A model for evaluating intervention strategies to control salmonella in the poultry meat production chain

M. J. NAUTA*, A. W. VAN DE GIESSEN AND A. M. HENKEN

Microbiological Laboratory for Health Protection (MGB), National Institute of Public Health and the Environment (RIVM), P. O. Box 1, 3720 BA Bilthoven, The Netherlands

SUMMARY

A model of the transmission of salmonella through the poultry meat production chain is developed, to predict the effects of intervention strategies for salmonella control. The model first describes the situation before intervention in terms of salmonella prevalences at flock level and some transmission parameters. After single control measures are translated into effects on these transmission parameters, the effects of sets of control measures (intervention strategies), can be calculated with the model. As research data are lacking, the model input parameters were derived from expert opinion. As an example, the effects of two intervention strategies proposed for the Dutch poultry industry are predicted. A sensitivity analysis is performed to indicate where the most effective control measures may be expected. Additionally, the reliability of the model predictions is studied by an uncertainty analysis. The use of the model as a tool for policy makers deciding about salmonella control strategies is discussed.

INTRODUCTION

Salmonella is recognized world-wide as a major cause of human diarrhoeal illness. For example, the annual incidence of salmonellosis in the general population in The Netherlands is estimated to be 700/100 000 (0·7%) [1]. Poultry meat is recognized as one of the major vehicles of infection with *Salmonella* spp. [2]. From a public health point of view, the importance of the serotype *S. enteritidis* as a contaminant in the poultry meat production chain does not exceed that of other *Salmonella* spp. However, *S. enteritidis*-infections have predominantly been associated with the consumption of eggs or egg-containing foods [3, 4]. Based on serotyping studies, it is estimated that poultry meat and eggs are implicated in approx. 20 and 40%, respectively, of the total number of salmonella infections in The Netherlands (W. Van Pelt, Dutch National Salmonella Centre, unpublished observations).

It is generally recognized that a reduction in human

salmonella infections should be achieved primarily by control of these organisms in food animals, especially poultry and pigs. In The Netherlands, the government has focussed on the control of salmonella in poultry as a first step and has set a goal to reduce salmonella in poultry meat (and eggs) to a minimal level within a few years [5]. To achieve this goal, several control measures have been proposed in one or more stages of the production chain, from grandparent stocks to the slaughterhouses. Combinations of control measures have been integrated in larger plans for the reduction of salmonella. One of those plans [6] is now implemented in the Dutch poultry meat production industry. As the transmission of salmonella through the production chain is complex, such integrative plans are the only way to achieve the desired reduction. However, the complexity of the process hampers the prediction of the ultimate effects of such plans.

In this paper we will show that modelling can be a helpful tool to evaluate the effect of intervention in a complex process like the transmission of salmonella

* Author for correspondence.

through the poultry meat production chain, and illustrate how modelling can be used to predict the effects of combinations of control measures. First, a model is formulated describing the transmission of salmonella over the consecutive stages of the poultry meat production chain. Next, the status of salmonella contamination in The Netherlands at the beginning of 1997 in the poultry meat production chain, as estimated by an expert panel, is implemented in the model. After estimating the effects of single control measures on the model parameters, the expected effect of intervention plans that consist of combinations of those measures can be calculated with the model.

In a subsequent sensitivity analysis we study the effect of small deviations of the estimated input to the final output, for two reasons. First, it is important to know which parameters are the most sensitive to change, as this indicates where better knowledge on the process of transmission is important. Second, if the model results are more sensitive to change of a certain parameter, this may indicate that intervention affecting this parameter is more effective. Hence, sensitivity analysis may be an aid in deciding where interventions should be aimed.

The inputs of the model, the estimated prevalences of salmonella and the estimated effects of control measures, are derived from expert opinion, because they are not available from research data. This implies that the input may be biased and unprecise, and consequently the output, the model predictions, may be inaccurate. Hence, to evaluate the reliability of the model predictions, we additionally perform an uncertainty analysis.

The main purpose of our paper is to illustrate how a rather simple model of the transmission of salmonella through the poultry meat production chain can be used as a tool in risk management, and help policy makers in choosing between different intervention strategies.

The specific evaluation of two intervention strategies for the Dutch situation is used as an example.

METHODS

Model description

The primary poultry meat production chain can be described as a chain of consecutive production stages t including grandparent stock (GPS) (elite flocks are not taken into account), breeder hatcheries (bH), rearing parent stock (rPS), parent stock (PS), com-
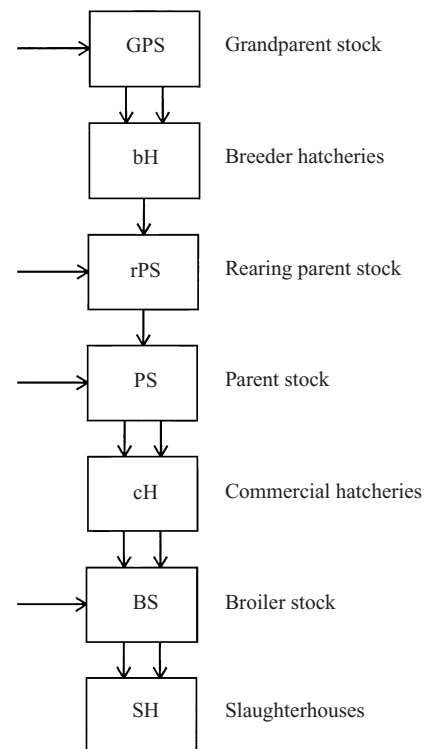
Fig. 1. The primary poultry meat production chain. The model describes how the salmonella prevalence at flock level may increase at each consecutive stage, due to transmission *dependent* on the salmonella contamination at the preceding stage (double vertical arrows) or transmission *independent* of this contamination (horizontal arrow).

mercial hatcheries (cH), broiler stock (BS) and slaughterhouses (SH) (see Fig. 1).

In the model, the prevalence of *Salmonella* spp. at flock level (equivalent to the probability that a flock is infected) at a certain production stage t is expressed by the parameter Q_t, with $0 \leqslant Q_t \leqslant 1$. (Below, for reasons of generality, a batch of eggs in a hatchery is referred to as a flock as well.) In general, potential routes of transmission existing in all stages of the production chain can be assigned to one of two categories: transmission dependent on salmonella contamination in the preceding production stage and transmission independent of this contamination. The prevalence of contaminated flocks and thus the probability of contamination at a certain production stage (Q_t) can be described as a function of the probability of dependent transmission at stage t (R_t dependent on Q_{t-1} and the probability of independent transmission at stage t (expressed as P_t) (Fig. 2). As a flock may become contaminated via both a *dependent* and an *independent* route of contamination, simple summing up of both probabilities, would result in an overestimation of Q_{t+1}. Therefore, the model uses the
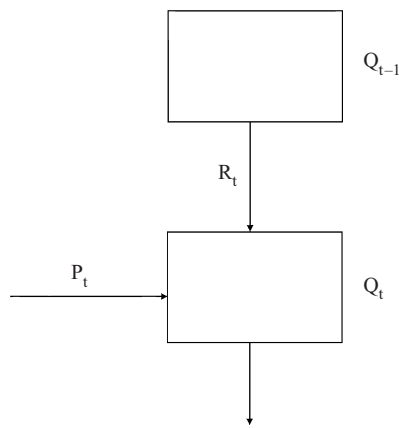
Fig. 2. Simplified representation of the two main contamination routes at all stages of the poultry meat production chain. The prevalence of contamination at flock level at a certain production stage, Q_t, depends on the probability of dependent transmission, R_t (dependent on salmonella contamination in the preceding production stage Q_{t-1}) and the probability of independent transmission from the environment, P_t (not dependent on Q_{t-1}).

probability that contamination of a flock at a certain production stage does *not* occur, which can be described by the following equation:

$$1 - Q_t = (1 - P_t)(1 - R_t). \tag{1}$$

The independent route of transmission consists of contamination from the environment such as contamination transmitted by vermin or by the feed. By definition, this contamination would occur even if the prevalence in the previous stage of the chain was zero. The main determinants of the probability of contamination of the flock by this route are hygienic conditions (either at the farm or during feed production), type of farming and length of stay of the flocks at a farm: the better the hygiene and the shorter the stay, the lower the value of the probability of contamination of the flock via this route, P_t.

The dependent route of transmission, roughly, consists of two components, i.e. (i) cross-contamination between flocks at the farm or during transport and (ii) increase of the contamination level due to the fact that each flock may be composed of progeny from multiple parent flocks. (In the hatcheries, for example, the eggs in one batch usually originate from different flocks. If any of these flocks is infected, the probability that the batch of eggs is infected is large.) In both cases, the probability of contamination is dependent on the prevalence of salmonella in the preceding production stage (if salmonella is absent in the preceding production stage, no dependent transmission occurs). The probability that flocks become

contaminated by dependent transmission at stage t (R_t) can be described by the equation:

$$R_t = (k_t + 1)Q_{t-1}, \tag{2}$$

where

$Q_{t-1} =$ the prevalence of *Salmonella* spp. in the preceding production stage,

$k_t =$ the increase of the contamination level due to dependent transmission ($k_t \geqslant 0$ and $(k_t + 1)Q_{t-1} \leqslant 1$). Thus, if $k_t = 0$, the level of contamination does not increase by dependent transmission of salmonella. As $k_t \geqslant 0$, it is assumed that the prevalence never decreases along the production chain without extra control measures (see below).

So, at each stage the following general formula can be derived to describe the prevalences over the chain:

$$Q_t = 1 - (1 - P_t)(1 - (k_t + 1)Q_{t-1}). \tag{3}$$

The next step is to evaluate the effects of control measures that intervene in the production chain. For this purpose, many control measures can be considered. Each of these measures will be expected to have an effect on the transmission process, either by lowering the probability of transmission by one of the two transmission routes (i.e. an effect on P or k), or by directly lowering the salmonella prevalence (an effect on Q). This can be implemented in the model by the parameters ef_{Pt}, ef_{kt} and ef_{Qt} which represent the relative effect of the control measure on the parameters P, k and Q at stage t respectively. (So for example, if by a control measure 75% of the positive flocks are eliminated at stage t, Q_t will decrease by 75%, so $ef_{Qt} = 0 \cdot 25$.)

After the effects of the control measures are specified, the predicted prevalence after intervention can be calculated at each stage t by:

$$Q'_t = (1 - (1 - P_t \times ef_{Pt})(1 - (k_t \times ef_{kt} + 1)Q'_{t-1})) \times ef_{Qt}, \tag{4}$$

where the accent in Q'_t indicates the value of Q_t after change due to intervention. Equation (3), is recovered by setting each effect parameter at $ef = 1$. With equation (4) it is possible to estimate the effect of any set of control measures for which the effect of each individual measure has been estimated. This will be illustrated below.

Model implementation

In general the model can be implemented by the following procedure:

(1) Description of the situation before intervention by (a) estimating the prevalences Q, (b) deriving the model parameters P and k, by combining knowledge of the relative importance of both transmission routes and model equation (3).

(2) Evaluation of the effects of intervention strategies by (a) estimating the effects of single control measures on the parameters P, k and Q by estimating ef_{Pt}, ef_{kt} and ef_{Qt} respectively, (b) calculation of the model with equation (4) for each set of control measures considered.

In this study the estimates mentioned above were obtained from a Dutch expert panel, including representatives from both the poultry industry and governmental authorities. First, at the start of 1997, this expert panel agreed upon some 'consensus' estimates for the poultry meat production chain in The Netherlands [5]. Later, in 1998, we discussed the model and asked the members of this panel to fill up a written questionnaire to give their individual point estimates of the prevalences and the effects of control measures for the situation in The Netherlands at the start of 1997. These individual estimates made it possible to perform an uncertainty analysis (see below). The means of these individual estimates, which may differ from the 'consensus' estimates of 1997, are used for the calculations in this study.

The effects of two different intervention strategies (sets of control measures) will be considered here. The first (plan I) is one of the strategies proposed by the expert panel mentioned above [5]. The second one (plan II) is based on the obligatory control measures imposed on the Dutch poultry industry by a special committee of the product boards for livestock, meat and eggs (PVE) in The Netherlands [6].

Sensitivity analysis

A sensitivity analysis was performed for the effect of a small change in each of the effect parameters ef_{Pt}, ef_{kt} and ef_{Qt} at each stage t, on the final prevalence of salmonella at flock level after slaughter, Q'_{SH}. This small deviation from ef was arbitrarily set at $\Delta ef = 10^{-6}$ and the effect on Q'_{SH}, expressed as a deviation $\Delta Q'_{SH}$, was calculated with equation (4). Then, the relative sensitivity was calculated as

$$\frac{\Delta Q'_{SH}}{\Delta ef} \times \frac{ef}{Q'_{SH}} \quad [7].$$

The larger the relative sensitivity, the larger effect a (small) change in the effect parameter will have on the prevalence at flock level after slaughter.

Uncertainty analysis

In an uncertainty analysis the uncertainty in the input parameter estimates is quantified to get an impression of the resulting uncertainty in the model output and thus of the reliability of the model predictions. Although several sophisticated mathematical modelling tools exist for this purpose, the available data do not allow a refined method in this study. We chose the following, rather simplistic, procedure to process the estimates of the individual experts: the members of the expert panel were asked to individually fill in a questionnaire and estimate both the prevalences Q_t at the different stages of the poultry production chain (the situation at the start of 1997), and the effects of a set of control measures. Subsequently, we recalculated the model for the effect of intervention strategy II for each expert individually. The uncertainty in the estimates of the prevalences was separated from that of the estimates of the effects of control measures, resulting in three separate analyses: (a) The individual estimates of both the prevalences and the effects; (b) the individual estimates of the prevalences and the mean of the estimates of the effects; (c) the mean of the estimates of the prevalences and the individual estimates of the effects. When recalculating the model, it was assumed that $P_{BS} = P_{rPS}$ (see below).

RESULTS

Description of the situation in The Netherlands at the start of 1997

The expert panel estimated the prevalences at flock level (Q_t) of salmonella in the different production stages of the poultry meat chain. The means of the estimates of nine experts are given in Table 1. These means are generally lower than the 'consensus' estimates of the prevalences that the members of the panel agreed upon earlier [5].

For the derivation of the values of the parameters P_t and k_t for each stage t, it was agreed that, in all production stages (apart from the broiler stage), either dependent or independent transmission could be neglected. For example, transmission of salmonella from the hatchery environment or slaughterhouses independent from salmonella contamination in the preceding production stage was considered negligible,

Table 1. *Survey of the model parameter values in the different production stages of the Dutch poultry meat chain. The salmonella prevalences at flock level* (Q_t, *as a fraction*) *are the means of the individual estimates of nine experts. The parameter values* P *and* k *are derived from the values of* Q, *using equation* (3). *In each stage, except the broiler stage* (*BS*), *one of the transmission routes was considered negligible*

Production stage	Salmonella prevalence at flock level (Q)	Probability of independent contamination (P)	Increase in dependent contamination level (k)
Grandparents (GPS)	0·037	0·037	
Breeder hatcheries (bH)	0·062		0·70
Rearing parents (rPS)	0·080	0·019	
Parents (PS)	0·148	0·074	
Commercial hatcheries (cH)	0·303		1·05
Broilers (BS)	0·364	0·019	0·16
Slaughterhouses (SH)	0·551		0·52

Table 2. *Description of the two intervention strategies for which the effect has been evaluated with the model. Each intervention strategy consists of a number of control measures at different stages in the production chain. The given effects of the control measures are the means of the effects as estimated by the individual members of the expert panel*

Intervention strategy	Control measure	Effect
Plan I*	Elimination of salmonella positive flocks at GPS	$ef_{Q,GPS} = 0·13$
	Logistic breeding in bH and cH	$ef_{k,bH} = 0·24$
		$ef_{k,cH} = 0·27$
	Logistic slaughter at SH	$ef_{k,SH} = 0·24$
Plan II†	Additional hygiene at GPS, rPS, PS and BS	$ef_{P,GPS} = 0·76,$
		$ef_{P,rPS} = 0·64,$
		$ef_{P,PS} = 0·54,$
		$ef_{P,BS} = 0·70$
	Logistic breeding in bH and cH	$ef_{k,bH} = 0·24$
		$ef_{k,cH} = 0·27$

* Based on one of the proposals of the expert panel [5].
† Based on the control measures prescribed for the poultry industry by the product boards of Livestock, Meat and Eggs in The Netherlands [6]

whereas the increase of the contamination level during the GPS and (r)PS stage was fully attributed to independent transmission from the environment. By setting either $k_t = 0$ or $P_t = 0$, it is easy to calculate the matching parameter from equation (3).

One exception to this general rule was made. In the broiler stock stage the increase in prevalence could not be attributed to one route of transmission only: neglecting k would lead to a $P_{BS} = 0·087$, implying a relatively bad hygiene in broiler farms. According to the expert panel this was not realistic as, on the contrary, the hygiene of the broiler farms is judged to be good in The Netherlands. As the panel considered the situation in the broiler stock comparable to the situation in the rearing parents stock, where the animals also stay for a relatively short period, P is set at the same value in both stages throughout this study. Then, to obtain, for example, the prevalence $Q_{BS} = 0·364$ when $P_{BS} = 0·019$, it can be derived from equation (3) that $k_{BS} = 0·16$, representing an estimate of cross-contamination during transport or at the farm.
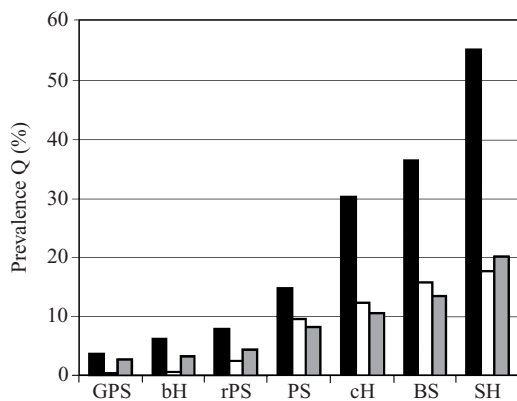
Fig. 3. The effect of the intervention strategies as predicted by the model. The bars represent the prevalence at flock level (Q) in The Netherlands per stage of the production chain. From left to right: Estimated situation 'start 1997'; model predictions plan I; model predictions plan II.

Evaluation of the effects of intervention

The effects of all the single control measures considered were estimated in terms of effects on one of the model parameters ef_P, ef_k or ef_Q. For example, logistic breeding (i.e. keeping eggs from contaminated flocks strictly separated from those from not-contaminated flocks) will lower the increase in prevalence by dependent transmission. The mean estimate of the expert panel of this effect is 76 % reduction of k in the breeder hatcheries and 73 % in the commercial hatcheries. So in this case $ef_{k,bH} = 0·24$ and $ef_{k,cH} = 0·27$. Likewise, logistic slaughtering (a procedure in which salmonella-negative flocks are slaughtered daily before salmonella-positive flocks) will also affect k, whereas hygienic measures will affect the independent transmission parameter P, and elimination of positive flocks will affect the prevalence Q directly.

Once the effects of single control measures are estimated, the model can be used to evaluate the effects of sets of control measures, defined as 'intervention strategies'. As an example, the effects of two intervention strategies are predicted with the model. These strategies and their effects, as estimated by the expert panel, are given in Table 2.

The model prediction is given in Figure 3. It shows that due to intervention the prevalence at flock level after slaughter (SH) reduces from 55 to 18 % by plan I and to 20 % by plan II.

Sensitivity analysis

A sensitivity analysis is performed on the effect parameters ef_{Pt}, ef_{kt}, and ef_{Qt} at all stages t of the production chain. This was done for all three situations shown in Figure 3: 'start 1997', plan I and plan II. The results are given in Table 3, and are generally explained by two observations. First, the sensitivity of the goal parameter Q_{SH} for ef_Q is often rather high and increases along the chain. This is a direct consequence of the structure of the model. From equation (4) it is easy to see that at each stage t, Q_t will be more sensitive to $ef_{Q,t}$ than to $ef_{P,t}$ or $ef_{k,t}$. The connection between stages is via Q_t, and the further down the chain, the closer the relation to Q_{SH}. Therefore the sensitivity is highest at the end of the chain. Second, for the other effect parameters, $ef_{P,t}$ and $ef_{k,t}$, the sensitivity is relatively high at those stages where the relative increase in prevalence Q_t is large.

For the situation 'start 1997', Table 3 shows that the sensitivity of Q_{SH} is highest for ef_Q (at all stages), ef_P (at GPS and PS) and ef_k (at cH and SH). This implies that control measures affecting these parameters are predicted to be the most effective in lowering Q_{SH}. Plan I reduces the values of some of these parameters (i.e. $ef_{Q,GPS}$, $ef_{k,cH}$ and $ef_{k,SH}$), and as a consequence the sensitivity of Q_{SH} for these parameters is decreased. Therefore, additional effort in control measures that affect these parameters (e.g. more effective logistic breeding) will be less effective than in the original situation. In contrast, the additional hygiene measures of plan II do not really lower the effect of the ef_P parameters. This implies that even more stringent hygienic measures may be effective.

To illustrate the value of the sensitivity analysis, we ran the model with mean estimates of the prevalences and effects, for an alternative plan with four control measures, selected on the basis of the sensitivities for the situation without intervention: (i) eliminating positive flocks at the GPS ($ef_{Q,GPS} = 0·13$), (ii) additional hygienic measures at PS ($ef_{P,PS} = 0·54$), (iii) logistic breeding at cH ($ef_{k,cH} = 0·27$) and (iv) logistic slaughtering at SH ($ef_{k,SH} = 0·24$). This resulted in a final prevalence of 13 %, lower than each of the intervention plans discussed above.

Uncertainty analysis

Eight (of nine) individual experts of the panel provided us with all the necessary estimates. Figures 4 and 5 show the results of the first analysis (a) discussed in the Methods section, where the model is run once for each expert's estimates. In Figure 4 the

Table 3. *The relative sensitivity of the prevalence after slaughter,* Q_{SH}, *(in %) for each effect parameter* ef *at the different stages of the production chain, for three different situations. The relative sensitivity depends on the values of the model parameters that describe the situation of concern. A control measure aimed at an effect parameter with a larger relative sensitivity will have a higher impact on the prevalence after slaughter*

Stages (see Fig.1)	1997*			Plan I†			Plan II‡		
	ef_Q	ef_P	ef_k	ef_Q	ef_P	ef_k	ef_Q	ef_P	ef_k
GPS	36	36	—§	5	5	—	34	34	—
bH	36	—	15	5	—	1	34	—	5
rPS	48	11	—	21	16	—	47	12	—
PS	95	43	—	88	66	—	90	42	—
cH	95	—	49	88	—	20	90	—	20
BS	100	3	13	100	10	12	100	9	12
SH	100	—	34	100	—	11	100	—	34

* The estimated situation at the start of 1997, with parameter values given in Table 1.
† Predicted situation after implementation of intervention strategy I ([5], see Table 2 and Fig. 3).
‡ Predicted situation after implementation of intervention strategy II ([6], see Table 2 and Fig. 3).
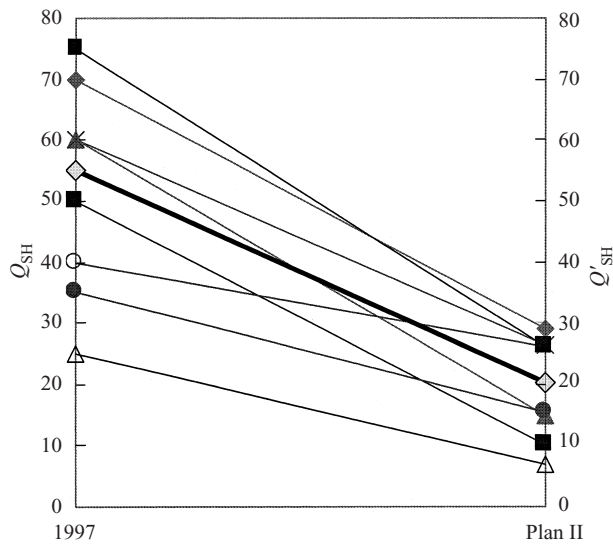§ If $P_t = 0$ or $k_t = 0$, the matching relative sensitivity is zero.

Fig. 4. Estimated prevalences after slaughter in 1997 (Q_{SH} (%), left) and the model prediction for these prevalences after implementation of intervention strategy II (Q'_{SH} (%), right). The thick line connects the mean prevalence estimate (left), and the model prediction with mean estimates for the model parameters (right). The thin lines connect the prevalence estimate and the model prediction per expert, for eight experts, and give an impression of the large uncertainty around the means. The relative effect of the intervention strategy, which is plotted in Figure 5, is $1 - Q'_{SH}/Q_{SH}$.
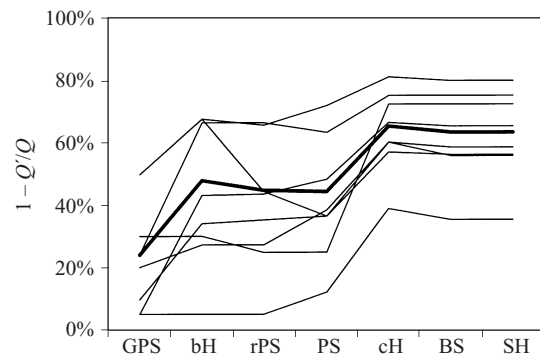

Fig. 5. The relative effect of intervention strategy II, according to model calculations for each individual expert (thin lines), and according to the mean estimates (thick line). The effect (in %) is defined as one minus the predicted prevalence (Q'_t) divided by the estimated prevalence for the start of 1997 (Q_t) at each stage t. 0 % relative effect means that $Q'_t = Q_t$, so intervention has no effect. 100 % relative effect means that the prevalence Q'_t has dropped to zero. Note that the uncertainty as expressed by the differences between the experts is similarly large all over the chain, and that, due to additional measures, the relative effect (roughly) increases further down the chain for all experts.

estimated prevalences after slaughter before intervention (Q_{SH}) are compared with the predicted prevalences after implementation of plan II (Q'_{SH}). In 1997 the estimated prevalences were 25–75 %; after implementation of plan II the predictions were 7–29 % (s.d. 8·5 %). Roughly, parallel lines in Figure 4 indicate that the predicted effects of the intervention plan are similar, whereas crossing lines indicate dissimilar effects. In Figure 5 the relative effects of intervention are expressed as $1 - Q'/Q$, and are shown for each expert along all stages of the chain. It shows that the uncertainty of the model predictions is rather large, and of the same order of magnitude all over the production chain. As a worst case scenario, the smallest predicted effect of plan II is only 35 %, when the prevalence after slaughter drops from 40 % in 1997 to 26 %. In two additional analyses only the

differences in estimated prevalences (*b*) and only the differences in estimated effects (*c*) are used (see Methods section). In analysis (*b*) Q'_{SH} was 6–29%, s.d. 7·2%, in analysis (*c*) Q'_{SH} was 11–30%, s.d. 5·7%. This implies that the uncertainty in the final model predictions is equally caused by both uncertainty in prevalence estimates of the situation at the start of 1997 and uncertainty in estimates of the effects of control measures.

DISCUSSION

A model has been developed to evaluate intervention strategies to control salmonella in the poultry meat production chain. As model input, the prevalences at flock level at all stages of the chain before intervention were used, as well as the effects of single control measures. The model gave predictions of prevalences at flock level along the chain, after intervention. As the model is rather non-specific in describing the transmission dynamics, and the model input is not based on actual research data, exact quantitative model predictions should be treated with caution. However, the model uses the same information on prevalences and effects of control measures as policy makers, choosing between alternative intervention strategies. As the model is easily implemented in a spreadsheet on a PC, it can be used to quickly evaluate the consequences of different opinions, and indicate which are crucially discordant. This may help to structure the debate on optimal intervention strategies, and point to the most important gaps in knowledge. Therefore, the model might be useful as an additional, relatively objective tool for policy makers.

In the specific case described in this paper, the prevalences at the different stages of the production chain in The Netherlands at the beginning of 1997 were estimated by an expert panel. The prevalence at flock level after slaughter was estimated at approx. 55%. Then, using the model, the prevalence after introduction of the obligatory intervention plan currently implemented in the Dutch poultry meat production chain (plan II, [6]) is predicted to be approx. 20%. Additional control measures may reduce this prevalence even further. At present, a monitoring programme in which these prevalences are measured has started as part of the implementation of the intervention plan. Therefore, more reliable data will become available in the future, which may give an impression of the accuracy of the model predictions. However, these data will not give additional in-

formation on the original situation in 1997, and will therefore not allow a full evaluation of the present study. Validation of the model will only be possible if more scientific data are available.

The sensitivity analysis indicates for which parameters a small change has a large effect on the prevalence at flock level after slaughter. This analysis can be used to identify the most important transmission processes in the chain and to choose the most effective control measures. Generally, the highest sensitivities are related to transmission processes that are responsible for the largest relative increase in prevalence. As might be expected, control measures that affect these specific processes are predicted to be the most effective. Also, the sensitivity analysis indicates high effectivity of control measures that directly lower the prevalence, eg by destruction of positive flocks. Regarding the sensitivity analysis for the situation in 1997, one might propose a set of control measures consisting of eliminating positive flocks at the grandparent stock, additional hygienic measures at the parent stock, logistic breeding in the commercial hatcheries and logistic slaughter in the slaughterhouses. As we have shown, this is predicted to be very effective.

An uncertainty analysis can be used to quantitatively assess the reliability of the predictions. Ideally, the experts should be asked to give a probability distribution of each estimate they provide, to express the uncertainty in each individual estimate [8]. These probability distributions could be linked in a large stochastic model, as currently applied in microbial risk assessment [9, 10]. However, in the model presented here this approach has not been used for three reasons. First, the individual experts have a different expertise and use different methods to come to their estimates, and for each individual expert the prevalence and effect estimates for the different stages are mutually dependent. As a consequence, probability distributions of the uncertainty around the different estimates cannot be interpreted comparatively, not even at single stages or for single experts. Second, as prevalences Q_t increase along the chain, the range of values that the prevalence at one stage can take depends on the prevalence at other stages. When prevalences are described by probability distributions, these distributions may overlap. In that case the distributions will have to be adapted interdependently. Third, a full uncertainty analysis, including the unravelling of all these complex dependencies and the construction of a large stochastic model which results in a probability distribution of

the uncertainty, would suggest a misleading accuracy. Neither the simplifying assumptions in the model itself, nor the generalization of the transmission routes, can be properly evaluated in such an uncertainty analysis, whereas these aspects are highly relevant for the reliability of the final model predictions.

As an alternative, we incorporated the estimates for each individual expert and compared the resulting model predictions in a simplified uncertainty analysis. Doing so, the model input of the different experts was not mixed up and the problems mentioned above are circumvented. It shows that the general tendency predicted by the model is the same among experts. Quantitatively, however, the model predictions for all individual experts show a large variability. As shown in Figures 4 and 5, with implementation of plan II, the predicted prevalence after slaughter may be 7–29%, so that the relative effect of intervention is 35–80%. This is the result of both differences in estimated prevalences along the chain, and differences in the estimated effects of control measures. The estimation of the latter is especially complex. In the model, these effects represent the effects of practical control measures on the salmonella prevalence or a theoretical transmission parameter, summarized for all farms and industries of the country as a whole. In estimation of these effects some aspects can easily be overlooked. Also, it may be impossible to fit a control measure into the model, for example when it is aimed at one or two serotypes only (such as vaccination against $S.$ enteritidis). To include such control measures, the model should be refined and extended. Nonetheless, when a control measure is proposed, this is done because it is expected to be effective in some way. Using the model, we are able to consider this effectiveness more specifically, which may be worthwhile in its own right.

For decisions about intervention strategies by policy makers, more aspects than those modelled here will have to be considered. The most important one among those is probably the economic consequence of an intervention strategy. For example, it is easy to see that withdrawal of all positive broiler flocks from the human consumption chain (intervention at $ef_{Q,BS}$) will be highly effective, but also extremely expensive. To deal with this problem, the model has been extended with an economic component [11]. It illustrates that the final decisions about optimal intervention strategies will always need profound deliberation, which may be facilitated by using models.

REFERENCES

1. De Wit MAS, Hoogenboom-Verdegaal AMM, Goosen ESM, Sprenger MJW, Borgdorff MJW. A community based survey in four regions of The Netherlands into the incidence and burden of gastro-enteritis and Campylobacter- and Salmonella-infections. Bilthoven: National Institute of Public Health and Environmental Protection, 1996. RIVM report nr. 149101014.
2. Bryan FL, Doyle MP. Health risks and consequences of Salmonella and Campylobacter jejuni in raw poultry. J Food Prot 1995; **58**: 326–44.
3. Anonymous. Salmonella enteritidis phage type 4: chicken and egg. Lancet 1988; ii: 720–2.
4. St. Louis ME, Morse DL, Potter ME, et al. The emergence of grade A eggs as a major source of Salmonella enteritidis infections. JAMA 1988; **259**: 2103–7.
5. Anonymous. Stappenplan salmonella pluimveehouderij, kuikenvlees en consumptie-eieren' Ede, The Netherlands: By order of the ministries of Public Health, Welfare and Sports (VWS) and Agriculture, Nature Management and Fisheries (LNV), 1997.
6. Anonymous. Plan van aanpak salmonella en campylobacter in de pluimveevleessector, version 97.08. Rijswijk, The Netherlands: Product boards of Poultry, Meat and Eggs (PVE), 1997.
7. Cascante M, Franco R, Canela EI. Sensitivity analysis. A common foundation of theories for the quantitative study of metabolic control. In: Voit EB, ed. Canonical non linear modeling. S-System approach to understanding complexity. New York: Van Nostrand Reinhold, 1991: 76–9.
8. Vose D. Quantitative risk analysis: a guide to Monte Carlo simulation modelling. Chichester: Wiley and Sons, 1996.
9. Bemrah N, Sanaa M, Cassin MH, Griffith MW, Cerf O. Quantitative risk assessment of human listeriosis from consumption of soft cheese made from raw milk. Prev Vet Med 1998; **37**: 129–45.
10. Cassin MH, Lammerding AM, Todd ECD, Ross W, McColl RS. Quantitative risk assessment for Escherichia coli O157:H7 in ground beef hamburgers. Int J Food Micro 1998; **41**: 21–44.
11. Van Horne PLM, Nauta MJ, Van Middelkeep JH. The cost of fighting against salmonella in poultry meat. World Poultry, special issue on salmonella, 1999: 48–9.