filariae. It is further characterised by the fact that following the death and disintegration of adult worms and decline in microfilaraemia, a sharp fall occurs in the level of antibody titre also. A definite correlation between the worm burden, circulating microfilariae and complement fixing antibody titre has also been reported in cotton rats and *Mastomys natalensis* infected with *Litomosoides carinii*[8,9]. Similarly, Pacheco[7] has also reported a high level of antibody titre in dirofilariasis of dogs during the period of active migration of larvae.

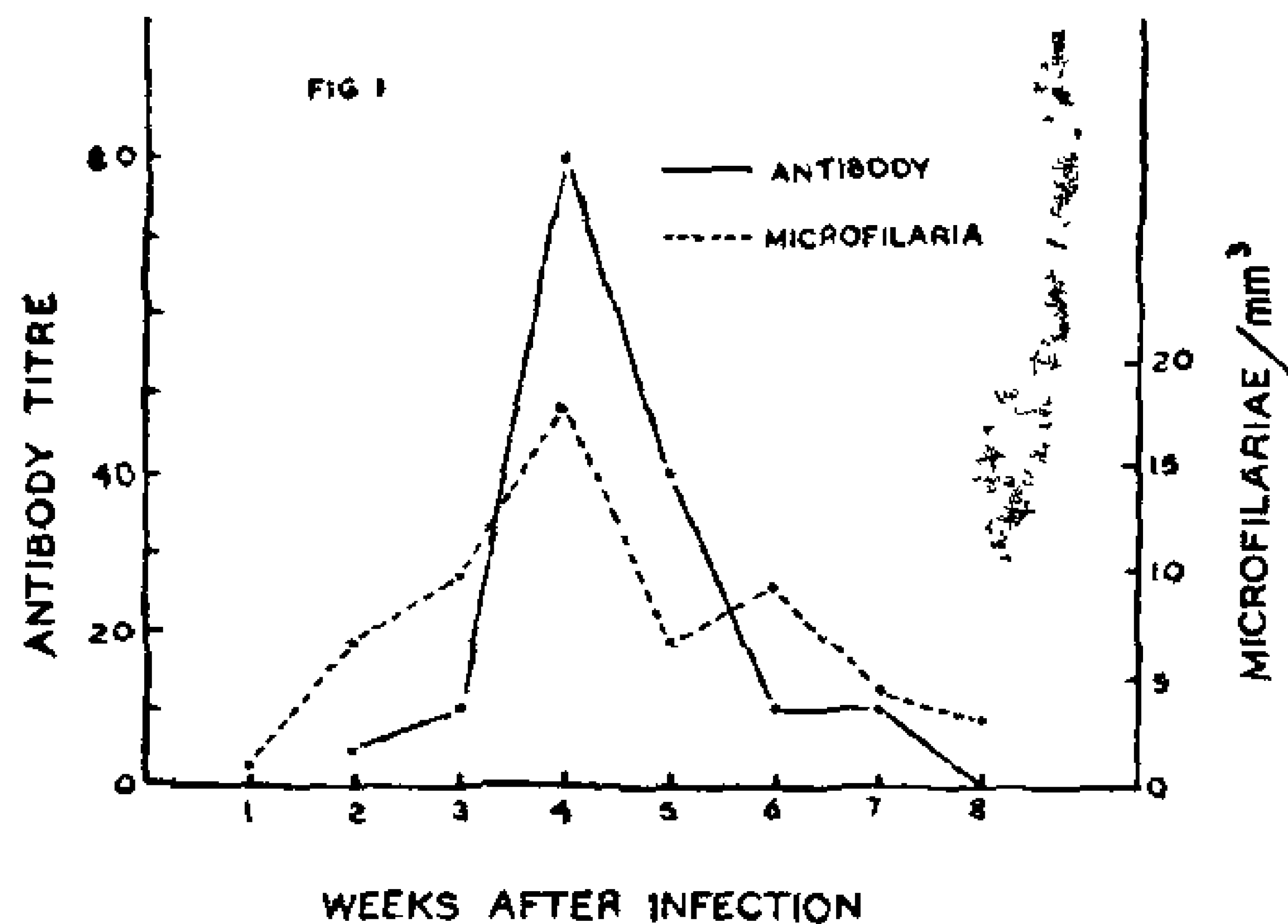


FIG. 1

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Department of Zoology, Section of Parasitology, Aligarh Muslim University, Aligarh (U.P.), India,
ABDUL BAQUI.
JAMIL A. ANSARI.

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## FAILURE OF IMMUNO-SURVEILLANCE AND RECURRENCES OF HERPES LABIALIS

*In vitro* lymphocyte stimulation to herpes simplex virus (HSV) antigens has been demonstrated in humans[1-3].

The *in vitro* specific stimulation of peripheral blood lymphoid cells in humans by antigen (HSV type 1), using $H^3$-tdr uptake method[4] and microculture technique is reported in this communication. The details of microculture technique have been described earlier[5]. Cultures were set up in quadruple using hepes-buffered Eagle's medium, supplemented with 5% foetal calf serum and 2-mercaptoethanol (60 $\mu$M/ml) in 0·1 ml of the medium. Heat inactivated cell (BHK21)—associated antigen of HSV1 (N102) acted as the source of viral antigen. A dose of $10^6$ pfu ($10^{-2}$ dilution) gave optimum stimulation response; this dose was therefore used for the study of antigen-specific lymphocyte stimulation in microcultures. BHK21 cell extract ($10^{-2}$ dilution) acted as control in all the stimulation experiments. Cultures were harvested using the skatron multiple cell culture harvester. Venous blood samples of patients suffering from recurrent cold sores were collected during acute ($\pm$ 1 day) stage (2) and during remissions (6). Venous blood samples of HSV1 seropositive normals (6) and HSV1 seronegative normals (6) acted as controls.

Significant HSV1 antigen-specific stimulation could be obtained with patient's peripheral blood samples, collected during remissions and also in normal seropositive controls. Venous blood samples of seronegatives did not respond to HSV1 antigens. Moreover, HSV1 antigen-specific stimulation responses with samples collected during acute stage (at the time of development of recurrent cold sores) were poor (Fig. 1). No stimulation with BHK21 cell extract ($10^{-2}$ dilution) was obtained in any of the lymphocyte stimulation experiments.

Failure to detect a HSV1 antigen-specific response during acute stage could be explained on the basis of (*a*) development of immuno-suppression following recurrence of the lesion or (*b*) by induction of suppressor cells or (*c*) by peculiarity of lymphoid cell traffic. A role for immuno-suppression is ruled out because lymphocyte stimulation responses to PHA during the acute stage were nearly as good as those observed later (Fig. 1). Induction of suppressor cells is a possibility because these patients were carrying the virus for 6–15 years. Another explanation could be the infection of the macrophages and/or the lymphocytes, but a point, against this is that PHA responses are not affected. It can, however, be argued that PHA proliferative response may not require accessory cells, while for antigen-specific stimulation to HSV1 they are needed (Fig. 2).

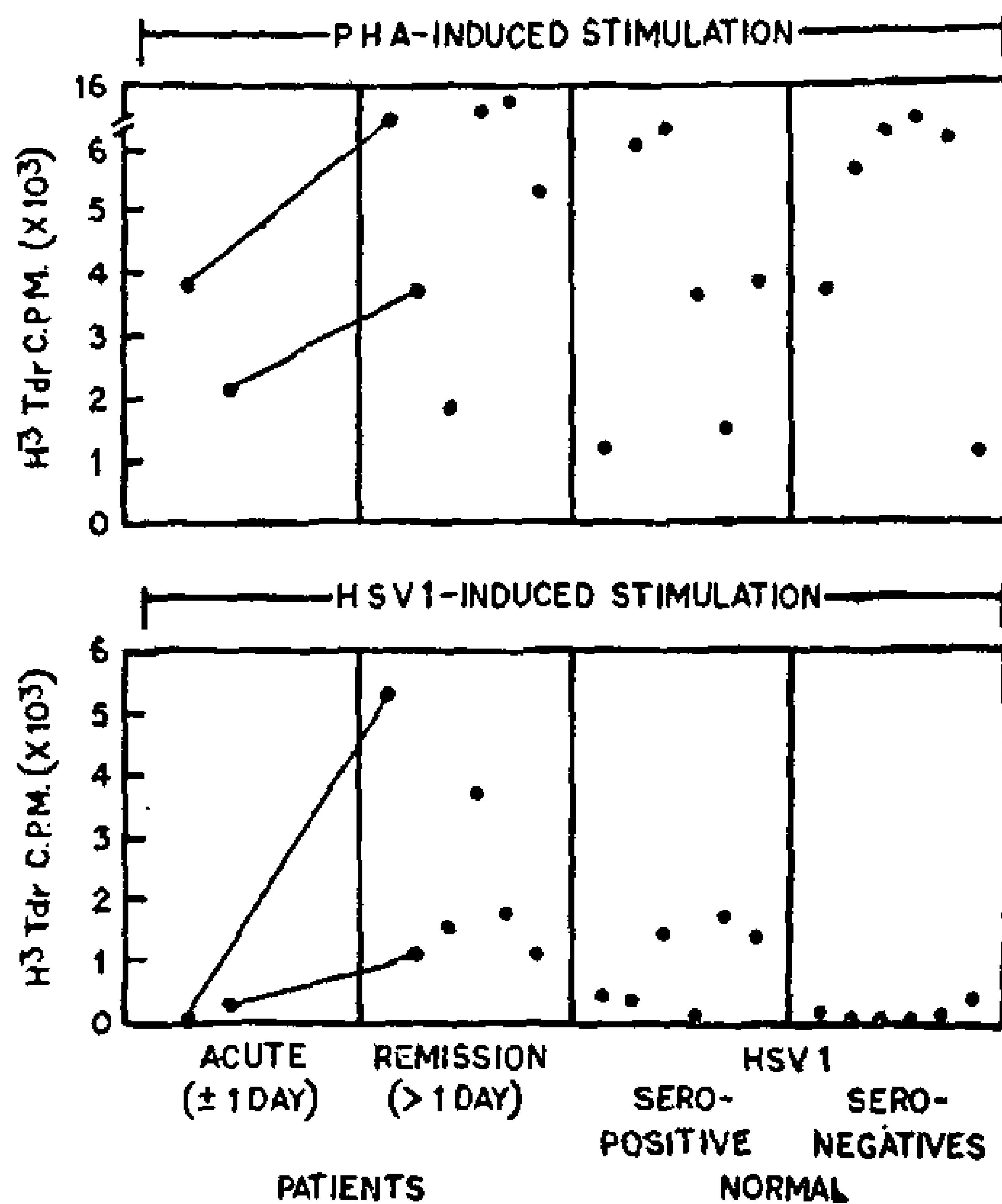


FIG. 1. Shows the result of *in vitro* lymphocyte stimulation to HSV1 antigens and phytohaemagglutinin (PHA) in humans.

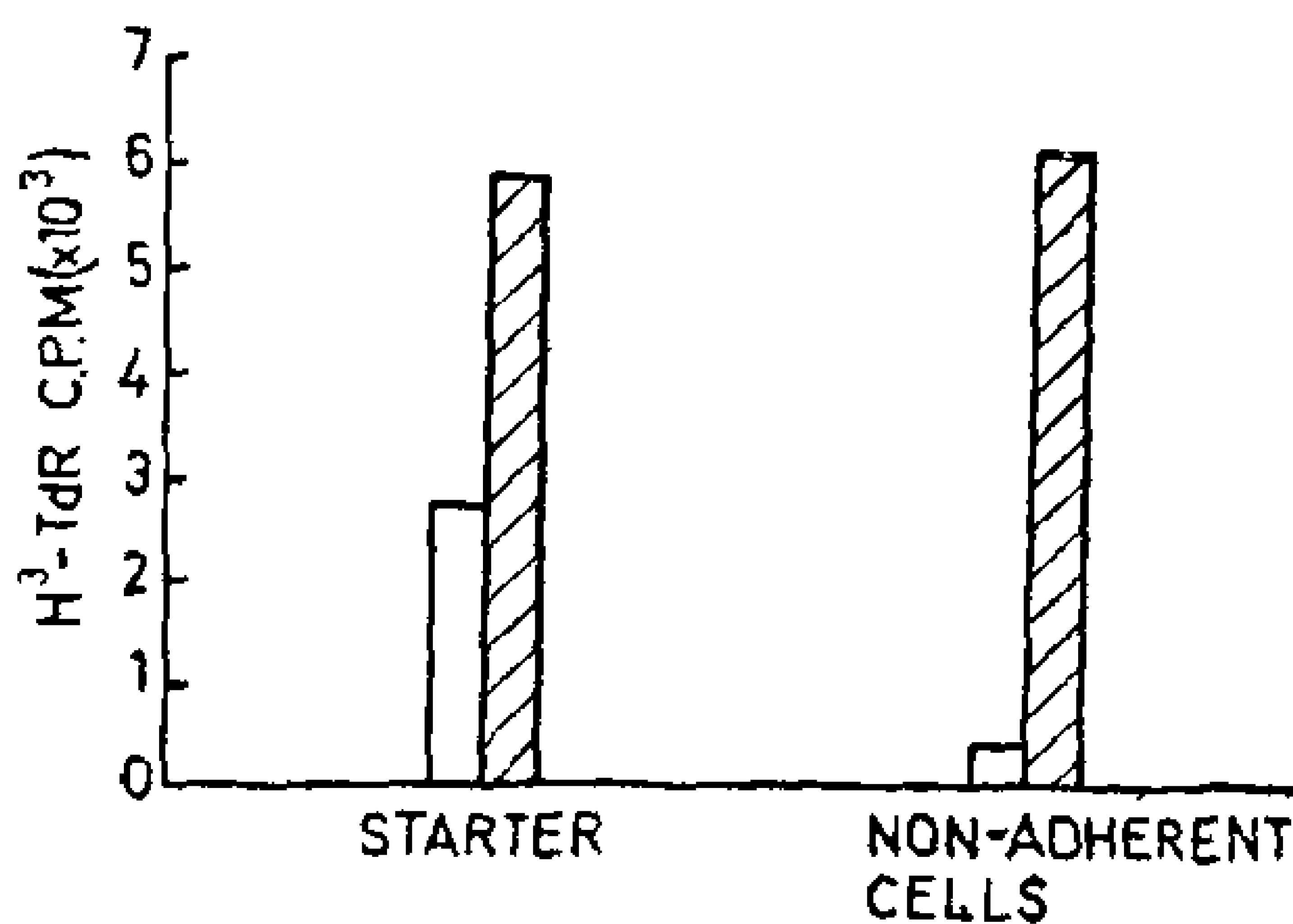


FIG. 2. Shows the result of lymphocyte stimulation of non-adherent cells. ☐ antigen (HSV1)—specific stimulation ; response with PHA.

These results can also be explained simply on the basis of peculiarities of lymphoid cell traffic. An antigen-specific response with peripheral blood cells during the acute stage might not have been obtained because the antigen-reactive cells are 'homing' to distant sites (lymph nodes, spleen, etc.) leading to a failure of immuno-surveillance mechanism.

It was concluded that recurrences of Herpes labialis occurred at the time of minimum lymphocyte reactivity to HSV1 antigens in blood, possibly subsequent to a failure of immuno-surveillance mechanism.

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Department of Experimental Pathology,
The Medical School,
Vincent Drive,
Birmingham, England B15 2TJ.
and
Department of Oral Pathology,
The Dental School,
Birmingham, England,
*November* 15, 1978.

A. K. KAPOOR.*
M. BASU.

* Present address : Department of Pathology, King George's Medical College, Lucknow, U.P., India.

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## MORPHOGENETIC ABNORMALITIES CAUSED BY TREATMENT OF EGGS OF *DYSDERCUS CINGULATUS* WITH JH ANALOGUES

THERE are a number of reports that exogenous juvenile hormone (JH) and JH analogues block embryonic development in insects when applied to eggs prior to a critical stage[1,2]. However, very few attempts have been made so far to study the internal details of these abnormal embryos. The present study summarises the changes in the external and internal organization of the embryos after treatment of the eggs of a pyrrhocorid bug, *Dysdercus cingulatus* with JH analogues.

Eggs after oviposition, after germ band formation and after blastokinesis were used for the study. Farnesyl methyl ether (FME) and ZR 777 (Kinoprene) were dissolved in acetone. One $\mu$l of the solution containing different doses of ZR 777 (0·25, 0·125, 0·06, 0·03, 0·015, 0·0075, 0·004, 0·0009 and 0·0001 $\mu$g per egg) and FME (2·0, 1·5, 1·0, 0·5 and 0·25 $\mu$g per egg) were topically applied using a Hamilton microliter syringe. Controls were treated with 1 $\mu$l of acetone. Twenty-five eggs per group were treated at a time. The experiments were repeated six times. The results were pooled and summarised. For whole mounts, embryos were fixed in Carnoy's fluid and