The impact of an African swine fever outbreak on endemic tuberculosis in wild boar populations

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Abstract

Animal tuberculosis (TB) is a widespread infectious disease caused by the *Mycobacterium tuberculosis* bacteria belonging to the *Mycobacterium tuberculosis complex* (MTC) that can persist in reservoir wildlife hosts. Wild boar (*sus scrofa*) are a key reservoir for MTC and an increasing trend in wild boar density is expected to lead to an increase in TB prevalence with spill-over to livestock. MTC infection is presently controlled through a variety of strategies, including culling. African swine fever (ASF) is a virulent, viral infection which affects wild boar and is spreading across Eurasia and Oceania. ASF infection leads to near 100% mortality at the individual level, can cause a dramatic decrease in population density and may therefore lead to TB control. In this study we develop a mathematical model to examine the impact of ASF introduction into a wild boar population that supports endemic TB. Our model results indicate that an ASF infection will reduce wild boar population density and lead to a decrease in the prevalence of TB. If ASF persists in the local host population the model predicts the long-term decline of TB prevalence in wild boar. If ASF is eradicated, or fades-out in the local host population, the model predicts a slower recovery of TB prevalence in comparison to wild boar density after an ASF epidemic. This may open a window of opportunity to apply TB management to maintain low TB prevalence.

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Animal tuberculosis (TB) is a widespread infectious disease caused by bacteria belonging to the *Mycobacterium tuberculosis complex* (MTC), mainly *M. bovis* and *M. caprae* , that can persist in wildlife reservoir hosts. Wild boar (*Sus scrofa*) are a key reservoir for MTC on the Iberian Peninsula and an increasing trend in wild boar density is expected to lead to an increase in TB prevalence with spill-over to livestock. MTC infection is presently controlled through a variety of strategies, including culling. African swine fever (ASF) is a virulent, viral infection which affects wild boar and is spreading across Eurasia and Oceania. ASF infection leads to near 100% mortality at the individual level, can cause a dramatic decrease in population density and may therefore, paradoxically, contribute to TB control. In this study we develop a mathematical model to examine the impact of ASF introduction into a wild boar population that supports endemic TB. Our model results indicate that an ASF infection will reduce wild boar population density and lead to a decrease in the prevalence of TB. If ASF persists in the local host population the model predicts the long-term decline of TB prevalence in wild boar. If ASF is eradicated, or fades-out in the local host population, the model predicts a slower recovery of TB prevalence in comparison to wild boar density after an ASF epidemic. This may open a window of opportunity to apply TB management to maintain low TB prevalence.

1 Introduction

Animal tuberculosis (TB) is a widespread multi-host disease caused by infection with *Mycobacterium bovis* and closely related members of the *M. tuberculosis* complex (MTC) and leads to increased host mortality [Barasona et al., 2016]. TB has a significant economic impact on the livestock industry due to test and slaughter schemes and movement restrictions [Gortázar et al., 2015], [Picasso-Risso et al., 2020], [Schiller et al., 2011] and within the European Union there has been funding and a long-term policy to reduce and eradicate TB in cattle [Kubik et al., 2016]. Other intervention measures include reducing indirect contacts among host species [Barasona et al., 2013], [Wilber et al., 2019], vaccinating wildlife [Díez-Delgado et al., 2018], and culling wildlife [Boadella et al., 2012], [Tanner et al., 2019a]. Nevertheless, there is an increasing prevalence of infection among cattle herds in Europe, with the EU herd prevalence at 0.4% in 2008 and at 0.9% in 2018 [EFSA and ECDC, 2019]. A key issue for the control of TB is its persistence in wildlife reservoirs from which it can spill-over to livestock [Barasona et al., 2019]. The primary reservoir species vary with location and include European badgers (*Meles meles*) in the British Isles, cervids in North America, brushtail possums (*Trichosurus vulpecula*) in New Zealand and buffalo (*Syncerus caffer*) in South Africa, among others [Fitzgerald and Kaneene, 2013], [Gortázar et al., 2015]. In mainland Europe, and in particular on the Iberian Peninsula, wild ungulates such as the Eurasian wild boar (*Sus scrofa*) and red deer (*Cervus elaphus*) act as the primary reservoir of infection [Gortázar et al., 2015], [Gortázar et al., 2012], [Naranjo et al., 2008], [Santos et al., 2020]. Due to increasing habitat suitability and decreasing hunting pressure, the density of wild ungulates has seen an unprecedented increase over the last decades [Burbaitė and Csányi, 2010], [Massei et al., 2015], [Milner et al., 2006] and this presents a challenge for TB management.

Wildlife species can harbour multiple infectious agents and this can be facilitated by the increase in density of wild ungulates. In wild boar, the spread of African swine fever (ASF) is a cause for current concern as it leads to high levels of mortality and has the potential to spill-over to domestic pigs resulting in subsequent losses in pig production [Barongo et al., 2016], [Sánchez-Vizcaíno et al., 2013]. As such, ASF is listed as a notifiable disease by the World Organisation for Animal Health (OIE) [Jurado et al., 2018], [Vergne et al., 2016]. Outbreaks are currently causing global concern; in Europe an outbreak in Georgia and the Russian Federation in 2007 had spread to Ukraine, Belarus, Estonia, Latvia, Lithuania and Poland [Depner et al., 2017] and recent outbreaks have been reported in the Czech Republic, Belgium, Slovakia and several south-east European countries [Miteva et al., 2020]. Germany, the EU's largest pork producer, is the most recently affected country [Kość and Standaert, 2020]. ASF is likely to continue to spread and therefore have a widespread global impact on domestic pig production and wild boar abundance.

The persistence of infectious disease is known to be linked to host population density with the potential for disease eradication if the host population decreases below a threshold density [Anderson and May, 1979]. This has underpinned the use of culling as a management strategy to control wildlife diseases [Barlow, 1996], [Tanner et al., 2019b], [Woodroffe, 1999]. Since ASF outbreaks typically lead to significant reductions in host

population density this is likely to have impacts for the prevalence and persistence of co-circulating pathogens, such as MTC. With the continued spread of ASF it is therefore important to examine the potential impact of an ASF outbreak on the persistence of TB.

Mathematical models have played a key role in understanding the processes that drive epidemiological dynamics in wildlife populations [Anderson and May, 1979], [Keeling and Rohani, 2008]. In this study we develop a mathematical model for a wild boar host population that can be co-infected with MTC and ASF. Given the marked effect of ASF on wild boar population density and knowing that wild boar culling can contribute to TB control, we hypothesize that ASF emergence will have a significant impact on wild boar TB. Therefore, our aim is to understand the consequences of an ASF outbreak on the prevalence and persistence of endemic TB in wild boar. To do this we incorporate the disease dynamics for ASF, detailed in O'Neill et al. [O'Neill et al., 2020], into a model of TB in wild boar [Tanner et al., 2019a]. Our model system is parameterised to be representative of the wild boar TB system in different regions in Spain. However, the findings apply in general as we examine the impact of ASF in regions of high density and high endemic TB prevalence and in regions of low density and low endemic TB prevalence. Degradation of ASF in wild boar carcasses can play a key role in the ASF epidemiological dynamics [Berg et al., 2015], [O'Neill et al., 2020], [Probst et al., 2017], and so we explore the outcome with two different rates of degradation, a high and a low rate. In particular, the high rate represents the rapid degradation of the pathogen as seen in places with high temperatures or with abundant obligate scavengers, such as vultures. We then consider how disease control measures introduced to eradicate ASF will impact the dynamics of TB. Beyond assessing the potential impact of ASF on the epidemiological dynamics of TB our findings add new perspective to the theory on the interaction and co-existence of multiple pathogens in a single host species.

2 Methodology

We intend to extend the model seen in Tanner et al. 2019 [Tanner et al., 2019a], which represents the dynamics of tuberculosis in wild boar, to include co-infection of African swine fever, as represented in O'Neill et al. 2020 [O'Neill et al., 2020]. A full model description is presented in the supplementary information. Here we describe the key infection processes for TB, in section 2.1, and for ASF, in section 2.2.

2.1 The Wild Boar Tuberculosis Model

The model of Tanner et al. 2019 [Tanner et al., 2019a] considers a wild boar host population that is split into three age-classes: piglets (P), yearlings (Y) and adults (A). The three different age classes are required as each class has distinct properties in terms of their demographic and infection dynamics. The age-classes are further split into susceptible (subscript S), infected (subscript I) and generalised (subscript G) classes to reflect the TB disease status of the population. Generalised individuals can also release free-living TB pathogen particles, with density F, into the environment. The model is given below:

(1)

Here, $N = P + Y + A$ represents the total wild boar population, where $P = PS + PI + PG$; $Y = YS + YI + YG$; $A = AS + AI + AG$ and G is the total number of generalised individuals, $G = PG + YG + AG$. Yearlings and adults give birth to susceptible piglets at rate b. This takes two values, $b = log\ 4$ and $b = log\ 7$, for scenarios representative of low and high TB prevalence regions, respectively. The total population is regulated through a crowding parameter, q, that acts on the birth rate, and is related to the carrying capacity, K, since . We choose the values, $K = 5.95$ or $K = 27$, for low and high prevalence TB regions, respectively. Maturity from piglets to yearlings and yearlings to adults occurs at rate m and piglets, yearlings and adults may die of natural causes at rate d. This set-up for the demographic dynamics has previously been used to assess wild boar TB interactions [Díez-Delgado et al., 2018], [Tanner et al., 2019b].

The model assumes infection can occur through direct frequency-dependent interactions (since wild boar tend to congregate in social groups) between susceptible and generalised individuals with transmission coefficients $\beta_{\Delta\Pi}$, $\beta_{\Delta\Psi}$ and $\beta_{\Delta A}$, for piglets, yearling and adults respectively, or through environmental contact with the free-living MTC, with transmission coefficients $\beta_{\Phi\Pi}$, $\beta_{\Phi\Psi}$ and $\beta_{\Phi A}$. Piglets and yearlings are assumed to be

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three times more susceptible to infection than adults [Martín-Hernando et al., 2007]. Infected individuals are not infectious but can progress to the generalised (infectious) class at rates ϵ_Π, ϵ_Ψ and ϵ_A, for the different age-classes, respectively. In low prevalence regions, where resources are not limited, it is assumed that $\epsilon_\Pi = \epsilon_\Psi = \epsilon_A$ [Tanner et al., 2019a]. However, for high prevalence regions, where resources (particularly water) are scarce and overall health is impaired (similar to conditions in central and southern Spain), it is assumed that piglets and yearlings progress from the infected to the generalised class at three times the rate of adults ($\epsilon_\Pi = \epsilon_\Psi = 3\epsilon_A$) [Tanner et al., 2019a]. The model assumes that free-living MTC is shed from generalised wild boar at rate λ and decays at rate μ_Φ . The level of environmental transmission is scaled through the parameter ω which increases when environmental conditions become more severe to reflect, for example, aggregation at limited water holes. Finally, it is assumed that all wild boar in the generalised class suffer disease induced mortality at rate a and that all adult and yearling classes are culled due to hunting at constant rate c .

In this study we consider two parameter sets that include a range of wild boar densities and TB prevalences found in Spain: (i) where the wild boar density is $4km^{-2}$ and TB is endemic with a prevalence of 10% , representative of regions in northern Spain, or similar, where water is not a limiting factor and no feeding takes place [Muñoz Mendoza et al., 2013] and (ii) where the wild boar density is $12km^{-2}$ and TB is endemic with a prevalence of 60% , representative of regions in central Spain, or similar, where wild boar are supplementary fed but where water resources are scarce in summer [Vicente et al., 2013]. A full description of the parameters and their values (which are taken from Tanner et al. 2019 [Tanner et al., 2019a], except where stated) is given in the supplementary information.

2.2 The Wild Boar African Swine Fever and Tuberculosis Model

We extend the wild boar TB dynamics to include co-infection with ASF following the methodology presented in O'Neill et al. 2020 [O'Neill et al., 2020] that develops a wild boar ASF model. For the ASF epidemiological dynamics we consider the following classes: S , uninfected and susceptible to infection; I , infected and able to transmit the virus; C , survivor individuals which do not transmit the virus but can revert to the infected (I) class and D , infected carcasses which can transmit the virus. The definition of survivor individuals arises from the type 1 'survivors' definition used in Ståhl et al. [Ståhl et al., 2019] where such individuals will invariably die but have the potential to excrete virus in association with the resurgence of viraemia. An overview of the ASF infection dynamics is shown schematically in Figure 1.

ASF infection is assumed to affect all age-classes of wild boar equally [O'Neill et al., 2020]. It is assumed a susceptible can become infected with ASF due to direct contact with an infected individual via frequency dependent transmission, β_Φ or due to environmental transmission through contact with an infected carcass, β_E . A proportion, ρ , of infected individuals suffer disease induced mortality at rate $\gamma = 365/5$, reflecting an average lifespan of 5 days for an individual with ASF [Gallardo et al., 2015]. A proportion, $1 - \rho$, of infected individuals can instead enter the survivor class, which does not incur disease induced mortality. Survivor individuals can revert to the infected class at rate $\kappa = 12/6$ implying that on average individuals remain survivors for 6 months [Gallardo et al., 2015]. Host individuals that die due to the infection become infected carcasses which degrade at rate μ . Further details of the wild boar ASF model can be found in O'Neill et al. 2020 [O'Neill et al., 2020] and the combined TB and ASF infection model is shown in full in the supplementary information with details of the parameter values. Note, in the TB and ASF full model we do not assume direct interference between competing pathogens. However, there will be indirect interference due to parasite induced changes in host population density.

In this study we consider ASF control measures in the form of culling and the removal of carcasses. Culling is applied at rate b_C , across all age and infection classes whilst the removal of carcasses is applied at rate r . The value b_C corresponds to a culling proportion equal to 1- e^{-bC}, per year, of the total population and the value r corresponds to an average time till the removal of a carcass of $1/r$ years.

3 The Impact of African Swine Fever on endemic Tuberculosis

We undertake model simulations for the introduction of ASF into the TB endemic system, for scenarios that

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are representative of TB infection status found in wild boar in Spain, including regions of high and low TB prevalence and scenarios that represent high and low ASF infected carcass degradation rates.

For the scenario with high TB prevalence and a high rate of ASF infected carcass degradation the introduction of ASF leads to a rapid population reduction of *85%* (Figure 2). There is a peak in ASF infection around 3 months after its introduction and in the model ASF persists at low prevalence (*0.8%*) in the long-term. The impact and persistence of ASF leads to a reduction in the host population density which in turn leads to a decrease in TB prevalence. Note, the decrease in TB prevalence is slow and in the long-term TB prevalence stabilises to a reduced level. For the scenario with high TB prevalence and a low rate of ASF infected carcass degradation the introduction of ASF leads to a near eradication of the host population, with a *99%* reduction in the host population density (Figure 3). The impact of ASF is more severe as the low rate of carcass degradation increases the potential for ASF transmission. ASF infection peaks after 2 months and persists for 2 years but the model results indicate local disease fade-out. In the long-term this allows the host population to return to the density prior to the ASF outbreak. The impact on TB is a gradual reduction in prevalence from *60%* to less than *20%* within 8 years, and then an increase in TB prevalence back to *60%* at a rate that lags behind the increase in population density.

Although ASF leads to near eradication of the population, the TB prevalence does not drop below *20%* . TB is not eradicated in the model as the component of the effective reproductive ratio that arises through the frequency dependent transmission is independent of host density and is greater than *1* (see supplementary information, section S3, for more details).

In the low TB prevalence (and lower initial host density), high carcass degradation rate scenario ASF establishes slowly and becomes endemic at low prevalence (Figure 4). This leads to a slow decrease in the host population of around *35%* which then leads to a slow decrease in TB prevalence. In the long-term TB would be eradicated as the TB infection parameters for this scenario mean that the reduction in host population density due to endemic ASF is sufficient to reduce the effective reproductive ratio for TB below *1* (see supplementary information, section S3). The low TB prevalence and low carcass degradation rate scenario (Figure 5) is similar to the equivalent high TB prevalence case with an ASF outbreak followed by rapid population crash and fade out of ASF before the population then recovers. There is a reduction in TB prevalence to low levels and a slow increase in TB prevalence following population recovery. The slow decrease and then increase in TB prevalence is due to the effective reproductive ratio for TB dropping below *1* for low host population densities and increasing above *1* as the host population returns to its density prior to the ASF outbreak. A common finding across all scenarios is that the epidemiological dynamics of ASF are rapid whereas those of TB are slow and highlights a difference between acute and chronic infections.

4 Applying ASF control

Due to the economic and population impact of ASF on farmed hosts control measures to eradicate ASF are applied following its detection. In ASF endemic regions, such as the Baltic countries, infected wild boar are recorded for several years after the initial introduction and suggest there could be poorly understood pathways that facilitate the persistence of the virus at very low densities [Miteva et al., 2020], [O'Neill et al., 2020]. ASF maintenance could also be caused by continued new introductions of the virus from adjacent affected regions and countries [Miteva et al., 2020]. Recommended measures under these epidemiological circumstances include continued active and passive disease surveillance, intense efforts of carcass detection and removal, and continuous and intensive hunting of wild boar to maintain low population densities, both to slow down the speed of infection spread and to monitor progress through active disease surveillance [Miteva et al., 2020]. We repeat the scenarios outlined in section 3 but with the inclusion of ASF control measures. We consider two control measures: the culling of the host population (with culling rate b_C) and the removal of ASF infected carcasses (with removal rate r). The measures are implemented as soon as ASF enters the population, and cease when ASF has been eradicated from the population (defined as the time when the total number of ASF infected and survivor individuals, $I + C$, is less than *0.1%* of the initial host density).

For control that focuses on carcass removal (a high carcass removal rate, $r = 200$ and low culling rate b_C

$= 0.2$) the results are similar regardless of the ASF and TB scenario. We show results for the high TB prevalence and low carcass degradation rate scenario (Figure 6). Results for other scenarios can be found in the supplementary information (Figure S1). ASF control prevents a severe ASF infectious outbreak and ASF is eradicated within 3 years. There is an approximate *40%* drop in total population and only *10%* drop in TB prevalence. This method of control eradicated ASF without a significant drop in host population density and therefore will have little impact on TB prevalence.

For the culling-based control measure (a low carcass removal rate,$r = 26$ and high culling rate $b_C = 0.7$) the outcome depends on whether the initial TB prevalence is high or low but is independent of the natural carcass degradation rate. We show results representative of a low carcass degradation rate with results representative of a high carcass degradation rate given in the supplementary information (Figure S2). For scenarios representative of a high TB prevalence region there is a rapid reduction in host population density, of approximately *95%* , and a more gradual reduction in TB prevalence (Figure 7). Once ASF is eradicated the total population recovers but the increase in prevalence of TB to its original value lags behind that of the increase in host population. For scenarios representative of a low TB prevalence region (Figure 7), there is an approximate drop of *80%* in the total population and slow decrease in TB prevalence. Following ASF eradication the host population recovers to its original density but TB prevalence increases slowly.

5 Discussion

The insight gained from our mathematical model confirmed the hypothesis that the emergence of ASF could have a significant impact on the prevalence of TB in wild boar, with a reduction in TB prevalence in response to an ASF outbreak. If ASF persists in the local host population the model predicts the long-term decline of TB prevalence in wild boar. If ASF fades-out in the local host population the model predicts a slower recovery of TB prevalence in comparison to wild boar density after an ASF epidemic. This may open a window of opportunity to apply TB management to maintain low TB prevalence. Whilst there are ongoing efforts to prevent ASF re-emergence in Spain, after its successful eradication in 1995 [Mur et al., 2012], and elsewhere across the globe, the ongoing ASF spread in Eurasia [Nielsen et al., 2019], [Jo and Gortázar, 2020] makes such an unfortunate scenario more practical.

Our model study adds new perspective to the theory on the co-existence of multiple pathogens. Classical infectious disease model studies [Anderson and May, 1979], [Bremermann and Thieme, 1989], that include multiple pathogens which exclusively infect the host and that consider density dependent infection transmission, indicate that the pathogen with the greatest reproductive ratio can out-compete other pathogen. Exclusion of competing pathogens occurs as disease-induced mortality suppresses the host density to levels where only one pathogen can persist [Bowers and Boots, 2003]. Coexistence of multiple pathogens requires co-infection or super-infection mechanisms that include trade-offs balancing the pathogens ability to transmit the infection against disease-induced mortality [Begon and Bowers, 1995], [Hochberg and Holt, 1990], [Nowak and May, 1994], [Levin and Pimentel, 1981]. Our study includes co-infection of the host population by different pathogens that infect the host through density-dependent and frequency-dependent mechanisms. In particular, the component of the pathogen reproductive ratio arising through frequency-dependent transmission does not depend on host density (see supplementary information, section S3) and we show that this transmission mechanism can promote pathogen coexistence and prevent the pathogen exclusion that arises due to disease-induced suppression of the host density.

ASF infection leads to high levels of disease-induced mortality in wild boar and an ASF outbreak can cause a rapid and significant decline in population density [O'Neill et al., 2020], [Depner et al., 2017], [Gallardo et al., 2015], [Korennoy et al., 2014], [Simulundu et al., 2017]. Therefore, an ASF outbreak in wild boar populations that support endemic TB will act in a similar manner to host population culling and can have an impact on TB prevalence and MTC infection levels [Tanner et al., 2019a], [Tanner et al., 2019b]. Our model results indicate that an ASF infection will reduce wild boar population density and lead to a decrease in the prevalence of TB. The decrease in population density leads to a rapid decrease in the number of TB infected individuals but with the decrease in TB prevalence occurring on a longer time scale. This is due to ASF infecting all TB classes equally, TB being a chronic long-lived infection compared to ASF and

due to the direct, frequency dependent transmission of TB at low wild boar density. When ASF persists in the host population a reduction in wild boar population density is seen due to the increase in population level mortality. This leads to a long-term reduction in TB prevalence and the potential for TB eradication. In some model scenarios where ASF transmission in the environment (from infected carcasses) is high, the ASF outbreak causes a severe reduction in host population density and while ASF persists in the medium term it can fade-out, in the local host population, in the long-term. This further highlights the importance of environmental transmission from infected carcasses in the persistence of ASF and the role obligate scavengers may play in reducing environmental transmission [Berg et al., 2015], [O'Neill et al., 2020], [Probst et al., 2017]. Infection fade-out is common for highly virulent, acute infections [Chenais et al., 2019], [Cross et al., 2005], [Macpherson et al., 2015], [White et al., 2014] and may occur locally for ASF but is unlikely to do so over a regional scale due to the spatial spread of infection (for example, ASF has persisted at the regional scale in Baltic countries since 2014, [Nurmoja et al., 2020]). Model results indicate that following ASF fade-out the local wild boar population density will increase and slowly return to pre-ASF infection levels. The increase in MTC infection lags behind the increase in population density and therefore the return to pre-ASF infection levels of TB is predicted to occur on a longer time scale. This should not be misinterpreted for TB control as TB will still increase in the long-term. However, the slow increasing rate of TB infection may open a window of opportunity to apply TB management and maintain low TB prevalence. This could include bio-safety measures that reduce wildlife-cattle contact rates [Barasona et al., 2013], the culling of wild boar [Boadella et al., 2012] or the introduction of TB vaccinations for wild boar [Díez-Delgado et al., 2018]. We recognise that our model only considers a single host system and in a natural system there are several species that can act as a TB reservoir [Gortázar et al., 2015], [Gortázar et al., 2012], [Naranjo et al., 2008]. Therefore, there is the potential for inter-species transmission which could accelerate the increase in TB prevalence during the wild boar population recovery phase. However, there is evidence that wild boar are the key reservoir species for TB in Spain [Naranjo et al., 2008], [Santos et al., 2020] and that a decrease in wild boar TB infection levels leads to a reduction in TB prevalence in other wildlife [Tanner et al., 2019a], [Boadella et al., 2012].

ASF infection has widespread and severe impacts on wild boar and domestic pig production [Barongo et al., 2016], [Sánchez-Vizcaíno et al., 2013] and is listed as a notifiable disease by the World Organisation of Animal Health (OIE) [Jurado et al., 2018], [Vergne et al., 2016]. Control measures to manage ASF are likely to be introduced following detection [Bellini et al., 2016], [Jurado et al., 2018] and while these would diminish the impact of an ASF outbreak they may also limit host population mortality and therefore the impact of ASF on TB control. Furthermore, resources that are used to target TB control may be redistributed to focus on ASF management and this could have unforeseen consequences for TB management. A consequence of the Foot-and-Mouth outbreak in the UK in 2001 [Vial et al., 2013] and a likely consequence of the effort to tackle COVID-19 [Gortázar and de la Fuente, 2020], is a shift in resources to control the emerging disease at the expense of a reduction in the direct efforts to control endemic disease. Therefore, any efforts to control emerging pathogens should be considered as part of a joint strategy to control the multiple pathogens that share a host.

In this study we have used a mathematical modelling framework to assess the potential impact of an ASF outbreak on the epidemiological dynamics of wild boar. We have made particular reference to the situation in Spain where wild boar are the key reservoir host for TB. However, the key findings that ASF can lead to a marked reduction in host density and hence a rapid reduction in TB infected individuals but a more gradual reduction in TB prevalence generalise beyond the system in Spain. Our results highlight the interaction and impact of co-infecting pathogens on the population and epidemiological dynamics of their host and the need to consider multiple pathogens when attempting to control a primary infectious outbreak.

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Author Contributions

X.O. and A.W. performed mathematical analysis. All authors contributed to the production of the manuscript. All authors gave final approval for publication.

Competing Interests

The authors declare no competing interests.

Data Availability Statement

Data sharing is not applicable to this article as no new data was created or analysed in this study.

Ethical Statement

The authors confirm that the ethical policies of the journal, as noted on the journal's author guidelines page, have been adhered to. No ethical approval was required as this is a research article with no original data.

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Figure 1: A visual representation of the wild boar ASF model presented in O'Neill et al. 2020 [O'Neill et al., 2020]. The nodes represent the different ASF infection classes: S , ASF susceptible,I , ASF infected, C , ASF survivor individuals and D , infected carcasses. The arrows show the possible entry and exit routes into and out of each respective class.

Figure 2: Population densities and prevalence for the model described by the equations (S1-S5), in the supplementary information, for the scenario with high carcass degradation rate and a high tuberculosis prevalence. Results are shown over a 25-year period. (A) shows total host density (blue), hosts susceptible to TB (green), hosts infected with TB (magenta), hosts with generalised TB infection (red) and the combined infected and generalised hosts (black). (B) shows the total host density (blue), hosts susceptible to ASF (green), hosts infected with TB (magenta), ASF survivor density (red) and the infected carcass density (black). (C) shows TB prevalence with infected prevalence,I_{TB}/N , (dotted); generalised prevalence,G/N , (dot-dash) and total prevalence, $(I_{TB}+ G)/N$, (solid). We used default parameter values and $K = 27$, $\beta = \lambda\nu\ 7$, $\varsigma_\epsilon = 3$, $\omega = 1$ and $d = 52$.

Figure 3: Population densities and prevalence for the model described by the equations (S1-S5), in the supplementary information, for the scenario with low carcass degradation rate and a high tuberculosis prevalence. Results are shown over a 25-year period. (A) shows total host density (blue), hosts susceptible to TB (green), hosts infected with TB (magenta), hosts with generalised TB infection (red) and the combined infected and generalised hosts (black). (B) shows the total host density (blue), hosts susceptible to ASF (green), hosts infected with TB (magenta), ASF survivor density (red) and the infected carcass density (black). (C) shows TB prevalence with infected prevalence,I_{TB}/N , (dotted); generalised prevalence,G/N , (dot-dash) and total prevalence, $(I_{TB}+ G)/N$, (solid). We used default parameter values and $K = 27$, $\beta = \lambda\nu\ 7$, $\varsigma_\epsilon = 3$, $\omega = 1$ and $d = 13$.

Figure 4: Population densities and prevalence for the model described by the equations (S1-S5), in the supplementary information, for the scenario with high carcass degradation rate and a low tuberculosis prevalence. Results are shown over a 25-year period. (A) shows total host density (blue), hosts susceptible to TB (green), hosts infected with TB (magenta), hosts with generalised TB infection (red) and the combined infected and generalised hosts (black). (B) shows the total host density (blue), hosts susceptible to ASF (green), hosts infected with TB (magenta), ASF survivor density (red) and the infected carcass density (black). (C) shows TB prevalence with infected prevalence,I_{TB}/N , (dotted); generalised prevalence,G/N , (dot-dash) and total prevalence, $(I_{TB}+ G)/N$, (solid). We used default parameter values and $K = 5.95$, $\beta = \lambda\nu\ 4$, $\varsigma_\epsilon = 1$, $\omega = 0.1$ and $d = 52$.

Figure 5: Population densities and prevalence for the model described by the equations (S1-S5), in the supplementary information, for the scenario with low carcass degradation rate and a low tuberculosis prevalence. Results are shown over a 25-year period. (A) shows total host density (blue), hosts susceptible to TB (green),

hosts infected with TB (magenta), hosts with generalised TB infection (red) and the combined infected and generalised hosts (black). (B) shows the total host density (blue), hosts susceptible to ASF (green), hosts infected with TB (magenta), ASF survivor density (red) and the infected carcass density (black). (C) shows TB prevalence with infected prevalence,I_{TB}/N , (dotted); generalised prevalence,G/N , (dot-dash) and total prevalence, $(I_{TB}+ G)/N$, (solid). We used default parameter values and $K = 5.95$, $\beta = \lambda\nu\ 4$, $\varsigma_\epsilon = 1$, $\omega = 0.1$ and $d = 13$.

Figure 6: Population densities and prevalence for the model described by the equations (S1-S5), in the supplementary information, for the scenario with low carcass degradation rate and a high tuberculosis prevalence under specific ASF control measures. Results are shown over a 25-year period. (A) shows total host density (blue), hosts susceptible to TB (green), hosts infected with TB (magenta), hosts with generalised TB infection (red) and the combined infected and generalised hosts (black). (B) shows the total host density (blue), hosts susceptible to ASF (green), hosts infected with TB (magenta), ASF survivor density (red) and the infected carcass density (black). (C) shows TB prevalence with infected prevalence, I_{TB}/N , (dotted); generalised prevalence, G/N , (dot-dash) and total prevalence,$(I_{TB} + G)/N$, (solid). Parameters are as in Figure 3, with the culling parameter $b_C = 0.2$ and carcass removal rate $r = 200$.

Figure 7: Population densities and prevalence for the model described by the equations (S1-S5), in the supplementary information, for the scenario with low carcass degradation rate and either a high tuberculosis prevalence (A-C) or a low tuberculosis prevalence (D-F), under specific ASF control measures. Results are shown over a 25-year period. (A&D) show total host density (blue), hosts susceptible to TB (green), hosts infected with TB (magenta), hosts with generalised TB

infection (red) and the combined infected and generalised hosts (black). (B&E) show the total host density (blue), hosts susceptible to ASF (green), hosts infected with TB (magenta), ASF survivor density (red) and the infected carcass density (black). (C&F) show TB prevalence with infected prevalence, I_{TB}/N , (dotted); generalised prevalence, G/N , (dot-dash) and total prevalence,$(I_{TB} + G)/N$, (solid). For A-C parameters are as in Figure 3 and for D-F parameters are as in Figure

5. For all plots, the culling parameter $b_C = 0.7$and carcass removal rate $r = 26$ are used.

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