Decoys and dilution: the impact of incompetent hosts on prevalence of Chagas disease

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Abstract

Biodiversity is commonly believed to reduce risk of vector-borne zoonoses. This study focuses on the effect of biodiversity, specifically on the effect of the decoy process (additional hosts distracting vectors from their focal host), on reducing infections of vector-borne diseases in humans. Here, we consider the specific case of Chagas disease and use mathematical population models to observe the impact on human infection of the proximity of chickens, which are incompetent hosts for the parasite but serve as a preferred food source for vectors. We consider three cases as the distance between the two host populations varies: short (when farmers bring chickens inside the home to protect them from predators), intermediate (close enough for vectors with one host to detect the presence of the other host type), and far (separate enclosed buildings such as a home and hen-house). Our analysis shows that the presence of chickens reduces parasite prevalence in humans only at an intermediate distance and under the condition that the vector birth rate associated with chickens falls below a threshold value, which is relative to the vector birth rate associated with humans and inversely proportional to the infection rate among humans.

1 Introduction

Biodiversity is commonly considered a means for reduction of vector-borne zoonoses risk though it is not always true [1], [2]. Species diversity consists of two elements - species richness: number of species, and species evenness: proportional representation by each species. Adding any host to a vector-host system can reduce or can increase the disease risk. The reduction in disease risk due to the diversity in species is known as the dilution effect. The strength of dilution effect in a system depends not simply on the measures of species richness [3], it also depends on the abundance of dilution hosts relative to focal hosts [4]. The opposite effect is known as the rescue effect when the disease risk is increased. The determination of type of effect is governed by a couple of factors where the competency of the added host is one of the most important ones.

Based on the competency of additional host(s), the effect of distraction of vectors from their suitable host(s) can be broadly divided into two cases – *decoy effect* and *alternative or incompetent hosts' effect*. Decoy effect involves adding any incompetent (incapable of transmitting the disease) host whereas alternative hosts are capable of transmitting pathogens, but not as much as the focal host. The use of non-human decoys (e.g. livestock) to divert feeding mosquitoes away from humans may reduce vector-borne infections in the short term, but the increase in successful blood meals has the potential to cause long-term increases in mosquito populations and thereby increase the risk of subsequent human exposure [1], [2]. In the last decade, many studies have investigated how biodiversity can help to reduce the incidence of infections of vector-borne zoonoses. Results from many of those studies indicate that it is more difficult than previously thought to predict the effect of biodiversity loss on the spread of vector-borne disease.

In 2010, Johnson and Thieltges showed that the strength of dilution effects depends on the relative abundance of dilution hosts relative to focal hosts [4]. Two years later, in 2012, Ostfeld and Keesing

suggested that increases in species richness will not always decrease disease risk; indeed, in some cases diversity will cause an increase in infection risk [5]. In 2014, Miller and Huppert (2014) tried to see the effect of host diversity on the prevalence of disease infections [6]. Their study showed the basic reproduction number, R_0, is not necessarily monotonic as a function of species diversity. Thus, the richness in host population can amplify or can dilute disease prevalence depending on vectors' preference of host. These works challenge the universally established idea that biodiversity always helps to reduce the disease risk. So, the challenge lies in identifying when and for what types of host–parasite interactions we are likely to find evidence of a negative relationship between diversity and disease.

This study shifts the context from sylvatic to domestic where we study the case of Chagas disease, also known as American trypanosomiasis. This is a potentially life-threatening illness caused by the protozoan parasite, *Trypanosoma cruzi* (*T. cruzi*). It is found mainly in 21 Latin American countries, where it is mostly vector-borne. The vector involved in the transmission of the parasite to humans is a triatomine bug, also known as a 'kissing bug'. An estimated 8 million people are infected worldwide, mostly in Latin America. It is estimated that over 10,000 people die every year from clinical manifestations of Chagas disease, and more than 25 million people risk acquiring the disease [7]. Cases of Chagas disease have also been noted in the southern United States [8]. According to the World Health Organization (WHO), vector control remains the most useful method to prevent Chagas' infection [7].

Domestic animals play an important role in the domiciliary transmission of *T. cruzi* [9]. In 1998, Gürtler et al. investigated the influence of humans and domestic animals on household prevalence of *T. cruzi* in vector populations. Their result shows the indoor presence of chickens reduces the proportion of infected vectors, but increases the number of infected vectors in households [9]. However, they didn't investigate anything related to the impact of presence of chickens on the prevalence of human infections. In 2007, Gürtler et al. studied the role of domestic cats and dogs in *T. cruzi* infection [10]. In that process, they performed an entomological and sero-parasitological survey in two rural villages in Argentina. Both cats and dogs are found as epidemiologically important sources of infection for bugs and householders where dogs are nearly three times more than cats. Researchers believe the preventive management of domestic animals is an essential approach to the control of Chagas disease [9]. As a consequence of this belief, a community-based intervention was developed in 2014, based on domestic animal management by De Urioste-Stone et al. and implemented in two cities in Guatemala [11]. This community intervention promoted chicken management as one of the means for reduction of Chagas disease infections.

So, this study aims to identify conditions, if any, under which the presence of one common domestic animal–chickens–can reduce the vector-human interaction and eventually decrease human disease risk for Chagas. Here chickens are the additional host, which is completely unsuitable for the parasite. So, this work adds to research on species richness, specifically on the presence of an additional host. In this study, we investigate whether this inclusion of an incompetent host (decoy) dilutes or strengthens the force of infection. Chagas disease transmission occurs primarily in rural homes in Latin America. Studies have shown that the practice, common in countries like Argentina, of bringing chickens (brooding hens) into the home for protection of eggs and chicks against predators and then leaving them outside once grown, affects domestic vector populations [12].

Usually, the presence of incompetent hosts reduces the number of encounters between the vectors and the focal host. Eventually it leads us to the perception that this reduces the disease risk. However, some earlier works, where chickens are considered to be in bedroom areas, already proved this perception wrong [9]. The practice among rural areas shows that the residence of chickens changes with time. Thus, the distance between chickens and humans is variable, rather than fixed. This fact motivates us studying the impact of the presence of chickens at varying distances from humans. In our analysis, we consider three different cases depending on the proximity of two hosts, humans and chickens. To analyze these cases, we develop models for transmission separately for each case using dynamical systems.

2 Model Development

This work considers three different cases regarding the distance of the incompetent host (chickens) from the focal host (humans): (1) far distance case, (2) intermediate distance case, and (3) short distance

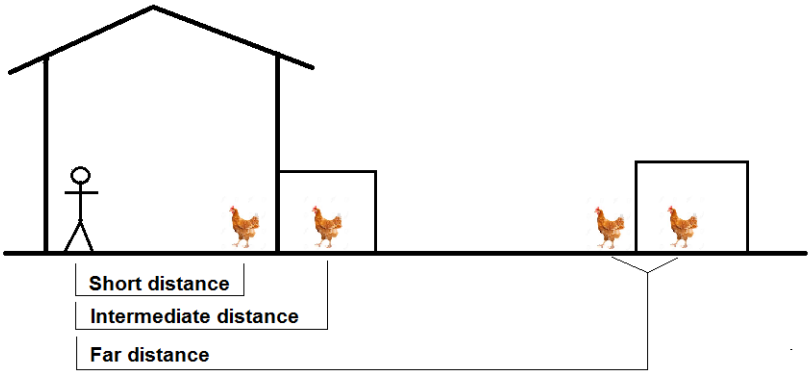


Figure 1: Portrayal of all the three cases

case. These cases are determined by the places where chickens are kept by the villagers. Most of the year, villagers keep their chickens either in a place separated from the houses or in some part of their houses. We consider the first of these two scenarios the 'far distance case' while we consider the other the 'intermediate distance case'. However, we consider the scenario 'short distance case' when chickens are brought indoors or very close to indoors to ensure their safety at a very young age.

Here, we are interested in studying the mean-field results rather than the range of possible variations, just to see whether the force of infection is strengthened or weakened by the presence of chickens. So, we are not using a stochastic model even though our population is small, rather we are using deterministic model.

In order to focus on the effects of the presence of incompetent hosts, we model only two host populations: primary and incompetent. The presence of other competent domestic hosts such as dogs can be incorporated by converting to a transmission-equivalent number of humans using the vectors' known feeding preferences.

To begin with, we consider the case when chickens sleep in nests separated from the house, either a free-standing hen-house or part of barn or other building (case of far distance). So, whenever bugs start to leave humans for inadequate availability of meals, they can easily and quickly find chickens as a source of their meals. However, here the vectors are unable to anticipate the presence of chickens while they are with humans.

A general compartmental model is used for describing the above mentioned idea mathematically. Here, the two hosts are humans (H_1) and chickens (H_2). Usually, some vectors are associated with humans and others are associated with chickens. However, no infections occur for the vectors (S_{v2}) who bite chickens since chickens are incompetent hosts. The per capita migration rates are independent of hosts' population density as vectors can not anticipate the presence of hosts due to the distance. Here, we assume the probability of vertical transmission for H_1 is p and all host demographics are at equilibrium. This is a special case (setting all the parameters related to strain I as zero) of the host switching model of [13]. All these ideas are depicted in Figure 2 and described by the system (1).

We next consider the scenario when chickens are kept a little bit closer to houses (case of intermediate distance). In this case, chickens live in a hen-house connected to the house, or in a different part of the house than the humans. Here, the proximity allows bugs staying with one host to sense the presence of other hosts and so vectors switch between hosts (humans and chickens) whenever they need. Certainly, the migration rates for vectors between hosts are determined by the availability of blood-meal sources. So, this migration between hosts is dependent on the target host's density. The model in this case is similar to the previous one, except the migration rates. The per capita migration rates are $m_{12}H_2$

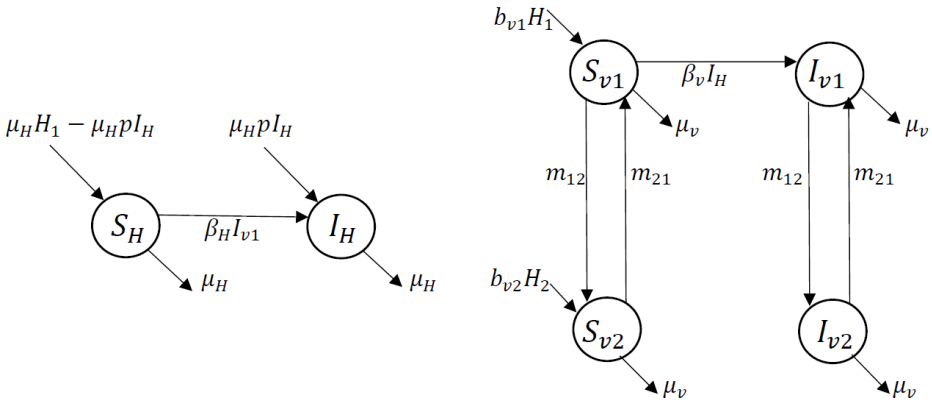


Figure 2: Flow diagram for 'far distance', system (1), where movements of vectors are independent of hosts' density.

for humans to chickens and $m_{21}H_1$ from chickens to humans. This case is visualized in Figure 3 and described by the system (2).

$$
\begin{aligned}
\frac{dS_H}{dt} &= \mu_H H_1 - \mu_H p I_H - \beta_H I_{v1} S_H - \mu_H S_H \\
\frac{dI_H}{dt} &= \mu_H p I_H + \beta_H I_{v1} S_H - \mu_H I_H \\
\frac{dS_{v1}}{dt} &= b_{v1} H_1 - \beta_v I_H S_{v1} - \mu_v S_{v1} - m_{12} S_{v1} + m_{21} S_{v2} \\
\frac{dI_{v1}}{dt} &= \beta_v I_H S_{v1} - \mu_v I_{v1} - m_{12} I_{v1} + m_{21} I_{v2} \\
\frac{dS_{v2}}{dt} &= b_{v2} H_2 - \mu_v S_{v2} - m_{21} S_{v2} + m_{12} S_{v1} \\
\frac{dI_{v2}}{dt} &= m_{12} I_{v1} - m_{21} I_{v2} - \mu_v I_{v2}
\end{aligned}
\tag{1}
$$

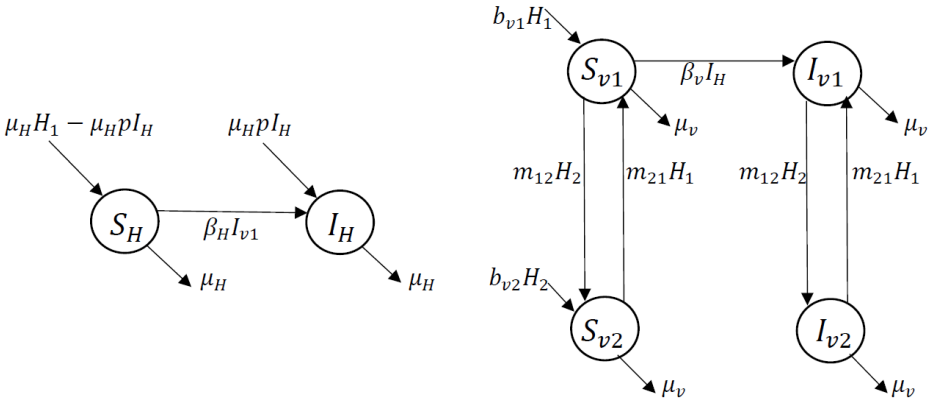


Figure 3: Flow diagram for 'intermediate distance', system (2), where movements of vectors are host density dependent.

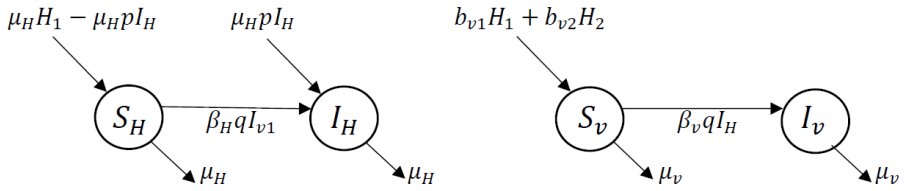


Figure 4: Flow diagram for 'short distance', system (3), where vectors don't need to migrate.

$$
\begin{aligned}
\frac{dS_H}{dt} &= \mu_H H_1 - \mu_H p I_H - \beta_H I_{v1} S_H - \mu_H S_H \\
\frac{dI_H}{dt} &= \mu_H p I_H + \beta_H I_{v1} S_H - \mu_H I_H \\
\frac{dS_{v1}}{dt} &= b_{v1} H_1 - \beta_v I_H S_{v1} - \mu_v S_{v1} - m_{12} H_2 S_{v1} + m_{21} H_1 S_{v2} \\
\frac{dI_{v1}}{dt} &= \beta_v I_H S_{v1} - \mu_v I_{v1} - m_{12} H_2 I_{v1} + m_{21} H_1 I_{v2} \\
\frac{dS_{v2}}{dt} &= b_{v2} H_2 - \mu_v S_{v2} - m_{21} H_1 S_{v2} + m_{12} H_2 S_{v1} \\
\frac{dI_{v2}}{dt} &= m_{12} H_2 I_{v1} - m_{21} H_1 I_{v2} - \mu_v I_{v2}
\end{aligned}
\tag{2}
$$

In the last case, chickens are brought so close to humans that vectors do not need to migrate to collect their meals (case of short distance). Now, vectors can bite and take blood meals from whomsoever they want. It is not anymore a host switching case, rather host sharing. So, all the vectors are sharing both of the host populations. Here, we assume that vectors bite humans a proportion q of the time. This case is a special case of host sharing model of [13] where all the parameters related to strain I set as zero. This model is portrayed in Figure 4 and represented by the system (3).

$$
\begin{aligned}
\frac{dS_H}{dt} &= \mu_H H_1 - \beta_H q I_{v1} S_H - \mu_H p I_H - \mu_H S_H \\
\frac{dI_H}{dt} &= \beta_H q I_v S_H + \mu_H p I_H - \mu_H I_H \\
\frac{dS_v}{dt} &= b_{v1} H_1 + b_{v2} H_2 - \beta_v q I_H S_v - \mu_v S_v \\
\frac{dI_v}{dt} &= \beta_v I_H S_v - \mu_v I_v
\end{aligned}
\tag{3}
$$

Table 1 summarize the variables for all of our models.

Table 1: Model variables with definition

Variable	Definition
S_H	Susceptible humans(focal host)
I_H	Infected humans
S_{v1}	Susceptible vectors associated with humans
I_{v1}	Infected vectors associated with humans
S_{v2}	Susceptible vectors associated with chickens
I_{v2}	Infected vectors associated with chickens

3 Parameter estimation

While estimating parameters, we try to take the values from the same geographical context (Argentina) to make our analysis more appropriate. Some of these parameter estimates are very rough, and we include them here primarily in order to generate illustrative qualitative trends. This study considers *Triatoma infestans* as the vector since this is the most common vector of *T. cruzi* in South America, including Argentina [14], [15], [16].

Table 2: Estimation of average lifespan for *Triatoma infestans* while feeding only on humans and chickens (base data are taken from [14])

Feeding pattern	Stage	Duration	Lifespan by gender	Lifespan by host's	Mean lifespan
Fed on humans	Egg to Nymph V	29.2 wks	45.5 wks	46.05 wks	41.2 wks ($\frac{41.2}{52}$ year)
	Adult as male	16.3 wks			
	Egg to Nymph V	29.2 wks	46.6 wks		
	Adult as female	17.4 wks			
Fed on chickens	Egg to Nymph V	18.9 wks	34.0 wks	36.35 wks	
	Adult as male	15.1 wks			
	Egg to Nymph V	18.9 wks	38.7 wks		
	Adult as female	19.8 wks			

During our careful literature review, we do not find any documented data for infection rates for humans and for vectors (β_H and β_v respectively). To estimate these values we use the method from [17] which gives the following formulas for our case

$$\beta_H = \frac{\mu_H(1-p)y_H}{(1-y_H)I_v},\ \beta_v = \frac{\mu_v y_v}{(1-y_v)I_H}$$

where y_H and y_v represent the prevalence of the disease in humans and chickens respectively. We take 27.81% (y_H) for humans [18] and 4.1% (y_v) for vectors [19], and multiply the household size and the number of bugs in a house by these prevalence values to find the value of I_H and I_v. In our literature review, we find the value 0.09 (documented as 9%) [20] for probability (proportion) of vertical transmission (p). For the human death rate, (μ_H) we take the reciprocal of their average lifespan and get $\frac{1}{77.5}$/year [21]. However, we do not get any direct documented data for vectors' death rate (μ_v). So, we use different data from the study done in 2015 by Medone et al. [14] and do our own estimation to find average lifespan for *Triatoma infestans* [Table 2] and finally take the reciprocal to get $\frac{52}{41.2}$/year as value for μ_v. Finally, using our own formula the infection rates are obtained as

$$\beta_H = \frac{\frac{1}{77.5}/year\times(1-0.09)\times\frac{27.81}{100}}{(1-\frac{27.81}{100})\times(26\times\frac{4.1}{100})vector}\text{=0.004/vector-year,}$$

$$\beta_v = \frac{\frac{52}{41.2}/year\times\frac{4.1}{100}}{(1-\frac{4.1}{100})(5\times\frac{27.81}{100})human}\text{=0.041/human-year.}$$

In our literature review, We do not find any documented data for vectors' birth rate per human (b_{v1}). So, we use the total vector population in disease free state from 4 to do back-calculation for estimating b_{v1}. Setting migration rates (m_{12} and m_{21}) as zero in N^*_{v1} for intermediate case, we get $N^*_{v1} = V_1 = \frac{b_{v1}H_1}{\mu_v}$ and eventually we get the formula:

$$b_{v1} = \frac{\mu_v V_1}{H_1}$$

This study find documented value for household size as 5 persons [22] and for bugs per infested house as 26 (1429 bugs in 55 houses, only the domiciliary cases are considered since we are looking for vec-

Table 3: Summary of estimated model parameters

Par.	Definition	Value	Units	Reference
β_H	Infection rate for human	0.104	1/vector-year	This study
β_v	Infection rate for vectors	0.206	1/human-year	This study
p	Probability of vertical transmission in humans	0.09	-	[20]
b_{v1}	Vectors birth rate (per human)	2.95	vector/human-year	This study
b_{v2}	Vectors birth rate (per chicken)	14.75	vector/chicken-year	This study
μ_H	Death rate for human	1/77.5	1/year	[21]
μ_v	Death rate for vectors	52/41.2	1/year	This study
m_{12}	migration rate from humans to chickens in (2)	$\frac{365}{(14\times15)}$	1/chicken-year	This study
m_{21}	migration rate from chickens to humans in (2)	$\frac{365}{(14\times5)}$	1/human-year	This study
q	proportion of time at which vectors fed on humans	1/6	-	[23]

tors' birth rate per human) (V_1) [9]. Unfortunately, the vectors' data we have is from houses where other hosts (dogs and cats) live also. In our literature review, we get 2.0 dogs and 0.5 cats per house [22]. So, to make the value of b_{v1} exactly per human we use the equivalence relation (based on the vectors' feeding pattern) among hosts done by Gürtler et al. [23] where they show one dog or cat is equivalent to 2.45 (mean of 2.3 and 2.6) humans. After doing some basic arithmetic, we find the equivalent number of persons per household is 11.125 (we use this as H_1 only for the estimation of b_{v1}, otherwise we use 5 as the value of H_1). Using this equivalent value in the above formula for b_{v1} we obtain

$$b_{v1} = \frac{(\frac{52}{41.2})/year\times 26 vectors}{11.125 human} = 2.95 vector/human - year$$

Since, vectors fed on chickens five times more than humans [23], we multiply the value of b_{v1} by 5 to get the value for b_{v2} which gives 14.75/chicken-year.

For estimating migration rate from chickens to humans (m_{21}), we take the time duration of vectors' last feeding to seeking a new host from [24], convert it to year, take the reciprocal of it and finally divided by household size which gives $\frac{365}{14\times5}$/human-year. For estimating the value of m_{12}, we similarly use the number of chickens/household, which is 15 [22] and get $m_{12} = \frac{365}{14\times15}$/chicken-year. And, we have $\frac{1}{6}$ (documented as five times more fed on chickens compare to humans) [23] for the proportion of time at which vectors fed on humans (q). All the estimated parameters summarized in Table 3.

4 Analysis

The goal of this study is to observe the impact of the additional incompetent host on the prevalence of Chagas disease among humans. The equilibria and the basic reproduction number (R_0) are primary indicators for such observations.

To find the equilibria of all three different dynamical systems, we set every single equation equal to zero for each model separately and solve. In this process, we find the total vector population (N^*_{v1}) from the disease-free equilibrium; those are shown in Table 4. We also get the infected human population (I^*_H) from the endemic equilibrium. Even though we are interested in observing the behavior of the infected population class, we still need to know the basic reproduction number (R_0) as it plays a very important role in interpreting the behavior of any infectious disease. To find the expression for R_0 we use the next generation method [25]. The expressions for R_0 and I^*_H for all three cases are in Table 5.

The expressions for R_0 and I^*_H clearly manifest that I^*_H is positive in all three cases iff R_0 >1. Now,

Table 4: N^*_{v1} for all three cases

Far distance	Intermediate distance	Short distance
$\frac{b_{v1}H_1+b_{v2}H_2\left(\frac{m_{21}}{m_{21}+\mu_v}\right)}{\mu_v\left(1+\frac{m_{12}}{m_{21}+\mu_v}\right)}$	$\frac{b_{v1}H_1+b_{v2}H_2\left(\frac{m_{21}H_1}{m_{21}H_1+\mu_v}\right)}{\mu_v\left(1+\frac{m_{12}H_2}{m_{21}H_1+\mu_v}\right)}$	$\frac{b_{v1}H_1+b_{v2}H_2}{\mu_v}$

Table 5: R_0 and I^*_H for all three cases, note N^*_{v1} is a function of H_2 in each case

Case	R_0	I^*_H
Far distance	$\frac{p}{2}+\sqrt{\frac{p^2}{4}+\frac{\beta_H\beta_v H_1 N^*_{v1}}{\mu_h\mu_v\left(1+\frac{m_{12}}{m_{21}+\mu_v}\right)}}$	$\frac{-\mu_H(1-p)\mu_v\left(1+\frac{m_{12}}{m_{21}+\mu_v}\right)+\beta_H\beta_v H_1 N^*_{v1}}{\beta_v\left[\mu_H(1-p)+\beta_H N^*_{v1}\right]}$
Intermediate distance	$\frac{p}{2}+\sqrt{\frac{p^2}{4}+\frac{\beta_H\beta_v H_1 N^*_{v1}}{\mu_H\mu_v\left(1+\frac{m_{12}H_2}{m_{21}H_1+\mu_v}\right)}}$	$\frac{-\mu_H(1-p)\mu_v\left(1+\frac{m_{12}H_2}{m_{21}H_1+\mu_v}\right)+\beta_H\beta_v H_1 N^*_{v1}}{\beta_v\left[\mu_H(1-p)+\beta_H N^*_{v1}\right]}$
Short distance	$\frac{p}{2}+\sqrt{\frac{p^2}{4}+\frac{\beta_H\beta_v H_1 q^2 N^*_{v1}}{\mu_H\mu_v}}$	$\frac{-\mu_H(1-p)\mu_v+\beta_H\beta_v H_1 q^2 N^*_{v1}}{\beta_v\left[\mu_H(1-p)q+\beta_H q^2 N^*_{v1}\right]}$

to check the impact of the presence of our incompetent host, chickens (H_2), we define I^*_H as a function of H_2 and then take the derivative of this newly defined function with respect to H_2. The expressions of these derivatives for far distance and short distance cases are given in Table 6. From the expressions, it is evident that these derivatives are always positive, which implies bringing chickens into the system always makes the situation worse for humans.

However, the consequences for the intermediate distance case are not straightforward. Here, the value of the derivative $I^{*\prime}_H$ (with respect to H_2) either can be positive or can be negative depending on certain conditions. In our analysis, we find housing chickens at an intermediate distance from humans can cause the prevalence of Chagas disease among humans to be slowed down only if

$$b_{v2} < \frac{m_{12}}{m_{21}}\left[\frac{\mu_H(1-p)}{\beta_H H_1}\mu_v K + b_{v1}(1+K)\right], \tag{4}$$

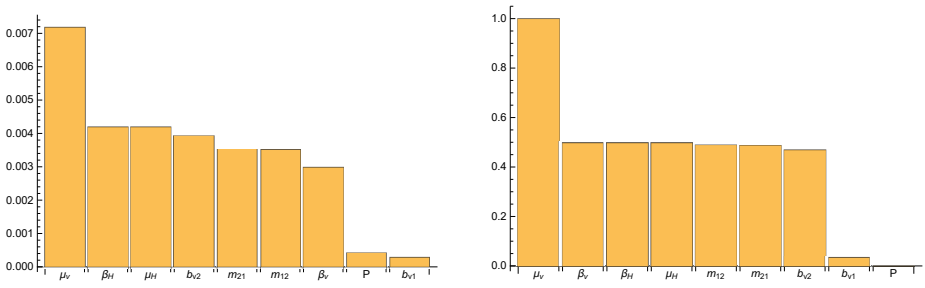
where $K = \frac{\mu_v(1+\frac{m_{12}H_2}{m_{21}H_1+\mu_v})}{\beta_v H_1(1+\frac{m_{12}H_2}{m_{21}H_1+\mu_v})^{-1}+\mu_v(1-\frac{m_{12}H_2}{m_{21}H_1+\mu_v})}$.

The above condition (4) on b_{v2} can only be true if

$$m_{12}H_2 < (m_{21}H_1+\mu_v)\sqrt{1+\frac{\beta_v H_1}{\mu_v}} \tag{5}$$

Table 6: Derivatives of I^*_H with respect to H_2

Far distance	$\frac{\mu_H(1-p)\beta_H\beta_v m_{21}b_{v2}H_1\left[1+\frac{\beta_v H_1}{\mu_v\left(1+\frac{m_{12}}{m_{21}+\mu_v}\right)}\right]}{\beta_v(m_{21}+\mu_v)\left(\mu_H(1-p)+\beta_H\left[\frac{b_{v2}H_2}{\mu_v\left(1+\frac{m_{12}+\mu_v}{m_{21}}\right)}+\frac{b_{v1}H_1}{\mu_v\left(1+\frac{m_{12}}{m_{21}+\mu_v}\right)}\right]\right)^2}$
Short distance	$\frac{\mu_H(1-p)\beta_H b_{v2}\mu_v(q\beta_v H_1+\mu_v)}{\beta_v[q\beta_H(b_{v1}H_1+b_{v2}H_2)+\mu_H(1-p)\mu_v]^2}$



(a) Sensitivity indices for I_H^* (b) Sensitivity indices for R_0

Figure 5: Sensitivity Analysis for all model parameters

Here, the second addend in (4) is directly proportional to b_{v1}, and the first term is inversely proportional to both β_v and β_H. Thus this condition is easy to satisfy when vectors have easy access to humans (high b_{v1}) or disease transmission (β_H and β_v) is low. So, the presence of chickens is helpful in this case if the birth rate of vectors with chickens is less than a certain threshold value which is relative to the birth rate of vectors with humans and inversely proportional to the infection rate among humans.

A sensitivity analysis of the potential endemic prevalence of Chagas disease (I_H^*) and R_0 indicates that (fortunately) neither of them is very sensitive to the model parameters which are more difficult to estimate well. Partial rank correlation coefficients for both quantities were computed for all model parameters (Figure 5). Both measures were most sensitive to vector longevity, μ_v, which is well known. All sensitivity indices for I_H^* were extremely low (less than 0.01), and all sensitivity indices for R_0 except μ_v's were less than 1/2. One of the next highest sensitivity indices in both cases was that of host longevity, μ_H, also well known. Remarkably, neither measure (I_H^* and R_0) is sensitive either to birth rates (b_{v1} and b_{v2}), or to infection rates (β_v and β_H), or to migration rates (m_{12} and m_{21}). These sensitivity analyses show that the parameters not known well are less influential and the most influential parameters are known well. So, the results of this study will not be significantly affected even if the actual values of our estimated parameters vary significantly from our estimation.

To facilitate interpretation, here we illustrate our results numerically for only the helpful case (intermediate case) in brief. At baseline (our estimated parameter values), we get $R_0 = 1.58$ and we also find the condition $b_{v2} < 19.57$/chickens-year at which the presence of chickens is helpful in reducing prevalence of Chagas disease in humans. In our analysis, we find R_0 strictly decreasing function of m_{12} and strictly increasing function of m_{21}. However, R_0 increases for up to a certain number of chickens and then start to decrease (Figure-6). This implies that for our parameter values the presence of chickens can reduce the infections in humans depending on the number of this incompetent host. Now, the condition for making the presence of chickens helpful becomes easier to satisfy as migration of vectors from

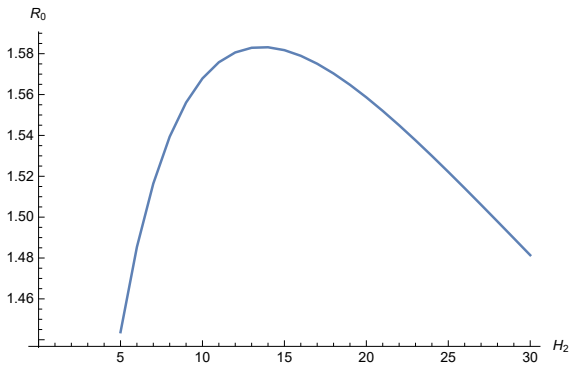


Figure 6: Behavior of R_0 as number of chickens (H_2) varies

humans to chickens increases and it becomes difficult as migration from chickens to humans increases. However, an increase in the number of chickens makes it easy to satisfy the condition for ensuring the presence of chickens helpful in reducing the prevalence in humans. All the numerical values here are based on our parameter estimations which can be different with other set of parameter values. However, the qualitative result will be the same regardless of parameter values.

Our results and analyses show that the presence of an incompetent host, in our case chickens, can reduce the prevalence of Chagas disease in humans under certain conditions only if chickens are placed at an intermediate distance from humans.

5 Discussion

The case when farmers bring their chickens inside the house has a positive impact on disease prevalence. Though the presence of the incompetent host may distract a significant fraction of vectors from humans to chickens at the beginning, however, in the long run the vector population will increase so much that the number of vectors biting humans is greater with chickens than without chickens. So, it is easily understandable why this close presence of chickens to human is not helpful for the reduction of human infections of our concern disease. For the far case where vectors can not anticipate the location of chickens, the decoy process does not help to reduce human infections. Here, vectors try to stay with humans as long as they can survive since they can't see any alternative food sources around them. So, by the time when a portion of them start to leave humans, the infections are already spread among humans at a large scale. Consequently, this case is not helpful for the purpose of controlling the prevalence of infections among humans.

In the remaining case, when chickens reside at a distance (adjacent to humans) such that vectors can detect the presence of remaining host while staying with the other, vector populations begin to migrate from humans to chickens in search of their blood-meals. The vector population with chickens will increase with time for having enough food sources and at some point they will start to move towards different directions in search of new blood-meal sources. Among those directions one will go back to humans. However, the net effect of vectors' migration from humans to chickens and from chickens to humans will reduce infections among human under some certain conditions. This will happen as most of the vectors will switch from humans to chickens before people in houses are infected that much.

So, the presence of chickens in households in the usual scenario, when chickens are kept at farther distance from people's living place, is not helpful to reduce the infections of Chagas disease among humans. Also, having chickens in bedrooms or very close to bedrooms has no positive impact on the reduction of human infections. The presence of chickens in houses can only help to reduce the prevalence of Chagas disease among humans when villagers keep their chickens at a distance which allows the vectors to anticipate the location of other hosts, but does not allow vectors to share both of the chickens and humans as their blood meal sources. Also, this scenario, in terms of disease prevalence in humans, has a negative relation with the increase in number of chickens. So, it can be concluded by saying that the decoy process, by the presence of an incompetent host, does not always help to reduce the disease prevalence among humans.

Results of this study will open a new door for the control of Chagas disease infections among humans. Ensuring the presence of chickens at a certain distance from household with satisfying certain conditions will enable us to reduce the infection of Chagas disease in humans.

However, implementation of the outcomes of this study depends on the distance from where vectors can sense the presence of hosts. Triatomine vectors' host detection achieved by identifying the presence of couple of factors such as distinctive odors from different odorants (including CO_2), water vapor and heat [26], [27]. Unfortunately, we find only one documented data source which says Triatomine bugs can identify humans presence from 2 meters by detecting heat [27]. So, further work can be done to explore the minimum and maximum distance from where vectors can detect the presence of chickens and humans as well, and then those explored values along with the outcomes of this study can help public health officials to implement effective policies in order to improve the scenario of Chagas disease infections in humans.

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