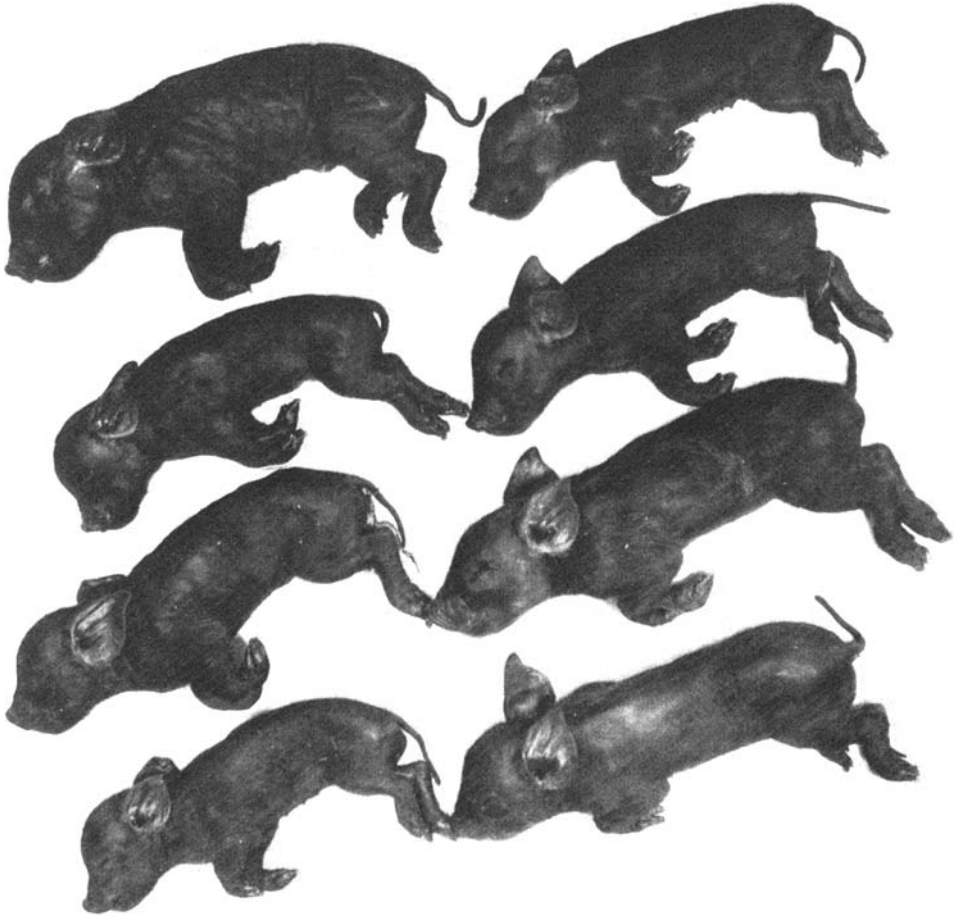


PIGS BORN WITHOUT EYE BALLS

FRED HALE

Texas Agricultural Experiment Station, College Station, Texas



A LITTER OF EYELESS PIGS

Figure 7

Progeny of normal Duroc-Jersey sow all showing complete absence of eye balls. Since both the parents were normal-eyed it is practically certain that this defect is not hereditary. It is rather to be taken as an example of the profound effect that vitamin deficiency may have upon development.

A REGISTERED Duroc-Jersey gilt receiving a ration deficient in Vitamin *A* at the Texas Agricultural Experiment Station, farrowed on March 29, 1932, eleven pigs, all of which were born without eye balls. Ten of the pigs were alive at birth, one

lived four days, one lived three hours, while the others died within five minutes after birth. The gestation period for the litter was 111 days, and their average birth weight before nursing was 1 pound 14 ounces. This gilt was placed on the vitamin *A* deficient ra-

tion when four months of age, and was bred 160 days later. She was kept in a pen with a concrete floor, and was fed in a self-feeder. Thirty days after being bred, she became too weak to get up. Cod liver oil was then supplied in daily doses of two ounces for 20 consecutive days. The gilt gained enough strength to move about the pen and eat regularly five days after she was started on the cod liver oil. After 20 days, the cod liver oil was given every other day for one week, after which it was discontinued, as the gilt appeared to be strong and had a good appetite. Forty-six ounces of cod liver oil were given in all. The litter of pigs was farrowed 53 days after discontinuing the cod liver oil.

Hereditary eye defects have been noted in rabbits by Guyer,* and there is also an "eyeless" recessive character in *Drosophila*, but a litter of eyeless pigs has not previously been noted.

Since both the sire and dam of this litter of pigs had normal eyes, this

defect, if hereditary, would have to be the result of a recessive factor, and in which case both sire and dam must have been heterozygous.

Assuming that both sire and dam were heterozygous for a single factor, the chance that all of the eleven pigs in the litter would be homozygous recessive for this character are only one in approximately four million.

Further evidence that this defect is not hereditary is furnished by one litter of eleven and one litter of eight pigs with normal eyes, sired by the same boar but farrowed by different sows.

While the cause of this abnormality has not been fully determined, and the study is being continued, evidence points to a vitamin *A* deficiency as the causal factor. The condition is illustrative of the marked effect that a deficiency may have in the disturbance of the internal factors that control the mechanism of development.

*GUYER, M. F. 1924) "Further Studies on Inheritance of Eye Defects Induced in Rabbits," *Jour. Exp. Zool.*, 38:449-474.



Sickle-Cell Anemia in the White Race

Rosenfield and Pincus [in *Amer. Journal of Med. Sci.*] state that a review of the literature of sickle cell anemia in the white race reveals only one previous case in which no evidence of a possible admixture of Negro blood can be discovered. A second case is cited in which the data are incomplete for such a conclusion. The authors report a third case in a family in which three generations show the sickling trait and at least five generations are known to be of the white race from a region where Negroes are practically unknown. The ethnologic and clinical features of the subject are discussed by the authors. They conclude that in the future more cases of sickle cell anemia in white persons will be discovered. The reasons for this statement are as follows: First, since attention has been called to the occurrence of the sickling trait in the white race, more

frequent examinations of the blood for sickle cells will be made, especially in those patients presenting the syndrome of an atypical hemolytic icterus. Thus, more cases of the type described by the authors may be discovered. Second, since it is known that the sickling trait is a dominant character in its hereditary transmission and since interbreeding between the Negro and the white races is more or less constantly taking place in many regions, including this country, one may in the future generations expect the presence of this peculiar blood trait in an increasing number of apparently white descendants. Because of the tendency to deny such descent, no history will be obtained of such racial origin in affected individuals, thereby increasing the number of apparently pure white cases of sickle cell anemia.

Journal Amer. Med. Assn., Feb. 4, 1933.