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Réception des nouveaux Associés étrangers sous la coupole de l'Institut de France

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I started my scientific career as a biologist fascinated with the natural world and in particular with the ecology of the complex life cycles that many parasitic organisms have evolved to exploit their host populations. The disciplines of ecology, and medical or veterinary epidemiology, were very much separate areas of scientific study with their own terminologies and concepts. The 1960s and 70s had witnessed an explosion of interest in ecology and especially population biology and species interactions. In parallel, mathematical tools had begun to occupy their rightful place serving as a template for the study of population dynamics and species interactions - a position they had long occupied in population genetics.

As a young scientist I wanted to focus an emerging understanding of population biology on the study of the pathogens that caused disease in wildlife, livestock and humans. I wished to redirect infectious disease epidemiology from its statistical routes of description, towards a framework founded on an understanding of the dynamics of populations and on the co-evolutionary pressures acting on both pathogen and host. An important part of this journey was a period of collaboration with Robert May which laid the foundations for two articles in *Nature* on the population biology of infectious diseases (Anderson & May, 1979; *Nature* 280, 361-7; 455-461). This research attempted to create a theoretical framework for the study of all infectious agents whether they had simple or complex life cycles, or irrespective of the type of host, human or animal. A side discovery of this work was a refutation of the conventional wisdom that evolution always drives parasites to reduced pathogenicity to their hosts, based on the premise - why destroy your environment? Theory showed clearly that pathogens will strive to maximise their reproductive success - and this can often lead, as in the case of the rabbit viral disease, myxomatosis, to increased virus virulence.

The creation of a mathematical template for the study of host and pathogen populations led to considerations of what actions might lessen their impact. Could theory generate guidelines on optimal control policies and predict the impact of defined intervention strategies (Anderson & May, 1991 *Infectious Disease of Humans: Dynamics and Control*, Oxford University Press)? The public health community in the early 1980s was somewhat sceptical of simple mathematical theory being able to predict outcome. The first test for simple theory came in

the field of mass vaccination programmes to control directly transmitted viral and bacterial infections. Analysis predicted that post the introduction of mass vaccination in young children, a trough of susceptibility would be created in children just older than those immunised as a consequence of the herd immunity created, lessening their exposure to infection in subsequent years (Anderson & May, 1982 *Science* 215,1053-1060; Anderson & May 1985 *Nature* 318, 323-329). This trough would then be maintained as they aged and would create problems if infection at an older age was more serious than if acquired early in life. This is the case for the rubella virus, which if acquired in the first trimester of pregnancy can induce serious disease in the unborn child (i.e. congenital rubella syndrome). The prediction was tested and confirmed when Finland introduced age and time stratified population based serology in the late 1980s (Ukkonen & Bronsdorff, 1988 *Scan. J. Infect. Dis.* 20:255-259).

My research has produced insights of application in both the interpretation of observed pattern and in the design of interventions. The work includes determining the impact of mass chemotherapy on helminth infections of humans and the optimal intervals between rounds of drug treatment in the developing world (Anderson & May, 1982 *Nature*, 297, 557-563) , analysing the main determinants of the pattern of the AIDS epidemic in different parts of the world and its demographic impact (Anderson and May, 1988, *Nature* 333, 514-522; Anderson et al, 1988 *Nature* 332, 228-234), predicting the course of the bovine spongiform encephalopathy (BSE) epidemic in cattle and the associated exposure of humans to contaminated beef (Anderson et al, 1986 *Nature* 382, 779-788), deciding how best to control the foot and mouth virus (FMD) in cattle and sheep in the UK in 2001 (Ferguson et al, 2001 *Science* 292,1155-1160), what control interventions for the SARS virus might induce the greatest impact (Riley et al, *Science* 300, 1961-1966), and charting the characteristics of an infection that determine what interventions are likely to be most effective (Fraser et al, 2004 *Proc. Nat. Acad. Sci.*101, 6146-6151). My current research is examining how low efficacy vaccines for HIV-1 and the malarial parasites will impact on transmission and public health.