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Spite, xenophobia, and collaboration between hosts and parasites

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Results of current computer simulations suggest that a pathogen-mediated version of Hamilton's spite hypothesis may play an unexpected role in the evolution of host-parasite interactions. Theory predicts that hosts' ability to influence parasite transmission to conspecifics is a neutral character provided that transmission routes are random. There are two possible deviations from this. Firstly, vertical transmission is biased in favour of kin, and this selects hosts to decrease pathogen transfer. Secondly, horizontal transmission is biased against kin and this selects hosts to increase pathogen transfer to conspecifics. Unfortunately, we typically do not know transmission statistics in relation to genetic kinship within a host population, thus cannot easily predict whether there is a selective pressure upon hosts either to transmit pathogens or not to transmit. Here I aim to show that even simple descriptive information on host and parasite life-histories may be used to make rough estimations about transmission routes and selection pressures. Interspecific predictions suggest that pelagic hosts, passively aerial hosts, and the intermediate hosts of pathogens transmitted along the predator-prey transmission routes are not selected to influence pathogen transmission. Intraspecific predictions suggest that parasitism and disease should facilitate emigration, and immigrants surrounded by non-related conspecifics should adaptively enhance pathogen transmission to conspecifics. This behaviour may select social animals, like humans, for an adaptive xenophobia in the context of an intraspecific arms race.

At the origin of the theory of kin selection, Hamilton (1970) proposed that altruism is expected to occur between any two animals of a population which are more closely related, and spiteful behaviour is expected to occur between any two individuals which are less closely related to each other than expected by chance. Spite was defined as a behaviour that harms conspecifics without a direct benefit to the self. There are few if any examples known for spite, and these examples are mostly limited within the context of infanticide (Gadagkar 1993). From the genetic point of view, however, harming non-relatives may not equal helping kin, because the benefits are diluted among not only kin, but the many non-kin that are not harmed. Moreover, there seem to be no easy practical ways for animals of identifying the least related conspecifics as compared to the several widespread modes of kin recognition. Thus, searching for the least related individuals within a population to harm them appears to be much less effective than exhibiting altruism toward the closest kin. However, animals harbouring chronic infections of virulent pathogens may face a different situation. Their ability to help relatives is reduced, whereas their ability to harm many others is greatly improved. It may well be adaptive to profit from this increased ability for spite by directing the flow of pathogens to non-related conspecifics.

Simulations in a host-parasite model system (Rózsa 1999) recently showed that influencing parasite transmission to conspecifics is a neutral character in hosts, provided that transmission routes are random. Under such circumstances, the harm caused by enhancing, or the benefits caused by reducing the transmission of virulent pathogens would equally affect all conspecifics regardless of their genotypes. Thus, there are no relative advantages or relative disadvantages attributed to different genotypes, and no selection occurs.

There are two possible deviations from random transmission routes. Firstly, vertical transmission is biased in favour of kin. In this case, hosts that harbour an allele to reduce pathogen transmission can focus this benefit upon kin who are likely to harbour copies of the same allele. This transmission route selects for a decrease of pathogen transfer. Secondly, horizontal transmission is also non-random, in the sense that genetic kin are affected less often than expected by chance. Under such circumstances, hosts that harbour an allele to increase pathogen transmission direct this harm preferentially toward non-related hosts, which are less likely to harbour similar alleles. Consequently, those who harbour an allele for promoting pathogen transmission would enjoy a relative advantage over those who do not harbour it. Horizontal transmission routes select for an increase of pathogen transfer, and this equals a pathogen-mediated version of Hamilton's spite hypothesis (Rózsa 1999).

Is it possible to select animals for an increase or decrease of pathogen transmission? A host-parasite interaction can be reasonably divided into three different phases: (1) infection into the host, (2) growth and multiplication within the host, and (3) transmission from one host to another. Both hosts and their parasites tend to exhibit different types of adaptations during these different phases, and these different adaptations are likely to be shaped by different components of host or parasite genomes, respectively (Fig. 1). Thus, the nature of host-parasite interactions can markedly differ from one phase to another. Undoubtedly, there is a conflict of interests between hosts and parasites during the first and the second phases, since hosts are adapted to avoid infection and to prohibit the growth and multiplication of invading pathogens, while parasites are adapted to evade these defences (Clayton and Moore 1997, Poulin 1998). However, once a parasite succeeds in entering a host individual, multiplying itself inside, and finally developing into an infective stage ready for transmission, then the previous host-parasite conflict does not necessarily affect the host's interest in the final phase of the interaction. Aspects of host behaviour such as degree of sociality, mating patterns (from monogamy to promiscuity), timing and location of defecation, contacts with potential vectors etc., are, arguably, characters with variable genetic components. Apart from other costs and benefits, these behavioural characters can potentially change the infected hosts' capability to distribute pathogens and parasites (Apanius and Shad 1994).

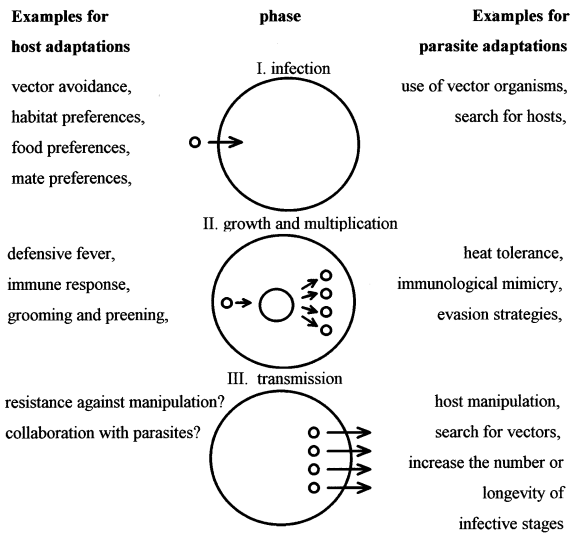

Fig. 1. Both hosts and parasites exhibit different adaptations in the three different phases of their interaction, and these different adaptations are likely to be shaped by different components of host or parasite genomes, respectively. Thus, it is reasonable to presume that selection for an optimal host adaptation in the third phase is not affected by the conflict of interest between hosts and parasites in the first and second phases.

Here, I wish to review some well-known biological phenomena, let them be either facts or just beliefs, in the light of the predictions yielded by the pathogen-mediated spite hypothesis. We just need to know the transmission statistics in relation to genetic relatedness within a particular host population, and then we can predict whether there is a selective pressure upon hosts either to transmit pathogens or not to transmit. Unfortunately, there are few if any data available in this field; however, even simple descriptive information on host and parasite life-histories may often help us to make rough estimations about transmission routes.

Interspecific predictions

Host animals often develop peculiar features when infected by parasites, such as modified behaviour, size, shape, or colour that may sometimes appear to enhance transmission of pathogens to new hosts. According to conventional wisdom, this phenomenon is rooted in a conflict of interests, say, an arms race between hosts and parasites. In this context, pathogen-induced modifications in host structure and function are interpreted (1) either as an adaptation of parasites to manipulate hosts, (2) or an adaptation of hosts to harm parasites, (3) or a non-adaptive side effect of their interaction (Moore and Gotelli 1990, Poulin et al. 1994). Indeed, some pathogen-induced modifications obviously promote pathogen transfer, and such modifications often do not emerge before the parasite develops into its infective stage of life and becomes ready to invade new hosts (see e.g. Poulin et al. 1992).

There is also an alternative interpretation of this phenomenon. Let us presume that there is no conflict between hosts and parasites, and hosts are not victims of parasitic manipulations. Rather the observed phenomenon is interpreted (1) either as a parasite adaptation which is neutral for the hosts, (2) or a shared adaptation mutually beneficial for both host and parasites, (3) or a non-adaptive accidental side effect of the interaction – which I do not deal with here.

Manipulative efforts of a pathogen may reduce host survival and reproductive success, and in this sense it may well be an integral part of pathogen virulence. Under such circumstances, hosts are selected for counter-adaptations to obstruct manipulations. On the other hand, however, one particular level of virulence may either come with or without manipulation, and manipulations are not necessarily virulent. This is particularly clear for the case of lethal pathogens and their ecological equivalents, parasites that castrate hosts. Though most of the known manipulators are not castrators, however, it may be worth taking a castrator as an example, such as the acanthocephalan *Plagiorhynchus*. This pathogen firstly castrates the isopod intermediate

host, and then manipulates it to increase its chance to become a prey of the definitive host (Moore 1983). After castration, there is no direct cost of being manipulated for an isopod: its dilemma resembles that of a criminal who has already been sentenced to death, but still has to decide whether to collaborate with the hangman or not.

Arguably, the nature of transmission routes influences the selection pressures on host behaviour in relation to pathogen transfer. Parent-offspring transmission routes dominate the pathogen transfer in many host-parasite species pairs. This is a kind of vertical transmission, thus predictably selects hosts to decrease their contribution to pathogen transfer. Meanwhile, it also selects pathogens to decrease virulence (Ewald 1994, 1995) due to a trade-off between competitive abilities versus persistence (Keeling 2000). Not surprisingly, hygienic behaviours of animals resulting in a decrease of parasite transfer often occur within the family circle. Furthermore, even without family bounds, individuals neighbouring each other in space and time often tend to be more closely related than expected by chance, causing a vertical bias of transmission (Wilkinson 1997) and giving precedence to adaptive hygienic behaviours.

Which life-history can randomise transmission routes? Firstly, currents of the physical environment can induce a randomising effect in planktonic or nektonic animals which float or swim freely in pelagic habitats. Moreover, small and aerial animals are often blown by the wind, thus they do not tend to be surrounded by genetic kin. In the case of parasite species with indirect developmental cycles (which utilise more than one host species in their life cycle) one pelagic or passively aerial species can randomise the transmission routes within both host species. Provided that *Anopheles* mosquitoes are frequently dispersed by the wind, the indirect transmission of *Plasmodium* from one human to another is randomised too.

On the other hand, terrestrial or benthic animals, and those living in aquatic or terrestrial vegetation, and species inhabiting small bodies of water are all likely to be surrounded by their genetic kin, even if they exhibit some degree of active dispersion from natal territories. Parasite transfer is likely to be vertically biased in these populations – except for pathogens with an indirect life cycle involving two or more host species of markedly different body size. In this case, pathogens move across a large scale of space and time while living in the large-bodied and long-lived host, which randomises transmission routes within the population of small-bodied and short-lived hosts. On the contrary, small-bodied and short-lived hosts cannot randomise transmission within the populations of large-bodied hosts. Having a look at reviews summarising most of the published cases of host manipulation by parasites (e.g. Moore and Gotelli 1990, Poulin 1994, 1995) yields in an interesting result. One can conclude that the vast majority (100 out of the 114 cases reviewed by Poulin 1994) of manipulative parasites observed up to date exhibit an indirect life cycle, and they manipulate the small prey (intermediate host) rather than the large predator (definitive host). Naturally, the possibility of a publication bias in favour of complex life cycles cannot be excluded.

Ewald (1994, 1995) interpreted this phenomenon in the context of the evolution of virulence. He argued that predator-prey transmission results in a non-vertical character of transmission routes within the prey populations, and therefore it selects pathogens for an increase of virulence. In this context, manipulating a host to become a prey is a particular manifestation of high virulence. This theory can explain which pathogens may afford to increase virulence; however, it cannot tell us why manipulations are included in some cases and not in others. The arguments presented here suggest that parasitic manipulations tend to occur particularly in those host species which are not subjected to selection pressures to influence the flow of pathogens to conspecifics.

Which life-histories can induce a horizontal bias of transmission routes? Sexual preferences for outcrossing inherent in many species can induce a horizontal bias in case of sexually transmitted diseases. In spite of that, infected hosts do not tend to increase sexual activity to enhance the flow of sexually transmitted pathogens (Lockhart et al. 1996). One possible explanation might be that increasing pathogen transmission along the sexual routes incurs a reduction of sexual partners' reproductive success, which may well be a cost much higher than the benefit of transmitting pathogens horizontally.

Intraspecific predictions

Provided that a host individual is chronically infected by virulent pathogens and cannot recover from disease, emigration from the spatial vicinity of kin may result in an advantage of redirecting the flow of pathogens from kin to strangers (Rózsa 1999). This response to a virulent infection may yield more advantages than suicide, which is also a possible way to prevent the infection of surrounding kin (McAllister and Roitberg 1987, Müller and Schmid-Hempel 1992; but see also Poulin 1992). Indeed, parasitic infection is known to correlate positively with emigration in cliff swallows (*Hirundo pyrrhonota*) (Brown and Brown 1992) and flour beetles (*Tribolium confusum*) (Yan et al. 1994). When reviewing 60 published cases of parasite-induced changes in host spatial activity, Poulin (1994) found an increase of mobility in 23 cases. Contrary to his expectations, parasites that did not appear to benefit from host manipulation had the greatest impact on host behaviour. Though mobility was examined at a small

spatial scale in these experiments, however, these results prompt the question whether infected individuals are always victims of parasitic manipulations or may benefit from an adaptive capability to emigrate.

Whether or not host emigration was induced by pathogens or other causes, an immigrant is likely to be surrounded by conspecifics less closely related genetically than expected by chance. Under such circumstances, an adaptive individual strategy is to increase the transmission of pathogens and parasites. At this point, hosts and parasites may share a common interest and collaborate with each other to enhance transmission – supposing that the host is unable to recover from disease. Naturally, eliminating pathogens and merging into the population of local natives would be an even more advantageous alternative for an immigrant animal, but this possibility is not open to all.

The adaptive value of emigration as a disease-dependent feature can explain the adaptive value of xenophobia aimed at prohibiting immigration, apparently characterising at least some of the social species (Freeland 1976, Peck 1990). These two adaptations may evolve in an arms race-like manner within the gene-pool of the same population, since their manifestations are limited to different situations, i.e. to different levels of parasitism.

Human behaviour in relation to pathogen transfer

Arguably, human behaviour may also be influenced by past selective pressures that were exerted by virulent parasites and pathogens upon our ancestors. In our species too, immigrants can be hypothesised to transmit pathogens horizontally. Moreover, non-reproductive sex is common in humans, characterised by a preference for outcrossing, and it is not necessarily limited within the boundaries of families or small social groups, giving rise to a possibility for a horizontal bias in pathogen transfer. In particular, prostitutes and homosexuals may transfer pathogens horizontally without the disadvantage of decreasing their own reproductive success by harming the health of reproductive partners. Consequently, when behaving instinctively, immigrants, prostitutes and homosexuals may be hypothesised to transmit pathogens more readily than the so-called "typical natives".

Theoretically, when behaving instinctively, humans are also predicted to show intraspecific counter-adaptations, such as xenophobia manifested in "typical natives" to exclude immigrants, prostitutes and homosexuals from the context of social and sexual relations. When behaving instinctively, they are predicted to blame immigrants, prostitutes and homosexuals for transmitting pathogens either ignorantly or purposely, even without any cause for such denouncing accusations. Indeed, the local majority of human societies often attribute bad body smell or other simple symptoms of low hygienic standards to immigrants or ethnic minorities with no evident reason for doing that (Gould 1981).

Since inherent xenophobia is so vivid in human societies, I see no possibilities to examine explicitly whether immigrants or any other groups of humans may have a specific role in distributing pathogens in a society. It may be worth noting, however, that even the medical organisations of the United Nations – organisations which are hopefully not so blinded by prejudice and racism as many of us – approve that migrants (UNAIDS 1998), sex-workers and homosexuals (WHO 1999) play a specific role in the spread of the current AIDS epidemic.

One could also argue that the pure existence of human xenophobia is in itself a fact which supports the view that horizontal transmission of pathogens by immigrants exerted a selective pressure upon our ancestors. However, this is a poor argument in itself, since the pathogens transmitted randomly or horizontally are also predicted to develop a higher virulence than those transmitted vertically by "typical natives" (Ewald 1994, 1995).

Anyway, this is just an adaptationist argument, which does not necessarily yield realistic predictions (Pigliucci and Kaplan 2000), particularly in the case of modern human behaviour. For those readers who believe that our behaviour is partially shaped by past adaptations, the hypothesis outlined above may help to understand the obvious fact that humans are prone to xenophobia. Evidently, attributing an adaptive value to a particular behaviour in early hominid societies does not mean that this behaviour is acceptable either morally or politically in a modern society.

Overall, the idea that different types of transmission routes select parasites and pathogens for different levels of virulence became widely accepted in recent years. Now we face an emerging new hypothesis which suggests that different types of transmission routes also select infected hosts for different contributions to the transmission of parasites and pathogens to conspecifics. A surprising new aspect of host-parasite interactions is – as already exemplified by our own history – that resistance often comes together with collaboration.

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