BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
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NAME
Rustom Antia

POSITION TITLE
Professor, Dept of Biology

eRA COMMONS USER NAME
rantia

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
<thead>
<tr>
<th>INSTITUTION and LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indian Institute of Technology, Bombay</td>
<td>M.Sc.</td>
<td>1978-1983</td>
<td>Physics</td>
</tr>
<tr>
<td>University of Massachusetts, Amherst</td>
<td>Ph.D.</td>
<td>1984-1990</td>
<td>Mol. and Cell. Biology</td>
</tr>
<tr>
<td>Imperial College, London</td>
<td>Post-doc</td>
<td>1990-1994</td>
<td>Mathematical Biology</td>
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A. Personal statement

I have been modeling the dynamics of immune responses and host-pathogen interactions since my postdoctoral fellowship at Imperial College in 1991. I approach problems in immunology and epidemiology from an ecological and evolutionary viewpoint.

At the within-host level, I view the immune system as a complex web of interconnected species (host cells, immune cells, resources, etc.), which can be perturbed by the introduction of invading pathogens. Studies from my group in the past decade focused on developing a quantitative framework for the understanding of very basic questions in immunology including: (i) Immune diversity: how diverse is the immune system (how many different αβ T cell receptors are expressed by the T cells in an individual), and how does this affect the number of naïve cells which are specific to a given pathogen? (ii) Generation of immune responses: what are the rules for the expansion and differentiation of T cells following infection and how does it depend on the duration of stimulation by antigen? (iii) How is immunological memory generated, and what dictates the longevity of this memory? (iv) What controls infections, and in particular can we determine the contributions of resource limitation, innate and adaptive immunity in the initial control of infections, and how does this affect the generation of resistance following antimicrobial treatment? (v) Why do pathogens harm their hosts and to what extent can we predict the emergence of novel pathogens and the evolution of their virulence?

More recently we have been extending these studies to consider persistent infections and aging, which are problems of relevance to human health. We are extending our earlier studies in the following directions. (i) What prevents pathogens from being cleared shortly after infection, why do some cause persistent infections? (ii) What are the rules that govern the behavior of immune cells if the pathogen persists, and why do immune cells display an exhausted (dysfunctional) phenotype? (iii) Can we use carefully timed post-exposure treatment shortly after initial infection to generate protective immunity (i.e. treatment as a vaccine)?

I have a rather small but productive group (a graduate student and a couple of post-docs) that works very closely with experimental immunologists (Rafi Ahmed at the Vaccine Center at Emory, and Joseph Blattman at Arizona State University) and statisticians and epidemiologists (Ira Longini’s group at the University of Florida). This allows us to rigorously integrate modeling with experimental validation and develop a quantitative understanding of the dynamics of infections.

B. Positions and honors

Positions and Employment
2009 - : Samuel Candler Dobbs Professor of Biology, Emory University
2007 - : Professor, Department of Biology, Emory University
2000 - 2007 : Associate-Professor, Department of Biology, Emory University
1994 - 2000 : Assistant Professor, Department of Biology, Emory University
Other Experience

2004-2007: Regular member of the Modeling and Analysis of Biological Systems (MABS) study section of the NIH.

Ad-hoc reviewer for NSF, NIH, Welcome Trust, and other agencies.


C. Selected 15 peer-reviewed publications (in chronological order)

Most relevant to the current application


Additional publications of relevance to the field


D. Research Support.

**Ongoing Research Support:**

NIH 1U01 GM70749 Longini/Halloran (PI) 5/01/2009 to 4/31/2014

Project Title: Containing Bioterrorist and Emerging Infectious Diseases

(continuation of MIDAS collaborative grant below.)

PI: Longini, I.M. Role: collaborator

Description: The overall objective of this research is to develop, validate, and implement mathematical and statistical models for the transmission and within-host dynamics of naturally occurring infectious diseases and bioterrorism agents.

My work of this project focuses on modeling the within-host dynamics of infectious diseases such as influenza, and linking the within-host dynamics of infection with the transmission of infections between hosts.

My part of the grant is about $80,000 direct costs per year.

**Completed Research Support**


Project Title: Quantitative models of CD8+ T-cell memory

Role: PI

NIH 1U01 GM70749 5/01/2004 to 4/31/2009

Project Title: MIDAS collaborative grant on Containing bioterrorist and emerging infectious diseases

PI: Longini, I.M. Role: collaborator
NIH 1RO1 AI 49334     7/01/2001 to 6/01/06 extension thro' 2/07
Project Title: Quantitative models of CD8+ immune memory
PI: Antia, R.

R29 GM54268         7/01/1996 to 6/30/2001 extension thro' 6/02
Project Title: Population Biology and Genetics of Antimicrobial Action
PI: Antia, R.