The use of satellite data in modeling population dynamics and prevalence of infection in the rodent reservoir of Junin virus

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Abstract

We present the first results of a simple numerical model of rodent population dynamics and viral infection for Junin virus, etiologic agent of Argentine hemorrhagic fever (AHF), in its host, Calomys musculinus. In contrast to the more common statistical approach, the model incorporates satellite-derived environmental data in a causal approach. In addition, the model incorporates specific biological characteristics of the host, such as birth rate and longevity. Theoretical and actual rodent population densities are compared with several years of capture data at locations on the Argentine pampas. The model appears to be a good tool for simulating dynamics of populations using remotely sensed data. Results are in agreement with field data showing maximum population densities during the autumn in most localities. The differences between simulated population densities and field observed values indicate that, although computer simulation is useful to obtain some aspects of population dynamics, improvements should be introduced in order to generate more robust results.

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1. Introduction

Satellite data have become important sources for a variety of biological and bio-geographical studies.

Depending on the way in which remote sensing can be combined with ecological models, these broad range of applications has been grouped in four categories (Plummer, 2000). For example, considering just the spatial scale (running from global to species level), remotely sensed data provides estimates of variables required to drive ecological process models of pre-
Argentine hemorrhagic fever (AHF) is a rodent-borne viral infectious disease that occurs in the humid pampas of central Argentina. The etiologic agent is Junin virus (JUNV), a member of the Arenaviridae family. AHF is a severe systemic disease with hemorrhagic and neurological manifestations. An important epidemiological characteristic of AHF is the progressive geographic extension into new areas and the disappearance of the disease from the oldest areas (Birkby, 2001). Finally, at species level, most of the works are oriented to analyze through multivariate statistic different variables (vegetation, air and surface temperature, humidity, and elevation) to access species distribution models and maps (Guisan and Zimmermann, 2000).

“Landscape epidemiology” is a relatively new interdisciplinary approach that involves the characterization of eco-geographical areas where diseases are transmitted. Landscape epidemiology is a second generation application of remotely sensed data where the target (the vector or reservoir host) can not be seen directly with satellite images. It is an holistic approach, which takes into account the relationships and interactions between the different elements of ecosystems under the assumption that the biological dynamics of the population of a host or vector are driven by landscape elements such as temperature and vegetation. A principal goal of this discipline is to develop risk maps for specific diseases to aid the formulation of a “health early warning system” (HEWS). There are several pioneering examples of the application of remotely sensed data to vector-borne diseases (Linthicum et al., 1987; Rogers and Randolph, 1991; Pope et al., 1992; Wood et al., 1992; Rogers and Randolph, 1993; Hay et al., 1996; Rogers et al., 1996; Beck et al., 1997, 2000).

The conditions associated with high risk of rodent-borne disease outbreaks can be monitored from remote sensors and, retrospectively, be related with reservoir population characteristics and the resulting human disease cases. Environmental information from remote
sensors can be an important tool for developing indicators, risk maps, and population models (Mills and Childs, 1998). These models can contribute to public health decision making by identifying specific times and locations that may pose a disease threat.

Greenness indexes integrate environmental information as temperature, precipitation, and soil properties. One of the most used index derived from remote sensed data is the normalized difference vegetation index (NDVI; Rouse et al., 1974) which is calculated as a ratio between the near infrared and red regions of the electromagnetic spectrum. The NDVI values rank from −1.0 to 1.0, where an increasing positive value indicates increasing green vegetation.

Investigators using remotely sensed data in studies of rodent-borne disease have successfully developed predictive models for risk areas of hantavirus pulmonary syndrome (HPS; Glass et al., 2000), and for Sin Nombre virus infection in deer mice (Boone et al., 2000). In both cases, an statistical approach was used. Boone et al. (2000) demonstrated a statistical relationship among the prevalence of infection in rodents at 144 field sites and environmental conditions such as vegetation type and density, elevation, slope, and hydrological features. Remotely sensed data were used to obtain a vegetation type map based on the normalized difference vegetation index, derived from Landsat. Glass et al. (2000) also derived a statistical relationship relating HPS case sites to precipitation, elevation, and the six non-thermal Landsat bands. In this case, the relationship with the remotely sensed data (the original Landsat bands) was direct.

Recently, a human HPS risk algorithm allowed to identify sites showing meaningful ecological differences between high and low risk conditions. In spite of the strong relationships found among different variables as HPS risk of sites, abundance and population structure of the reservoir, and the values of Landsat bands; the factors that link those variables remain to be determined (Glass et al., 2002a,b). This fact encouraged us to look for a different approach, which might explain that kind of relationship.

The use of causal models seems to be a less efficient method to develop predictive tools for operational use in the public health sector. Nevertheless, this complementary approach, which incorporates remote sensing in the development of numerical models of rodent population dynamics and virus transmission, might be very heuristic. For example, simulating the effects of modifying environmental parameters, or those parameters describing the biological characteristics of host species, could lead to insights into both the ecological and virological aspects of host–virus dynamics.

Because of its extensive area, economic importance, and high population density (Maiztegui et al., 1986), the AHF endemic area is particularly suitable for the application of the broad scale monitoring offered by space-based sensors. In this paper, we present an initial version of a model of C. musculinus population dynamics and viral infection, which uses remotely sensed data in a causal approach in contrast to the more common statistical approach. In addition, we present theoretical analyses and comparisons with experimental field data.

2. The model

The population model presented here is mainly based on the equations proposed by Kirchner and Roy (1999) describing the temporal dynamics of a single species population. That paper presents a theoretical study of the effects of longevity on the trajectory of populations of infected and non-infected individuals. It suggests that, under certain conditions, a longer life span is less advantageous for the host population. This theoretical approach has several similarities with the association between JUNV and C. musculinus. For this reason we used Kirchner and Roy’s (1999) model as a starting point for our modeling.

Kirchner and Roy’s (1999) model assumes the existence of two sub-populations, “X” representing the non-infected rodents and “Y” the infected rodents. Both “X” and “Y”, are expressed as a fraction of the ecosystem carrying capacity for the species. The sources and sinks for each class are schematically presented in Fig. 1. The temporal equation for the non-infected rodents is:

\[
\frac{dX}{dt} = \alpha (1 - N)X - \beta XY - \frac{X}{\tau_{\text{dead}}} 
\]

(1)

and for the infected population

\[
\frac{dY}{dt} = \beta XY - \frac{Y}{\tau_{\text{mi}}}
\]

(2)

where X is the non-infected host population as a fraction of carrying capacity, Y the persistently infected host
Fig. 1. Sources and sinks of each sub-population classes in the model. Dashed arrows show outputs and full arrows means input in each sub-population. \( \alpha \): Birth rate, \( \tau \): mean life span, \( \gamma \): immunization rate, \( X \): proportion of non-infected sub-population, \( Y \): proportion of infected sub-population, \( Z \): proportion of immunized sub-population (all in terms of carrying capacity), \( N \): total population (\( X + Y + Z \)), \( X/\tau \): non-infected host mortality rate, \( Y/\tau \): infected host mortality rate and \( Z/\tau \): immunized mortality rate.

Population as a fraction of carrying capacity and \( N \) is the total \( C. \) musculinus population, in this case \( N = X + Y \). In this non-dimensional approach the maximum possible value for “\( X \)” or “\( Y \)” is 1. Then \( 1 - N = 1 - (X + Y) \) is the free fraction of the carrying capacity (available to be occupied by new births). \( \tau \) (tau) is the maximum “mean life span” (equal for both, infected, and non-infected rodents) producing a non-infected host mortality rate \( X/\tau \), \( \alpha \) (alpha) the potential (per capita) reproductive rate in absence of carrying capacity constrains, \( \beta \) (beta) the average number of non-infected hosts that an infected host can infect during its lifetime, the term \( \beta XY \) the infection rate and \( m \) in the general case accounts for differential mortality rate. In the case of \( C. \) musculinus it is equal to 1, because infection with JUNV is assumed to have no negative effect on the rodent. Eqs. (1) and (2) can be discretized and then solved numerically in a simple finite differences scheme. In this process, IDL language was used and the time step was 1 day.

It is important to note that births are not included in Eq. (2) because intrauterine infection from persistently infected females has a detrimental effect on the offspring as was stated above.

Laboratory studies of \( C. \) musculinus infection with JUNV revealed that some individuals develop an immunizing infection. These immunized rodents cannot infect other non-infected rodents and they cannot be re-infected. To include this new rodent class in the model, we add an equation describing the population of immunized hosts, “\( Z \)”, including an immunization rate \( \gamma \) (gamma), as follows:

\[
\frac{dZ}{dt} = \gamma Y - \frac{Z}{\tau m} \tag{3}
\]

2.1. Model parameters

To improve our simulation, we used parameters for \( C. \) musculinus that were extracted from our database and from the literature: \( \tau \) was obtained from De Villafate and Bonaventura (1987); \( \alpha \) derived from De Villafate (1981) and Hodara et al. (1984); \( \beta \) obtained from data included in Sabattini et al. (1977) and information on \( \gamma \) values was obtained from Sabattini et al. (1977) and Mills et al. (1994). The values used to simulate population growth of the host of JUNV are presented in Table 1.

Because our goal is to develop an alternative method for using satellite information in characterizing parameters related to rodent-borne diseases, we are interested in identifying those parameters from Table 1 that could be associated with environmental conditions that can be monitored using remote sensors. We selected NDVI derived from the AVHRR instrument on board, the
Table 1

Comparison of original Kirchner and Roy (1999) and *C. musculinus* model parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Kirchner and Roy</th>
<th><em>C. musculinus</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>$\tau$</td>
<td>7 months</td>
<td>51</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>11.7 births per life span</td>
<td>12 per life span</td>
</tr>
<tr>
<td>$\beta$</td>
<td>3 per life span</td>
<td>0.12 per month</td>
</tr>
<tr>
<td>$m$</td>
<td>1</td>
<td>0.12 per month</td>
</tr>
</tbody>
</table>

*C. musculinus* model parameters were obtained from the literature cited on the text.

NOAA satellite as a remotely sensed variable representative of the environmental condition. NDVI was chosen because this greenness index integrates environmental information, which influence biological parameters of host populations and, indirectly, measures variables such as the quantity and quality of refuge and food (Suárez-Seoane et al., 2002).

Different biological aspects of *C. musculinus* have seasonal changes, in some cases they have a direct relationship with vegetation variables: for example, changes in social structure of *C. musculinus* during harvest and plowing season have been repeatedly reported (Crespo et al., 1970; Polop et al., 1982) due to the increase of death by agricultural machines, the increase of predation by birds and related to changes in plant species composition (Busch et al., 2000). The structure of the populations shows a growing feature with young individuals as most important group in Autumn (April), but is less evident later in the year. A decrease in the mean body weight of rodent trapped (De Villafañe et al., 1988), and a high number of “missed” rodents in closed plots during the winter (Polop, unpublished data) suggest the mean life span in *C. musculinus* is shorter in this season.

Also the reproductive cycles in *C. musculinus* run from September to June (Mills et al., 1992a,b), showing a seasonality of the pregnancy prevalence that decrease after June (De Villafañe et al., 1988).

With this knowledge, we assume in a parsimony way a linear dependence of both the reproductive rate “$\alpha$” and the mean life span “$\tau$” on NOAA AVHRR NDVI data. So, when NDVI increases (summer) we propose that “$\alpha$” and “$\tau$” take their maximum values, and when NDVI has lower values (winter) we assume, based on experimental evidence that birth rate decreases and mortality increases (“$\alpha$” and “$\tau$” decreases). NDVI data were obtained from decadal (10 days) series available at “daac.gsfc.nasa.gov”. For each locality where we ran the model, we used NDVI image series of a single pixel value ($8\times8$ km of resolution) corresponding with its geographical coordinates. Hereafter, when we refer to localities we are speaking about the rural agricultural area near to small towns with a typical radius of about 2 km. Each locality’s rural environmental conditions are described by a pixel. To feed the model, an interpolation of decadal data is used in order to obtain an NDVI value corresponding to each time step. In the model, these NDVI values result in “$\alpha$” and “$\tau$” values within the limits defined by laboratory and field values (Table 1), ranging from 0.3 to 1 offspring per month and from 2.5 to 7 months of mean life span.

3. Study area and field data

Our study area included parts of the Argentinean humid pampas, in southern Santa Fe and northern Buenos Aires provinces. This region includes the AHF-endemic area, and the large quantity of historical field and epidemiologic data available for the last 25 years, which gives us a cognitive base in order to contrast the model results.

3.1. Study site and rodent trapping

Our data set represents AHF studies conducted from 1991 to 1994. Rodent trapping was conducted at three sites within the AHF-endemic area: Pergamino falls, in southern Santa Fe and northern Buenos Aires provinces. Two localities, Alcorta and Máximo Paz, were selected from the AHF-epidemic area, which currently has a high incidence of disease.
4. Testing results

When we use *C. musculinus* parameter values (Table 1) to run the model with an infected and a non-infected sub-population (as originally did Kirchner and Roy, 1999), the results were very similar to those of the original model (Fig. 3 in Kirchner and Roy, 1999). In the next step, we included the immunized sub-class "Z" in Eq. (3) using the same parameter values. Fig. 1 shows the schematic representation of this model. In both cases, the proportions of different host categories evolve to stationary values as described by Kirchner and Roy (1999). Nevertheless, the effect of including another rodent category produces an increase in the total population and inversely lowers stationary values for infected sub-population ("Y") by almost 50% (Fig. 2). Note that "Y" represents individuals that can produce human infection.

Fig. 2. Evolution of *C. musculinus* population considering non-infected (X), permanent infected (Y), and immunized (Z) sub-populations. Parameter values used in this specific case are *X*₀ = 0.1, *Y*₀ = 0.02, *r* = 7 months, *β* = 12 per life span, *α* = 11.7 per life span, and *γ* = 0.15.
In the original model, there was no factor that would account for seasonal variation in habitat quality. As we describe above, this factor is included in our model using NDVI as a variable that modulates both $\alpha$ and $\tau$. When NDVI is included (Fig. 3), the host population does not evolve to a final stationary state. Instead, we obtain a cyclic pattern during the simulated 4-years period showing an annual oscillation with maxima during early Autumn (March, April). The total population pattern agrees with previously reported seasonal field peaks of *C. musculinus* (Crespo et al., 1970; Mills and Childs, 1998; Mills et al., 1992a,b).

The permanent infected rodent also show this seasonal pattern with 1 or 2 months delay compared with a non-infected sub-population and also the field infection distribution showed the highest values at the end of summer and autumn (Calderon paper in preparation).

Although NDVI, as well as $\alpha$ and $\tau$, have annual maximum in summer, the model produces maximum rodent numbers in Autumn, implying a lag effect between $\alpha$ or $\tau$ and $X+Y+Z$. If that delay is not taken into account, we cannot expect a significant correlation between NDVI and the total number of rodents.

![Graphs](image)
This effect can also be observed in Fig. 3, where NDVI curve for that period is compared with the modeled total population peaks.

Fig. 4a, presents the curve for the total rodent population corresponding to the previous simulation together with a curve for which the life span has been artificially reduced (maximum of 3.5 months) and maintaining its NDVI dependence in the equation. In contrast to the result obtained by Kirchner and Roy (1999) that showed a better fitness for the entire population, in our case, the total population was lower during the entire simulation.

In Fig. 4b, we have lowered NDVI values during the first year to simulate extreme environmental conditions such as a long and harsh winter (i.e., very low NDVI values) and a mild summer, which have been suggested to affect \( C. \) musculus densities. In this case, virus extinction occurs for almost 3

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**Fig. 5.** (a) Total population modeled by using averaged NDVI values from three localities (Maximo Paz, Pergamino, and Alcorta) compared with the average normalized trap success of the same places. Time is measured in months starting in January 1991. (b) Simulated (predicted) and captured total populations of \( C. \) musculus in Maximo Paz, Pergamino, and Alcorta. Trapping was conducted once per season and trap success was transformed to fraction of the carrying capacity (for more details see the text). The starting Y axis values were estimated by the mean field trap values of summer season for the all period (January 1991 to December 1994).
years before finally reappearing. Mathematically, virus recovered because "Y" never actually reached zero. In nature such a recovery might result from the introduction of virus from an adjacent population via immigration. About month 25 (i.e. the summer’s end of the second year) the model predicts a large number of non-infected *C. musculinus* but very few infected mice.

![Graph](image.png)

Fig. 6. (a) (Mills, 1992a; Fig. 1) Mean monthly trap success (number of captures of *Calomys musculinus* per 100 trap nights) and monthly number of confirmed cases of Argentine hemorrhagic fever (AHF) in southern Santa Fe, and northern Buenos Aires provinces, Argentina, March 1988–August 1990. (b) Total and infected sub-populations of a model run with a reproductive rate reduced to 0.6 of α. The input NDVI is an average of the values for the localities in the Argentine hemorrhagic fever endemic zone in order to achieve better agreement with field data reported in (a). Period March 1988–August 1990.
5. Comparative results

We attempted to evaluate the capability of our model, to describe the temporal and spatial variations of rodent populations monitored at several localities in the study area during 1991–1994. For each locality, the model was run using the corresponding pixel values of NDVI from NOAA decadal imagery during the simulation period and yield results as fractions of the carrying capacity. Field data are presented as total relative population densities derived from standardized trap success. These density values were normalized using the maximum trap success at each locality (from 1991 to 1994) as the carrying capacity value (equal to 1) to compare it with the modeled results.

Fig. 5a presents the normalized measured total population density and the simulated population density \((X + Y + Z)\) averaged for three localities: Máximo Paz, Alcorta, and Pergamino. Fig. 5b includes the measured and simulated data for each locality. Those localities were chosen because they were representative of the endemic area while presenting no anomalous patterns. A better agreement is observed with the mean curve than with individual curves. Nevertheless, some general aspects of our field data, as the seasonal variation in population dynamics with maximum values in the autumn, are accurately predicted by the model. A better fit of the model to field data might be obtained by exploring different algorithms for translating trap success to fraction of the carrying capacity.

Mills et al. (1992a, especially Fig. 1 therein which was inserted in this paper as Fig. 6a) and Mills and Childs (1998) provided data from a longitudinal study of *C. musculinus*, showing that both rodent populations and AHF cases increased in 1990. We applied our model to those data by running the model using mean NDVI data from a large portion of the endemic area from March 1988 to August 1990. Fig. 6b presents numerical results obtained with a model with a decreased reproductive rate \((0.6\text{ of its original value})\). Although there is not an absolute agreement, the model recovers the essential aspect of that field data: an increase of the total *C. musculinus* population during 1989 and the increase of infected mice some months later. It is possible that our original “\(i\)” values from laboratory data are overestimated due to optimal environmental conditions in the laboratory and because permanent contact between males and females was maintained in the laboratory.

6. Discussion and conclusions

We have presented a methodological approach based on a simple numerical model of rodent populations and viral infection that incorporates causality in the form of environmental information from remote sensing. The model is a useful tool for simulating hypothetical scenarios varying both environmental conditions and biological parameters of rodent populations. We selected NDVI as our indicator of environmental condition because it is readily available, biologically meaningful, and easily interpreted.

When immunized sub-population and seasonal variation is incorporated via NDVI, the theoretical conclusion of Kirchner and Roy (1999) regarding the advantage of having a shorter life span is not corroborated. In our model, when life span is artificially reduced while maintaining an association with NDVI, the total population was lower throughout the simulation. A more complete and accurate study of this effect could be done with a model that includes both population cohort structure and seasonal variation of population parameters.

In agreement with Mills et al. (1991) and De Vil-lafain et al. (1988), whom proposed a long and harsh winter as favorable condition to the extinction of the virus in nature; low NDVI values (such as might result from these climatic conditions) resulted in virus extinction.

These results demonstrate the utility of this kind of approach for studying and simulate population dynamics of rodents and its links with epidemiological aspect of the disease: because the natural infection with Junin virus is low (Mills et al., 1994), we used a very low proportion of carrying capacity as starting values for infected and non-infected rodents to represent the rates between each sub-population densities observed in field traps. The preliminary results of concordant with field trapping data are demonstrating seasonal variation with autumn peaks in rodent population densities. These seasonal changes are more clear in endemic AHF localities than in historic or surrounding places of the disease (Polop, personal communication) suggesting an important role of environmental condi-
tions in population dynamic and indirectly infection dynamic. The sole environmental variable in our model, NDVI, reaches maximum values in summer. NDVI, within the context of the structure of the mathematical model explained much of the variation in rodent population density and prevalence of viral infection. With some modification in population parameters, our attempts to model previously reported historic data (Mills et al., 1990a) provided results that were consistent with those data in terms of the variation in time of total population densities (Fig. 5). Also the proportion of permanent infected mice is quite in agreement with AFH cases (Fig. 3).

Nevertheless, several additional considerations might allow the improvement of our model. The NDVI decadal AVHRR product uses the maximum NDVI value in each 10 days period. In some cases, including the humid pampas agricultural landscape, rodent populations in winter could be more sensitive to minimum than to maximum values of NDVI. Because we are constrained by the period of available field data, we cannot use data from others satellites. In future studies, involving other field data sets, biological parameters may be modeled as a function of variables derived from other satellites with different temporal and spatial resolution (as EVI, enhanced vegetation index, from MODIS sensor; LAI leaf area index; SAVI soil adjusted vegetation index and GVI vegetation index derived from AVIRIS instrument). Additionally, in future studies, we will incorporate other variables related to soil moisture, land use patterns, and different vegetation types.

With respect to field measurements, our model overestimated some population peaks while underestimating others. These differences may be due limitations of the model as well as the way in which field data were transformed into fractions of carrying capacity. The derivation of fractions of the carrying capacity should be the topic of further study. The proposed functional dependence of rodent birth rate and longevity on NDVI should be carefully assessed, as should the possibility of incorporating the influence of NDVI on other biological parameters, either directly or through its effect on carrying capacity.

From the point of view of rodent-borne disease research, and as was pointed out by Mills and Childs (1998), remote sensors could play an important role in the development of predictive models of reservoir population dynamics. Toward that end, we have introduced what we feel is a useful causal approach to the application of satellite-derived information to problems relating to human diseases.

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