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The role of vaccination and treatment in controlling infectious diseases: A mathematical outlook

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This is the era of diseases. Almost every year we are struck by a new epidemic or disease making our survival more difficult. Reasons are many. There are three basic ways of withstanding the attacks of diseases, namely, (i) early identification and checking the spread of a disease; (ii) development of a suitable vaccine; and (iii) Medical treatment. First one is the best one – a preventive measure. However this is possible only in a well prepared and developed society which is not the case with most of the world. Vaccination is also a preventive measure. But we need to understand the structure and behavior of virus to prepare suitable vaccine. It is useful only for susceptible population but not for the infected ones. Also arrival of vaccine-resistant strains of viruses reduces its success. Diseases like AIDS and new viruses such as Ebola still await suitable vaccines. Here comes the role of cure – treatment process. For chronic diseases such as Chagas, AIDS etc., treatment is the only way out. Thus, vaccination and treatment work at two different stages and directions. In a recent exposition, the authors (the present speaker with Dr. M. Naresh Kumar) have studied the influence of vaccination and treatment in checking the spread of diseases by formulating a mathematical model under fairly general conditions. The model consists of susceptible, infected and recovered populations. Parameters and functional relations of the model represent the various activities of the disease environment. Several conditions on parameters and functions are obtained that force (i) a disease free environment or (ii) endemic environment. It is noticed that treatment is playing a major role in controlling the disease when compared to the influence of vaccination especially when the system is influenced by time delays in incubation. At the same time, an estimate on vaccination effort is provided for creating a disease free environment. Numerical examples are provided to check the theoretical results. Thus, 'an experiment on paper' is done. Owing to the ability of mathematics to interpret available information, predict for future and project for all circumstances, the stage is well set to utilize outcomes of these mathematical results. This helps in designing appropriate strategies for containing spread of diseases in a population

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Human immune responses to *Plasmodium falciparum* infection: Molecular evidence for a suboptimal TH α β and TH17 bias over ideal and effective traditional TH1 immunity

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Using microarray analysis, we showed up-regulation of Toll-like receptors 1, 2, 4, 7, 8, NF- κ B, TNF- α , p38-MAPK and MHC molecules in human peripheral blood mononuclear cells following infection with *Plasmodium falciparum*. We report herein further studies based on time-course microarray analysis with focus on malaria-induced host immunity. Results show that in early malaria; selected immunity-related genes were up-regulated including α , β and γ interferon related genes, as well as genes of IL-15, CD36, chemokines (CXCL10, CCL2, S100A8/9, CXCL9, and CXCL11), TRAIL and IgG Fc receptors. During acute febrile malaria, up-regulated genes included α , β and γ interferon related genes, IL-8, IL-1 β IL-10 downstream genes, TGFB1, oncostatin-M, chemokines, IgG Fc receptors, ADCC signaling, complement-related genes, granzymes, NK cell killer/inhibitory receptors and Fas antigen. During remission, genes for NK receptors, immunoglobins, and granzymes/perforin were up-regulated. When viewed in terms of immunity type, malaria infection appeared to induce a mixed TH1 response, in which α and β interferon driven responses appear to predominate over the more classic IL-12 driven pathway. In addition, TH17 pathway also appears playing a significant role in the immunity to *Plasmodium falciparum*. Gene markers of TH17 (neutrophil-related genes, TGFB1 and IL-6 family (oncostatin-M)) and TH α β (IFN- γ and NK cytotoxicity and ADCC gene) immunity were up-regulated. Initiation of TH α β immune response was associated with an IFN- γ response which ultimately resulted in moderate-mild IFN- γ achieved via a pathway different from the more classic IL-12 TH1 pattern. Based on these observations, we speculate that in *Plasmodium falciparum* infection, TH α β /TH17 immunity may predominate over ideal TH1 response.

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