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Editorial

Microbiome, Immunity and HIV Infection: Is there a Link?

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Human body and its activities remain harmonized with its environment until a third factor disturbs the balance of the two. A large part of our system is constituted of various kinds and counts of microorganisms which in totality are referred to as 'Microbiome'. Various microbial species inhabit different locales of our body playing a fixed role in co-ordination with our immune system [1]. Without this co-ordination either the microbes are thrown away by the immune system or the immune system is distressed by the disturbance of the microbes residing in and on our body. The most important niche wherein microbes reside in larger quantities and are most interactive being there is our gut. Gut metagenomics has paved way to know more and more about these microbes and has endowed with varied evidences about the role of microbes in controlling, supporting or disturbing our metabolic and immune status [2].

The role of gut microbiome in maintaining intestinal homeostasis and maintenance of mucosal barrier function and regulation of innate and adaptive immune responses is known now [3]. There are reports underlining a difference in gut microbial populations when analyzed in infected individuals and compared with healthy individuals especially in case of HIV infection [4,5]. At the same time, there are evidences that the resident microbial communities in the gut play a direct role in dictating immune activation or immune suppression which is the real driver in HIV disease progression. Further to this, analysis of microbial communities with respect to their metabolites, has informed that different microbial communities dominantly secrete different molecules like succinate, lactate, butyrate etc. which in turn dictates immune system. For instance, a report about small chain fatty acids (SCFAs) describes their role in cell migration, cytokine production, phagocytosis, and apoptosis in a type- and concentration-dependent manner. High levels of SCFAs induce the production of IL-8, IL-6 and IL-β by peripheral blood mononuclear cells (PBMCs) in vitro. Butyric acid decreased both the formation of HIV provirus and the production of HIV viral protein p24 by macrophages [6]. On the other hand, succinate has been identified as a metabolite in innate immune signaling, which enhances interleukin-1β production during inflammation [7]. Ladinsky and co-workers [8] have done an interesting study using electron tomography (ET) to image HIV-1 in gut-associated lymphoid tissue (GALT) of HIV-1infected humanized mice. They have also discussed that the CD4 depletion starts in the gut much before it becomes apparent in peripheral blood and lymph nodes. They have enlisted several features of gut which facilitate its susceptibility to HIV infection. Of interest herein, is the evidence that the GI mucosa includes high levels of proinflammatory, HIV-1 stimulatory cytokines produced by exposure to antigens in the external environment. This again confirms the role of microbial communities in creating this kind of environment.

During my presentations for the advocacy of exploring gut connections with HIV infection and its progression, the issues emerged like (i) site of infection is not the gut, then how come gut microflora can influence establishment of infection? (ii) Vaginal flora is almost all Lactobacilli and remains dominated with lactic acid; there is no chance for other to survive and play a role. It was really tough for me to answer such queries that time. However, I am convinced now that thinking other way is also not wrong. With new evidences, it appears that microbial dysbiosis is the early event in the establishment of HIV infection. It also seems convincing that a stage is set prior to the establishment of HIV infection at gut. Microbial translocation and other events occur later when the HIV infection proceeds. Hence the role of commensal microorganisms in allowing HIV to establish is gaining much importance and we may track a link between the gut metagenome, immunity and HIV infection with some more efforts. The studies with all advancement of experimentation should focus on elucidating the transition events during first exposure to HIV and the role of resident or changing microflora in allowing or restricting the infection. For instance, Hirao et al. developed and demonstrated the utility of the simian ligated intestinal loop model, which most closely recapitulates the anaerobic gut microenvironment. This model is capable of capturing the *in vivo* dynamics between microbes, the gut epithelium, and immune cell populations during the viral infection [9]. Hopes are high that targeting this link would give us a better tool to fight HIV infection in a manageable and host-friendly way.

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