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EFFECT OF PRAZIQUANTEL ON THE TEGUMENT OF
SCHISTOSOMA JAPONICUM AND SCHISTOSOMA MEKONGI

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Summary

Adult S. japonicum, S. mekongi and S. mansoni were incubated in vitro in MEM medium containing praziquantel at doses of 0.1, 1 to 10 µg/ml. At 5,15,30,45 and 60 min. the survival rate of parasites were estimated by observing the body's contractility and movement, and the changes of tegument surface and ultrastructure were studied by SEM and TEM. In term of species, S. mekongi is generally more resistant to the drug than S. japonicum; and females are more resistant than males of the same species. By contrast, at these doses, the majority of both sexes of adult S. mansoni are still viable at the end of the experiments. When the parasites' surfaces were examined by SEM, the severity of damage ranges from S. japonicum, S. mekongi to S. mansoni. The males show more damage than the females of the same species, and the anterior part tend to be damaged more than the posterior part. The morphological expression of surface damages are composed of surface blebbing, followed by swelling of blebs and some ridges, and later disruption of these structures. Finally, the lesion and erosion of the surface are observed followed by desquamation in some area, particularly at longer time and higher doses. Interestingly, some major surface structure such as sensory papillae, and spines still remain unaffected at all doses. . When examined by TEM, the sequence of morphological changes are generally similar at all doses ; however, at higher dose the changes are fast and their sequence can be

easily observed. The earliest sign of changes is the depolymerization of the microtrabecular network in the basal and the apical zones which result in the formation of non-membrane bound vacuoles of various sizes. Subsequently, the tips of ridges that become vacuolated are enlarged which could be equivalent to blebs observed in the SEM, and that some are disrupted which appear as lesion spots in SEM. Finally, the microtrabecular network in the middle layers also breaks down, and various-sized vacuoles form throughout the tegument. Finally, tegument is invariably disrupted and detached from the parasites body. At higher dose, the myofilaments of most muscle cells are also depolymerized. From this sequence of morphological changes, it is concluded that the primary effect of praziquantel is on the microtrabecular network : those in the basal and apical zones are most liable to depolymerization by this drug. By contrast, other important organelles of the tegument, ie., mitochondria and microtubules are relatively unaffected by praziquantel, and so are the 10 nm filaments in S. mekongi. It is possible that praziquantel causes the depolymerization of microtrabecular network in the tegument and myofilaments in the muscle cells through its induction of Ca^{2+} influx into the parasites and the detail mechanism was discussed.