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Delayed frequency dependence, immunology, and the paradox of virus diversity

Rhinoviruses, the most common cause of the common cold, are arguably the most successful disease on earth. People spend about one year of their lives partially incapacitated by tiny viruses armed with only 10 genes. Over the years, each of us will be infected by most of the over 100 different types, which differ from each other in subtle but barely understand ways. Rhinoviruses use their 10 genes to take over the cells in our noses, hide from the parts of the immune system that generate antibodies, and disappear without doing serious harm.

Mathematical models to understand these devious viruses must link the immunological processes that determine symptoms and the development of resistance, mutational processes that generate new virus genotypes, and population processes that spread infections between individuals. An model of the immune response generates delayed frequency dependence, wherein the successful virus today is one that was rare last year. Including delays and competition within the broad framework of the neutral theory of biodiversity requires novel methods of approximation in order to understand viral biodiversity and evolution.