Molecular Analysis of the Pathophysiology of Cardiomyopathy

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Recent progress in molecular and cellular biochemistry in life science has resulted in the evolution of medical knowledge and techniques. This progress has contributed not only to analysis of the pathophysiology of diseases but also to the development of techniques for the monitoring and diagnosis of disorders.

Taking this opportunity, we designed this symposium to integrate the recent advances made by the various institutes using molecular biological techniques in the rapid expanding field of basic research of cardiomyopathy. By bringing together investigators ranging in their interests from molecular biology to immunology on the pathogenic process of cardiomyopathy, we have attempted to present a fairly comprehensive coverage.

Kawaguchi et al demonstrated the enhanced phosphatidyl inositol turn-over in cardiomyopathic hamster hearts (BIO 14.6 and BIO 53.58). They suggested that this abnormal PI response leads to the death of myocytes via elevation of intracellular calcium level. T. Ito et al observed deletions and point mutations of mitochondrial DNA in patients with dilated or hypertrophic cardiomyopathy. These genetic abnormalities appeared to affect the function of the mitochondrial respiratory chain resulting in the degeneration of myocytes, and to clarify the problems involved in the treatment of the disease. Nishi et al reported genetic factors in the immune system which may induce susceptibility to dilated cardiomyopathy. Their results revealed that HLA-DQB1 and immunoglobin lambda light chain genes are closely linked to the susceptibility of dilated cardiomyopathy. Seko et al and Fukuta et al also suggested that immunological processes play an important role in the degeneration of myocytes in dilated cardiomyopathy. Seko reported that cell-mediated cytotoxicity is mainly involved in the myocardial cell damage observed in viral myocarditis. Fukuta demonstrated the presence of anti-heart antibodies in the serum of patients with dilated cardiomyopathy. These observations strongly suggested that hypersensitive and long lasting immunological reaction induces gradual myocardial cell damage resulting in dilated cardiomyopathy.

Viral infection has been strongly implicated in the induction of cell-mediated cytotoxicity involved in the pathogenesis of dilated cardiomyopathy. To confirm this anticipation, Kitaura et al revealed the viral existence in endomyocardial biopsy samples obtained from patients clinically diagnosed as having dilated cardiomyopathy using PCR gene amplification. Through the formal presentation and informal discussion at this symposium, we certainly obtained better understanding of the problems of cardiomyopathy, especially dilated cardiomyopathy and the formulation of new approaches to their solution. In closing the symposium, we would like to express sincere appreciation to Prof. R. Kusukawa, the chairman of the 55th Congress of Japanese Circulation Society for his generous support and to all the speakers for their excellent presentations, without which it would have been impossible to organize the symposium and bring it to a successful conclusion.