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Editorial

Making the pathogen: Evolution and adaptation in parasitic protists

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> Life arose as a living molecule or protogene, the progression from this stage to that of the ameba is at least as great as from ameba to man. All the essential problems of living organisms are already solved in the one-celled (or, as many now prefer to say, noncellular) protozoan and these are only elaborated in man or the other multicellular animals. . . The change from protogene to protozoan was probably the most complex that has occurred in evolution, and it may well have taken as long as the change from protozoan to man.
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> George Gaylord Simpson, The Meaning of Evolution: A Study of the History of Life and of its Significance for Man (1949), 16.
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> Animals, whom we have made our slaves, we do not like to consider our equal.
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> Charles Darwin, The B notebook (1837/8).

Parasitic protozoa have been a significant threat to human heath and civilization for millennia. They continue to influence and mould human society and behaviour greatly, and are thus a high priority for research and public health campaigns worldwide. The many challenges that these organisms present, in terms of huge variations in lifestyle, host–vector interactions, drug sensitivity and immune evasion mechanisms are all, of course, a product of their diversity and coevolution with their hosts. It is a coevolution that conceivably originated in the Cambrian, coincident with the radiation of major animal lineages, including that leading to extant terrestrial arthropods [1], and in all likelihood long before the rise of vertebrates [2].

Successful parasitism is a balance between exploiting host resources, pathogenesis, replication rate and transmission efficiency, and must be such that, for the most part, the needs of the host remain met. We are just beginning to understand how such adaptations function, and with the advent of genome sequencing the opportunity to assess evolution and change is now present. Whilst reductionist, it is also possible to start to reconstruct and build up again from individual genes to whole systems, pathways and organelles as well as intact organisms and their detailed evolutionary histories.

This special issue considers the evolution of organelles and gene families in parasites, together with some more theoretical considerations of how new functions and compartments can evolve. These are subjects that interest us because, in part, many of the variations in organelle and cell surface biology documented for parasites play key roles in virulence. Yet, from comparative genomics of the free-living relatives we're also beginning to realise that the biology often associated with the adaptation to parasitism can pre-date this transition and may even facilitate, rather than be the consequence of, assuming life as an obligate parasite [3–5]. The major lineages of parasites that mainly concern human health are focused on in this special volume are the Kinetoplastida, Apicomplexa and Entamoebids, which are part of the Excavata, SAR and Amoebozoa supergroups respectively. Additional important parasites also reside within the heterolobosids of the Excavata and are briefly discussed.

The authors consider the nucleus and the nuclear envelope and how emerging work is demonstrating the contributions that conserved and lineage-specific proteins make to control of gene expression and hence immune evasion mechanisms. Also considered are energy producing systems in the mitochondrion and how these functions have altered and may contribute towards adaptations to host resources, together with discussions of the cytoplasm, peroxisomes, acidocalcisomes, membrane trafficking pathways, the cytoskeleton and finally the critical proteins at the parasite surface. These latter close the circle as they are frequently the home of gene products that have special mechanisms within the nucleus for their expression and also act as major contributors towards immune evasion and host parasite interactions, and which has emerged as a major point of evolutionary change.

The great evolutionary divergence separating parasite and host is reflected in many features. One of the more obvious is the presence of apparently lineage-specific genes, as well as genes with extremely low degrees of similarity between host and pathogen, and where the ability to predict function becomes challenging. Many of these are thought to arise either as the direct result of selective pressure, or to have achieved fixation through the frequent bottlenecking that parasite populations experience, essentially each time they are transmitted. This latter process serves to accelerate the potential fixation of alleles and provides an important

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additional driver for the evolution and diversification of parasitic organisms [6]. Significantly, differing solutions to the same or similar biological problems are frequent, with distinct or very divergent mechanisms and players facilitating these various roles. These processes have frequently been considered as attractive drug targets, but in many instances this promise remains to be fulfilled. Perhaps comfortingly, we find that it is possible to map functions using conserved marker genes, and that reconstructing and predicting parasite processes and cellular architecture from sequence data is indeed possible and highly valuable.

Simpson was of course correct in his insight of the massive gulf between parasites and man. Remarkably, modern evidence suggests that the origin of life at ~3.5 bya and the radiation of eukaryotes at ~1.5 bya are surprisingly similar in that it took 2 by to evolve the eukaryotic form, and a further 1.5 by to attain the present levels of diversity within eukaryotic organisms. Returning to our quote from Darwin, we know that parasites have been our constant companions for many millennia and continue to resist our attempts at enslavement. As the articles in this volume we hope attest, they are, in terms of age and sophistication, very much our equals.

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