Preface

The group of pattern recognition receptors (PRRs) includes families of Toll-like receptors (TLRs), NOD-like receptors (NLRs), C-type lectin receptors (CLRs), RIG-I-like receptors (RLRs), and AIM-2-like receptors (ALRs). Conceptually, receptors constituting these families are united by two general features. First, they directly recognize common antigen determinants of virtually all classes of pathogens (so-called pathogen-associated molecular patterns, or simply PAMPs) and initiate immune response against them via specific intracellular signaling pathways. Second, they recognize endogenous ligands (since they are usually released during cell stress, they are called damage-associated molecular patterns, DAMPs), and, hence, PRR-mediated immune response can be activated without an influence of infectious agents. Thus, PRRs play the key role in performing the innate and adaptive immune response. In addition, many PRRs have a number of other vital functions apart from participation in immune response realization.

The fundamental characters and diversity of PRR functions have led to amazingly rapid research in this field. Such investigations are very promising for medicine as immune system plays a key role in the vast majority if not all human diseases, and the process of discovering the new aspects of the immune system functioning is rapidly ongoing. The latest discoveries in the field of genomics led to understanding of the fact that inherited variation in genes encoding PRRs may alter their expression and activity, modulating the risk and clinicopathological features of various diseases. These diseases may be divided into four major groups: infectious diseases, autoimmune and allergic disorders, cardiovascular diseases, and cancer. With respect to the first and second mentioned groups, the role of genomic variation in PRRs in their development is relatively simple. The enhanced PRR activity leads to the effective prevention of infectious diseases, whereas it may also launch and promote various autoimmune processes; conversely, diminished PRR functioning will increase the chance to catch an infectious disease, simultaneously reducing the risk of autoimmune and allergic disorders. For cardiovascular diseases and cancer, the situation is much more complicated, resembling a double-edged sword for cancer and the chain of probabilities for cardiovascular diseases.

Hence, in this book we consider the role of the structural genomic variation in PRRs in the development of these two groups of diseases. First, we describe the

role of PRRs in the construction of the entire immune system, and then the structure and functioning of TLRs and NLRs is considered in detail, since they are the most significant and studied groups of PRRs. Secondly, we briefly relate to the conception of the structural genomic variation in modern biology. The main part of the book is devoted directly to the role of inherited variation in PRRs in cancer and cardiovascular diseases. In the last chapter, we summarize the situation in the field, indicate the key directions, and suggest a conception of integrative systems of genomic risk markers for the prevention of various pathologies. To the best of our knowledge, this is the only book analyzing the field of PRR genomics and its practical applications. We prepared this book with the hope that it would be useful for a wide audience, particularly immunologists, cancer researchers, cardiologists, epidemiologists, microbiologists, geneticists, as well as for Ph.D., graduate and undergraduate students of biomedical faculties and their lecturers.

Kemerovo, Russian Federation

Anton G. Kutikhin
Arseniy E. Yuzhalin

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Kutikhin, A.G.; Yuzhalin, A.E.

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