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Microparasite species richness in rodents is higher at lower latitudes and is associated with reduced litter size

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Parasite species loads are expected to be higher in the tropics and higher parasite species richness to have cumulative effects on host physiology or demography. Despite being regularly assumed or predicted, empirical evidence on species–latitude patterns is scarce or contradictory and studies on the impacts of concomitant infections have mainly been done at host intra-specific level. Broad generalizations are then very hard, if not spurious.

By focusing on rodent species and their non-eukaryotic microparasites (i.e. viruses and bacteria), we investigated, using a comparative approach, microparasite species richness across rodent species according to the latitude where they occur. We also explored the links between rodents' reproductive traits, latitude and microparasite species richness.

We find for the first time in rodents that virus species richness increases towards tropical latitudes, and that rodent litter size seems to decrease when microparasite species richness increases independently from the latitude. These results support the hypotheses that rodent species in the tropics effectively harbour higher parasite species loads, at least in terms of species richness for viruses, and that parasite species richness influences rodent life-history traits. Although some other factors, such as seasonality, were not taken into account due the lack of data, our study stresses the idea that chronic microparasite infections may have detrimental effects on their rodent host reservoirs, notably by affecting litter size.

A recent meta-analysis suggested that parasite-associated mortality may be higher at tropical latitudes for a broad range of animals, including arthropods, molluscs, amphibians, birds, fishes and mammals (Robar et al. 2010). Interestingly, one explanation was related to the 'enhanced incidence of multiple infections', at tropical latitudes 'which tends to favour the evolution of more virulent parasites'. In the literature, multiple infections refer to simultaneous (i.e. co-infections) or sequential infections of hosts by different parasite species (or clones of the same parasite species) at the individual, population or species level.

The study by Robar et al. (2010) raises two important points. The first one is related to the predicted higher parasitic species loads at tropical latitudes. The second one concerns the expected effects of multiple infections. Broadly speaking, the assumption of Robar et al. (2010) is based on an expected link between multiple infections at tropical latitudes and their enhanced effects on the host's demography. Patterns of multiple infections per se across host species (i.e. at interspecific level) have not been well investigated to date and few data are still available in the literature. On the contrary, parasite species richness, which is related to the number of parasite species encountered at various geographical scales and prone to infect a host at individual, population and species level is a metric regularly investigated across host species. Then, if multiple infections imply co-infections and differ to parasite species richness per se, we can make the assumption that higher parasite species richness may increase the occurrence of co-infections, simply due to higher availability of parasite species. Unfortunately, to date, we are not aware of any study that links higher parasite species richness at tropical latitudes with stronger impacts on reproduction or survival in vertebrates, except in humans (Guégan et al. 2001; but see also Møller 1998 for the potential major parasitized-related impacts on bird immune systems in the tropics).

Concerning the first point, the probability to be infected by a higher number of parasite species at tropical latitudes seems a priori rather straightforward given that parasite species richness is expected to follow the same latitudinal gradients as observed for free-living species richness (Poulin 1995, Nunn et al. 2005). The main mechanism to explain higher parasite species richness classically refers to the particular biotic and abiotic conditions that prevail within tropical environments (Guernier et al. 2004, Nunn et al. 2005). Indeed, higher parasite mortalities as a result of harsher conditions far from the equator are expected to be a strong constraint for parasite transmission at higher latitudes. This may affect both free parasitic infective stages and intermediate hosts or vector-borne arthropods, which are largely implicated in the

transmission of major parasites in the tropics. However, and despite the often claimed or 'predicted' findings, comparative studies about the parasitic loads in the tropics and in temperate zones are still scarce and have given discrepant results. Focusing on mammals, a well-studied host group for parasites, only one study succeeded in finding a strong positive correlation between proximity to the Equator and total parasite species richness in humans (Guernier et al. 2004). Lindenfors et al. (2007) and Krasnov et al. (2004) found just the opposite for helminths and carnivores and for fleas and rodents, respectively. Nunn et al. (2005) concluded that parasite species richness increases toward lower latitudes in primates only for protozoans, but not for viruses or helminths. Finally, across mammalian species, Poulin (1995) and Bordes et al. (2010) did not find any correlation between helminth species richness at intra- or inter-specific levels and latitude. Helminth species richness was however higher in the Afro-Ethiopian area than in the Neartic and Paleartic ones (Bordes et al. 2010).

Considering now the second point, which concerns the effects of multiple parasitic infections, we are also aware that polyparasitism is rarely considered as a parasitic pressure per se (reviewed by Bordes and Morand 2009a). The majority of studies still focus on single host/single parasite systems despite the substantial empirical evidences that polyparasitism and co-infections are the rule in natural ecosystems (Mc Kenzie 2005, Pullan and Brooker 2008). Consequently, studies related to the effect of multiple infections are still scarce. Moreover, the few studies about polyparasitism effects on host mortality or host reproduction have all been done at intraspecific level (Lello et al. 2005, Davidar and Murton 2006, Jolles et al. 2008, Munson et al. 2008). At the interspecific level, comparative studies used data on parasite species richness due to the lack of data on co-infections (e.g. Šimková et al. 2008 for the effect of parasite species richness on reproduction across fish species).

Therefore, the aims of our study were to fill these two gaps by exploring: 1) the existence, or not, of higher parasite species richness at tropical latitudes; and 2) the related effects of higher parasite species richness on the host demography across host species.

Specifically, we focused on rodents and their non-eukaryote microparasites (i.e. viruses and bacteria) due to the abundant literature available for these two parasitic taxa and also because there is empirical evidence, at intraspecific level, that viruses and bacteria may affect the demography of their mammal host species (Jolles et al. 2005), and more specifically of rodents (Kallio et al. 2007, Burthe et al. 2008). Moreover, we are not aware of any previous comparative study that has investigated the latitudinal effects on microparasite species diversity in this host group despite their strong potential implications as reservoirs of major zoonotic agents (Meerburg et al. 2009).

Material and methods

Virus and bacteria screened

A preliminary review of the literature (notably Piesman and Gern 2004 and Meerburg et al. 2009) allowed us to identify groups/families of microparasites with broad geographical distribution across the world and/or large repartition inside a biogeographic realm. We then retained three virus groups (Hantaviruses (genus *Hantavirus*, family Bunyaviridae, which includes the Sin Nombre, Puumala and Hantaan viruses), Arenaviruses (genus *Arenavirus,* family Arenaviridae, which includes the Lassa fever virus and Lymphocytic choriomeningitis virus) and Poxviruses (genus *Orthopoxvirus*, family Poxviridae)) and five bacteria, the *Borrelia* spp. (agents of Borreliosis in Paleartic and Neartic areas, i.e. Lyme disease), *Bartonella* spp. (agents of widespread infections in diverse mammalian species around the world), *Leptospira* spp. (agents of Leptopsirosis around the world), *Orientia tsustsugamushi* (agent of Scrub typhus in Asia) and *Yersinia pestis* (agent of plague).

Data on microparasites

The purpose of our study was to investigate the existence of latitudinal gradients in microparasite species richness of host species (i.e. the influence of the latitude on the number of microparasite species encountered in the host species for a given rodent species). We compiled data from published literature by searching the Science Citation Index for the years 1965–2010 with various combinations of keywords: Hantavirus* (or the other viruses or bacteria cited above) or microparasite* or pathogen* and rodent* or small mammal* or mammal*. Additional references, which were not identified during search with keywords, were collected from the quoted literature. The list of collected published citations is put in Supplementary material Appendix A1. Typically, the information extracted from published studies concerned the number of all rodent species examined for a given pathogen and the circulation (i.e. presence) of this pathogen among individual hosts, inferred through serological tests and/or pathogen isolation from different organs and/or molecular detection. If no information was clearly available on the host sample size we did not retain the reference. Taxonomy and phylogeny of rodents follow Binida-Emonds et al. 2007.

We could collect information on 107 different rodent species that belong to eight families (Caviidae, Cricetidae, Dipodidae, Geomydae, Heteromyidae, Muridae, Nesomyidae and Sciuridae) and live in five different biogeographic realms (i.e. Neartic, Neotropical, Paleartic, Afro-Ethiopian and Oriental) (see Supplementary material Appendix A2 for a list of the rodent species with data on bacteria and virus richness). Twenty-three species were found in the Southern Hemisphere and 84 species in the Northern Hemisphere.

Data on host rodent species

We estimated the mean latitudinal range and range of latitude of each rodent species according to the geographic distribution maps extracted from the IUCN Red List database (IUCN 2010). In our study, geographical areas ranged from 28°S to 59°N. Information on host demography across rodent species was obtained from Ernest (2003). We gathered data on host species body mass and litter size. We did not include data on host longevity as they were too scarce and mainly obtained from animals in captivity. These life-history data are also included in Supplementary material Appendix A2.

Confounding variables

Many previous studies established that parasite species richness in mammals might be affected not only by the host sample size, but also by its phylogeny, body size and various other ecological traits (Poulin 2007, Ezenwa et al. 2006, Bordes et al. 2009, 2010). Thus, in order to investigate latitudinal gradients, and following previous studies (Nunn et al. 2005, Lindenfors et al. 2007, Bordes et al. 2010), we controlled the host sample size and host phylogeny as confounding variables.

To address the problem of host sampling effort, we included measures of sampling effort as covariates of multiple statistical analyses. A first measure of sampling effort was the number of host individuals investigated (i.e. screened). However, and unfortunately, not all rodent species were screened for all microparasite species retained (Supplementary material Appendix A1) as the detection of microparasites depends on the use of specific tools (serology, isolation or molecular identification). The number of microparasite species detected in a given host species depends on both the screening effort (number of screening tests) and the host sampling size (number of individual hosts screened). Microparasite species richness may be biased by these both parameters, and thus we used both variables as two covariates in statistical analyses. Microparasite species richness may also depend on the geographical distribution of hosts.

We then included latitudinal range of host distribution as potential determinant of microparasite species richness.

Comparative analyses

We tested whether the investigated variables showed evidence of phylogenetic signal based on the parameter λ (Pagel 1999, Freckleton et al. 2002). We used R (R Development Core Team 2008) packages APE and GEIGER (Paradis et al. 2004). Values of parameter λ were compared between the real phylogeny and a star phylogeny (i.e. polytomic tree without phylogenetic structure).

As λ was significantly different from 0.0 for rodent body mass, we used the computer program CAIC (Purvis and Rambaut 1995), as the phylogeny of the rodents investigated is not fully resolved (which is an imperative when using the package APE). We used information on rodent phylogeny from Binida-Emonds et al. (2007) and we calculated the independent contrasts with the computer program CAIC (Purvis and Rambaut 1995). To confirm the proper standardization of contrasts, we regressed the absolute values of standardized contrasts against their standard deviations. We found no significant relationships, suggesting that it was not necessary to transform branch lengths before computing standard deviations (Garland et al. 1992). Contrasts were then analyzed using standard multiple regressions, with all intercepts forced through the origin (Garland et al. 1992).

In order to investigate a potential link between litter size and microparasite species richness, we controlled for sampling effort and phylogeny but also for latitude and host body size as these two variables may strongly affect host reproductive traits (Hayssen 2008).

Determinants of microparasite species richness

We first performed standard multiple regressions using independent contrasts, with the intercept forced to be zero, with microparasite species richness as dependent variable and number of host samples, number of screening tests, mean latitude, range of latitudinal variation and host body mass as independent variables.

We second selected the best subset selection of variables using Mallows' statistic. Mallow's Cp. This statistics helps to find the best model in selection procedure (such as stepwise regression) among several predictor variables and to assess the fit of the model. Mallow's Cp is an estimate of the mean squared prediction error, which avoids the overfitting with the increase of the number of predictors in a standard multiple regression. The optimum model selected is then a compromise among the sample size, the effect sizes of the different predictors, and the degree of collinearity between the predictor variables (Daniel and Wood 1980)

Determinants of litter size variation

We obtained the residual values of virus and bacteria species richness by regressing these variables with the number of screening tests and/or host sample size using standard regressions on independent contrasts. We used these residuals in the subsequent analysis.

We then performed a standard multiple regression with litter size as the dependent variable and host body mass, mean latitude, virus species richness (residuals) and bacteria species richness (residuals) as independent variables using independent contrasts.

Results

Phylogenetic signal

As on rodent body mass and rodent litter size showed significant phylogenetic signal (Table 1) we used the independent contrasts method to control for this potential phylogenetic effect. There were no significant phylogenetic signal on the number of microparasite tested, host sample size and the microparasite richness (Table 1, all $p = 1$).

Table 1. Measures of Pagel's phylogenetic signal (λ) for the traits used in this study. Phylogenetic signal was significantly nonzero for (p-values indicate significance levels when testing whether λ differs 0 in a likelihood ratio test).

Variable	λ	Loglikelihood ratio	p
Microparasites tested	0.808	175.99	1.00
Host sample size	0.360	130.26	1.00
Microparasite species richness	0.770	167.71	1.00
Virus species richness	0.886	122.11	1.00
Bacteria species richness	0.736	107.65	1.00
Latitude (mean)	0.498	424.00	<0.0001
Rodent body mass	0.428	53.537	0.0012
Rodent litter size	0.545	44.905	0.0059

Microparasite species richness and latitude

We performed multiple regression with microparasite species richness as dependent variable and number of host samples, number of screening tests, mean latitude, range of latitudinal variation and host body mass as independent variables.

By selection of the best subset of independent variables we found that, in rodents, microparasite species richness depended on both the number of screening tests and on the latitude (Mallows Cp = 1.18) (Table 2). Microparasite species richness as a whole (i.e. for both viruses and bacteria) increased at lower latitudes (Table 2, Fig. 1A), independently of host latitudinal range distribution. However, when assessed separately the best subset of independent variables, only the virus species richness increased with the decrease of the latitude, contributing to the general trend observed (Cp = 0.67) (Table 2). No such a trend was observed for bacteria (Cp = –1.69) (Table 2).

Rodent litter size and microparasite species richness

Using multiple regressions, we found that, in rodents, litter size was linked both by microparasite species richness (using residuals of the general regression model of Table 2) and latitude (Table 3). Specifically, rodents' litter size increased at higher latitudes (Table 3, Fig. 1B) and decreased with higher microparasite (both bacteria and virus species) richness (independently from the latitude) (Table 3, Fig. 1C). The relationships remained statistically significant when removing the few outliers.

Discussion

This comparative analysis shows: 1) the existence in rodent species of a negative correlation between microparasite species richness and latitude: rodent host species in the tropics harbour in general more virus species than those living at higher latitudes; 2) rodents harbouring higher virus and/or bacteria species have reduced litter size, independently of body size, phylogeny and latitude. Our analyses and results also emphasize the need to control for potential bias linked to the microparasite detection (i.e. the number of microparasites screened), by obviously showing that the detection of microparasites is dependent on screening techniques and not only on the number of hosts sampled (as for macroparasite detection).

These findings seem to support the hypothesis that enhanced parasite richness-related effects could be linked to the higher incidence of multiple infections and their stronger effect on the host physiology and reproduction, and that these effects might be exacerbated in the tropics where higher microparasite species richness is observed.

Higher microparasite species richness at lower latitudes

Our analysis is the first to link virus and bacteria species richness with latitude in wild mammal species. The few previous studies, which focused on carnivores (Lindenfors et al. 2007) or primates (Nunn et al. 2005), have not linked virus or bacteria species richness and latitude. The only previous study that linked higher virus richness with tropical latitude was the study by Guernier et al. (2004) in humans. This might be due to the existence of some specific factors that enhance microparasite transmission in humans and rodents in the tropics (but not in carnivores and primates). Interestingly, Guernier et al. (2004) found an effect of latitude on microparasite richness but only for indirectly-transmitted virus. Moreover, the increase in protozoan species richness observed in primates in the tropics (Nunn et al. 2005) was also mainly related to an increase in vector-borne protozoans, as sixty four percent of all protozoan species considered in this study were vector-borne. Taken together, these results suggest the importance of arthropod-vector transmission in the observed higher microparasite species richness in the tropics.

However, if vector transmission had a significant role in the latitudinal pattern, we should have observed higher bacteria species richness at lower latitudes considering that most of the bacteria included in our analysis are vector-transmitted. On the contrary, our results show that the increase in microparasite species richness concerns only viruses, which are in our analysis all directly-transmitted. This suggests the existence of some other mechanisms at work to explain the observed pattern. Directly-transmitted parasites may more often depend on levels of host contacts and/or host densities (Anderson and May 1979). We can hypothesize that higher rodent densities, often observed in areas with more anthropogenic disturbance (Utrera et al. 2000, Suzan et al. 2008) and particularly in the tropics (Alessa and Chapin 2008), may promote higher virus transmissions (Kuenzie et al. 2001). Accordingly, the lower density of carnivores and primates in tropical areas (Harcourt 2006, IUCN 2010) might help to

Table 2. Multiple regressions on the potential determinants of overall microparasite species (both viruses and bacteria) richness, virus species richness and bacteria species richness with host sample size, number of screening tests, host species body mass and host species latitude as independent variables (using independent contrasts) with selection of the best subset of independent variables (using Mallows' statistic).

Dependent variable	Subset of independent variables	Sum of squares	Slope	F-test (p)	R^2, F-total (p)
Viruses + Bacteria	no. of microparasites screened	7.3	0.70	103.08 (< 0.001)	
	latitude	0.40	–0.02	5.65 (0.02)	
					$R^2 = 0.81$
					$F_{2.56} = 52.20$ (< 0.0001)
Viruses	no. of microparasites screened	1.72	0.37	24.98 (< 0.001)	
	latitude	0.31	–0.02	4.43 (0.03)	$R^2 = 0.57$
					$F_{2.56} = 13.63$ (< 0.0001)
Bacteria	no. of microparasites screened	1.4	0.38	63.99 (< 0.001)	$R^2 = 0.73$
					$F_{1.57} = 63.99$ (< 0.0001)

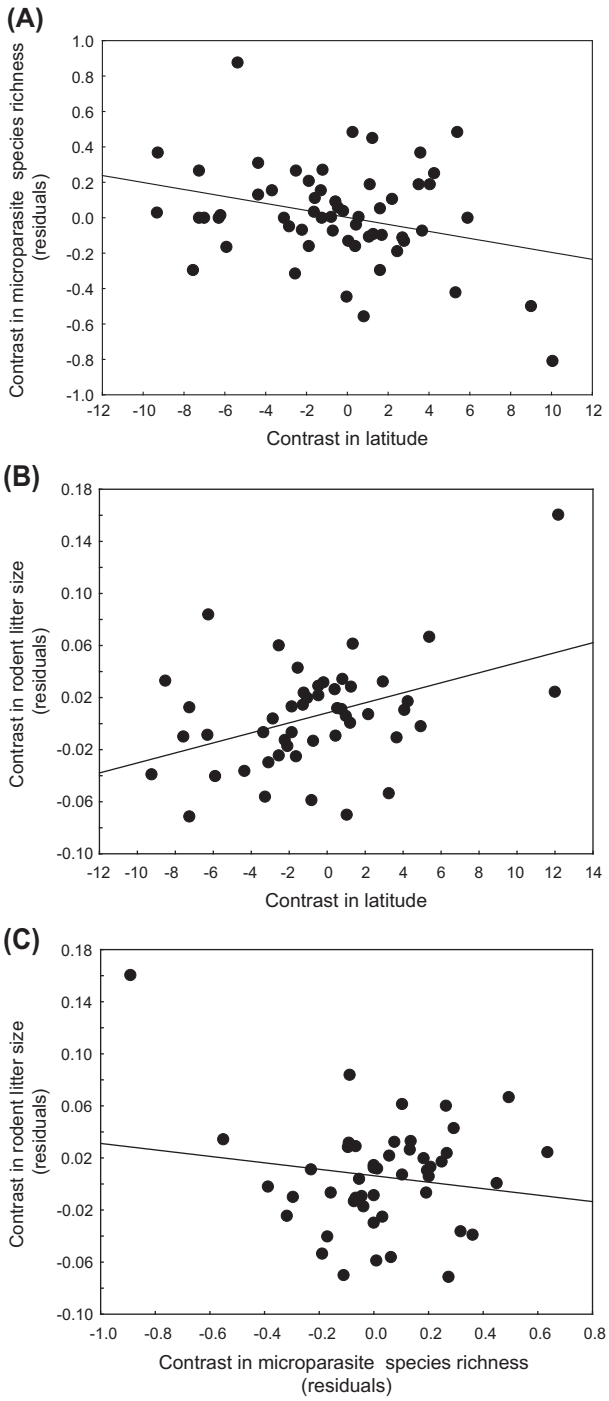

Figure 1. Partial relationship between (A) microparasite (bacteria and viruses) species richness and latitude, (B) rodent litter size and latitude and (C) rodent litter size and microparasite (bacteria and viruses) species richness using independent contrasts (using residuals from the general regression modelling in Table 2) (all regressions remained statistically significant when removing outliers).

explain why no latitude trend for the microparasite richness was observed in these two categories of mammals.

There is however also the possibility that the type of vectors may matter. Although the vectors of protozoan parasite species in primates or viruses in humans are mainly mosquitoes (see for examples the transmission modes of *Plasmodium* spp., *Hepatocystis* spp. or yellow fever virus), flies (*Trypanozoma brucei*) or bugs (see the transmission modes of *Trypanozoma cruzi* in humans and primates, or of *Trypanosoma rangei* in primates), the bacteria in the present study are transmitted by fleas (plague, *Bartonella* spp.), ticks (*Borrelia* spp.) or chigger mites (*Orientia tsutsugamushi*). Although some studies showed higher tick species richness at lower latitudes (Cumming 2000), this appears not to be the rule. For examples, flea species richness is reduced at lower latitudes when compared to more temperate areas (Krasnov et al. 2004), and Oribatid mite species richness increases from the boreal regions towards the southern temperate regions, but it does not further augment in the tropics (Maraun et al. 2007). The lack of relationship between bacteria species richness and latitude could then be explained by the discrepancies we observed in the geographical distribution of arthropod species vectors with latitude.

Other parameters that can enhance pathogen species richness, such as the number of mammal or bird species in a given region, were not considered in this study (Dunn et al. 2010). They however could be an important component of the latitudinal effect on the microparasite species richness in rodents.

Litter size and latitude in rodents

Litter size has already been reported to be positively correlated with latitude in rodents (Hayssen 2008) and our results confirm these previous findings. Interestingly, no tested explanation has been proposed for such a pattern. Here, we suggest that the reduced litter size observed in rodents may be linked to higher parasite species richness, independently of latitude. Latitude per se is, however, hardly a determinant of diversity (Brown 1995). Rather, various factors (including abiotic factors such as climate or biotic factors such as parasites or the intensity of predation) associated with latitude should operate independently or synergistically to explain the observed patterns (Schemske et al. 2009). Seasonal reproduction linked to climatic factors could explain the observed link between litter size and latitude. Many mammals reproduce seasonally due to hard foraging conditions in some (or many) parts of the year. We may then hypothesize that the observed reduced litter size at lower latitudes could be related to seasonality in reproduction in temperate or arctic areas, a pattern prone to promote higher litter size during reproductive events at higher latitudes contrary to tropical latitudes. Unfortunately, we were not able to test the effects of seasonality per se due to the lack of data for most of the investigated rodents. However, there are some recent empirical arguments that minimize the effects of seasonality. At all latitudes mammals may reproduce seasonally and even in the tropics many habitats can be strongly seasonal as those in more temperate latitudes (reviewed by Bronson 2009). For example, if the cloud forest mice *Peromyscus nudipes* ovulates throughout the year in Costa Rica, it cannot maintain pregnancy during the dry season because of insufficient food (Heideman and Bronson 1993). In north Burkina Faso, Sahelian rodents living in habitats where food is only abundant during the rainy season reproduce only during this period (Sicard and Fuminier 1996). Moreover, and importantly, we are not aware of any study linking higher litter size to seasonality per se.

Table 3. Multiple regressions on the potential determinants of rodent species litter size with host body mass, host latitude, virus and bacteria species richness (virus richness and bacteria richness were corrected for both host sample size and number of microparasites screened using residuals of the GRM of Table 2) as independent variables (using independent contrasts).

Independent variables	Sum of squares	Slope	F-test (p)	R^2, F-total (p)
Host body mass	<0.01	–0.11	0.82 (0.37)	
Latitude	0.02	0.42	11.19 (0.002)	
Virus richness (residuals)	0.01	–0.30	5.43 (0.02)	
Bacteria richness (residuals)	0.01	–0.30	5.57 (0.02)	
				$R^2 = 0.31$
				$F_{4.45} = 5.04$ (0.002)

Seasonality is a potential factor that may explain reduced litter size in rodents at tropical latitude, but clearly this hypothesis remains to be tested in relation to other factors. Our results, taken together with those of Robar et al. (2010), bring some support to the idea that higher parasitic pressures at tropical latitudes, eventually combined with other factors such seasonality, could affect rodents' life- history traits.

Parasite species richness, resistance and reduced fertility

The demographic effects of polyparasitism have been mainly studied at the host intraspecific level (Davidar and Murton 2006, Jolles et al. 2008, Munson et al. 2008) by comparing individual hosts harbouring one or few parasites species with individual hosts that harbour more parasite species. Moreover, they all focused on the mortality rates, except the study by Šimková et al. (2008), which linked higher parasite species richness and reduced gonad size across female fish species. Our study, despite being only correlative (as the previous ones), strongly suggests that higher parasite species richness, potentially positively linked to higher co-infections, can negatively affect host demography. Importantly, to date, there is no consensual explanation or related mechanisms able to explain the higher observed impacts. The key factor could be the virulence of parasites because there is a positive evolutionary relationship between transmission and virulence (defined here as any negative effect of a parasite on its host). In other words, any factor that enhances transmission in a given area (i.e. potential higher host densities in the tropics) could explain not only the higher parasite species richness at lower latitudes but also the higher virulence of the parasites encountered in the tropics. Our results also highlight the ongoing debate about resistance and tolerance in host–parasite co-evolution (Råberg et al. 2008, Svensson and Råberg 2010). Briefly, resistance is a way of minimizing the enemy's successful attacks (notably by the immune defences), whereas tolerance is a way of minimizing the impact of these attacks on the host fitness (for example by altering a life-history trait such as fertility). It was suggested recently that biologists have over-emphasized resistance (Svensson and Råberg 2010). In accordance with the tolerance hypothesis, an increase in the reproductive output (i.e. increased litter or clutch size) in response to parasitism has sometimes been observed in mammals and birds, at intraspecific level, in single host / single parasite models (Soler et al. 2001, Kristan 2004). Shifts in life-history traits have been proposed in presence of chronic parasitic infections due to expected reduced survival and/or negative impacts on future reproductive opportunities (Agnew et al. 2000). Life-history theory predicts that parasitized hosts should increase their reproductive effort by earlier sexual maturity and/ or higher reproductive output (Agnew et al. 2000, Kristan 2004). For example, wild-derived mouse *Mus musculus* increased their litter size when infected by the nematode *Heligmosomoides polygyrus (*Kristan 2004). Despite the existence of these alternative and potential adaptive responses, our results seem to suggest that higher parasite species richness across host species may affect rodent reproductive traits. These negative impacts could be then related to resistance to parasites and tradeoffs between resistance and reproduction mediated by enhanced costly immune responses. This interpretation is in accordance with previous comparative studies that highlighted higher immune investments in tropical birds (Møller 1998) or in mammals harbouring higher helminth parasite species (Bordes and Morand 2009b).

Reservoirs with non-pathogenic infections: a weak paradigm?

All the viruses and bacteria screened in our study persist in their host populations for long times and may thus serve as reservoirs for future infections. These chronic infections, due to the fact that the immune response is inefficient in eliminating the parasite, are classically supposed to be asymptomatic or to induce minor pathogenicity for their "reservoir" host (Easterbrook and Klein 2008). However, there is recent empirical evidence for viruses that such chronic infections may affect the host demography, particularly by reducing host survival (Kallio et al. 2007, Burthe et al. 2008) and also fecundity (Feore et al. 1997). From this perspective, our comparative approach supports the idea that viruses and bacteria that cause chronic infections in rodent species may have negative effects on their rodent hosts, particularly by reducing their litter size.

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Supplementary material (available online as Appendix 019314 at <www.oikosoffice.lu.se>). Appendix A1, A2.

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