What is Myoparasitology?

This Hot BAM is devoted to different aspects of host-parasite relationships, in which both skeleton and cardiac muscle tissues are involved.

Some parasites, such as the nematode Trichinella, have chosen skeleton muscles as the ideal habitat for growing and developing. To this end they enter the muscle cells and induce modifications in these cells which become “nurse cells” so allowing parasite to survive. T. spiralis behaves as true intracellular organism whereas T. pseudospiralis remains free among muscle fibers. Knowledge about this host-parasite relationship is continually increasing and we are beginning to understand the fine molecular mechanisms underlying muscle modifications induced by the parasite also. A Trichinella spiralis MyoD homologue has recently been cloned. These aspects will be discussed in detail in the reviews by Stewart and Gabryel and collaborators. The clinical implications of muscle invasion by Trichinella spiralis will be elucidated in the paper by Kociecka.

Also protozoa, for example Toxoplasma gondii or Sarcocystis, are able to penetrate striated muscle cells and remain there for some time, causing damage to the host cells. Lindsay and collaborators review the host-parasite relationships in these two infections.

In some cases the most important consequences of parasitic infection are not at skeleton but cardiac muscle level and Chagas’ disease is a unique example of this. The review by Tanowitz and collaborators elucidates very clearly all aspects of this heart involvement. Trypanosoma cruzi is responsible for very severe impairment in the functioning of this organ by different mechanisms ranging from induction of an autoimmune response to dysregulation of cardiocyte function due to alteration in the integrity of signal transduction components. The parasite per se can cause perturbations in biochemical pathways in both endothelial and myocardial cells and the final result is coronary microvascular spasm which is responsible for the focal pathology.

The heart may also be involved indirectly as a consequence of parasite infection, for example an increase in levels and activation of inflammatory cells such as eosinophils and mast cells which occurs during trichinellosis. The paper by Paolocci and collaborators describes the morphological aspects of myocarditis in experimental trichinellosis and presents some new aspects with respect to the better known model of myocarditis in another experimental helminthic infection such as toxocariasis.

A particular and little known aspect of muscle parasitism is Microsporidia muscle invasion which causes myositis. In particular it has been reported that parasites of Pleistophora genus are responsible for myositis in immunodepressed patients; this suggests that an immune response dysfunction is indispensable for muscle invasion by these parasites. Pozio reviews this parasitic infection.

The aim of this Hot BAM is to demonstrate to myologists, that parasitic infections can be very interesting models for studying muscle modifications and, to parasitologists that both skeleton and cardiac muscle tissues are very useful for studying the host-parasite relationship, the so-called “myoparasitology”.

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