

Special Issue

Oncogenic Driver Mutations and Signaling Pathways in Tumorigenesis and Therapy in Lung Cancer

Message from the Guest Editor

Lung cancer poses a significant global health challenge as its incidence and mortality rates continue to rise. The initiation, progression, and response to therapy for lung cancer are heavily influenced by oncogenic driver mutations and dysregulated signaling pathways. This Special Issue, titled “Oncogenic Driver Mutations and Signaling Pathways in Tumorigenesis and Therapy in Lung Cancer”, aims to delve into the complex landscape of oncogenic drivers and signaling cascades involved in lung tumorigenesis and treatment. By uncovering the molecular mechanisms behind cancer progression, our goal is to pinpoint therapeutic targets and propel precision medicine approaches to improve patient outcomes. Authors are encouraged to submit original research articles, reviews, and meta-analyses that advance our knowledge of oncogenic driver mutations and signaling pathways in lung cancer, specifically focusing on tumorigenesis and treatment strategies. Studies involving the manipulation of oncogenic driver mutations in animal models to investigate tumorigenesis and treatment responses are particularly welcome.

Guest Editor

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Deadline for manuscript submissions

closed (31 May 2025)



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About the Journal

Message from the Editor-in-Chief

Cancers (ISSN 2072-6694) is an international, online journal addressing both clinical and basic science issues related to cancer research. The journal will continue its open access format, which will certainly evolve to ensure that the journal takes full advantage of the rapidly changing world of information and knowledge dissemination. It publishes high-quality clinical, translational, and basic science research on cancer prevention, initiation, progression, and treatment, as well as other related topics, particularly to capture the most seminal studies in the rapidly growing area of immunology, immunotherapy, and tumor microenvironment.

Editor-in-Chief

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