THE CHANGING INCIDENCE OF LEFT DISPLACED ABOMASUM AND UTERINE TORSION SINCE THE 2001 UK FMD OUTBREAK

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Introduction: The UK FMD epidemic of 2001 was the largest the world had ever experienced (Rossides, 2002), resulting in the slaughter of over 4 million animals in support of the control strategy and a further 2.5 million on welfare grounds (Kitching et al 2005). Inevitably a tragedy on this scale ushers in an era of change. Impact analysis assesses the response in a time series to a discrete event or intervention input (Makridakis and Wheelright 1987). This article describes an approach to quantifying the impact of the outbreak on left displaced abomasum (LDA) and uterine torsion diagnoses made by NADIS, a network of 40 veterinary practices and 6 veterinary colleges monitoring diseases in cattle, sheep and pigs throughout the UK. Since its inception in Jan 1997 it has returned monthly counts of veterinary treatments for endemic disease. The object of this study was to test the null hypothesis that there had been no change in incidence or seasonality of LDA and uterine torsion from before the 2001 FMD outbreak to after.

Materials and methods: The NADIS data from January 1997 to December 2007 was analysed, bracketing the 2001 UK FMD outbreak (19th February 2001 to 30th September 2001). During that period the population of dairy cows serviced by NADIS vets remained roughly stable at 100,000 cows (M: Howe, pers.comm.). A general model for a time series with a single intervention is given by the following formula:

\[ Y_t = m_t + N_t \]

where

- \( Y_t \) = count of outcome variable at time t
- \( m_t \) = the change in the mean function attributable to the intervention
- \( N_t \) = the underlying, unperturbed time series usually modeled on the pre-intervention data using an ARIMA process.

There are two types of intervention, a pulse intervention for a one-time event such as a disease outbreak where the input variable has a value of 1 for the period of the epidemic and 0 otherwise, or a continuing intervention, such as a law change, that assigns a value of 1 to the input variable after the date of the law change. Examination of time series plots indicated that after FMD the change in LDA and uterine torsion incidence appeared permanent, so a continuous intervention was used in both models. All analyses were undertaken using R v2.10.1 (R Development Core Team 2004). The Box-Cox method was used to select the best variance stabilising power transformation for each dependent variable. LDA data were transformed using a power of \( \lambda = 0.2 \) and uterine torsion data required log transformation. A Box-Jenkins ARIMA model was fitted to the transformed pre-FMD data and the estimated ARIMA values were then fitted to the whole data set. Examination of the residuals, figure 1, showed that neither LDA nor uterine torsion was well described using the pre-FMD models. Both residual plots contained extensive runs of positive residuals after the FMD outbreak. A new ARIMA model was re-fitted to the whole LDA or uterine torsion data set including a post-FMD dummy variable with a value of one from October 2001 through to December 2007. Plots of the average diagnoses for the periods Jan 1997 to Dec 2000 and Jan 2002 to Dec 2007 were made to check for any changes in seasonality. The strength of the observed seasonality was assessed using the coefficient of determination of the autoregressive regression model (R2Autoreg) fitted to the data (Moineddin et al 2003).
Results:

Figure 1 Residual time series plots from ARIMA models fitted to pre-FMD data for a) Left Displaced Abomasum diagnoses b) Uterine torsion diagnoses per month. The period of the 2001 FMD outbreak is shown hatched in grey. The final models fitted to both data sets were a first-order autoregressive non-seasonal Box-Jenkins model with the following formula: $Y_t = \delta + \phi Y_{t-1} + wE_t + at$

Where:

$Y_t$ = count of diagnoses at time $t$ and $Y_{t-1}$ = count of diagnoses at time $t-1$

$\delta$ = intercept, $\phi$ = autoregression coefficient and $w$ = dummy variable coefficient

$E = 1$ if $t$ = October 2001 to December 2007 otherwise $E = 0$

$at$ = random error at time $t$

Model output for LDA data (SE)

$\delta = 1.8623 \ (0.0385)$

$\phi = 0.4112 \ (0.0794)$

$w = 0.3726 \ (0.0512)$

Model output for Uterine Torsion data (SE)

$\delta = 2.066 \ (0.0816)$

$\phi = 0.3003 \ (0.0834)$

$w = 0.3012 \ (0.1086)$

After the FMD outbreak there was a 150% increase in LDA from 22.4 to 55.8 diagnoses per month and a 35% increase in uterine torsion diagnoses from 7.9 to 10.7 per month. Both the absolute number of uterine torsion cases and the proportion of total dystocia caused by uterine torsion increased, from 11.3% (95% CI 10.0-12.6) pre-FMD to 15.5% (95% CI 13.9-17.2) post-FMD, $P = 0.0001$. The goodness of fit of the models refitted with a dummy variable was assessed using the log likelihood test, both were highly significant ($P < 0.00001$).
For LDA and uterine torsion the overall $R^2_{Autoreg}$ was 0.69 and 0.43 respectively, indicating moderate to strong seasonality. Changes in seasonality after FMD were moderate. For LDA, the late summer peak became more pronounced (figure 2a). Uterine torsion became more seasonal with a pronounced July peak (figure 2b).

**Discussion:** Reports of both conditions increased after the FMD outbreak, particularly LDA. Lawrence et al, 2010 attributed causality to the relationship between the FMD outbreak and a subsequent 60% increase in mastitis treatments by NADIS vets. For LDA and uterine torsion, though, the most logical explanation for the observed increases is not a causal link but that the cows, farming practices or both changed markedly after the outbreak. The exact nature of these changes is unclear. Both LDA and uterine torsion are strongly associated with calving but clearly peak at different periods of the year (figure 2), and therefore must have some different risk factors. A change in the age of the population is one potential cause, as there was widespread retention of older cows following the outbreak (Mason 2005). Another potential link is abdominal space. Aubry et al 2008 found that cows were 5 times more likely than heifers to develop uterine torsion. They suggested that this was a function of size with cows having a larger abdominal cavity. The development of an LDA also requires abdominal space to allow abomasal migration, so larger cows may be at greater risk for LDA. The two diseases may also be linked by a fetal size effect. Frazer et al 1996 showed fetal size influenced the risk of uterine torsion; for LDA, there is no proven direct link, but, as a key factor in the development of LDA is reduction in rumen size because of fetal and uterine growth, larger fetuses may impact more on rumen size and thus increase abdominal space post-partum. Larger fetuses also increase the risk of other peripartal diseases, e.g. dystocia, RFM, indirectly increasing the risk of LDA (Correa et al 1993). Nutrition is also likely to be important. Shaver, 1997 showed that poor nutritional management, particularly excessive concentrates pre-partum resulted in reduced rumen fill and decreased abomasal and ruminal motility. As well as influencing LDA risk, these factors could also impact on uterine torsion because of their impact on abdominal space. Further research is required to better understand the common risk factors between LDA and uterine torsion. Future work with this dataset will involve examining the data at the practice/vet level looking for evidence of reporting bias or regional differences.

**Conclusion:** The incidence of LDA and uterine torsion has increased substantially on farms serviced by NADIS veterinarians since the 2001 FMD outbreak. This increase is probably the result of changing feed practices combined with increased cow size.

**Key words:** changing incidence, left displaced abomasums, uterine torsion, UK FMD outbreak.

**References:**


