ABSTRACT Based on the daily records on turkeys' mortalities for the series of flocks placed on different farms in a relatively compact geographical area for the period of approximately 2 yr and other relevant explanatory variables, the goal of the research was to design a decision model to determine whether or not to use the fluorquinolone antibiotic, sarafloxacin, to prevent spiking mortality of turkeys. The core of the designed decision model is the forecasting model that attempts to ex-ante predict the cumulative flock mortality for the period between 8 and 28 d of age. Forecasts were generated with the parameters of the linear regression model where continuous values of daily mortalities served as a dependent variable. The decision variable is a binary (yes/no) choice variable, where “yes” means “go ahead with treatment” and “no” means “do nothing”. If the predicted cumulative mortality for the period between 8 and 28 d of age exceeds 9% of the total initial placement, the model generates a “yes” signal. If the predicted cumulative mortality for the same period is below 9% of the total initial placement, the model generates a “no” signal. The results indicate a reasonable accuracy of the prediction model where the number of correct prediction increases and the number of incorrect predictions falls very fast as the forecasting window shortens. The intervention decision model could help veterinarians in making decisions on whether or not to treat the suspect flocks.

(Key words: Poult Enteritis Mortality Syndrome, intervention decision model, forecasting, turkey)

INTRODUCTION

In late summer of 1991, an apparently new disease affecting young turkeys characterized by sudden, high mortality occurred in a few flocks in a geographically restricted area along the North Carolina/South Carolina border. Because of the clinical similarity of this disease to spiking mortality of broiler chickens (Craig, 1991; Davis et al., 1996) it became known as spiking mortality of turkeys (SMT). However, preliminary findings suggest that the two diseases are likely quite different from each other. During the next 2 yr, although more flocks were affected, the disease remained localized to the same area. Since 1994, there has been a marked increase in the occurrence of the disease and it has affected flocks in all turkey-producing areas of North Carolina and in several Southeastern states.

Spiking mortality of turkeys is defined as an infectious, transmissible disease, of unknown cause that affects turkeys primarily between 8 and 28 d of age characterized by mortality equal to or exceeding 9% during this period with at least 3 consecutive d of mortality equal to or exceeding 1%/d. A milder form of SMT was suspected from epidemiological studies and subsequently confirmed by placing sentinels in affected flocks. This form of the disease was named excess mortality of turkeys (EMT), which is defined as a mortality between 2 and 9% during the 8 to 28 d age period without 3 consecutive d with mortality equal to or greater than 1%/d. To encompass both the SMT and EMT forms, the name Poult Enteritis Mortality Syndrome (PEMS) was proposed and adopted by the turkey industry. Because of recent management changes that result in turkeys being exposed at an older age, the disease may occur outside of the normal 8 to 28 d age period. Severe diarrheal disease and stunting occur when older birds are affected, but mortality tends to be lower.

Spiking mortality of turkeys occurs most commonly in the late spring and summer, typically appearing in the 21st wk of the year. It has been estimated from
mortality records that around a quarter million turkeys died from SMT during the summer of 1994. One company reported losses of nearly a million dollars a week from SMT during the summer of 1995. Losses from morbidity ("stunting") likely exceed those from mortality. It is feared that SMT will likely continue to occur each year until effective measures to prevent and control it are developed. Although several approaches to control SMT, or lessen its impact on a flock, have been tried, only the use of a fluoroquinolone antibiotic, sarafloxacin, has shown any benefit. Treatment with sarafloxacin substantially reduces mortality, but has little impact on morbidity (unpublished data). Because diarrheal diseases are so common in young turkey flocks and most are not SMT or PEMS, it would be beneficial to be able to predict which flocks are likely to have SMT in order to effectively use the antibiotic. The judicious use of antibiotics both from the cost-efficiency and the selection of resistant strains of bacteria standpoints cannot be overemphasized.

The cause of SMT is unknown. One or more viruses are thought to be responsible for initiating the disease and causing stunting and immunosuppression; secondary bacterial proliferation in the immunologically impaired turkey is considered the cause of the high mortality. Initially there was evidence of possible alphavirus infection (Ficken et al., 1993) but continued examination of birds from affected flocks has failed to confirm a role for this group of viruses. More recently, coronaviruses have been associated with affected flocks (Goodwin et al., 1995). Clinical findings of high, sudden, unexpected mortality suggested a toxicity but there has been no evidence to support this suspicion. All available evidence indicates SMT is caused by infectious agents but nutritional or environmental factors may contribute to the severity of a given outbreak.

Why the disease occurs most frequently during the warmer periods of the year remains an enigma. Darkling beetles have been found to be capable of transmitting enteric disease agents of turkeys (Despins et al., 1994). Other arthropods, especially flies, are suspected of also being capable of carrying the infectious organisms, which could account for outbreaks occurring so frequently in late spring and early summer.

In order to use the antibiotic effectively to control the disease, the first step is to design an intervention decision model. The core of the decision model is a forecasting model that relies on the daily disease dynamics to ex-ante predict the cumulative turkey mortality for the period between 8 and 28 d of age. The next section discusses the model and the data in detail. The last section presents the estimation and prediction results.

**MATERIALS AND METHODS**

**Modeling Spiking Mortality**

Designing a forecasting model involves several steps: selecting the format for the dependent variable, choosing the set of explanatory variables, determining the model’s functional form, and making assumptions about the statistical properties of residuals. The structure of the specified model will determine the choice of the appropriate econometric technique to be used to estimate the model’s parameters.

In this study, we use a standard linear regression model in which the dependent variable assumes continuous values of daily mortalities. Based on what has been known about the disease so far, we hypothesize that daily mortality can be explained by a set of explanatory variables including its own history (i.e., lagged daily mortality values, history of the disease on the farm, and presence or absence of the disease in the preceding flock), temperature, humidity, flock size, and the presence and the number of overlapping generations on the farm. Controlling for those affects, in all other aspects, we assume that farms and flocks are approximately homogenous. Given the geographical proximity of the farms and relatively similar technology they use, the assumption that the differences among farms and flocks are negligible does not seem overly restrictive. This assumption allows us to pool the data from different farms and different flocks together by vertically stacking daily mortalities into one column vector.

For the purposes of explaining the incidence of mortality today (and then predict the occurrence of mortality tomorrow), we exploit information in the historical data on mortality through the use of three groups of explanatory variables. First, we use lagged daily mortality values. Due to the nature of the data set, in which observations from different farms and different flocks were pooled together, using lagged values of the dependent variable as explanatory variables would cause unwanted causalities. For example, at the point where the first flock ends and the second flock begins, the behavior of the second flock’s mortality would be erroneously explained by the first flock’s characteristics. In order to avoid this problem, the data set was manipulated by deleting the necessary number of observations (depending on the number of lags included in the model specification) at the beginning of each flock such that each flock’s mortality is explained only by its own lagged values. An empirical question to be answered here is how many lags (days) to include in the model specification. Ideally, this should be determined based on the observed characteristics of the disease. However, at this point too little is known about the daily dynamics of the spiking mortality, and we had to rely solely on the statistical techniques. Secondly, we use dummy variables to single out farms with the previous incidence of the disease on the farm. The variable assumes the value of 1 for farms on which SMT had previously occurred, and 0 in cases where there was no history of SMT on the farm. Finally, we use another set of dummy variables to identify flocks immediately following flocks in which SMT had occurred. The variable assumes the value of 1 for the flock whose immediately preceding flock was diagnosed with SMT.
and 0 elsewhere. The expected signs for all those variables are positive, meaning that we anticipate the current incidence of the disease to be positively correlated with its own history.

The second critical set of explanatory variables are meteorological observations. The main characteristic of the disease is its seasonality. The correlation between warm weather and the incidence of the disease has been well documented. Additionally, a pronounced mortality has been noticed in periods of unusually high nightly temperatures or abnormally high lows. Based on these observations, two different explanatory variables were included in the model. The first one measures the maximum temperature recorded on a given day, and the second measures the difference between the maximum and the minimum temperature on a given day. Maximum temperature serves as a proxy for seasonality, and the range variable measures the importance of the day and night temperature differences on mortality. We expect the sign of the maximum temperature to be positive and the sign of the temperature range variable to be negative. In other words, as the maximum temperature increases, mortality of turkeys should rise, and as the temperature range increases (i.e., the nights get cooler), the mortality decreases.

In addition to the positive correlation between temperature and mortality, it has also been hypothesized that humidity or dew point may be important factors in explaining the outbreak of the disease. To test these hypotheses, two explanatory variables were included in the regression model: relative humidity and dew point. The expected sign on both variables is positive, which is to say that we expect mortality to increase as humidity and dew point rise.

The third group of explanatory variables is of secondary importance but can provide valuable information for understanding the disease through testing whether hypothesized variables have statistically significant effects on daily mortality. The flock daily size variable was included in the model to capture the effects of factors such as congestion and litter moisture. The variable was defined as the difference between the total initial placement and cumulative mortality up to that day. The expected sign for the daily size variable is positive. Finally, we also experimented with the data on overlapping generations. By recording the dates of entry and exit of individual flocks, we were able to count the number of different turkey generations simultaneously present on each farm. This information was included in the model through the use of binary (dummy) variables measuring the simultaneous presence or absence of different age cohorts on a given farm. We expect multiple age cohorts farms to be more likely affected by the spiking mortality than single age cohort ("all in – all out") farms.

**Data Description**

The data set used in this research contains observations on daily mortalities from 1,050 turkey flocks collected on farms in the southwestern part of North Carolina (mainly Union county) in the period between December, 1993 and November, 1995. Observations were collected during the first 42 d of each flock. Of 1,050 flocks, 64 satisfied the definition of spiking mortality in turkeys (i.e., with the cumulative mortality between 8 and 28 d of age in excess of 9% of the initial placement). For the entire sample period, the mean, standard deviation, and minimum and maximum of cumulative mortality rates for flocks with no SMT were 2.06, 1.64, 0.00, and 8.81, respectively. These values increased to 16.46, 7.99, 9.01, and 46.40 for the SMT flocks. The time pattern of cumulative mortality rates by flock is presented in Figure 1. The statistics show that the
summer increase in the mortality rates culminates in June and July. The mortality actually begins to increase in April and returns to its normal level in September. Whereas the first 3 and the last 4 mo of the calendar year are not statistically different from each other, the difference between the monthly means for the April to August period is statistically different from other monthly means.

Aside from the daily mortality data, we also collected data on outside temperature, relative humidity, and dew point. Outside temperature data consist of daily minimum, daily maximum, and mean data obtained from the weather station in Monroe, NC. Relative humidity and dew point data are daily observations for the Charlotte, NC area. The summary of the monthly averages of daily mortalities and temperatures are presented in Table 1.

The data used for estimation of the linear regression models' parameters contains 34,818 observations on daily mortalities from 829 turkey flocks. The remaining 9,282 observations at the end of the period (221 flocks) were set aside to be used in the out-of-sample prediction and model validation. Among these 221 flocks, there were 13 cases of SMT.

### RESULTS AND DISCUSSION

#### Model Estimation

The standard linear regression model used in estimation is of the following form:

$$Y_t = X_t' \beta + \epsilon_t, \quad t = 1, 2, \ldots, n$$  \[1\]

where it is assumed that vector of explanatory variables $X_t$ is nonstochastic; coefficient vector $\beta$ is constant over time; and $\epsilon_t$ is a Gaussian random variable with zero mean, variance $\sigma^2$, and the covariance Cov($\epsilon_t, \epsilon_{t-j}$) = 0 for $j \neq 0$.

Numerous model specifications were tried. The ordinary least squares (OLS) estimates of the three best models are presented in Table 2.

The obtained results seem to indicate that daily mortality can be best explained by the previous mortalities $1$ ($Lmort1$), $2$ ($Lmort2$), $3$ ($Lmort3$), and $4$ d ago ($Lmort4$), maximum daily temperature ($Tmax$), the difference between maximum and minimum daily temperature ($Range$), daily flock size ($Size$), and the history of the spiking mortality on a given farm ($DfarmS$). The adjusted coefficient of determination of 0.6759 indicates that close to 68% of the variability in the dependent variable (mortality) was explained by the model, i.e., by the selected explanatory variables. Most of the variables are significant ($P < 0.01$) and have the expected sign.

The decision to include only 4 d lagged mortality in the model was made solely on the statistical grounds. The application of the Box-Jenkins (1976) approach to randomly selected flocks indicates, in general, that the first four lags are more important. The fifth and sixth lagged values of daily mortality, when included in the model, turned out to be statistically insignificant. An interesting
and puzzling result is the negative and statistically significant coefficient on Lmort3. Unlike other lagged mortalities, whose impact on the current mortality seemed to be positive, the mortality of 3 d ago exhibited negative impact on today’s mortality. If an effect of some treatment takes on average 3 d to materialize, the negative sign of Lmort3 may be capturing that effect.

The effect of maximum temperature on daily mortality is positive, as expected, and the sign of the temperature Range coefficient is negative. Seasonality of the disease is best described by interpreting these two meteorological variables together. As weather gets warmer, we expect to see an increase in daily mortality. However, if the difference between night and day temperatures is large, the relative effect of warm weather decreases. Alternatively, the Range coefficient may be interpreted as an indicator of humidity in the sense that a higher range of daily temperatures signals a lower humidity. In this case, as the humidity decreases, the relative effect of high temperatures also decreases. The magnitude of the Tmax coefficient indicates that if the maximum temperature increases by 1 F, other things being equal, we expect additional 0.148 turkeys to die. The effect of the Size variable is positive, as anticipated, and significant. It is true that mortality increases with density; however, the estimated magnitude of the effect is extremely small. The dummy variable DfarmS, defined as 1 for farms on which SMT has previously occurred, and 0 in cases where there was no history of SMT on the farm, is positive. Two models using dew point (Dewp) as one of the explanatory variables were also tried; one including the maximum temperature (Tmax) and the other including the average daily temperature (Tmean). In both cases, the dew point coefficient ended up being insignificant.

We also experimented with other explanatory variables such as relative humidity, dew point, dummy variables for flock histories of SMT and EMT, dummy variable for the farm history of EMT, and dummy variables for the presence and the number of overlapping generations on the same farm. However, none of these variables contributed significantly to explaining the behavior of the dependent variable, so they were excluded from the version of the model used for forecasting.

**Predictions**

Estimated models from the previous section can be used to generate out-of-sample predictions of their respective dependent (left-hand-side) variables. The model actually used to generate predictions was Model 1. In the linear regression model, the dependent variable was defined in terms of daily mortality. The prediction of the occurrence of spiking mortality was constructed by adding up individual daily forecast from 8 until 28 d of age. If the sum of individual daily predictions between Days 8 and 28 exceeds 9% of the initial placement, the model generates a “yes” signal, otherwise the model generates a “no” signal.

For simplicity, assume that the true model is given by the following model:

\[ Y_t = a + b_1 Y_{t-1} + b_2 X_t + u_t \]  

where \( Y_t \) is the dependent variable (daily mortality); \( Y_{t-1} \) is the lagged value of daily mortality; and \( X_t \) is another exogenous independent variable, say, temperature. The estimation of the model [2] will produce estimates of the coefficients \((a, b_1, b_2)\). Hence, the one-period-ahead prediction (forecast) of \( Y_t \) is given by:

\[ \hat{Y}_{t+1} = a + b_1 Y_t + b_2 \hat{X}_{t+1} \]  

where variables with hats denote predictions. Notice that in order to predict \( Y_{t+1} \) one needs to know \( Y_t \) and \( X_{t+1} \). The value of \( Y_t \) is the current value of \( Y \) and hence it is known. The value of \( X_{t+1} \) is the future value of \( X \) and hence it is not known and has to be predicted. Following the same routine, the two-period-ahead prediction of \( Y_t \) becomes:

\[ \hat{Y}_{t+2} = a + b_1 \hat{Y}_{t+1} + b_2 \hat{X}_{t+2} \]  

Notice that in this case, to generate a two-step-ahead forecast of \( Y_t \), we need not only the two-period-ahead forecast of \( X \) but also the one-period-ahead forecast of the dependent variable \( Y \) obtained previously by expression [3]. The same routine can be repeated if one wants to generate predictions further out into the future. For example, standing at Day 7 and having all information available up to that time, we can forecast the entire 8 to 28 d window and sum up individual daily mortality predictions to obtain the prediction of the cumulative mortality needed to forecast the SMT. One can easily see that the quality of forecasts as measured by the prediction error is going to deteriorate as one forecasts further into the future.

As mentioned earlier, the data on 221 flocks were saved for out-of-sample validation. The predictions are generated by using the observed values for Tmax and Range instead of the required out-of-sample forecasts. This restriction is fairly mild, as the contemporaneous correlation coefficient between daily temperatures in 1994 and 1995 equals 0.7316. The initial flock placement is known at Day 1, so this number is always available at the beginning of the forecasting exercise. The first set of predictions starts at Day 7 and predicts mortalities from Day 8 to 28, the second set of predictions starts at Day 8 and predicts Day 9 to 28, and the last one starts at Day 26, predicting only Days 27 and 28. In addition to generating point predictions, we also calculated their 95% prediction intervals. If the upper prediction limit of the cumulative mortality exceeds 9%, the model would generate a “yes” signal. The problem in constructing the prediction interval is how to obtain the correct standard errors because the model predicts the daily mortality rates, whereas the required cumulative mortality rate is obtained by summing up daily mortalities from Day 8 to 28.
One approach to solving this apparent problem is to bootstrap the prediction interval. The main part of the data set used to estimate the model parameters containing 829 flocks was used to generate in-sample predictions and compare them to their respective true numbers. The procedure was repeated 20 times, i.e., for each day between Days 7 and 26. For each of the 20 iterations, 829 prediction error bootstraps were generated. The empirical distribution of the bootstraps enables the calculation of the standard error of these prediction errors. These standard errors were then used in constructing a 95% prediction interval as the upper and the lower limits of the generated out-of-sample point estimates described earlier.

The summary of the prediction results is presented in Table 3, where we report the out-of-sample predictions made at Days 7, 11, 15, 19, 23, and 26. The results indicate that as we try to predict more distant future the prediction interval gets wider and the model loses its predictive power. For example, at Day 7, the model predicts 100% of the observed SMT cases correctly. However, the model also incorrectly predicts the occurrence of SMT 188 times in cases when it actually did not happen. Therefore, part of that relatively high percentage of correct SMT signals is the consequence of the extremely wide confidence interval. At Day 11 the model correctly predicted 69.2% of the SMT cases (which is less than Day 7) but overprediction declined to only 56 wrong signals. At the same time, the model correctly predicted 73.1% of the “no” signals (less than 9% cumulative mortality) with only 7 false alarms. Therefore, part of that relatively high percentage of correct “no” signals is the consequence of the extremely wide confidence interval. As we get closer to the expiration of the time window, the predictions improve. At Day 23, the model generates “yes” signals with 84.6% accuracy with zero false signals. At the same time, the model generates “no” signals with perfect accuracy with only two wrong signals. At Day 26, the model again predicts all 13 of the observed SMT cases correctly, but this time the number of wrong “yes” signals is only 1. As far as the “no” signals go, the model accurately predict 99.5% of the 208 flocks with no SMT with zero wrong signals.

In summary, the predictive power of the model increases as we get closer to the Day 28. The results indicate that the number of correct predictions increases and the number of incorrect predictions falls very fast as the forecasting window shortens. The presented model could be used by veterinarians or flock managers to make delicate decision whether to begin the expensive antibiotic treatment of suspect flocks or not. The model is far from being perfect but provides a reasonable decision making tool in absence more reliable predictors.

REFERENCES