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VIIth Complement Genetics Workshop and Conference

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It is often difficult to identify the beginning of a scientific field. In this issue, Chester Alper, who has personally made many key contributions to its advancement, has reviewed the dawn of complement genetics, revealing that already 80 years ago, a complement-deficient guinea pig was identified, and has also recalled that it was only 30 years ago when the first description of a genetic polymorphism was discovered independently by three groups for the third component and published in the United States in 1968 and a year later in the former East Germany. These observations have initiated a new field of genetic research. The first international workshop on complement genetics was held in Bonn, Germany, in 1972. It focussed on a single component, C3. Twenty scientists discussed methods, nomenclature, formal and population genetics of C3 polymorphism. Thereafter, complement genetics workshops have been held in Cambridge in 1977, in Cologne in 1979, in Boston in 1982 and in 1986, and in Mainz in 1989 and very recently, in 1998, again on the banks

of the Rhine, only 150 km away from Bonn, the cradle of this workshop series.

The results of the reference typings of the recent 1998 workshop have been compiled in this issue. Many reference typing laboratories all around the world took part in comparative investigations on selected complement components. It was the aim of this workshop, as of the previous ones, to update most of the known complement polymorphisms by standardising reference typing and proposing nomenclature revisions. Emphasis has been laid on complement components of the major histocompatibility complex (MHC) class III cluster, especially C4, as before, on components of the terminal complement complex (TCC or MAC) gene cluster (C6, C7 and C9), and on complement receptor 1 (CR1) of the regulators of complement activation (RCA) gene cluster. For C2, C8, factor I, and other components of the RCA cluster, we have to refer to the previous proceedings [1].

High voltage agarose gel electrophoresis and isoelectric focusing, combined with hae-

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molytic overlay, immunoblotting, or immunofixation have, for years, been the most important technique for the assessment of complement allotypes. Despite the fact that allo-specific monoclonal antibodies are available for C4, C3 and C7, genetic typing of polymorphic variants by electrophoretic methods has maintained its dominant position. However, using recombinant DNA technology, tremendous progress has been accomplished over the last 15 years in the definition of the genetic basis of the complement system, including polymorphisms and deficiencies. Most of the approximately 40 genes coding for C components or subunits, receptors and regulators have been cloned, sequenced and assigned to chromosomal locations. Protocols for both protein and DNA typing methods have recently been compiled and discussed in great detail – despite some overlap, both publications ‘complement’ each other quite well [2, 3].

Apart from the historical overview and the reference typing reports, this issue also contains reviews of selected presentations at the meeting covering

- gene organisation and evolution of the MHC class III complement genes (Yu),
- deficiency of C1q and its implications for development of autoimmune disease (Botto),
- gene organisation and evolution of the terminal complement genes (Hobart),
- deficiencies of terminal complement genes (Fukumori).

An overview of these and other aspects of the meeting, including the application of polymorphisms to a wide range of problems, such as association with disease, structure-function relationships, as well as phylogenetic and population genetic studies, is concurrently published elsewhere [4].

We hope that this issue will help the workshop participants review the meeting and assist the interested reader in obtaining the latest information on complement genetics. Scientists working with a particular component may contact the authors for any questions, particularly on polymorphisms and deficiencies both on the protein and on the molecular level. We especially hope to encourage young scientists to join us in this exciting task and conclude with the optimistic view that it will not take another nine years before we have a chance to meet again at the VIIIth Complement Genetics Workshop.

Last but not least we would like to acknowledge gratefully the financial support we have received for organising this meeting. The VIIth Complement Genetics Workshop was sponsored the Deutsche Forschungsgemeinschaft (DFG), the Academy of Sciences and Literature in Mainz, as well as by travel grants from the BIOMED 2 concerted action (Complement in Infection and Chronic Disease, secretary M.R. Daha, Leiden) via the European Complement Network (ECN, secretary R. Würzner, Innsbruck).

The Editors

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