

Brief Communication

**ARTHROGRYPOSIS AND ASSOCIATED DEFECTS IN PIGS:
INDICATION OF SIMPLE RECESSIVE INHERITANCE**

In December/January of 1982/83, two litters of pigs containing piglets with congenital deformities were born in a pig herd in Trøndelag, Norway. Relevant herd data were obtained in cooperation with the local veterinary practitioner and the farmer. Piglets with gross anatomical defects were subjected to post mortem examination. These examinations were carried out either at the State Veterinary Laboratory in Trondheim, by the local veterinary practitioner or by the author. The herd, which was kept under modern conditions of husbandry, comprised 27 sows. The diet consisted mainly of a premixed fodder ("Svinefôr III") with a whey supplement and small amounts of herring meal and hay. Breeding was carried out both by artificial insemination and by use of a herd boar. Studies of the herd pedigree showed that this boar appeared both as the sire and as the great grandsire of the affected piglets (Fig. 1).

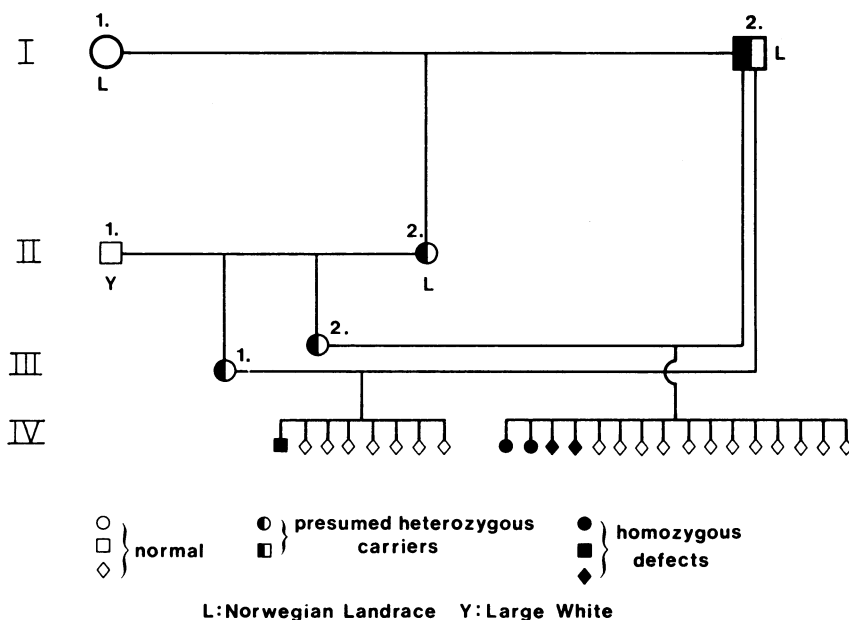


Figure 1. Pedigree showing probable inheritance pattern of arthrogryposis with associated defects.

The 2 litters concerned comprised 24 piglets in all. The 5 defective piglets were born to full term and were of normal size. They were all alive at birth but infirm to a varying extent. The following gross pathological changes were found on post mortem examination in all 5:

1. *Arthrogryposis*. Contracture of the flexor musculature in all 4 limbs resulting in fixation of the joints. The thighs were especially poorly developed. Articular surfaces and joint function, examined separately after cutting the tendons, were normal. In 2 of the piglets, the limbs appeared to be shortened.

2. *Brachygnathia inferior et superior*. Additionally, one of the piglets showed vertebral abnormalities. The tail was stunted and distorted. Radiographic examination revealed vertebral defects and there was discontinuity in the boundary area between the thoracic and lumbar column.

Arthrogryposis (crooked joints) is a common congenital defect often reported in man and animals (*Selby et al.* 1971), in which there is persistent joint curvature caused by muscle contracture (*Nes et al.* 1982). Though the condition may occur alone, it is most often seen associated with deformities of the jaw, the palate and/or the axial skeleton (*Huston et al.* 1978). As with most congenital abnormalities, both hereditary and environmental causes have been implicated in arthrogryposis. In contrast to other species, evidence for a hereditary basis of arthrogryposis (and associated defects) in pigs has rarely been reported in newer literature. More often cases being due to different environmental factors, such as plant teratogens and various drugs are described (*Apokodje & Barker* 1971, *Crowe & Swerczek* 1974, *Edmonds et al.* 1972, *Leipold et al.* 1973).

Idwall (1952) described cases with congenital deformities of the hindlimbs in Swedish large white pigs. The hindlimbs were misshapen (crooked). These defects were associated with abnormalities of the jaw. A simple recessive mode of inheritance was suggested, as all the piglets had been sired by the same boar and had the same maternal grandsire.

A probable mode of inheritance in the herd described above is shown in Fig. 1. The boar I.2 was both the great grandsire and the father of the affected piglets. A hypothesis of simple recessive inheritance (ratio of no. of malformed to no. of normals = 1:3) can be tested with the chi-square test. In this case there

were 5 malformed out of 24, and thus $\chi^2 = 0.22$ $df = 1$: $0.5 < P < 0.7$. The hypothesis cannot be rejected.

The sire I.1 has, according to the farmer, fathered more than 150 litters. No further piglets with anatomical defects have been born. Furthermore, each of the dams III.1 and III. 2 and their full sister (not included in Fig. 1) have produced 3 healthy, normally sized litters since the birth of the affected litters. This would seem to indicate that the genetic combination involved is the very factor which leads to manifestation of the defect. Several environmental factors have been described as causing arthrogryposis in pigs (*Huston et al.* 1978). In the present case it has not been possible to reveal any changes in nutrition or other environmental factors from the summer of 1982 onwards. The litters of generation IV in Fig. 1 are 25 % large white and 75 % Norwegian landrace crosses (the sire I.1 was a large white). The extent to which this might influence the manifestation of the defective genes, e.g. by omission of modifying genes, is impossible to evaluate on the basis of this material. Test matings were not possible, as the boar was slaughtered immediately after the birth of the first deformed piglets.

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