

PLASMODIUM SPECIES RELATED IMMUNE
RESPONSE
IN MALARIA PATIENTS

A Dissertation Project submitted to Nirma University
In Partial fulfilment of requirement for
The Degree of
MASTER OF SCIENCE
IN
BIOCHEMISTRY/MICROBIOLOGY



Submitted by:

ANJALI M. KAIMAL (16MBC001)
DEVANSHI H. VADODARIYA (16MMB023)

Under the Supervision of:

DR. PRANATI SAR
(DST-SERB YOUNG SCIENTIST)

INSTITUTE OF SCIENCE, NIRMA UNIVERSITY
AHMEDABAD-382481
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NIRMA
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NAAC ACCREDITED 'A' GRADE

CERTIFICATE

This is to certify that the thesis entitled “*Plasmodium* species related immune response in Malaria Patients” submitted to the Institute of Science, Nirma University in partial fulfillment of the requirement for the award of the degree of M.Sc. in Biochemistry and Microbiology, is a record research work carried out by **Anjali M. Kainal (16MBC001)** and **Devanshi Vadodariya (16MMB001)** under the guidance of **Dr. Pranati Sar**. No part of the thesis has been submitted for any other degree or diploma.

Prof. Sarat Dalai

(Director I/c)

Director
Institute of Science
Nirma University
Ahmedabad



Dr. Pranati Sar

(Dissertation Guide)

Date:

Place: Ahmedabad

Institute of Science, Nirma University

Sarkhej-Gandhinagar Highway Ahmedabad 382 481, INDIA, Ph +91-02717-241900/01/02/03/04, +91-79-30642753, Fax +91-02717-241916

E-mail director.is@nirmauni.ac.in, Website www.nirmauni.ac.in

DECLARATION

I hereby kindly declare that the thesis entitled "***Plasmodium* species related immune response in malaria patients**" submitted to **Institute of Science, Nirma University, Ahmedabad** in partial fulfillment of the requirement for the award of the degree of M.Sc in **Microbiology** is a record of an original work done under the guidance of **Dr. Pranati Sar.** The results embodied in the thesis have not been submitted to any other University or Institute for the award of any degree or diploma.

Dt: 26th APRIL, 2018

Place: Institute of Science
Nirma University
Sarkhej-Gandhinagar highway
Ahmedabad-382481



Anjali M. Kaimal

Devanshi H. Vadodariya

ABSTRACT

Malaria, one of the major engenders of mortality and morbidity worldwide is caused by *Apicomplexa* parasites. These parasites of *Plasmodium* genus have a multiplex life cycle in the definitive and intermediate host.

It is seen that with the onset of *Plasmodium* infection there occurs the down regulation of MHC II expression which pivotal role in the antigen display to T cells such that it can discriminate self from nonself. MHC class II molecule expression prompted by TLR, thus, not only enables effective antigen display for the activation of adaptive immunity, but also regulates the positive feedback mechanism enhancing TLR-mediated responses. To address this, first we estimated parasitic load by using Real time assay in clinical samples of malaria patients and also distinguished the *Plasmodium* species parasite present in the samples. Then the MHC II gene expression (DRB expression) was examined in correspondence to the parasitic load at RNA level by q-PCR. From which the down-regulation of MHC II expression was observed with the increase in the parasitic load. Along with this the TLR 4 and TLR 9 expression was analyzed which was elevated by regulation of MHC class II. And with respect to the TLR 4 and TLR 9 expression, cytokine profiling was determined by ELISA.

Therefore, in our study we have inferred that the suppression of MHC II expression is *Plasmodium* species specific and TLR mediated immune response is enhanced by MHC class II molecules.