Many steps are involved in physiological signal transduction to end into the final biological response. A cascade of reaction starts from the original signals or factors which act on the cell membrane, followed by chain reactions, which leads to translocation of specific signal proteins to the nucleus. Once in the nucleus, transcription factors are formed and subsequent translation leading to generation of protein(s) necessary for the final biological response. By understanding the sequence of these reactions, it is possible to intervene at different steps to stop an abnormal or overactivated reaction to control certain pathological conditions. Connective Tissue Disorders (CTD) exemplified by Rheumatoid Arthritis (RA), lupus and others as Dermatomyositis (DM), scleroderma have been shown to be involved in a mutated or over activated sequence of reactions. In lupus, an excessive formation of a stimulator called Blys or BAFF results in overactive B cells, hence, augmented plasma cells and consequent release of autoantibodies that form deleterious complexes in various organs and tissue in the body resulting in dysfunction and morbid effects. Also, various cytokines including TNF, INF and Interleukins are excessively released to add to the deleterious effects on normal body functions. The same applies to RA in which overactive T and B cells and associated cytokines exert cytotoxic effects on joints and body system targeting and damaging effectors components of tissues. Biological agents including antibodies and fusion proteins have been devised for blocking and targeting the following mis-regulated sites: growth signal and factors, affected cells, receptor sites, components of cascade reactions and released cytokines. Examples include the following: IL1 inhibitors (Anakinra), IL6 inhibitors (Toclizumab), CD20 inhibitors (Rituximab), CD 22 inhibitors (Epratuzumab), BAFF (Blys) inhibitors (Belizumab), APRIL inhibitors (Atacicept), Co-stimulation inhibitor (Abatacept) and TNF inhibitor (Infliximab and similar). These agents mainly target the components responsible for the pathological pathway and call for the immune system to coordinate for the removal of the offending components of the disease. No doubts, these agents have been considered as an armamentarium to stop the progress of the above mentioned disabling, aggressive pathological conditions for the goal of curing and improving quality of life. Although these are considered as recent breakthrough in the field of targeted immunotherapy for CTD, high cost presents a barrier for the wide clinical use of these agents. In addition, they might increase the susceptibility to infection because of the immune depressing effects of these biologics beside the activation of dormant diseases and potential cancerous conditions and possible precipitation of allergic manifestations.

Biography

Fouad Moustafa Sharabi is a Professor Emeritus of Pharmacology and Toxicology at the Faculty of Pharmacy, Alexandria University, Egypt. He has teaching and research experience with professional background including pharmaceutical, hospital and clinical pharmacy services. Previously, he was enrolled in academic leadership positions: Head of Pharmacology and Toxicology Department, then as Vice Dean of students’ affairs and Acting Dean of the Faculty of Pharmacy. Presently, he is the Head of the National Scientific Committee for Faculty Promotion of Professors and Associate Professors in the specialization of Pharmacology and Clinical Pharmacy of the Faculties of Pharmacy in the Egyptian universities. Additionally, he was involved in various activities including continuing education services, drug development for pharmaceutical companies, certified Specialist of Medical Analysis and Administrative Work (previous General Coordinator of Clinical Pharmacy program). He was honored by the University of Alexandria Appreciation Prize 2014 and has participated in several local and international scientific meetings and invited lectures and coauthor of several international textbooks.

fouadmost@hotmail.com