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Animal disease incidence and indemnity eradication programs[☆]

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Abstract

There are many options for controlling the spread of animal diseases. Some diseases have been treated as public sector problems and many nations have tried to control disease spread by purchasing sick animals from farmers. Government agencies have purchased breeding stock that might transmit diseases. Government agencies have purchased animals that might otherwise have gone to the slaughterhouse, thereby keeping pathogens out of the food supply. Our hypothesis is that when it is not immediately obvious to farmers or private sector buyers which animals carry or transmit diseases, a government indemnity program's success is not assured. Instead, disease control depends on farmers' ability to respond to the relative prices they face. We examine the incentives created by prices (indemnity payment levels) government agencies choose. The scrapie indemnity eradication program in the United States (1952–1992) provides a natural laboratory for measuring the responsiveness to government-set prices. We show that government-set prices played a major role in determining the program's outcome: the supply of infected animals was price elastic. We argue that short-run movements in relative prices and the number of infected animals offer a practical method for assessing program effectiveness. © 2000 Elsevier Science B.V. All rights reserved.

Keywords: Indemnity eradication programs; Scrapie

1. Introduction

The Federal government compensates individuals for a wide array of property losses in the United States. It often indemnifies property owners following natural disasters. It also influences environmental quality, and human and animal health through payments made to individuals for their property. The fifth amendment to the constitution requires the government to com-

pensate individuals when private property is taken for public use. USDA, as well as agriculture departments in many other countries, has mitigated the effects of some infectious animal diseases by making indemnity payments for sick animals. The payments made to farmers to control animal diseases differ from *many* indemnity programs because program existence creates nontrivial changes in incentives. These changes make programs differ from *all* other indemnity programs because animals reproduce, thereby eliminating upper bounds on program expense. In this paper we examine the behavior induced by one such program. We show that by identifying the incentives created by government-set prices, we can find conditions for evaluating program success.

[☆] The opinions expressed here are those of the authors and do not necessarily reflect views of the US Department of Agriculture.

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With a disaster-relief program, forecasting program impacts and expense is relatively straightforward. These program decisions are ad hoc. In the aftermath of a natural disaster, a specific area is delineated and designated as able to receive compensation. When residents have no or limited insurance coverage, they have no reason to reject Federal compensation. In effect, disaster relief is undertaken knowing how much that relief will cost.

Reducing or eradicating an animal disease is a different problem because animals are not fixed in quantity. Finding those that are infected depends on the technology for diagnosing disease, and the government must compete with the private sector for sick animals if diseases are difficult to detect. If it is not immediately obvious that an animal is infected, the animal may remain in production or be sent to slaughter. So, the indemnity payment will be a means for the government to induce farmers to allow destruction of some animals. What the indemnity does to the number of sick animals is not obvious.

The insurance industry has long recognized that indemnity payment levels and the conditions under which payments are made affect the behavior of insurance buyers and profits. Insurance companies have developed various types of risk-sharing insurance coverage, including coinsurance and deduction-based coverage, to reduce behavior that raises the frequency with which they make payments. However, outside of the insurance industry, the impacts of indemnity payment price levels on behavior have not been studied.

Conceptually, indemnity payments can influence both private and public sector behavior. For example, sometimes the indemnity comes from the budget of the government agency responsible for making payments. That is, indemnity payments compete with and may compromise other agency activities.¹ In this case, a regulatory agency that is both required to protect public health and is legally mandated to indemnify the industry it regulates may have less incentive to find health and safety violations than an agency that

imposes fines for violations. Alternatively, suppose a government agency indemnified contractors against especially large environmental risks inherent in production. Indemnification against unusually hazardous risks generates a more competitive supply for the agency, but eliminates the gains from risk sharing.²

The Animal and Plant Health Inspection Service (APHIS) of USDA has run indemnity programs to control and to eradicate brucellosis, tuberculosis, hog cholera, scrapie, and other animal diseases, of which the first two are communicable to humans. Here, we examine the history of the scrapie indemnity eradication program. This program is a natural laboratory for exploring behavioral responses to indemnity payments. Over the 41-year history of the program, indemnity payment levels and the extent to which flock depopulation was required upon finding scrapie varied. Also, APHIS recorded each confirmed scrapie case.³

We investigate the price-responsiveness of sheep farmers to indemnity payment levels. We assume that farmers responded to relative prices, regardless whether prices were established in the private or public sector. We test the hypothesis that as payments increased, farmers increased their efforts to find scrapie-infected sheep within their flocks. In effect, we estimate the supply of scrapie-infected sheep offered to the Federal government through its scrapie indemnity eradication program. Our price elasticity estimates reveal the indemnity price levels and program characteristics over which farmers could have easily expanded the number of sheep offered to the Federal government. Equivalently, the estimates show the degree of control the government had over the number of animals removed from production. We conclude by arguing that, for similar programs, program success could be judged without having to observe long-run relations among variables. Instead, short-run movements in relative prices and in the number of animals offered to a program indicate whether the program has reduced disease incidence.

¹ Until 1988, the US Environmental Protection Agency faced these choices. The agency was required to compensate pesticide owners for pesticides that could not be used under emergency suspension. Three pesticides were withdrawn from use under this requirement (McKenna and Cuneo and Technology Sciences Group Inc., 1991). Whether the legal mandate led to fewer emergency suspensions than would a different financing scheme is unknown.

² Some branches of the military assume responsibility for environmental cleanup costs and indemnify contractors against unusually hazardous risks (U.S. General Accounting Office, 1994).

³ Confirmed scrapie-positive sheep were defined by APHIS as those from which a diagnosis was made histologically or on the basis of mouse-inoculation testing at the National Veterinary Services Laboratory (Wineland et al., 1998, p. 714).

2. Scrapie and the eradication program

Scrapie is a degenerative disease affecting the central nervous system of sheep and goats. In the United States, scrapie primarily has been reported in the Suffolk breed (USDA, APHIS, VS, 1998a). Early signs include behavioral changes followed by rubbing against fixed objects, hence the name. Scrapie is classified as a transmissible spongiform encephalopathy (TSE). Many animal species are at risk from TSE diseases. The TSE family of diseases includes (among others) bovine spongiform encephalopathy (BSE, commonly known as mad cow disease), chronic wasting disease in deer and elk, transmissible mink encephalopathy, and classical and new-variant Creutzfeldt–Jakob disease in humans (USDA, APHIS, VS, 1998a, b, c, d). All the diseases display a prolonged incubation period of months or years and are progressive, debilitating, neurological illnesses. They are always fatal (Detwiler, 1992).

For purposes of control, the important scientific questions have not yet been answered. Detwiler notes that the cause of scrapie has been debated for many years. Initially, arguments were between a genetic cause and an infectious origin. The current debate allows for both factors, but the means of natural transmission are not understood. The agent responsible for TSE's is smaller than the smallest known virus and has not been completely characterized. Currently, there are three main theories on the nature of the agent: (1) it is an unusual virus, (2) the agent is a prion — an exclusively host-coded protein that is modified to a protease-resistant form after infection, and (3) the agent is a virino — a small, noncoding regulatory nucleic acid coated with host-derived protective protein (USDA, APHIS, VS, 1998a). The agent is extremely resistant to heat and to normal sterilization processes. Detwiler stated:

“The extent to which scrapie is transmitted by a contaminated environment, including pens, barns, feed, water, bedding and other fomites is unknown. The remarkable resistance of the agent to inactivation leads one to believe that it may survive in the environment for a number of years. (p. 504)”

Existence of the disease has raised several concerns. The first case of scrapie was diagnosed in the United States in 1947. Then, scrapie raised productivity and trade questions, as some countries placed restrictions

on imports of sheep, embryos, semen, and other ovine products from countries where the disease existed (Detwiler, 1992). Conditions exist that encourage such restrictions to continue: Australia and New Zealand are recognized by the United States as being free of scrapie (USDA, APHIS, VS, 1998a).

The disease raised concerns that it might be the cause of BSE in the UK (Brown, 1998). That is, changes in the rendering process that occurred around 1980 could have allowed the scrapie agent to survive and infect cattle. Brown noted these linkages have been disputed. But scrapie does not appear to directly compromise human health. USDA, APHIS, VS (1998a) stated:

“There is no scientific evidence to indicate that scrapie poses a risk to human health.”

Historical records indicate the disease was recognized in the early 1700s, so a lack of evidence for human health risks is compelling. But the Federal government has not ignored possible linkages. In 1997, the Food and Drug Administration issued a regulation prohibiting the use of most mammalian protein in the manufacture of animal feeds given to ruminants (USDA, APHIS, VS (1998b)).

However, if scientists can establish a causal linkage between scrapie and BSE, the issues raised by the existence of scrapie will be amplified. BSE raises human health issues as well as trade and government budget exposure questions. The United Kingdom Department of Health tallies the human health effects. From 1985 through November 1998, the Department found 35 new-variant Creutzfeldt–Jakob disease cases it classified as either definite or probable (United Kingdom Department of Health, 1999). Financial effects are easier to detect. The EU imposed a ban on British beef exports for 2 years following the UK government's admission of a possible link between BSE and the new-variant Creutzfeldt–Jakob disease. Various government programs led to the slaughter of over 4 million cattle in the 1996–1998 period (Reaney, 1998).

In 1952, the Secretary of Agriculture declared a state of emergency in an attempt to eradicate scrapie in the United States. Initially, the program focused on total flock depopulation. Once the disease was confirmed, the flock was quarantined and depopulated. All exposed sheep sold from the flock were traced and slaughtered. From 1957, source flocks were depopulated. There were periods in which depopulation was

not emphasized. The bloodline/surveillance program required maternal bloodlines of an infected sheep to be removed and all other animals were placed under 42-month surveillance (Detwiler, 1992). The indemnity payment also was adjusted. The indemnity rate started out as \$ 75 per head for registered sheep and \$ 25 per head for grade. For fiscal years 1976–1978 (October 1–September 30), the rate increased to \$ 90 for registered and \$ 40 for grade. Beginning in 1979, the rate shifted to two-thirds of the appraised value, with a maximum of \$ 300 for registered (Wineland et al., 1998).

An entirely different approach began in fiscal year 1993. A voluntary flock certification program was initiated. Essentially, this is a label indicating an absence of disease. In fiscal year 1993, farmers were also offered a one-time payment equivalent to the indemnity (Wineland et al., 1998 and Detwiler, 1992). That is, farmers were assured payments would end after 1993. Farmers appear to have responded to the last-chance offer. The number of confirmed cases peaked in 1993, more than double the previous year or succeeding years (see Fig. 1).

3. The economic problem and eradication programs

The structure of costs and benefits of publicly financed eradication programs are different from many

health and safety programs, but are similar to many investment problems. Programs like regulations requiring childproof caps on medicine bottles require on-going expenses for caps and offer the potential for on-going reductions in poisonings, so long as parents leave the caps on (Viscusi, 1998). Claiming that a problem can be eradicated is equivalent to saying that an up-front expense may result in never having to face the problem again. That is, immediate expenses can be compared with benefits that accrue over a very long period.

But putting a publicly-financed eradication program in place and receiving a stream of benefits are not identical. There is some possibility that programs will not be successful. Eradication programs may have design flaws because scientists rarely have a complete understanding of the biology of crop and livestock pests and pathogens. Scientists cannot forecast and account for all the ways pests and pathogens can adjust to control measures. Scientists' understanding of scrapie is far from complete.

In the more recent periods 1965–1975 and 1983–1993, the indemnity program depopulated along bloodlines rather than flocks. This emphasis suggests program designers had relatively greater certainty of a genetic, rather than environmental mode of disease transmission. Or, at the least, a genetic link was considered a necessary condition for transmission. The practical importance of such a conclusion is that only a small proportion of the sheep population would

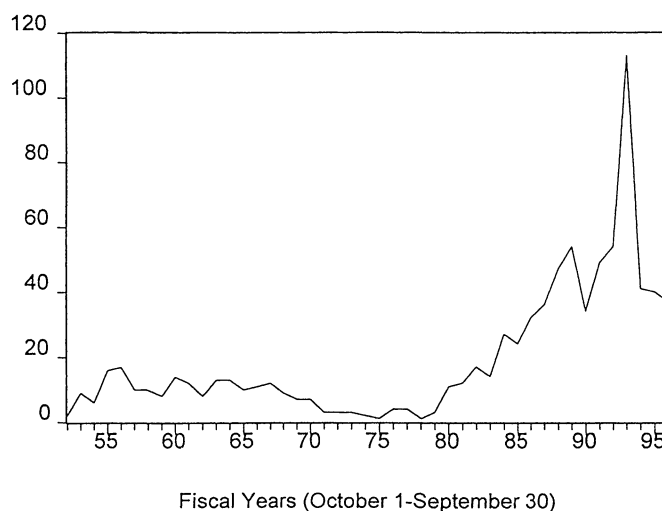


Fig. 1. Annual number of confirmed scrapie cases, 1952–1996.

therefore have the characteristic that makes animals susceptible to the disease. If sheep with that characteristic could be removed from the sheep population, the disease would disappear. Alternatively, if the disease were caused by an uncontrollable environmental condition, and all sheep were susceptible, there would be no reason a priori to suspect the disease could be eradicated. Instead, it would repeatedly arise spontaneously regardless of government actions.

The following simple model reveals the problem for eradicating an undesirable genetic characteristic. The model shows that the initial size of the problem (the proportion of the population susceptible to the disease and hence capable of passing on susceptibility) and the effectiveness at finding susceptible animals determines program success. Forecasting the outcome of the program is difficult because prices are likely to influence effectiveness of finding susceptible animals.

For simplicity, assume there is a long-lived breeding stock denoted Q . Q is assumed constant with new animals added only as replacements for animals discovered susceptible. Let S_t be the number of susceptible animals in the population, identified or not, during year t . Assume that S_t grows, depending on breeding practices, at annual rate g . Let F_t be the number found and replaced. As there is some positive probability that replacements are susceptible, assume that the proportion of susceptible animals within the set of replacements is identical to the proportion susceptible within the current population. Then, the number susceptible would evolve following Eq. (1).

$$S_t = (1 + g)S_{t-1} - \left(1 - \frac{S_{t-1}}{Q}\right) F_{t-1} \quad (1)$$

Assume that a fixed proportion of the susceptible animals are found and replaced each year.

$$F_t = fS_t \quad (2)$$

Then, S_t can be specified parametrically, as in Eq. (3).

$$S_t = (1 + g - f)S_{t-1} + \frac{fS_{t-1}^2}{Q} \quad (3)$$

Suppose the program is very effective at finding susceptible animals and there are few animals to find. Temporarily assume away any growth in the susceptible population. Let $f=0.9$ and the initial proportion susceptible be 0.01. In this case, it would take just over

2 years to drive the number of susceptibles to 1% of the initial level and just over 3 years to reach one-tenth of 1% of the initial level. In effect, the problem would be eradicated quickly as the susceptible animals become extinct. On the other hand, if $f=0.01$ and the initial proportion susceptible were 0.9, it would take 21 years to realize a 2% reduction in the proportion susceptible. Of course, if $g>f$ the number would grow to Q .

When a government agency offers a bounty for a sick animal, it makes f a function of the indemnity payment, $f=f(p)$. An increasing bounty means the rewards for searching for susceptible animals among the existing stock rises. Assuming that the marginal productivity of farmers' search efforts are positive, $(df/dp)>0$, more susceptible animals should be discovered. In effect, f rises along with the rising indemnity payment. In this case, the impact of an increase in the indemnity payment is to lower the number of susceptible animals.

$$\left. \frac{\partial S_t}{\partial p} \right|_{g=0} = \frac{df}{dp} \left[\frac{S_{t-1}^2}{Q} - S_{t-1} \right] \leq 0 \quad (4)$$

As $(S_{t-1}/Q) \leq 1$, the term in brackets is nonpositive, making Eq. (4) nonpositive.

There are two ways to produce susceptible animals. Farmers can search within their flocks, finding some proportion of the temporarily fixed quantity of susceptible animals. Alternatively, for diseases where transmission mechanisms are known, they can manufacture new susceptible animals. If it is physically possible to breed for infections or susceptibility, there is a price that will make the returns from such breeding positive. This possibility creates a unique moral hazard problem. In effect, the impact of an increase in the indemnity payment depends on the relative productivity of the technology for transmitting disease among animals and the technology for searching for susceptible animals. Eq. (5) allows prices to affect both means of producing susceptible animals. The sign of Eq. (5) is ambiguous because $(dg/dp)>0$ but the remaining term is nonpositive.

$$\frac{\partial S_t}{\partial p} = \frac{dg}{dp} + \frac{df}{dp} \left[\frac{S_{t-1}^2}{Q} - S_{t-1} \right] \quad (5)$$

In the scrapie case, we assume that since scientists are still unsure how the disease is transmitted, farm-

ers are unlikely to have discovered a practical means for breeding for susceptibility and kept the discovery secret from the scientific community. Thus, indemnity price increases should have reduced (or left unchanged) the susceptible subpopulation.

Physical characteristics of the problem and the impact of prices on the effectiveness with which infected animals are identified determine whether an eradication program will quickly succeed or never make a detectable impact on the problem. Technology was not advanced enough for the scrapie eradication program to search for susceptible animals. Instead, the program offered a bounty for confirmed cases, a subset of the susceptible animals.

4. Estimating the price incentive

Several analysts have speculated that prices influenced scrapie reporting. Wineland et al. (1998) included a graphic overlaying confirmed cases on the nominal indemnity payment, suggesting a connection between indemnity payment levels and the number of reports. USDA, APHIS, VS, 1991 was more explicit:

“The reporting of scrapie has been notably influenced by the real (inflation-adjusted) value of the indemnity payment. (p. 3)”

However, the agency report did not suggest the hypothesis has been tested empirically.

Similar speculation accompanied both the British and Portuguese indemnity programs to control BSE. Several public health officials argued that prices determine whether programs are successful in removing diseased animals from the food supply. Summarizing remarks of Dr. Richard Lacey, Reuters reported

“... farmers are under enormous pressure not to report cases of BSE because the UK government has cut compensation levels for sick animals (Reaney, 1998).”

The Wall Street Journal summarized remarks of Ramiro Doutel Mascarenhas, vice director of the veterinary section of the Ministry of Agriculture in Lisbon:

“... farmers who report diseased animals are paid more than they are worth, so there is no financial incentive to send a sick animal to the slaughterhouse (Stecklow, 1998).”

Fig. 1 shows the time pattern of reported cases since from the beginning of the eradication program in 1952 through its end in 1992, along with succeeding years covered by the voluntary certification program. Clearly, neither program eradicated the disease. But even if the eradication program had been partially successful, infected sheep would be increasingly difficult to find. In that case, the incentive to find additional animals created by rising indemnity payment levels would decline. That is, the supply of infected sheep would become increasingly price inelastic. Here, we show that the supply of scrapie-infected sheep was price elastic and that the level at which the indemnity payment was set offered a strong behavioral incentive.

We let the quantity of confirmed scrapie cases, the supply of infected sheep offered to the Federal government, depend on the relative prices farmers anticipate (P_t^e). A desired supply is thus a function of expected prices (Eq. (6)).

$$Q_t^* = \beta_0 + \beta_1 P_t^e + \varepsilon_t \quad (6)$$

Neither variable is directly observable.

Like all other agricultural commodities, it takes time to produce sheep, whether healthy or ill. Thus, complete responses to relative prices may not occur immediately. Instead, the supply of scrapie-infected sheep may adjust partially each period (Eq. (7)).

$$\Delta Q_t = \lambda(Q_t^* - Q_{t-1}) + \eta_t \quad (7)$$

The error terms ε_t and η_t are assumed to be normal, independently and identically distributed random variables with mean zero and constant variance. Substituting Eq. (7) into Eq. (6) eliminates Q_t^* , one of the unobservable variables (Eq. (8)).

$$Q_t = \lambda\beta_0 + \lambda\beta_1 P_t^e + (1 - \lambda)Q_{t-1} + v_t \quad \text{where} \quad v_t = \lambda\varepsilon_t + \eta_t \quad (8)$$

A ewe is a capital asset and farmers continually face the choice of whether to continue using the ewe to produce slaughter lambs or to sell the asset at its market value. The scrapie indemnity program offered farmers an additional option for their infected sheep. They could sell to the Federal government. Here, we focus on the choice between accepting the asset's salvage value and selling to the Federal government and the future productivity of maintaining an animal in the breeding stock.

Assuming the ewe market is efficient, we know the price should be equivalent to the present value of the expected profits from slaughter lambs. That is, if we had a consistent series of market prices for breeding stock, we should find little difference from the present value of expected profits from slaughter lambs. Empirically, the two series would move together if we could measure them both. However, no consistent data on ewe prices exist as these are quite heterogeneous assets, with values likely varying among breeds. We use the current price of slaughter lambs as a proxy for the value of a ewe.

Unlike the slaughter lamb price, the indemnity payment was set by fiat and thus need not bear any relation to market prices. We model the major measurable choices that faced sheep farmers with potentially scrapie-infected sheep. Our working hypothesis is that it was not immediately obvious that all infected sheep were in fact infected. That is, some infected sheep may have been sold for slaughter. The major alternative use of a scrapie-infected sheep was to acknowledge the illness and receive the indemnity payment. Thus, we model expected relative prices: the maximum indemnity payment relative to the price of slaughter lambs. We define the relative prices farmers faced as:

$$P_t = \frac{\text{Maximum indemnity payment in period } t}{\text{Market price of slaughter lambs in period } t} \quad (9)$$

When indemnity payments increased relative to the slaughter lamb price (representing the value of a ewe in production) rise, farmers may have looked harder for infected animals. Similarly, when indemnity payments were reduced relative to slaughter lamb prices, the incentive to find infected animals was reduced. In that case, fewer confirmations of scrapie would be anticipated.

Because indemnity payments changed following Federal government fiscal years, we constructed all variables on a fiscal year basis. Variables were transformed to natural logarithms so parameter estimates could be interpreted as elasticities. The dependent variable is a (log transformed) count of the number of confirmed scrapie cases in the United States each fiscal year. APHIS maintained records of each confirmed scrapie case. From confirmation dates, we constructed fiscal year totals.

We calculated an annual slaughter lamb price also on a fiscal year basis and constructed the relative price series. Slaughter lamb prices received by farmers are from the National Agricultural Statistics Service annual price survey reports (US Department of Agriculture, National Agricultural Statistics Service, Agricultural Statistics Board).

We cannot observe expected prices, only realized prices. We assumed that farmers extrapolated from past prices when forecasting prices and could observe cyclical movements in prices. Thus, we let (log transformed) relative prices follow an extrapolative model.

Identifying the ARIMA model that the relative price series followed strongly suggested a random walk. Autocorrelations of the relative price series declined slowly and the partial autocorrelations spiked at lag 1 and nowhere else. After differencing, no significant autocorrelations or partial autocorrelation were significantly different from zero. Dickey–Fuller tests confirmed the need to difference once. None of the three Dickey–Fuller test statistics approached critical values for the undifferenced data. After differencing, all three exceeded 1% critical values, strongly suggesting stationarity of the differenced series. Augmented Dickey–Fuller tests showed exactly the same results as lagged dependent variables were uniformly insignificant. Thus, our model for expected prices is Eq. (10).

$$P_t^e = P_{t-1} \quad (10)$$

During two periods (1957–1964 and 1976–1982), participating in the program had some negative consequences. If a farmer sold one or more confirmed scrapie-infected sheep to the government, the government would depopulate the flocks containing those sheep. Also, if the sheep had been purchased, the flock from which the infected sheep had come were depopulated. We assume these consequences would have had a chilling influence on future transactions among farmers and would have made a farmer less likely to participate in the program. In other years, flock depopulation was not emphasized (bloodline/surveillance program). For convenience, we refer to the years in which the bloodline/surveillance program was in effect as the unrestricted period. Consistent with our price variable, we defined the dummy variable equal to 1 for the years in which these negative consequences were not enforced in the previous year (unrestricted period) and 0 for all other years (restricted period).

Table 1
Supply function parameter estimates

	Estimates	<i>t</i> -statistics
λ	0.5170	4.9305
β_0	-1.7429	-2.5963
γ_0	2.9915	5.7637
γ_1	0.4004	1.8791

Table 2
Estimated price elasticities of supply

	Short-run	Long-run
Less restrictive period	1.7536	2.9915
More restrictive period	1.5466	3.3919

The estimated model contained a constant, the expected relative prices, a dummy variable shifting the price slope ($\beta_1 = \gamma_0 + \gamma_1 D_t$) and a partial adjustment parameter.

$$\ln Q_t = \lambda \beta_0 + \lambda(\gamma_0 + \gamma_1 D_t) \ln P_{t-1} + (1 - \lambda) \ln Q_{t-1} + e_t \quad (11)$$

Eq. (11) was estimated with non-linear least squares, yielding parameter estimates and asymptotic *t*-statistics (Table 1). Estimating in double-log form yields the short-run (one-period) price elasticities. During the unrestricted period price elasticity equals $\lambda(\gamma_0 + \gamma_1)$ and price elasticity equals $\lambda\gamma_0$ when the indemnity payment carried severe penalties. The long-run price elasticity during the unrestricted period is $\lim_{\lambda \rightarrow 1} \lambda(\gamma_0 + \gamma_1) = \gamma_0 + \gamma_1$ and at the restricted period $\lim_{\lambda \rightarrow 1} \lambda\gamma_0 = \gamma_0$. Estimates of the four price elasticities are reported in Table 2.

The diagnostic statistics (Table 3) show the model performs well. The Jarque–Bera test indicates no reason to reject the null hypothesis that the residuals are independent and normally distributed. We tested for

model misspecification following Ramsey's RESET procedure. To run the test, we followed recommendations of Ramanathan (1995, p. 290), adding fitted values of the dependent variable up to fourth power to Eq. (11). We also tested a lower order for powers of fitted values. Neither test indicates any evidence of misspecification. Serial correlation in the error terms would be problematic as the model incorporates a lagged dependent variable. The Lorange multiplier (LM) tests for serial correlation suggest the residuals are white noise. Thus, there is no reason to suspect bias in the parameter estimates.

The adjustment coefficient ($\lambda = 0.5170$) indicates that just over half of the gap between realized and desired supply closed in 1 year. The speed of adjustment ($\lambda^{-1} = 1.9342$) indicates full adjustment occurred in just under 2 years.

The short-run price elasticity $\lambda\gamma_0$ was 1.75 during the less restrictive periods, indicating that a 1% rise in the indemnity payment, relative to the market price of slaughter lambs, yielded a 1.75% increase in the number of confirmed scrapie cases (Table 2). Similarly, a 1% increase in the price of slaughter lambs relative to the indemnity payment, yielded a 1.75% reduction in confirmed cases. In the more restrictive period, the price response was slightly less, but still elastic. The long-run impact of a one-period price shock was approximately twice the short-run impact. A 1% price shock was associated with a 3.39% change in the number of confirmed cases, over a nearly 2-year period in the less restrictive years.

By itself, Fig. 1 might suggest that over the long run, the number of cases was rising. However, the model explains 81% of the variation in the (log-transformed) number of cases and, as Table 3 suggests, the residuals appear to be white noise. That is, the increase appears to be largely due to changing relative prices and program characteristics.

Table 3
Supply function diagnostic statistics

Tests			Breusch–Godfrey serial correlation LM test			Jarque–Bera residual test	
Ramsey's RESET							
Additional variables (in logs)	<i>F</i>	<i>p</i> value	<i>H</i> ₀	χ^2	<i>p</i> value	χ^2	<i>p</i> value
\hat{Q}_t^2 and \hat{Q}_t^3	0.4934	0.6149	$\rho_1 = 0$	0.0200	0.8875	1.7950	0.4076
\hat{Q}_t^2 , \hat{Q}_t^3 , and \hat{Q}_t^4	0.3243	0.8078	$\rho_1 = \rho_2 = 0$	0.4475	0.7995		

These results are not surprising when viewed in terms of relative prices. The indemnity payment relative to the price of slaughter lamb followed a downward trend over the 1962–1977 period. The number of confirmed cases also trends downward over that period. Following the 333% increase in the indemnity payment in 1978, the number of confirmed cases began trending upward for more than a decade.

5. Conclusions

We examined the history of the program intended to eradicate scrapie in sheep. Our econometric model of the supply of confirmed scrapie cases indicated price elastic responses throughout the program's history. That is, expansion and contraction of the number of confirmed cases was more than proportionately responsive to relative prices, and adjustments in the number offered to the Federal government were relatively easy to make.

Finding any response to changing relative prices suggests that more infected animals remained in production than would have under higher indemnity payments. Within the observed relative price range, the price elastic response suggests that higher indemnity payments early in the program's history would have yielded substantially more confirmed cases. Whether, say, a higher payment during the 1950s might have yielded a declining long-term pattern of confirmed cases, is unknown however.

A program that is reducing disease incidence should show supply of confirmed cases becoming price inelastic. As the most easily identified cases are removed first and the population of susceptible animals shrinks, the marginal cost of identifying new cases should rise. Thus, the price elasticity of supply should approach zero, and will be zero when the disease is eradicated.

There are at least two explanations for finding the generally price elastic response. First, it is possible that the indemnity payment was too low to significantly reduce the susceptible subpopulation. A payment outside the range of our data might have elicited a less elastic response. Second, the disease itself may have characteristics that enable it to resist eradication. It is possible that the disease is easily spread among unrelated animals and that there is an environmental reservoir that repeatedly infects animals. Under these

conditions, no indemnity payment could eradicate the disease.

Taking a long-run perspective on a program can yield ex post observations about program effectiveness. Such observations could be useful when agencies are contemplating new programs. However, to evaluate on-going programs, a more rapid evaluation method is necessary.

There are signals that indicate, in the short run, whether an eradication program is responding in a desirable way to indemnity payments. A program that is working will show the number of reports of illness falling, approaching zero. But the fall has to be conditional on the existence of incentives to report illnesses. That is, the fall has to be the result of increasing difficulty in finding infected animals rather than in a lack of incentive to look. If relative prices (indemnity payments relative to market price) are rising or constant through time and the number of reports falls, one could conclude that infected animals are becoming more difficult to find.

It is possible to construct a short-term decision rule because livestock markets adjust quickly to changing relative prices. That is, the time period over which prices and reports need to be observed is relatively short. The long-run price response in the scrapie case is just under 2 years. In the very short run, rising relative prices could induce additional reports. The short-run response is to bring forth additional effort to find and report infected animals. But, for diseases with characteristics amenable to eradication, having found infected animals and therefore reduced the susceptible portion of the population, infected animals will forever be more difficult to find. Thus, observing the pattern of relative prices and reported infections over a few marketing years might yield information sufficient to judge a program. A combination of falling reports and rising relative prices might not prove the program will be successful, but continually rising reports in the face of rising prices suggests eradication will not occur.

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