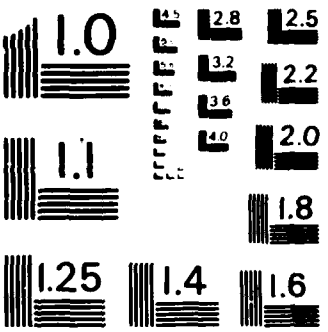
AD-A170 910 IMMUNE RESPONSES IN PARASITIC DISEASES(U) KANSAS UNIV MEDICAL CENTER KANSAS CITY D J STECHSCHULTE ET AL. 01 SEP 82 DAMD17-74-C-4136 1/1

UNCLASSIFIED F/G 6/5 NL



PHOTOGRAPH THIS SHEET

AD-A170 910

DTIC ACCESSION NUMBER

LEVEL

INVENTORY

IMMUNE RESPONSES IN PARASITIC DISEASES
ANNUAL REPORT - SEPT. 1982
(July 1974 - June 1975)

DOCUMENT IDENTIFICATION

DISTRIBUTION STATEMENT A
Approved for public release;
Distribution Unlimited

DISTRIBUTION STATEMENT

ACCESSION FOR	
NTIS GRA&I	☑
DTIC TAB	☐
UNANNOUNCED	☐
JUSTIFICATION	
BY	
DISTRIBUTION /	
AVAILABILITY CODES	
DIST	AVAIL AND/OR SPECIAL
A-1	

DISTRIBUTION STAMP

COPY INSPECTED

DTIC
ELECTE
AUG 2 0 1986
S D
D

DATE ACCESSIONED

DATE RETURNED

86 8 20 022

DATE RECEIVED IN DTIC

REGISTERED OR CERTIFIED NO.

PHOTOGRAPH THIS SHEET AND RETURN TO DTIC-DDAC

DTIC FORM 70A DEC 83

DOCUMENT PROCESSING SHEET

PREVIOUS EDITION MAY BE USED UNTIL STOCK IS EXHAUSTED.

AD_____

IMMUNE RESPONSES IN PARASITIC DISEASES

Annual Report

Daniel J. Stechschulte, M.D.

Herbert B. Lindsley, M.D.

September 1982

(July 1974 - June 1975)

Supported by

U. S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, Maryland 21701

Contract No. DAMD 17-74-C-4136

University of Kansas Medical Center
College of Health Sciences and Hospital
Kansas City, KS 66103

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) IMMUNE RESPONSES IN PARASITIC DISEASES		5. TYPE OF REPORT & PERIOD COVERED Annual Report July 1974 - June 1975
		6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) Daniel J. Stechschulte, M.D. Herbert B. Lindsley, M.D.		8. CONTRACT OR GRANT NUMBER(s) DAMD17-74-C-4136
9. PERFORMING ORGANIZATION NAME AND ADDRESS University of Kansas Medical Center College of Health Sciences and Hospital Kansas City, KS 66103		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 62770A.3M162770A802.00.045
11. CONTROLLING OFFICE NAME AND ADDRESS US Army Medical Research and Development Command Fort Detrick Frederick, Maryland 21701		12. REPORT DATE 1 September 1982 13. NUMBER OF PAGES 7
14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)		15. SECURITY CLASS. (of this report) Unclassified 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

None

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Acquired resistance to infection and the development of sterile immunity is dependent upon specific immune responses. The responses include the proliferation of thymic or T and bursal-equivalent or B lymphocytes and their production of lymphocyte mediators and immunoglobulins respectively. Although recent evidence indicates that T and B cell cooperation modulates the total immune response and that both lymphocyte populations produce selected lymphocyte mediators, an investigation of the immune response to infection can still be approached by exploring the cellular and (Con't on reverse side)

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

Item #20 - Continued:

humoral systems independently. Monospecific reagents will be developed for the rat immunoglobulins in order to specifically measure the antibody response to Trypanosoma rhodes ense infection.

AD_____

IMMUNE RESPONSES IN PARASITIC DISEASES

Annual Report

Daniel J. Stechschulte, M.D.

Herbert B. Lindsley, M.D.

September 1982

(July 1974 - June 1975)

Supported by

U. S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, Maryland 21701

Contract No. DAMD 17-74-C-4136

University of Kansas Medical Center
College of Health Sciences and Hospital
Kansas City, KS 66103

Summary

Acquired resistance to infection and the development of sterile immunity is dependent upon specific immune responses. The responses include the proliferation of thymic or T and bursal-equivalent or B lymphocytes and their production of lymphocyte mediators and immunoglobulins respectively. Although recent evidence indicates that T and B cell cooperation modulates the total immune response and that both lymphocyte populations produce selected lymphocyte mediators, an investigation of the immune response to infection can still be approached by exploring the cellular and humoral systems independently. Monospecific reagents will be developed for the rat immunoglobulins in order to specifically measure the antibody response to Trypanosoma rhodesiense infection.

Foreword

The research studies conducted at the University of Kansas Medical Center between July, 1974 and June, 1975 under a contractual arrangement with U.S. Army Research and Development Command were designed to further understand the immunopathogenic mechanism in rats infected with Trypanosoma rhodesiense. In conducting the research described in this report, the investigator(s) adhered to the "Guide for Laboratory Animal Facilities and Care," as promulgated by the Committee on the Guide for Laboratory Animal, Resources, National Academy of Sciences-National Research Council.

Report - July, 1974 - June, 1975

Acquired resistance to infection and the development of sterile immunity is dependent upon specific immune responses. The responses include the proliferation of thymic or T and bursal-equivalent or B lymphocytes and their production of lymphocyte mediators and immunoglobulins respectively. Although recent evidence indicates that T and B cell cooperation modulates the total immune response and that both lymphocyte populations produce selected lymphocyte mediators, an investigation of the immune response to infection can still be approached by exploring the cellular and humoral systems independently.

The host immune response of any species to an infection with African trypanosomiasis is not completely understood. The rat was initially selected as a model in which to study Trypanosoma rhodesiense infections, because of: (1) the availability of monospecific reagents to the recognized rat immunoglobulins and therefore the antibodies within each class; (2) the demonstrated histocompatability of inbred rat strains permitting adoptive cell transfer studies, and (3) the probability that this host-parasite relationship can be studied as a chronic infection, better suited for a detailed kinetic analysis of the immune response. When the trypanosomal antigens are characterized, the humoral immune response to infection will be analyzed by standard in vitro techniques, and these antigens will also be evaluated for their ablity to stimulate lymphocyte proliferation. A notable feature of African trypanosomiasis is the dramatic rise in IgM levels which return to normal with successful

treatment of the infection. Characterization of this IgM response in the rat may provide a useful insight into the effectiveness of IgM antibodies as a mechanism of protection against T. rhodesiense infection. Rats demonstrating resistance to infection presumably have acquired protective antibody and/or a lymphocyte population that specifically recognizes the parasite at a vulnerable stage in its life cycle. The effects of passive transfer of antibody and adoptive transfer of educated lymphocytes into syngeneic rats prior to the initial infection will test this presumption. Protective antibody, defined by its ability to confer a delay in patency, a suppressed parasitemia, or a decrease mortality to rats experience a primary infection, will be defined in terms of immunoglobulin class and in vitro antibody titer. The recent development of techniques such as affinity column chromotography, which allows quantitative recovery of virtually pure populations of T and B lymphocytes, should permit an evaluation in syngeneic rats of the effects of each cell type on subsequent infections.

These studies have demnstrated that the predominate humoral immune response in rhodent T. rhodesiense infection is in the IgM and IgG 1 classes. What role these antibodies play in host defense mechanisms and tissue injury are undetermined.

END

DTIC

9-86