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Trypanosoma cruzi infection in dogs along the US-Mexico border: R_0 changes with vector species composition

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ABSTRACT

Infection with *Trypanosoma cruzi,* etiological agent of Chagas disease, is common in US government working dogs along the US-Mexico border. This 3145 km long border comprises four states: Texas (TX), New Mexico (NM), Arizona (AZ) and California (CA) with diverse ecosystems and several triatomine (a.k.a., kissing bug) species, primary vectors of *T. cruzi* in this region. The kissing bug (Heteroptera: Reduviidae) community ranging from CA to TX includes *Triatoma protracta* (Uhler), *Triatoma recurva* (Stål) and *Triatoma rubida* (Uhler) and becomes dominated by *Triatoma gerstaeckeri* Stål in TX. Here, we ask if *T. cruzi* infection dynamics in dogs varies along this border region, potentially reflecting changes in vector species and their vectorial capacity. Using reversible catalytic models of infection, where seropositivity can be lost, we estimated an R_0 (Estimate ± S.E.) of 1.192 ± 0.084 for TX and NM. In contrast, seropositivity decayed to zero as dogs aged in AZ and CA. These results suggest that dogs are likely infected by *T. cruzi* during their training in western TX, with a force of infection large enough for keeping R_0 above 1, i.e., the disease endemically established, in TX and NM. In AZ and CA, a lower force of infection, probably associated with different vector species communities and associated vectorial capacity and/or different lineages of *T. cruzi*, results in dogs decreasing their seropositivity with age.

1. Introduction

Chagas disease, caused by *Trypanosoma cruzi*, is a common vector-borne infection in humans, dogs and wildlife across the Americas (Gürtler and Yadon, 2015). Triatomine bugs (Heteroptera: Reduviidae: Triatominae), commonly referred to as 'kissing bugs', vector *T. cruzi* and require bloodmeals throughout all life stages (Lent and Wygodzinsky, 1979). *T. cruzi* is shed in the bugs' feces and infection can occur through introduction of infected feces into the bite site or mucous membrane, by the consumption of infected feces or whole infected bugs (Gerberg, 2008), and by congenital transmission (Carlier et al., 2015). Dog infections, or parasite exposure revealed by serology, have been reported in Argentina (Gürtler et al., 2006), Brazil (Dias et al., 2016), Paraguay (Fujita et al., 1994), Venezuela (Crisante et al., 2006), Colombia (Jaimes-Dueñez et al., 2017; Mesa-Arciniegas et al., 2018; Ramírez et al., 2013), Panamá (Fung et al., 2014; Pineda et al., 2011; Saldaña et al., 2015), Costa Rica (Montenegro et al., 2002), Nicaragua (Roegner et al., 2019), Mexico (Arce-Fonseca et al., 2017; Estrada-Franco et al., 2006) and the US (Beard et al., 2003; Elmayan et al., 2019; Kjos et al., 2008; Meyers et al., 2020; Williams et al., 1977). Several factors suggest dogs, among several mammalian species, are key reservoirs for *T. cruzi*, including that they are frequently fed upon by kissing bugs, commonly infected, achieve a *T. cruzi* parasitemia capable of infecting vectors, and share *T. cruzi* discrete typing units (DTUs, which are *T. cruzi* genotypes based on several distinctive molecular markers) with humans and vectors (Travi, 2018). Collectively, these studies confirm that dogs are not only viable sentinels of human disease (Fung et al., 2014; Gürtler et al., 2006; Tenney et al., 2014) but also suggest that Chagas disease control

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needs to consider dogs when designing and applying disease control strategies for human infections (Travi, 2018), a perspective also suggested by results from multi-host mathematical models (Bartsch et al., 2017; Collaborating Group on Chagas Disease Modeling, 2019; Flores-Ferrer et al., 2019; Lee et al., 2018).

In the US, *T. cruzi* infections in dogs are common in the southern states. *T. cruzi* has been diagnosed in shelter dogs (Elmayan et al., 2019; Hodo et al., 2019; Tenney et al., 2014), companion dogs in low-income rural communities (Curtis-Robles et al., 2017b), hound dogs (Barr et al., 1995; Curtis-Robles et al., 2017a), dogs living in national parks (Curtis-Robles et al., 2018c) and US government working dogs (Meyers et al., 2017; Meyers et al., 2020). This last population is of special interest given these dogs are highly trained and play a critical role in security along the US-Mexico border (Meyers et al., 2017). The US-Mexico border is 3145 km long, includes a wide range of ecosystems and spans four states: Texas (TX), New Mexico (NM), Arizona (AZ) and California (CA). In TX, and parts of NM border region *Triatoma gerstaeckeri* is present and has frequently been found with high proportions (near 100 %) of individuals infected with *T. cruzi*, even when outnumbered by other triatomine species (Curtis-Robles et al., 2018b; Kjos et al., 2009; Ramsey et al., 2015; Rodriguez et al., 2021; Wood and Wood, 1938; Wood and Wood, 1961), while elsewhere in NM, AZ and CA *T. protracta, T. recurva* and *T. rubida* are the dominant vectors (Behrens-Bradley et al., 2019; Curtis-Robles et al., 2018b; Klotz et al., 2014a; Martínez-Ibarra et al., 2012; Ramsey et al., 2015; Shender et al., 2016a; Wood, 1934). Although *T. gerstaeckeri* (Curtis-Robles et al., 2018a; Pippin, 1970; Wozniak et al., 2015), *T. protracta* (Curtis-Robles et al., 2018a; Wood, 1934; Wozniak et al., 2015), *T. recurva* (Behrens--Bradley et al., 2019; Klotz et al., 2014b) and *T. rubida* (Curtis-Robles et al., 2018a) have been commonly found infected with *T. cruzi*, several traits associated with vectorial capacity of kissing bugs, such as invasion of peridomestic environments, shorter defecation time, shorter feeding time and high probability of defecation when still bloodfeeding or shortly after engorgement, suggest that *T. gerstaeckeri* may be the most efficient vector of these four species. For example, *T. cruzi* infected *T. gerstaeckeri* was the most common species found in peridomestic kennels hosting US government dogs in TX (Meyers et al., 2017). Moreover, defecation times have been shown to be faster in *T. gerstaeckeri* (Pippin, 1970) when compared with *T. rubida, T. recurva* and *T. protracta* (Martínez-Ibarra et al., 2012). In addition, *T. gerstaeckeri* nymphs are more likely to defecate on hosts compared with nymphs from *T. rubida, T. recurva* and *T. protracta* (Martínez-Ibarra et al., 2012; Pippin, 1970; Reisenman et al., 2011). Even when outnumbered by other kissing bug species *T. gerstaeckeri* has been found with a higher parasite infection prevalence (Rodriguez et al., 2021). High parasitic prevalence in vectors is key for a more effective *T. cruzi* transmission (Cohen and Gürtler, 2001; Rabinovich et al., 1990), and more generally for vector-borne pathogen transmission, which increases with contact frequency between hosts and infected vectors (Dye, 1992; Massad and Coutinho, 2012). Contact can also be broadly defined, also including the ingestion of infected kissing bugs, which is assumed as the main route of *T. cruzi* transmission to dogs (Bradley et al., 2000; Kribs-Zaleta, 2006; Rowland et al., 2010; Saldaña et al., 2015). As kissing bug defecation still plays a major role for maintaining the *T. cruzi* life cycle, it might be expected that dogs will be more likely to get infected by *T. cruzi* in areas where *T. gerstaeckeri* is present when compared with areas where *T. protracta, T. recurva* and *T. rubida* are dominant vectors.

The higher vectorial capacity of *T. gerstaeckeri* might render the *T. cruzi* force of infection higher in regions where *T. gerstaeckeri* is present like TX and NM compared to areas dominated by *T. rubida, T. recurva and T. protracta* like AZ and CA. This hypothesis can be tested by comparing seroprevalence by age patterns in US Customs and Border Protection (CBP) working dogs, a population with restricted mobility associated with their service along the US-Mexico border (Meyers et al., 2017), and where major differences for *T. cruzi* exposure risk might be driven by regional differences in vector species composition and vectorial capacity. Our goal is to analyze cross sectional seroprevalence data for dogs stationed in TX, NM, AZ and CA using reversible autocatalytic models to estimate the force of infection and the antibody decay rate. We use these parameters, in addition to dog life expectancy estimates from vertical life tables (Chaves, 2018), to estimate the basic reproduction number, R_0, i.e, the number of new infections generated by an infected host in a susceptible population (Dietz, 1993). We expect R_0 to be higher in eastern areas of the US-Mexico border where *T. gerstaeckeri* is present than further west along the border given differences in vector species composition and vectorial capacity.

2. Materials and methods

2.1. Data

2.1.1. Study population

CBP working dogs are mainly Belgian Malinois and German Shepherds born in Europe and imported to the US as puppies. All dogs are trained for 3–6 months, mainly at a facility in El Paso, Texas, but some are also trained in Virginia. Following training, dogs are deployed to one of nine management areas along the border, and movement outside of their management area is limited (Meyers et al., 2017). Data were combined from two cross-sectional studies: the first was carried out between November 2015 and April 2016 to collect blood samples from dogs along the TX-NM border with Mexico (Meyers et al. 2017), and the second from March 2017 to May 2018 from dogs along the AZ-CA border with Mexico (Meyers et al., 2020). In both cross-sectional surveys around 60 % of the working dogs were sampled. Inclusion criteria were: (i) dogs 6 months or older, and (ii) to be in training or active duty. For each dog we recorded: age, breed and sex, among other variables that showed no significant association with *T. cruzi* infection (Meyers et al., 2017). Here it is important to stress that dogs have very restricted motion patterns once they are assigned to a border post, thus their seroprevalence patterns likely reflect their exposure to *T. cruzi* as mediated by the interaction with local vectors. For *T. cruzi* testing, at least 1 mL of blood was collected by venipuncture and aliquoted into no-additive and EDTA tubes. Samples from each dog were screened for anti-*T. cruzi* antibodies using the Chagas Stat-Pak rapid immunochromatographic test (ChemBio, NY) following the manufacturers protocol. Chagas STAT-PAK was designed for human use but has been validated in dogs (Nieto et al., 2009). Any band development was considered positive and band absence negative. Total sample size included 902 dogs which were separated by location into two groups; TX-NM ($n = 528$) and AZ-CA ($n = 374$) for analysis. These regions divide the border based on the presence/absence of *T. gerstaeckeri* and similar *T. cruzi* seroprevalence by age patterns. For example, seropositive samples at older ages, e.g., above 6 years, were more commonly observed in TX and NM but not in AZ and CA (Table S1). Table 1 shows the sex, age, breed, and *T. cruzi* seroprevalence for the two populations we modeled in this study.

2.2. Ethical clearance

All samples were collected in adherence with protocol number 2015–0289 approved by Texas A&M University's Institutional Animal Care and Use Committee on 08/17/2015. Written consent, from CBP personnel, was received for each dog included in this study.

3. Mathematical modeling

3.1. Autocatalytic model of infection

Infection seroprevalence commonly tends to monotonically increase with age in natural populations (Anderson and May, 1991; Dietz and Schenzle, 1985). The resulting curves have been widely analyzed with autocatalytic models (Awerbuch, 1994; Muench, 1959). These models study infection, or more precisely pathogen exposure since antibodies

Table 1
Age, sex, breed and *Trypanosoma cruzi* seroprevalence in US government working dogs along the border with Mexico.

Group	Age interval (years)	Sex		Breed			*T. cruzi* seroprevalence
		Male	Female	Belgian Malinois	German Shepherd	Other	
TX-NM	Below 2	55	25	43	29	8	11.25
	2–4	111	46	77	59	21	20.38
	4–6	72	37	55	34	20	18.35
	6–8	73	48	60	39	22	21.49
	Above 8	40	21	27	21	13	21.31
AZ-CA	Below 2	10	5	7	4	4	13.33
	2–4	105	39	69	55	20	4.17
	4–6	67	24	41	34	16	10.99
	6–8	50	35	28	40	17	2.35
	Above 8	25	14	19	13	7	2.56

(or serological evidence of exposure), not pathogens, are detected (Calzada et al., 2015). The force of infection is estimated as a function of the abundance of susceptible (seronegative) and seropositive (exposed) individuals in a host population (Anderson and May, 1991). Infections in catalytic models can be irreversible, an approach we have previously used to study cutaneous leishmaniasis in dogs (Calzada et al., 2015; Chaves, 2018), or reversible when individuals can recover from infection (Muench, 1959). For example, antibody titer can wane to levels below the detection threshold (Umezawa et al., 1996). The lack of seropositive saturation close to 100% for dogs of older ages (Table 1), suggests that either antibodies decay or some dogs are able to self-cure, a phenomenon that has been unambiguously shown in mice infected with *T. cruzi* (Tarleton, 2013) and, to some extent, in humans where seronegative conversions have been documented (Alvarez et al., 2012; Bertocchi et al., 2013; Dias et al., 2008; Olivera et al., 2010). Apparent self-cure has also been reported for dogs in Argentina (Castañera et al., 1998; Gürtler et al., 2006). Biologically, the lack of seropositive saturation could also be due to mortality by a disease commonly causing lethal acute infections at early ages, but in general Chagas disease in dogs is mostly a chronic disease (Andrade and Andrade, 1980). The simplest reversible catalytic model describing this type of dynamic is represented by the following non-linear partial differential equation:

$$\frac{\partial S(a,t)}{\partial t} + \frac{\partial S(a,t)}{\partial a} = \lambda(1 - S(a,t)) - \gamma S(a,t) \tag{1}$$

where S is the proportion of seropositive individuals, i.e., those diagnosed as exposed to a pathogen, based on a positive serological diagnostic test. Eq. (1) can be solved, as a function of age, denoted by a, at any given time t. For example, time t can represent the slice of time when a cross sectional epidemiological study was performed. The model in (1) assumes all hosts are born susceptible and become seropositive following pathogen exposure, which is a function of the force of infection (λ). The model also assumes λ is age independent, and that seropositive individuals can lose the marker, e.g., antibodies, used for diagnosis at a rate (γ). Solving Eq. (1) we find that (S) as function of age (a) is:

$$S_a = \frac{\lambda\left(1 - e^{-(\lambda+\gamma)a}\right)}{(\lambda+\gamma)} \tag{2}$$

Which allows the estimation of the force of infection (λ) and antibody decay rate (γ) by fitting seroprevalence estimates as function of age to Eq. (2). An interesting property of Eq. (2) is that as the host population ages, i.e., $a \to \infty$, the equation reaches a limit where seroprevalence asymptotically reaches a plateau:

$$S_{a\to\infty} = \frac{\lambda}{\lambda+\gamma} \tag{3}$$

This can be seen for the TX and NM population (Table 1) where values reach a near constant value after 2 years of age. By contrast in AZ and CA, infection seems to have two peaks, below 2 years and after 4 years, monotonically approaching 0 after those peaks. In that case we can assume that $\lambda = 0$ when solving Eq. (1) as function of age obtaining the following result:

$$S_a = S_0 e^{-\gamma a} \tag{4}$$

Where S_0 represents seroprevalence peaks before S asymptotically (when $a \to \infty$) becomes zero:

$$S_{a\to\infty} = 0 \tag{5}$$

3.2. Relaxing the assumption of an age independent force of infection

The force of infection for a pathogen can also be age-dependent, for example when individuals from a given age class are more likely to be exposed to a pathogen. Assuming a negligible antibody decay rate we can estimate an age-dependent force of infection, solving Eq. (1), with the following equation:

$$S_a = 1 - e^{-\int_0^a \lambda(a)} \tag{6}$$

In the absence of a functional form this equation can be solved by the numeric integration of a smooth function that can be obtained using smoothing splines (Bjørnstad, 2022). Smoothing splines are a non-parametric form of regression where the response, or dependent variable, can take any shape as long as it is continuous and the first and second derivative exist for all points within the range where the regression is fit (Faraway, 2006).

To test the robustness of assuming a constant, i.e., age independent, force of infection we fitted the model described by Eq. (6).

3.3. Inferring life expectancy and survival in dogs from cross sectional data

Life expectancy, e_0, is a parameter necessary to estimate the basic reproduction number of a disease, R_0, since it is necessary to know whether a host, on average, is more likely to die before being infected (Anderson and May, 1991). Life expectancy is estimated using the survival schedule function, l_a, which is a function of mortality (Carey, 2003). Life expectancy, e_0, is the expected time units a newborn host will survive until its death in the host population (Carey, 2001, 2003). e_0 is defined by the following equation:

$$e_0 = \sum_{a=0}^{\infty} l_a \tag{7}$$

Demographic theory has shown that a population age structure can reveal information about its mortality patterns, and this information can be used to estimate both l_a and e_0 (Keyfitz and Caswell, 2005). The method that estimates survival based on a population age structure is known as vertical life table, and data from cross-sectional studies are amenable to this type of analysis (Chaves, 2018). Vertical life tables can be used under two key assumptions: (i) that populations are at equilibrium, i.e., that populations have a size that does not considerably change through time and (ii) that the population structure is pyramidal,

meaning that there are more younger individuals than older individuals in the population (Krebs, 1998; Southwood, 1978). Both conditions are met by the dog populations we studied (Table 1), and are a common pattern in natural populations (Roach and Carey, 2014). Thus, for each given age class, which in this study is set to one year, we estimated the age specific period survival, p_a, as the ratio between the abundance of individuals (N_{a+1}) of age $a+1$ with respect to individuals of age a, (N_a):

$$p_a = \frac{N_{a+1}}{N_a} \tag{8}$$

Which can then be used to reconstruct l_a, the probability of surviving to age a (Carey, 2003), as follows:

$$l_{a+1} = p_a l_a \tag{9}$$

Where for the first age class, i.e., $a = 0$, we have:

$$l_0 = 1 \tag{10}$$

A common problem in the estimation of vertical life tables is that l_a needs to be a decreasing function of age, such that $l_{a+1} \leq l_a$. Therefore, if an age class $a+1$ has more individuals than age class a, the curve needs to be smoothed to obtain an appropriate $\widetilde{l_a}$ estimate (Chaves, 2018). Thus, the reconstructed l_a was smoothed using the lowess algorithm (Cleveland and Devlin, 1988):

$$\widetilde{l_a} = lowess(l_a) \tag{11}$$

The lowess smoothing also allows the estimation $\widetilde{l_a}$ for age classes with missing data, as was the case for age class 1 in TX and NM, and age classes 1 and 2 in AZ and CA (Table 1), given the condition presented by Eq. (9), which sets a maximum value for the l_a function, allowing the interpolation of $\widetilde{l_a}$ values. The raw outcome of the lowess smoothing is likely to include estimates falling outside the range from 0 to 1, where l_a is defined. This can be corrected using a standard procedure for transforming the range of probability functions (Ross, 2014), where the value for the oldest age is subtracted from all ages:

$$\widehat{l_a}' = \widetilde{l_a} - \widetilde{l_\infty} \tag{12}$$

then resulting values are divided by the smoothed estimate for age 0:

$$\widehat{l_a} = \widehat{l_a}' \Big/ \widetilde{l_0} \tag{13}$$

Where $\widehat{l_a}$ is a function whose range is between 1 and 0.

3.4. R0 estimation

R_0 is a parameter widely used to assess the likelihood of epidemic outbreaks or the endemic persistence of a pathogen in a population (Anderson and May, 1991; Dietz, 1993). Its value generally indicates the average number of new infections generated by an infection in a susceptible host population (Antonovics et al., 1995). For the transmission dynamics described by Eq. (1), R_0 also implies that a disease is endemically established in a population when its value is above 1. Here it is also worth commenting that in this study R_0 derived from Eq. (1) is an abstraction of the several biological processes involved in the transmission of a zoonotic vector borne disease. This means that the number of new *T. cruzi* infections in dogs will grow following the estimated R_0 under the implicit assumption of a steady and similar transmission dynamics between vectors, dogs and other reservoir species involved in the life cycle of *T. cruzi*.

For the model described in Eqs. 1 and 2, where the force of infection is constant and under the "weak homogeneous mixing" assumption (i.e., that new infections are a function of susceptible hosts), Anderson and May (1991) showed that R_0 is inversely proportional to the equilibrium fraction of susceptible hosts (1-S^*):

$$R_0 = \frac{1}{1 - S^*} \tag{14}$$

Following a procedure described in detail by Chaves (2018), where the proportion of susceptible hosts is equated to the age structured ratio of seronegative individuals over the age structured population, according to the following equation:

$$(1 - S^*) = \frac{\frac{N}{\lambda+\gamma}\sum_0^\infty \left(\gamma + \lambda e^{-(\lambda+\gamma)a}\right) l_a}{N \sum_0^\infty l_a} \tag{15}$$

We have that R_0 is related with parameters from Eq. 2, and the host population life expectancy, e_0, and survival schedule, l_a, by the following equation:

$$R_0 = \frac{e_0(\lambda + \gamma)}{e_0\gamma + \lambda \sum_{a=0}^{\infty} e^{-(\lambda+\gamma)a} l_a} \tag{16}$$

In Eqs. (15) and (16) the assumption that susceptible hosts are only those seronegative does not imply the absence of superinfections (Aron, 1983), i.e., the existence of new infections in seropositive hosts, something observed in parasitic infections. In a model like the one described in (2) superinfection could be reflected by a decrease in the antibody decay rate (γ) as antibody production gets boosted by repeated exposure where superinfections occur (Alonso et al., 2019; Aron, 1988a, b).

4. Software and parameter estimation procedures

All parameter estimations were made with the R language for statistical computing version 3.6.1 (R Core Team, 2019).

Models presented in Eqs. (2), (4), and (6) were fitted, respectively, to data from TX-NM and AZ-CA, employing maximum likelihood functions (Bolker, 2008) presented in the supplementary online material S1. The maximum likelihood models that we fitted assumed a normal error:

$$Error \sim N(0, \sigma^2) \tag{17}$$

where σ is the standard deviation of the error.

To fit the models, we organized the databases according to dog age and divided each one in 16 quantiles, a number large enough to warrant prevalence estimates based on at least 23 dogs in each quantile. For each age quantile we estimated the average age and seroprevalence. For the TX-NM data, the resulting 16 data points, plus the origin, were used to fit the model of Eq. (2) and Eq. (6). We compared these models using the Akaike Information Criterion, AIC, a metric for model selection based on likelihood maximization and parameter minimization (Faraway, 2004). For the AZ-CA data, we fitted a model for the full dataset, only considering the 16 data points from the quantiles, and compared its AIC with a model that fitted separated curves for individuals below and above 3.85 years of age.

To test for differences in dog breed proportions between the TX-NM and the AZ-CA populations, as function of age, we employed G tests. These tests are maximum likelihood ratio tests whose null hypothesis is that proportions are the same between the samples compared, and have an approximate chi square distribution (Sokal and Rohlf, 1994).

Smoothed survival curves were estimated employing the command "lowess" (Cleveland and Devlin, 1988) with a smoother span of 2/3, 3 iterations per local fit, and local fits covering 1/100 of the age range.

The R_0 estimate for TX-NM had its error estimated using the partial derivative method (Ku, 1966), based on the errors previously estimated for e_0, λ and γ. We did not estimate R_0 for AZ-CA given that, by definition as presented in Eq. (3), no new infections are generated in AZ-CA and R_0 is constrained to be below 1.

5. Results

In accordance with patterns recorded in Table 1, *T. cruzi* seroprevalence by age curves in dogs from TX-NM (Fig. 1A) and AZ-CA (Fig. 1B) were very different. In TX-NM seroprevalence monotonically increased with age (Fig. 1A), while in AZ-CA seroprevalence simply decreased with age, but had two peaks—one at 2.00 years and one at 3.85 years (Fig. 1B). Parameter estimates for the autocatalytic model fitted to the TX-NM data are presented in Table 2, where it is worth highlighting that the force of infection ($\widehat{\lambda}$) is smaller than the antibody decay rate ($\widehat{\gamma}$). Nevertheless, seroprevalence reached a plateau of 20 % as the dog population aged, suggesting *T. cruzi* is endemically established in the CBP working dog population of TX-NM. The model assuming a constant force of infection (AIC= −53.98) outperformed the model assuming an age-dependent force of infection (AIC = −52.62, the smoothed age dependent function is presented in Fig. S1) and, qualitatively, the prevalence fit was very similar to the one assuming at age-independent force of infection (Fig. S2). These results suggest that assuming a constant force of infection is a sound assumption.

Table 3 shows two sets of parameter estimates for the AZ-CA curves. In Table 3, we only present results for the model with two set of estimates given the AIC of the model with a breakpoint (AIC = −49.03) at 3.85 years of age outperformed the model without a breakpoint (AIC = −29.91). Table 3 shows the initial seropositive fraction ($\widehat{S_0}$) was larger for the older dog group, those aged 3.85 years or older, than for the younger dogs (below 3.85 years Table 3). The estimated initial seropositive fractions ($\widehat{S_0}$) for the age interval starting at 2 years in AZ-CA was similar to the estimate for that age in the TX-NM population (Fig. 1A). Similarly, for the age interval starting at 3.85, *T. cruzi* seroprevalence also coincided with the TX-NM population (Fig. 1A). Both seroprevalence estimates are around 20 %. In the AZ-CA population the antibody decay rate ($\widehat{\gamma}$) decreased with age (Table 3), a result suggesting that younger dogs remain seropositive for a shorter time than older dogs. This pattern might reflect dog population breed composition, which was significantly different when comparing dogs below and above 4 years (Table 1). For dogs below 4 years of age the proportion by breed was similar in both the TX-NM and AZ-CA populations (G = 0.72348, d.f. = 2, P-value = 0.6965), but for dogs that were 4 years or older, the breed proportions were significantly different (G = 27.593, d.f. = 2, P-value = 1.019e-06). Indeed, the AZ population proportionally had more German Shepherds than Belgian Malinois in age groups above 4 years when compared with the TX-NM population (Table 1).

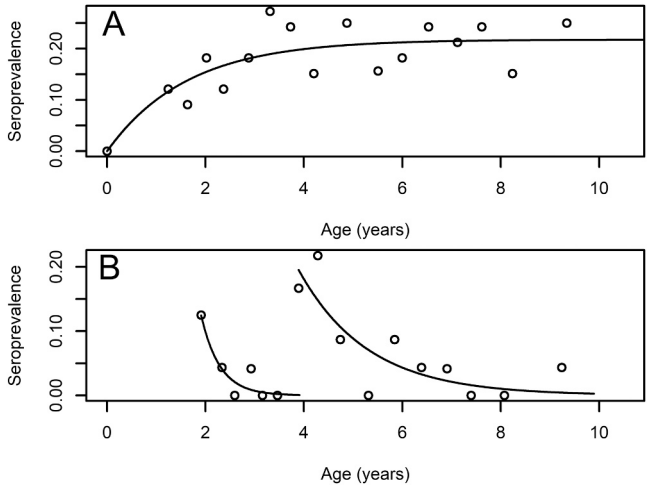

Fig. 1. Age specific *Trypanosoma cruzi* seroprevalence in US government working dogs. Seroprevalence by age curves for dogs from (A) Texas and New Mexico, the curve is based on parameters presented in Table 2 (B) Arizona and California, the curves are based on parameters presented in Table 3.

Table 2
Maximum likelihood parameter estimates for the reversible autocatalytic model explaining *Trypanosoma cruzi* seroprevalence by age in US government working dogs from Texas and New Mexico.

Parameter	Estimate	95% CL	S.E.	Z	P-value
Force of Infection ($\widehat{\lambda}$)	0.135	0.084–0.253	0.033	4.070	4.71E-05*
Antibody decay rate ($\widehat{\gamma}$)	0.483	0.249–1.018	0.150	3.216	0.0013*
Error standard deviation ($\widehat{\sigma}$)	0.041	-	-	-	-

*Statistically significant (P < 0.05)

The smoothed survival curves can be seen in Fig. 2. In both TX-NM (Fig. 2A) and AZ-CA (Fig. 2B) populations survival decreased, in a nearly linear fashion, with age. As shown in Table 4 life expectancy for both populations was similar, being, on average, slightly longer for TX-NM than AZ-CA. The value of R_0 above 1 for the TX-NM population confirms that *T. cruzi* is endemically established in that population (Table 4).

6. Discussion

Our modeling clearly shows that *T. cruzi* infection dynamics in US government working dogs differ between TX-NM and AZ-CA. The R_0 estimate for TX-NM is above 1 and indicates that *T. cruzi* infection is endemically established in these dogs, while a contrasting pattern was observed in dogs from AZ-CA region, where seropositivity decreased as dogs aged. A plausible explanation for this difference is that in TX-NM the force of infection is higher because *T. gerstaeckeri*, the more dominant vector species in TX and present in NM (Curtis-Robles et al., 2018b; Kjos et al., 2009; Ramsey et al., 2015; Rodriguez et al., 2021; Wood and Wood, 1938; Wood and Wood, 1961), likely has a higher vectorial capacity than *T. protracta, T. recurva* and *T. rubida*, the dominant vector species in AZ and CA (Behrens-Bradley et al., 2019; Curtis-Robles et al., 2018b; Klotz et al., 2014a; Martínez-Ibarra et al., 2012; Ramsey et al., 2015; Shender et al., 2016a; Wood, 1934; Wood and Wood, 1938). The higher vectorial capacity could be derived from a shorter feeding and defecation time by both nymphs and adults of both sexes (Pippin, 1970), a pattern contrasting with *T. protracta, T. recurva* and *T. rubida*, where nymphs, and *T. rubida* adult males, are less likely to defecate soon after feeding (Martínez-Ibarra et al., 2012; Reisenman et al., 2011). This vectorial capacity-based inference is made under the assumption that transmission mainly occurs via defecation from vectors. However, differences in dominant vector species might still account for the observed differences assuming transmission is primarily oral, via the ingestion of infected bugs, a phenomenon commonly reported for dogs and other mammals (Gerberg, 2008; Rowland et al., 2010; Saldaña et al., 2015). *T. gerstaeckeri* is a species that commonly invades dog kennels and working stations, as inferred by the high frequency of this species being encountered by personnel caring for US government working dogs in TX (Meyers et al., 2017). *T. gerstaeckeri* also has a documented behavior where invading man made habitats is common (Kjos et al., 2009; Wozniak et al., 2015). In contrast, *T. protracta, T. recurva* and *T. rubida,* although able to invade houses and man-made habitats (Reisenman et al., 2014), likely mainly thrive within more ecologically restricted habitats, such as rodent nests (Ryckman, 1986; Wood, 1934; Wood and Wood, 1938, 1961). This pattern is reinforced by kissing bug bloodmeal composition, as only *T. rubida* and *T. protracta* adults have been shown to feed on humans and dogs (Stevens et al., 2012), unlike what has been observed for *T. gerstaeckeri* where both adults and nymphs have been shown to feed on humans and dogs (Kjos et al., 2013). Here, we also want to highlight that although most of the transmission could be due to a few dominant vectors, in nature vector borne disease transmission can be related to the community of vectors, as has been described for sand

Table 3
Maximum likelihood parameter estimates for the best model explaining *Trypanosoma cruzi* seroprevalence decrease with age in US government working dogs from Arizona and California.

Age	Parameter	Estimate	95 % CL	S.E.	Z	P-value
< 3.85 years	Initial seropositive fraction ($\widehat{S_0}$)	0.124	0.087–0.162	0.016	7.713	1.23E-14*
	Antibody decay rate ($\widehat{\gamma}$)	2.613	1.400–6.364	0.723	3.614	0.00030*
	Error standard deviation ($\widehat{\sigma}$)	0.016	0.010–0.032	0.005	3.450	0.00056*
≥ 3.85 years	Initial seropositive fraction ($\widehat{S_0}$)	0.195	0.122–0.274	0.034	5.671	1.42E-08*
	Antibody decay rate ($\widehat{\gamma}$)	0.714	0.298–1.484	0.236	3.026	0.0025*
	Error standard deviation ($\widehat{\sigma}$)	0.041	0.027–0.073	0.010	3.997	6.41E-05*

* Statistically significant ($P < 0.05$)

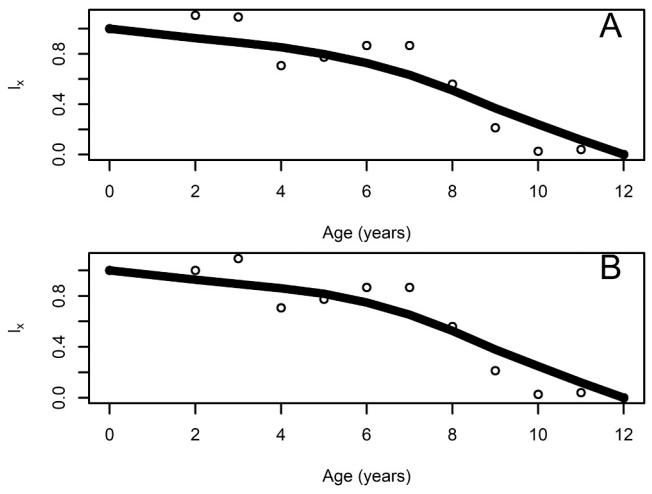

Fig. 2. Age specific survival in US government working dogs. Data for dogs from (A) Texas and New Mexico (B) Arizona and California. In panels (A) and (B) solid lines are the smoothed estimates obtained with the lowess method applied to the vertical life table survival estimates represented by the open circles.

Table 4
Life expectancy, e_0, and *Trypanosoma cruzi* basic reproduction number, R_0, in US government working dogs from Texas & New Mexico (TX-NM) and Arizona & California (AZ-CA). All parameters are indicated as the estimate ± S.E.

Parameter	TX-NM	AZ-CA
e_0	7.887 ± 0.039	7.870 ± 0.038
R_0	1.192 ± 0.084	Constrained to be below 1

fly (Diptera: Psychodidae) vectors of leishmaniasis (Chaves and Añez, 2004, 2016; Rigg et al., 2019a; Rigg et al., 2021) and mosquito (Diptera: Culicidae) vectors of arboviruses (Chaves et al., 2011; McMillan et al., 2020; Petruff et al., 2020; Reisen et al., 1992) and malaria parasites (Hurtado et al., 2018b; Iwashita et al., 2014; Rigg et al., 2019b). For example, in Texas there are at least seven kissing bug species all of which are vectorially competent to transmit *T. cruzi* (Curtis-Robles et al., 2018b). Nevertheless the most abundant, also having traits that potentially increase *T. cruzi* transmission is *T. gerstaeckeri* (Curtis-Robles et al., 2018a,b). Thus, further integration of carefully designed field observations with modeling studies are necessary to establish how a differential vector species composition translates into patterns like the ones we describe in this study, where reference to dominant vectors across the borders actually might represent changes in species composition that we could not study with the currently available data.

Life expectancy was very similar in dogs from both TX-NM and AZ-CA, being close to 8 years, implying dog demography was an unlikely factor affecting *T. cruzi* transmission dynamics in these two populations. However, the approximate 8 years life expectancy more than doubles what we had estimated for mixed breed dogs in a rural community from Panamá (Chaves, 2018). This situation seems critical for the endemic establishment of *T. cruzi* infection in dogs from TX-NM, whose R_0 might go below 1 if dogs died younger. For example, the low force of infection we observed in TX-NM, combined with the high mortality pattern we inferred using vertical life tables in dogs from rural Panamá (Calzada et al., 2015), would imply an average dog is more likely to die before being exposed to *T. cruzi,* a pattern that would result in the disease eventually dying out in the host population (Anderson and May, 1991; Dietz, 1993).

The peaks in *T. cruzi* seroprevalence observed around 2 and 4 years in AZ-CA dogs likely represent infections acquired at the training center in El Paso, western Texas, and their history of *T. cruzi* exposure prior to being deployed to AZ and CA. In principle, this may suggest that both populations had similar patterns of *T. cruzi* exposure, before the AZ-CA population started to live in a region with a lower, perhaps insignificant, force of infection. An interesting phenomenon observed in AZ and CA was the change in antibody decay rate, which was almost 4 times faster in younger (below 3.85 years) than older dogs (3.85 years or older). This might be related to dog breeds given that proportionally German Shepherds were more common among older AZ-CA dogs. Another possibility for this observation of a faster antibody decay rate in younger dogs is that younger working dogs, especially below 1 year of age, may be more likely to die from an acute *T. cruzi* infection than older dogs (Kjos et al., 2008), something that our model cannot test, given that our model implicitly assumes no age-specific variation in mortality associated with *T. cruzi* infection. However, we want to highlight that this assumption about mortality patterns in dogs is partially supported by ontologically independent lines of evidence. First, as we already mentioned there is evidence suggesting self-cure in dogs (Castañera et al., 1998; Gürtler et al., 2006) and seronegative conversions have been observed in other mammal hosts (Tarleton, 2013). Fatal Chagas myocarditis is a common pathology in US working dogs that is only noticed after the sudden death of dogs that otherwise seemed healthy but had chronic infections (Meyers et al., 2021). More generally Chagas disease is a chronic infection in dogs, where mortality is observed at older ages and lethal pathologies, in general, develop with time (Andrade and Andrade, 1980; Andrade et al., 1981; Meyers et al., 2021). At older ages, given multiple competing mortality risks, removing a specific cause for mortality has little impact on overall survival (Carey, 1989). This, in principle, supports that assuming a negligible increase in mortality due to *T. cruzi* infection is an appropriate assumption for modeling mortality in dogs exposed to *T. cruzi*.

Our observations, however, warrant further research, given that we have previously observed that German Shepherds have a lower seroprevalence than Belgian Malinois, across all ages, in US government working dogs from TX (Meyers et al., 2017). However, our modeling results suggest that when German Shepherds are more common in the dog population, the dog population tends to be seropositive over a longer time period, given the seropositive period is the inverse of the antibody decay rate (Anderson and May, 1991). Understanding the influence of dog breed gets further complicated by the age difference in the two groups, but definitely highlights the possibility that dog breed is

a factor likely influencing the risk for *T. cruzi* exposure. Thus, it is not clear if the mechanism is related to dog breed behavior, for example, different probability of ingesting kissing bugs or related to breeds having a differential immune response when challenged by *T. cruzi* parasites, or just a result of changes in the antibody decay rate related with dog age. This question can be answered by performing behavioral experiments offering engorged/non-engorged kissing bugs to different dog breeds and ages, or longitudinally examining differences in the expression of immune molecular markers associated with anti-*T. cruzi* immune responses (Meyers et al., 2017). Similarly, the disparity in estimates for antibody decay rates is suggestive that superinfections might occur in Chagas disease in dogs, as the antibody decay rate was smaller in TX-NM when compared with any of the two estimates from AZ-CA, and this is something to be expected when superinfections happen (Aron, 1983).

Additional factors that require further research regarding the patterns we report here, and that might act in conjunction with regional differences in vectorial capacity that reflect vector species composition along the US-Mexico border shaping a differential *T. cruzi* exposure risk, are differences in wildlife reservoirs and circulating *T. cruzi* genotypes (Discrete Typing Units [DTUs]). While some of the wildlife reservoirs are common across the US-Mexico border, e.g., raccoons, *Procyon lotor* (Hodo et al., 2020), and Virginia opossum, *Didelphis virginiana* (Zecca et al., 2020) some might have a more restricted distribution such as the white-throated woodrat, *Neotoma albigula*, which is considered a primary host for triatomines in Arizona (Behrens-Bradley et al., 2019; Stevens et al., 2012) and California (Orin, 2011; Ryckman, 1986; Shender et al., 2016a; Shender et al., 2016b). Since host associations with DTUs have been found, this might influence the composition of circulating DTUs, probably also modulating dog immune response, pathology, or even dog infection probability, to circulating *T. cruzi* parasites (Curtis-Robles et al., 2016; Hodo and Hamer, 2017; Jansen et al., 2015; Hodo et al., 2020; Ramirez et al. 2013). Similarly, our analyses of DTUs, a common system to classify *T. cruzi* genotypes (Cura et al., 2015; Zingales et al., 2012), along the border suggests, based on data from kissing bugs, that while TcI, TcIV and mixed TcI/TcIV lineages are common in TX and NM, the DTU composition in AZ and CA might be different, given that only a few samples from AZ were classified as TcI (Curtis-Robles et al., 2018a), and samples from California are dominated by TcI (Brenière et al., 2016; Shender et al., 2016b). In the three US government working dogs, from TX and NM, where *T. cruzi* parasites were typed one had a TcIV infection and the rest mixed TcI/TcIV infection (Meyers et al., 2017). This preliminary data is suggestive about potential differences in the response of dogs to *T. cruzi* infections when TcIV is present in a population. A fully open question is whether the substructure of TcI (Zingales et al., 2012), the most commonly detected DTU in triatomines in the southern USA (Curtis-Robles et al., 2018a), might drive the differences we report here. As more *T. cruzi* samples from AZ-CA are typed into DTUs it will become more clear, if beyond dominant vector species and/or vector species composition differences, parasite genotypes also play a role shaping the contrasting *T. cruzi* infection dynamics we describe here.

Similarly, a case could be made to study potential changes in the force of infection related to changes in environmental conditions. In principle this might explain some of the variability around the force of infection curves, as it is common for vector-borne diseases to have interannual fluctuations related to global climatic phenomena like the El Niño Southern Oscillation, ENSO (Hurtado et al., 2014; Hurtado et al., 2018a). Some of these cycles have been linked to changes in abundance and infection of vectors (Chaves et al., 2014; Poh et al., 2019). That type of variability might be an alternative hypothesis that could be tested to explain differences between seroprevalence peaks above and below 3.78 years of age in AZ-CA, where transmission in years where environmental conditions exacerbate transmission can lead to differences in exposure and seroprevalence linked with different age cohorts. The spline based approach that we used to estimate an age dependent force of infection can potentially be used to measure associations between the force of infection and interannual large scale climatic phenomena like ENSO.

Finally, as extensively argued by Travi (2018), dogs are not only a major link to understanding and managing the eco-epidemiology of *T. cruzi* infection as a zoonotic disease with implications for human infections, but also serve a potential role to aid in the control of Chagas disease. Dogs serve many important functions as companion animals and working dogs provide diverse services to society, thus being ubiquitous in the human environment. Moreover, working dog training is costly and maximizing their life span is also desirable from an economic perspective (Curtis-Robles et al., 2017a; Meyers et al., 2017). Accordingly, host-targeted insecticides (Busselman et al., 2023; Reithinger et al., 2005; Travi, 2018) is one example of how dogs could help reduce triatomine populations and limit *T. cruzi* transmission in dogs and humans.

CRediT authorship contribution statement

Study Design: **LFC, ACM, GLH, SAH**; Model Development: **LFC**; Funding acquisition: **SAH, LFC**, First draft preparation: **LFC, ACM**; Data Collection and Management: **ACM, RCR, JPS, GLH, SAH**, Other Resources: **CH, RCR, JPS, GLH**; Draft comments and edits: All Authors.

Declaration of Competing Interest

All authors declare that no conflict of interest exist.

Data availability

The raw data cannot be shared given its sensitivity for US National Security. The code used to fit the models is included as a supplement.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.epidem.2023.100723.

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