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Short Communication

Human Immunodeficiency Virus (HIV) Immunogenetics and the Pathogenesis of Host-Virus Interactions: A Short Communication

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DESCRIPTION

Human Immunodeficiency Virus (HIV) remains a global health challenge, affecting millions worldwide. The complex interaction between the virus and the host immune system determines the course of infection, disease progression and response to treatment. Key to understanding these dynamics is HIV immunogenetics, which explores how genetic variations in both the virus and the host influence susceptibility, immune response and clinical outcomes. The study provides a complete exploration of HIV immunogenetics, involve viral diversity, host genetic factors, immune evasion mechanisms, implications for vaccine development [1-3].

HIV and immunogenetics

HIV is a retrovirus that primarily targets Cluster of Differentiation 4 ($CD4^+$) T cells, plays important role in adapt immune responses. Upon infection, HIV integrates into the host genome and progressively impairs immune function, leading to Acquired Immunodeficiency Syndrome (AIDS) if untreated [4]. The variability in disease progression among individuals infected with HIV enhance the influence of host genetics on viral control and immune response [5].

Viral diversity and evolution

HIV exhibits remarkable genetic diversity due to its high mutation rate and rapid replication cycle. This diversity is categorized into two main types: HIV-1 and HIV-2, with HIV-1 being the predominant cause of the global pandemic. Within HIV-1, distinct subtypes (clades) and Circulating Recombinant Forms (CRFs) further contribute to viral diversity, impacting transmission dynamics, immune recognition and treatment efficacy [6].

HIV-1 subtypes: HIV-1 is classified into four groups (M, N, O, P), with Group M responsible for the majority of infections worldwide. Group M is further divided into subtypes (A-D, F-H, J, K) and numerous recombinant forms, each exhibiting unique genetic signatures that influence viral fitness and pathogenicity.

Impact on vaccine development: The genetic variability of HIV presents a significant challenge for vaccine development. Vaccine candidates must elicit broad and potent immune responses against diverse viral strains, necessitating innovative approaches to overcome antigenic variation and immune evasion strategies.

Host genetic factors influencing HIV susceptibility

Host genetic factors plays an important role in determining susceptibility to HIV infection, disease progression and response to Antiretroviral Therapy (ART). Genetic variations affecting immune cell receptors, cytokines and antiviral factors influence the interaction between HIV and the host immune system [7,8].

CCR5 delta32 mutation: The C-C Chemokine Receptor Type 5 (CCR5) receptor on $CD4^+$ T cells serves as a co-receptor for HIV entry into cells. Individuals homozygous for the CCR5 delta32 mutation (CCR5Δ32) are resistant to HIV infection, as the mutation disrupts CCR5 expression and impairs viral entry.

HLA variants: *Human Leukocyte Antigen (HLA)* genes encode Major Histocompatibility Complex (MHC) molecules involved in presenting viral antigens to T cells. Specific HLA alleles, such as HLA-B57:01 and HLA-B27:05 are associated with slower disease progression and enhanced immune control of HIV by presenting conserved viral epitopes to Cytotoxic T Lymphocytes (CTLs).

TRIM5α and APOBEC3G: Innate antiviral factors, such as TRIM5α and APOBEC3G, restrict HIV replication through mechanisms that include viral capsid destabilization and hypermutation of viral genomes. Genetic variants influencing the activity or expression of these factors impact viral susceptibility and control.

Immune evasion strategies of HIV

HIV employs multiple strategies to evade host immune responses, exploiting host genetic diversity and immune cell vulnerabilities:

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Viral mutagenesis: High mutation rates during viral replication generate diverse viral quasi-species, enabling escape from neutralizing antibodies and cytotoxic T cells.

Downregulation of MHC expression: HIV proteins (e.g., Nef, Vpu) interfere with MHC class I expression on infected cells, evading CTL recognition and immune surveillance.

Immune modulation: HIV proteins (e.g., Tat, Vpr) manipulate host immune responses by altering cytokine production, impairing dendritic cell function and inducing immune exhaustion in $CD4^+$ T cells [9].

Implications for vaccine development

Developing an effective HIV vaccine remains an urgent global health priority. Insights from HIV immunogenetics inform vaccine design strategies aimed at eliciting durable and broadly protective immune responses:

Conserved epitopes: Targeting conserved regions of the HIV genome, such as the CD4 binding site and the Membrane-Proximal External Region (MPER) of Glycoprotein 41 (Gp41), may induce broadly neutralizing antibodies (bnAbs) capable of cross-reacting with diverse viral strains.

T cell responses: Vaccine candidates stimulating strong $CD4^+$ and $CD8^+$ T cell responses against conserved viral epitopes may enhance viral control and reduce transmission rates.

Adaptive immunity: Understanding host factors influencing immune responses to HIV vaccines, including HLA polymorphisms and cytokine profiles, guides personalized vaccine approaches to individual genetic backgrounds.

Viral-host interactions

HIV immunogenetics focuses on advancing our understanding of viral-host interactions and optimizing therapeutic interventions:

Genome-Wide Association Studies (GWAS): Identifying host genetic variants associated with HIV susceptibility, disease progression and treatment outcomes informs precision medicine approaches and clinical management strategies.

Host-virus dynamics: Characterizing viral evolution, immune escape mechanisms and host factors influencing viral reservoir establishment and persistence underpins efforts to achieve sustained virologic remission (functional cure) in HIV-infected individuals.

Gene editing technologies: Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR-Cas9) and other gene editing tools enable targeted modification of host cells to enhance HIV resistance, disrupt viral latency and engineer immune cells for adoptive cell therapies [10].

CONCLUSION

HIV immunogenetics illuminates the complex genetic landscape underlying host-virus interactions, immune responses and disease outcomes. Genetic variations in both the virus and the host shape susceptibility to infection, influence viral control mechanisms and impact clinical trajectories in HIV-infected individuals. Advances in genomic technologies, coupled with insights into viral diversity and immune evasion strategies and prepare for personalized therapeutic approaches and preventive strategies. By the genetic complexities of HIV, to make an effective vaccine, improved treatment regimens and ultimately, a world free of HIV/AIDS. HIV immunogenetics enhance the pivotal role of genetic diversity in shaping the course of HIV infection and informs strategies for advancing HIV prevention, treatment and cure initiatives.

REFERENCES

1. Deeks SG, Walker BD. Human immunodeficiency virus controllers: mechanisms of durable virus control in the absence of antiretroviral therapy. Immunity. 2007;27(3):406-416.
2. Wilson MJ, Torkar M, Haude A, Milne S, Jones T, Sheer D, et al. Plasticity in the organization and sequences of human KIR/ILT gene families. Proc Natl Acad Sci U S A. 2000;97(9):4778-4783.
3. Uhrberg M, Valiante NM, Shum BP, Shilling HG, Lienert-Weidenbach K, Corliss B, et al. Human diversity in killer cell inhibitory receptor genes. Immunity. 1997;7(6):753-763.
4. Lanier LL, Corliss BC, Wu J, Leong C, Phillips JH. Immunoreceptor DAP12 bearing a tyrosine-based activation motif is involved in activating NK cells. Nature. 1998;391(6668):703-707.
5. Letvin NL, Walker BD. Immunopathogenesis and immunotherapy in AIDS virus infections. Nat Med. 2003;9(7):861-866.
6. Naranbhai V, Carrington M. Host genetic variation and HIV disease: from mapping to mechanism. Immunogenetics. 2017;69:489-498.
7. Bashirova AA, Bleiber G, Qi Y, Hutcheson H, Yamashita T, Johnson RC, et al. Consistent effects of TSG101 genetic variability on multiple outcomes of exposure to human immunodeficiency virus type 1. J Virol. 2006;80(14):6757-6763.
8. Boniotto M, Crovella S, Pirulli D, Scarlatti G, Spano A, Vatta L, et al. Polymorphisms in the MBL2 promoter correlated with risk of HIV-1 vertical transmission and AIDS progression. Genes Immun. 2000;1(5):346-348.
9. Javanbakht H, An P, Gold B, Petersen DC, O'Huigin C, Nelson GW, et al. Effects of human TRIM5α polymorphisms on antiretroviral function and susceptibility to human immunodeficiency virus infection. Virology. 2006;354(1):15-27.
10. Pereyra F, Addo MM, Kaufmann DE, Liu Y, Miura T, Rathod A, et al. Genetic and immunologic heterogeneity among persons who control HIV infection in the absence of therapy. J Infect Dis. 2008;197(4):563-571.