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THE TRANSMISSION DYNAMICS OF *BRUCELLA ABORTUS* IN THE YELLOWSTONE BISON HERD

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The transmission dynamics of *Brucella abortus* in the Yellowstone bison herd.*

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Abstract

Brucella abortus has been present in the Yellowstone bison herd since its introduction by infected cattle in the early years of this century. Ironically, it is now perceived to be a threat to the local cattle industry. Determining the risk of transmission of *B.abortus* from wildlife to cattle requires a quantitative understanding of the transmission dynamics of pathogens within the host population. We have developed a mathematical epidemiological model for *B.abortus* that explicitly

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acknowledges that three different routes of transmission may operate: pseudo-vertical transmission from an infected female to a suckling calf; horizontal transmission as an STD; or "diagonal transmission" from aborted foetus to animals that contact that foetus. Comparison of the observed serology profiles with those generated by the model suggests that transmission is dominated by the diagonal pseudo-vertical routes from infected female to suckling calf. It is determined that the basic reproduction ratio of brucellosis in Yellowstone bison is only just above one, and that transmission to cattle via aborted foetuses is probably negligible.

1 Introduction

Brucellosis is a chronic disease of mammals caused by bacteria of the genera *Brucella*. Although rarely fatal the disea : causes abortion in cattle and other ungulate hosts. Milk and meat products from infected herds are not suitable for human consumption, and significant restrictions are placed on the movement of cattle from areas where outbreaks have occurred. Although there have been no recorded cases of transmission of *Brucella abortus* between wildlife and cattle in the United States, APHIS (the U.S. Department of Agriculture's Animal and Plant Health Inspection Service) regards the eradication of *B.abortus* in Yellowstone bison as a major priority (Baskin, 1998). Attempts to control brucellosis in Yellowstone have focused on removal and slaughter of animals, an approach that has been controversial and, when combined with an unusually bad winter, has reduced the largest herd of bison in the U.S. from a population of around 4000 to one of around 1200 animals. Achieving a sustainable brucellosis-free herd will require a quantitative understanding of the transmission dynamics of *B.abortus*.

Standard epidemiological methods have provided some important insights into the population dynamics of brucellosis in Yellowstone bison (Dobson & Meagher, 1996). However, the chronic nature of the disease and apparent differences in the susceptibility of male and female bison, adults and juveniles, to infection imply that a more detailed framework is required than an unstructured *SIR* model. We present a model structured on the age and sex of the herd that separates the potential routes of disease transmission. We compare the age-prevalence curves derived from the model with data from a bison cull, and are able to deduce some aspects of the epidemiology of the disease, in particular the relative importance of different routes of transmission.

2 The brucellosis model

2.1 Host population dynamics

The results of surveys of the Yellowstone bison herd over a 90 year period are presented in Fig.1. The data clearly show an exponential increase in the size of the herd since the 1960s (in the absence of bison culling), with no evidence of density-dependent population regulation. The estimated exponential coefficient of the increase is 0.106.

check

We divide the herd (size N) into N_j juvenile bison, N_f adult females and N_m adult males. The equations for the host population dynamics are therefore

$$\frac{dN_j}{dt} = b\left(N_f - \phi I_f\right) - d_j N_j - a N_j \tag{1a}$$

$$\frac{dN_f}{dt} = \frac{a}{2} N_j - d N_f \tag{1b}$$

$$\frac{dN_m}{dt} = \frac{a}{2} N_j - d N_m \tag{1c}$$

Adult female bison calve every second year (Ref?), hence we set the birth rate $b = 0.5$ year^{-1}. The juvenile and adult death rates are d and d_j year^{-1} respectively, I_f female bison have brucellosis of which a proportion ϕ abort, and bison are considered to be juveniles (on average) until a^{-1} years old. We take the age of maturity to be 2.5 years (ref), and hence $a = 0.4$.

The population structure at the 1991-92 cull showed a high proportion of juveniles in the herd (see Fig.2). Setting a minimal juvenile death rate of $d_j = 0.005$, an adult death rate of $d = 0.09$ and $\phi = 0$ the rate constants for population growth are $(0.106, -0.601, -d)$. The population would then have 21.9% juveniles. Setting $\phi = 0.1$, $I_f/N_f = 0.08$ the rate constants are $(0.105, -0.600, -d)$. Hence, our demographic parameter values are consistent with the observed population structure and growth rate, and setting epidemiological parameters to realistic non-zero values does not change the population dynamics of the herd significantly.

2.2 Disease dynamics

The population dynamics of *B.abortus* within the herd may be described by a compartmental model of the *SIR* type with age structure, leading to a set of 5 coupled differential equations. These describe the numbers of juveniles, adult females and adult males that are infectious (I_j, I_f, I_m), and the proportions of adult female and male bison that have been infected, but are no longer infectious (R_f, R_m). We assume that an infectious juvenile remains infectious until it becomes an adult. We identify three possible routes of transmission: pseudo-vertical transmission from an infected female to a suckling calf; horizontal transmission as a sexually transmitted disease (STD); or "diagonal transmission" from an aborted foetus to animals that contact that foetus (see Williams et al., 1997). Hence we obtain the equations

$$\frac{dI_j}{dt} = \beta_v b(1-\phi) I_f + \beta_{jd}\phi b S_j \frac{I_f}{N_f} - (d_j + a) I_j \quad (2a)$$

$$\frac{dI_f}{dt} = \frac{a}{2} I_j + \beta_{fd}\phi b S_f \frac{I_f}{N_f} + \beta_{fh} S_f \frac{I_m}{N_m} - (d + \gamma_f) I_f \quad (2b)$$

$$\frac{dI_m}{dt} = \frac{a}{2} I_j + \beta_{md}\phi b S_m \frac{I_f}{N_f} + \beta_{mh} S_m \frac{I_f}{N_f} - (d + \gamma_m) I_m \quad (2c)$$

$$\frac{dR_f}{dt} = \gamma_f I_f - dR_f \quad (2d)$$

$$\frac{dR_m}{dt} = \gamma_m I_m - dR_m \quad (2e)$$

As the population is growing exponentially we cannot analyse the epidemiology in terms of steady states. However, an increase in the size of the herd results in an increase in the area that it occupies, without significant change in local population density. Hence all disease transmission is frequency dependent (for a discussion see de Jong et al., 1995). Under these circumstances it is possible that although the population size is increasing the prevalence of brucellosis may remain constant. To examine this we rewrite equations (2) in terms of prevalences (setting $Z_j = I_j/N_j$ etc. and $W_f = R_f/N_f$ etc.) to obtain

$$\frac{dZ_j}{dt} = \beta_v b(1-\phi) \frac{N_f}{N_j} Z_f + \beta_{jd}\phi b X_j Z_f - b(1-\phi Z_f) \frac{N_f}{N_j} Z_j \quad (3a)$$

$$\frac{dZ_f}{dt} = \frac{a}{2}\frac{N_j}{N_f} Z_j + \beta_{fd}\phi b X_f Z_f + \beta_{fh} X_f Z_m - \left(a\frac{N_j}{N_f} + \gamma_f\right) Z_f \quad (3b)$$

$$\frac{dZ_m}{dt} = \frac{a}{2}\frac{N_j}{N_m} Z_j + \beta_{md}\phi b X_m Z_f + \beta_{mh} X_m Z_f - \left(a\frac{N_j}{N_m} + \gamma_m\right) Z_m \quad (3c)$$

$$\frac{dW_f}{dt} = \gamma_f Z_f - \frac{a}{2}\frac{N_j}{N_f} W_f \quad (3d)$$

$$\frac{dW_m}{dt} = \gamma_m Z_m - \frac{a}{2}\frac{N_j}{N_m} W_m \quad (3e)$$

where the proportions of juvenile, adult female and adult male bison that

are susceptible are

$$X_j = 1 - Z_j = \frac{S_j}{N_j} \tag{4a}$$

$$X_f = 1 - W_f - Z_f = \frac{S_f}{N_f} \tag{4b}$$

$$X_m = 1 - W_m - Z_m = \frac{S_m}{N_m} \tag{4c}$$

2.3 Pseudo-steady states

In order to have constant proportions in each epidemiological state we find a steady state solution to equations (3), recognising that the population size is still increasing (see equations 1). Setting the right-hand sides of equations $(3d)$, $(3a)$, $(3b)$, and $(3e)$ to zero, in that order, we obtain

$$W_f = \frac{2\gamma_f N_f}{aN_j} Z_f \tag{5a}$$

$$Z_j = \frac{(\beta_v b\,(1-\phi)\,N_f/N_j) + \beta_{jd}\phi b}{(b\,(1-\phi Z_f)\,N_f/N_j) + \beta_{jd}\phi b Z_f} Z_f \tag{5b}$$

$$Z_m = \frac{\left(\gamma_f + aN_j/N_f\right) Z_f - \left(\frac{a}{2}N_j/N_f\right) Z_j - \beta_{fd}\phi b X_f Z_f}{\beta_{fh} X_f} \tag{5c}$$

$$W_m = \frac{2\gamma_m N_m}{aN_j} Z_m \tag{5d}$$

Hence we have defined (Z_j, Z_m, W_f, W_m) as functions of Z_f. We now set $dZ_m/dt = 0$ in equation $(3c)$ leading to

$$Z_f = \frac{\left(\gamma_m + aN_j/N_m\right) Z_m - \left(\frac{a}{2}N_j/N_m\right) Z_j}{\beta_{md}\phi b X_m + \beta_{mh} X_m} \tag{6}$$

Equation (6) is then solved numerically, and the result substituted into equations (5) to obtain the pseudo-steady states $\left(Z_j^*, Z_f^*, Z_m^*, W_f^*, W_m^*\right)$.

2.4 Age-prevalence curves

When the disease is in a pseudo-steady state, the proportion of new-born bison that are infected with brucellosis is

$$z_j(0) = \frac{\beta_v (1-\phi) Z_f^*}{1 - \phi Z_f^*} \tag{7}$$

Hence we use $z_j(\tau)$ for the prevalence of brucellosis in bison at age τ, and for $\tau \in (0, 1/a)$

$$\frac{dz_j}{d\tau} = \beta_{jd} \phi b Z_f^* (1 - z_j) \tag{8}$$

Making the approximation that all bison become adults at age $1/a$, we can write $z_j(1/a) = z_f(1/a) = z_m(1/a)$ and obtain the age-prevalence curves for the adult population from

$$\begin{aligned}
\frac{dz_f}{d\tau} &= \left(\beta_{fd}\phi b Z_f^* + \beta_{fh} Z_m^*\right)(1 - z_f - w_f) - \gamma_f z_f && \text{(9a)}\\
\frac{dz_m}{d\tau} &= \left(\beta_{md}\phi b Z_f^* + \beta_{mh} Z_f^*\right)(1 - z_m - w_m) - \gamma_m z_m && \text{(9b)}\\
\frac{dw_f}{d\tau} &= \gamma_f z_f && \text{(9c)}\\
\frac{dw_m}{d\tau} &= \gamma_m z_m && \text{(9d)}
\end{aligned}$$

The explicit solutions to equations (9) are

$$\begin{aligned}
z_j(\tau) &= 1 + (\gamma_1 - 1) e^{-\gamma_2 \tau} && \text{(10a)}\\
z_f(\tau) &= e^{\gamma_3/a} \frac{\gamma_3 (1-K)}{\gamma_5 - \gamma_3} e^{-\gamma_3 \tau} + e^{\gamma_5/a} \frac{\gamma_5 K - \gamma_3}{\gamma_5 - \gamma_3} e^{-\gamma_5 \tau} && \text{(10b)}\\
z_m(\tau) &= e^{\gamma_4/a} \frac{\gamma_4 (1-K)}{\gamma_6 - \gamma_4} e^{-\gamma_4 \tau} + e^{\gamma_6/a} \frac{\gamma_6 K - \gamma_4}{\gamma_6 - \gamma_4} e^{-\gamma_6 \tau} && \text{(10c)}\\
w_f(\tau) &= 1 - e^{\gamma_3/a} \frac{\gamma_5 (1-K)}{\gamma_5 - \gamma_3} e^{-\gamma_3 \tau} - e^{\gamma_5/a} \frac{\gamma_5 K - \gamma_3}{\gamma_5 - \gamma_3} e^{-\gamma_5 \tau} && \text{(10d)}\\
w_m(\tau) &= 1 - e^{\gamma_4/a} \frac{\gamma_6 (1-K)}{\gamma_6 - \gamma_4} e^{-\gamma_4 \tau} - e^{\gamma_6/a} \frac{\gamma_6 K - \gamma_4}{\gamma_6 - \gamma_4} e^{-\gamma_6 \tau} && \text{(10e)}
\end{aligned}$$

where

$$\begin{aligned} \gamma_1 &= \frac{\beta_v (1-\phi) Z_f^*}{1-\phi Z_f^*} & \gamma_2 &= \beta_{jd} \phi b Z_f^* \\ \gamma_3 &= \beta_{fd} \phi b Z_f^* + \beta_{fh} Z_m^* & \gamma_4 &= \beta_{md} \phi b Z_f^* + \beta_{mh} Z_f^* \\ \gamma_5 &= \gamma_f & \gamma_6 &= \gamma_m \end{aligned} \tag{11}$$

and

$$K = z_j(1/a) = 1 + (\gamma_1 - 1)\, e^{-\gamma_2/a} \tag{12}$$

Note that

$$\begin{aligned} x_f(\tau) &= 1 - z_f(\tau) - w_f(\tau) \\ &= e^{\gamma_3/a} (1-K)\, e^{-\gamma_3 \tau} \end{aligned} \tag{13}$$

with a similar expression for $x_f(\tau)$.

3 Transmission dynamics

3.1 Estimation of the transmission coefficients

Data from the 1991-92 bison cull in Yellowstone Park are presented in Fig.2. The fitted curves that are also shown were obtained using by minimising the sum of the squares of the deviations of equations (10) over all possible values of γ_i, $i = 1 \ldots 6$. For this purpose we substituted two (out of fourteen) culture positive females at age four for the anomalous result zero (see Dobson & Meagher, 1996) to reduce the effect of this outlier. We assumed that "culture positive" equated to "infectious" and sero-positive equated to "infectious plus removed", hence the curves presented in Figs.2a,b show $w_f + z_f$ and $w_m + z_m$, and those in Figs.2c,d show z_f and z_m respectively. All numerical calculations were performed using Mathcad 8 Professional (Mathsoft Inc., 1997). The results are discussed below.

3.1.1 Transmission to juvenile bison

We obtained $\gamma_1 = 0.071$ and $\gamma_2 = 0.124$. Recall that β_v is a probability, and cannot exceed one. From equations (11) we then have

$$\phi < \frac{Z_f^* - \gamma_1}{Z_f^*(1-\gamma_1)}$$

and hence $Z_f^* > \gamma_1$. The data collected in the 1991-92 cull (Fig.2) suggest a lower prevalence, so we assume from here a value $Z_f^* = 0.08$, which leads to a maximum value $\phi = 0.117$ for which $\beta_v = 1$ and $\beta_{jd} = 26.4$.

3.1.2 Transmission to adult bison

We obtained $\gamma_3 = 0.033$ and $\gamma_4 = 0.091$. Equations (11) define relationships between β_{fd} and β_{fh} (similarly β_{md} and β_{mh}) of the form

$$\frac{\beta_{fd}}{\beta_{fdmax}} + \frac{\beta_{fh}}{\beta_{fhmax}} = 1 \tag{14}$$

where $\beta_{fdmax} = \gamma_3/\left(\phi b Z_f^*\right) = 7.08$, $\beta_{fhmax} = \gamma_3/Z_m^* = 0.24$, $\beta_{mdmax} = \gamma_4/\left(\phi b Z_f^*\right) = 19.5$ and $\beta_{mhmax} = \gamma_4/Z_f^* = 1.14$. Hence we have upper bounds for the transmission coefficients.

3.1.3 The period of infectiousness

We obtained $\gamma_f = \gamma_5 = 0.98$ and $\gamma_m = \gamma_6 = 0.60$, suggesting that female and male bison remain infectious for approximately one year and twenty months respectively.

3.2 The basic reproduction ratio

An expression for the basic reproduction ratio, R_0, may be derived using the next-generation matrix (Diekmann et al., 1990). Entries in this matrix are

the number of secondary cases due to a typical primary case introduced into a fully susceptible population, during its period of infectiousness. We then have $R_0 = \rho(M_0)$, where ρ signifies spectral radius. The matrix is defined by

$$M_0 = \begin{pmatrix} 0 & (F \to J) & 0 \\ (J \to F) & (F \to F) & (M \to F) \\ (J \to M) & (F \to M) & 0 \end{pmatrix} \tag{15}$$

and for example $(F \to J)$ signifies the number of secondary cases in juveniles due to a primary case in females. The zero entries occur because juveniles are only infected by females, and males cannot infect males. If the population size were constant we would have

$$\begin{aligned} (F \to J) &= \frac{\beta_v b(1-\phi) + \beta_{jd}\phi b N_j/N_f}{d+\gamma_f} & (J \to F) &= \frac{a/2}{d_j + a} \\ (F \to F) &= \frac{\beta_{fd}\phi b}{d+\gamma_f} & (M \to F) &= \frac{\beta_{fh}}{d+\gamma_m} \\ (J \to M) &= \frac{a/2}{d_j + a} & (F \to M) &= \frac{\beta_{md}\phi b + \beta_{mh}}{d+\gamma_f} \end{aligned} \tag{16}$$

Using the parameter values derived above we have

$$M_0 = \begin{pmatrix} 0 & 1.23 & 0 \\ 0.494 & (0, 0.389) & (0.345, 0) \\ 0.494 & 1.07 & 0 \end{pmatrix}$$

where two numbers in parentheses signify the range of values as β_{fd} goes from zero to β_{fdmax}.

In our case the population size is not constant but increasing. We define the quantity R_p to be the spectral radius of the matrix M_p, similar in structure to matrix M_0 in equation (15), but with the entries equal to the increase in prevalence among one population group (juvenile, female or male)

due to a unit increase in prevalence among another group, in a susceptible population. Hence the entries in M_p are

$$
\begin{aligned}
(F \to J) &= \frac{(\beta_v b\,(1-\phi)\,N_f/N_j) + \beta_{jd}\phi b}{(aN_j/N_f) + \gamma_f} & (J \to F) &= \frac{\frac{a}{2}N_j/N_f}{bN_f/N_j} \\
(F \to F) &= \frac{\beta_{fd}\phi b}{(aN_j/N_f) + \gamma_f} & (M \to F) &= \frac{\beta_{fh}}{(aN_j/N_m) + \gamma_m} \\
(J \to M) &= \frac{\frac{a}{2}N_j/N_m}{bN_f/N_j} & (F \to M) &= \frac{\beta_{md}\phi b + \beta_{mh}}{(aN_j/N_f) + \gamma_f}
\end{aligned}
\tag{17}
$$

With our parameter values we have

$$
M_p = \begin{pmatrix} 0 & 1.95 & 0 \\ 0.125 & (0, 0.346) & (0.289, 0) \\ 0.125 & 0.743 & 0 \end{pmatrix}
$$

Note that the components $(F \to M)$ of matrices M_0 and M_p are proportional to γ_4, hence R_0 and R_p depend only on the estimate of γ_4 and we have no information on the relative contribution of diagonal (β_{md}) and horizontal (β_{mh}) routes to the total transmission to males.

Sensitivity analysis?

3.3 The washout phenomenon

In Fig.3 we present values of R_0 and R_p calculated for transmission coefficients as specified above, and values of β_{fd} in the range from zero to β_{fdmax}. It can be seen from the figure that over most of the feasible range of transmission coefficients R_0 is close to one, and for most parameter values just above one (range $1.08 - 0.997$), whereas R_p is less one (range $0.744 - 0.697$). This implies that if the herd were to remain at a constant size then brucellosis would remain endemic, but that the population is increasing at a rate that precludes a constant prevalence. Hence we have a "washout" situation, with the number of diseased animals increasing but the prevalence of the disease

reducing. This type of phenomenon has been discussed previously in relation to macroparasites of wild animals (Roberts & Dobson, 1995).

3.4 Routes of transmission

In Fig.4 the different routes of transmission are illustrated, with their relative magnitudes. The magnitudes are presented as the expected numbers of secondary cases due to a single primary case in a fully susceptible population. Where the data are unable to distinguish between routes, ranges of values are given. It can be seen that female to juvenile transmission is most important, being approximately one third (pseudo)vertical and two thirds diagonal. Female to male transmission is next in importance, but the proportion of transmission that is horizontal or diagonal cannot be distinguished from the data. Female to female (diagonal) and male to female (horizontal) routes are relatively unimportant, and if transmission to male bison is predominantly via aborted foetuses they can be regarded as dead-end hosts that play no further part in transmission.

4 Discussion

It is clear from the data presented in Fig. 1 that the size of the Yellowstone Park bison herd is increasing. However, an increase in herd size does not imply an increase in local population density or inter-animal contacts (see de Jong et al., 1995, and Gueron & Levin, 1993, for similar behaviour in wildebeest). It could be argued that making the assumption of frequency-dependent disease transmission prejudges the results where they relate to the effectiveness of a culling programme, but this turns out to be an over-simplistic view. The data presented by Dobson & Meagher (1996, Fig.4)

add in impact of culling on R_0

vaccination

show little evidence for an increase in prevalence with herd size, and would support the idea of a pseudo-steady state. Our results ($R_p < 1$) suggest that such a situation would not persist. Of course, a constant prevalence with an increasing herd size would still imply an increase in the numbers of bison with disease.

The data that we have used to estimate our parameters are inadequate, and therefore the parameter estimates must be treated with caution even if they do seem to be approximately correct. The sample of bison was not random, the sample size was small, and prevalence in females appears to be too low to be consistent with other observations. The result $R_p < 1$ implies that a pseudo-steady state is not sustainable, but the curves were fitted to the data under the assumption that prevalences had been constant over the lifetimes of the existing herd members. In addition, the assumption that culture positive animals are infectious, and those sero-positive but not culture-positive are "removed", requires further examination. Despite these difficulties the data presented in Fig.2 and the fitted curves show remarkably good agreement and lead to a possible description of the transmission dynamics of the disease.

A striking feature of the data is the high prevalence of brucellosis in juvenile bison. Vertical transmission is indistinguishable from transmission via lactation in the model, but requires that the mother does not abort. It has been observed that abortion is uncommon among wild bison (Meyer & Meagher, 1997), giving us confidence in our estimate of ϕ. However, a low abortion rate implies that the route via environmental contamination (diagonal) must be efficient as the prevalence in juveniles is too high to be explained by vertical transmission alone. The data do not provide enough information to distinguish the relative importance of "diagonal" (environmental contamination) and horizontal (sexual) transmission to adult male bison. The horizontal

route is the only way by which an infected male can transmit to another bison, so if this were not important the male could be regarded as a"dead end host".

The implications that these results have for the control or eradication of brucellosis from the Yellowstone herd ~~are not immediately apparent~~. The results suggest that the prevalence of disease in the herd may be reducing with time ($R_p < 1$), but as the herd size is increasing and $R_0 > 1$ the number of infected animals is increasing. However, the results also suggest that R_0 is very close to one, so almost any control measure should succeed in eradicating brucellosis over time. As all transmission is frequency-dependent, reducing the herd size will not on its own reduce prevalence. ~~Once again, though~~, it will reduce the number of infected bison, and ~~if done by culling will reduce~~ the time during which a positive animal is infectious, ~~and hence R_0~~. ~~Similarly~~, it would only be necessary to vaccinate a small proportion of bison to reduce R_0 below one.

Although the results of this study suggest that diagonal transmission from aborted foetuses is important, the main routes of transmission are from adult female to juvenile bison. The proportion of infected females that abort appears to be about 10%. Adult males take little part in disease transmission, and may even be dead-end hosts. Hence reducing transmission to juveniles by vaccination could be a more effective means of control than culling adult animals.

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2. Proportions of male and female bison that were sero-positive (a,b) and culture-positive (c,d) in Yellowstone park in the 1991-92 cull, with standard errors. The continuous lines were obtained by fitting equations (10) to the data.

3. Values of R_0 and R_p calculated from equations (15) with $p = \beta_{fd}/\beta_{fdmax}$ ranging from 0 to 1 and β_{fh} determined by equation (14).

4. Routes of transmission of brucellosis between bison. The numbers on arrows are the expected numbers of secondary cases due to a single primary case in a fully susceptible population. Where the data are unable to distinguish between routes, ranges of values are given.

Bison in Yellowstone

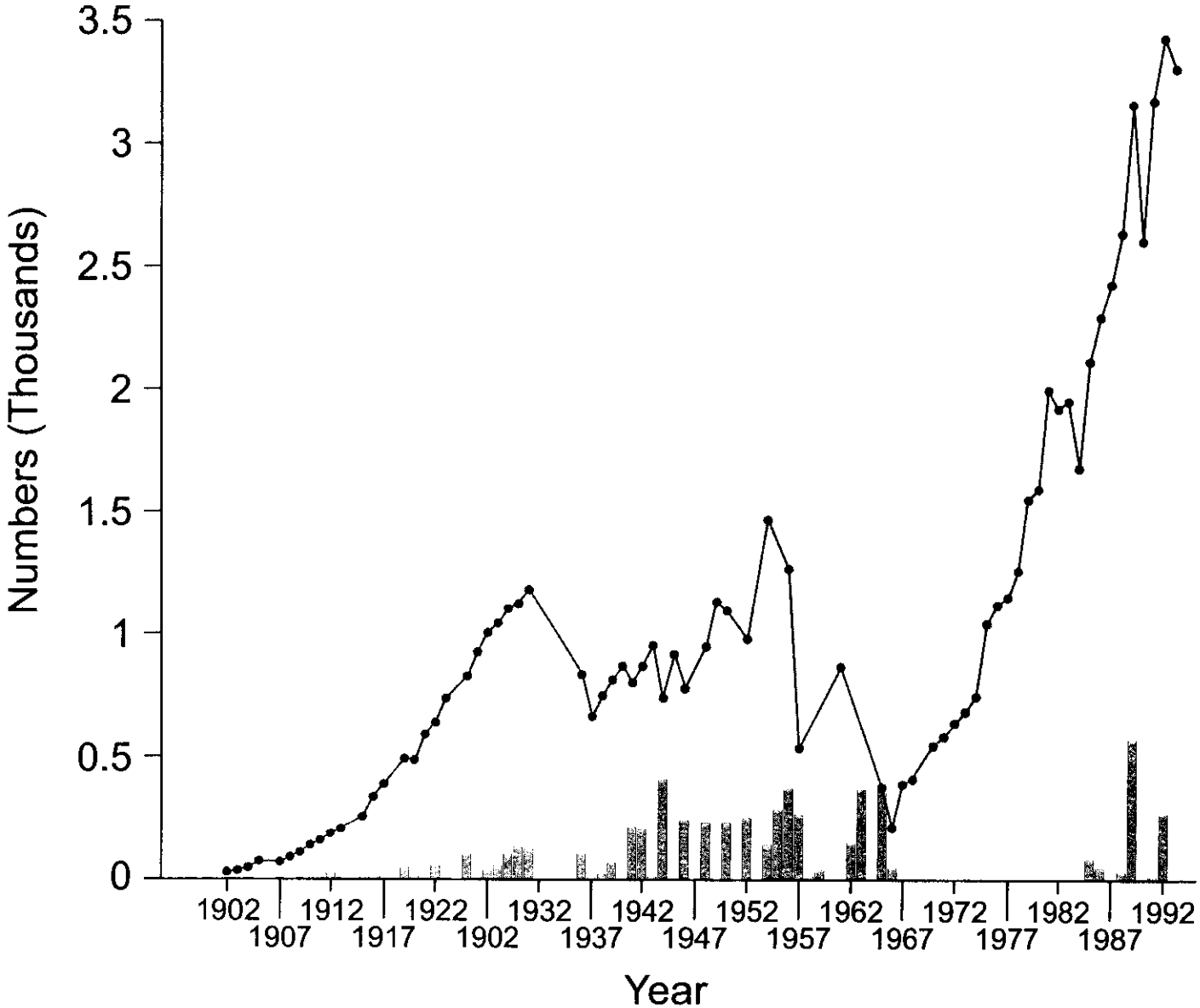
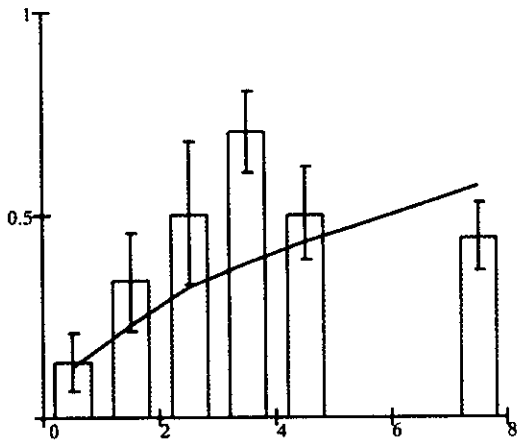
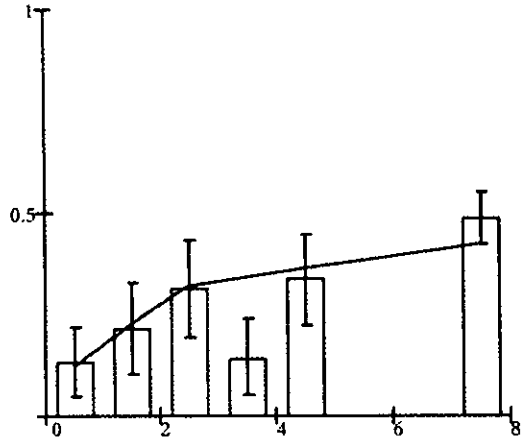


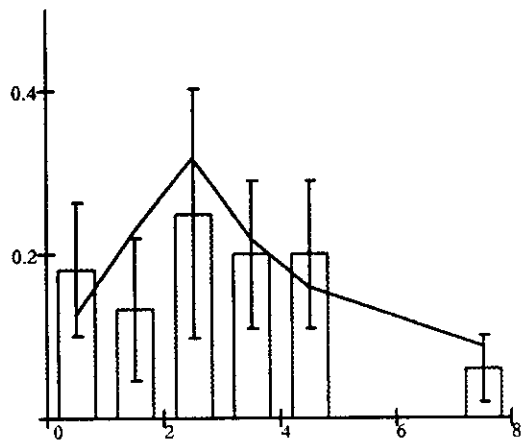
Fig. 1



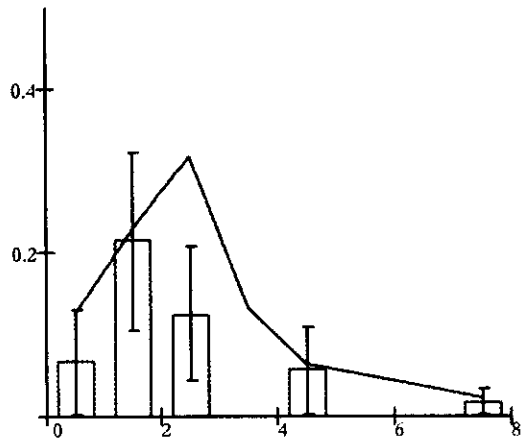
Male- Sero



Female- Sero



Male- Culture



Female- Culture

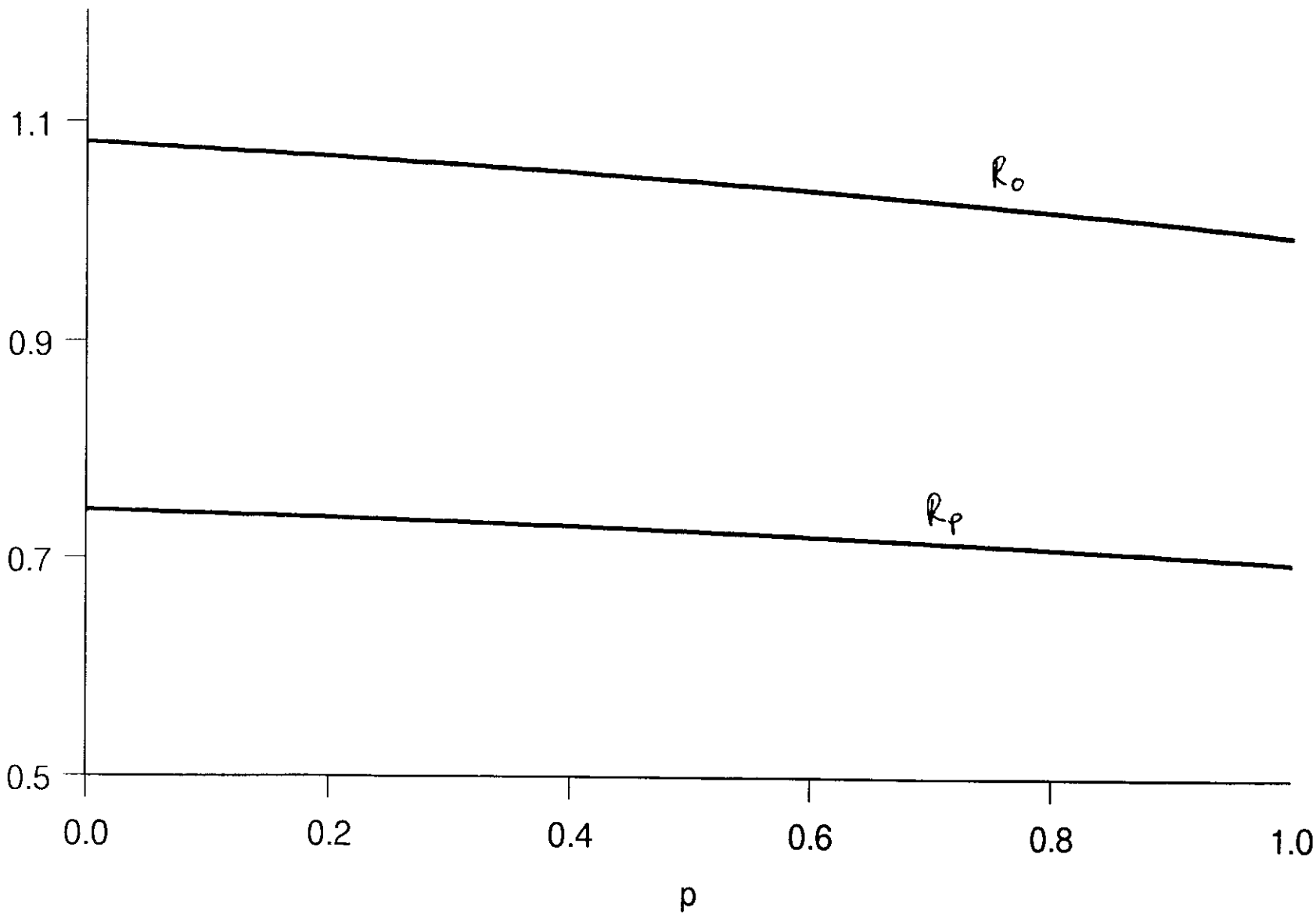


Fig 3

Fig-4

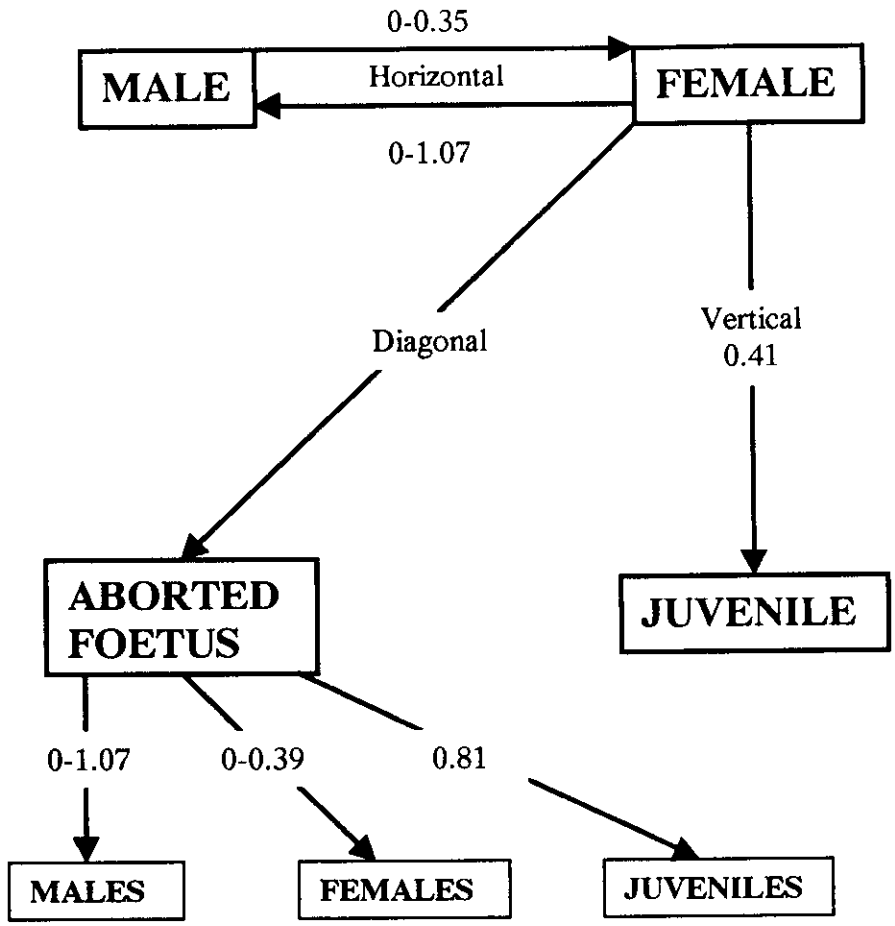


Figure 4. Routes of transmission of brucellosis between bison. The numbers on arrows are the expected numbers of secondary cases due to a single primary case in a fully susceptible population. Where the data are unable to distinguish between routes, ranges of values are given.