neuraminidase treatment is known to enhance the adhesion reaction[13].

Thus it is summarized that the microfilariae of *L. carinii* but not of *D. viteae* possess a large number of WGA binding sites. The binding of other lectins is poor in comparison and appears to be predominantly non-specific. N-acetyl glucosamine residues are primarily contributed by chitin which by the enzymatic action appear to be the prominent polysaccharide of the sheath of *L. carinii* microfilariae. Trypsin may be used to exsheath this species of microfilariae *in vitro*. Treatment of the microfilariae with the enzyme exposes a large number of new lectin binding sites which increases the vulnerability of the parasite to cellular adhesion.

29 April 1987; Revised 5 January 1988

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NEWS

"WHO COLLABORATING CENTRE ON DRUG INFORMATION" AT CDRI LUCKNOW

The World Health Organization has established a "WHO Collaborating Centre on Drug Information" at the Central Drug Research Institute, Lucknow. The National Information Centre for Drugs and Pharmaceuticals (NICDAP), a sectoral discipline-oriented information centre under the NISSAT Plan of the DSIR, Ministry of Science and Technology, Govt. of India, operating at CDRI since October 1977, has been chosen for this purpose. This collaborating centre is the first of its kind established by WHO on drug information and the collaboration with WHO has become effective from February 1988 for a period of four years. The centre will act as a focal point for information activities in the area of drugs and pharmaceuticals for the 11 member-countries of the South East Asian region which include, besides India, Bangladesh, Bhutan, Burma, Indonesia, Democratic Republic of Korea, Maldives, Mongolia, Nepal, Sri Lanka and Thailand.

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ACTIVATION OF SERUM COMPLEMENT BY ORGANOCHLORINE INSECTICIDES, DDT AND ENDOSULPHAN

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ABSTRACT

A baseline information is provided on the mode of interaction of DDT and endosulphan with serum complement. Both the insecticides activated C_3 (the third component of the complement system) to C_3b when incubated with normal human serum, C_3 activation occurred through alternative pathway as evidenced by the insignificant alteration of total haemolytic complement (CH_{50}) *in vitro*, maintenance of activation despite the removal of Ca^{2+} by ethylene glycol tetra acetic acid and blockade of activation due to the chelation of both Ca^{2+} and Mg^{2+} by ethylene diamine tetraacetic acid.

INTRODUCTION

RECENT developments on complement research have placed this group of serum proteins at the key position of immunopathology and immunoregulation[1-3]. Complement activation has been detected in association with many pathological conditions including several allergic manifestations[4-6]. In a variety of such situations, the etiology of complement activation remains unknown, especially when the activation occurs through antibody independent alternative pathway. At this juncture, it appears appropriate to study the interaction of environmental chemicals, particularly, the persistent ones with the complement system. This communication reports our findings on the mode of interaction of two chlorinated insecticides, DDT and endosulphan with complement system.

MATERIALS AND METHODS

DDT (1,1-bis-(*p*-chlorophenyl) 2,2,2-trichloroethane, technical grade and endosulphan [α,β-1,2,3,4,7,7-hexachlorobycyclo (2,2,1)-heptene (2)-bis-hydroxymethylene(5,6) sulphite) Thiodan, Hoechst] were procured from the Hindustan Insecticide Limited, India. Antisera to human C_3 were purchased from Immunodiagnostics, India.

Serum samples from normal human subjects were incubated with varying concentrations of DDT and endosulphan for 30 min at 37°C.

Activation of C_3 was monitored by crossed immunoelectrophoresis[7]. During the first dimension run, 5 μl of the insecticide treated and untreated serum samples were applied in the well, punched on a 1% agarose gel, coated on a microscopic slide (3 ml). Electrophoresis was run for 2 h at 200 V. Bromophenol blue was applied along with the sample (or in a parallel well) to detect 5 cm run. After the first dimension run, the gel was cut into two longitudinal slabs from the middle and the individual slab was transferred at the edge of another plate (5 × 7.5 cm) horizontally. The plate was coated with 5 ml of 1% agarose containing 2% anti C_3 and the second dimension run was performed at 100 V for 6 h in a perpendicular dimension. At the end of the electrophoresis, the gels were washed, dried and stained by coomassie brilliant blue. EDTA of 0.01 M concentration was added in gel and tank buffer to prevent C_3 activation during electrophoresis.

In separate experiments, serum samples were incubated for 30 min at 37°C with 10 mM ethylene glycol tetra acetic acid (EGTA) or ethylene diamine tetra acetic acid (EDTA) respectively to chelate Ca^{2+} or (Ca^{2+} and Mg^{2+}) both prior to insecticide treatment. Parallel controls remained untreated with DDT or endosulphan. C_3 activation was followed by crossed immunoelectrophoresis.

Total haemolytic complement in the treated and the untreated sera was determined[8] by estimating CH_{50} levels.

RESULTS

Effect of DDT and endosulphan on C_3 activation

Figures 1 and 2 represent the crossed immunoelectrophoretic pattern of insecticide treated and untreated normal human sera (NHS). Appearance of a higher C_3b peak served as the index of C_3 activation. As is evident from the figures, both the insecticides activated C_3 to C_3b at all the concentra-