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# Sex against virulence: the coevolution of parasitic diseases

Dieter Ebert and William D. Hamilton

Parasites – here broadly defined as damage-producing organisms, including microbial pathogens, traditional parasites and small herbivores – are ubiquitous and influence either directly or indirectly almost every conceivable level of biological organization. The impact parasites have on the evolution and ecology of their hosts depends on their virulence, the driving force in host–parasite coevolution. Virulence, *per se* beneficial for neither parasite nor host, cannot be a property of a parasite alone; rather, it is a product of the host–parasite interaction. Different host genotypes from the same population do not suffer equally when infected with the same parasite strain, and different parasite strains cause variable levels of virulence in the same host genotype<sup>1–3</sup>.

Most studies on the evolution of virulence have concentrated on parasite evolution, assuming that virulence is maintained by genetic trade-offs between virulence and other fitness components of the parasite. For example, parasite-induced host mortality was shown to be negatively correlated with host recovery rate (which contributes to parasite mortality) in Australian rabbits infected with the myxoma virus<sup>4,5</sup> and positively correlated with the multiplication rate of a microsporidian parasite in *Daphnia* hosts<sup>3</sup>. Therefore, it has

**Reciprocal selection is the underlying mechanism for host–parasite coevolutionary arms races. Its driving force is the reduction of host lifespan or fecundity that is caused by a parasite. Parasites evolve to optimize host exploitation, while hosts evolve to minimize the ‘parasite-induced’ loss of fitness (virulence). Research on the evolution of virulence has mostly emphasized the role of parasite evolution in determining virulence. However, host evolution, accelerated by sexual recombination, contributes to the evolution and expression of virulence as well. The Red Queen hypothesis predicts that genetic variation among host offspring facilitates selection for reduced virulence. Here, we outline a synthesis between current thinking about the evolution of virulence and the evolution of sex.**

been suggested that to maximize fitness a parasite should optimize the trade-off between virulence and other fitness components<sup>5</sup>. This optimality concept for the evolution of virulence, however, largely neglects genetic variation among hosts in their interaction with parasites. Such variation results in differential reproductive success among hosts and would, in the absence of parasite evolution, lead to reduced virulence. Given the high evolutionary rate of parasites<sup>4,6</sup>, host evolution can often be ignored in a first approximation, but for a better understanding of the evolution of virulence it is essential to understand the host’s evolutionary response and in particular the role of genetic recombination in host evolution.

It has been suggested that sexual reproduction of hosts is a means to overcome the disadvantage of the low evolutionary rate that an asexual host would have in comparison with its rapidly evolving parasites<sup>7–9</sup>. Combining current theory of the advantage of genetic recombination and outbreeding with the theory on the

evolution of virulence, one would predict that hosts continuously evolve to reduce virulence, while their parasites evolve to keep virulence as close as possible to an optimal level for their own life histories. In this arms race, a high

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Dieter Ebert was at the NERC Centre for Population Biology, Imperial College at Silwood Park, Ascot, Berks. UK SL5 7PY; he is now at the Institut für Zoologie, Universität Basel, Rheinsprung 9, CH-4051 Basel, Switzerland; William Hamilton is at the Dept of Zoology, Oxford University, South Parks Road, Oxford, UK OX1 3PS.

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**Box 1. Testing the Red Queen: do parasites express higher virulence in host genotypes to which they are adapted?**

- (1) Variation in virulence reactions is expected in both hosts and parasites. 'Wild-sampled' hosts will vary in fitness when exposed to a single parasite strain and 'wild-sampled' parasite strains will vary in virulence and reproductive success when applied to a single host line.
- (2) Wild-sampled parasite strains will initially evolve an increase in virulence and reproductive success when kept in monoclonal host populations.
- (3) Parasites taken from one host population should be, on average, less virulent in hosts from other populations. Virulence in these novel hosts should, on average, decrease with decreasing genetic similarity between the host of origin and the novel host. 'Average' should be stressed here, since occasional highly virulent poorly reproducing parasites can be expected.

**Box 2. Testing the Red Queen: does outbreeding and host genetic diversity hinder parasite adaptation?**

- (1) Parasites kept in monoclonal host cultures should evolve higher levels of virulence than parasites in multiclonal host populations. To avoid inevitable selection on host genotypes, the genetic composition and gene frequencies of the mixed host populations must be kept constant throughout the selection period. This can be done by continually reconstituting the host population from the same mixture of the same stocks. Without such replacement, selection for the least susceptible host genotype will occur and confound the result. The same replacement procedure must be done in the monoclonal host populations, to avoid selection on mutants and provide comparable handling and interference.
- (2) Parasites kept in monoclonal host cultures should lose (reduce) their virulence when combined with other host clones, even if they were previously adapted to these clones.
- (3) Parasites adapted to a monoclonal host population should express, on average, lower virulence in sexually outbred descendants of their hosts compared with inbred or asexually produced offspring. Variance of virulence and parasite success, however, should be high in the outbred offspring generation, and particularly high variance due to new homozygous combinations may become apparent in grand offspring and in later descendants from the outcrosses.

**Box 3. Testing the Red Queen: do parasites mediate selection and polymorphism in natural host populations?**

- (1) Natural host populations that suffer intense parasite pressure should maintain higher levels of genetic variability than host populations without parasites. In clonal or cyclic parthenogenetic host populations, in which the whole host genome represents one linkage group, this effect can be investigated by correlating diversity of genetic markers with some measure of parasite selection pressure (e.g. mean parasite richness or abundance) across host populations. For sexually reproducing hosts, the allelic diversity of the defence loci can be studied (e.g. MHC haplotypes in vertebrates).
- (2) In clonal and cyclic parthenogenetic host populations, linkage and selection by parasites are likely to produce genotype frequencies that differ between parasitized and uninfected hosts. In host-parasite systems where genes involved in defence are known (e.g. the MHC in vertebrates), the frequency of these genes can be studied directly.

**Box 4. Testing the Red Queen: do parasites induce temporal changes in their host populations?**

- (1) Following prediction (2) in Box 3, if associations between parasites and host genotypes are detected and host generation time is short, parasite-mediated selection may be tested by re-sampling the same host population and monitoring changes in the frequency of the particular genotype (clonal markers or MHC genes, respectively).
- (2) When parasite strains can be stored unchanged (e.g. by freezing of microbial pathogens), their re-introduction into the same host population many host generations later should reveal a change in their average virulence – most often a reduction in virulence. Due to ongoing host evolution, some originally highly virulent strains are likely to have become much less virulent.
- (3) A similarity should be found between patterns of virulence in hosts over space and genetic distance [hosts vary spatially; see prediction (3) in Box 1] and changes in virulence when a genetically 'frozen' parasite is applied to the same local population over time [hosts vary temporally; see prediction (2) in Box 4].

evolutionary rate would benefit both opponents. Since parasites already have a very high evolutionary rate intrinsic to their short life cycle, hosts would be selected for increased evolutionary rates, even if this has costs. Sexual recombination could provide such an increase<sup>7,8,10,11</sup>. The principal underlying assumption for this hypothesis is that genetic variation for host-parasite interaction exists within populations and gives differential fitness to both the host and the parasite. Such genetic variation has been shown for different host-parasite systems [e.g. Refs 2,12,13, including variation at the major histocompatibility complex (MHC) in relation to infectious diseases in humans (severe cerebral malaria<sup>1</sup> and chronic lyme arthritis<sup>14</sup>)]. Given continuance of such variation, genetic recombination creates novel gene combinations. In a sexual population, every host constitutes a genetically unique environment for the parasite. Therefore, parasite adaptation to one host genotype is only of temporary benefit. Host diversity hinders evolution towards an optimal level of virulence, and we expect a level reflecting not only the evolution of the parasite to optimize host damage, but also the evolution of the host to minimize damage<sup>15,16</sup>.

**Observing the evolution of virulence**

The hypothesis that virulence in naturally coevolving populations is on average sub-optimal for the parasite allows us to make testable predictions. If host evolution is experimentally restricted by reducing host genetic variability, parasites would be expected to adapt to the predominant host genotype by shifting virulence upwards, towards their optimum. There is some empirical evidence that virulence increases during adaptation to a new host genotype. Influenza virus increases in virulence and multiplication rate when propagated experimentally within chickens with the same genetic background<sup>17,18</sup>. Virulence of the measles virus increases when transmission occurs between siblings, compared with transmission between non-related members of the same host population<sup>19,20</sup>. The primate malaria agent *Plasmodium knowlesi* initially causes mild infections in humans; however, after 170 artificial human to human transfers, its virulence had risen sharply, indicating its adaptation to the new host environment (discussed in Ref. 21). The well-known case of initial decrease of virulence of the myxoma virus in Australian rabbits does not contradict this. The above prediction assumes that host and parasites are in a dynamic balance and it is when this holds that slower evolutionary change of the host will allow the parasite to increase virulence. The myxoma virus, however, was released to control rabbits and was therefore chosen to be as deadly as possible<sup>1</sup>. Since it was in a non-natural host the optimal level of parasite virulence was far off the optimum – in this case, far above the optimum. The strong decline of myxoma virus virulence over the first few years after its release indicates the strong potential of parasites to respond to changes in their genetic environment. Although ecological factors might have contributed to all these described changes in virulence, adaptation to the novel host genotype appears to us to be the more likely explanation.

The adaptation of a parasite to a new host genotype that was formerly rare or absent often results in the loss or a decline of virulence in the host of origin<sup>17</sup>. This finding was used in the development of vaccines, by employing strains in immunization programs that became non-virulent for their normal host after being kept for some time in foreign hosts (e.g. vaccines against yellow fever virus and poliovirus<sup>22,23</sup>). This process emphasizes the dynamic, 'Red Queen' nature of host-parasite interactions<sup>7,10</sup>.

## Local adaptation

Support for the coevolutionary hypothesis of virulence also comes from studies using the reverse of the above argument. Namely, a parasite that infects a novel host (a host of a different genotype or population from that to which the parasite is adapted) should initially have, on average, a lower virulence in this new host, compared with that in its original host. In nearly all experiments where parasites were brought into contact with novel hosts, virulence and transmissibility decreased; this has been shown for viruses<sup>24</sup>, fungi<sup>25</sup>, helminths<sup>26,27</sup>, protozoans<sup>3</sup> and herbivores<sup>28–30</sup>. The reduction in virulence and transmissibility was stronger the more the novel hosts differed genetically from the host with which the parasite was associated before the experiment<sup>3</sup>. Experiments that did not find significant advantages for the parasite in its original host showed very high levels of genetic interactions between hosts and parasites<sup>31–33</sup> and this could contribute to the masking of local adaptation. Failure to detect local adaptation in cases where it is present can have various causes. Statistical power is weak when the within 'genetic unit' (e.g. host population, geographic area) variation is much larger than the variation explained by genetic isolation. Misjudgment of the scale of local adaptation might also lead to problems; for example, if parasites adapt to individual hosts rather than to host populations, detection of local adaptation across host populations might be difficult. Detection of local adaptation may also be hindered by acquired immunity of hosts, maternal effects on resistance, asymmetric gene flow between populations (source and sink populations) and insufficient time for adaptation. To our knowledge, no evidence has been presented against local adaptation.

Attention-attracting as they may be, cases where parasites showed devastating effects after accidental introduction into new host populations (e.g. rinderpest in Africa, Dutch elm disease, chestnut blight, HIV) appear to be exceptions<sup>3,13,16</sup>. There are likely to have been numerous failed introductions that have passed unnoticed. Most studies conducted under controlled conditions (see above) clearly show that parasites cause, on average, most harm in the host populations to which they are adapted.

## Testing the Red Queen hypothesis

In summary, genetic diversity of host populations appears to be crucial in hindering the parasite to evolve an optimal level of virulence. Sexual recombination benefits an outcrossing host through the production of variable offspring. In the context of host–parasite relationships, novel and rare genotypes have intrinsic advantages and may be selected. Asexual offspring, in contrast, cannot escape the antagonistic advances made during the previous generations by their parent's parasites. Genetic variability among hosts forces a parasite to adapt anew whenever it encounters a new host genotype<sup>10,34</sup>. The more different genotypes a host population consists of, the lower the frequency of each and the smaller the chance that a parasite will encounter the same genotype in successive hosts.

So far, direct evidence for the benefits of genetic diversity for host populations is weak, although numerous studies suggest such benefits. The vulnerability of monocultures to pathogen attack is notorious. Cereal monocultures have been shown to be more prone to attack by rapidly evolving, clone-specific, fungal diseases than genetically mixed cultures are<sup>35</sup>. Also, virulence of viruses was suggested to be higher in human populations with low genetic diversity at their MHC than in MHC-diverse populations<sup>20</sup>. Studies in natural populations are difficult, because frequency depend-

ence of the host–parasite arms race is time-lagged, and therefore one cannot expect a positive correlation between the parasite prevalence and frequency of host clones<sup>36</sup>.

The theory that sex is advantageous in the presence of rapidly evolving parasites<sup>7,8</sup> became known as the Red Queen hypothesis. Support for the 'sex against parasites' hypothesis has come mainly from comparative studies (e.g. Refs 9,37–39). Experimental studies are so far limited, with some support coming from the effects of herbivores on long-lived plants<sup>40,41</sup>. There is a strong need for experimental tests to clarify our understanding of arms races and the advantage of outbreeding. Boxes 1–4 summarize testable predictions derived from this review. Our predictions are stated for clonal host populations, but they are valid for all host organisms that can be brought to a lower level of genetic diversity than that found in normal wild populations (for example, by cloning, by using offspring from crossed inbred lines, or by using full-sib families). It is also helpful if strains of parasites and/or host can be 'genetically frozen' over time, to be used as a control in later experiments<sup>42</sup>. We hope that these predictions will stimulate research on the coevolution of sex and virulence.

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# Sexual selection and the brain

Lucia F. Jacobs

Sexual dimorphisms in the nervous system have been well studied, although largely as models for brain development, and not for their behavioral or evolutionary significance<sup>1</sup>. Most studies of sex differences in cognitive mechanisms similarly lack an explicit evolutionary analysis, despite the recent inroads of evolutionary theory into the field of human cognitive neuroscience<sup>2</sup>. Thus, the time is ripe to apply our understanding of sexual selection to the neural bases of behavior and to analyze sexual dimorphisms in the light of sexual selection hypotheses.

The theory of sexual selection was born from Darwin's observations of conspicuous sex differences in morphology and behavior: 'That these characters are the result of sexual and not of ordinary selection is clear, as unarmed, unornamented or unattractive males would succeed equally well in the battle for life and in leaving numerous progeny, if better endowed males were not present'<sup>3</sup>. It was obvious to Darwin that such conspicuous traits, particularly those displayed only during the breeding season, are involved in mate selection. Not all sexually selected traits are conspicuous,

**Sex differences are intrinsically interesting, particularly in the brain. When sexually dimorphic structures mediate learning, and when such learning ability is necessary to compete for mates, then such differences are best understood within the framework of sexual selection. By categorizing recent studies of sex differences in the brain by their role in mate competition, theories of sexual selection can be used to predict and characterize the occurrence of dimorphisms among species with different mating systems.**

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Lucia Jacobs is at the Dept of Psychology, University of California, Berkeley, CA 94720, USA.

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however, and when a sex difference consists of an enhancement of a cognitive or perceptual ability, disentangling the separate actions of natural and sexual selection is difficult. Sex differences could, after all, arise from other differences between males and females, such as different feeding habits. However, if such differences arise in the struggle for mates, not merely in the struggle for existence, then we can assume that they are subject to sexual selection. An example of this is the immunocompetence theory of sexual selection: if sexual selection favors the evolution of indicators of good health, then sexual selection will lead to the evolution of indicator structures, such as bright plumage<sup>4</sup>.

Learning ability and its underlying neural plasticity, like immune plasticity, allow an organism to track and respond to a changing environment. Thus the ability to recognize and recall previous encounters is a fundamental property of both immune and neural systems, and such properties may be subject to enhancement by sexual selection as well as natural selection. If females, for example, can assess a male's ability to learn more information, or learn more quickly than