

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



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Introduction

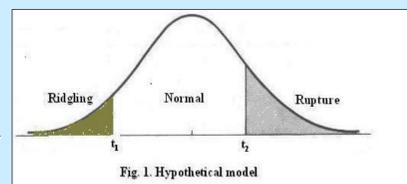
Many studies observed that ridgling and scrotal rupture were associated with abnormal size of inguinal canal (e.g. Warwick 1926; Ashdown 1963; Wensing 1975). A scrotal rupture develops when there is an abnormally large inguinal canal, through which intestines move into scrotum. Ridgling is a condition 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 the development after birth. Based on the association between anatomical structures and anomaly conditions, some scientists hypothesized that ridgling and scrotal rupture are caused by undersize and oversize of the inguinal canal, respectively. Ridgling and scrotal rupture are phenotypes of a single trait (referred to as “inguinal size” here). Three phenotypes, including ridgling, normal condition and scrotal rupture, can be observed for this hypothetical trait. The purpose of this study is to check this hypothesis based on a large data set of piglets.

Data

Data for this study were collected from a German nuclear population of Landrace during the period from 1979 to 1982. After editing, 104,006 piglets in 10,801 litters, originated from 136 boars and 4055 dams, were included in the analysis. ridgling and scrotal rupture were inspected and recorded shortly before weaning at 21 days of age.

Methods

Based on the hypothesis described above, the model in Fig. 1 was assumed: “Inguinal size” is a threshold trait of two threshold values, t_1 and t_2 . Its underlying liability is normally distributed and influenced by genetic effects and systematic and random environmental effects. An animal shows ridgling when its liability is smaller than threshold value t_1 , and shows scrotal rupture when its liability is larger than threshold t_2 . The animal is normal when its liability is between t_1 and t_2 . To check the hypothetical model, the data were analyzed as follows:



Coincidence rates of ridgling and scrotal rupture in litters were investigated. **Genetic correlations** between ridgling and scrotal rupture were estimated using the approach of Mikami and Fredeen (1979). The genetic correlations were estimated based on both full-sib and half-sib models.

Heritabilities were estimated for ridgling, scrotal rupture and the hypothetical trait, “inguinal size”. After model fitting process, the linear models used for the three traits are all the same. It included the fixed effect of litter size born, the fixed year and season effects, the interaction effect between years and seasons and the random sire effect. The residual effect was assumed to be identically independently normally distributed. The data were divided into parity 1 and parity 2 in this analysis.

Results

Coincidence rates of ridgling and scrotal rupture in litters: Of 10,801 litters observed, there are 9,717 litters with at least one male pig. 7.45% (724 litters) of these 9,717 litters have at least one ridgling and 9.11% (885 litter) have at least one pig with scrotal rupture. Under the assumption of independence between the two anomalies, it is expected that 66 litters (0.679%) have both ridgling and scrotal rupture. However, the coincidence of the two anomalies was observed in 106 litters, which is significantly higher ($p < 0.001$) than the expected number due to chance. Because a piglet is rarely affected by both ridgling and scrotal rupture, the incidences of the two anomalies happening in a litter are not independent. Once a ridgling appears in a litter, the probability that the litter has at least one piglet with scrotal rupture will be reduced. This means that the coincidence of the two anomalies is expected to be smaller than 66 litters. The observed coincidences of 106 litters are beyond explanation by random errors alone.

Genetic correlation on liability scale

The estimated genetic correlation between scrotal rupture and ridgling was 0.30 based on full-sib analysis and 0.49 based on half-sib analysis.

Heritabilities of scrotal rupture, ridgling and “inguinal size”: Heritability estimators are listed in Table 1.

Table 1. Heritability estimates of ridgling, scrotal rupture and “inguinal size”

	Ridgling	Inguinal size	Scrotal hernia
First parity	0.095	0.084	0.106
Second parity	0.083	0.066	0.070

Discussions and Conclusion

The results showed that in spite of the associations between the anomalies and inguinal size, ridgling and scrotal rupture are not attributable to undersize and oversize of the inguinal canal, respectively. The hypothesis that ridgling and scrotal rupture are phenotypes of a single trait is rejected, based on the evidences obtained in this study: (1) If the two anomalies are the same trait, then ridgling and scrotal rupture correspond to rough measurements of the hypothetical trait “inguinal size”. In linear model analysis, it would lead to a larger residual variance and a lower heritability estimator. However, the estimated **heritability** for ridgling and scrotal rupture is larger than that for “inguinal size”. (2) It is expected based on the hypothesis that ridgling (undersize inguinal canal) and scrotal rupture (oversize inguinal canal) should be negatively correlated. Actuality, the estimated **genetic correlation** between the two anomalies is positive. The positive genetic correlations of 0.2 and 0.73 were also reported by Mikami and Fredeen (1979) and Knap (1986), respectively. (3) Opposite to the expectation from the hypothesis, the observed **coincidence rate** of the two anomalies in a common litter was significantly higher ($p < 0.001$) than the expected rate under the assumption of independence between the two anomalies.

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