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Short Communication

A Brief Note on Cell Mediated Immune Responses

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ABSTRACT

Tuberculosis is essentially a lung illness, and its spread is dependent on the productive infection of this vital organ. The acquired cellular immune response to Mycobacterium tuberculosis (Mtb) infection by aerosol is extremely slow to be produced and expressed within the lung. This slowness permits infection to take hold and compels the acquired response to take place in the setting of an inflammatory location that the bacteria has started and manipulated. Mtb possesses a range of surface chemicals that interact with the innate response, and this interaction, combined with the immune response's auto-regulation by numerous mechanisms, resulting in bacterial growth control that is less than optimum. Understanding of the mechanisms that mediate the development, expression, and modulation of the immune response in the lungs is necessary for improving current vaccine tactics. We also need to figure out how to trigger both known and new immune protection responses without triggering immunological pathology.

Keywords: Phagocyte; Immune-response; Inflammation

DESCRIPTION

The interaction of bacterial virulence and host resistance, which are two distinct and independent variables, results in tuberculosis pathogenesis; this has been known for a long time. In recent years, however, sophisticated technologies have enabled us to characterize the role of individual bacterial and host components to unprecedented levels [1]. Identifying virulence factors and therapeutic targets inside the bacterium, as well as components of the host's immune system that can be enhanced and even transformed by vaccination, is the targeted end point of research to describe bacterial and host components of this terrible disease. We discussed briefly only about early modern classic literature in this review. We concentrated on current research that has benefited from the maturing of tools that made accessible as a result of the publishing of the mouse and bacterial genomes. These technologies, together with the ability to genetically modify the bacterium, have expanded the ability to adjust the disease model in a controlled manner [2].

The direct effect of tuberculosis on global public health is widely acknowledged. Indeed, when one third of the world's population is exposed, the fact that just 5% of those exposed get sickness is not consoling when the case rate is 8 million new cases per year. When the ability of HIV infection to diminish immunity to Mycobacterium tuberculosis (Mtb) infection is taken into account, the implications for the spread of both drug-sensitive and drug-resistant tuberculosis are frightening. While the disease's tremendous public health impact justifies scientists' intense interest, immunologists have been interested by the sickness and the immunological pathologic lesions it causes since the discipline's beginnings [3].

Immunologists are interested in this topic because the lymphocyte response to mycobacterial infection is involved in both immunity and pathogenesis. While there is limited to no immunity in the absence of an acquired cellular response, the absence of this response also inhibits the creation of the characteristic caseation associated with pathogen transmission. The effects of HIV infection on the development of tuberculosis are possibly the greatest evidence to support this claim [4].

Tuberculosis is an indicator disease for HIV patients, appearing when CD4 counts are still significantly higher than those that predispose to other opportunistic diseases. When the immunological pathologic repercussions of Mtb infection in AIDS patients are examined, however, there is a significantly different clinical state and inflammatory response. There is a dominating granulocytic infiltration with necrosis, but not the characteristic caseous necrosis found in tuberculosis granulomas

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that are not infected with HIV. The mouse model in which the CD4 molecule is genetically altered shows a considerable inclination toward granulocytic involvement [5]. The acquired cellular response, which is predominantly represented by CD4 T cells, thereby provides protective immunity while also promoting the formation of mononuclear lesions and caseous necrosis, both of which are essential for transmission. The seeming contradiction of a strong cellular immune response at the location of an un resolving disease is due to the duality of the acquired cellular response's role.

CONCLUSION

Our understanding of the cellular response to Mtb infection has greatly expanded. However, we have yet to identify the sort of acquired cellular response that mediates immunity and should thus be generated by vaccination, severely limiting our ability to develop innovative vaccines. We're also unable to test new vaccines in humans because we lack a reliable predictor of protection. Our ability to define the human cellular response to infection and disease is improving because to advancements in locally available technology in areas with high disease incidence.

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