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SHOULD A BREEDER TELL?

by

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Breeders of stud stock should have one common end in view, the improvement of the particular breed in which they are interested. This object, however meritorious it may be, unfortunately has to take second place to pecuniary gain. In this country at least, most breeders must make their breeding operations pay, or else get out of the business. In order to make a living, the breeder has to give the public what it wants, though he may well know that something different would be asked for if the public were educated as to the real values of breeding stock.

Considering one branch of Animal Production, the Dairy Industry, the viewpoint of the rank and file has undergone considerable change during the last few years. The dairyfarmer now uses a pedigree bull, and looks for butterfat backing. He wants a bull from a dam with a high record of production and also expects the other females in his bull's pedigree to have proved themselves as milkers. In this respect, up to a recent date, one high record has been regarded as sufficient, and so the breeder has been encouraged to go in for C.O.R. testing. Under this system it is not necessary to test the whole herd and a few of the breeder's best cows may receive extra care and be fed to an uneconomic degree. The high records so obtained are certainly a good advertisement for the breeder, and may on this account justify the extra expense, but they tend to create the impression that all pedigree animals are superior animals. It is not generally realised to what degree almost any cow would increase her yield under better, though uneconomic, conditions. I wonder how dairyfarmers would react if they had the chance of reading Bulletin No.188 of the Iowa Experimental Station and could see the wonderful improvement in the milk yield of a scrub dairy cow under a superlative environment.

Under the C.O.R. system, the cows tested are not a fair sample of the breed, for the breeder naturally selects those animals that he thinks will do best under test. If one of these cows should not perform creditably, and this has been known to happen, her yield is not published, and in the case where she seems unable to come up to a comparatively low standard, her test is discontinued before completion. These failures are certainly not advertised, but they should be taken into consideration when estimating the value of their sires and dams.

The old system of phenotypic selection is giving way to the consideration of the genotype through the progeny test, and this test demands the disclosure of a breeder's failures as well as his successes. This sometimes places the breeder in an awkward position; to disclose his failures may injure the reputation of a fashionably bred sire and entail considerable financial loss. Very few breeders will publicly admit that they have any failures; is this a satisfactory position? One breeder thought that he was doing the right thing in getting rid of his duds and conveniently forgetting all about them. This gentleman was heard to remark "When I have any failures, I cut their throats and forgets all about 'em." He was probably unaware that he was doing as much harm to his own breeding operations as any one else's. Culling low producers will not produce a general improvement in germ plasm for higher producing levels. The poor cows will always be in the herds to cull unless the owners follow a breeding programme that will eliminate the germ plasm that is responsible for low production.

The dairyfarmer has been advised to line breed, so when the time comes to replace a bull that has been a successful sire, he goes back to the same breeder and buys a related animal. This is a sound practice, unless in the germ plasm of the favoured herd hidden imperfections, or even perhaps, lethal factors, exist.
There may, however, be a skeleton in the cupboard. The favoured herd may contain no low-producing animals, but if any have appeared, and have been quietly culled, or if any calves have been still-born or have died shortly after birth through abnormalities, the intending purchaser has the right to know.

One bull from such a herd would do little or no harm to the new owner's breeding operations, for it is not certain that such a bull would carry the undesirable factors, or even if he did, it is not certain that he would be mated with any cows that also carried the undesirable factors, and so disclose them in the offspring. But this has been known to happen. An unfortunate case occurred in the herd of one of our most successful and prominent dairy farmers. His herd contains a Jersey cow which probably holds a record for lifetime production. During her long life she has only presented her owner with three heifer calves, two of which had to be destroyed because of the fact that they were born with defective lower jaws. They were sired by the same pedigree bull.

But if a dairy farmer went back to such a breeder at a later date for other related bulls, to mate with the progeny of the earlier ones, then there is a danger. In support of this I will quote a letter to the Editor of the New Zealand Farmer Weekly, published in the issue of the 23rd January, 1941.

The writer says: "I have a number of heifers that have overshot top jaws (or should I say undershot?); the lips of each jaw are in some cases three inches apart. The second bull I used was sired by a full brother of the first bull, and my third was the same. I had no misshapen jaws until I used the latter bull. I have never mentioned this to the breeder of the bulls, as I have a great respect for him, but I hesitate to buy from him again. I have never seen any cattle like those mentioned; they all have a long, narrow nose, long from the eyes. (Signed) Farmer, Hawera.

There is also a danger if the dairy farmer inbreeds with the progeny of a bull from such a herd. In support of this I will quote from "NOTES ON A NEW RECESSIVE LETHAL FACTOR", a paper by Dr. H.E. Annett in the February issue of the Journal of Genetics.

"The author has been practising intensive inbreeding with a Milking Shorthorn bull. This appeared to be desirable since the latter's daughters proved to be far better producers than their dams. . . . . . . . the bull has sired 110 calves and 28 of these were out of his own daughters. Of these 28 calves five were abnormal, whereas no such abnormality appeared in the remaining 82 calves. The abnormality consisted in a deformity of the lower jaw, which was only half the normal length. Since such a calf cannot suck its dam, and, even if it survived, could not subsequently graze, we have apparently to deal with a lethal factor."

I must confess that I was the breeder of Dr. Annett's bull, but I can say in self-defence that I did not realise that the lethal factor existed in my herd. When Dr. Annett first informed me of the fact that he had recorded several cases of defective lower jaw in the calves of the bull that he had purchased from me, I was loth to agree that inbreeding had brought to light an inherent defect in my bull. I was inclined to attribute the condition to malnutrition of the dam during pregnancy, possibly through mineral deficiency. I was confirmed in this belief because I had never heard of the condition previously and could not remember a case occurring in my own herd, though for many years past I had been inbreeding very considerably. I recognised, however, that it was possible for a mutation to happen, giving rise to a single dose of the recessive gene in Dr. Annett's bull.

Shortly after this, Professor Snyder, of the Ohio State University, reported the discovery, in a Milking Shorthorn herd in central Ohio, of a recessive lethal factor which he named "parrot-beak". In the heterozygous condition the calves are perfectly normal, and show no evidence of carrying the lethal factor. In the homozygous condition the factor produces calves with an abnormally shortened lower jaw, in which the molar teeth are impacted. These calves never live more than a few hours and may even be born dead.

Though Dr. Annett had not noted the impacted molars, it appeared probable that the condition that he and Professor Snyder had discovered were one and the same. Then a case of "parrot-beak" occurred in my own herd, sired by the sire of Dr. Annett's bull. Other cases of "parrot-beak", including impacted molars, came to my knowledge, all in cattle bred from my stock.

For many years I had held a prominent place among the breeders of Milking Shorthorn cattle in New Zealand, and the fact that a hidden and unsuspected hereditary undesirable abnormality had been discovered in cattle bred by me, and descended from the most valuable blood lines in my herd, made it imperative that I should discover, if possible, to what extent the factor responsible was present in my herd, and from what source it had been introduced.

This led me to make a careful search through my records and to my surprise I found a solitary case had occurred some ten years previously, in 1928. This case had not impressed itself on my memory. At the time I had probably put it down to an accident in the development of the foetus, a somatic variation that was not hereditary.

Pedigrees are the basis of the study of heredity in cattle, but their value depends absolutely on the care with which the records have been kept from which they have been compiled. As many family histories as possible of the character to be studied should be collected. They must then be carefully examined to see if a reasonable explanation can be given of the method of inheritance of the character concerned. I know that I can trust my own records, and the charts that I prepared led without any doubt to the conclusion that the factor for "parrot-beak" had been introduced to my herd by the purchase of a Coates Herd Book bull in 1915. This is interesting, as it seems to indicate that the American cases and my own cases have a common origin. The American Milking Shorthorns are also founded on Coates Herd Book (British) stock, mainly of the Bates strain. Bates, who inbred to an extraordinary degree, may have experienced losses from "parrot-beak" in his famous Duchess family which is supposed to have died out from lack of fertility, intensified by excessive inbreeding. If so, Bates certainly kept his troubles to himself, as most breeders have done in the past.

The sire of the two cases of "parrot-beak" in my herd is dead and gone. I have had no other cases, though still inbreeding with the same stock. My comparative freedom from the condition can best be explained by the biometrician, but purchasers of my stock have not all been so lucky. Sixty odd sons of the bull referred to have been distributed throughout the length and breadth of New Zealand. I have not questioned all the purchasers but have learned of the appearance of "parrot-beak" in four local herds, where inbreeding has been practised with stock bred by me. In some of the cases, the ratios work out splendidly, showing without a doubt that the character is inherited as a simple autosomal recessive.

I have collected a considerable amount of material and have considered publishing it, but have been restrained by the fact that such publicity might cause financial loss to some of the purchasers of my stock. In New Zealand there has been, and still exists, considerable rivalry between two strains of Milking Shorthorns, one founded on Coates Herd Book (British) stock and the other founded on Darbalara and Illavarra (Australian) stock.
Recently, several Coates Herd Book bulls have been imported at considerable expense. Disclosure of the fact that "parrot-beak" has its source in Coates Herd Book stock may cause considerable loss to those importers.

My problem is certainly "SHOULD A BREEDER TELL".

As a dairyfarmer, "parrot-beak" has not caused me any financial loss, but as a breeder of pedigree cattle I should like to be free from it. On the assumption that the recessive factor for "parrot-beak" is still carried by some of my cows, and I cannot be certain that this is not so, I can only prevent its reappearance in my herd by being sure that my herd sire is free from it. By using a succession of such bulls I could eliminate "parrot-beak" altogether. So I have tested out my last two herd sires by mating them to their own daughters. This course is recommended by Wriedt. In his book "Hereditari in Live Stock" he asserts that until a bull has given at least twenty normal calves in matings with his own daughters he cannot be regarded as free from lethal factors.

Speaking of lethal factors Wriedt says: -(And he is worth quoting at some length).

"As a result of the relation between the prominent individuals of a breed we find that the genes of the great sires are widely spread throughout the breed. The recessive factors found in the great sires in a single dose will descend to one-half of the progeny, and they in their turn will hand the factor to one-half of their descendants.

Great sires, then, have the power of passing on their recessive genes to the entire breed. When a few generations have come and gone an attempt will be made to in-breed with the great sires as a basis, in many instances recessive homozygotes will then be derived. This is a fact of great practical importance, because lethal factors are very widely spread. Among cattle seven recessive lethal factors have been already noted, among horses one, among swine one, and among sheep two, and this in spite of the fact that investigations of lethal factors among live-stock are not very general.

It is evident that the presence of lethal recessive factors of the type here described involves a marked depreciation in value in all meat-producing animals. In such animals the offspring represent the only, or at any rate the main, product.

In milk-producing animals and in the horse the conditions are somewhat different. In milk-producing cattle the calf does not represent the all-dominating product, and in the horse the broodmare is a working animal as well as a breeder.

From a superficial view it might seem to be of relatively little importance that every eighth calf is lost in a herd of milk-producing cattle due to the presence of a lethal recessive. But the elimination of 20 calves in three years within a single herd - an actual case encountered in our investigation - cannot be regarded as unimportant from an economical point of view. Moreover, the possibility of more than one sub-lethal gene being present within the same herd cannot be disregarded. Though Mohr and I have not as yet discovered such cases, we found that both the gene for "hairless" and that for "amputated" have probably come together in one and the same herd.

However, the loss of every eighth calf is of great importance from a breeding point of view. In Denmark, one of the most prominent milk-producing countries, we may, according to Fredriksen, estimate that up to 20 per cent. of a herd are sorted out each year. In order to replace these individuals in a herd of 100 cows it is necessary to rear 22 heifer-calves annually. It must also be taken into account that at least 10% of the heifers are dropped due to disease or infertility.
before the first calving.

According to the Danish statistics for the year 1923, 86 calves per 100 cows were born. Half of these calves are heifers. Of these, some are apt to die from diarrhoea and other diseases shortly after birth. According to Fredriksen, about 38 viable heifers per 100 cows in one year may be regarded as fair output.

Provided that the rearing of heifer-calves from any cow in the herd could be recommended, this would represent an ample margin, and the loss of every eighth heifer-calf would be relatively unimportant from an economical point of view. However, herds are very rare in which every cow is satisfactory from a breeding point of view. In most herds one must be content if half of the cows are valuable enough as regards milking capacity and butterfat percentages to be used as breeders. Under these conditions less than 20 heifer-calves are at hand for rearing, and this number is too small for the renewal of the herd. It should be remembered that the general physical condition of the calf must be taken into account.

Thus in milk-cattle the occurrence of lethal factors is of much greater economical importance than indicated by the meat value of the eliminated calves. Moreover, the psychological consequences cannot be disregarded. The possibility of breeding expensive animals giving rise to malformed monsters and stillbirths is in itself apt to cause a marked fall in the prices paid."

Although I have had no recurrence of "parrot-beak" in test matings, I have recently recorded the birth of a heifer with a bob tail. I have not had it X-rayed, but on careful examination it appears to possess only two coccygeal vertebrae and these seem to be ankylosed. The question now arises as to whether this abnormality is due to a genetic cause or to some accident in development which is not hereditary. I am up against the same problem that arose when I experienced the first case of "parrot-beak". I cannot find any record of the birth of a bob tail calf, though a flock of "notail" sheep has been recently established in the U.S.A. at the South Dakota Experimental Station.

I shall perhaps never solve this problem, as the numbers that I deal in are so small, but if bob tail should be hereditary and other cases occur from similar matings, then perhaps I could establish a breed of bob tail cattle if such were desirable. The heifer seems to suffer inconvenience from the lack of a tail, and from the point of view of cleanliness it would certainly be an advantage in the milking shed. This and other problems raises the question:

"SHOULD THE BREEDER BE TOLD — AND HOW?"

The value of the New Zealand Society of Animal Production here becomes apparent. Here is a body that can review operations as at present being conducted, pick out what is faulty and suggest remedies. How to hammer this advice home is a very important problem which requires our immediate attention. By arriving at some method of reducing the time-lag between the new discoveries in the field of Animal Genetics and their application, we would be doing a service to our country, even in war time.