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When Rabbits became Human (and Humans, Rabbits): Stability, Order, and History in the Study of Populations

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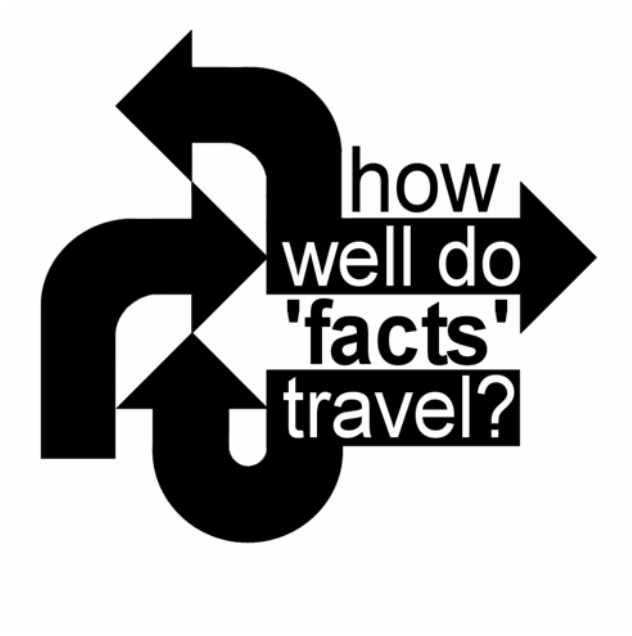

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When Rabbits became Human (and Humans, Rabbits): Stability, Order, and History in the Study of Populations[1]

Paul Erickson and Gregg Mitman

Abstract

"Population" is often a significant unit of analysis, and a point of passage for facts and models moving between the natural and social sciences, and between animals and humans. But the very existence of a population is a "fact" fraught with challenges: What distinguishes a population from an economy, an ecosystem, a society? Are populations simply memory-less aggregates of solitary individuals, or do they constitute groups with unique histories and agency? Looking at how populations of humans and populations of rabbits were thought of in terms of one another, this paper examines several interlinked episodes in the history of "population" as an organizing concept in 20th century science, tracking the transfer of facts from rabbit populations to human populations (and vice versa) through economics, infectious disease modelling, and macro-histories. What happens when rabbits become human, and when humans become rabbits?

In both the natural and social sciences, the "population" is often a significant unit of analysis, structuring inquiry across a wide range of disciplines – statistics, evolutionary biology, ecology, demography, to name a few. Not surprisingly, "population" has been recognized as an obligatory point of passage for facts and models moving between fields, between the natural and social sciences, and between animals and humans. Thus Darwin could find inspiration for his theory of natural selection by reading Malthus's seminal work of demography, the 1798 *Essay on Population*, and interdisciplinary programs in "population studies" flourish throughout universities today that attempt to blend

[1] Prepared for "How Well Do Facts Travel?" workshop, "Facts at the Frontier," LSE, 16-17 April 2007

ecology, demography, evolutionary biology, and epidemiology in varying proportions.

Michel Foucault has argued that the emergence of the population is connected to the rise of modern capitalism and the nation state, whereby "numerous and diverse techniques for achieving … the control of populations" has led to new forms of "power situated and exercised at the level of life, the species, the race, and the large-scale phenomena of the population." Foucault's claim raises the question: What constitutes a population? And why have so many different fields of inquiry seized upon this ambiguous entity to serve as their fundamental unit of analysis, and what is the relationship between "facts" about population displayed through graphs, charts, and models, assumptions about animal and human behaviour, and strategies for population regulation and control? The very existence of a population is a "fact" fraught with challenges. Are populations entirely arbitrary collections of individuals, and how are they delineated? Are facts about populations somehow irreducible to facts about their members? For example, the fitness statistics of population genetics are transparently aggregative facts about populations, as are the risk coefficients produced by traditional epidemiology; but other facts about populations may not be as obviously so. For example, are populations characterized by structures that transcend the individual? What distinguishes a population – which may consist of anything from atoms to animals – from an economy, a community, an ecosystem, a society, or any of the other units of analysis that define the human sciences of economics, sociology, anthropology, or history? The question of population structure leads to a final question concerning how populations change over time. Are populations simply memory-less aggregates of solitary individuals, or do they constitute groups with unique histories and agency? To date, these sorts of questions have not been analyzed in a unified and comparative manner.

As a first cut at this analysis, this paper examines several interlinked episodes in the history of "population" as an organizing concept in 20th century science. These episodes are chosen so as to focus attention on the passage of facts between animal populations and human populations. The 1920s saw a renewed focus on populations in the biological sciences from a number of disciplinary fronts. In the world of animal ecology, Charles Elton proved a key figure in making visible the ways in which populations regulated, and were controlled by, natural and human economies. For Elton, a focus on population provided a convenient way of bridging academic research and the interests of his patrons, who were concerned with fluctuations in output of resources drawn from fish stocks, populations of fur-bearing animals, or rangelands. Thus, while Elton established the facts of population fluctuations in animals, during the 1920s these facts moved freely between ecologists and economists interested in understanding the causes of business cycles in human economies. A second chapter in this story begins in the 1950s, when a successful application of a virus, myxomatosis, to control rabbit populations in Australia produced one of the most famous natural experiments of host-parasite co-evolution ever performed. For a number of reasons unique to the 1960s and 1970s, the story of the rabbits and myxomatosis came to be seen as a glimpse of the past (and future) of humanity's encounters with disease. Myxomatosis became the pre-eminent example of how disease regulated populations across the animal-human divide, and a model that biologists such as Jared Diamond and historians such as William McNeill drew upon to write best-selling books highlighting disease as a natural force in shaping the course of human history. Finally, in the 1980s, these conversations facilitated a theoretical convergence between population ecology, evolutionary biology, and epidemiology to form new links between the scientific study of disease in animals and in man. These connections continue to this day

in the form of increasingly sophisticated mathematical models for the ecology of diseases like HIV/AIDS, BSE, avian influenza, and other emerging infectious diseases. Throughout this unfolding history of population, a central problem emerges: when rabbits become human, and when humans become rabbits, what is the differential fate of facts in the travel across this asymmetrical divide?

Lemmings and Stockbrokers: Charles Elton and the "Institutionalist" Approach to Fluctuations in Animal Populations

Populations of animals in nature tend to fluctuate. This fact is often associated with the names Alfred Lotka and Vito Volterra, who independently wrote down a set of coupled nonlinear first order ordinary differential equations that described the interaction between populations of predator and prey species. The linearized (small-displacement) solutions to these equations trace out sinusoids in time, with predators and prey chasing each other in never-ending cycles of feast and famine, boom and bust. [2]

These sinusoids are often taken as biological facts in themselves, yet the 1920s also saw the emergence of several major empirical research programs devoted to establishing the facts of population

[2] For reviews of this literature, see M.E. Solomon, "The Natural Control of Animal Populations" *Journal of Animal Ecology* 18.1 (May 1949), pp. 1-35; cited on p. 1. On the history of the Lotka-Volterra equations, see Kingland, *Modeling Nature: Episodes in the History of Population Ecology* (Chicago: University of Chicago Press, 1985). See also Alfred Lotka, *Elements of Physical Biology* (Baltimore: Williams and Wilkins Company, 1925); Vito Volterra, "Variazioni e fluttuazioni del numero d'individui in specie animali conviventi" *Mem. R. Acad. Naz. Lincei Cl. Fis. Mat. e Nat.* 62.3 (1926), pp. 31-112; Vito Volterra, *Leçons sur la théorie mathématique de la lutte pour la vie* (Paris: Gauthier-Villars, 1931); Volterra, "Principes de biologie mathématique" *Acta Biotheoretica* 3 (1937), pp. 1-36; AJ Nicholson, "The Balance of Animal Populations" *Journal of Animal Ecology* 2.1 (1933), pp. 132-178; A.J. Nicholson and V.A. Bailey, "The Balance of Animal Populations, Part I" *Proceedings of the Zoological Society of London* 3 (1935), pp. 551-598.

fluctuations in nature. [3] The scientists who performed this research were not philosophically minded theorists like Lotka but hard-nosed ecological managers who sought to create knowledge that would be of service to the commercial and administrative organizations of the late British Empire. Chief among these was Charles Elton, the grandee of the Oxford "school of imperial ecology" (to borrow a phrase of Peder Anker), expert in fur-bearing animals for his patrons in the Hudson Bay Company, and eventual director of the Oxford Bureau of Animal Population, started with funds from his powerful friends in the New York Zoological Society. [4]

Elton first drew attention to the problem of fluctuating animal populations in a famous 1924 article on the subject. Here, Elton drew upon a range of data, especially the records of the Hudson Bay Company 's fur-trading operations in Canada, to assemble a historical record of fluctuations in populations of fur-bearing animals (including rabbits) over nearly a 100-year period. These fluctuations were indeed significant, with rabbits in particular expanding or contracting by a factor of 10 over a couple years. However, at first blush, the data were pretty cryptic, producing graphs of seemingly random squiggles with no discernable amplitude or period. To help make sense of things, Elton turned to a fellow faculty member at Oxford, GMB Dobson, who instructed Elton in a kind of numerical Fourier analysis that would extract fluctuations of a given period from the data. Crunching through the numbers, Elton discovered what he thought were some significant patterns. The fluctuations appeared to be coordinated over wide geographical ranges, which suggested that some common cause must be responsible.

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[3] The separateness of these two programs comes across especially forcefully in Solomon's extensive literature review, "The Natural Control of Animal Populations," and in CHD Clarke's "Fluctuations in Populations" *Journal of Mammalogy* 30.1 (February 1949), pp. 21-25.

[4] On Elton, see Peter Crowcroft, *Elton's Ecologists: A History of the Bureau of Animal Population* (Chicago: University of Chicago Press, 1991); and Peder Anker, *Imperial Ecology: Environmental Order in the British Empire, 1895-1945* (Cambridge, MA: Harvard University Press, 2001).

Specifically, after removing some shorter-period fluctuations from the data, he found that the overall pattern of fluctuations was closely correlated with the 11-year sunspot cycle. He therefore concluded that changing output from the sun was the primary cause of population fluctuations. [5]

Of course, this result didn't explain everything about the fluctuations, and Elton spent the balance of the 1920s analyzing more data and trying to find more correlations. During this time he also became an ecological consultant to the Hudson Bay Company, which allowed him and his students access to HBC data in return for Elton's expertise in predicting fur yields. [6] Elton also cultivated connections with other powerful friends. Thus in 1931 he was invited to join Canadian wildlife officials and academic experts for a conference on "biological cycles" at the country house of Copley Amory near the town of Matamek Factory. Amory was a Boston Brahmin, a hunting and exploration enthusiast, and an investor in Newfoundland fisheries. As a result he was keenly interested in understanding why the 1930s were such miserable years for the fisheries, as they clearly were (one Matamek participant who caught a fish on lunch break was so surprised that he threw it down on the conference table when he returned). More generally, Canadian wildlife was in a sorry state, and folk wisdom seemed to indicate that this situation ran in cycles of about ten years. As coverage of the conference in *Time* magazine reported, sled dogs were ill and game was scarce, leading to significant human suffering in the Canadian economy. These

[5] Charles S. Elton, "Periodic Fluctuations in the Numbers of Animals: Their Causes and Effects" *British Journal of Experimental Biology* (1924), pp. 119-163.
[6] See Anker, *Imperial Ecology*, p. 107. The Company opened its archives (based in London) to the public precisely in 1931, although the reasons for the decision are unclear. By the 1940s, the Hudson Bay Company data, published and commented upon by Elton and his students, had come to be seen as something of a "biological canon" among scholars (see Clarke, "Fluctuations in Populations," *op. cit.*).

topics, and Elton's significant sunspot theory, set the agenda for the Matamek conference.[7]

The agenda at Matamek made it clear that understanding the nature of population cycles would thus be significant for understanding the ups and downs of both animal and *human* populations. This point comes across best in a presentation made by Ellsworth Huntington, an economist and geographer at Yale who presented data showing that financial panics in the United States were associated with droughts and crop failures, which ultimately depended on fluctuating climatic conditions. Here, Huntington drew on the business cycle theories of Henry Ludwell Moore, an American institutionalist economist and a founder of econometrics.[8] In light of the international economic meltdown then unfolding, Huntington's suggestion apparently sparked a great deal of interest in the attendees. Did fluctuations in climate operate upon humans and animals in a similar fashion, inducing common patterns of changing intellectual or reproductive activity, shifts in food sources, or the emergence of new diseases? Could these produce the common patterns of fluctuations seen in human and animal populations? The stage seemed set for a remarkable traffic in facts about business cycles in human and animal populations.

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[7] "Canadian Ecology" *Time* 10 August 1931. See also Ellsworth Huntington, "The Matamek Conference on Biological Cycles, 1931," in *Science* for an overview of the conference.

[8] On Moore, see Philip Mirowski, "Problems in the Paternity of Econometrics: Henry Ludwell Moore," *History of Political Economy*, 22 (Winter 1990), pp. 587-609. For an overview of the business cycle debate in the 1920s, see the review papers of Warren Persons, "Theories of Business Fluctuations" *Quarterly Journal of Economics* 41.1 (November 1926); on Moore's theory of climate-induced cycles see Moore, *Economic Cycles: Their law and cause* (New York, 1914) and Moore, "The Origin of the Eight-Year generating Cycles" *Quarterly Journal of Economics* (1921). Huntington had been interested in Moore's ideas since his years as a newly-minted Ph.D. in the 1910s; see Huntington, "Climatic Variations and Economic Cycles" *Geographical Review* 1.3 (March 1916), pp. 192-202 for a review of Moore's theories. Moore was part of an established tradition dating to the work of William Stanley Jevons, who in the 1860s had hypothesized that sunspots were responsible for regular fluctuations in economic activity; moreover, Moore's climatic determinism would remain part of the debate well into the 1930s.

What kind of facts about populations could travel across the human-animal divide, breached so briefly at Matamek? In general, the existence of population cycles and of a macro-climatic cause at work in both humans and animals were facts that could travel. Moreover such facts seemed to travel largely from animal populations to human populations.

However, it is equally revealing to look at what facts *didn't* travel particularly well at Matamek, and here Elton's earlier work provides some guidance. During much of the 1920s, Elton's thinking about population was greatly influenced by the work of his Oxford colleague Alexander Carr-Saunders. The 1922 book that made Carr-Saunders' academic name (and which strongly influenced Elton) applied Darwinian theories of evolution and Malthusian demography to the study of human societies. Using as evidence anthropological literature documenting human practices of infanticide, war, and sexual relations, Carr-Saunders argued that humans develop social customs designed to keep their populations close to an "optimum number" given their state of technology, the conditions of their environment, and so forth. Populations often did fluctuate around this "optimum number" due to a number of factors including "the fluctuations in the number [of humans] desirable, the erratic action of certain causes of elimination, such as war and disease, and migrations."[9] However, in spite of the recent conflict with Germany, Carr-Saunders remained optimistic that rising population densities did not inevitably lead to war and pestilence, since changes in human customs and social structures (for example, those governing relations between the sexes) might permit still greater densities of population to develop without threat of calamity.

[9] Alexander Carr-Saunders, *The Population Problem: A Study in Human Evolution* (Oxford, 1922), cited on p. 477. On Carr-Saunders, see "Obituary: Sir Alexander Carr-Saunders, 14 January 1886-6 October 1966" *Population Studies* 20.3 (March 1967), pp. 365-369.

Elton drew heavily on Carr-Saunders' work, as well as his earlier studies of population fluctuations, in writing his 1927 *Animal Ecology*, which is commonly viewed as a founding text of population ecology. Citing *The Population Problem*, Elton noted, "the question of the desirable number on a given area has received a great deal of attention from people studying the ecology of human beings. It is found that there is an optimum density of numbers for any one place and for people with any particular standard of skill." [10] In the case of animals, however, "standard of skill" in manipulating the environment or desired "standard of living" was not as significant in determining optimum number as the wholly biological facts of reproductive rates, quantities of food consumption, location in the food chain, and so forth. Species at the top of the food chain who would not succumb to predation by larger animals were a bit of a special case, and Elton seemed to be at a bit of a loss to explain their regulation. Parasites and disease could help account for keeping these populations in check; microbes rather than larger predators helped to turn a trophic hierarchy into a loop. Other animals apparently chose to limit their reproduction to replacement rate for reasons that were not entirely clear.

Elton's invocation of Carr-Saunders' results in *human* demography represents a significant departure from the conception of population dominant in his 1924 paper or in the Matamek conference. In *Animal Ecology*, populations teeter on the brink of becoming *communities* (a term that Elton borrowed from V.E. Shelford) or *economies*, entities characterized by facts about structure, interacting behaviours, and internal regulation. Yet in the final analysis, Elton saw little point in pursuing these sorts of analogies. They were, he insisted again and again, just a convenient and familiar language, signifying nothing: only the

[10] Elton, *Animal Ecology*, 6th printing (London: Sidgwick and Jackson, 1953), cited on p. 113.

facts about population numbers and their correlations with external driving forces are important. And at Matamek, the notion that rises and falls in population might be due to the internal social and behavioural structure of populations received scant attention.

Another potential – yet fraught – stimulus for traffic in facts between human and animal populations was the evident role of disease in population regulation. At the Matamek conference, the attendees had begun to grant more agency to disease-causing parasites – both bacteria and viruses – in the generation of population cycles. While "most members of the conference expected that the sunspot cycle of 11.2 years would figure prominently at Matamek,"[11] the conference revealed that there were in fact many natural cycles each with its own period. Instead, "In the production of cycles among animals an even greater part seems to be played by bacterial parasites and the diseases to which they lead than by the larger parasites." Professor R.G. Green of the University of Minnesota reported on his study of tularaemia in rabbits, a tick-borne disease that became more virulent at the peak of the rabbit cycle and helped to bring down population numbers. However, "for some unknown reason," "the virulence of the tularaemia bacteria decreases rapidly" toward the end of the population collapse, permitting the remaining rabbits to recover.[12] Similarly, Elton and several of his students presented their work on epidemics of encephalitis among arctic foxes, showing that in this case the virulence of the parasite remained strong throughout while the remaining foxes possessed significant immunity to the disease.

This emphasis on the role of disease and parasitism in regulating population densities had been present in Elton's work since at least the 1924 paper, and he paid attention to the relationship between disease outbreaks and population fluctuations in several other papers through the

[11] Huntington, "The Matamek Conference on Biological Cycles, 1931," p. 231.
[12] Huntington, "The Matamek Conference on Biological Cycles, 1931," p. 233.

1920s.[13] However, Elton generally emphasized the role of macro-climatic conditions in generating population fluctuations, in which disease was essentially a proximate agent *ex machina* causing the population decline, albeit one of particular interest to humans looking to prevent outbreaks of animal-borne illnesses like plague. However, Elton felt that facts about disease did not travel easily between human and animal populations. As he noted in a 1931 paper, "Up to the present time it has been customary to believe that wild animals possess a high standard of health, which is rigidly maintained by the action of natural selection, and which serves as the general, though unattainable, ideal of bodily health for a highly diseased human civilization." Animals, Elton argues, do not inhabit a pristine state of nature: rather, like "civilized" humans, they are constantly afflicted by epidemic disease. Because the former assumption has been overcome only recently, "the systematic study of disease in wild animals forms one of the latest branches of animal ecology." Disease then, had the potential to be a structuring force, not simply imposed from without but acting differentially *within* the population.[14]

Yet the implications of disease for highlighting new facts about the internal dynamics of populations seem to have escaped both Elton and the Matamek attendees: they remained focused on gathering facts about the correlation between population numbers and external stimuli, and disease was simply an intermediate cause in accomplishing these fluctuations. By contrast, later disease ecologists, such as Stephen Boyden and Robert May, came to see how that subtle shift – from disease as intermediate cause to disease as structuring agent – fundamentally transformed populations from an ensemble of statistical mechanics to a structured social unit.

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[13] C.S. Elton, "Plague and the Regulation of Numbers in Wild Mammals" *Journal of Hygiene* 24 (1925), pp. 138-163.
[14] Elton, "The Study of Epidemic Diseases Among Wild Animals" *Journal of Hygiene* 31.4 (October 1931), pp. 435-356, cited on p. 435.

Elton's work and the Matamek conference thus emphasized some facts about "populations" while downplaying others, and this permitted the establishment of some common ground between researchers who studied human populations (like Huntington) and experts on animal populations. However, the traffic in facts between human and animal populations was limited by a number of factors that were at once intellectual, social, cultural, and institutional. It is impossible to imagine Elton's interest in population fluctuations outside of his consultancy to the Hudson Bay Company and the significant patronage that he enjoyed from men like Copley Amory. For such sponsors, the facts that mattered were the quantity of crops and harvests and their pattern over time, not the intricacies of animal population structures. By contrast, in his earlier work, Elton had drawn on the demography of Carr-Saunders to suggest an interpretation of populations as "communities" marked by common norms of behaviour. He had also suggested that the omnipresence of disease served as an "internal" regulator of population in both humans and animals. Yet these features of human populations made little impression on the ecologists' understanding of animal populations (save in metaphor), nor were such facts really capable of making the return trip, bringing lessons from animal populations back to human societies.

Rabbits and the Future of Humanity: Ratcliffe, Fenner, and Myxomatosis in Australia

In the years that followed Elton's work on population cycles and their causes had become recognized as a classic in ecology, the start of a new direction of inquiry. Yet as we saw at Matamek, Elton's original explanation of the cycles was increasingly questioned. An author of a review article on population cycles would write in 1949, "For a while after Elton's inaugural paper (1924), grateful professors could include a period

on sunspots and wildlife cycles in their lectures. Now the matter is in a state much less satisfactory for the classroom, but much more so for research. Most measurable cosmic influences are open to the objection that they should operate uniformly over a large area, whereas local variations are characteristic of the best known cycles."[15] In addition, the facts of Elton's foxes and the models of predator-prey cycles developed by Lotka and Volterra remained separate: a logical explanation for the never-ending back and forth between animals and their parasites seemed clear, but the specifics of the process were yet to be discovered in any particular case. "The mathematicians tell us that under certain circumstances the relationships of animals and their predators or parasites or diseases could result in a cycle. The trouble is that where any organism is found killing animals during a cyclical die off it is so easy to postulate ways in which the required premises are satisfied that we have so far developed no basis of experimentation."[16] Within 10 years, this author's hopes would be fulfilled by an example emerging, not surprisingly, from the network of ecologists studying population cycles in the practical contexts afforded by the institutions of the late British Empire.

Francis Ratcliffe was typical of the generation of Anglophone biologists, like Thompson or Elton, who developed close associations with Imperial institutions after World War I. A student of Huxley and Elton at Oxford (BA, 1925), Ratcliffe also spent time working in London with the Empire Marketing Board before accepting an offer from Australia's Council for Scientific and Industrial Research to study the ecology of the flying fox and desertification in Queensland and New South Wales. His

[15] C.H.D. Clarke, "Fluctuations in Populations" *Journal of Mammalogy* 30.1 (1949), pp. 21-25; cited on p. 22.
[16] Clarke, "Fluctuations in Populations," p. 22; see also P. DeBach and H.S. Smith, "Are Population Oscillations Inherent in the Host-Parasite Relation?" *Ecology* 22 (1941), pp. 363-369 for a summary of where the Lotka-Volterra-Nicholson-Bailey strand of theorizing on host-parasite relationships and population fluctuations stood in the 1940s.

knowledge of entomology also proved invaluable to the Australian Imperial Force, which commissioned him a major and put him in charge of combating malaria, scrub fever, and dengue in the south-western Pacific theatre during the war. Afterwards, he spent a year as a fellow with Charles Elton's Bureau of Animal Population in 1948, returning to Australia to head up a new Wildlife Survey Section of the Commonwealth Scientific and Industrial Research Organization (CSIRO, which replaced CSIR). There, he would spend more than a decade leading the wildlife survey section and supervising efforts to control invasive species in Australia, most notably the European rabbit that had been introduced in the 19th century and that was responsible for the desertification of Australia's prized rangelands.[17]

Getting rid of the rabbits was no easy task, and after nearly a century of minimal progress using guns, dogs, and poison, the Australians finally resorted to biological warfare in the late 1940s. In 1949 Ian Clunies Ross, the director of CSIRO, ordered Ratcliffe to begin field trials of myxomatosis, a pox virus that harmlessly coexisted with South American rabbit populations but proved deadly when transmitted (either by mosquitoes or fleas) to European rabbits. Although the first few attempts at inoculation in Australia did not lead to sustained transmission, sometime in December-January 1950-51, the disease managed to escape from one of the trial sites in the Murray River Valley. Borne by mosquitoes, it spread throughout the river basins of inland New South Wales killing literally millions of rabbits. To this day, the rabbits have not regained their numbers before 1951, making the introduction of myxoma

[17] See "Ratcliffe, Francis Noble" in *Australian Dictionary of Biography* (http://www.adb.online.anu.edu.au/biogs/A160070b.htm, at 10 June 2007). On the centrality of CSIR and agricultural and industrial applications of ecology in Australia, see Martin Mulligan and Stuart Hill, *Ecological Pioneers: A Social History of Australian Ecological Thought and Action* (Cambridge: Cambridge University Press, 2001), especially chapter 7. Ratcliffe certainly knew the damage the rabbits could do from his study of desertification in the 1930s; see Ratcliffe, *Flying Fox and Drifting Sand*, pp. 323-325 for the significance of rabbits in his final report on the project.

virus one of the most successful programs of biological control ever carried out. However, the CSIRO ecologists did begin to notice revivals (and collapses) in rabbit populations in subsequent years, exactly as Elton's work might have suggested. In December 1952, a weaker strain of the virus (90% mortality as opposed to 99.8% mortality) was discovered in the Lake Urana study in New South Wales. And by the late 1950s, the mortality associated with the virus fell from 90% to 25%, and continued to fluctuate thereafter.[18]

Similar phenomena were also observed in Europe at about the same time. In June of 1952, inspired by the success of the rabbit control program in Australia, the French physician Paul Delille introduced a strain of the virus to rabbits on his country estate. The virus (thereafter known as the "Lausanne" strain of the virus) had been recently isolated in Switzerland from an infected Brazilian animal. Within months, spread by mosquitoes and also the European rabbit flea, the virus began to move rapidly across France killing between 35 and 45% of those animals infected. However, by 1955 scientists began to detect attenuated strains of the virus and despite occasional outbreaks of high virulence, overall mortality due to the disease continued to decline thereafter. Not long after the initial outbreak in France, the virus spread to England, where it was given a helping hand by farmers and foresters. Its history there closely paralleled that observed in France. By 1962, only 30% of virus samples in Britain were found to be of the highest virulence (grade V), and after another decade the original highly virulent strain had disappeared entirely.[19]

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[18] Frank Fenner and Bernardino Fantini, *Biological Control of Vertebrate Pests: The History of Myxomatosis, an Experiment in Evolution* (Oxford: Oxford University Press, 1999). See also Fenner's autobiography, *Nature, Nurture, and Chance*, chapter 6, available online at epress.anu.edu.au. The mortality figures come from Fenner and Ratcliffe, *Myxomatosis* (Cambridge: Cambridge University Press, 1965), p. 345.

[19] See Fenner and Fantini, p. 225, for a discussion of Thompson's career. The discussion of myxomatosis in France and the UK is based on Fenner and Fantini,

In Australia, the person to explore the biological causes of these subsequent fluctuations in the rabbit population was Frank Fenner, a new professor of microbiology at the John Curtin School of Medical Research in Canberra. The myxomatosis epidemic occurred at a propitious time for Fenner: he had studied virology at Australian National University in Melbourne with Macfarlane Burnet and was just then striking out on his own, looking for a new virus to make the focus of his research. Burnet had suggested that his student take over one of his projects on the genetics of influenza, but Fenner wanted to steer his own course. So when he heard of the rabbit control program and its use of myxoma virus, Fenner immediately contacted Ian Ross at CSIRO, which was also located in Canberra. With Ratcliffe and his team performing the fieldwork, Fenner and his graduate students conducted the laboratory experiments that clarified how the virus was transmitted, the immunological responses of the rabbits, and the changes in virulence of the virus from year to year. Fenner's work clarified many of the lingering mysteries left by Elton's work: how the rabbits acquired immunity to the virus, whether passive or active; the extent to which changing virus virulence or rabbit immunity was responsible for fluctuations; the significance of the mode of transmission (e.g., mosquitoes versus fleas) for the epidemiology of myxomatosis; and so forth. Their work clearly had practical applications for the management of the rabbit population in terms of finding optimal vectors for the virus in different areas, and suggesting the introduction of more virulent forms of the virus as the rabbits developed immunity.[20]

Biological Control of Vertebrate Pests, pp. 211-233. As in Australia, the leader of myxomatosis research in Britain was also a student of Elton's, Harry V. Thompson, who joined the ministry of agriculture in 1946 to direct the study of wild mammals and birds that impacted agriculture. Thompson was subsequently involved in numerous commissions that studied myxomatosis and rabbit control.

[20] These experiments are described in detail in Fenner and Fantini, *Myxomatosis* (1999); and in Fenner and Ratcliffe, *Myxomatosis* (1965).

For the most part, during the 1950s and early 1960s, this kind of laboratory study of myxomatosis remained relatively obscure – a case in the emerging field of animal virology, pioneered by Burnet and his students. This changed in 1965 when Fenner and Ratcliffe published a detailed yet highly readable account of their work, titled simply *Myxomatosis*. The work integrated, for the first time, Ratcliffe's population ecology of the rabbit, performed in the tradition of Elton, with Fenner's immunological and microbiological perspective on the disease. The crowning moment of the book came in the final chapter, when the authors presented an overview of population cycles in different species of animals: plague in African gerbils, myxomatosis in rabbits, and others. What would the future hold for such relationships between populations and their diseases? The authors identified "two possible climax associations." The first was a situation like that experienced by South American rabbits, who developed substantial resistance to an otherwise deadly disease. The second was one "in which myxomatosis still caused a moderately severe disease with an appreciable mortality, much as smallpox does in human communities." In the latter situation, the disease would lead to a never-ending sequence of population fluctuations caused by occasional outbreaks in the disease.[21]

Not surprisingly the example of myxomatosis rapidly became *the* textbook example of dynamic and evolving host-parasite systems within ecology.[22] It also rapidly became an epidemiological fact to which evolutionary biologists and historians could point in explaining the role of disease in human history. While reviewers of the book noted its relevance to virologists, professional ecologists, and other specialists, many also recognized that the work had much broader appeal:

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[21] Ratcliffe and Fenner, *Myxomatosis* (1965), p. 347.
[22] See, e.g., David Pimentel's "Population Regulation and Genetic Feedback" *Science* 159 (29 March 1968), pp. 1432-1437.

> The book's greatest appeal, however, should be to those who are interested in the natural history of disease. Without doubt man's own evolution has been greatly affected by racial experience with plagues of various types, ranging from malaria, typhus and smallpox to tuberculosis and other similar diseases; great die-offs in population create conditions favourable for evolutionary change. Nearly all virulent diseases, newly introduced, have become attenuated with time by mutual adaptations of host and parasites. The Australian investigators are to be congratulated on providing such a lucid and well-documented account of how such modifications actually take place.[23]

As the review suggests, *Myxomatosis* could be read in the tradition of *The Natural History of Disease*, the *magnum opus* of Fenner's teacher Macfarlane Burnet: as a great narrative of the emergence and recession of a disease and its host.

Fenner's intellectual trajectory after 1965 reflects the comments on the future of myxomatosis with which he closed the book, and illustrates the broader significance of disease and population in this period. In 1968, having left the laboratory the previous year to become director of the John Curtin School of Medical Research in Canberra, Fenner prepared a major revision of Burnet's *Principles of Animal Virology* (re-titled *The Biology of Animal Viruses*) that surveyed knowledge of the pathogenesis, epidemiology, and ecology of a wide range of viruses. Here myxomatosis appeared as a constant point of comparison for viruses afflicting human populations such as measles, polio, and smallpox. In discussing the evolutionary changes of virus and host, for example, he juxtaposed the ongoing saga of the myxoma virus and the rabbits with the similar story of humans and influenza viruses. Just as CSIRO established a monitoring program for rabbits and myxoma in Australia, the World Health Organization had established an Influenza Surveillance Program in 1952

[23] Lawrence Kilham, "Myxomatosis" in *Science*, New Series, Vol. 150, No. 3700. (Nov. 26, 1965), pp. 1146+1151.

that “provided the opportunity for observing continuing evolutionary changes in the influenza viruses” as it underwent regular cycles of outbreaks and pandemics.[24]

Fenner’s discussion of epidemiology in human and animal populations reveals some interesting differences. While the evolution of host and virus was a feature of both human and animal populations, the creation of new viral *diseases* (not new viruses) was generally “due to human intervention in some natural situation, or to changes in the social habits of man.” Fenner went on to note:

> Man is distinguished from all other living things by the speed with which major changes have occurred in the form of his social organization; within a few thousand years he has changed from isolated societies consisting of at the most a few hundred hunters and food-gatherers to the vast conurbations of modern man. By comparison, the social organizations of all non-domesticated animals are static. The pattern of viral infection sustained by any type of animal upon its social contacts with its fellows....[25]

Fenner then proceeds to trace out what viral diseases were likely to have flourished at different points in the grand progressive history of human social organization. Fenner was here entertaining the question of what happens when humans become rabbits, and such a move depended upon the introduction of sociality into the equation. But it was a social life of limited kind, in which long periods of stasis were punctuated by rapid transformation in modes of production. Fenner begins by discussing the epidemiology of primitive close-knit tribes of hunter-gatherers; next are the epidemics of concentrated agricultural societies.

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[24] Frank Fenner, *The Biology of Animal Viruses* Volume II (New York: Academic Press, 1968), p. 770.

[25] See Fenner, *The Biology of Animal Viruses*, pp. 778-787: “New Viruses and New Viral Diseases.” See also Fenner’s remarks in “The Effects of Changing Social Organization on the Infectious Diseases of Man” in *The Impact of Civilization on the Biology of Man* ed. S.V. Boyden (Canberra: Australian National University Press, 1970).

Agricultural societies in turn gave rise to a colonial social order, during which time a lively traffic in human and animal diseases sprang up to cause mass displacements and extinctions. The tale of the European rabbit's initial boom in Australia, followed by an equally spectacular collapse upon the introduction of a new pathogen from a foreign environment, was the quintessential example of the instabilities caused by this mode of social organization. Finally, the apotheosis of this colonial order is the freedom of movement promised by globalized air travel, which Fenner argues has the potential to undermine all geographical barriers to the spread of disease. Moreover, programs of disease eradication such as the World Health Organization's campaign against smallpox were likely to be unsuccessful because of such features of social integration on a global scale.[26] Despite his scepticism of eradication campaigns, Fenner would shortly accept an invitation to serve on the WHO committee overseeing smallpox eradication, and he would also chair the committee that certified the global eradication of the disease in 1979.[27]

Having left research behind to take the directorship of a major medical research institute, Fenner also lived vicariously by encouraging a number of his colleagues to continue the integration of ecology with the study of disease epitomized by his collaboration with Ratcliffe on *Myxomatosis*. Among these was Stephen V. Boyden, a collaborator of René Dubos at the Rockefeller Institute and former WHO program director who came to the John Curtin School in 1960. In 1966, he became

[26] See "New Viruses and New Viral Diseases" pp. 778-787. See also Fenner's remarks in "The Effects of Changing Social Organization on the Infectious Diseases of Man" in *The Impact of Civilization on the Biology of Man* ed. S.V. Boyden (Canberra: Australian National University Press, 1970).

[27] Warwick Anderson, "Natural Histories of Infectious Disease: Ecological Vision in Twentieth-Century Biomedical Science" *Osiris*, 2nd Series, Vol. 19, Landscapes of Exposure: Knowledge and Illness in Modern Environments. (2004), pp. 39-61. For Fenner's reflections on the eradication campaigns, see Fenner, "Nature, Nurture, and My Experience with Smallpox Eradication" *Medical Journal of Australia* 171 (1999), 638-41.

head of the Urban Biology Group at the Curtin School and embarked on an investigation of what he called "the biology of civilization." In this capacity, Boyden was instrumental in calling a 1968 conference in Canberra to consider "the impact of civilization on the biology of man."[28] Fenner's presentation was essentially an elaboration on the final chapter of *Biology of Animal Viruses* in which he speculated on the future of human diseases in a globalizing world. Boyden's paper, "Cultural Adaptation to Biological Maladjustment," was a clear companion piece to Fenner's, seeking to clarify the role of culture in adapting humans to disease conditions for which they were biologically maladapted. It was an important step in taking seriously the question of what facts travel when humans become the models for rabbits in disease ecology. However, as he noted, this kind of adaptation could not solve every health problem, and it liable to provoke unintended consequences that could harm heath. This work formed the basis for Boyden's concept of "eco-deviation," a "condition of life, experience by an individual or by a population, which represents a significant deviation from the conditions of life to which the species has become genetically adapted through natural selection."[29] With Fenner's encouragement, Boyden would apply this concept to study the human ecology and epidemiology of Hong Kong.

Fenner's reference to myxomatosis in his musings on the relationship between infections and social organization found an especially receptive audience among historians in the 1970s who looked to disease to structure grand narratives of human history. William McNeill's 1976 *Plagues and Peoples*, a sweeping account of the major outbreaks of disease that shaped world history, drew extensively upon

[28] For the edited conference proceedings, see S.V. Boyden, *The Impact of Civilization on the Biology of Man* (Canberra: Australian National University Press, 1970).
[29] Boyden, Sheelagh Millar, Ken Newcombe and Beverley O'Neill, *The Ecology of a City and its People: The Case of Hong Kong* (Canberra: Australian National University Press, 1981). Cited on p. 98.

the history of myxomatosis in framing its narrative. So too did Jared Diamond's later book, *Guns, Germs, and Steel* (1997). Both followed the stage model of human disease ecology—from hunter-gathers, to agricultural societies, to urban civilization—that Fenner outlined in his analysis of host-parasite evolution. But ultimately rabbits were the model upon which these accounts of disease, population, and history were based. Apart from these punctuated equilibria, there was little internal to the population that determined its history from within; rather, that history was largely determined by macro-environmental forces that structured the population from without.

In the hands of these historians, myxomatosis emerged in the 1960s and 1970s as a kind of cautionary tale for humans infatuated with the high modernist ideals lying behind the WHO's eradication campaigns. The existence of unstable futures for populations in relation to disease undermined progressive visions of history that ended in human triumph over their parasites: instead, the facts strongly suggested that mankind had to make peace with his parasites. However, the use of facts about mxyomatosis in framing histories of disease in human societies raised still more unanswerable questions about the use of "population" as a unit of analysis bridging the human-animal divide. The insistence of Fenner and his students that only humans could produce "eco-deviations" was especially problematic. Could animal populations, like humans, possess social structures that might also change in historical time? Were humans the analogue for rabbits? Or could humans be less "historical" than they seemed – less capable of choice and agency in ordering their affairs and prone to catastrophic plagues that ultimately would overthrow their political order and system of beliefs about the world? If the latter were true, then rabbits would have a lot more to tell historians such as McNeill and Diamond. These questions remained on the table as the

methodology of choice of studying populations became significantly more mathematical and theoretical during the 1970s.

From Gibbs Ensemble to Society: Robert May's Kinetics of Disease

By the late 1960s Fenner and his colleagues developed a new ecological perspective on disease that had moved some distance from Elton's aggregate ecology. Mathematical conceptions of animal populations, by contrast, remained largely wedded to metaphors and models inspired by the hegemonic science of days past: physics. Alfred Lotka's famous predator-prey equation was part of his broader program of "physical biology," which was inspired by the conceptual apparatus of thermodynamic's statistical mechanics. Here is how Lotka described this program:

> It would seem, then, that what is needed is an altogether new instrument; one that shall envisage the units of a biological population as the established statistical mechanics envisage molecules, atoms, and electrons; that shall deal with such average effects as population density, population pressure, and the like, after the manner in which thermodynamics deal with the average effects of gas concentration, gas pressures, etc.; that shall accept its problems in terms of common biological data, as thermodynamics accepts problems stated in terms of physical data; and that shall give the answer to the problem in the terms in which it was presented. [30]

From this conception of population as thermodynamic ensemble, Lotka was able to derive his famous coupled differential equations to describe the unfolding dynamics of interacting populations of predators and prey. Predators consumed prey at a rate proportional to the product of the numbers of two species present – an assumption that flowed from

[30] Alfred Lotka, *Elements of Physical Biology* (Baltimore: Williams and Wilkins Company, 1925), p. 39.

what Volterra would call the "method of encounters," the notion that individuals in a population interacted randomly like "molecules of gas in a closed container." [31] Of course, Volterra, the primary agent in spreading Lotka's approach among ecologists, was a mathematical physicist, and physicists were significant players in discussing the mathematics of the Lotka-Volterra equations in subsequent years. This literature tended to focus on developing analogies between results associated with the "ensembles" of statistical physics (for example, the ergodic theorem) and results about populations. [32]

The overlap between physics and biology is undoubtedly what drew a young physicist, Robert May, to the mathematics of populations in the 1960s. May had been a prodigy, gaining note for his work in quantum statistical mechanics and rising to full professor of theoretical physics at his *alma mater* of Sydney University (Australia) while only in his early 30s. While there, he fell in with the ecologist Charles Birch and began attending an interdisciplinary seminar with the other ecologists focused on the emerging field of population biology. According to Birch, May "thought the mathematics of the population biologists was pretty archaic and he thought he could do something about it." [33] Accordingly he went on leave to Princeton in 1972 to collaborate with Robert MacArthur, who was reckoned to be one of the most mathematically sophisticated population biologists at the time. May was only able to produce one paper with MacArthur, who died tragically young in the fall of that year, but by then

[31] See Kingsland, p. 110.
[32] See e.g. E.H. Kerner, "A Statistical Mechanics of Interacting Biological Species" *Bulletin of Mathematical Biophysics* 19 (1957), pp. 121-146; Kerner, "Further Considerations on the Statistical Mechanics of Biological Associations" *Bulletin of Mathematical Biophysics* 21 (1959), pp. 217-255; Kerner, "Gibbs Ensemble and Biological Ensemble" in *Towards a Theoretical Biology* Vol. 2, ed. CH Waddington (Edinburgh University Press, 1969), pp. 129-139; N.S. Goel, S.C. Maitra, and E.W. Montroll, "On the Volterra and other Non-Linear Models of Interacting Populations" *Review of Modern Physics* 43 (1971), pp. 231-276. Since the early 1970s, the Lotka-Volterra equations have made regular appearances in *Review of Modern Physics.*
[33] Mulligan and Hill, *Ecological Pioneers*, pp. 178-9.

he was established enough in the field with his own papers that he would assume MacArthur's chair in the Zoology department in the following year.

May's initial interest was a pretty natural one for a physicist. A number of ecologists (including MacArthur and Elton) had suggested that that the stability of population numbers was related to the trophic diversity of the food web in which the populations were embedded, and by the late 1960s this had become almost an article of faith within ecology and a justification for wholesale conservation measures. Elton's observations were purely empirical: he was convinced that rabbit populations in Canada were as volatile as they were because of the relative lack of diversity in sub-Arctic environments, whereas the bio-diverse tropics rarely witnessed the kind of extravagant carnage of the rabbit epidemics. MacArthur by contrast drew upon the entropy definition of information, suggesting that greater trophic "entropy" corresponded with greater stability over time. It was a result that a statistical physicist like May would love. But by the early 1970s, May felt he had poked some theoretical holes in the connection between diversity and stability. By examining the mathematics of the multi-species Lotka-Volterra equations, May presented "a clear caveat against any simple belief that increasing stability is a *mathematical* consequence of increasing trophic web complexity."[34] Adding more trophic links was not like adding more molecules to a thermodynamical system: the fluctuations in macroscopic variables did not get proportionally smaller. In 1974, May published the results of this work in his landmark *Stability and Complexity in Model Ecosystems*.[35]

[34] Robert May, "Stability in Multispecies Community Models," *Mathematical Biosciences* 12 (1971), pp. 59-79; cited on p. 62.
[35] Robert May, *Stability and Complexity in Model Ecosystems* (Princeton, NJ: Princeton University Press 1974).

If the stable regulation of population numbers was not simply a combinatorial outcome of trophic diversity, then what actually did regulate populations? May was convinced that the answer would emerge from a systematic investigation of the co-evolution of populations and their diseases and parasites. Assuming the traditional statistical view of populations embraced by both population genetics and epidemiology, as homogeneous randomly mixing and evolving collectives, May asked: what would be the future of a population of simultaneously evolving animal hosts and parasites? Would the disease evolve avirulence, would the host population develop immunity, or would the equilibrium yield unstable population fluctuations? Although May did not mention myxomatosis, it was precisely these questions that Ratcliffe and Fenner had asked (but had not been able to answer in generality) in the closing chapter of their 1965 book. In this vein, May began collaboration with Roy Anderson, an epidemiologist at King's College and an expert on tropical diseases such as helminth infections. Their initial work culminated in a pair of articles published in the *Journal of Animal Ecology* in 1978 suggesting that, from a mathematical perspective, evolving host-parasite systems could spawn a dizzying array of mathematically plausible patterns.[36]

However, doubtless under Anderson's influence, May's focus began to move from his initial theorizing about the effects of disease on population stability to more applied questions concerning the dynamics of disease transmission in *human* populations. In two review articles

[36] See e.g., Roy Anderson and Robert May, "Regulation and Stability of Host-Parasite Population Interactions, I. Regulatory Processes" *Journal of Animal Ecology* 47 (1978), pp. 219-247; and Anderson and May, "Regulation and Stability of Host-Parasite Population Interactions, II. Destabilizing Processes" *Journal of Animal Ecology* 47 (1978), pp. 249-267. May was far from the only person theorizing about the dynamics of host-parasite interaction in the 1970s; see also Park's experiments on *Tribolium* and the subsequent theoretical work of Levine and Pimentel (described in Pimentel, *op. cit*.)

published in *Nature* in 1979,[37] Anderson and May called attention to the implications of theoretical population biology for the management of disease in human populations. While they noted that interest in the role of disease in regulating animal populations had increased during the 1970s, still relatively few works had examined the relevance of this literature to human populations. Indeed as they noted "such phenomena are largely responsible for the dramatic differences between age-specific survival probabilities for people in developed and underdeveloped countries." Moreover, citing the historical works of William McNeill and Frank Fenner's article in *The Impact of Civilization on the Biology of Man,* they noted that "the broader patterns of human history are to be interpreted in terms of the evolving relationships between man and his diseases."[38] To explain these patterns, it was essential to move from the perspective of traditional epidemiology – in which host and parasite populations did not evolve or change their structure or behaviour – toward a more dynamic and richly detailed view of populations. Finally, following up on the *Nature* manifesto, Anderson and May organized a conference, held in Berlin in the spring of 1982, on "Population Biology of Infectious Diseases." The conference was essentially the Anderson-May collaboration writ large, bringing together epidemiologists and public health practitioners – including Fenner, fresh from his leading role in the global smallpox eradication campaign – and evolutionary modellers such as May and William D. Hamilton.

The emphasis of the conference lay on overcoming the disciplinary barriers that prevented a synthetic population biology of infectious disease from forming. Most significantly, as May noted in his introduction, practical programs of disease control were often conducted on the basis

[37] Anderson and May, "Population Biology of Infectious Diseases: Part I" *Nature* 280 (2 August 1979), pp. 361-367; and Robert May and Roy Anderson, "Population Biology of Infectious Diseases: Part II" *Nature* 280 (9 August 1979), pp. 455-461.
[38] Anderson and May, "Population Biology of Infectious Diseases: Part I" p. 361.

of intuition without input from modellers. While readily available technical measures for controlling a disease (e.g., vaccines) could lead health officials to conclude that mass eradication was possible, more ecologically sensitive models could show that eradication efforts might well make the overall incidence of disease worse. One major case study of the volume, the failure of the World Health Organization's malaria eradication campaign in the 1950s and 1960s, made precisely this point. Early models of malaria transmission by British epidemiologists Ross and Macdonald had turned the tide of professional opinion in favour of eradication, but they failed to take into account a number of critical complications, including the effects of concentrated non-compliance with vaccination regimens, and the evolution of mosquitoes resistant to DDT. What was needed was a new generation of models that encompassed the behaviour of host populations, from sanitation habits to sexual practices, the ecology of their environs, and the co-evolution of the host and the parasite. To meet the challenges set by global disease control organizations like the WHO, human and animal populations needed to be studied within the same modelling framework.[39]

While control of disease in human populations was clearly the ultimate goal of the Berlin conference, animal diseases – and myxomatosis in particular – also received a great deal of attention. The conference report on the "Evolution of Parasites and Hosts" devoted many pages to a discussion of Fenner and Ratcliffe's myxomatosis research effort, holding it up as a model for the study of host-parasite interaction. Not surprisingly, in the years that followed, Anderson and May drew upon Fenner's data to provide empirical grounding for their models. Myxomatosis provided an example of a particular evolutionary strategy

[39] On the failure of the malaria eradication program, see especially PEM Fine et al., "The Control of Infectious Disease Group Report" in *Population Biology of Infectious Diseases* eds. RM Anderson and RM May (New York: Springer Verlag, 1982), especially pp. 132-135.

that the virus could pursue, based on the relationship between its virulence, transmissivity, and the recovery rates of infected individuals. Using historical data from both Australia and England, it was possible to explore the relationship between these quantities for a given host-parasite pair to determine the set of possible co-evolutionary trajectories.[40] By the early 1990s, when he was called upon to consider to implications of his modelling for emerging viruses such as HIV, he would note that "Frank Fenner is the real hero of this part of my essay,"[41] for providing the first such data for constructing phenomeno-logical models of host-virus co-evolution – models that would subsequently be tested against human-disease co-evolution.

Most significantly, the conference seems to have marked a final step in May's gradual shift away from his initial conception of population as an ensemble of statistical mechanics toward something more closely resembling a "community" or "society." Certainly, *human* populations were characterized by facts about their social structure, a point made most eloquently by Anderson and May in a 1982 article outlining strategies for control of schistosomiasis in developing countries. This disease was especially problematic since it cast a wide ecological web linking sewage disposal, snail populations, and water use issues, among others, so that traditional vaccination and eradication campaigns were not always an optimal approach. As Anderson and May put it, "Human helminth infections are good examples of the discrepancy between our knowledge of how to treat an individual and how to control the infection in a community." Selective treatment, targeted at key social groups affected in the transmission cycle, was therefore advantageous. "These benefits,"

[40] See Anderson and May, "Coevolution of Hosts and Parasites" *Parasitology* 85 (1982), pp. 411-426; and Anderson and May, "Epidemiology and Genetics in the Coevolution of Parasites and Hosts" *Proc. Roy. Soc. Lond. B.* 219.1216 (22 October 1983), pp. 181-313.
[41] May, "Ecology and Evolution of Host-Virus Associations" in *Emerging Viruses* ed. Stephen Morse (New York: Oxford, 1993), pp. 58-68.

they concluded, “will be further enhanced if, as has been suggested, the heavily infected individuals are predisposed to this state, not by the laws of chance, but as a consequence of genetic, behavioural or social factors.” [42] The elements of the population were no longer Lotka’s molecules but socially interacting individuals.

Finally, it is worth noting that this conception of “population” as “society” was not confined in May’s mind to collectives of humans afflicted with helminth parasites. When two scientists from the UK’s ministry of agriculture presented the Royal Society with a historical review of myxomatosis in England, May and Anderson were two of the most eager discussants afterward. The talk seemed to indicate that rabbit resistance was increasing more rapidly in Britain than in Australia, but why? Suggested May: “Behavioural changes are also alleged in Britain, for example involving rabbits living above ground and hence in a less suitable habit for a flea vector.” The ministry scientists said they knew of no such changes, although they suggested (with May concurring) that it would make a very interesting study. Similarly, when the suggestion arose to introduce flea vectors in New Zealand to improve rabbit control, May noted: “Even if a vector were introduced, there would not necessarily be transmission. In birds there are quite sensitive dependencies between group living patterns and densities and the propagation of disease.”[43] The remaining rabbits in New Zealand may well have established new social orders that would defeat the best efforts of human wildlife managers. In May’s mind, the rabbits were on the brink of humanity – and perhaps vice versa as well.

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[42] Anderson and May, “Population Dynamics of Human Helminth Infections: Control by Chemotherapy” *Nature* 297 (17 June 1982), pp. 557-563; cited on p. 563.
[43] J. Ross and A.M. Tittensor, “The Establishment and Spread of Myxomatosis and its Effect on Rabbit Populations” *Phil. Trans. R. Soc. Lond.* B 314 (1986), pp. 599-606; cited on pp. 602-3.

Conclusion: Facts about Populations

At what point did rabbits become human, or humans rabbits? Reviewing the history related here it becomes clear that transfer of facts from rabbit populations to human populations (and vice versa) occurred in stages and is still ongoing. Charles Elton and his economic doppelganger Henry Ludwell Moore (as channelled through Ellsworth Huntington at the Matamek Conference) were able to share some facts about populations, but not others. They could agree on a vision of populations as aggregates, whose behaviour in time is assumed to be driven by events outside themselves, whether sunspots or inexplicable revolutionary shifts in the mode of production. By contrast, other facets of populations – their resemblance to "communities" or "economies" possessing history, memory, social roles and behavioural interactions – did not travel as well across the human-rabbit divide. As we saw in subsequent conceptions of population, scientists were halting in their attempts to draw realistic lessons for the history (and future) of humanity from the experience of rabbit populations. When they initially did, accounts such as those authored by McNeill and Diamond tended to exhibit a rigid determinism: they were the histories of herds, not the histories of societies or civilizations. It may only be in the work of ecologically-minded modellers of disease, such as May, that an unbiased traffic in facts between human and animal societies is taking place. Yet even here, the outcome is not yet clear. Are rabbits looking more like humans, or are humans looking more like rabbits? With the burgeoning field of disease ecology poised to become the *de facto* welfare economics for an age of globalized disease threats, this question merits much more attention than it has yet received.

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