

Penicillin estimations were carried out on blood samples of twelve guinea pigs which had survived treatment with penicillin three weeks previously, and on twelve which had not been previously dosed, at one hour after an intramuscular dose of 20,000 units. There was no significant difference in the average levels of the two groups, these being 0.6 and 0.5 units respectively. The levels at three hours after inoculation had fallen to zero in all but six animals, which had a level of 0.15 units or less. These levels were unexpectedly low. Blood from guinea pigs injected four hours previously with penicillin was not toxic for normal guinea pigs in subcutaneous doses of 5-10 c.c.

P. STUART
G. SLAVIN

Veterinary Laboratory,
New Haw,
Weybridge, Surrey.
Oct. 17.

¹ Rivière, C., Thely, M., and Gauton, G., *C.R. Acad. Sci., Paris*, **244**, 1856 (1947).

² Hauduroy, P., and Rosset, W., *Ann. Inst. Pasteur.*, **75**, 67 (1948).

Genetic Variability and Artificial Insemination

IN *Nature* of November 11, S. L. Rabasa¹ discusses the reduction in genetic variability which can take place in a population bred by artificial insemination. The problem he raises is not peculiar to populations bred in this way, but is a general one which must be faced in any population in which the parents of later generations are selected out of the whole number of adults available. The reduction in genetic variability resulting from using only a small number of animals as parents, and the loss of vigour which usually accompanies the unavoidable inbreeding, has to be weighed against the benefits resulting from the genetic superiority of the parents selected—a genetic superiority which can be increased only if the selected fraction is allowed to decrease. Artificial insemination makes it possible to have a smaller ratio of parents to non-parents but otherwise leaves the situation unchanged.

Of course, this problem arises in closed populations only. In most countries at the present time the artificial insemination system is an appendage of normal pedigree breeding, and the bulls at artificial insemination stations are renewed each generation from the pedigree herds. It is the practice of the pedigree breeders which determines the genetic structure of the breeds, not the practice of the artificial insemination centres. However, when the great potentialities of the artificial insemination system as a method of constructive breeding are fully used, the problem will assume considerable importance.

The loss of genetic variation in small populations has been discussed extensively by Wright², who has shown that in a closed population with M males and F females between which mating is at random, the fraction of genetic variance lost per generation is $\frac{1}{8M} + \frac{1}{8F}$. As F is usually very much greater than M , the most important factor is, not the male-female ratio, but the actual number of males used in the population. It follows that the decline in variability is quite slow, even when the number of bulls is very small. Thus, a closed population, using two bulls at a

time, would take at least eleven generations to halve the genetic variance. Far more serious, in the early stages at any rate, is the decline in vigour which has been found to accompany the unavoidable rise in inbreeding. Experiments in dairy cattle are not sufficiently extensive to determine the exact rate of decline in yield or the extent to which it is an invariable accompaniment of inbreeding; but present indications are that a rise of 0.10 in the inbreeding coefficient is accompanied by a decline in vigour leading to a reduction of 2 per cent in milk yield.

Although there is a definite danger to be faced in increasing the intensity of selection through the use of artificial insemination, it is one which can be overcome by taking the appropriate steps. If a breed is divided into several sub-units, each centred on its own artificial insemination system, and if there is a small amount of controlled migration between centres, the danger, both of inbreeding and loss of variability, can be overcome. A fuller discussion of this problem can be found in a paper by Wright³ and a discussion with particular reference to dairy cattle in an article by Rendel and Robertson⁴.

J. M. RENDEL
A. ROBERTSON

Animal Breeding and
Genetics Research Organization,
Institute of Animal Genetics,
Edinburgh 9.
Nov. 30.

¹ Rabasa, S. L., *Nature*, **166**, 821 (1950).

² Wright, S., *Genetics*, **16**, 97 (1931).

³ Wright, S., Proc. 32nd Ann. Meeting, Amer. Soc. Animal Production, **18** (1939).

⁴ Rendel, J. M., and Robertson, A., *Scot. Agric.*, **30**, 79 (1950).

It has been stated by Rabasa¹ that in order to avoid inbreeding and the loss of genetic variability, artificial insemination should be used for a few generations only. It should be pointed out that much of his reasoning would already apply in most pure breeds of livestock, where Lush² has demonstrated for cattle, horses, pigs and sheep that, because of inbreeding, the number of males actually used is only equivalent to about twenty or thirty with equal chances of parenthood, though, of course, the census number is far larger. For example, from a study³ of the Shorthorn breed in Britain, the rate of increase in the level of inbreeding at the time of the study (1925) would correspond to an effective number of twenty-one bulls.

In spite of these small effective numbers, the level and rate of increase of inbreeding in these breeds are not high, and it should be possible while using artificial insemination in commercial herds to keep inbreeding permanently down to a 'safe' level. Robertson and Rendel⁴ have discussed progeny-testing with artificial insemination in dairy cattle as being the most promising method of improvement in Great Britain.

G. M. WRIGHT
Applied Mathematics Laboratory,
Crop Research Division,
Department of Scientific and Industrial Research,
Christchurch, New Zealand.
Jan. 18.

¹ Rabasa, S. L., *Nature*, **166**, 821 (1950).

² Lush, J. L., *Amer. Nat.*, **80**, 318 (1946).

³ Wright, Sewall and McPhee, H. C., *J. Agric. Res.*, **31**, 377 (1925).

⁴ Robertson, A., and Rendel, J. M., *J. Genet.*, **50**, 21 (1950).