INSIGHT
CURTIS M. LIVELY

Curt Lively was born in 1954 in Louisiana, USA, but grew up in the desert regions of Arizona. He received a BS degree in Zoology from Arizona State University in 1977, and a Ph.D. in Ecology and Evolution from the University of Arizona in 1984. He then conducted post-doctoral research at the University of Canterbury in New Zealand (1984–89) and at Rutgers University (1989), before joining the faculty at Indiana University in 1990. He has worked in freshwater, marine, and terrestrial environments to address questions on predator-prey interactions, the evolution of phenotypic plasticity, the evolution of sexual reproduction, and host-parasite coevolution. He has also conducted theoretical studies on the evolution of induced defense, the evolution of parasite virulence, the evolution of sex/recombination, and the ecology of symbiont-mediated defense against pathogens. – Address: Department of Biology, Indiana University, Bloomington, IN 47401, USA. E-mail: clively@indiana.edu

I was a Wiko Fellow from April through June of 2011. The time that I spent in residence was too short, but it was fascinating to interact with so many interesting people from such a wide range of disciplines. I very much hope to return someday.

I am a biologist, trained in ecology and evolution. I was part of the "Antonovics disease group". I spent most of my time at Wiko working on a difficult theoretical problem concerning the rate of evolutionary change (the fundamental theorem of natural selection). I was particularly interested in understanding how the rate of change in fitness caused by natural selection compares to the rate of change in fitness caused by environmental deterioration. For example, a beneficial mutation that increases the range of

foods that an organism is able to consume can lead to an increase in the organism's population size. However, as the mutation spreads, the population size grows and competition for resources will increase. Hence, there is a feedback: the mutation increases fitness, which increases population size, which then leads to a decrease in fitness. I wanted to understand the details of this process and relate it to standard mathematical models in population genetics. I was able to tackle this problem by relying heavily on the theoretical work of a former Wiko Fellow, Steve Frank. My ultimate goal was to expand the study to understand host-parasite coevolution. In this case, the parasite causes the environmental deterioration for the host, and vice versa; and both host and parasite may be evolving at very rapid rates.

I am not trained as a mathematician, so I found the work challenging. I don't think that I would have made any progress on this project had I not been at Wiko, which allowed for long periods for concentration. I also greatly benefited from working with the disease group. Mike Boots and Janis Antonovics are pioneers in modeling infectious diseases, and they are both exceptionally good at explaining their approaches to the problem. I also learned from, and became fascinated by, the non-scientists at Wiko. They were able to clearly explain their projects, and they did so with great enthusiasm. Several of their talks stood out for the depth of scholarship (and emotion) that they conveyed.

Perhaps the biggest insight for me came from a question that Herbert Muyinda asked during my Wiko presentation. He asked: what could we do to combat Schistosomiasis in Africa? (Schistosomiasis affects hundreds of millions of people, mainly in Africa and SE Asia; the disease is caused by a worm that cycles between humans and freshwater snails.) I answered that, after careful study, I would consider increasing the genetic diversity of the local snail population by adding uninfected snails drawn from several different locations. Someone replied that this addition of snails would also increase snail density, and thereby increase the intrinsic rate of disease spread. I saw the point, but it was not immediately clear to me which effect would be greater: the positive effect on disease spread caused the increase in host density, or the negative effect on disease spread caused by the increase in host genetic diversity. So, I worked on this problem. Over much of the parameter space, I found that the negative effect greatly outweighs the positive effect. Thus increasing host genetic diversity could reduce disease spread even when associated with a temporary increase in host population size. Thank you Herbert for that question!

On the whole, my time at Wiko was magical. It was a great honor for me to be part of this group.