Elasticity and substitutability of food demand and emerging disease risk on livestock farms

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Abstract

Disease emergence in livestock is a product of environment, epidemiology, and economic forces. The environmental and epidemiological factors contributing to novel pathogen emergence in humans have been studied extensively, but the two-way relationship between farm microeconomics and outbreak risk has received comparably little attention. We introduce a game-theoretic model where farmers produce and sell two goods one of which (e.g. pigs, poultry) is susceptible to infection by a pathogen potentially dangerous to humans. We model market effects and epidemiological effects at both the individual farm level and the community level. The addition of a second good into this modeling framework ensures that producing a unit of livestock has an opportunity cost. We find that in the case of low demand elasticity for livestock meat, the presence of an animal pathogen causing large production losses can lead to a bistable system where two outcomes are possible, depending on the economic inputs into the system. One outcome is successful disease control. The second outcome, a potentially dangerous one, is a stable equilibrium where farmers slaughter their animals at a low rate, face substantial production losses, but maintain large herds because of the appeal of high meat market prices, therefore maintaining disease circulation. We show the potential epidemiological benefits to (i) policies aimed at stabilizing livestock product prices, (ii) subsidies for alternative agricultural activities during epidemics, and (iii) diversifying agricultural production and sources of proteins available to consumers.

Keywords: health economics, veterinary epidemiology, livestock epidemics, livestock production, food safety, game theory

1 Introduction

Livestock farming systems have played a major historical role in the evolution and establishment of major human pathogens (Wolfe et al., 2007). Today livestock farming is still a predominant driver of pathogen emergence in humans (Rohr et al., 2019). In addition, certain livestock pathogens that are not transmissible to humans, such as Peste des Petits Ruminants (PPR) and Newcastle Disease, are a continuous threat to livelihoods in lower and middle income (LMIC) countries in Africa and Asia, and they undermine the efficiency of our use of environmental resources to cover the protein needs of an increasingly larger human population (de Haan et al., 2015). Other infectious diseases such as African swine fever (ASF), which were previously circumscribed to restricted areas, are now propagating to a large number of countries causing major supply shocks and threatening the livelihood of low income populations (Mason-D’Croz et al., 2020). Difficulties in identifying and implementing effective disease control programs have raised questions of the added value of policy optimization based on an economic rationale, indicating that the incorporation of strategic responses of actors of animal production into epidemiological models may be necessary.

While the impact of strategic behavior of economic agents in the face of epidemics has attracted significant attention over the past two decades (Verelst et al., 2016), few studies have addressed the specific issue of behavioral responses of livestock farmers to the spread of diseases in livestock populations and their market effects. The incentivization of *ex ante* disease prevention through enhanced farm biosecurity has been the focus of most theoretical behavioral studies so far, with recommendations mostly tailored to the livestock sector of industrialized countries (Hennessy, 2007; Beach et al., 2007; Kobayashi and Melkonyan, 2011).

Zoonotic risk due to poor hygiene occurs primarily in LMICs where livestock producers lack access to financial capital and technology, which limits their capacity to invest in infrastructure or veterinary interventions for disease prevention (Hennessy, 2007; Ebata et al., 2020). A majority of smallholder farmers in LMICs rely on a low input production system based on free grazing or use of swill for animal feeding, which is incompatible with most biosecurity requirements (Penrith et al., 2021). Free-grazing animals from different farms frequently intermix and are exposed to contaminated material present in the environment. Vaccination constitutes a key preventive response, but, depending on the disease and the socio-economic context, vaccines against the considered pathogen may simply not exist yet - this is the case of ASF (Dixon et al., 2019) - or their delivery to farmers may be impaired by constraints in production, transport

and storage (Tago et al., 2017). Surveys in LMICs show a strong positive correlation between herd size and vaccine uptake (Marsh et al., 2016; Delabougliise et al., 2020), suggesting that vaccination is more likely to be adopted in large-scale farming systems; however, the vast majority of livestock producers are still smallholders.

The appeal of smallholder farmer partly comes from its diversification of income sources, a major risk-coping strategy of rural households (Dercon, 2002). Keeping livestock on a limited scale or with low fixed investments in building or equipment allows farmers to easily adapt their production size or temporarily empty their farm to invest in other economic activities, in response to epidemics or market shocks. As a consequence, farmers may react to epidemics affecting either market prices or their productivity by modulating their flock/herd sizes or switching focus to a different species (rather than investing in prevention). The common nature of species changes in farmed livestock at the household level, in low and middle income countries, is supported by empirical observations. In Nigeria, a general household survey was conducted by the National Bureau of Statistics (5000 households, 2010-2016) at three different time points (Nigeria National Bureau of Statistics, 2011, 2013, 2016). For a given livestock species category present in the household, the average probability that the species/category would be absent in the next survey was 32% for large ruminants, 29% for small ruminants, 66% for pigs, and 39% for poultry. The average probability that a present species was absent in the previous survey was 30% for large ruminants, 30% for small ruminants, 67% for pigs, and 39% for poultry. This suggests that removals and introductions of new species are common. A recent prospective study conducted on a monthly time-scale — 1087 poultry flocks followed on 53 farms in southern Vietnam (Delabougliise et al., 2019) — showed that each month an average farm had a 30% probability of introducing a poultry species absent in the previous introduction, and 44% of poultry introductions included at least one new species absent in the previous poultry introduction (**Figure 1**). Additionally, depopulation of chickens was influenced by the presence of outbreaks with poultry sales occurring earlier (i.e. in younger age animals) when outbreaks were occurring and later (i.e. in older age animals) two months after outbreaks (Delabougliise et al., 2020).

Figure 1: Bar plot illustration of the species shifts that occurred in the 53 monitored poultry farms in the Mekong delta region over the 20-month survey period in 2015-2017. The height of each bar is proportional to the number of birds introduced at each month of the study period, and it is scaled to a maximum number of introduced birds in each farm. The introductions of poultry species present in the last introduced batch are displayed in gray, while the introductions of poultry species absent in the last introduced batch are displayed in orange. On average new poultry species were introduced in 30% of farm-months.

70 Socio-economic surveys conducted in northern Vietnam have revealed that commer-
71 cial pig farmers react to epidemics of Classical Swine Fever or Porcine Reproductive
72 and respiratory Syndrome by expanding their stock of breeding sows, rather than
73 slaughtering them, in order to produce a larger pool of fattened pigs in anticipation of
74 production shortage and high prices (Pham et al., 2017). Broiler chicken farmers em-

75 ploy both strategies, most of them reducing their purchase of day-old chicks upon being
76 informed of an epidemic, while some of them do the exact opposite, as they aim at
77 being ready to sell adult chickens during price peaks following epidemics (Delabougli
78 et al., 2016). These empirical observations underline the need of new modelling ap-
79 proaches incorporating farmers’ responses to variations in both disease risk and prices.

80
81 Epidemics of livestock diseases affect markets through supply and demand shocks
82 (Rich et al., 2005). Supply shocks originate from livestock product shortage due to
83 a drop in production caused by (i) the disease itself, (ii) policies implemented to con-
84 trol the epidemics, or (iii) the downsizing of farm production by farmers out of fear.
85 Demand shocks are due to information obtained by consumers on disease occurrence
86 in the livestock population leading them to decrease their demand for products of the
87 affected species. Depending on the relative intensity of these two shocks, market prices
88 may evolve in opposite ways. Empirical studies of consumers’ response to food safety
89 risks like Salmonella contamination in chickens or Bovine Spongiform Encephalopathy
90 (BSE) in cattle showed that consumers’ demand for animal products quickly adapts
91 to information received on health hazards, although the magnitude of changes in the
92 concerned products’ demand varies according to the considered pathogen and socio-
93 cultural context (Dahlgran and Fairchild, 2002; Taylor et al., 2016; Yeung and Morris,
94 2001; Yang and Goddard, 2011). The scare caused by H5N1 avian influenza (AI) in-
95 troduction in Europe in 2005-2006 temporarily turned southern European consumers
96 away from poultry meat (Mazzocchi et al., 2007). A study using time series of house-
97 holds’ purchase data showed that Italian consumers reduced their demand for poultry
98 by about 20% in response to AI-related information (Beach et al., 2008). During the
99 first H5N1 epizootic in 2004 Vietnamese consumers turned away from poultry, resulting
100 in a compensatory increase in demand for pig meat and resulting losses and benefits
101 for the poultry and pig sectors respectively (ACI, 2006; Burgos et al., 2008). 74% of
102 respondents of a survey conducted in Hanoi reported that they stopped eating poultry
103 (Figue and Fournier, 2008). However, consumers also seem to have quickly recovered
104 a large part of their previous poultry consumption level after the reported incidence
105 of the disease dropped, and the disease-induced supply shortage resulted in rises of
106 poultry market prices and high profits for the poultry farms which were still operat-
107 ing at this time. Similar price responses to H5N1 epizootics were observed in other
108 countries like Egypt (Hassounah et al., 2012) and Turkey (Saghaian et al., 2008). The
109 recent spread of ASF in China, started in mid-2018, has led to a fall in pig production
110 of nearly 50% in this country, resulting in a more than twofold increase in the price
111 of pork meat and a more moderate rise in the price of substitute meats (including
112 chicken, beef and mutton), despite rising concerns of Chinese consumers for the safety

113 of animal products (Ma et al., 2020).

114

115 The present study aims at providing a theoretical framework for the modelling of
116 the joint dynamics of livestock pathogens and livestock markets when farmers change
117 their herd size or species in response to changes in market prices, perceived risk of mor-
118 tality, or mandated depopulation. We determine how elasticity of demand for livestock
119 meat, the substitutability between food products, and consumer preference for healthy
120 animals can influence the outcome of diseases control policies and suggest potential
121 directions for improvement of these policies.

122

123 2 Model

124 2.1 Description of the system

125 We model a system composed of a population of households practicing the breeding
126 and fattening of animals of a given species for the purpose of meat production. The
127 breeding-fattening model applies to most ruminant and pig farms and to smallholder
128 chicken farms where farmers aim at producing mature animals eligible for slaughter
129 and consumption while keeping some adults as breeders to ensure self-renewal of their
130 herd or flock. Each farmer keeps one herd and aims at optimizing herd size and
131 sell rate. Similar to the model proposed by (Nerlove and Bessler, 2001) we consider
132 that herds are entirely self-renewed, i.e. departing animals are replaced through the
133 breeding of adult animals present in the herd. Farmers fatten and sell their surplus
134 animals i.e. the difference between the number of newborn animals and the number
135 of replacement animals needed to renew the breeding herd. The meat obtained from
136 slaughtered animals is sold to consumers. Households have multiple activities and
137 are simultaneously engaged in the supply of an alternative good. The two goods are
138 assumed to be perfect substitute-in-production, i.e. their production consumes the
139 same resources. In order to produce more of one good the farmer needs to reduce the
140 production of the other one, and the rate of substitution between the two goods is a
141 given constant. In other words, animal production is assigned an opportunity cost in
142 our model. Animals of the considered species are susceptible to an infectious disease,
143 which is transmitted through direct contact between infected and susceptible animals.

144 2.2 Farm economic model

145 The farm dynamics and the utility maximization problem faced by households are
146 illustrated in **Figure 2**. Households' basic income is derived from the sale of animals

147 of the considered species and of units of the alternative product. This basic income
 148 is the product of the number of units produced and market price for each product.
 149 Additionally, farmers may receive indemnities from the state to compensate the losses
 150 incurred due to the infectious disease. A farmer f 's utility is therefore defined as:

$$U_f = \rho_f n_f P + \rho'_f n'_f P' + P_s \theta I_f \quad (1)$$

151 The farmer f simultaneously adjusts four variables, namely the number n_f of ani-
 152 mals of the species of interest present in the farm, the number n'_f of units the alternative
 153 production, and their sell rates ρ_f and ρ'_f . P and P' are the market prices, with indi-
 154 vidual farmers being price-takers (the farmer population is considered sufficiently large
 155 to ensure atomicity of the market). I_f is the number of infected animals, θ the mortal-
 156 ity rate of infected animals and P_s is the amount of indemnity provided by the state
 157 for each animals dead as a result of infection. Productions n_f and n'_f both consume
 158 household resources, mainly labor and land. The resource limit of the farm is noted
 159 M_F , considered equal across the population of farmers. The constraint implies:

$$M_F \geq \omega n_f + n'_f \quad (2)$$

160 As stated earlier, the two goods are considered to be perfect substitutes-in-production:
 161 any unit decrease in n_f enables farmers to allocate some farm resources to ω more units
 162 of the alternative production : $\frac{\partial n'_f}{\partial n_f} = -\omega$.

163 All newborn animals are susceptible (S_f) to infection and, in the course of their life,
 164 can be sold, die, or be infected with the pathogen. Infected animals (I_f) can likewise
 165 die or be sold, but their mortality rate is higher. The herd dynamics are described by
 166 the following set of differential equations:

$$\begin{aligned} \dot{S}_f &= \alpha (S_f + I_f) - \mu S_f - \rho_f S_f - \lambda S_f \\ \dot{I}_f &= \lambda S_f - \mu I_f - \rho_f I_f - \theta I_f \\ n_f &= S_f + I_f \end{aligned} \quad (3)$$

167 Where α is the breeding rate of the livestock species, μ is a natural mortality rate
 168 (with $\mu < \alpha$), λ is the force of infection (externally determined, in the individual-farm
 169 part of the model), i.e. the rate of new infections per susceptible animal per time

unit, and θ is the removal rate of infected animals resulting from disease-induced mortality or government-initiated mass culling operations implemented in contaminated herds to halt disease spread. Note that α and μ are constant in this model, i.e. the breeding and basic mortality rate depend solely on biological properties of animals and their environment. The same sell rate ρ_f applies to susceptible and infected animals. For an alternative model where farmers can destock sick animals at a higher rate, see (Delabouglise and Boni, 2020). A discussion of our choice of modelling an endemic equilibrium at farm level is provided in **Supplementary information 1**.

Figure 2: Illustration of the system dynamics. Households allocate their limited resources to two different production sectors in an optimal way in order to maximize their income. Resource allocation is based on market prices and expected productivity of the livestock herd, which in turn is constrained by losses of animals due to disease infection. The demographic and epidemiological dynamics of the animal herd are also displayed with blue sub-compartments and arrows: the herd is entirely self-renewed, animals are all born susceptible and can transit to an "infected" category in the course of their life through disease infection. The sell rate applied by farmers ensures demographic stability. Therefore a large proportion of infected animals and/or high rate of departure due to disease ($\theta I/n$) reduces the proportion of animals available for sale (i.e. the sell rate ρ).

We make three preliminary observations. First, there is a unique positive value of

180 sell rate ρ_f ensuring herd demographic stability (i.e. constant population size) (see
 181 **Supplementary information 2**). The sell rate ρ_f is therefore strictly determined
 182 by the external infection pressure λ and the severity of disease θ . If $\lambda = 0$ (in a
 183 disease-free population) or $\theta = 0$ (the disease has no effect on mortality) then farmers
 184 simply destock at a rate equal to the surplus, i.e. the difference between breeding
 185 and mortality rate $\rho_f = \alpha - \mu$, hereafter referred to as ρ_0 . With increasing disease
 186 incidence and/or disease severity, farmers reduce ρ_f : more adult animals need to be
 187 kept for breeding rather than being sold in order to compensate the loss of animals
 188 due to the disease. All farms in our model have the same properties and face the same
 189 force of infection λ , thus for an individual farmer we write $\rho_f = \rho(\lambda)$ to indicate that
 190 the force of infection influences the sell rate (as empirically observed in Delabougliše
 191 et al. (2020)). The second observation is that, for high values of either λ and θ the herd
 192 goes extinct regardless of the value of ρ_f . This outcome is obtained when the excess
 193 departure due to disease mortality exceeds the self-renewing capacity of the herd, i.e.
 194 when:

$$\lambda \frac{\theta - (\alpha - \mu)}{\theta + \mu} > \alpha - \mu \quad (4)$$

195 The third observation is that the expected proportion of infected animals in the
 196 herd (Z) is directly determined by λ :

$$\frac{I_f}{n_f} = Z(\lambda) = \frac{\lambda}{\lambda + \mu + \theta + \rho(\lambda)} \quad (5)$$

197 The sell rate of alternative product ρ'_f can also be assumed to be limited by pro-
 198 ductivity constraints but independent of λ and therefore equal to a constant noted ρ'_0 .
 199 The marginal utility function of herd expansion (i.e. adding one more animal to the
 200 herd) can be defined as:

$$\frac{\partial U_f}{\partial n_f} = \rho(\lambda)P + \theta Z(\lambda)P_s - \omega \rho'_0 P' \quad (6)$$

201 With $\rho(\lambda)P + \theta Z(\lambda)P_s$ and $\omega \rho'_0 P'$ being the marginal benefit and marginal oppor-
 202 tunity cost, respectively, of livestock herd expansion. By adding one livestock head
 203 to their herd, the farmers forego the opportunity of producing ϵ units of alternative
 204 product. The optimal livestock herd size is therefore determined by market prices P
 205 and P' , the level of losses of livestock incurred, which is captured by $\rho(\lambda)$, and the

206 revenue earned from state indemnities, which is captured by $\theta Z(\lambda)(\lambda)P_s$.

207 **2.3 Disease dynamics in a population of farms**

208 In addition to the individual farm model, we track the total number of susceptible
 209 ($S = \sum_f S_f$) and infected ($I = \sum_f I_f$) animals in a region or community with many
 210 farms. These classical dynamics are non-linear and dependent on the total number of
 211 infected animals, and they determine the community-level force of infection λ that is
 212 experienced by individual farms.

$$\begin{aligned}\dot{S} &= \alpha(S + I) - \mu S - \rho S - \beta SI \\ \dot{I} &= \beta SI - \mu I - \rho I - \theta I \\ n &= S + I\end{aligned}\tag{7}$$

213 with β as the community-level transmission parameter. The force of infection λ expe-
 214 rienced by a single farm is:

$$\lambda = \beta I = \beta n - (\mu + \theta + \rho)\tag{8}$$

215 And the reproduction ratio of the disease, the number of infections caused by a single
 216 infected animals in a fully susceptible population, is:

$$R_0 = \frac{\beta n}{\mu + \theta + \rho}\tag{9}$$

217 Transmission is density dependent, i.e. the reproduction ratio is positively correlated
 218 with the size of the animal population and the establishment of the disease in the
 219 population is only possible if n is above a given threshold n_T , which results in $R_0 > 1$.

220 **2.4 Livestock meat price**

221 We consider, without loss of generality, that the slaughter of an animal produces one
 222 unit of meat so that the total quantity of meat (q) and alternative product (q') produced
 223 by the population of farmers at a given time are:

$$\begin{aligned} q &= \rho n \\ q' &= \rho' n' \end{aligned}$$

224 We do not model the intermediate transactions occurring between producers and con-
 225 sumers and assume the farm-gate price is equal to the retail price. We also assume
 226 that livestock meat cannot be stored, so that the price at a given time is determined
 227 by demand and number of livestock slaughtered. A consumer chooses the quantity
 228 of livestock meat q and alternative product q' purchased based on two criteria: price
 229 and perceived quality, the later being affected by the presence of the disease in the
 230 consumed animal. We assume that consumers do not know the origin of the purchased
 231 meat but are sufficiently aware of the current disease prevalence and, therefore, able
 232 to estimate the risk Z that the purchased meat come from an infected animal. Hence
 233 the expression of livestock meat market price is:

$$\begin{aligned} P &= a - bq - cq' - dZ \quad \text{if } a - bq - cq' - dZ > 0 \\ &= 0 \quad \text{if } a - bq - cq' - dZ < 0 \end{aligned}$$

And

$$\begin{aligned} P' &= a' - b'q' - cq \quad \text{if } a' - b'q' - cq > 0 \\ &= 0 \quad \text{if } a' - b'q' - cq < 0 \end{aligned}$$

234 With a , a' , a'' , b , b' , c , and d constant positive parameters. See **Supplementary**
 235 **information 3** for a detailed description of the demand model and price formation.
 236 While the demand model does not have constant own-price and cross-price elasticities,
 237 b and b' are related to the own-price elasticity of demand for the two products: a
 238 high value of b indicates that consumers are inflexible in their consumption of livestock
 239 meat and the quantity of livestock meat consumed is not highly responsive to changes
 240 in meat price P . The parameter c relates to the potential substitution effects between
 241 the consumption of the two products and, thereby to the cross-prices elasticities. The
 242 parameter d is the disutility associated with the consumption of a unit of meat origi-
 243 nating from an infected animal. Prices can be modified by specific policies, for example
 244 the introduction of a ceiling on livestock meat would mean that P cannot be above a
 245 given value, while a subsidy on price P' would mean that farmers earn a revenue from
 246 the sale of alternative products above the market price. This is developed in more
 247 details in section 3.4.

2.5 Criteria of equilibrium and stability

Farmers maximize their income by optimizing their herd size n_f while the sell rate ρ takes a unique value ensuring herd demographic stability. The sum of all herd sizes n determines both market prices P and P' and the infection pressure λ farmers are facing. Any stable equilibrium population size n needs to satisfy two conditions (Thomas, 1984):

$$\frac{\partial U_f}{\partial n_f} = 0 \quad (10)$$

$$\frac{\partial^2 U_f}{\partial n \partial n_f} < 0 \quad (11)$$

Condition (10), i.e. equality between marginal benefit and cost of herd expansion, ensures n is an equilibrium while condition (11) ensures the equilibrium is stable. Hereafter we will refer to Nash equilibria allowing and not allowing disease circulation as endemic and disease-free respectively.

3 Results

Our economic-epidemiological livestock model where individual farms simultaneously produce goods and disease risk admits a maximum of three Nash equilibria, two stable and one unstable (see proof in **Supplementary information 4**). Specifically, the system has either a single stable Nash equilibrium, or it has three Nash equilibria (two stable, one unstable).

3.1 Uniqueness and conditions of stability of the disease-free equilibrium

In the absence of the disease, the herd sell rate ρ is constant and equal to ρ_0 . Marginal benefit of herd expansion is a linear decreasing function of n , which results in a single disease-free equilibrium (DFE) combination of market price and population size $\{P_{\text{DFE}}, P'_{\text{DFE}}, n_{\text{DFE}}, n'_{\text{DFE}}\}$ (see demonstration and mathematical formulation of equilibria in **Supplementary information 4**).

The food demand model has own-price elasticities governed by b and b' – the two coefficients that dictate how inflexible consumers are in the quantity of livestock meat that they consume – and the coefficient c describing the substitutability between these two products. The own-price elasticities at disease-free market equilibrium are:

$$\begin{aligned}\epsilon &= \frac{\partial q}{\partial P} \frac{P_{\text{DFE}}}{q_{\text{DFE}}} = -\frac{P_{\text{DFE}}}{\rho_0 n_{\text{DFE}}} \frac{b'}{bb' - c^2} \\ \epsilon' &= \frac{\partial q'}{\partial P'} \frac{P'_{\text{DFE}}}{q'_{\text{DFE}}} = -\frac{P'_{\text{DFE}}}{\rho'_0 n'_{\text{DFE}}} \frac{b}{bb' - c^2}\end{aligned}\tag{12}$$

276 Similarly, we can derive an expression of the cross-price elasticities of demand for the
277 livestock and alternate product at disease-free equilibrium:

$$\begin{aligned}\epsilon_c &= \frac{\partial q}{\partial P'} \frac{P'_{\text{DFE}}}{q_{\text{DFE}}} = \frac{P'_{\text{DFE}}}{\rho_0 n_{\text{DFE}}} \frac{c}{bb' - c^2} \\ \epsilon'_c &= \frac{\partial q'}{\partial P} \frac{P_{\text{DFE}}}{q'_{\text{DFE}}} = \frac{P_{\text{DFE}}}{\rho'_0 n'_{\text{DFE}}} \frac{c}{bb' - c^2}\end{aligned}\tag{13}$$

278 where high values of b and b' are indicative of inelastic demand for the livestock meat
279 and the alternative product respectively. A high value of c is indicative of a high
280 cross-price elasticity between the two alternative products: with a higher value of c ,
281 more consumers are likely to increase their demand for one product in response to an
282 increase of the price of the other. The stability of the disease-free equilibrium depends
283 on $R_0(n_{\text{DFE}})$. If $R_0(n_{\text{DFE}}) < 1$ the disease cannot persist in the population and any
284 introduced pathogen ultimately goes extinct. If $R_0(n_{\text{DFE}}) > 1$, the population can be
285 invaded by the disease, in which case livestock production and consumer demand are
286 altered. Analytically, we can show that the disease-free equilibrium is unstable if and
287 only if:

$$n_{\text{DFE}} > \frac{\alpha + \theta}{\beta} = n_T, \tag{14}$$

288 As in traditional epidemiological models, when the community's total population size
289 is above a threshold value n_T . We can observe that a sufficiently large disease-induced
290 mortality rate θ reduces disease transmission and enables the stabilization of a disease-
291 free equilibrium as it shortens the time period during which infected animals can trans-
292 mit the disease. We denote θ_T as the threshold value of θ above which the disease-free
293 equilibrium is stable (see a mathematical formulation of θ_T in **Supplementary infor-**
294 **mation 4**). Note that a large breeding rate α has the same effect as a large θ , since it
295 implies that farms have rapid turnover in their animal populations, and the pathogen
296 is not able to invade or establish; this effect is not present in human disease models.

3.2 Conditions of stability of the endemic equilibrium

At endemic equilibrium, provided the disease causes some mortality ($\theta > 0$), the sell rate ρ is affected by the infection risk λ which, in turn, is determined by the population size n . In the face of increasing risk of mortality due to disease, farmers have to lower their sell rate in order to maintain the balance between births and departures. Consequently, the marginal return of herd expansion $\frac{\partial U_f}{\partial n_f}$ is no longer a linear decreasing function of the community size n . The sign of $\partial U_f / \partial n_f$ is determined by a continuous cubic function of n , shown in **Supplementary information 4**. For very large n , $\partial U_f / \partial n_f$ is ultimately negative, ensuring that in the absence of disease-free equilibrium, there is at least one endemic equilibrium (as illustrated in **Figure 3**).

This also implies that, under some specific conditions, for a range of intermediate values of n , $\frac{\partial U_f}{\partial n_f}$ can be an increasing function of n . Qualitatively, we observe that this counterintuitive outcome is obtained when the demand for livestock meat is inelastic and the disease-induced mortality is high (i.e. low ϵ , high θ). Under this condition, when the community size n is moderate but sufficient to sustain disease transmission, a limited increase in community size paradoxically results in a reduction in meat production and an increase of meat market price P , as the steep increase in the proportion of infected animals compels farmers to considerably reduce their sell rate. The positive relationship between $\frac{\partial U_f}{\partial n_f}$ and population size allows the establishment of three Nash equilibria, as illustrated in **Figure 3** (bottom right graph). The intermediate equilibrium, characterized by $\frac{\partial^2 U_f}{\partial n \partial n_f} > 0$, is unstable. In this situation of system bistability, farmers have two stable strategies: (i) keeping a low herd size, ensuring $R_0 < 1$, good productivity ($\rho = \rho_0$) and moderate sale price $P = P_{\text{DFE}}$, or (ii) keeping a large herd size, with $R_0 > 1$ and productivity undermined by disease losses ($\rho < \rho_0$) which is compensated for by high sale price $P > P_{\text{DFE}}$.

3.3 Effect of disease-induced livestock mortality on herd size

The effect of disease-induced mortality on the animal population is strongly dependent on the elasticities of demand for livestock meat and the alternative product. Under endemic equilibrium, a high disease-induced mortality rate forces farmers to sell fewer animals. If the demand for livestock meat is elastic, the limited price increase caused by the shortage of livestock meat does not compensate for a farmer's production loss, and farmers would tend to reduce their herd size in order to redirect their resources to the production of the alternative good; this in turn reduces disease prevalence (top **Figure 3**). If demand is sufficiently inelastic, then the price increase offsets the production loss and farmers expand their herd to increase profits from the high livestock

333 meat sale price, which in turn increases disease prevalence (bottom **Figure 3**). Note
 334 that the relationship between herd size and disease-induced mortality is non-linear,
 335 with a peak in animal population when θ is close to the livestock natural growth rate
 336 $\alpha - \mu$.

337
 338 When $\theta < \theta_T$ the stable equilibrium is endemic. When $\theta > \theta_T$ the disease-free equilib-
 339 rium is stable. The equilibrium is unique in the case of elastic demand for livestock
 340 meat, as there is no benefit for farmers in maintaining a herd size above n_T (top **Figure**
 341 **3**). When livestock meat demand is inelastic, bistability may occur, with two simul-
 342 taneously stable equilibria (one endemic, one disease-free; **Figure 3**). At the endemic
 343 equilibrium, herds are kept sufficiently large to allow the disease to circulate. The high
 344 mortality of livestock due to disease forces farmers to limit the sale of their animals,
 345 but this loss is compensated for by the high meat market prices, providing sufficient in-
 346 centive for farmers to maintain large herds. One equilibrium is clearly socially optimal
 347 (the disease-free equilibrium) while the second (the endemic equilibrium) is character-
 348 ized by ongoing disease circulation, low consumer welfare due to high meat prices, and
 349 inefficient use of farm resources.

350
 351 As illustrated in the supplementary **Figure S1 (supplement to Figure 3)**, inelastic
 352 demand for the alternative product considerably reduces variation of animal popula-
 353 tion size. The reason is that any expansion of the livestock population results in a
 354 decreased production of alternative good. If the demand for the alternative good is in-
 355 elastic, the resulting increase in P' , encourages farmers to maintain a high production
 356 of the alternative good and limit livestock herd expansion. Likewise, when livestock
 357 herd size is reduced and production of the alternative good is increased, the resulting
 358 drop in P' encourages farmers to rebalance by shifting resources back into livestock
 359 production. As a consequence, bistability is less likely: the range of values of θ allowing
 360 both disease-free and endemic equilibria is smaller.

Figure 3

Figure 3. Effect of the level of disease-induced removal rate (θ) on the system Nash equilibria population sizes (n), numbers of infected animals per herd (I), and livestock market prices (P). All variables are scaled to be simultaneously represented on the same graph. Stable and unstable equilibria are represented with solid and dotted curves respectively. The relationship is explored for own-price elastic (low b , top graph) and own-price inelastic (high b , bottom graph) demand for livestock meat. In both cases, the own-price elasticity of the demand for the alternative product is high (low b' , $\epsilon' = -4$), and there is no substitution effect between the consumption of the two products ($c = 0$), no food scare ($d = 0$), and no state indemnities ($P_s = 0$). See **Figure S1 (supplement to Figure 3)** for an alternative setting with inelastic demand for the alternative product. Additionally, three smaller graphs display the relationship between the marginal benefit (blue curve) and marginal opportunity cost (orange curve) of expanding the one's herd size (n_f) for households in response to increasing population size (n) under three conditions: no infectious disease, disease with low disease-induced departure rate ($\theta = 0.1$), and disease with high disease-induced departure rate ($\theta = 0.35$). Dots indicate Nash equilibria which are either disease-free and stable (gray dots), endemic stable (black dots), or endemic unstable (white dot). Dotted lines and dashed lines indicate disease-free equilibrium herd size (n_{DFE}) and threshold population size enabling disease propagation (n_T), respectively.

380

3.4 Effect of demand structure on farmers's strategic response and policy implications

The results in the previous section show that inelastic demand for livestock products can exacerbate livestock epidemics and undermine efforts aimed at eradicating an infectious disease. If a government initiates a control program based on the rapid detection and culling of infected animals or animals located in infected farms, which is equivalent to an increase of disease-induced mortality, we see that the an expansion of herd size is a stable strategic response for farmers, and that this strategy allows for persistence of the disease at levels of culling which would normally result in disease eradication. This scenario corresponds to the bistability shown in **Figure 3, bottom right** and displayed in **Figure 4A**. Similarly, this strategic response may allow a highly pathogenic infectious agent to maintain its circulation in a livestock population when it would be expected to go extinct if the farmers did not increase their herd size. In this section we explore different modifications to the demand structure and policy interventions which

395 may solve this perverse effect.

396

397 Introducing a substitution term between the consumption of the two products ($c > 0$)
398 or a consumer's aversion for livestock disease ($d > 0$) both decrease the likelihood of
399 bistability. When the two products are substitutes, the drop in livestock production
400 caused by the disease results in a subsequent increase in the alternate product's price,
401 as consumers partly compensate for decreased livestock meat with an increased de-
402 mand for the alternative product, thereby increasing the marginal opportunity cost of
403 herd expansion when the disease circulates (**Figure 4B**). Consumer's aversion towards
404 meat originating from infected livestock (the "food scare" effect) enables a decrease
405 of the endemic equilibrium livestock meat price and thereby decreases the marginal
406 benefit of livestock herd expansion at endemic equilibrium (**Figure 4C**).

407

408 Introducing the payment of an indemnity for each animal lost by farmers as a re-
409 sult of the disease ($P_s > 0$) worsens the situation, as it increases the marginal benefit
410 of herd expansion when the disease is circulating (**Figure 4D**): farmers are willing to
411 stock more animals not only because of the high sale price of the survivors, but also be-
412 cause disease losses are compensated by state indemnities. On the contrary, enforcing
413 a ceiling on livestock market price P sets a limit to the marginal benefit farmers derive
414 from expanding their herd and thereby reduce the likelihood of endemic equilibrium
415 (**Figure 4E**). Finally, a subsidy on the sale price of the alternative product in case of
416 endemicity (subsidies are only granted if the disease circulates) have positive effects,
417 as it increases the marginal opportunity cost of herd expansion (**Figure 4F**).

Figure 4: Illustration of the effect of some variations in demand structure and policy intervention on bistability of disease-free and endemic stable Nash equilibria. Lines indicate baseline (dashed) and new (solid) marginal benefits (blue) and marginal costs (orange) of herd expansion for increasing values of the community size n . (A) Baseline scenario with inelastic consumer demand. Effects of changes in the demand structure include (B) the introduction of a substitution term between the two products supplied by farmers ($c > 0$), (C) aversion of consumers towards consumption of meat from diseased animals ($d > 0$). Policy interventions include (D) the payment of an indemnity to farmers for each dead animal as a result of the disease ($P_s > 0$), (E) enforcement of a ceiling on livestock market price P , and (F) a subsidy to the sale price P' of the alternative product. The vertical dashed grey line is the threshold community size enabling sustained disease circulation (n_T)

4 Discussion

In most countries, livestock meat is a necessity good characterized by inelastic demand. In general, meats from different animal species are consumption substitutes with positive cross-price elasticities (Shibia et al., 2017; Wadud, 2006; Chen et al., 2015; Fayaad

et al., 1995). Supply shocks caused by livestock epidemics tend to drive livestock meat prices up, as exemplified by the current situation of the pig industry in China, where African Swine Fever is still circulating despite government policy responses based on rapid detection of outbreaks, preventive culling of pigs and movement controls. Despite the risk of disease-induced production losses, a USDA report indicated that farmers were willing to restock early or expand their herd after the onset of the ASF epidemic, in anticipation of the benefits obtained from the sale of surviving animals (USDA, 2019). An economic analysis confirmed that the benefits obtained by the Chinese industry as a result of the supply shocks exceeded the costs resulting from production losses, with up to 40% excess returns compared to baseline predictions during the 2019 spring festival, when the demand for pork was highest (Chen et al., 2020; Xiong et al., 2020; Reuters, 2019). Increasing net profits seems to be encouraging the rapid rebuilding of the pig breeding population, and the production is increasing faster than expected (Reuters, 2021). However, this expansion of pig farms is unequally distributed across production systems. Although accurate official data are missing, reports indicate a large fraction of Chinese small scale farms interrupted or quit pork production while large scale farms are rapidly expanding in number size (Xiong et al., 2020; Reuters, 2020b). This concentration of the pig industry partly owes to governmental support benefiting mostly, if not exclusively, to large scale producers, like financial subsidies and loans, and a downgrading of environmental regulations and land use restrictions, while in some provinces small scale farms were banned from restocking (Chen et al., 2020; Xiong et al., 2020). Owing to economies of scales and easy access to financial credits, large scale farms are also more likely to implement preventive actions to limit production losses due to infectious diseases, giving them a competitive advantage over small scale producers. Our model does not capture such dynamics as we did not incorporate heterogeneity in farm sizes and costs of risk-reduction measures.

The co-occurrence of infectious disease and market dynamics can be observed with other types of animal products. The recent mass culling of farmed minks in Europe resulted in a supply shock on the global fur market. Consequently, even though the farming of minks and other wild animals are among the suspected drivers of the emergence of SARS-CoV-2 in humans (Zhou and Shi, 2021; NPR, 2021), the Chinese mink industry currently benefits from the rise in fur prices (Reuters, 2020a).

When disease spread is facilitated by a high density of animals, the most critical aspects of disease control are the evolution of the size of the livestock population in response to epidemics, their resulting control policies, and their market effects. Our model shows that in the context of inelastic demand for meat of the affected species,

the adaptation of herd size by farmers can undermine the benefits of a disease control policy based on the culling of affected herds. A previous theoretical work showed the potential perverse effects of culling policies when the indemnities provided to farmers encourage an expansion of their farm size (Boni et al., 2013). Here we demonstrate that, even without indemnities, the market response alone may drive an overstocking of animals with adverse epidemiological and economic effects.

The policy messages we derive from our analysis are twofold. First, in the presence of inelastic demand for livestock meat, animal disease control policies should be complemented with economic measures aimed at discouraging an expansion of the herds of susceptible animals. One option is to limit livestock meat price increase. While enforcing a ceiling on market price may be impracticable, a temporarily removal of entry barriers to imports of livestock products, enabling the supply of the domestic market with imported meat, can contribute to maintain domestic prices at a low level. Our results show that state indemnities provided to farmers to compensate the loss of animals due to disease have perverse effects, but removing these subsidies would not be acceptable for farmers confronted with large income losses, especially if these losses are due to depopulation measures initiated by the government. A potential solution would be to encourage farmers to invest in an alternative production, by conditioning a fraction of the state indemnities on the stocking of an alternative species of livestock, preferentially a close consumption substitute to the species affected by the disease. In the case of ASF in China, a recent study underlined the benefits of subsidizing poultry production as complementary to supporting the pig industry, as poultry is a close consumption substitute to pigs and has the added advantage of being less resource-consuming than pig and beef and rapidly available to consumers because of its short production cycle (Ma et al., 2020). Second, livestock disease control benefits from a diversification of protein production and consumption. The co-existence of different types of food productions satisfying the protein needs of consumers, based on livestock, fish, insects or vegetables, encourages consumers and producers to respond to epidemics by replacing susceptible animals with their substitutes in their farms and in their diets respectively, instead of overstocking animals of the susceptible species. Excessive stocking of susceptible animals can also be averted if information made available to consumers on ongoing disease outbreaks lessen their demand for the meat of the susceptible species, especially if the disease has some zoonotic potential. However, food scares may have long-lasting effects on patterns of meat consumption which can outlive the disease epidemics and are not easily predictable (Taylor et al., 2016).

The main limitation of our model is that it only captures one aspect of farmers' adap-

tation to disease risk, i.e adjustment of herd size, leaving aside other aspects such as vaccination, disinfection, and rapid sale or slaughter of sick animals (Delabougliise et al., 2020). The rapid sale of sick animals can be considered as a farmer-initiated increase of the removal rate associated with disease infection to avoid feeding animals which are likely to die before maturity. The results are based on the assumption that the transmission of the disease is to some extent density dependent, i.e. that its spread is facilitated by a high population size. This assumption holds for most cases of infectious disease transmitted through direct contact within and between herds, but it might not be relevant to all epidemiological settings. For example, when disease transmission is mainly mediated by long-distance trade movements, disease transmission may be dependent on the frequency of animal transports and movement network properties rather than on the number of farmed animals (Delabougliise and Boni, 2020).

With a consistently high frequency of disease emergence observed at the interface between animals and humans and rising concerns around the environmental impact of the livestock industry, it is increasingly clear that prospects for the development of livestock production systems are tightly linked to their ability to reduce the burden of infectious disease and effectively manage the risk of livestock epidemics. Our analysis underlines some potential effects of strategic reactions of farmers to disease epidemics, with joint economic and epidemiological dynamics potentially leading to an overstocking of animals. This adverse outcome may be corrected in the short term by providing the right incentives to farmers, and in the long term by favoring diversification of protein production systems.

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5 Supplementary information

5.1 Supplementary information 1. Hypothesis of farm-level endemicity

Our assumption of stable herd-level disease prevalence is arguably simplistic. For most known cases of highly contagious livestock diseases, endemicity can be maintained at the population level by a sustained between-farm circulation but the disease is unlikely to remain endemic in a single farm, since the reduction of the number susceptible individuals is too rapid after pathogen introduction into the herd to sustain the chain of infections. However, farms undergo frequent reintroductions of the disease causing short episodes of high mortality. However, on the long term these different epidemiological patterns may result in the same level of losses due to mortality, and resulting adjustment in the sell rate, as farmers account for the expected risk of losses due to episodes of high mortality in their herd management plan.

5.2 Supplementary information 2. Farm demographic model

following from the differential equation (3), any herd demographic equilibrium must satisfy:

$$\rho_f^2 - (\alpha - (\theta + \lambda + 2\mu))\rho_f - \theta\alpha - (\mu + \lambda)(\alpha - \theta - \mu) = 0 \quad (15)$$

The only equation parameter farmers can modify is ρ_f . Therefore, a necessary condition for demographic stability is that there is a positive solution ρ to the quadratic equation (15). And (15) only admits one unique positive solution if and only if:

$$\begin{aligned} \theta &< \alpha - \mu \\ \text{or} \\ \lambda &< \frac{(\theta + \mu)(\alpha - \mu)}{\theta - (\alpha - \mu)} \end{aligned} \quad (16)$$

5.3 Supplementary information 3. Food demand model

We formulate the following expression of the consumer's utility function:

$$U = f(q, q', q'', Z) \quad (17)$$

With q and q' the consumed quantity of product originating from the considered livestock species and the alternative product respectively and q'' the quantity of all other

consumption goods not produced by the farmer population. Consumer utility is affected negatively by the information consumers receive on the disease prevalence in the livestock population ($Z = I/n$). Consumers make their consumption choices to maximize f under the following budget constraint:

$$M_C \geq qP + q'P' + q''P''$$

With P , P' , and P'' the market prices of the corresponding products and M_C the budget limit of consumers. For the sake of mathematical tractability, we posit a polynomial expression of f , where the disutility effect associated with disease information is considered to be strictly additive:

$$U(q, q', q'', Z) = K + aq + a'q' + a''q'' - \frac{b}{2}q^2 - \frac{b'}{2}q'^2 - cq q' - dqZ \quad (18)$$

With K , a , a' , a'' , b , b' , c , and d constant positive parameters. From the budget constraint equation we derive:

$$\frac{\partial q''}{\partial q} = \frac{P}{P''} \text{ and } \frac{\partial q''}{\partial q'} = \frac{P'}{P''}$$

Hence $a'' \frac{P}{P''}$ and $a'' \frac{P'}{P''}$ are the decrease (increase) in consumption of all other goods resulting from the purchase (sale) of a unit livestock meat and alternative product respectively. In all equations we consider, without loss of generality $P = a'' \frac{P}{P''}$ and $P' = a'' \frac{P'}{P''}$. Therefore marginal utility of a consumer associated with an increased consumption of each product has a simple reduced form:

$$\begin{aligned} \frac{\partial U}{\partial q} &= a - bq - cq' - dZ - P \\ \frac{\partial U}{\partial q'} &= a' - b'q' - cq - P' \end{aligned} \quad (19)$$

While the demand model does not have constant own-price and cross-price elasticities, b and b' are related to the own-price elasticity of demand for the two products: a high coefficient b means that the marginal utility of livestock meat consumption steeply decreases with q , making consumers inflexible in their consumption of livestock meat and the quantity of livestock meat purchased rather irresponsive to changes in meat price P . c relates to the potential substitution effects between the consumption of the two products and, thereby to the cross-prices elasticities: if $c > 0$ the marginal utility of livestock meat consumption also decreases with q' as the alternative product satisfies some of the nutritional needs also covered by the consumption of livestock meat. Note,

however, that c is strictly lower than both b and b' , since the two products do not have the exact same characteristics.

5.4 Supplementary information 4. Identification of system equilibria

When the disease is endemic ($R_0 > 1$ or $I > 0$), we find a general formulation for disease prevalence $Z = I/n$ and the population-average herd sell rate ρ :

$$Z = 1 - \frac{\alpha}{\beta n - \theta}$$

$$\rho = \alpha - (\theta + \mu) + \frac{\theta \alpha}{\beta n - \theta}$$

Which leads to the following formulation of the marginal utility of herd expansion at equilibrium. Critically, this marginal utility depends upon the epidemiological status of the population, i.e. whether the disease does not invade ($R_0 < 1$) or invades ($R_0 > 1$):

$$\begin{aligned} \frac{\partial U_f}{\partial n_f} \Big|_{R_0 < 1} &= A(\alpha - \mu) - A' - (B(\alpha - \mu)^2 + B' - 2C(\alpha - \mu)) n \\ \frac{\partial U_f}{\partial n_f} \Big|_{R_0 > 1} &= \frac{1}{\mathbf{x}^2} \left(\frac{1}{\beta} \left(-B' - B(\alpha - (\theta + \mu))^2 + 2C(\alpha - (\theta + \mu)) \right) \mathbf{x}^3 \right. \\ &\quad + \left(-A' + A(\alpha - (\theta + \mu)) - B' \frac{\theta}{\beta} - B \frac{\alpha - (\theta + \mu)}{\beta} (3\theta \alpha - \theta(\theta + \mu)) \right. \\ &\quad + \left. 2C \frac{\theta}{\beta} (2\alpha - (\theta + \mu)) - d(\alpha - (\theta + \mu)) + P_s \theta \right) \mathbf{x}^2 \\ &\quad + \alpha \left(A\theta - B \frac{\theta^2}{\beta} (3\alpha - 2(\theta + \mu)) + 2C \frac{\theta^2}{\beta} - D(2\theta - (\theta + \mu)) - P_s \theta \right) \mathbf{x} \\ &\quad \left. + \alpha^2 \left(-B \frac{\theta^3}{\beta} + D\theta \right) \right) \end{aligned} \tag{20}$$

$$\begin{aligned} \text{With } \mathbf{x}(n) &= \beta n - \theta & R_0(n) &= \frac{\beta n}{\alpha + \theta} \\ A &= a - c\rho' M_F & A' &= \rho' \epsilon (a' - b' \rho' M_F) \\ B &= b & B' &= b' \rho'^2 \epsilon^2 \\ C &= c\rho' \epsilon & D &= d \end{aligned}$$

$\frac{\partial U_f}{\partial n_f}|_{R_0 < 1}$ is continuous, linear and strictly decreasing in n . There is a threshold value of n , noted n_T allowing disease propagation ($R_0(n) > 1$ if and only if $n > n_T$).

$$n_T = \frac{\alpha + \theta}{\beta}$$

Which corresponds to $\mathbf{x} = \alpha$. The equilibrium disease-free herd size is unique and takes the value:

$$n_{\text{DFE}} = \frac{A(\alpha - \mu) - A'}{B(\alpha - \mu)^2 + B' - 2C(\alpha - \mu)}$$

If $R_0(n_{\text{DFE}}) > 1$ ($n_{\text{DFE}} > n_T$) then the disease-free equilibrium is unstable. Otherwise the disease-free equilibrium is stable, since $\frac{\partial^2 U_f}{\partial x \partial n_f}|_{R_0 < 1} < 0$. We can also define a threshold value for the disease-induced removal rate θ , noted θ_T , above which the disease-free equilibrium is stable.

$$\theta_T = \beta \frac{A(\alpha - \mu) - A'}{B(\alpha - \mu)^2 + B' - 2C(\alpha - \mu)} - \alpha$$

$\frac{\partial U_f}{\partial n_f}|_{R_0 > 1}$ is continuous and not linear on $\mathbf{x}$, while $\mathbf{x}$ is a linear increasing function of n . Based on equation (20), it is obvious that the equilibrium values of $\mathbf{x}$, satisfying $\frac{\partial U_f}{\partial n_f}|_{R_0 > 1} = 0$, are the roots of a cubic equation on the interval $[\alpha; +\infty[$. Also, the marginal utility function is continuous on n_T , i.e. $\frac{\partial U_f}{\partial n_f}(n_T)|_{R_0 < 1} = \frac{\partial U_f}{\partial n_f}(n_T)|_{R_0 > 1}$, ensuring full continuity of the function over the whole range of possible values of n . The term in $\mathbf{x}^3$ being strictly negative ($b' > c$ and $b > c$) the marginal utility tends towards $-\infty$ for extreme values of $\mathbf{x}$. We can distinguish two situations:

Situation 1: The disease free equilibrium is unstable ($n_{\text{DFE}} > n_T$). Then $\frac{\partial U_f}{\partial n_f}(\mathbf{x} = \alpha) > 0$. On the interval $[\alpha; +\infty[$, there is either one stable equilibrium value x^* (i.e. satisfying $\frac{\partial U_f}{\partial n_f} = 0$ and $\frac{\partial^2 U_f}{\partial x \partial n_f}(x^*) < 0$) or three equilibria x^* , two being stable ($\frac{\partial^2 U_f}{\partial x \partial n_f}(x^*) < 0$), and the other unstable ($\frac{\partial^2 U_f}{\partial x \partial n_f}(x^*) > 0$).

Situation 2: the disease free equilibrium is stable ($n_{\text{DFE}} < n_T$). Then $\frac{\partial U_f}{\partial n_f}(\mathbf{x} = \alpha) < 0$. On the interval $[\alpha; +\infty[$, there is either no equilibrium value or two equilibria x^* , one being stable ($\frac{\partial^2 U_f}{\partial x \partial n_f}(x^*) < 0$), and the other unstable ($\frac{\partial^2 U_f}{\partial x \partial n_f}(x^*) > 0$).

Consequently, the system has either one Nash equilibrium or three Nash equilibria two of which being stable. If $n_{\text{DFE}} < n_T$ ($\theta > \theta_T$), one of these equilibria is disease-free, with $n = n_{\text{DFE}}$.

5.5 Supplementary information 5. Figure S1 - supplement to Figure 3

Figure S1 (supplement to Figure 3)

Figure S1

Figure S1 (supplement to Figure 3). Effect of the level of disease-induced removal rate (θ) on the system Nash equilibria population size (n), number of infected animals per herd (I) and livestock market price (P). All variables are scaled to be simultaneously represented on the same graph. Stable and unstable equilibria are represented with solid and dotted curves respectively. The relationship is explored for own-price elastic (low b , top graph) and own-price inelastic (high b bottom graph) demand for livestock meat. In both cases, the own-price elasticity of the demand for the alternative product is low (high b' , $\epsilon' = -0.2$), and there is no substitution between the consumption of the two products ($c = 0$), no food scare ($d = 0$), and no state indemnities ($P_s = 0$). Additionally, three smaller graphs display the relationship between the marginal benefit (blue curve) and marginal opportunity cost (orange curve) of expanding the one's herd size (n_f) for households in response to increasing livestock population size (n) under three conditions: no infectious disease, disease with low disease-induced departure rate ($\theta = 0.1$), and disease with high disease-induced departure rate ($\theta = 0.35$). Dots indicate Nash equilibria which are either disease-free and stable (gray dots), endemic stable (black dots) or endemic unstable (white dot). Dotted lines and dashed lines indicate disease-free equilibrium herd size (n_{DFE}) and threshold population size enabling disease propagation (n_T) respectively.