

DEXAMETHASONE DOES NOT REDUCE THE WORM BURDEN IN MICE INFECTED WITH *in vivo* OBTAINED SCHISTOSOMULES OF *Schistosoma mansoni*

Marco V. HERMETO, Alan L. MELO, Rosilene S. BICALHO, Antonio P. VARGAS,
Fábio J. O. FAVARETTO & Leógenes H. PEREIRA

The cercaria of *Schistosoma mansoni* must penetrate the skin of the vertebrate host to undergo many structural and physiological changes, originating a new organism, the schistosomule. This period is critical not only regarding to the changes themselves, as well as facing the host defenses⁷.

It is assumed that the effect of glucocorticoid therapy on the *S. mansoni* experimental infection, reducing the worm burden and retarding the parasite development, is patent when these drugs are given during the week of infection, when the parasite is passing through by this critical adaptation period^{1,2}.

In a previous *in vivo* experimental study, a worm burden reduction was shown in mice infected with *S. mansoni* cercariae and treated with dexamethasone in the first week of infection. This reduction is an early process, presumably affecting the skin and/or the lung phases, hence the numbers of lung schistosomules were reduced from the second day of infection until the eighth day⁵. Possibly the glucocorticoid could interfere with the early processes of larval adaptation to the vertebrate host, as cercaria-schistosomule transformation. Thus, in the present report, aiming to confirm this hypothesis, schistosomules obtained *in vivo* from the peritoneal cavity were used to infect mice, thus eliminating the cercaria-schistosomule transformation phase.

Ten albino male mice ($20 \pm 2g$) received an intraperitoneal inoculation with about 1,500 cercariae of *S. mansoni* (LE strain) shed by laboratory-reared *Biomphalaria glabrata*. These mice were sacrificed 4 hours after inoculation, and about 30% of the schistosomules were recovered as previously described⁶. These larvae were appropriately washed with saline and inoculated into 30 male naive mice (about 100 organisms/animal), by subcutaneous route. These animals were treat-

ed with dexamethasone (50 mg/Kg, subcutaneously) 1 hour before infection. Treatment with the same dose was also carried out from the second to the eighth day of infection. An untreated group of 20 mice infected with schistosomules served as control. Perfusion of the portal system for worm counts was performed six weeks after infection. The experiment was repeated twice with 10 mice/groups.

As can be seen in table 1 (results obtained from 3 different experiments), no statistical differences (Students't test) were verified in the worm burden of mice infected with peritoneal-obtained schistosomules and treated with dexamethasone, in relation to the untreated groups. The same was also observed in the *S. mansoni* sex distribution in treated and control mice.

TABLE 1

Mean number and standard deviation of worms recovered from the portal system of mice infected with schistosomules of *Schistosoma mansoni* and treated with dexamethasone.

Experiment	group	mean number of worms recovered		
		male	female	total
I	treated(30)	14.4 \pm 3.7	13.1 \pm 4.4	27.2 \pm 4.1
	control(20)	13.0 \pm 3.2	13.9 \pm 2.8	27.5 \pm 4.3
II	treated(10)	16.4 \pm 3.2	15.9 \pm 2.9	32.3 \pm 5.3
	control(10)	15.7 \pm 2.6	16.8 \pm 1.7	32.5 \pm 4.2
III	treated(10)	16.2 \pm 2.2	13.6 \pm 1.9	29.8 \pm 2.8
	control(10)	14.3 \pm 2.8	15.0 \pm 3.0	29.3 \pm 4.5

Our results suggest that dexamethasone can act during the critical phase of *S. mansoni* larvae adaptation to the vertebrate host. This possibility

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Grupo Interdepartamental de Estudos sobre Esquistossomose, Instituto de Ciências Biológicas da Universidade Federal de Minas Gerais. CEP 31.270-901 Belo Horizonte, MG, Brasil.

is attractive, as some results obtained with the peritoneal cavity model indicate an evident slow-down effect of dexamethasone during the cercaria-schistosomule transformation^{3,4}. Further studies are in progress, attempting to clear up this troublesome effect of the corticoids.

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