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Stage-specific homocytotropic antibody response of *Mastomys natalensis* to *Dipetalonema viteae* infection[1]

Short communication

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Attempt was made in the present study to investigate whether filaria-specific homocytotropic antibody (HcAb) level of host was uniform throughout or related to prevalence of specific life-stage of the parasite, using *Dipetalonema viteae* in *Mastomys natalensis* as a working model and assaying antibody by passive cutaneous anaphylaxis (PCA).

Male mastomys (6 weeks old, GRA Giessen strain) were infected by inoculating 50 infective larvae (L_3) of *D. viteae* isolated from infected ticks, *Ornithodoros moubata* (Worms et al., 1961). Animals were also infected by implantation of adult male and female worms (5–8 numbers) and microfilariae (0.2 million) subcutaneously. Soluble somatic antigen was prepared separately from adult male and female worms, L_3 and microfilariae (mf) by the method of Singh et al. (1985). Protein content of the preparations was estimated by the technique of Lowry et al. (1951).

Microfilariaemia/5 mm^3 of tail blood of animals was assessed at weekly or fortnightly interval starting from day 60 post L_3 exposure or days 4 and 7 postimplantation. Blood for serum was collected from retro-orbital plexus of animal on days 20 and 30 of infection and thereafter at monthly interval along with matched uninfected controls.

PCA test was performed in homologous, healthy recipients (12 weeks old) following the method of Soulsby et al. (1977). After initial evaluation, a quantum of 250 μg of antigen protein was found optimal for positive PCA reaction. PCA test was also performed after heating sera samples at 56° C for 1 h to exclude the possibility of interference of IgG type of reagin in the test.

The study showed the presence of HcAb against all the three important life stages e.g. mf, L_3 and adults of *D. viteae* (Fig. 1). L_3-specific HcAb did not last longer possibly due to short persistence of this life-stage in the host. The peak level was detected within 40 days and total disappearance occurred before day 150 post exposure (p.e.). HcAb response of host to adult stage was detected on day 20 and persisted for longer period i.e. up to day 240 p.e. The persistence of antibody to adult parasites even after their death could possibly be due to cross-reactions with other developing forms. High adult-specific HcAb titre as detected on day 60 coinciding with the appearance of mf in circulation could also be due to enhanced common antigenic stimuli from mf and adults. However, this does not explain why adult-specific antibody disappeared on day 240 p.e. when mf still persisted. The plausible reason could be that major antigenic stimuli (excretory and secretory product) specific to adults slowly disappeared with the death of worms and the quantum of 250 μg of adult antigen in PCA test was insufficient to detect cross reacting mf antibody during that period. Almost similar pattern of

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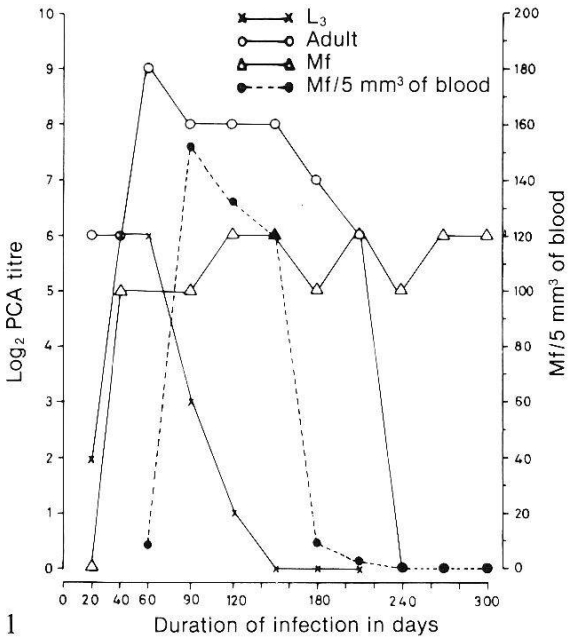

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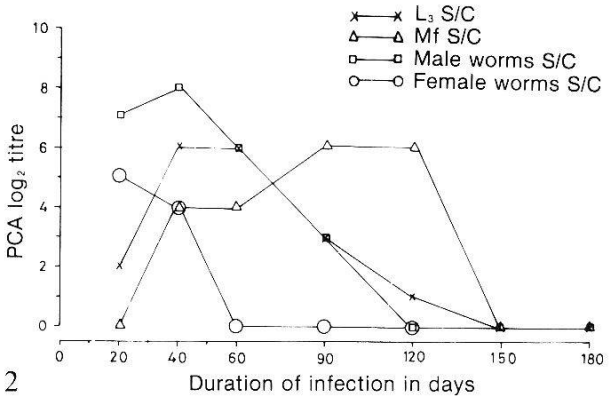

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Fig. 1. Stage-specific homocytotropic antibody response of *Mastomys* during the course of *D. viteae* infection. Fig. 2. Homocytotropic antibody of *Mastomys* to implanted stage-specific infection.

HcAb response with adult antigen was observed earlier by Benjamin and Soulsby (1976) in mastomys infected with *Brugia pahangi.* In spite of very long persistence of mf, anti-mf titre never reached to a very high level and low titre persisted up to the end of present observation period (i.e. 300 days) when amicrofilaraemia was set in. Apparently mf appear to be more antigenic and their sensitization effects last longer. Wederhof and Wenk (1975) reported stage-specific protection using live mf, though it was not clear from their study whether IgE played any role in that. However, Haque et al. (1981) reported IgE-mediated in vitro killing of *D. viteae* mf by macrophage-rich peritoneal exudate cells. Detection of mf-specific PCA titre as early as day 40 p.e. could be due to antigenic stimulus from uterine secretions of gravid female worms. Nevertheless, some cross-reactivity with other life-stages can not be ruled out. The nature of stage-specific HcAb response as observed with adult male, female or mf-implanted infection was by and large similar to L_3-induced infection (Fig. 2). Apparently male worms were found to be more potent in inducing HcAb response than their female counterparts which might be due to earlier death of the latter sex (thus lacking E-S products).

Apart from eliciting hypersensitivity reaction, the role of filaria-specific IgE in acquired resistance against filarial infection remains unanswered. Though there are certain reports regarding its role in functional immunity to helminth parasites (Ogilvie et al., 1966; Sadun and Gore, 1970; Gusmao et al., 1981) its involvement in protective immunity in filariasis is yet to be conclusively proved. The present study shows that stage-specific HcAb develop during filarial infection but their association in eliminating any particular life-stage or infection as a whole is not clear. The homocytotropic antibody detected in the present study was heat labile.

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