

Furazolidone treatment of cutaneous leishmaniasis

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BERMAN & LEE (1983) reported a high antileishmanial activity for furazolidone against amastigotes in human macrophages and suggested a clinical trial with this drug. *In vitro* it was six times more effective than nifurtimox, a drug we have investigated in human leishmaniasis (MARSDEN *et al.*, 1979; GUERRA *et al.*, 1981). At a time when glucantime was not available furazolidone was given to eight patients with

done is a very different situation to this small clinical study. To date it has not been possible to do *in vitro* tests with *L. braziliensis braziliensis* since it does not adapt to the macrophage system. Furazolidone is poorly absorbed and only about 5% of an oral dose appears in the urine (M. S. Wolfe, personal communication). Probably this is a factor accounting for our poor results.

Table—Results of Furazolidone treatment

Patient number	Before treatment		Immuno-fluorescent antibody titre	After treatment	
	Leishmanin skin test	Amastigotes detected		Amastigotes detected	Clinical result*
1	Neg	+	1:40	+	A
2	+	+	1:80	+	A
3	+	+	1:80	0	A
4	Neg	+	0	+	A
5	+	+	1:80	—	A
6	+	+	1:40	—	A
7	+	+	1:320	—	H
8	+	—	0	—	A

*1 month after cessation of treatment A—active
H—healed

cutaneous leishmaniasis of one to six months duration in an area in which *Leishmania braziliensis braziliensis* is the dominant parasite infecting man. A daily divided oral dose totalling 8 mg/kg body-weight was given for 10 days and this course repeated within a month. After a further month if there was no improvement patients received glucantime which was then available. The results are set out in the table. All patients had histology compatible with leishmaniasis and amastigotes were observed in most patients. The leishmanin skin test and fluorescent antibodies to *Leishmania* were positive in most patients. Seven patients failed to respond to furazolidone and parasites were seen in three of four biopsies after treatment. The one patient who healed after the drug had a significant fall in the fluorescent antibody titre seven months later (1:40). This could be the result of furazolidone therapy or simply the natural evolution of the disease, since spontaneous cure is common even with *L. braziliensis braziliensis* infections (MARSDEN *et al.*, 1984).

Furazolidone is widely used in the USA for the treatment of giardiasis, especially in children (WEBSTER, 1960; CRAFT *et al.*, 1981). From our results it does not benefit patients with cutaneous leishmaniasis in the same way as does glucantime (LLANOS-CUENTAS *et al.*, in press). These seven patients who failed to heal responded to glucantime therapy (20mg Sb^{III}/kg body-weight for 30 days).

Evidently *L. tropica* tested *in vitro* against furazoli-

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