CONGENITAL HYPOPLASIA OF VULVA AND VAGINAL VESTIBULE IN PIGS.
R. H. C. NOGUERA*, M. A. G. CORDEIRO*, J. C. P. SILVA, and E. F. NASCIMENTO.
Veterinary School, Federal University of Minas Gerais, Brazil. Box 567.

The congenital defects in domestic animals constitute a group of pathologic entities with a broad anatomic, functional, and causal spectrum. Nevertheless, the predominant trend is to consider these defects an occasional occurrence, without other involvements except the eventual loss of some individuals. As far as swine is concerned, no less than 36 different alterations of this kind, regarding all the organic systems, eyes and chromosomal constitution were presented in recent review (Huston et al., 1970). The congenital malformation, which is the subject of this report, i.e., an hypoplasia of both vulva and vaginal vestibule, contrary to what commonly occurs, was characterized by a significant clinical expressivity and a numerous emergence in a short period of time. According to Niederle and Cohrs (1970), Roberts (1971), Johnson and Rendel (1972), Smith et al. (1974), the vulval and vaginal hypoplasia is a partial manifestation of hypoplastic female genitalia, as frequently reported in interspecies. Jensen and Kennedy (1970) without any reference to etiology, say that hypoplasia can affect either vulva or vagina, separately, but they do not register the species in which this alteration was detected. Hull et al. (1940) described the presence of intersexuality in bovine. Leopold and Saperstein (1975) the vaginal stenosis associated to ancrected stenosis, also in bovine. In connection with swine, Thonke (1967) reported a case of vulval hypoplasia thoroughly, and Hansen-Melander (1972) related a similar case, in a Landrace sow with normal karyotype. Thus, we can infer that the present paper register a congenital defect in swine not yet reported.

Twelve out of 22 cases of congenital anomalies detected were related with vulval hypoplasia. The boars (5) and the sows (60) did not show any phenotypic abnormality on visual examination. The data of the affected gilt presented a small opening, with a reduced diameter, and the urine was eliminated through a jet, which was some distance, with great difficulty. The morphoclinal significance of this abnormality was invariable. The post mortem examination of the affected gilts corroborated the definitive diagnosis of vulval-vaginal hypoplasia, since the vulval anomaly was associated with an equally slight defect in vaginal vestibule. The other parts of genitalia as well as the pair of gonads, were normal and structurally. Afterwards, through the farmer's filiation, it was remarked that the affected gilts, which belonged to seven litters, descended from two Landrace boars directly. Based upon the origin of the reproducers, the racial constitution of the swine herd, and the results obtained with the prophylactic measures, the most acceptable etiologic hypothesis is heredity. The genetic agent could be a mutant autosomal recessive gene, sex limited, with complete penetrance. The prophylaxis used consisted in sending the genetically infected animals (two boars and seven sows) to slaughter. Their respective descendants were kept for fattening and posterior slaughtering. These preventive measures resulted in the animals were followed-up for three years and no defect could be detected. In the literature that was consulted, no description of congenital hypoplasia comprising both vulva and vaginal vestibule in sows was found, except for some cases of intersexuality, usually accompanied by more severe genital malformations. As far as the form merely vulval of the abnormality is concerned, although uncommon, it was possible to obtain some information about its occurrence in swine. Thonke (1967) reported this alteration in a gilt, suggesting genetic etiology, since the same fact was observed in a complete sister and in a fullsister of the above mentioned gilt.

Unfortunately, the suspected boar was slaughtered before the hypothesis of genetic etiology could be raised, and consequently the crossbreeding tests could not be performed. In the present study, it was not possible to test both reproducers responsible for the outbreak of the cited defect, either. Another occurrence of vulval hypoplasia in sow was related by Hansen-Melander and Melander (1972). It concerned with a Landrace animal, whose vulva had a narrow diameter (2-3 mm) and the urogenital meatus besides an underdeveloped clitoris - was localized at one site of the vaginal vestibule. From this point on, no defect could be detected in the genitalia, and the karyotypic analysis showed a normal female with 38,XX chromosomes. Hull et al. (1940) described this defect regarding a descendent of a Jersey bull, which were as a last resort submitted to surgery on the occasion of parturiation. Leopold and Saperstein (1975), detected in three Jersey herds the existence of 14 cows with vaginal stenosis associated to ancrected stenosis. Paternal lines of all affected cows were traced to a common ancestor and those animals had very serious difficulties on the occasion of parturition. New authors, based on pedigree, suspected the presence of a recessive gene, with low frequency, inducing a clitoral manifestation of the femininity. Thus, although completely prejudicial to the species under natural conditions, the vulval hypoplasia in cow does not impair fertility.

REFERENCES