Do parasites live in extreme environments? Constructing hostile niches and living in them

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SUMMARY

We develop the hypothesis that parasites do not invade extreme environments, i.e. hostile hosts, but rather 'create' them. We argue that parasites may have driven the evolution of the constitutive and adaptive immune system. This leads to several implications. First, parasites respond to 'genes to kill' by 'genes to survive' and this triggers an indefinite selection of measures and counter-measures. Second, these coevolutionary arms races may lead to local adaptation, in which parasite populations perform better on local hosts. Third, the evolution of the immune system, whose responses are predictable, may allow parasites to specialize, to evade and even to manipulate. Finally we show that the correlations between the increase in the antibody repertoire, the expansion of MHC loci and parasite pressures support our hypothesis that both host complexity and parasite pressures can be invoked to explain the diversity of antibodies, T-receptors and MHC molecules.

Key words: immune system, host niche, host specificity, MHC.

INTRODUCTION

When parasites exploit organisms which live in extreme environments (for instance at the most extreme temperatures which are still compatible with life), their free-living stages are exposed to extreme conditions. This is one aspect of the adaptation of parasites to extreme environments. A different question is: can the environment where parasites live (i.e. the host) be qualified as extreme or not?

In recent years, it has been increasingly perceived that organisms modify their environments and that these changes can, in their turn, have consequences for selective pressures exerted by these environments. These changes apply to other organisms in the same ecosystem and to the organisms responsible for the environmental alteration itself. The way an organism modifies natural selection for other organisms sharing its ecosystem relates to the concept of ecosystem engineers developed by Jones, Lawton & Shachak (1994, 1997). The way an organism modifies natural selection for itself is closer to the concept of niche construction developed by Odling-Smee, Laland & Feldmann (1996), and by Laland, Odling-Smee & Feldmann (1996, 2000).

Parasites have already been considered as ecosystem engineers. For instance, Thomas *et al.* (1998) and Poulin (1999) have shown that the behaviour of the cockle *Austrovenus stutchburyi* was altered by metacercariae of the trematode *Curtuteria australis* in such a way that parasitized individuals were unable to bury themselves and remained exposed at the surface of the sediment. In the area under study (shores of New Zealand), cockles are one of the rare hard surfaces where an intertidal community of benthic invertebrates (limpets, anemones, bryozoans, barnacles, etc.) can establish. When a cockle was infected by *C. australis*, limpets (*Notoacmea helmsi*) were significantly more frequent and anemones (*Anthopleura aureoradiata*) less frequent, in relation to the depth at which the substrate provided by the cockle was available. The trematode was thus an ecosystem engineer since it modified the quality of the environment for organisms not directly involved in the parasite–host association.

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PARASITES CONSTRUCT HOSTILE NICHES

Regarding the influence that an organism may have on the selective pressures exerted on itself (niche construction), the best possible example is provided by parasite-host relationships themselves because 'living in a living environment' always provokes a dramatic change in the quality of the niche. A living organism which would otherwise provide the parasite with a peaceful habitat and an indefinite source of energy soon becomes a killing machine possessing an impressive battery of weapons (phagocytic cells, cytotoxic cells, antibodies, enzymes, etc.) (Frost, 1999).

Parasites do not invade extreme environments. They 'create' them as we may argue that parasites

have driven the evolution of the constitutive and adaptive immune system.[1]

This has several important evolutionary consequences. One is that parasites respond to 'genes to kill' by 'genes to survive' (Combes, 1995). This triggers an indefinite selection of measures and counter-measures which are probably one of the main supporting examples of the Red Queen hypothesis (Van Valen, 1973). In their turn, such arms races provide one of the most likely explanations for maintenance of sex (see for instance Hamilton, Axelrod & Tanese, 1990; Ladle, 1992) and for sexual selection (the quest for 'good genes' which constitutes the basis of Hamilton and Zuk's 1982 hypothesis).

Second, these coevolutionary arms races and the short generation times of parasites as compared to those of their hosts, may lead to local adaptation, in which parasite populations perform better on local hosts. Indeed, several mathematical models (Gandon *et al.* 1996; Morand, Manning & Woolhouse, 1996) and empirical studies (Lively, 1989, 1999; Xia, Jourdane & Combes, 1998; Moné, Mouahid & Morand, 2000) have shown that parasites are more compatible or better infective to their local hosts, i.e. in sympatric conditions, than to geographically distant hosts, i.e. in allopatric conditions (see Kaltz & Shykoff, 1998). Although the underlying processes and mechanisms are still poorly known, they represent one of the aspects of the survivorship of parasites in the hostile niche.

HOSTILE NICHE AND HOST SPECIFICITY

Bristow (1988) remarked that, in predator-prey systems, specialization arises when a particular defence mechanism has been selected in prey, for instance a thick carapace, the secretion of toxic substances, or an homochromy with the milieu. When such adaptations are selected in prey, counter-adaptations are usually selected in only a small number of predators and allow them to specialize on the resource. Combes & Théron (2000) suggested that specialization in parasites could be the result of a similar process: parasites would be specific (in general) because adaptations necessary to survive in the hostile environments they have 'created' in hosts are extremely costly. Since immunity is achieved through different pathways in different host species (in the same way that different plant species produce different compounds as an adaptation to combat herbivorous insects), parasites specialize to survive in a limited number of these hostile environments. It does seem that the efficiency of evasion mechanisms of a parasite or parasitoid is better when it exploits few host species than when it exploits many (Dupas & Boscaro, 1999) which is an indirect argument for the above statement. 'Extreme' environments following infection in hosts have the same evolutionary consequences as plant defences and trigger the emergence of specialist parasites.

The immune response to a given parasite implies a rather precise cascade of specific and non-specific mediators within a defined temporal sequence (see Davey, 1990). This means that immune responses are predictable, which allows parasites to specialize, to evade and even to manipulate (see Frost, 1999).

Host specificity can thus be the result of building a hostile niche and living in it.[2]

PARASITES AND THEIR EVER-EVOLVING HOSTILE ENVIRONMENT

The sophistication of immune systems has obviously increased from 'lower' to 'higher' organisms. This is especially true when MHC molecules, T-receptors and the antibody repertoire of vertebrates are considered. This 'ever-evolving hostile environment' might have had two different causes, acting in parallel. (1) Through evolution, the increasing complexity of free-living organisms has provided pathogens with an increasing diversity of niches opened to colonization, the result of which was an increasing diversity of parasites. Klein (1991) argued that, in order to have an impact on MHC evolution, a parasite must coevolve over a long period of time, also emphasising which parasites may play this role. We may argue that it is not a parasite species nor a parasite group *per se* (say trypanosomes as exemplified by Klein, 1991) that is responsible for MHC evolution, but rather a community of parasite species, i.e. parasite species richness (but see Paterson, Wilson & Pemberton, 1998). By using parasite species richness as an indicator, Morand (2000) showed that helminth species richness is low in reptiles and amphibians (from 3·7 to 6·7 parasite species per host species) and high in birds (14·0) and mammals (12·0) (Poulin & Morand, 2000; Morand,

[1] Ectoparasites 'cumulate' classical pressures from the abiotic and biotic environments with pressures due to immunological responses of the hosts. For instance, ectoparasites living on the gills of fish are directly affected by external factors such as water temperature, water currents, activities of cleaners (Hart *et al.* 1990). Cleaner fishes in particular have a strong influence on their client's ectoparasite load (Grutter, 1996, 1999; Arnal & Côté, 1998). Endoparasites themselves are submitted to factors that can be qualified as extreme and do not originate in immunity, such as pH, enzyme activities, fluid movements, etc. (Sukhdeo, 1997).

[2] This does not mean that specialization always arises from the hostility of the niche. For instance specialization may occur from a 'lock-and-key' relationship between two anatomical features of the host and the parasite (see Morand *et al.* 2000 for a positive relationship between gopher hair-shaft diameter and louse head-groove width).

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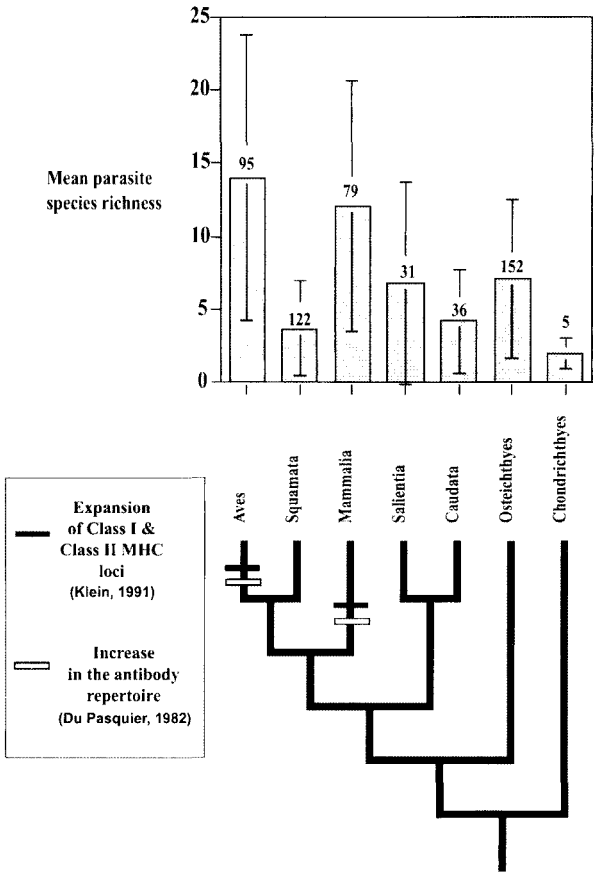

Fig. 1. Correlation between the increase of the antibody repertoire, expansion of MHC loci and parasite richness in vertebrates.

2000) (Fig. 1). Although the helminths are not the only pathogens that may have an effect, we may expect a co-variation in parasite diversity, in such a way that host species harbouring a high species richness of helminths should also harbour a high diversity of protozoans, bacteria or viruses.

The high diversity of parasites was responsible for increasing pressures on immune systems, which responded by more and more sophisticated weapons, especially in vertebrates (Klein, 1991). Lower vertebrates possess fewer specific antibodies than birds and mammals, respectively fewer than 500000 as compared with between 10^7 and 10^9 (Du Pasquier, 1982; see Frost, 1999). Similarly, the emergence of the MHC in the lower vertebrates was followed by dramatic expansion and duplication of MHC genes in birds and mammals (Klein, 1991). MHC genes are not neutral and their persistence and the level of their polymorphism can be explained by balancing selection caused by parasites and pathogens (Takahata, 1990). Other comparative studies should be carried out both inter- and intra-specifically in order to confirm the hypothesis of a parasite-driven force. In response to the evolving immune systems (a 'Red Queen' process ...), pathogens acquired adaptations to escape recognition, using for example antigens similar to host self antigens, to provoke immunosuppression or to provoke non-specific lymphocyte polyclonal activation, which results in the dilution of the specific response against the parasite (Reina-San-Martin, Cosson & Minoprio, 2000).

(2) However, it is probable that pathogens were not alone in being responsible for the complexity of immune systems. We may hypothesize that antigenic diversity increased in proportion to structural complexity of organisms and that the development of the immune system also has been influenced by the necessity to become tolerant to an increasing number of self antigens.

In the current state of knowledge, we think that the correlations between the increase in the antibody repertoire, the expansion of MHC loci and parasite pressures (Fig. 1) support the hypothesis that *both* host complexity and parasite pressures can be invoked to explain the diversity of antibodies, T-receptors and MHC molecules.

CONCLUSION

As a rule, extreme environments and/or hostile environments are defined on the basis of the difficulty of maintaining life and are thus characterized by a low diversity in term of species richness (see for instance Vernon, Vannier & Trehen, 1998). Contrary to this, living in a hostile niche seems to promote both specialization and diversification in parasites. Poulin (1992) and Sasal, Desdevises & Morand (1998) found that high host specificity is correlated with high species diversity in fish parasites.

Finally, it must be stressed that the notion of the extreme environment is somewhat anthropomorphic. For an animal adapted to live in a desert (usually referred to as an extreme environment), it can be lethal to be transported to a temperate climate (supposed to be not extreme). Once an organism is adapted to an environment, this is the environment where its fitness is best, whether we qualify it as extreme or not. It is the same for parasites. It might be concluded that it is more comfortable to live inside a polar bear than on the pack ice. The polar bear does live in an extreme environment. Its parasites do not.

REFERENCES

ARNAL, C. & CÔTÉ, I. M. (1998). Interactions between cleaning gobies and territorial damselfish on coral reefs. *Animal Behaviour* **55**, 1429–1442.

BRISTOW, C. M. (1988). What makes a predator specialize? *Trends in Ecology and Evolution* **3**, 1–2.

COMBES, C. (1995). *Interactions Durables. Ecologie et Evolution du Parasitisme*. Paris, Masson.

COMBES, C. & THÉRON, A. (2000). Metazoan parasites and resource heterogeneity: constraints and benefits. *International Journal for Parasitology* **30**, 299–304.

DAVEY, B. (1990). *Immunology. A Foundation Text*. New Jersey, Prentice Hall.

DU PASQUIER, L. (1982). Antibody diversity in lower

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vertebrates – why is it so restricted? *Nature* **296**, 311–313.

DUPAS, S & BOSCARO, M. (1999). Geographic variation and evolution of immunosuppressive genes in a *Drosophila* parasitoid. *Ecography* **22**, 284–291.

FROST, S. D. W. (1999). The immune system as an inducible defense. In *The Ecology and Evolution of Inducible Defenses* (ed. Tollrian, R. & Harvell, C. D.), pp. 104–126. Princeton, Princeton University Press.

GANDON, S., CAPOWIEZ, Y., DUBOIS, Y., MICHALAKIS, Y. & OLIVIERI, I. (1996). Local adaptation and gene-for-gene coevolution in a metapopulation model. *Proceedings of the Royal Society of London, Series B Biological Sciences* **263**, 1003–1009.

GRUTTER, A. S. (1996). Parasite removal rates by the wrasse *Labroides dimidiatus*. *Marine Ecology Progress Series* **130**, 61–70.

GRUTTER, A. S. (1999). Cleaner fish do clean. *Nature* **398**, 672–673.

HAMILTON, W. D., AXELROD, R. & TANESE, R. (1990). Sexual reproduction as an adaptation to resist parasites (a review). *Proceedings of the National Academy of Sciences, USA* **87**, 3566–3573.

HAMILTON, W. D. & ZUK, M. (1982). Heritable true fitness and bright birds: a role for parasites? *Science* **218**, 384–386.

HART, B. L., HART, L. A. & MOORING, M. S. (1990). Differential foraging of oxpeckers on impala in comparison with sympatric antelope species. *African Journal of Ecology* **28**, 240–249.

JONES, C. G., LAWTON, J. H. & SHACHAK, M. (1994). Organisms as ecosystem engineers. *Oikos* **69**, 373–386.

JONES, C. G., LAWTON, J. H. & SHACHAK, M. (1997). Positive and negative effects of organisms as physical ecosystem engineers. *Ecology* **78**, 1946–1957.

KALTZ, O. & SHYKOFF, J. A. (1998). Local adaptation in host–parasite systems. *Heredity* **81**, 361–370.

KLEIN, J. (1991). Of HLA, Tryps, and selection: an essay on coevolution of MHC and parasites. *Human Immunology* **30**, 247–258.

LADLE, R. J. (1992). Parasites and sex: catching the Red Queen. *Trends in Ecology and Evolution* **7**, 405–408.

LALAND, K. N., ODLING-SMEE, F. J. & FELDMANN, M. W. (1996). On the evolutionary consequences of niche construction. *Journal of Evolutionary Biology* **9**, 293–316.

LALAND, K. N., ODLING-SMEE, F. J. & FELDMANN, M. W. (2000). Niche construction, biological evolution, and cultural change. *Behavioral and Brain Sciences* **23**, 131–175.

LIVELY, C. M. (1989). Adaptation by a parasitic trematode to local populations of its snail host. *Evolution* **43**, 1663–1671.

LIVELY, C. M. (1999). Migration, virulence, and the geographic mosaic of adaptation by parasites. *The American Naturalist* **153**, S34–S47.

MONÉ, H., MOUAHID, G. & MORAND, S. (2000). On biogeographical history of *Schistosoma bovis* Sonsino, 1876 in the light of both intermediate host spectrum and compatibility in the mollusc–parasite association. *Advances in Parasitology* **44**, 99–138.

MORAND, S. (2000). Wormy world: comparative tests of theoretical hypotheses on parasite species richness. In *Evolutionary Biology of Host–Parasite Relationships: Reality Meets Models* (ed. Poulin, R., Skorping, A. & Morand, S.). Elsevier (in press).

MORAND, S, HAFNER, M. S, PAGE, R. D. M & REED, D. L. (2000). Comparative body size relationships in Pocket Gophers and their Chewing Lice. *Biological Journal of the Linnean Society* **70**, 239–249.

MORAND, S., MANNING, S. D. & WOOLHOUSE, M. E. J. (1996). Parasite-host coevolution and geographic patterns of parasite infectivity and host susceptibility. *Proceedings of the Royal Society of London, Series B Biological Sciences* **263**, 119–128.

ODLING-SMEE, F. J., LALAND, K. N. & FELDMANN, M. W. (1996). Niche construction. *American Naturalist* **147**, 641–648.

PATERSON, S., WILSON, K. & PEMBERTON, J. M. (1998). Major histocompatibility complex variation associated with juvenile survival and parasite resistance in a large unmanaged ungulate population (*Ovis aries* L.). *Proceedings of the National Academy of Sciences, USA* **95**, 3714–3719.

POULIN, R. (1992). Determinants of host-specificity in parasites of freshwater fishes. *International Journal for Parasitology* **22**, 753–758.

POULIN, R. (1999). The functional importance of parasites in animal communities: many roles at many levels? *International Journal for Parasitology* **29**, 903–914.

POULIN, R. & MORAND, S. (2000). The diversity of parasites. *Quarterly Review of Biology* (in press).

REINA-SAN-MARTIN, B., COSSON, A. & MINOPRIO, P. (2000). Lymphocyte polyclonal activation: a pitfall for vaccination design against infectious agents. *Parasitology Today* **16**, 62–67.

SASAL, P., DESDEVISES, Y. & MORAND, S. (1998). Host-specialization and species diversity in fish parasites: phylogenetic conservatism? *Ecography* **21**, 639–645.

SUKHDEO, M. V. K. (1997). Earth's third environment: the worm's eye view. *BioScience* **47**, 141–149.

TAKAHATA, N. (1990). A simple genealogical structure of strongly allelic lines and trans-species evolution of polymorphism. *Proceedings of the National Academy of Sciences, USA* **87**, 2419–2423.

THOMAS, F., RENAUD, F., DE MEEÛS, T. & POULIN, R. (1998). Manipulation of host behaviour by parasites: ecosystem engineering in the intertidal zone? *Proceedings of the Royal Society of London, Series B Biological Sciences* **265**, 1091–1096.

VAN VALEN, L. (1973). A new evolutionary law. *Evolutionary Theory* **1**, 1–30.

VERNON, P., VANNIER, G. & TREHEN, P. (1998). A comparative approach to the entomological diversity of polar regions. *Acta Oecologica* **19**, 303–308.

XIA, M., JOURDANE, J. & COMBES, C. (1998). Local adaptation of *Schistosoma japonicum* in its snail host demonstrated by transplantation of sporocysts. *ICOPA IX, Monduzzi Editore* (ed. Tada, I., Kojima, S. & Tsuji, M.), pp. 573–576.