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Foreword

"The important thing in science is not so much to obtain new facts as to discover new ways of thinking about them."
Sir William Bragg (1862–1942)

Since meiosis was first proposed by August Weismann, and demonstrated, by Edouard van Beneden, in the 1880s, it has become evident that this process is essential for the propagation of sexually reproducing species, at the same time resulting in the mixing of parental alleles that is an additional consequence of genetic recombination. It was not until 1931, in landmark papers by Harriet Creighton and Barbara McClintock as well as by Curt Stern, that cytologic evidence of crossing over was demonstrated, but the molecular basis for these events remained elusive for several decades, despite intense interest in the field. The past 20 years or so have seen the emergence of many different model systems for studying meiotic recombination, from budding and fission yeast, to humans. This menagerie of biologic systems has fostered an intense and lively environment in the field.

What has emerged from these studies is a picture of a stringently regulated process that guarantees accurate segregation of homologous chromosomes during the first meiotic division, while at the same time allowing recombination events to synchronize with the cell cycle, replication, and transcriptional machineries within the cell. The high level of stringency required to ensure fidelity of the meiotic process at the chromosomal level can only be fully appreciated when one considers the intricacy involved in orchestrating the breakage of DNA molecules and the subsequent formation of hybrid DNA. Such events must be strictly monitored to limit the frequency of crossover events and to ensure accurate repair of hybrid DNA. These processes are regulated by a large group of DNA repair and modification proteins, collectively termed the recombinational repair machinery.

How the fidelity of these events is ensured in different organisms of varying genome size differs, but the members of the recombinational repair machinery remain largely conserved. And thus the outcome of these events is efficiently maintained in all organisms but one, the human, in which the frequency of meiotic errors in oocytes is estimated to be as high as 20%. Why are humans so error prone? How is recombination so exquisitely regulated across other sexually reproducing organisms, resulting in less than 1% aneuploidy rates in most organisms? Only by taking full advantage of the different model systems and the myriad of experimental techniques now available, can we begin to answer these questions. The articles included in this single topic issue of ***Cytogenetic and Genome Research*** serve to illustrate the power of such comparative analyses. These papers are from experts in the field who study recombination in yeast, flies, mice and humans. My thanks go to them for their outstanding contributions to the field and, more specifically to this exciting volume.

It has been my pleasure to serve as guest editor for this issue of the journal, and I extend my heartfelt thanks to Harold Klinger and Michael Schmid for honoring me with the invitation to take on this responsibility. In addition, I would like to thank other members of the *CGR* team, particularly Barbara Grandinetti for all her hard work in co-ordinating this issue, and for coping with a novice editor such as myself. Finally, I thank all the members of the meiosis community who served as referees for these articles. Without the efforts of all these various groups – the authors, the referees and the *CGR* team – this issue would not have been possible. I hope that you, the reader, will enjoy this product of their collective labors.

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