THE ENZYMATIC MECHANISMS OF GENETIC INFORMATION IMPLEMENTATION DURING IMMUNE RESPONSE TO DIFFERENT FORMS OF ANTIGENIC DETERMINANTS

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Introduction

The herpes virus type 6 (HHV-6) includes to a relatively small cohort of microbial pathogens, capable of usurpation lymphoproliferative mechanisms for its effective persistentention. Such options found in particular other herpesviruses (Epstein-Barr virus, cytomegalovirus), human immunodeficiency virus (HIV), hepatitis C virus. Underlying these capabilities, is the ability to influence the two main mechanisms of maturation of T and B lymphocytes - somatic recombination and somatic hypermutation. Using these same mechanisms (ADA and CDA immunoenzimes, somatic recombination and hypermutation) the immune system resists viral agents while maintaining its stability.

Research goals

It is nesessary to compare direct substrate activity for the two main enzymatic regulators of the immune response: CDA and ADA in patients with viral infections caused by pathogens can disturb the implementation of genetic information. Also need to assess the condition of somatic recombination for normal and infected patients.

Results

Comparative evaluation of gene rearrangements processes in polyclonal populations of B cells were performed for lymphocyte populations from blood of healthy volunteers, patients with chronic HCV infection and monoclonal controls. The computational analyze an immunoglobulin genes families' usage and to investigate nucleotide replacement in CDR and FR regions was performed by bioinformatic approaches. Clonal status for HCV patients reviled itself as similar to normal polyclonal picture. High somatic hypermutation levels, which may provoke HCV by activating CD81 were compensated by GC/AT mutation pressure variation. During Epstein-Barr virus infectious mononucleosis was observed extremely intensive increase (up to 80 IU/l) of serum ADA levels, which is a consequence of intense T-lymphocytes proliferation. Being active mutagen, CDA, conversely characterized by low inflammatory-dependent variations in different forms of acute and chronic viral infections: 1,21±0,17 IU/l for HCV infection, 0,54±0,21 for HIV, 0.70±0.43 IU/l for HHV-6. Normal CDA actyvity variates betveen 0.58 and 4.91 IU/l with average 1,82±0,36 IU/l.

Conclusions

The immune system has developed many effective mechanisms to counter the usurpation of its function by different pathogens. The main role this is given to CDA and ADA– immune response intensity end specify enzymatic regulators.