

ASSESSING THE IMPACT OF DISEASE VECTORS ON ANIMAL POPULATIONS

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ABSTRACT

Many studies have attempted to assess the relative effects of different vectors of a disease on animal populations. To this end, three measures have been proposed: Vectorial efficiency, Vectorial capacity and recently Vectorial effectiveness (or Vectorial impact). In this study we relate these measures to derive some of their properties emphasising in the vectorial impact for its importance in both, population performance of parasites and the proportion of the prevalence of one parasite due to a given vector. We applied the quantitative expressions advanced in this study to a simple Chilean example with one parasite (*Trypanosoma cruzi*), two vectors (*Triatoma infestans* and *Mepraia spinolai*) and one animal population (humans: Chagas's disease).

KEY WORDS: disease vectors, vectorial impact, vectorial capacity

1. INTRODUCTION

One of the most important regulating factors of animal populations are infectious diseases (Anderson and May 1979, May and Anderson 1979, Holmes 1982, Dobson and Hudson 1986, Ewald 1995, Gulland 1995, Myers and Rothman 1995). Among these, vector borne diseases are particularly important in community ecology due to their impact on both geographical distribution and the abundance of animal populations (May and Anderson 1979, Dobson and Hudson 1986, Lehane 1993, Ewald 1994, Loye and Carrol 1995).

Although the interaction between vector insects and animals has received much attention, many problems relating to the estimation of entomological parameters of ecological interest, as well as the biology of vectors, have yet to be fully understood (Anderson 1993).

Many studies have attempted to assess the relative effect of different vectors of a particular disease on animal populations (Mc Donald and Cuellar 1968, Herath et al. 1983, Molineaux et al. 1988, Burkot et al. 1988, Mendis et al. 1990, 1992). To this end, the most commonly used measure, the vectorial capacity, attempt to measure the

vectors contribution to the parasite's potential daily reproductive rate (Garret-Jones 1964, Garret-Jones and Shidrawi 1969, Garret-Jones et al. 1980). However, this measure on its own is insufficient. It does not account for the individual risk of one animal to acquire an infectious disease when he has been bitten by a vector and neither about the relative impact of each one vector on a particular host population. Canals et al. (1993) have proposed that to fully evaluate the effect of vectors on animal populations, the following three issues must be addressed: i) what is the probability that a susceptible animal could become infected after being bitten by a vector insect? (Vectorial efficiency); ii) How many potentially infecting bites can a population of a particular vector insect distribute as a result of bites on one infected host? (Vectorial capacity); and iii) Of a population of infected animals, what proportion has been infected by a given vector? (Vectorial effectiveness or Vectorial Impact).

As we will see later, to answer these three questions several entomological parameters need to be estimated. Knowing each one it is possible to answer these questions and identify sensible parameters for the individual and population risk. Also, establishing relationships between the vectorial efficiency, vectorial capacity and vectorial effectiveness would permit answering several other questions such as: is a vector with high vectorial capacity, also an efficient transmitter? or does the population effect of a single vector depend on its own efficiency and vectorial capacity?, or does it depend on those of the other vectors?.

In this study we relate these three measurements to derive some of their properties. We emphasise in the vectorial impact because it is important in two senses: i) from the view point of the parasite it represents the relative importance of different members of the coterie of vectors on their population performance, over a particular definitive host, and ii) from the viewpoint of the definitive host it represents the proportion of the existing prevalence that is actually due to a given vector (the impact of the vector).

In addition we evaluate their utility by using them on a published data set.

2. THEORETICAL APPROACH

Consider the following events:

A_i : an individual vector of type i carries the infectious agent;

B_i : an individual is bitten by a vector of type i ;

C : the susceptible host gets infected.

The vectorial efficiency (E_i) can be expressed as :

$$E_i = P((C \cap A_i)/B_i) \quad [1]$$

which for convenience can also be written as:

$$E_i = P((C \cap A_i)/B_i) = P(C \cap (A_i \cap B_i))/P(B_i) = P(C/(A_i \cap B_i)) \cdot P(A_i \cap B_i)/P(B_i) \quad [2]$$

Strictly speaking A_i and B_i might not be independent. Such is the case of many blood sucking insects in which the presence of the infectious agent (parasite) can affect the biting frequency (Molyneux and Jefferies 1986). However, neglecting this effect, by assuming A_i and B_i are independent, we may obtain a simple expression for the vectorial efficiency:

$$E_i = P(C/A_i \cap B_i) \cdot P(A_i) \quad [3]$$

This way, an efficient vector is one which has both a high likelihood of carrying the infecting agent (large $P(A_i)$) and is a good transmitter of it (large $P(C/A_i \cap B_i)$).

We define Vectorial Impact (or effectiveness) (I_i) as the proportion of infected individuals that were caused by vector of type i . It corresponds to a conditional probability determined by the ratio of the probabilities of [being infected and having been bitten by an infecting agent i] to the probability of [being infected]. Considering only vectorial transmission, the denominator can be decomposed into all the different ways of being infected (*i.e.* different vectors). Thus, one host could be infected by a vector of type i , or by a vector of type j (but not by both vectors), or a vector of type m ...etc.

Using Bayes's theorem we get:

$$I_i = P(Q_i/C) = P(C/Q_i) \cdot P(Q_i) / \left\{ \sum_j P(C/Q_j) \cdot P(Q_j) \right\}$$

where Q_i = infecting access of a vector of type i . A good estimator of $P(Q_i)$ is: $P(Q_i) = P(A_i \cap B_i) / [\sum P(A_j \cap B_j)]$. Furthermore we note that $P(C/Q_i) = P(C/A_i \cap B_i)$. The difference between Q_i and $(A_i \cap B_i)$ is that, while an infected host might have been bitten by several vectors (*i.e.* $(A_i \cap B_i)$ and $(A_j \cap B_j)$ are not mutually exclusive), only one of them would infect the host (Q_i and Q_j mutually exclusive for all i and j).

With these considerations:

$$I_i = P(C/(A_i \cap B_i)) \cdot P(A_i \cap B_i) / \left\{ \sum_j P(C/(A_j \cap B_j)) \cdot P(A_j \cap B_j) \right\} \quad [4]$$

with $j = 1, 2, \dots, K$, K = number of vectors.

If we assume that A_i and B_i are independent, and that the probability of infection given a bite by a vector carrying the agent is the same for all vectors ($P(C/A_i \cap B_i) = P(C/A_j \cap B_j)$, for all i and j) we obtain a simplified expression:

$$I_i = P(A_i) \cdot P(B_i) / \left\{ \sum_j P(A_j) \cdot P(B_j) \right\}$$

Without assuming equal infectivity or independence of A_i and B_i , equation [4] can be written as :

$$I_i = \frac{P(C/A_i \cap B_i) \cdot P(A_i \cap B_i)}{P(C/A_i \cap B_i) \cdot P(A_i \cap B_i) + \sum_{j \neq i} P(C/A_j \cap B_j) \cdot P(A_j \cap B_j)}$$

Dividing both the numerator and the denominator by $P(B_i)$ we get:

$$I_i = \frac{E_i}{E_i + \sum_{j \neq i} P(B_j)/P(B_i) \cdot E_j}$$

which, dividing by E_i , yields:

$$I_i = \frac{1}{1 + \sum_{j \neq i} c_{ji} \cdot ER_{ji}} \quad , \quad c_{ji} = P(B_j)/P(B_i) \quad ER_{ji} = E_j/E_i \quad [6]$$

c_{ji} represents the relative probability of a given host is bitten by a vector of type j , with respect to the same probability for a vector of type i . ER_{ji} represents the relative efficiency of a vector of type j with respect to the vector i .

Now, in the next paragraph, we analyse how the vectorial capacity is related to the vectorial impact.

Vectorial capacity was defined by Garret-Jones (1964) as the product of: i) the biting rate of a vector (α_i), ii) the expectation of infective life (Γ_i), and iii) the biting habit (a_i). The biting rate of the vector is simply the product of the biting habit and the vectorial abundance (m_i). And in turn the biting habit is the product of the biting frequency (number of bites per unit of time) and the specific blood index (proportion of vectors having a specific type of blood, *i.e.* human blood). Thus, the vectorial capacity $CV_i = m_i \cdot a_i^2 \cdot \Gamma_i$, yields:

$$\alpha_i = m_i \cdot a_i = \frac{\sqrt{m_i \cdot CV_i}}{\Gamma_i} = k_i \sqrt{CV_i} \quad [8]$$

The number of bites on individuals of a host species (bn) can be modelled as following a Poisson distribution with mean $L_i = \alpha_i t$ (Bailey 1975), with t being the time period.

The probability that a host individual is bitten by a vector of type i is thus:

$$P(B_i) = P(bn \geq 1) = 1 - e^{-L_i}$$

Thus the ratio of probabilities that the vectors j and i will bite a particular susceptible host (c_{ji}) is given by:

$$c_{ji} = \frac{1 - e^{-k_j t \sqrt{CV_j}}}{1 - e^{-k_i t \sqrt{CV_i}}} \quad [9]$$

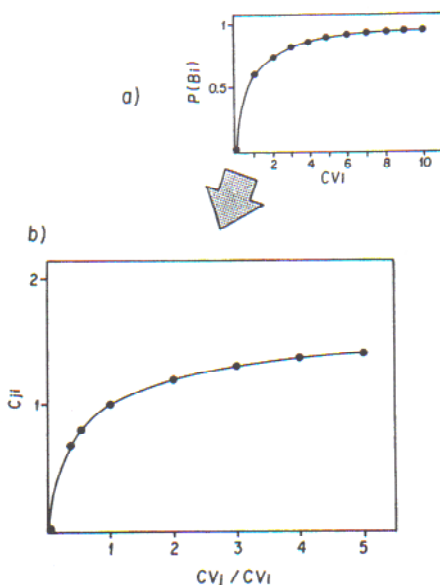


Figure 1. Relationship between the vectorial capacities (CV_i) and probabilities of being bitten by a vector of type i ($P(B_i)$). When CV_i is larger, the probability $P(B_i)$ is also high (a). A similar effect over c_{ji} is seen if CV_j / CV_i is high, when considering the abundance and the infective life as the same ($k_i = \sqrt{m_i / \Gamma_i} = \sqrt{m_j / \Gamma_j} = k_j$) (b). The later figure was built considering a constant value of CV_i (*i.e.* $CV_i = 1$) and varying CV_j from 1 to 5 times the value of CV_i .

The vectorial efficiencies are independent of CV_i , but the vectorial capacities modulate the c_{ji} coefficients (Figure 1), and these and the vectorial efficiencies determine the vectorial impact.

To simplify the interpretation of the relationships between CV_i , E_i and I_i , expression [6] can be rewritten as:

$$I_i = \frac{1}{1 + (k-1) \cdot R_{ki}}, \quad R_{ki} = \left[1/(K-1) \right] \cdot \sum_{j \neq i} (c_{ji} E R_{ji}), \quad K \text{ the Number of vectors [10]}$$

R_{ki} compares the parameters of the other $(K-1)$ vectors against those of vector i . The value of R_{ki} is zero when the disease is only caused by the vector i . Notice also that the vectorial impact (I_i) decreases with an increasing value of K (the total number of vectors). However, the rate of decrease of I_i , when k increases, is modulated by R_{ki} . If the efficiency and the vectorial capacity of the vector i are larger (small R_{ki}), adding vectors to the coterie will have a negligible effect on I_i (Figure 2). On the contrary, elimination of this vector i , could result in drastic changes in the vectorial effectiveness I_j of the other $(K-1)$ vectors. In contrast, for vectors with low vectorial efficiencies and vectorial capacities adding species to the coterie will result in a strong decreasing effect on I_i . Their elimination would provoke small changes in vectorial effectiveness of other vectors.

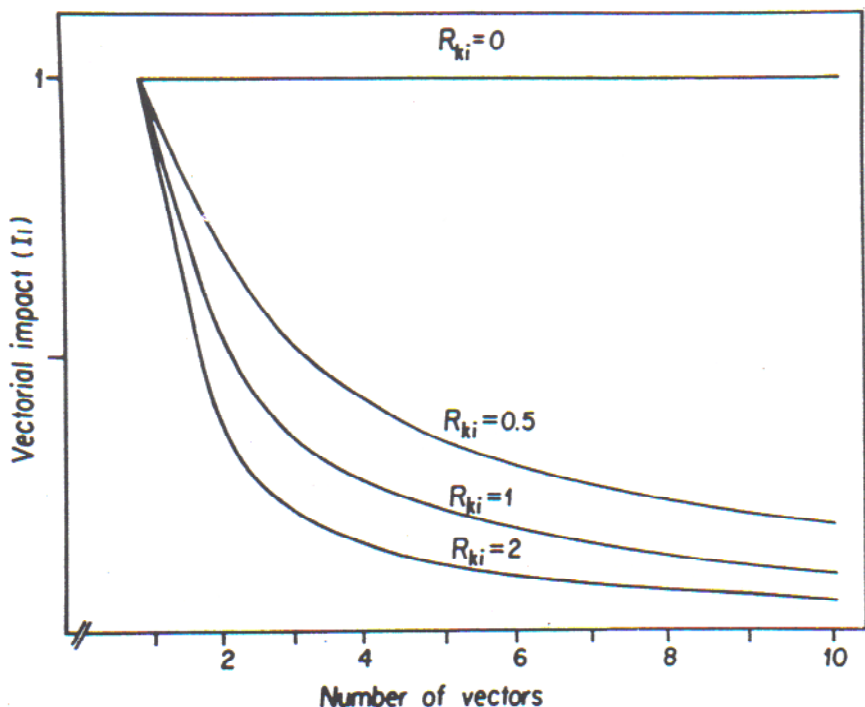


Figure 2. Relationship between effectiveness of "i" vector and the size of the vectors coterie (K), at different values of R_{ki} (see text).

3. EXAMPLE

There are two vectors of *Trypanosoma cruzi*, the agent causing Chagas's disease (American Trypanosomiasis), in Chile: *Triatoma infestans* and *Mepraia spinolai* (Hemiptera; Reduviidae). They are sympatric species, but *T. infestans* has domestic habits while *M. spinolai* prefers wild and peri-domestic habitats (Apt and Reyes 1990).

In this example, we consider the metropolitan zone of Santiago, the best studied zone in Chile with endemic Chagas's disease.

The probability that an individual becomes infected with *Trypanosoma cruzi* when bitten by one bug carrying the trypanosome, is approximately $P(C/A_i \cap B_i) = 0.01$ (Rabinovich and Rossel 1978). This estimation assumes a contamination by defecation on the bite site. However, while *T. infestans* always defecates on the host during a bite, *M. spinolai* does it only in 3.7% of bites (Canals et al., 1998a). Then, we consider $P(C/A_i \cap B_i) = 0.01$ in *T. infestans* and $P(C/A_i \cap B_i) = 0.00037 (= 0.01 \cdot 0.037)$ in *M. spinolai*. $P(A_i)$ is also different between the species, and both $P(A_i)$ and $P(C/A_i \cap B_i)$ are responsible for differences in their vectorial efficiencies (Table 1).

Table 1.- Vectorial efficiency (E_i), Vectorial capacity (CV_i) and Vectorial Impact (I_i) of Chilean vectors of Chagas's disease (American Trypanosomiasis): *Triatoma infestans* and *Mepraia spinolai*, on human populations in Santiago, Chile. The vectorial efficiency is the product of $P(A_i)$ and $P(C/A_i \cap B_i)$ ([3]); the vectorial capacity is $CV_i = m_i \cdot a_i^2 \cdot \Gamma_i$ ([7]); $ER_{ji} = E_j / E_i$ ([6]); $c_{ji} = P(B_j) / P(B_i)$ ([6] and [9]) and I_i from [10].

	$P(A_i)$	E_i	m_i	Γ_i	CV_i	c_{ji}	ER_{ji}	I_i
<i>T. infestans</i>	0.321	0.00321	34.44	121.26	12.50	15.816	32.10	0.998
<i>M. spinolai</i>	0.260	0.00010	4.59	160.66	0.106	0.063	0.030	0.002.

If we use [5] for I_i we obtain 0.951 and 0.049 for *T. infestans* and *M. spinolai* respectively. These are good approximations, but in our case the assumption $P(C/A_i \cap B_i) = P(C/A_j \cap B_j)$ is not valid.

The biting frequency of one bug has been estimated in 0.0799 bites/day for *infestans* and in 0.153 bites/day for *M. spinolai* (Canals et al., 1998a). The human blood indices are 0.684 in *T. infestans* (Schenone et al. 1985) and 0.0784 in *M. spinolai* (Canals et al., 1998b) in this zone. Then, the biting habits of *T. infestans* and *M. spinolai* are $a_i = (0.0799 \cdot 0.684) = 0.0547$ and $a_j = (0.153 \cdot 0.0784) = 0.0120$ human bites/ day respectively.

The population density (m_i) of *T. infestans* has been estimated as 34.44 individuals per human. In this zone the maximum capture rates (individuals per hour per human) are 480 for *T. infestans* (Canals et al. 1993) and 64 for *M. spinolai*. Assuming a constant ratio between maximum capture rate and population density, we estimate 4.592 individuals per human of *M. spinolai*. The expectation of infective life (Γ_i) of the trypanosome in each vector species has been estimated (Canals et al. 1993). With these parameters, using equations [7] to [10], we obtained vectorial capacities, and vectorial impact as shown in Table 1.

Our results show that *T. infestans* is a more efficient vector of the Chagas's disease than *M. spinolai*. This difference is supported mainly by a lower probability of *M. spinolai* to transmit the agent during a bite when he carries it. Also these vectors have a different vectorial capacity supported by differences in the vectorial abundance and the biting habits, particularly by a lower human blood index of *M. spinolai*. In agreement with these results the vectorial capacity of *M. spinolai* is lower than it of *T. infestans*. This is supported by a vectorial efficiency of *T. infestans* 32 times the vectorial efficiency of *M. spinolai* and also larger c_{ji} . In this zone *T. infestans* would be responsible for 99.8% of infections of humans by *Tripanosoma cruzi* (see Table 1). That virtually all Chagas's infection prevalence is caused by *T. infestans* has been proposed by previous authors (Apt and Reyes 1990, Schenone 1989).

The elimination of *T. infestans* would provoke a great decrease of Chagas's infection only initially, because the vectorial impact of *M. spinolai* would be 1. The future of *T. cruzi* in human hosts in Chile would depend on the extent to which *M. spinolai* population density and human blood index increased; both would be associated with an increase in their capability to colonise human dwellings.

4. DISCUSSION

We have put forward quantitative expressions which relate three concepts in vectorial ecology: Efficiency, Impact and Vectorial Capacity. We think that the definitions and the procedure advanced in this note can be used in field studies in order to clarify the effects of insect vectors on establishment of parasite sub-populations. It can also be used to assess the impact of particular vectors on hosts populations and to rationalise control policies. The three measures are useful in different senses. The vectorial efficiency assesses the risk of an individual becoming infected when he has been bitten by a vector, and the vectorial impact measures the population effect of a particular vector. Both measures are easy to estimate (see eq. [3] and [5]) and they are specially useful in human diseases. The vectorial impact is also conceptually useful. From the view point of the parasite it represents the relative importance of different members of the coterie of vectors on their population performance, over a particular definitive host. More to the point, it represents the proportion of sub-populations that a vector is able to disseminate on a particular host. This situation is analogous to that of the seed dispersers in which the disperser effectiveness represents the effects of the different dispersers on the performance of the plants (see Bustamante & Canals 1995). From the host standpoint the vectorial impact assess the population damage caused by a specific vector (*i.e.* the proportion of the existing prevalence is actually due to a given vector).

The vectorial capacity contributes in evaluating the vector contribution to the potential reproductive rate of a parasite species, but it is an absolute measure. This fact differences it from the vectorial impact which is a relative one. Also the vectorial capacity is not an impact measure, but differences in it between the vectors species plus differences in their vectorial efficiencies determines the vectorial impact.

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