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BRCA1 and BRCA2: Genome Instability and Tumorigenesis

Guest Editor:

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Deadline for manuscript submissions:
closed (20 April 2021)

Message from the Guest Editor

Inherited mutations in BRCA1 and BRCA2 (breast cancer genes 1 and 2) predispose individuals to a high risk of breast and ovarian cancer. BRCA1 and BRCA2 are well-established DNA damage repair proteins with roles in homologous recombination-driven double strand break repair, inter-strand crosslink repair, R-loop processing, and stalled replication fork repair. Increased genomic instability upon defective DNA damage repair in BRCA1- and BRCA2-deficient cells is considered to be one of the driver events in tumorigenesis.

In this Special Issue, we welcome reviews, original articles, and short reports that cover different aspects of BRCA1 and BRCA2 biology. These include, but are not limited to, molecular mechanisms that drive BRCA1 and BRCA2 mutant cancer, the role of BRCA1 and BRCA2 in different DNA damage repair pathways, haploinsufficiency for BRCA1 or BRCA2 functions, different isoforms of these proteins that contribute to tumorigenesis, mechanism-based treatment strategies for BRCA1/2 mutant cancer, and the mechanisms that drive chemotherapy resistance in BRCA1/2 mutant tumors. We look forward to your contributions.

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Message from the Editor-in-Chief

Genes are central to our understanding of biology, and modern advances such as genomics and genome editing have maintained genetics as a vibrant, diverse and fastmoving field. There is a need for good quality, open access journals in this area, and the *Genes* team aims to provide expert manuscript handling, serious peer review, and rapid publication across the whole discipline of genetics. Starting in 2010, the journal is now well established and recognised.

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