extracts may be due to the strong antiestrogenic action of the extracts[6] which probably change the growth and secretory functions of the cell[13], cell permeability[14] and also the uterine milieu required for the implantation of an egg.

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COMPLEMENT FIXATION TEST IN EXPERIMENTAL SETARIASIS

Setaria cervi, a cosmopolitan filariid worm of buffaloes, has a high rate of incidence among cattle and buffalo population in India. The parasite does not usually cause any significant pathology in the natural hosts. However, evidence is available which indicates that the infective stages of the parasite under strange circumstances may enter the central nervous system of buffaloes, deers, sheep and goats and develop to partial maturity causing severe pathological condition known as 'cerebro-spinal nematodiasis'[1-3].

The present work deals with the serological diagnosis of experimental setariasis by means of Complement Fixation Test (CFT). Adult worms, collected from freshly slaughtered buffaloes, were implanted intraperitoneally into 36 laboratory bred white rats[4]. Each rat received 5 adult worms. CFT was performed according to Kolmer[5]. Before carrying out main CFT, hemolysin, complement and antigen were titrated. Two units of hemolysin contained in 0·5 ml were used in main CFT. Normal guinea pig serum was used as a source of complement. Two full units of complement in 1 ml were employed. 1% alcoholic extract of the powdered worms, prepared according to the methods of Minning and McFadzean[6], Pacheco[7], was employed as an antigen. Antigen was used in a dilution of 1 : 80 in 0·5 ml amount. Weekly blood, from 12 microfilaria-positive rats was collected intracardially, and the serum was separated. Pooled antisera diluted from 1 : 5 to 1 : 640 were used in 0·5 ml. A positive reaction was taken as the one which gave 50% hemolysis, and hemolysis above 50% was taken as negative[7]. In the test, immune serum control, hemolytic control, normal serum control and antigen control were also included. A weekly record of microfilaraemia in all these rats was also maintained. Another 24 infected rats were sacrificed at weekly intervals to note the condition and survival time of adult worms in the peritoneal cavity of white rats.

Microfilariae appeared in the peripheral blood circulation of white rats after a latent period of 8 ± 2 days with a maximum microfilarial density during 3rd and 4th weeks of initial infection. Adult worms survived in the peritoneal cavity for about 4-5 weeks, thereafter, they started disintegrating. Dead and exhausted worms were found to be embedded in the peritoneal wall and intestinal mesentries. Complement fixing antibody was detected in infected rat sera in 2nd week of initial infection with a titre of 1 : 5. The antibody titre rapidly increased and reached its maximum by 4th week with a titre of 1 : 80 which was found to be coinciding with the peak microfilarial density (Fig. 1). No antibody titre was detected during 8th week.

The above observations indicate that there is a definite correlation between the level of antibody titre, level of microfilaraemia and survival of adult worms. Higher levels of antibody titre during 4th and 5th weeks followed by a sharp fall during later phase of infection indicate that maximum antigenic stimulus is generated by live adult worms and micro-

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.

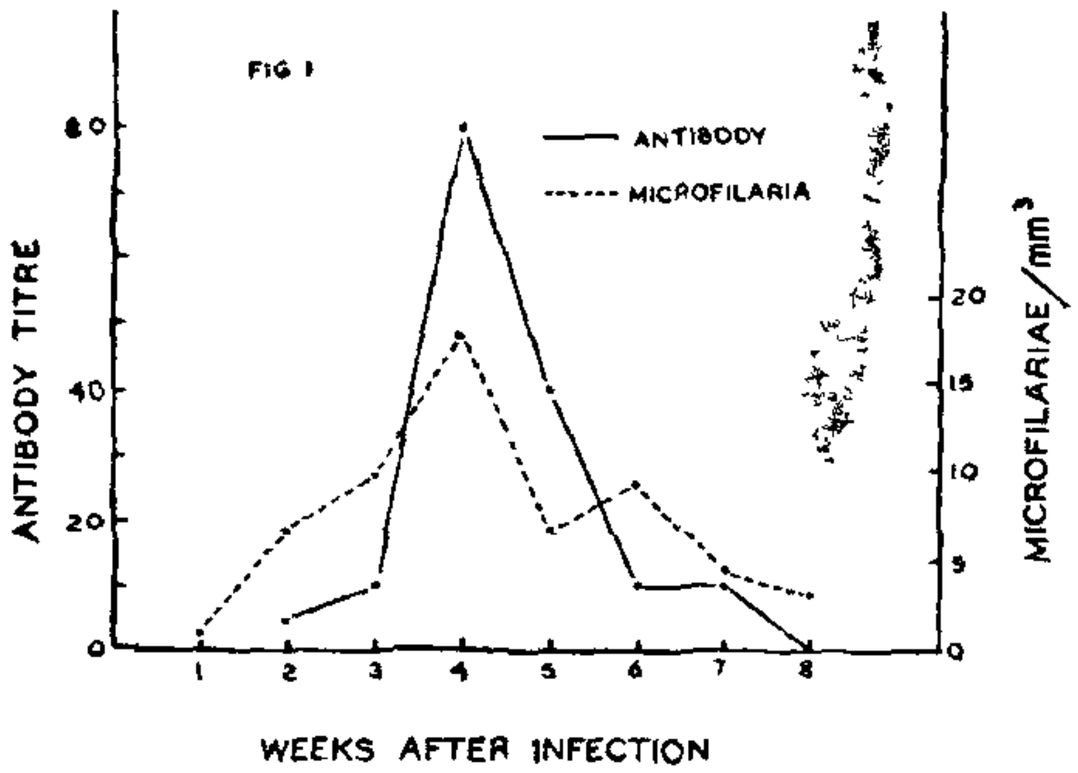

FIG. 1

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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 μM/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).