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Perspective

Treatment of Schistosoma through Praziquantel

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DESCRIPTION

The majority of schistosome infestations and many cestode infestations require the anthelmintic praziquantel. Praziquantel causes the cell membrane to become more permeable, which causes schistosomes to constrict. The medication also results in vacuolization and tegument disintegration in schistosomes. Compared to immature worms, the effect is more pronounced on adult worms. An enhanced calcium flow could be crucial. Inhibition of glucose uptake, a decrease in glycogen stores, and stimulation of lactate release are all secondary effects. Praziquantel only exclusively affects trematodes and cestodes; it has no effect on nematodes (including filariae).

Praziquantel works by paralysing the worms' muscles and inducing violent spasms in them. A fast Ca2+ influx inside the schistosome is present along with this paralysis and is likely what causes it. Another initial impact of praziquantel is morphological changes. The exposure of schistosome antigens at the parasite surface is also increased as a result of these structural changes. After that, the worms are either totally eliminated in the intestine or expelled in the stool. Praziquantel's peculiarity of being comparatively inefficient against young schistosomes is intriguing. Despite being effective at first, potency against schistosomes declines until it approaches a minimum at 3-4 weeks. The effectiveness then resumes its upward trend until it reaches its peak at 6-7 weeks. Schistosomiasis vaccines and medications frequently target Glutathione S-Transferase (GST), an important detoxifying enzyme in parasitic helminths. The only known target of praziquantel at this time is the calcium ion channels on schistosomes.

More than 250 million individuals worldwide are afflicted by schistosomiasis, a serious neglected tropical disease. Praziquantel (PZQ), an anthelmintic medication, has been used to treat schistosomiasis for more than a generation. PZQ is the preferred medication for treating schistosomiasis; it is effective against all major forms of the disease, while it is less effective against young parasites than it is against mature ones. PZQ, a derivative of the pyrazino-isoquinoline, is not thought to be harmful and often has minimal or only minor adverse effects.

In Sub-Saharan Africa, where schistosomiasis is widespread, mass drug administration has increased the emergence of diminished PZQ efficacy, portending the selection of drug-resistant variants of these organisms. For example, the synthesis of drug analogues, the rational design of pharmacophores, and the identification of novel compounds *via* extensive screening programs are all gaining interest in the synthesis of enhanced derivatives of PZQ. In this article, reports on the metabolism and mode of action of PZQ and its derivatives against schistosomes from the 1970s to the present are reviewed. Since more than 40 years ago, PZQ1 has been the medicine of choice for treating and preventing the spread of schistosomiasis due to its effectiveness, safety, affordability, and lack of alternatives. PZQ1, however, has limitations, such as its ineffectiveness against young schistosomes.

Furthermore, relying solely on one medication to treat a condition as prevalent in society as schistosomiasis runs the risk of promoting the growth and spread of drug resistance, especially given how frequently diminished susceptibility has been observed in both the field and the lab. There is an urgent need for novel therapies, such as PZQ1 resistance detection techniques and newer drugs with distinct modes of action from those of PZQ1. Several PZQ1 derivatives have new structures, yet the majority of them are effective enough to warrant further study in clinical trials. The mechanism of PZQ1's action and its metabolism must also be understood because knowing this information will make it easier to identify new targets and/or boost the effectiveness of this important and unique drug.

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