Introduction

Transmissible gastro-enteritis (T.G.E.) is a highly infectious viral disease of pigs characterised by vomiting, severe diarrhoea, high morbidity and mortality in pigs under two weeks of age. Pigs of all ages are affected within a susceptible herd.

The persistence of outbreaks and infection on pig farms is largely determined by the size of the herd and the nature of its production system. Underhill et al. (1) have shown that the carrier state may persist for more than one hundred days. Several workers have suggested that the term carrier pig may be an important source of virus and thereby provide the link between succeeding epidemics. (2) (3)

From this it would follow that moving recently recovered replacement breeding stock into susceptible herds is fraught with danger.

Practical experience in the U.K., however, does not substantiate those anachronistic and the circumstances surrounding the movement of recovered breeding stock, within ten weeks of their first clinical signs of T.G.E., into eighteen separate pig breeding farms is described.

History

Two Multiplier Breeder Units situated within the Surrey County of England, an area of high pig density, suffered outbreaks of T.G.E., confirmed in national laboratory testing, in the winter of 1968/69. Circumstantial evidence suggested that the infection introduced into these herds was either airborne or carried by birds. Each Multiplier Breeding Unit was subdivided into a breeding section and a gilt growth section. In each case, the gilt grower was affected at the outset.

Once the disease was suspected a standstill on breeding gilt sales was imposed and customers were advised. A planned exposure of stock in each section was undertaken to accelerate the onset of herd immunity, with a consequential loss of approximately 0.5% piglets per sow in the herd in each case.

Clinical signs persisted in the two gilt grower sections for ten and twelve days respectively. It was decided on a basis of previous field experience that breeding gilts, aged six to seven months, would be released for sale not less than forty days after the last clinical signs of the disease in each gilt grower section.

Outcome

During the first fourteen days after the resumption of sales from the two Multiplier Breeder Units, two hundred and sixty-six recovered gilts were supplied to eighteen commercial breeding units, together containing approximately four thousand sows.

The health history of these eighteen herds has been investigated and monitored over the subsequent twelve month period. It was found that one of these herds had been affected with T.G.E. in December 1968, being eleven months after an introduction of the recovered gilts, and at least three months since the recovered gilts were farrowed. Recently introduced breeding gilts, supplied to this herd by the original Multiplier Breeder Unit at the time of this outbreak, became clinically affected. Veterinary investigation of the circumstances surrounding the outbreak in this one affected herd from the sample of eighteen herds supplied, did not lead to the conclusion that infection had been introduced with replacement breeding stock.

It is normal practice within this national breeding pyramid to place newly introduced breeding stock into isolation for up to six weeks. The degree of isolation varies from farm to farm. It is common practice to introduce boars to these gilts while still in isolation. Such boars are, therefore, the first pigs in each herd to be the most closely exposed to potential carrier gilts. Most of these boars have now been blood sampled and laboratory testing disclosed no evidence of virus conversion.

The gilts supplied have farrowed down alongside susceptible sow and litter in the eighteen clean herds without initiating clinical disease resembling T.G.E.

Discussion

The dilemma presented to the veterinarian advising in the commercial field revolves around the problem of the infective carrier animal.

It is postulated that the persistence of the carrier state in recovered pigs is variable between those herds in which the disease was recently epidemic and those herds in which the disease is endemic. The factors considered in making a decision to release recovered gilts for sale include the presumption that T.G.E. is not endemic within the herd of origin.

Gilts do not necessarily seroconvert upon exposure to T.G.E. (4). Thus, the lack of positive titres in our "pedigree" boars may be of little significance.

Furthermore, clinical impressions have led to the hypothesis that within extensive management systems, gilts may become infected upon planned exposure to infection, but fail to become clinically affected until subsequently subjected to the consequences of farrowing (5).

It was postulated that each supplied herd would be at risk when recovered gilts first farrowed down in their new herds. The absence of clinical signs over the period of monitoring had extended into a winter when the incidence of T.G.E. in the Eastern Counties of England was high.

The complexities underlying the epidemiology of T.G.E., however, emphasize the need to view these findings with caution. They must be set in perspective against the background of present knowledge and in recognition of the needs for future research.

Selected References: