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Nowhere to run, rabbit: the cold-war calculus of disease ecology

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Abstract During the cold war, Frank Fenner (protégé of Macfarlane Burnet and René Dubos) and Francis Ratcliffe (associate of A. J. Nicholson and student of Charles Elton) studied mathematically the coevolution of host resistance and parasite virulence when myxomatosis was unleashed on Australia's rabbit population. Later, Robert May called Fenner the "real hero" of disease ecology for his mathematical modeling of the epidemic. While Ratcliffe came from a tradition of animal ecology, Fenner developed an ecological orientation in World War II through his work on malaria control (with Ratcliffe and Ian Mackerras, among others)—that is, through studies of tropical medicine. This makes Fenner at least a partial exception to other senior disease ecologists in the region, most of whom learned their ecology from examining responses to agricultural challenges and animal husbandry problems in settler colonial society. Here I consider the local ecologies of knowledge in southeastern Australia during this period, and describe the particular cold-war intellectual niche that Fenner and Ratcliffe inhabited.

Keywords Disease ecology · Epidemiology · Myxomatosis · Cold war biology · Invasion ecologies

1 Introduction

Six weeks before the end of World War II in Asia, and a couple of months after war stopped in Europe, virologist F. Macfarlane Burnet was proposing a laboratory experiment in epidemiology, a study that might illuminate how and why viruses spread. As director of the Walter and Eliza Hall Institute in Melbourne, Australia, Burnet had spent the previous decade trying to render the statistical analysis of

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infectious disease patterns more biologically complex—that is, he sought to align microbiology with evolutionary theory and ecological reasoning. Efforts to merge bacteriology with animal ecology, thereby revealing microbial population dynamics and interactions, had culminated in publication in 1940 of his *Biological Aspects of Infectious Disease*, a pioneering treatise in what became known as disease ecology (Burnet 1940; Anderson 2004, 2016). In July 1945, Burnet wrote to a promising young malaria researcher in the Australian Army: "There is a specially good opportunity in experimental epidemiology for the study of a virus disease."[1] Thus he hoped to lure Frank Fenner to the Hall Institute to investigate ectromelia virus in mice. British epidemiologist Major Greenwood had experimented with the virus before the war but did not know then of its relations to smallpox, which made fresh studies vital (Greenwood et al. 1936; Hardy and Magnello 2002; Amsterdamska 2005).[2] "I am very interested in the host-parasite relationship in all its aspects," Fenner responded from northwest Borneo, "and have been working away along these lines."[3] Burnet's invitation set Fenner on a research trajectory that began with experimental epidemiology in Melbourne, passed through mycobacterial inquiries with René Dubos at the Rockefeller Institute in New York City, and ended at the new Australian National University in Canberra where, as the first professor of microbiology, he would investigate viral ecology and, eventually, environmental degradation. Burnet remained an insistent guide and mentor for most of this period. Perhaps most pertinently, around 1950 Burnet urged his protégé to look into the outbreak of a viral disease of rabbits called myxomatosis, which was wreaking havoc among the Australian vermin. The emergent disease, Burnet asserted in an echo of his earlier summons to Fenner, might constitute an unparalleled experiment in epidemiology, only this time in the field.

Through his statistical studies in the 1950s of the natural history of the myxoma virus in rabbit hosts, Fenner would become for population biologist Robert M. May "the real hero" of disease ecology. The "moral" of his research was "that the co-evolutionary trajectories pursued by virus-host associations, and more generally by most pathogen-host associations, involve complicated trade offs between virulence, transmissibility, and host resistance" (May 1993: pp. 63, 64; May and Anderson 1982).[4] Fenner himself later declared that myxomatosis was "the best natural experiment on the co-evolution of viral virulence and host resistance available for a disease of vertebrates" (Fenner 1999: p. 639). His research into parasite-host relations in outback Australia came to inform popular accounts of disease ecology and species invasion, such as William H. McNeill's *Plagues and Peoples* (1976), Alfred W. Crosby's *Columbian Exchange* (1972), and Eric C. Rolls's *They All Ran*

[1] Burnet to Fenner, 2 July 1945, folder 143/6/1, box 3, F. J. Fenner papers, MS 143, Basser Library, Australian Academy of Science, Canberra, Australia (Fenner papers).

[2] Greenwood was professor of epidemiology and vital statistics at the London School of Hygiene and Tropical Medicine.

[3] Fenner to Burnet, 10 July 1945, folder 143/6/1, box 3, Fenner papers. See Fenner (1948). See also Fenner (2006) and Anderson (2013).

[4] Myxoma is a poxvirus, like ectromelia and smallpox, which causes skin tumors, blindness, fatigue and fever in European rabbits, usually resulting in death within twelve days. The virus was discovered in Uruguay in the late-nineteenth century.

Wild (1969). As historians Paul Erickson and Gregg Mitman suggest, "myxomatosis emerged in the 1960s and 1970s as a kind of cautionary tale for humans" (Erickson and Mitman 2007: p. 22). In the following decade, the advent of frightening new diseases, such as acquired immunodeficiency syndrome, prompted even greater interest in the ecological dimensions of microbiology (King 2004). The lesson was more redoubtable with each telling. Myxomatosis became the classic case study of parasite-host interaction—so much so that one might suppose that we are all rabbits now, with all our pathogens condensed into a single predatory poxvirus. The apparent homology should cause us to ask, with Erickson and Mitman, what might humans gain and what might we lose when we become rabbits? In tentatively answering this question, Erickson and Mitman focus shrewdly on the reframing of populations, on the transforming of society or community into biological collective or herd. Here I want to supplement their analysis by looking more closely at cold-war translations of myxomatosis modeling into precepts of human biosecurity, surveillance, and preparedness.

Since historians of science and medicine belatedly turned to the study of disease ecology, they have described multiple influences on the field's formation and composition, a veritable ecology of knowledge (Rosenberg 1979). In a provocative essay, Andrew Mendelsohn linked rising interwar interest in ecological interpretations of infectious disease to perceptions of epidemiological inadequacy in response to the influenza pandemic. As a result, leading statisticians such as Greenwood sought greater biological realism and complexity through experimental epidemiology (Mendelsohn 1998). That is, they framed epidemics as natural "configuration," not simply as "contamination" (Rosenberg 1992). Others have traced ecological inroads into general medicine back to early parasitology and tropical medicine, investigatory enterprises that are inherently complex, requiring attention to multiple natural actors—parasites, hosts, and vectors—in various environmental niches (Worboys 1988; Tilley 2011).[5] Recently, historians have considered more proximate institutional and practical pressures on the development of ecological frameworks for infectious diseases research. In the United States, involvement in veterinary and agricultural problems, mostly through state bureaus, fostered the ecological engagement of biologists like Theobald Smith and Karl Friedrich Meyer, and permitted a crossover into human concerns (Dolman and Wolfe 2003; Honigsbaum 2016). In Australia, the smaller scientific community was densely interwoven and interdependent, so medical research often articulated with veterinary and agricultural investigation, and different disciplines evolved a sort of mutualism. Thus Burnet saw himself as a natural historian as much as a medical scientist; his infectious diseases research included humans, birds, and other animals; and he advised the Council of Scientific and Industrial Research (CSIR) on its veterinary and agricultural inquiries, including its pest control projects. Burnet's biological enthusiasms and entanglements, together with institutional and practical demands, meant he was conversant with thriving Australian research in animal ecology, in particular A. J. Nicholson's work in Canberra, and familiar with the ecological reasoning of British savants such as Julian Huxley and Charles Elton

[5] For challenges to this argument, see Mendelsohn (1998) and Farley (1992).

(Anderson 2016). As we learn more about the multiple local origins of global disease ecology, we have come to appreciate its dependence on imperial cares and liabilities, and additionally its investment, often conflicted, in settler colonial development projects in places like the United States and Australia.[6]

The profusion of explanations for the inception of disease ecology before World War II contrasts with the deficiency in our knowledge of how the field matured and expanded after the war. Tracking Fenner's involvement in the ecological analysis of the myxomatosis epidemic among Australian rabbits offers us a revealing view onto post-war patterns of infectious diseases research. It shows the congruence of an ecological mind-set with prevailing cold-war thought styles, which were saturated with concerns about threat, surveillance, infiltration, and containment. It should be recalled that pest control in Australia during this period, including the deliberate liberation of myxoma virus, was a form of biological warfare. In attempting to acquire epidemic intelligence and to devise complex methods of disease surveillance and prediction, Fenner's ecological studies of myxomatosis offered techniques that could be applied equally well to biological warfare among humans (Dando 1999). During the cold war, disease ecology contributed to what anthropologists Stephen J. Collier and Andrew Lakoff have described as the "rationality of preparedness" in civil defense and biosecurity planning (Collier and Lakoff 2007, 2008; Fearnley 2010). Thus disease ecology was not only a colonial science: it took shape and grew apace in the cold war too. Fenner's rabbits were war casualties.

2 Biological control of rabbit vermin

As a haughty and conceited young Englishman, Francis N. Ratcliffe had first visited Australia in 1929 to survey native fruit bats, or flying foxes, in the northern tropics. A student of Elton and Huxley at Oxford, he was well equipped for the task. Ratcliffe soon became engrossed in the continent's natural history, though he flayed its degenerate white settlers, most of them "useless rotters."[7] "The Australian," he wrote, "is a foul-mouthed, incorrigibly lazy waster, whose chief pastimes are drinking and betting on horses." Organized labor seemed to impede any progress. "Unfortunately the independent, arrogant spirit of the people precludes the possibility of a Mussolini," he announced. "If one did appear, I may say, I should be one of the first to put on a black shirt."[8] Fond of biological scouting, Ratcliffe toured the country and met nearly all its zoologists, including Nicholson in the CSIR economic entomology division in Canberra, who was devising an intriguing model for the population dynamics of predators and their prey (Nicholson 1933; Kingsland 1996). Ratcliffe returned to England but Australian nature continued to captivate him. In 1935, he took up a new job at the expanding CSIR, based in Canberra with Nicholson, studying termites and pests of stored wheat, but finding

[6] This was anticipated in Grove (1995). See also Griffiths and Robin (1997) and Anker (2001).

[7] Ratcliffe to People [Family], 4 June 1929, box 8, Francis N. Ratcliffe papers, MS 2493, National Library of Australia, Canberra (Ratcliffe papers). A friend of Roy Campbell, the poet and fascist sympathiser, Ratcliffe's father Samuel was a conservative journalist and freethinker who dedicated his life to explaining England to Americans. See Powell (2001) and Mulligan and Hill (2001).

[8] Ratcliffe to Mother, 4 October 1929, box 8, Ratcliffe papers.

opportunities for excursions to survey soil erosion in central Australia (Schedvin 1984, 1987). This time he experienced in his adventures the terrifying harshness of the outback, and came to appreciate the hardiness and generosity of struggling white inhabitants. "We seemed to be looking around the bend of the earth," he wrote of his time in the red center, "later I was to be really scared—scared that something in my mind would crack, that the last shreds of my self-control would snap and leave me raving mad" (Ratcliffe 1938: p. 260). The sensitive Englishman pulled himself together sufficiently to describe his sojourn in the Never–Never in *Flying Fox and Drifting Sand* (1938), perhaps the most compelling of pre-war environmental narratives.

During the Pacific war, Ratcliffe was assigned as an entomologist to Land Headquarters in Cairns, in northern Queensland, where he investigated the transmission of malaria, scrub typhus, and dengue fever. He worked closely with fellow entomologist Ian M. Mackerras, formerly his senior colleague in Canberra, and came to know Fenner who was involved in the related atebrin (mepacrine) antimalarial trials among Australian troops (Anderson 2013; Walker 1952; Spencer 1994; Condon-Rall 2000; Sweeney 2003). Mackerras and Ratcliffe disdained the clinical researchers who, with the exception of Fenner, showed little understanding of the intricacies of mosquito ecology. In response, E. V. "Bill" Keogh, the wily director of army hygiene and pathology, deplored the ecologists' "irritating qualities" and "administrative deficiencies." He got the impression they believed they were the sole experts on malaria control.[9] He felt Radcliffe was "a good fellow but still has to learn that plenty of people with not quite his intellect know more about how to get results, crudely expressed and not quite what I mean which is really he can do with taking down a peg or two."[10] The top army medical brass may have slighted the entomologists' obsession with mosquito behavior, but Fenner continued to observe them intently, and attempted to learn more natural history.

After the war, Ratcliffe returned to the CSIR (from 1949 the CSIRO) in Canberra, where he set up the wildlife survey section, later the division of wildlife and ecology, a task that fixed his attention on the environmental devastation wrought by invasive rabbits. Introduced in the middle of the nineteenth century, rabbits had proliferated across the continent, running amok and destroying fragile ecosystems. For decades the Australian government had experimented with biological control mechanisms for invasive pests and vermin, so Ratcliffe, conforming to this institutional logic, or default procedure, began in the late 1940s to speculate on possible natural methods to exterminate rabbits.[11] "The

[9] Keogh to Ted Ford, 29 October [1943], Edward Ford papers, Royal Australasian College of Physicians, Sydney (Ford papers). A graduate in medicine and zoology from Sydney, and an expert in veterinary entomology and parasitology, Mackerras was appointed after the war as the first director of the Queensland Institute of Medical Research. As a Melbourne medical researcher before the war, Keogh was a rival of Burnet, and later he dedicated himself—through the army, philanthropic foundations, and the Anti-Cancer Council of Victoria—to promoting the research careers of the promising young men he befriended. Another Melbourne medical graduate, Ford became director of the School of Public Health and Tropical Medicine, dean of the Faculty of Medicine, and acting vice-chancellor of the University of Sydney.

[10] Keogh to Ted Ford, I October 1942, Ford papers.

[11] A compelling precedent was the introduction of the *Cactoblastis cactorum* moth from Argentina in the 1920s, which nearly eliminated prickly pear (*Opuntia*) from the brigalow country of Queensland and NSW. See Rolls (1969); Frawley and McCalman (2014); and Deveson (2016).

decision I came to was a purely personal one," he recollected, "made in solitude one might say." He had heard of earlier attempts to introduce myxoma virus into rabbit populations, which seemed uniquely vulnerable to that microorganism, but wondered how to contrive an epidemic across the country. As early as 1919, at the end of the influenza pandemic, a Brazilian virologist, Henrique de Beaurepaire Aragão, had written to Anton Breinl, the director of the Australian Institute of Tropical Medicine in Townsville, offering him some samples of myxoma virus to use against rabbit infestation. While based at the Fundação Oswaldo Cruz, Aragão conducted experiments around Rio de Janeiro, demonstrating that the virus killed rabbits without harming humans and other animals—but senior Australian physicians urged caution. Ever persistent, Aragão sent further virus samples to the New South Wales Department of Agriculture through the 1920s, to no avail. The state pest control experts surmised that the virus was not contagious and therefore could not spread readily through the rabbit population.[12] In 1934, Aragão tried again, mailing the frozen virus to C. J. Martin, the former head of the CSIR"s division of animal nutrition, who was living in Cambridge, England. With the continuing support of the CSIR, Martin undertook some desultory experiments in the English countryside (Martin 1936), then sent the strain to Lionel B. Bull, an overbearing, self-confident veterinary scientist, chief of the CSIR"s division of animal health in Melbourne.[13] Bull began trials of the myxoma virus on an Aboriginal reserve in the remote Spencer Gulf area of South Australia. "I was the first to conceive the idea," Bull recalled, "that a winged insect vector might make the spread of the disease sufficiently wide and fast to prove of practical value in the control of rabbit populations in the field."[14] But he was constrained to test the hypothesis in the desert, with no mosquitoes and only scattered fleas. "We took the view," Bull later wrote, "that myxomatosis might prove successful, even if its range were restricted, if used in regions where mosquitoes and possibly other winged insects would probably act as vectors."[15] But rabbit control was not the priority during World War II, so research lapsed.

Around 1950, Ratcliffe decided to try again to spread myxoma virus among rabbits. Although claiming to have conceived the program in solitary contemplation, he also was aware of Melbourne microbiologist Jean Macnamara's strident campaigning for action against the vermin. While working in Richard Shope's laboratory at the Rockefeller Institute in New York City she had learned about myxomatosis and its potential to control rabbits. In 1949, she wrote bluntly to

[12] For documentation of these early attempts, see folder 143/9/3, box 5, Fenner papers. On Breinl and the Institute of Tropical Medicine, see Anderson (2006). A conservative nationalist, Aragão became director of the Oswaldo Cruz Institute in the 1940s.

[13] Before becoming chief of the CSIR's division of animal nutrition in Adelaide (1930–33), Martin had taught physiology at the University of Melbourne and was director of the Lister Institute of Preventive Medicine, London (1903-30), where Burnet obtained his Ph.D. Burnet and Dora Lush at the Hall Institute had also begun to experiment with myxoma virus before the war: see Lush (1937). Bull also obtained a virulent strain of the virus from Shope's laboratory.

[14] Bull to Fenner, 13 December 1961, folder 143/9/1, box 5, Fenner papers. The Commonwealth director-general of health, J.H.L. Cumpston, who had reluctantly approved the importation of the virus, constrained the choice of experimental site.

[15] Bull to Fenner, 3 August 1955, folder 143/9/1, box 5, Fenner papers. See Bull & Mules (1944).

Melbourne newspapers criticizing what she thought was Bull's equivocation over the efficacy of myxoma virus, and demanding further trials.[16] After the release of the virus, Macnamara would turn her attention to Ratcliffe, castigating him for any reservations he might express about the program, and for his failure to give her proper credit. "Myxomatosis meant a great deal to her," Ratcliffe recollected, "and she regarded the scientists" job not to point out the limitations of the virus but to find ways of overcoming them. In her eyes, I had stood in the way of maximum development." Macnamara seemed to view him, and anyone else with the temerity to resist her, as "a boil on the bum of progress." "Until I had dealings with her, and saw her in operation in front of and behind the scenes," Ratcliffe wrote, "I don't think I knew the real meaning of vindictiveness."[17] For the remainder of his career, the bickering over myxomatosis would rankle. "I have come to the conclusion that the virus of myxomatosis is rather like a bad-luck fetish," Ratcliffe told the chairman of the Commonwealth Scientific and Industrial Research Organisation (CSIRO) in 1968. "It brings trouble and human discord to those who get involved with it."[18]

3 Mosquito populations and disease ecology

Ratcliffe arranged for the release of myxoma virus at a couple of field sites in southeastern Australia, mostly along the Murray River, in the autumn and winter of 1950. For months he waited. At first nothing happened, then at the end of the year he heard of a few myxomatosis cases some fifteen miles from the last field experiment. Within a few weeks, rabbits were succumbing all over New South Wales, especially along the watercourses (Ratcliffe et al. 1952). Early in 1951 Bull approached Burnet asking if he could take charge of the associated laboratory studies of changing myxoma virulence (or capacity to kill) and host resistance (manifested in case mortality), but Burnet, after consulting his friend Ian Clunies Ross, the chairman of the CSIRO, instead nominated his protégé Fenner. At a meeting on February 9, 1951, Burnet, Ratcliffe and Fenner accepted there "is a need for ecological study of inland mosquitoes" and agreed that Fenner "should make myxoma a major job."[19] In a few weeks, Fenner wrote to his mentor Dubos, explaining that he was "deflected from the mycobacteria by the extraordinary outbreak of myxomatosis." He went on:

> Mosquitoes are the important element in the local epizootics, and it seems that they must have been responsible for this astonishing spread. I intend to keep

[16] Melbourne *Herald* (11 May 1949). Macnamara had worked in the 1920s and 1930s with Burnet, differentiating strains of poliomyelitis virus and studying psittacosis. See Zwar (1984).

[17] Ratcliffe to Douglas Stewart, 28 February 1968, folder 143/9/1, I 1-3, box 4, Fenner papers. Stewart published Rolls (1969), which was full of praise for Macnamara and criticism of Bull and Ratcliffe.

[18] Ratcliffe to Frederick White, 28 February 1968, folder 143/9/1, I 1-3, box 4, Fenner papers.

[19] Extracts from Burnet diary, 9 February 1951, folder 143/9/1B, box 4, Fenner papers. As a veterinary scientist with the CSIR in Sydney, Clunies Ross studied host-parasite relations, especially sheep and hydatids, before the war.

> track of the virus and see whether any sign occurs of an alteration in the present 100% lethality of the virus for the rabbit. Over a couple of years it should be very interesting.[20]

Dubos was intrigued. "For all the reasons that you know," he responded, "I am most interested in your story of the outbreak of myxomatosis."[21] Later, he observed: "The advocates of the importance of biological warfare will be heartened by [your] findings. Epidemiologists will be eager to learn whether the epizootic carries over from one season to the other, and how soon a resistant population of rabbits will develop. You have some fine problems in natural resistance and immunity ahead of you."[22] The correspondence on the virus outbreak continued for years. "We now have quite a good set-up," Fenner wrote in 1952, "for following the evolution of the virus and host." "Are the rabbits becoming immune, or naturally resistant," Dubos asked insistently in 1954, "is the virus changing?"[23] The potential for tracking changes in host resistance and parasite virulence during the epizootic fascinated both scientists.

Fenner was well prepared for ecological research into a disease outbreak. The son of a geographer, from a nationalist family who shared the peculiar pre-war South Australian enthusiasm for literary fascism, Fenner had trained with the anthropologically minded medical professors at the University of Adelaide, most of them staunch, if outmoded, votaries of Lamarckian natural history (Anderson 2006).[24] At the beginning of the war, he had spent three months at the Sydney School of Public Health and Tropical Medicine, learning about malaria from Edward Ford, the cultivated, quizzical lecturer in bacteriology. With the army in Palestine, Fenner studied modern methods of malaria diagnosis in Saul Adler's laboratory at Hadassah Hospital. He worked excitedly on plasmodia, the malaria parasites, in between bouts with sand-fly fever and visits to Tel Aviv to listen to the orchestra play Brahms. "Working up there [in Jerusalem] for a few days revives my desire to do Trop Med Research rather than anatomy," he wrote home.[25] In 1942, promoted to major and transferred as physician and pathologist to the 2/2nd General Hospital in Cairns, Fenner tended to patients sent down from Papua and New Guinea with malaria and dysentery. Known as "Noffie" for his obsession with the *Anopheles* mosquito, which spreads malaria, he became a malariologist, reporting to Ford and cooperating with Mackerras and Ratcliffe.[26] "Everything about malaria," wrote

[20] Fenner to Dubos, 1 March 1951, folder 143/7, box 3, Fenner papers.

[21] Dubos to Fenner, 29 March 1951, folder 143/7, box 3, Fenner papers.

[22] Dubos to Fenner, 18 September 1951, folder 143/7/2A, box 3, Fenner papers.

[23] Fenner to Dubos, 17 November 1952, and Dubos to Fenner, 1 September 1954, folder 143/7, box 3, Fenner papers. In 1954, Dubos read Ratcliffe (1938), finding it "most interesting." See also Dubos (1958).

[24] The Lamarckian anatomist and anthropologist Frederic Wood Jones was a strong influence. Wood Jones introduced Fenner to his friend Ford. Fenner's family was loosely associated with the Jindyworobaks and the Australia First movement, though active in neither.

[25] Fenner to Everyone, 29 May 1941, folder 3C, box 4, Fenner papers.

[26] Keogh found Fenner frustrating at times: I "am worried about him [Noffie]," he wrote to Ford, "He is a pigheaded little bugger, fond and all as I am of him" (Keogh to Ford, February 1943, Ford papers). On connections between malaria research and disease ecology in this period, see Way (2015).

Lewis W. Hackett, an expert whom Fenner admired, "is so molded and altered by local conditions that it becomes a thousand different diseases and epidemiological puzzles. Like chess, it is played with a few pieces, but is capable of an infinite variety of solutions" (Hackett 1937: p. 266). Studying malaria in tropical Australia during the war fostered in Fenner an ecological sensibility, which further post-war experience with Burnet and Dubos would entrench.

4 Fieldwork correlated with some first class lab work

From 1950, Fenner took charge of the pathological and serological investigation of the rabbits, while Ratcliffe organized the field studies. Agents of the wildlife survey section inoculated rabbits with the myxoma virus at select sites, generally places where mosquitoes normally abounded. They assessed rabbit density, predation, and behavior, making daily counts of diseased and dead rabbits. As the epizootic gathered momentum, it swept through rabbit populations, with incredibly high mortality rates. Within months, it covered the Murray-Darling river basin, 1500 km from north to south and 1800 km from east to west. The effect on the rabbit population was catastrophic. Entomologists determined that the summer mosquito *Culex annulirostris* was responsible for transmission of the virus in this unusually dry season. Evidently the movement of infected insects was rapid and extended, more so than anyone had expected. With winter, the river-land epizootic diminished, though sporadic intense outbreaks occurred further afield, perhaps spread by *Aedes camptorhynchus*, another rabbit-biting mosquito. In later wetter seasons, *Anopheles annulipes*, a favored vector in such conditions, took over transmission. It soon became clear that myxomatosis would become enzootic in Australia, its distribution co-extensive with the dwindling presence of rabbits (Fenner 1952, 1953; Fenner and Ratcliffe 1965; see also Fenner and Fantini 1999; Bartrip 2008).

One of the wildlife officers involved in virus liberation and rabbit surveys later reflected on "the raw inexperience of Ratcliffe's team in regard to the research problems it had to deal with. There was a critical shortage of graduates in the years following the war and Francis was lucky to get what he got. But it left him holding a pretty weak hand in the first few years."[27] A zoology graduate from Sydney, Kenneth Myers had served with the Royal Australian Air Force (RAAF) intelligence unit in the Pacific, doing photographic reconnaissance, before his recruitment into myxomatosis studies in northeast Victoria and the Riverina district, where he focused on Lake Urana.[28] Myers would send Fenner as many rabbits and mosquitoes as he could muster, but the microbiologist always demanded more. In 1951 Fenner told Myers:

[27] Myers to Fenner, 8 May 1998, folder 143/9/9, box 7, Fenner papers.

[28] Ratcliffe's team included biochemists John H. Calaby and M. Lazarus; agricultural scientist Bernard V. Fennessy; veterinary scientist Roman Mykytowycz; and zoologists Myers and R. Brereton. Myers was later professor of zoology at the University of Guelph, Canada, but returned after ten years there to work again at the CSIRO.

> We would like any living wild rabbits which show evidence of having recovered from myxomatosis. Such animals would be useful both for providing us with stocks of immune serum, and for determining the rate of decline with various types of antibody to myxoma virus, essential preliminaries to serological surveys in the field.[29]

When Myers was not collecting rabbits, he snared mosquitoes. "My programme for the coming summer," he wrote, "entails night after night of exposure to mosquitoes down the river—I think I'll be the most bitten man in Albury [NSW] when this is over."[30] He wrote again to Fenner:

> It will take me all my time to supply you with mossies in the required numbers…. I plan to do as full a study as time will allow on adult movements and feeding performances of the river mosquitoes and their connection with disease performance.[31]

The wildlife officer reported regularly to the laboratory scientist, offering insights from the field, conveying "intelligence" on rabbits and insects. At the beginning of summer, for example, as temperatures increased, "*Aedes* started to bite at night and *Anopheles annulipes* burst into activity. The ecological set-up points to one or both of these factors as being the cause of the immediate spreading of myxomatosis throughout the countryside."[32] Another wildlife officer, W. R. "Bill" Sobey recalled: "Fieldwork, which I suppose is ecology really, is so frustratingly complex and intriguing, and myxo kept turning up new things to look at."[33] Myers agreed, and added in a letter to Fenner that he "especially would like to see my fieldwork correlated with some first class lab work."[34]

5 The lab-field borderland

As he accumulated specimens and intelligence from the field, Fenner made his laboratory—initially in Melbourne under Burnet's watchful eye, then in Canberra—into a center of calculation in the biological war against rabbits. He tested viral virulence by inoculating a particular strain into unexposed laboratory rabbits and seeing how long it took to kill them; he determined host resistance by following the responses of survivor field rabbits to a myxoma virus of known virulence; and he sought to understand how effectively the virus was transmitted. It was impossible to make sense of myxomatosis without passing through his laboratory, without coming to terms with his investigatory enterprise. Although distant from the field, the laboratory was at the frontline in unraveling parasite-host interactions (Latour and Woolgar

[29] Fenner to Myers, 14 March 1951, folder 143/9/1, box 5, Fenner papers.

[30] Myers to Fenner, 9 November 1951, folder 143/9/1, box 5, Fenner papers.

[31] Myers to Fenner, 6 December 1951, folder 143/9/1, box 5, Fenner papers.

[32] Myers to Fenner, 6 December 1951, folder 143/9/1, box 5, Fenner papers.

[33] Sobey to Fenner, 26 March 1998, folder 143/9/9, box 7, Fenner papers. A South African, Sobey completed his Ph.D. at Edinburgh before joining the CSIRO in 1953.

[34] Myers to Fenner, 6 December 1951, folder 143/9/1, box 5, Fenner papers.

1979). It was not that the field had turned into a laboratory, or that the field was simply translated into laboratory artifacts, and scaled up or down—rather, a heavily trafficked borderland emerged, bringing together both sites, and generating, or reframing at least, life forms and their collectives. The natural experiment in epidemiology revealed, or made thinkable, new hybrid "epistemic things" that could not be singularly assigned to either the laboratory or the field (Rheinberger 1997). Rather than the lab-field boundary simply being crossed, an enlarging borderland encompassing both scenes of investigation was rapidly inhabited by newly imagined rabbit and mosquito populations, entangled in hitherto unanticipated ways, in novel modes of being. Thus myxomatosis produced a sort of epidemiological "edge effect" (Anderson 2015).[35]

Dealing with multitudes of rabbit carcasses and dead mosquitoes was hard work, too much for Fenner alone. A cooperative fellow, he disliked solitary toil. Soon, Max F. C. Day, Gwendolyn M. Woodroofe, and Ian D. Marshall joined his laboratory, further expanding the lab-field nexus. A botany graduate from Adelaide, Woodroofe would spend her whole career at the ANU, eventually moving from myxomatosis to other arthropod-transmitted viruses. Trained in agricultural science at Melbourne, Marshall had heeded Macnamara's advice that he should ask Fenner for a position in the myxomatosis research team while undertaking his doctoral studies on arbovirus virulence and host resistance. A Sydney zoology graduate, awarded a Ph.D. at Harvard for termite research, Day was looking for a field investigation of an arthropod-borne virus, hoping to pin down the specificity of vector-parasite compatibility. Fenner was happy to put him in charge of the mosquitoes to discover how they transmitted the virus. "The work was intense but fascinating," Day recalled. "All the ecology was being done by [Ratcliffe's team], we were doing the lab stuff, and it meshed well together."[36] During the war, he had served as a scientific liaison officer in Washington, DC, transferring penicillin-production technology to Australia, a role he resumed in 1955 after his myxomatosis work, though the job had changed considerably. In the United States during the cold war he was operating more as a research intermediary and coordinator, building relationships between Australian and American scientists, especially those concerned with disease ecology and epidemic surveillance. "Nobody made as much impact as Macfarlane Burnet when he came to Washington," Day recollected. "I would get phone calls for two months in advance and for a fortnight after he had left, everybody wanting to see him." It was, Day said, a wonderful opportunity to gather intelligence.[37] In New York City, Dubos took Day aside once and told him Fenner's myxoma virus research "will do more to make the science of epidemiology a respected one than any other contribution."[38] But Day needed no encouragement to promote the myxomatosis inquiries across North America.

[35] Kohler (2002) ambiguously describes the adjacency of laboratory and field in terms of both frontier (or boundary) and borderland (or contact zone), leaning toward the latter.

[36] Max Day interviewed by Max Blythe (1993), Australian Academy of Science, http://www.sciencearchive.org.au/scientists/interviews/d/day.html, accessed 7 March 2016. As a child, Day was a keen butterfly collector, encouraged by the lepidopterist G. A. Waterhouse. Later, he became the first chief of the division of forest research at the CSIRO.

[37] Max Day interviewed by Max Blythe (1993). Among other activities, Burnet was advising the US department of defense on preparedness for biological warfare.

[38] Day to Fenner, 15 December 1955, folder 143/9/1D, box 4, Fenner papers. See Fenner et al. (1956).

Just as Ratcliffe and Fenner began to collaborate, their task was complicated by an outbreak of encephalitis in communities adjacent to the field experiments. The coincidence prompted speculation among physicians and the public that the two epidemics might be linked. Could the myxoma virus cause brain inflammation in humans? Fenner pointed out that the distribution of encephalitis and myxomatosis did not overlap significantly. Then pathologists detected the Murray Valley encephalitis virus—an agent different from myxoma virus, though also spread by mosquitoes—in autopsies of fatal human cases. Still, public controversy rumbled on until Burnet, Clunies Ross, and Fenner inoculated themselves with myxoma virus, and remained healthy. After three weeks, no antibody to the virus could be detected (Fenner and Ratcliffe 1965: pp. 280–281). Such dramatic proof of human invulnerability made the scientists celebrities.

6 Host-parasite co-evolution

Before long, the research of Fenner and Ratcliffe was demonstrating evolution of pathogen virulence and host resistance (Fenner 1957). In this respect, Myers' studies at Lake Urana were exemplary, offering a model for the project as a whole. A depression in the plains north of the Murray River, Urana flooded only in wet years, at other times providing pasture for grazing. A rim of sand hills to the south was threaded with rabbit warrens. The isolated rabbit population was large enough to ensure a few survivors after each epizootic; and mosquito vectors were plentiful. Myers counted some 5000 rabbits in September 1951, in the southern spring, before he started to inoculate them with a virulent strain of myxoma virus. By November he could find only fifty. Fenner's serological tests showed that most of the survivors had escaped infection, giving a case mortality rate of 99.8%. An attenuated form of the virus persisted around the lake, to which many of the survivors seemed inured. Breeding rapidly, and augmented by an influx from neighboring country, the rabbit population soon rebounded, reaching 550 in October 1952, when Myers again inoculated them. But before the new virulent strain could take effect, an epizootic of the attenuated strain emerged. Rabbit numbers again dropped sharply, down to 60 survivors, giving a case mortality rate this time of 90% (Myers 1954; Myers et al. 1954; Mykytowycz 1953).[39] "The presence of this attenuated virus," Fenner and Ratcliffe concluded, "permitted the survival of enough rabbits for effective selection for host resistance to occur" (Fenner and Ratcliffe 1965: p. 287).[40] As Fenner wrote to Dubos: "In areas where they are exposed to annual severe epizootics there may be selection of a more resistant race of rabbits."[41]

Worried about the long-term impact on their campaign, Fenner and Ratcliffe decided to compare the pattern of infection of the virulent strain with the attenuated

[39] A few years later, case mortality was 25%.

[40] On 16 August 1953, Ratcliffe wrote to Burnet: "I was most interested in your blueprint of how you thought strains of reduced virulence emerged in nature" (folder 143/9/1, I 1-3, box 4, Fenner papers). He continued, in reference to Lake Urana: "field studies of rabbit resistance are very laborious."

[41] Fenner to Dubos, 28 September 1954, folder 143/7, box 3, Fenner papers.

mutant. In 1955, the rabbits at Lake Urana were inoculated again with a virulent myxoma virus, which gave raised skin lesions, killing about 70% of the population. But in the later stages of the myxomatosis epizootic an attenuated strain, giving flat skin lesions—which presumably had persisted through the winter—replaced the virulent type (Fenner et al. 1957). It was clear, then, that under conditions of epizootic transmission attenuated strains enjoyed a selective advantage over the fully virulent virus, probably because their hosts lived longer (Fenner et al. 1956). "I believe," Ratcliffe later wrote, "that these … experiments rank as among the most important ever carried out in an epidemiological research programme."[42] The conclusions, however, did not augur well for continued biological control of rabbits. Over time, natural selection for host resistance and attenuated viruses, along with physical impediments to efficient pathogen transmission, would make myxoma inoculation less effective. "At this stage in the research programme," Ratcliffe recalled, "it became obvious to those in the know that the myxomatosis situation in Australia was complicated by what I can only call gremlins."[43] Increasingly, it would become less profitable to liberate highly virulent strains. Ratcliffe wanted to resort instead to poisoning the resistant survivors of the myxomatosis outbreaks. But when this proposal surfaced, Macnamara's "uneasiness and dissatisfaction grew," causing her to resume her militant crusade for further inoculations.[44] Ratcliffe, though, was unrepentant. Later he wrote:

> I think everyone with research experience would agree with me that it is extraordinarily difficult to decide what to do when a programme reaches the stage when the law of diminishing returns starts to apply. This stage was reached, for at any rate the wildlife side of the myxomatosis research programme, in 1956 or thereabouts, when we were being forced to recognize the full implications of insect transmission.[45]

By 1956, "there seemed little doubt that the great majority of inoculations represented wasted effort" (Fenner and Ratcliffe 1965: p. 297). Nonetheless, myxomatosis had become enzootic across the continent, fluctuating in severity according to vector activity and the proportion of susceptible individuals in the rabbit population, thereby producing "a patchwork pattern of localized outbreaks" (Fenner and Ratcliffe 1965: p. 298). Since the 1950s, rabbit numbers have stabilized at around 20% of the pre-myxomatosis level. As Fenner and Ratcliffe pointed out, the reduction in Australia's rabbit population "must be classed as an ecological event of the first magnitude" (Fenner and Ratcliffe 1965: p. 306). Even so, they warned, "the rabbit is a most formidable enemy, because no victory over it, short of total eradication, can ever be permanent" (Fenner and Ratcliffe 1965: p. 32).

[42] Francis N. Ratcliffe, Myxomatosis—Dame Jean Macnamara and the CSIRO, 1969 [typescript], p. 2, box 8, Ratcliffe papers.

[43] Ratcliffe, Myxomatosis, p. 2.

[44] Ratcliffe, Myxomatosis, p. 3.

[45] Ratcliffe, Myxomatosis, p. 6.

7 Conclusion: rabbits as cold war casualties

Reflecting on their epidemiological studies, Fenner and Ratcliffe (1965: p. 344) observed that myxomatosis had "provided a unique opportunity for the investigation of the influence of infectious disease on the evolution of a mammal." In particular, it was an unrivalled opportunity to document the selection of wild animals for resistance to an infectious disease. Fenner and Ratcliffe predicted that eventually resistance to myxoma virus would stabilize, and rabbit populations would reach equilibrium. "We could then envisage a climax association," they concluded, "in which myxomatosis still caused a moderately severe disease with an appreciable mortality, much as smallpox does in human communities" (Fenner and Ratcliffe 1965: p. 347). Of course, they were writing before the eradication of smallpox, which Fenner, as the preeminent expert on poxviruses, was to certify (Fenner 1983; Fenner et al. 1988).[46] The analogy with smallpox was a loaded one. Fenner and Ratcliffe recognized the similarity of their experiment with the introduction of European diseases into Indigenous populations, except that rabbits were an invasive species like the virus—in that sense, more like vulnerable white settlers than Aboriginal Australians.

"With hindsight," reflected Fenner and Bernardino Fantini, "myxomatosis could have been introduced into the Australian rabbit population much earlier than it was" (1999: p. 143). To explain the delay, they adduced concerns about specificity and host range; the distraction of a major conventional (and then nuclear) war; and the psychological resistance of some scientists to taking a risk of such magnitude. But the release of myxoma virus also occurred in a period when biological warfare more generally was becoming thinkable, even perhaps acceptable, in ways unimaginable before the late 1940s. Conditions of possibility for the ecological analysis and biological management of populations—whether human or vermin—changed with the onset of the cold war. Burnet and Fenner were called to advise the Australian and U.S. military on the feasibility of biological warfare during the 1950s. Neither of them did so with any enthusiasm, but they wanted their knowledge of disease ecology to inform operational decisions. Additionally, their ecological research was predicated on, and contributing to, growing international attentiveness to disease surveillance and epidemic intelligence. Fenner shared interests in the application of statistics to epidemiology with Alexander D. Langmuir, the founder in 1951 of the U.S. Epidemic Intelligence Service, later the Centers for Disease Control, an advocate for disease preparedness and what came to be called biosecurity.[47] The ecological lessons of myxomatoisis were adapted to cold-war mind-sets,

[46] Smallpox eradication focused on "surveillance and containment," whereas for myxomatosis it was all about inoculation, or spread, and surveillance.

[47] Fenner may have heard of Langmuir through mutual interests in wartime malaria research; certainly he knew his postwar advocacy of surveillance; and Fenner's collaborator D. A. Henderson (a student of Langmuir) later introduced them. See the correspondence in folders 29 and 48, box 6, Langmuir papers, Alan Mason Chesney Medical Archives, Johns Hopkins Medical School, Baltimore, MD. See also Langmuir (1965, 1971); Susser (1985); Declich and Carter (1994); Dando (1999); Brown and Fee (2001); and Fearnley (2010). To this should be added the "medical snooping" of Joseph E. Smadel (friend of Burnet and Fenner) and his acolytes in the early 1950s at the Walter Reed Army Institute of Research: see Anderson (2008): chapter 2.

sensibilities, and apprehensions; they enabled would-be germ warriors to calculate the outcome of their proposed biological interventions. Accordingly, post-war disease ecology became inextricably enmeshed in a military-medical complex, in the emerging biosecurity industry.[48] And so, in becoming rabbits during the cold war, humans were acquiring the biopolitical status of vermin, or bare life (Agamben 1998), vulnerable to biological attack under ecological supervision.

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[48] Fenner went on to write *The Biology of Animal Viruses* (1968). Later, he founded the Centre for Resources and Environmental Studies at the ANU and became an outspoken advocate of human population control. Ratcliffe was a founder of the Australian Conservation Foundation.

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