lactation, the decline in the yield of milk is similar in pregnant and non-pregnant cows. However, after the 5th month of pregnancy these cows begin to decline more rapidly in milk yield. It was suggested by Brody et al. (1923) that the difference in milk yield of about 450 lbs was due to the increasing divergence of nutrients from the mammary gland to the uterus for the growth and maintenance of the fetus.](image)
If the additional milk secreting cells grown during pregnancy begin to secrete milk prior to parturition, their contribution to milk yield is over-balanced by the lack of milk precursors in the blood. Their influence on milk yield is shown only after parturition upon the next lactation period.

Value of the Dry Period

It is usually recommended that cows be given a dry period of 30 to 60 days. If cows are of low persistency they may reach a level of milk yield where the value of the milk does not pay the cost of milking. On the other hand, there are many cows of higher persistency and productivity which are still yielding large quantities of milk up to 30 to 60 days before calving. Should these cows be dried up or continued to be milked up to calving time without a "rest period?"

It is commonly suggested that the purpose of the dry period is to increase the body stores so that any loss of calcium and phosphorus from the bones will be replaced and the cow will be in good condition at the time of calving. If this is the reason for the dry period, it is important for the cow to be well fed then.

However, most cows which are fed all they will eat during the last months of lactation, gain considerably in body weight (Fig. 3) while they are lactating. Thus, if improvement in condition is the chief reason for the dry period, this can be obtained by higher levels of feeding during the latter part of the lactation period.

Is there any benefit to the milk secreting cells of the udder in having a "rest period" before lactating again? In the first calf, it was shown that milk secretion (colostrum) begins to be secreted and the udder "bags up" prior to parturition. If the cows were dried off for 30 to 60 days, the cells of the udder would begin to secrete milk again 2 or 3 weeks before calving. Thus, the cells of the dry cow would actually be "resting" only for a short period during the dry period. Actually, there is no known physiological or endocrine reason for believing that milk secreting cells are benefited by a "resting" period.

As the persistency of dairy cattle increase by selection of such animals and high yields of milk may be obtained throughout the lactation period, the physiological need of a dry period should be explored. The loss of 30 to 60 days of productivity by all cows each year is a serious economic drain on the dairy industry. If cows can be milked continuously, year after year, without loss of productivity by better methods of feeding prior to parturition, this knowledge would be of great economic importance.

As pointed out in the section on prepartum milking, such a plan would necessitate a program of supplying immune bodies to the newborn calf because the milk after parturition would be free of colostrum. As the blood of cattle is rich in immune bodies, cattle blood could be mixed in the milk fed to the calf for several days.

While it is not recommended that all cows be milked continuously (that is without a dry period) until further research has been conducted, if the condition of the cow can be increased during the latter part of pregnancy by liberal feeding and if the production of the cow is well sustained, the program might be tried out on a limited basis to compare with similar cows given a dry period. It is recognized that some cows cannot be brought into a high condition while lactating intensely and such cows may require a dry period.
Summary

The objective of this series of bulletins is to attempt to explain why dairy cattle vary so greatly in their capacity to secrete large amounts of milk. It is suggested that high producing cows have received an inheritance (genes) which enables them to secrete optimal levels of all the hormones influencing the growth of the udder during pregnancy and early lactation. The hormones which stimulate udder growth have been described.

The present bulletin tells the story of the role of the lactogenic hormone in stimulating the secretion of milk by these cells beginning in late pregnancy and continuing throughout the lactation period.

It was shown that the initial secretion of the lactogenic hormone by the anterior pituitary gland is dependent upon the secretion of estrogenic hormone, the same hormone, which in conjunction with progesterone, stimulates udder growth during pregnancy. If the secretion of estrogen were deficient, it would have an adverse effect upon udder growth as well as upon the secretion of the lactogenic hormone.

Following parturition, with estrogen no longer being secreted, the continued secretion of the lactogenic hormone in optimal amounts is dependent upon regular milking.

The lactogenic hormone is secreted in the anterior pituitary during the interval between milking, then is discharged quickly into the blood following the stimulus of milking. It then passes via the blood to the udder to stimulate the cells to secrete milk during the interval between milkings.

The rate at which these cells secrete milk is primarily dependent upon the amount of lactogenic hormone discharged at each milking period. If the udder is large (many secreting cells) each cell may not receive an optimal amount of the lactogenic hormone unless the pituitary secretes a large amount of hormone. Thus the rate of secretion of lactogen is reflected in the rate of secretion of milk by each cell. If the udder is small, then less lactogenic hormone is required to maintain optimum amounts of hormone in each cell and a deficiency in lactogen secretion is less likely.

With the advance of the lactation period, the continued secretion of the lactogenic hormone at a high rate is of great importance. In cows of high persistency of milk secretion, this seems to be possible, but in most cows there is a gradual decline in milk yield caused by the decline in the secretion of the lactogenic hormone.

Thus, in cows with the capacity of high yearly milk yield, it is important that the pituitary not only secrete large amounts of lactogenic hormone at the peak of milk secretion, but that the hormone continue to be secreted at a high level throughout the lactation period.

Selection of dairy cattle with high persistency of milk secretion in conjunction with the use of dairy bulls whose daughters have displayed a high average persistency should be effective in the genetic improvement of dairy cattle.

In subsequent bulletins, the role of other hormones in stimulating intense milk secretion will be described.

References


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