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Immunocompetence: a neglected life history trait or conspicuous red herring?

Defending oneself against parasites is not something that should be undertaken lightly. As anybody who has experienced a course of antibiotics or antimalarial drugs can testify, artificial immunity can be costly and have dangerous side effects. But what about natural immunity against parasites? Although usually viewed as a benefit, can natural resistance also prove costly? And, if natural immunity can incur health risks as well as benefits, what are the consequences for our understanding of the evolution of both disease and disease-resistance? Such questions lie at the heart of the new research interest of 'ecological immunology'[1].

Costliness is an important concept in modern evolutionary theory. If activities were cost-free, all individuals would have the same perfect life history – long life, high fecundity, fast growth and lavish parental care. The fact that such a darwinian demon does not exist is explained by tradeoffs between costly activities – organisms have only a limited amount of resources and must allocate them prudently among several expensive activities. Traditionally, however, evolutionary biologists have restricted their treatment of life history tradeoffs to those involving the classic life history variables, such as the tradeoffs between offspring size and number, or between offspring number and survival. New work in ecological immunology suggests that immunocompetence (Box 1) could warrant inclusion in life history theory.

If we are to begin to understand the role of immunocompetence in the evolution of host life histories, we need to be able to answer a series of related questions. Why is parasite defence costly? What is the role of host–parasite coevolution? How can immunocompetence be measured in wild populations? These questions were the framework for a recent workshop on 'Parasite Defence and Trade-offs in Evolutionary Ecology' funded by the European Science Foundation's Population Biology Programme (27–30 October 1998). The workshop was organized by Ben Sheldon (Uppsala University, Sweden) and Simon Verhulst (University of Gröningen, The Netherlands), and was hosted by the Zoology Department at Uppsala University.

Why is parasite defence costly?

The best known hypothesis is that mounting an immune defence is energetically expensive. Hence, individuals must trade off energy devoted to immunological functions against energy devoted to other life history activities, such as growth, reproduction and survival. In a review of the nutritional demands imposed by immune function, Bob Lochmiller (Oklahoma State University, Stillwater, USA) showed that infection in mammals often leads to substantial increases in basal metabolic rate (BMR) and losses in body mass. He also presented indirect evidence for immune function being energetically expensive; for example, the observation that germ-free conditions could lead to up to 30% decreases in BMR and 30% increases in body weight in laboratory-raised chickens and mice. Doubts, however, were raised about the energy-cost hypothesis. For example, Claudie Doums and Gabriele Sorci (both at Université Pierre et Marie Curie, Paris, France), experimentally manipulated the energy budgets of bumblebees (*Bombus terrestris*) and house sparrows (*Passer domesticus*), respectively, and looked for associated changes in immune function. In neither case was there convincing evidence for a relationship between energy availability and relevant immune responses. Similarly, during cold-stress experiments, Lars Råberg (Lund University, Sweden) found no significant difference in BMR between individual blue tits (*Parus caeruleus*) that had been vaccinated to stimulate their immune system versus individuals whose immune system had not been experimentally stimulated.

A related perspective is that immune responses are costly because they use up specific nutrients. Both Anders Møller (Université Pierre et Marie Curie) and Jacqui Shykoff (Université de Paris-Sud, France) stressed the potential importance of carotenoids as a key mediator between immune function and biological signals. Carotenoids are perhaps best known to evolutionary biologists as the pigments that underlie many of the most extravagant examples of animal coloration. To immunologists and biochemists, however, carotenoids are better known as vital precursors in a huge number of immunological systems. Hence, carotenoid-based signals can provide accurate information about the bearer's ability to cope with their current parasite load: if an individual can afford to waste precious carotenoids on a colourful ornament then it must be in good shape[2]. Mike Siva-Jothy (University of Sheffield, UK) then pointed out that melanin-based pigmentation could play a parallel role in many invertebrate groups. In such organisms, an important element of immune defence is a host's ability to encapsulate foreign bodies, such as parasitoid eggs, and to melanize the subsequent capsule. Again, therefore, melanin-based signals in invertebrates might honestly signal the bearer's immunological capacity. Indeed, given that the main building block of melanin, the amino acid tyrosine, has a range of important immunological functions in vertebrates, melanin-based 'badges of status' might not be as cheap as commonly supposed.

Other mechanisms by which immunity might incur a cost were also proposed. Some of these provided evidence of a more direct link between immunity and fitness. Oliver Kaltz (Université de Paris-Sud), for instance, suggested that the main way in which certain plants defend themselves against anther-smut (a fungus) is to drop diseased male flowers before the pathogen has had time to develop and reproduce. For these species of plant, the cost of immunity can therefore be measured directly in terms of reduced gamete production. Similarly, Ken Wilson (University of Stirling, UK)

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suggested that caterpillars of the African armyworm (*Spodoptera exempta*) that melanize their cuticles as part of their defence against parasitoids and pathogens may increase their conspicuousness to avian predators. Here, the cost of immunity could be measured directly in terms of increased predator-induced mortality.

A completely different argument is that individuals might be limited in their ability to mount an immune defence, not because the defence is costly, but because it is dangerous. Lars Råberg showed that although a strong immune response might well have advantages for parasite resistance, it might also elevate the risk of self-attack (autoimmune disease) during which the host's immune system turns against the host's own cells. Moreover, Torbjörn von Schantz (Lund University) and Mike Siva-Jothy made cases that a strong immune response would be dangerous simply because such mechanisms often employ potentially toxic oxidative agents, such as free radicals, to eliminate foreign cells. Again, such agents could potentially act against the host's own DNA, proteins and lipids. Perhaps, they suggested, it is an ability to cope with this sort of 'oxidative stress' that is being signalled by secondary sexual characters.

Is host–parasite coevolution important?

Costliness is not the only factor affecting immune defence. Coevolutionary interactions between hosts and parasites might also prevent a host from mounting an effective immune defence even if the immune defence is relatively cost-free. The best-known version of this hypothesis is that hosts often lag behind their parasites in some sort of evolutionary arms race. Unfortunately, although this explanation has been well studied by theoreticians, it has received very few empirical tests. Both Dieter Ebert (University of Basel, Switzerland) and Ian Owens (University of Queensland, Brisbane, Australia) demonstrated protocols for overcoming this deficiency by 'arresting' the evolution of either the host or the parasite. Of particular interest in this context were Lex Kraaijeveld's (Imperial College London, UK) results from artificial selection experiments on *Drosophila melanogaster* populations that were known to have become locally adapted to particular strains of parasitoid wasps. These experiments confirmed the principle that evolutionary change in host immunity occurs via adaptations in both 'general' and 'specific' mechanisms of parasite resistance. General mechanisms are those that can be used to resist a wide range of parasite types, whereas specific mechanisms are those used to counter a specific parasite strain or species. The relative importance of these two mechanisms remains unclear.

A novel version of the coevolution hypothesis was provided by Minus van Baalan (Université Pierre et Marie Curie) and Jukka Jokela (ETH-Zürich, Zurich, Switzerland) who suggested that reduced immunocompetence could be an evolutionarily stable strategy either when parasites are very rare or, counter-intuitively, when parasites are very common. This latter prediction caused much interest because it provides a novel reason why some individuals might produce a weak immune response: if parasites are so virulent that resistance is futile, the host might as well withdraw its resources from its immune system and, instead, use them to produce offspring that might be able to disperse to a parasite-free environment.

How can immunocompetence be measured in wild populations?

Victor Apanius (Florida International University, Miami, USA) and Bob Lochmiller reviewed the components of vertebrate immune systems and the impressive battery of techniques now available to ecological immunologists for measuring the immunocompetence of individuals. The simplest methods rely on estimating the absolute, or relative, abundance of immunologically active cells. The problem with these observational techniques is that it is usually impossible to distinguish whether an abundance of such cells is due to a high level of natural immunity or is in response to a recent infection (Sabra Klein, Johns Hopkins School of Public Health, Baltimore, MD, USA). Similar problems surround the method of measuring the relative size of immunological organs, such as the spleen or bursa of Fabricius in birds (Charlotte Deerenberg, Max-Planck Institute, Andechs, Germany). Many researchers have therefore moved to 'challenge' techniques, which are based on experimentally exposing the host to a novel substance to see how they deal with a new parasite. Different challenge substances have already been developed to assess different aspects of the immune system, using either naturally occurring or novel antigens (Dennis Hasselquist, Lund University).

However, such approaches are not without their own problems. Matthew Evans (University of Stirling), for example, pointed out that the current battery of techniques do not assess potentially important aspects of the immune response, such as natural killer cells and innate defences. Also, the use of naturally occurring antigens can lead to the same difficulty as encountered when using the observational techniques: are high levels of immune function caused by high natural resistance or recent exposure to the antigen? Another complicating factor raised by Heinz Richner (University of Bern, Switzerland) is that an offspring's immunity during early life can be maternally derived and therefore unrepresentative of a naive immune system. Finally, a series of workers reported difficulties in replicating results either across populations (Mariusz Cichon, Uppsala University) or across techniques (Gabriele Sorci, Sabra Klein, and Ian Owens). But by far the most important unknown at present is whether an individual's response to immune challenges is a good index of its ability to cope with natural parasites. Until this is known,

Box 1. What is immunocompetence?

There can be few words in modern biology that raise so much interest, discussion and controversy as immunocompetence. For instance, in the past five years alone, nearly 500 papers have been published that use this word (*Science Citation Index*). Yet, despite this, there are few published definitions of immunocompetence, and this has added to the confusion that surrounds the field of ecological immunology.

In an attempt to generate a definition that workers in the field could agree on, we surveyed a dozen of our colleagues to get (semi-) independent views on the subject. To our surprise, the various definitions were broadly similar. Most agreed that it was an individual's ability to respond to a foreign antigen so as to minimize the fitness costs of infection. However, people did disagree on which mechanisms should be included in the 'response', with some restricting the term to include 'antigen-specific lymphocyte-mediated responses, with memory', whilst others included all 'innate, humoral and cell-mediated immune reactions'.

Interestingly, none of these definitions would include behavioural or physical resistance mechanisms, both of which might be important in minimizing the risk of infection. Is a bird that regularly preens itself to remove ectoparasites more immunocompetent than one that does not? Is a thick skin, which prevents mosquitoes biting, an indicator of high immunocompetence? Given that one definition of immunity is 'the ability of an organism to resist disease' (*Collins Concise Dictionary*, 1995), one could argue that the answer to both of these questions is yes. One respondent captured this problem by suggesting that the mechanisms should include any that are 'of interest to broad-minded immunologists', but this might prove too vague for a working definition.

Definitions of immunocompetence published in medical and immunology dictionaries may be too restrictive for evolutionary studies of host–parasite interactions, particularly when these include invertebrate hosts. A more all-encompassing definition might be:

Immunocompetence: a measure of the ability of an organism to minimize the fitness costs of an infection via any means, after controlling for previous exposure to appropriate antigens.

most challenge techniques will be viewed with caution by field biologists.

Prospects

The workshop raised at least as many questions as it answered. Some of these new questions were disarmingly straightforward. For example, in the final session of the workshop, Peeter Horak (Tartu State University, Tartu, Estonia) simply asked who had convincing evidence of a direct link between immunocompetence and fitness, or even between immunocompetence and disease resistance. His request was met by silence. Similarly, Mike Siva-Jothy asked whether evolutionary biologists could obtain an adequate understanding of the role of immunocompetence in life history evolution when they continued to treat immunology as a 'black box'. Was it really safe, he asked, to ignore the details? Or, using Lex Kraaijeveld's analogy, would we understand the Universe as well if we had never bothered learning about subatomic particles such as quarks? Continuing this theme, several workers expressed their concerns about using a single challenge technique to assess overall immunocompetence. Why is there so much variation across studies? Is it due to differences in experimental procedure, or differences in biology?

And what about the parasites? In a host-oriented meeting, only Margaret Mackinnon and Andrew Read (both from University of Edinburgh, UK) asked the basic question of why parasites damage their hosts in the first place.

Then, finally, there was the underlying worry over whether techniques and results could be extrapolated across taxa. For instance, are immunological truisms developed through studies of domesticated chickens and ducks really of relevance to wild populations of passerines? If ecological immunology is to realize its promise, these troublesome questions must be addressed. Until then, skeptics will continue to point at the uncertain foundations of the field and thus have an excellent excuse for ignoring its potential as a radical advance in life history theory.

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Habitat fragmentation and insect flight: a changing 'design' in a changing landscape?

The possible evolutionary implications of habitat isolation for flight morphology, such as wing reduction in birds on oceanic islands, are well recognized. However, the fragmentation of terrestrial habitats can also affect the morphological 'design' of species (or populations) in more subtle ways, via the costs and benefits of changing dispersal patterns. Theoretical models predict that dispersal in metapopulation systems is favoured during colonization and selected against once a population has been founded[1,2], and that genetic variation in dispersal-related traits can have a dramatic effect on the probability of metapopulation extinction following a fragmentation event[3].

In insects, changes in dispersal capability have mainly been studied for discontinuous traits, such as winged–wingless dimorphisms[4], but insects that use flight for behaviours other than dispersal might show more subtle responses. Empirical research on damselflies and particularly butterflies has provided interesting suggestions for different directional evolutionary change (Box 1). Damselflies were found to have a more 'mobile' morphology in fragmented landscapes[5], whereas time-series data of some butterflies suggested a morphological change towards less mobility when populations became more isolated[6].

Recent work by Jane Hill, Chris Thomas and Owen Lewis indicates heritable responses in the silver-spotted skipper butterfly (*Hesperia comma*)[7] when morphological traits among individuals from different habitat patches and metapopulations were compared. Together with another study from the same British research team on another rare butterfly (the silver-studded blue, *Plebejus argus*)[8], there is now a clear line of evidence that at least butterflies (but probably a wider range of flying insects) show evolutionary responses in flight morphology to changes in landscape structures. Such effects could be more general and of larger significance than has previously been realized.

Flight muscles and fragmentation

Hill and colleagues compared adult morphology in male and female *H. comma* butterflies at two spatial levels that are relevant to their population structure: different habitat patches (separated by <5 km) within a metapopulation, and different metapopulations (separated by >40 km). The metapopulations differed in degree of fragmentation (i.e. patch size and isolation) and in the colonization history of the butterfly. In the 1950s and 1960s, *H. comma* disappeared from many sites in the UK because of reduced grazing levels on short-turfed calcareous grasslands, but it spread again as habitat was restored during the past 20 years, although some areas have not been recolonized[9]. Hence, this system allowed the comparison of flight morphology of butterflies from recolonized and permanently occupied ('refuge') sites within metapopulations.

In contrast to earlier studies[5,6], comparisons were not based on direct measurements of wild-caught specimens, but on individuals collected as eggs from different sites and reared in a common environment. This is a more powerful approach because differences between sites will reflect genetic differences, a prerequisite to evolutionary change, more than local environmental differences (e.g. microclimate), which might influence phenotype production independently of fragmentation.

Among metapopulations, measures of total size (e.g. total dry mass and wing area) did not differ between sites, but there was significant variation in relative investment in the thorax, which mainly

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