

Are Ridgling and Scrotal Rupture Caused by Abnormal Sizes of Inguinal Canal?

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Ridgling and scrotal rupture are two of the most common congenital anomalies in pigs. The incidence rate ranges from 0.20 to 1.38% for ridgling and from 0.06 to 2.56% for scrotal rupture, according to studies on different populations. Early studies attempted to use single-locus Mendelian models to explain the genetic mode of ridgling and scrotal rupture. The segregation analyses of more recent studies (e.g. Althoff 1995; Stigler et al. 1991; Thaller et al. 1996) generally ruled out the simple Mendelian inheritance modes and suggested polygenic genetic model for the anomalies.

Many researchers observed that ridgling and scrotal rupture were associated with extreme conditions of inguinal size (e.g. Warwick 1926; Ashdown 1963; Wensing 1975). A scrotal rupture develops when there is an abnormally large inguinal canal, through which intestines move into scrotum. Ridgling is a condition with abnormally small inguinal canal so that either one or both testicles fail to descend fully into scrotum. A normal inguinal canal allows only testicles, not other organs, to migrate from the abdomen into scrotum during development after birth. Based on the association between anatomical structures and anomaly conditions, some scientists hypothesized that ridgling and scrotal rupture are phenotypes of a single trait (referred to as 'inguinal size' here). Three phenotypes, ridgling, normal condition and scrotal rupture, can be observed for this hypothetical trait.

The anatomical cause of the two anomalies has been studied statistically based on the Data collected from a nuclear population of German Landrace including 104,006 piglets in 10,801 litters, originated from 136 boars and 4055 dams. The results showed that in spite of these associations between the anomalies and inguinal size, that ridgling and scrotal rupture are not attributable to oversize and undersize inguinal canals, respectively. Instead, the size of inguinal canal seems to depend on the conditions of the two anomalies, based on the evidences presented in this study: (1) If the two anomalies are the same trait, then ridgling and scrotal rupture correspond to a rough measurements of the hypothetical trait 'size of inguinal canal'. In linear model analysis, it would lead to a larger residual variance and a lower heritability estimator. In actuality, the estimated heritability in visible scale for ridgling (0.095) and scrotal rupture (0.106) is larger than that for inguinal size (0.084). (2) The estimated genetic correlation between the two anomalies is positive and ranges from 0.30 to 0.49. The positive genetic correlations of 0.2 and 0.73 were also reported by Mikami and Fredeen (1979) and Knap (1986), respectively. (3) The observed coincidence rate of the two anomalies in a common litter was significantly higher ($p < 0.001$) than the expected rate estimated under the assumption of independence between the two anomalies.