SUMMARY
Experimental mycetoma like lesions developed in 24-30 days after subcutaneous injection of M. asteroides in guinea pigs. Although delayed hypersensitivity (DH) appeared in 4-5 weeks increased macrophage migration inhibition (MMI) and microbicidal activity (Mic-A) appeared after 7 weeks. When the lesions healed high cell mediated immunity (CMI) was present. CMI was transferred from guinea pigs possessing high CMI as shown by increased MMI and Mic-A to normal ones by spleen cell transfer. These recipients survived much longer than control animals after an intravenous challenge. Their tissues also did not show any growth of M. asteroides. Therefore it is felt that CMI plays an important role in protection against M. asteroides infection.

Guinea pigs were immunized with ribonucleic acid protein fraction (N-PRNA) from M. asteroides. They developed high CMI as shown by increased Mic-A and MMI which appeared in 14 and 25 days respectively. This persisted at least up to 70 days. However, this fraction did not induce any DH in immunized animals. The CMI induced by N-PRNA was protective against intravenous challenge with M. asteroides. The protection was seen in both actively and passively immunized guinea pigs. Guinea pigs were passively immunized by transfer of spleen cell suspension from donor guinea pigs actively immunized with N-PRNA and possessing high CMI. The protection was reflected in the prolonged survival, fewer lesions and less number of organisms in the tissues of the challenged
animals. When immunized animals were challenged subcutaneously they developed fewer sinuses which also healed very rapidly.

When guineapigs immunized with N-PRNA were challenged intraperitoneally with N.a.steroides, 25-40 days postimmunization, a period at which maximum CMI was seen, their macrophages exhibited specific invivo killing of the organisms. This was seen by the decrease in the intracellular viable count of N.a.steroides at 72 h. This invivo killing of N.a.steroides by these macrophages persisted up to 60 days. Macrophages from guineapigs immunized passively by spleen cells from donor actively immunized with N-PRNA also showed specific invivo killing of N.a.steroides. This showed that CMI was responsible for the effective killing of N.a.steroides by the macrophages. It was further supported by the observation that spleen cells from donors actively immunized with N.a.steroides when passively transferred to normal recipients conferred protection in these animals. Actively or passively immunized guineapigs were treated with antimacrophage serum and then challenged intracardially with N.a.steroides.

These guineapigs died in 5 days with large number of N.a.steroides in lungs, heart, kidneys, liver and spleen. There was no protection seen in these animals. The treatment with anti abolished the protection afforded by CMI, indicating that macrophages are essential for protection against experimental N.a.steroides infection.