A family of PP2 phosphatases in and parasitic protozoa: reply.

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How does the Sparganosis Occur?

Several species of helminths can absorb arachidonic acid (AA) from their environment and metabolize it to form prostaglandins. Schisosoma mansoni cercariae can form proplasmoid larvae, which were thought to have a role in the penetration of the cercariae into the host skin. S. mansoni cercariae release prostaglandin E2, which may regulate host macrophage and lymphocyte functions. Proplasmoid larvae are thought to be involved not only in the defense mechanism against host immune systems, but also in the penetration and the migration mechanism that may cause sparganosis.

In general, Diphyllobothrium larvae in fish, which have lower temperature and lower AA concentration, don't cause sparganosis in humans. However, S. erinaceieuropaei plerocercoids do cause sparganosis in humans. What happens when the plerocercoid changes from a host of lower temperature to that of higher temperature, and from higher to lower AA concentration? The hamster has an AA/arachidonic acid ratio (A/E) of 1.76 in serum, which is very similar to that of the human, 1.75 (Ref. 5); it is therefore a snake and hamster, respectively. The A/E plerocercoid was 29.33 and 12 in the skin. The penetration of the cercariae into the host were thought to have a role in the cercariae can form prostaglandins, which may cause sparganosis.

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Letters

A Family of PP2 Phosphatases in Plasmodium falciparum and Parasitic Protozoa: Reply

The life cycle of Plasmodium is complex. Even within the sexual phase, there are a number of distinct developmental processes. These include: gametocytogenesis, formation of both male and female gametocytes, fertilization (which results in the release of a single motile male gamete from a single male gametocyte), fertilization of the extracellular gametocytes, and ookinete development. The mechanisms by which these events are controlled are not yet understood, but will involve precise interplay of signaling pathways.

We have recently isolated a number of genes from Plasmodium falciparum encoding enzymes potentially involved in signal transduction, all of which are expressed specifically in the sexual eugonytic stages of the life cycle. These include two cyclases (DS, Carucci et al., unpublished; A.A. Witney et al., unpublished) and two protein phosphatases (PP-α, Ref. 1 (PPP-B), Ref. 2). On the basis of structural comparisons, PPP-α is most closely related to phosphatases in the type-1 subgroup whereas PPP-B shows greatest similarity to protein phosphatase type-2A (PP-2A).

The Comment article by Garcia et al. (this issue) discusses features of three α- threonine phosphatase homologues from P. falciparum. The first is the unusual ‘double’ PPP-2C that might be involved in a stress response; as it can complement the heat-shock response defect of a Schizosaccharomyces pombe strain with a PPP-C deletion. Second, the new putative type-2A phosphatase (MP0301) from the P. falciparum sequence database (Sanger Centre, Hinxton, UK), on which the basis of amino acid sequence, is likely to be a PP-2A. MP0301 contains the sequence CYRCG, which is closest to phosphatases in the type-2A subgroup whereas PPP-B shows greatest similarity to protein phosphatase type-2A (PP-2A).

The activity of PPP-B may be regulated by reversible phosphorylation of the N-terminal segment. The sequence of PPP-B contains the motif CYRCG, suggestive that it might also be highly sensitive to the inhibitor okadaic acid. However, our work has indicated that okadaic acid does not affect early gametocyte development in vitro (B.S. Hall et al., unpublished), implying that PPP-B might be involved in later sexual stage events. It is known that PP-2A exists as a heterotrimeric/heterozygous composed of a common core structure associated with different regulatory subunits (2). Clearly, identification and characterization of the potential regulatory subunits of PPP-B will give a greater understanding of the precise role of this molecule.

In addition experiments are in progress (in collaboration with A.F. Cowman, Walter and Eliza Hall Institute, Melbourne, Australia) to disrupt the genes we have isolated, in order to investigate their role in sexual development.

References


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Erratum

In Tables 1 and 2 of the article: How do protozoan parasites survive inside macrophages? By Christian Boggin and Martin Röllighoff (Parasitol. Today 15, 22–28, 1999) the word _protozoon_ should be spelt out as _Trypanosoma_ gen. (not _protozoon_ gen).

We apologize for the error.

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