albumin contains no carbohydrate in its molecules (*11, 12, 14*) may render it unnecessary for albumin to be transported to the Golgi to acquire carbohydrate before its discharging into cytosol. Presumably, albumin belongs to a class of proteins (*15*) whose molecules are synthesized by bound polysomes and discharged directly into the cytosol.

To establish the specificity of the immunocytochemical reaction, some critical control experiments have been conducted. The binding of Fab-peroxidase conjugate with intracellular albumin did not occur when the conjugate was previously treated with excess rat serum albumin. Incubation of specimens with horseradish peroxidase alone did not produce reaction product. When Fab-peroxidase conjugate against potato acid phosphatase was used, no reaction product was present, except for the expected endogenous peroxidase activity in peroxisomes and erythrocytes. When tissue section was incubated with 2 percent H_2O_2 at room temperature for 10 minutes before incubation with Fab-peroxidase conjugate, the reaction product was present at the site of albumin synthesis, although endogenous peroxisomes showed negative reaction. The last experiment indicated that the reaction product was not due to endogenous peroxidase activity. Furthermore, since only some liver cells could synthesize albumin, the albumin-negative cell adjacent to the positive cell served as another excellent control.

In addition to the liver, positive albumin synthetic activity has been observed in other tissues, including aortic endothelium, renal vascular and lymphatic endothelium, Bowman's capsular epithelium (Fig. 1d), proximal convoluted epithelium, interstitial cells, and the ascitic cell of the Chang rat hepatoma. The intracellular sites of localization of albumin in these tissues were exactly the same as those in the hepatocytes. Previously, investigators believed that only liver cells could synthesize albumin (*14*).

CHIN-TARNG LIN*
JEFFREY P. CHANG
Department of Human Biological Chemistry and Genetics, University of Texas Medical Branch, Galveston 77550

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* Present address: Taiwan University Hospital, Taipei, Taiwan, Republic of China.

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Insular Biogeography: Of Mice and Mites

Abstract. *The number of mite species using cricetid rodents in North America is related to the host distributional areas. The age and latitude of the distributional areas have unimportant effects on the number of mite species using a rodent species. The relation between species and area is analogous to species equilibrium numbers of island faunas.*

Terrestrial mammals often support a large and diverse assemblage of endo- and ectoparasites. We have examined the theories of island biogeography introduced by MacArthur and Wilson (*1*) and subsequently applied to insect communities of host plants (*2*) for their applicability to mice and mites.

Mites associated with mammals can be parasitic or phoretic. They range in size from the microscopic *Demodex* species (100 μm) to the relatively large and visible ticks (Ixodidae). The endoparasitic mites, ticks, and chiggers have been excluded from consideration, restricting the component community to those mites, found on the skin or hair of mammals, that either spend their entire lives on their hosts or remain on a mammal for a period of time sufficient for dispersal (*3*). Roughly three guilds, based on food preference (blood, tissue fluids, or hair) can be identified in this community with a fourth guild of phoretic mites (*4*). We have chosen those mites reported from North American cricetid rodents because they are well studied and represented by a large number of species. Distributional areas of the rodents were

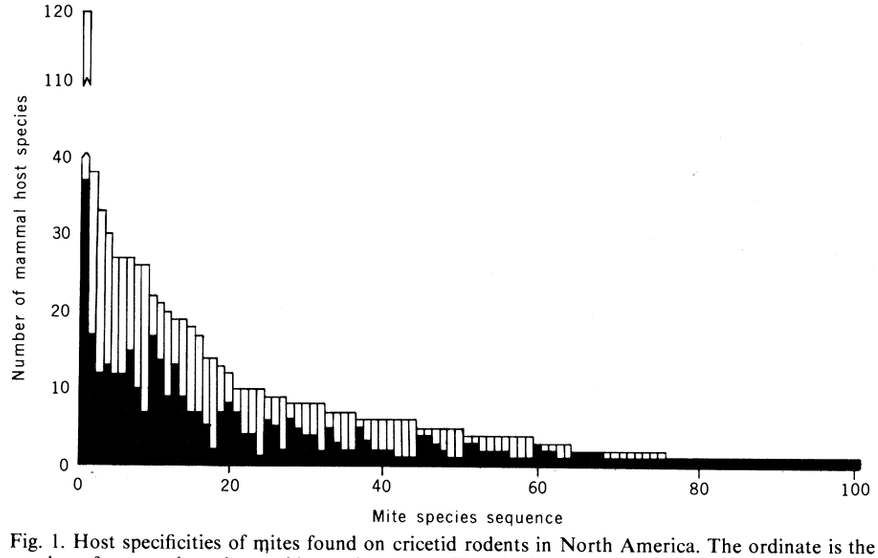

Fig. 1. Host specificities of mites found on cricetid rodents in North America. The ordinate is the number of mammal species used by a mite species. The abscissa represents each mite species ranked by the number of mammal species used as hosts, those with the fewest species farthest from the origin. The solid bars represent the number of cricetid species used; the hollow bars, all other mammal species used. One can conceive of the graph as ranking the mite species in order of increasing host specificity. [Based on data from Whitaker and Wilson (*6*)]

obtained from published maps (*5*) by means of a planimeter, and lists of mite species reported from each rodent were obtained from Whitaker and Wilson (*6*). Only those cricetids listed in Whitaker and Wilson were considered, yielding data on 40 rodent and 101 mite species (*7*). Records of mite species are derived from two types of sources: extensive surveys of the ectoparasitic fauna of mammals in a particular area (*8*) for which the number of mite species can be correlated with the number of specimens examined, and publications of a taxonomic and descriptive nature (*9*) in which typical hosts are reported, but not the number of animals examined. The mite species studied demonstrated varying degrees of host specificity as shown in Fig. 1, in which accidental hosts (those not known to be normal hosts, but on which the mite species is occasionally reported) are given equal value to the more typical hosts.

The resultant species-area curve (Fig. 2) fits the equation $S = kA^z$ found for island species, but A in this case represents host distributional area. The exponent in the above equation is known as the z-value in island biogeography and can be thought of as an index which measures the rate at which new species are added as area is increased. The z-value obtained (0.37) is lower than that reported by Opler (0.465) for leaf-mining Lepidoptera on oaks, or by Strong (1.10) for insect species on British trees, but is higher than the expected value for continental areas (0.12 to 0.17), being closer to that of actual island faunas (0.20 to 0.35) as reported by MacArthur and Wilson.

The species equilibrium postulated for island communities is not only a function of island size, but also of island age (*10*) and proximity to a species pool (*1*). The age of a host "island" would include both the age of the present host distribution as well as the evolutionary age of the host species. Opler dealt with this difficulty by choosing oak species that he judged to be of nearly identical ages. In his reevaluation of Southwood's data (*11*) on the insects of British trees, Strong concluded that evolutionary time was an insignificant component. The geographic ranges of North American cricetid rodents can be thought of as islands of identical age which appeared in their present form sometime after the final retreat of Pleistocene glaciers (*12*), but unlike Opler's oaks, these islands are very young, no more than 20,000 years old at their present sizes. Some of the rodent species are probably also no older than 20,000 years (*13*). No correlation between fossil age and number of mite species could be found at the generic level for the rodents studied. The importance of evolutionary age is therefore minimal. Such a contention can be strengthened by an examination of the mite fauna of the three introduced species of Old World mice and rats. (Muridae). *Mus musculus*, *Rattus norvegicus*, and *Rattus rattus* are distinguished by their close association with human activities and their cosmopolitan distribution. If one allows 10 million square miles (roughly the area of North America) for their distributional areas, the number of mite species reported from each rodent still falls roughly within the limits of our data (23, 24, and 15 mite species, respectively, for *Mus musculus*, *Rattus norvegicus*, and *Rattus rattus*). Of the total of 35 mite species reported from these rodents only 9 species, at most, are introduced and only 4 species, at most, are still endemic to one or more of the three rodents in North America (*14*). This suggests that the equilibrium number of mite species is rapidly attained since dates of introduction are about 1500 for *Rattus rattus* (*15*) and 1775 for *Rattus norvegicus* (*15*) and *Mus musculus* (*16*).

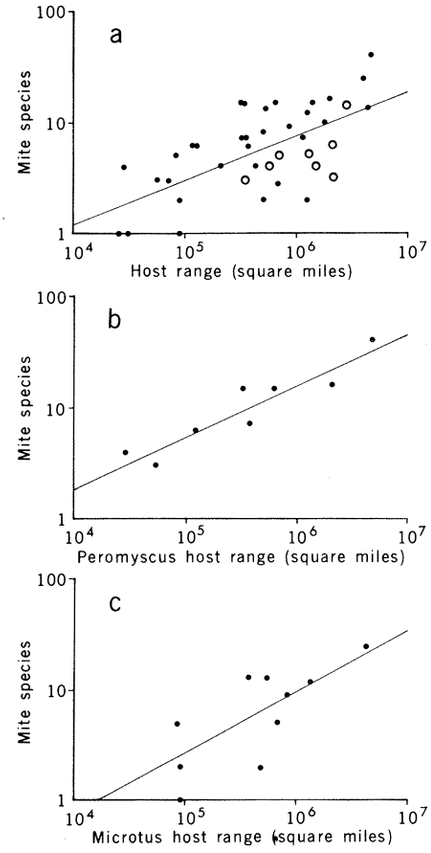

Fig. 2. Species to area relationships for mite species on cricetid rodents of North America. The open circles in (a) represent species whose ranges lie almost entirely above 50° latitude; the closed circles, all others. The regression lines are: $\log_{10}$ (No. spp.) = 0.363 × $\log_{10}$ (area) − 1.295 (r = .61, $P \leq .001$) for all species studied; $\log_{10}$ (No. spp.) = 0.454 × $\log_{10}$ (area) − 1.53 (r = .93, $P < .001$) for *Peromyscus* (b); $\log_{10}$ (No. spp.) = 0.549 × $\log_{10}$ (area) − 2.309 (r = .68, $P \leq .01$) for *Microtus* (c).

The species pool consists of all mite species on North American mammals. For species that are generalists in terms of host preference, the probability of colonization for a given host "island" is related to the degree of overlap of its geographical distribution with those of other hosts with which it can exchange mite species, rather than strictly distance to a species pool. The probability of colonization would therefore increase as the degree of overlap with other species increases. In general, as host areas increase, the amount of overlap with other mammal species ranges will increase. Host "islands" thus differ from other islands in that increasing the area can increase the immigration rate as well as decreasing the extinction rate for host species whose fauna are not strictly host-specific.

Opler and Strong, dealing with host species with more limited ranges than those in this report, could not examine what effect latitude could have on species number. In general, species number decreases along a latitudinal gradient (*17*). Although multiple regression analysis including latitude (as determined by the center of the host species range) did provide a better fit for the data (r = .74) compared to r = .61 without considering latitude), statistical analysis revealed that latitude failed to explain any significant amount of variance after the effect of area had been subtracted, while area did explain a significant amount of variance ($P < .001$) after the effect of latitude has been subtracted. In Fig. 2a, however, those rodent species found mostly north of 50° latitude generally fall below the regression line, having fewer mite species than would be expected from their distributional areas. This is probably due to a paucity of mammal species at northern latitudes. Below the 50° threshold the number of mammal species cannot be strictly correlated to latitude alone, which is probably the reason for the lack of significance of latitude on mite species diversity in our regression analysis.

We are aware that certain weaknesses in our data, such as the uneven intensity at which the mite fauna of each rodent species has been studied, could produce artifacts in our analysis. It is possible, by tabulating all species reported from a rodent rather than the number of species present at one time, to overestimate the equilibrium numbers if species turnover is very rapid. Similarly, the equilibrium numbers may be underestimated for poorly studied species. To estimate the effects of intensity of study we compared *Peromyscus* species,

which have been well studied in the last decade, with *Microtus* species, which have been studied less intensively, in Fig. 2, b and c. *Peromyscus* shows a much better fit (r = .93) to the equation than does *Microtus* (r = .68), suggesting that poorly studied species serve only to increase the variance and do not introduce artificial correlations into the data.

In conclusion, the number of mite species using a rodent species is a function of the rodent's distributional area and, to a small degree, its latitude. The fit to the species-area equation reported here suggests that island biogeography theories are plausible explanations for the data observed, although conclusive evidence would require that species turnover rates for host "islands" be measured (*18*). For mites on mammals we believe the resulting species equilibrium numbers are determined by immigration and extinction rates which act primarily in evolutionary time. One would not expect that increasing area would greatly increase the colonization rate in ecological time for host-specific mite species.

WILLIAM DRITSCHILO
HOWARD CORNELL
DONALD NAFUS, BARRY O'CONNOR
Department of Entomology, Cornell University, Ithaca, New York 14853

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Leukemia Virus–Induced Immunosuppression: Scanning Electron Microscopy of Infected Spleen Cells

Abstract. *Spleen cells from mice infected with Friend leukemia virus were examined by scanning electron microscopy. Whereas splenocytes from normal noninfected animals showed the expected morphologic classes of lymphocytes, including those with smooth surfaces and with numerous villous projections, an alteration of cell type was evident within a few days after infection. Friend leukemia virus caused a rapid decrease in the number of villous cells, with a concomitant increase in the number of cells with smoother surfaces. By the end of the first 1 to 2 weeks after infection the majority of cells were smooth, many showing distinct morphologic changes, including "holes" and a spongy appearance. Nearly all of the splenocytes were abnormal in appearance by days 17 to 30 after infection, with most showing a spongy topography. These changes paralleled the marked immunosuppression induced by Friend leukemia virus infection, as well as the appearance of virus-associated surface antigen on individual splenocytes. Topographic changes evident by examination with scanning electron microscopy were not readily apparent by either standard histology or transmission electron microscopy.*

Infection of susceptible strains of mice with an oncornavirus such as Friend leukemia virus (FLV) invariably leads to a marked and generalized immunosuppression (*1*). However, the mechanism whereby tumor virus infection depresses immunologic competence is still not clear. Earlier studies in this and other laboratories suggested that leukemia viruses preferentially affect antibody precursor cells rather than antibody-forming cells per se (*2*). Cell transfer studies with splenocytes from leukemia virus–infected mice, as well as transfer of bone marrow or thymocytes (or both) from infected animals, focused attention on the B lymphocyte as the most likely target of leukemia virus–induced immunosuppression (*3*). Furthermore, immunohistologic studies revealed a marked decrease in cells bearing immunoglobulin

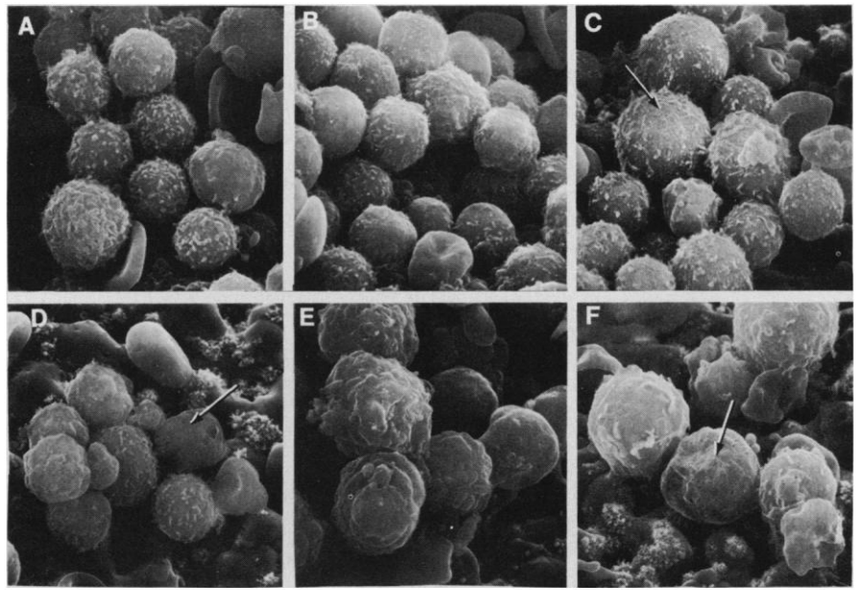

Fig. 1. Scanning electron micrographs of spleen cells. (A) Typical spleen cells from normal Balb/c mice showing villous and smooth cells; (B) spleen cells from 5-day infected mice showing moderate changes including fewer villous cells and one moderately "spongy" cell; (C) 7-day infected spleen cells, many of which are large and deformed with altered surface topography (arrow); (D) 10-day infected spleen cells with smoother surfaces and one highly spongy cell (arrow); (E) 17-day infected spleen cells most of which are large and deformed; (F) spleen cells from 30-day infected mice showing large, smooth surfaces, some with "holes" and spongy appearance (arrow) (× 2500).