FEATURES

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Role of the *Helicobacter pylori*–Induced Inflammatory Response in the Development of Gastric Cancer

Acacia Lamb and Lin-Feng Chen

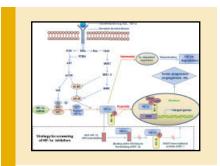
Helicobacter pylori (*H. pylori*) infection causes chronic gastritis and peptic ulceration and is the strongest risk factor for the development of gastric cancer. The pathogenesis of *H. pylori* is believed to be associated with infection-initiated chronic gastritis, which is characterized by enhanced expression of many inflammatory genes. *H. pylori* utilizes various virulence factors, targeting different cellular proteins, to modulate the host inflammatory response. In this review, the many different ways by which *H. pylori* initiates inflammation, leveling many "hits" on the gastric mucosa which can lead to the development of cancer is explored. Recent findings in understanding the pathogen-host interactions and the role of transcription factor NF-kB in *H. pylori*-induced inflammation is also discussed.

Recent Agents Targeting HIF–1 α for Cancer Therapy Yaozhong Hu, Jing Liu, and He Huang

Tuoznong Hu, Jing Liu, unu He Huung

The discovery of hypoxia-inducible factor-1 (HIF-1) has led to an increasing understanding of the mechanism of tumor hypoxia in the past two decades. As a key transcriptional regulator, HIF-1 plays a central role in the adaptation of tumor cells to hypoxia by activating the transcription of targeting genes, which regulate several biological processes including angiogenesis, cell proliferation, survival, glucose metabolism and migration. The inhibitors of HIF-1 in cancer have provided us a new clue for the targeting cancer therapy. This review will introduce the general knowledge of the biology characteristic of HIF-1 and mechanism of O_2 -dependent regulation. Moreover, a number of chemical inhibitors plus protein and nucleic acid inhibitors are included and classified mainly based on their different mechanism of inhibiting action. We also prefer to discuss the advantages of protein and nucleic acid inhibitors compared with chemical inhibitors.

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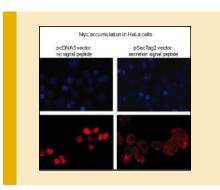
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Journal of Cellular Biochemistry

Conflict of Interests: Multiple Signal Peptides With Diverging Goals

Annunziata Venuto and Ario de Marco

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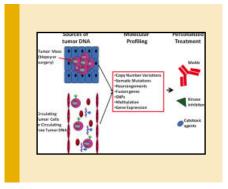


Peptide signal sequences attached to or embedded into a core protein sequence control its cellular localization and several post-translational modifications. However, misleading or cumbersome results may be generated when expressing recombinant proteins with modified signal peptides or single domains of larger proteins

Molecular Diagnostics and Personalized Medicine in Oncology: Challenges and Opportunities

Nicola Normanno, Anna Maria Rachiglio, Cristin Roma, Francesca Fenizia, Claudia Esposito, Raffaella Pasquale, Maria Libera La Porta, Alessia Iannaccone, Filippo Micheli, Michele Santangelo, Francesca Bergantino, Susan Costantini, and Antonella De Luca

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Increasing evidence demonstrates that target-based agents are active only in molecularly selected populations of patients. Therefore, the identification of predictive biomarkers has become mandatory to improve the clinical development of these novel drugs. Mutations of the epidermal growth factor receptor (EGFR) or rearrangements of the ALK gene in non-small-cell lung cancer, and BRAF mutations in melanoma are clear examples of driver mutations and predictive biomarkers of response to treatment with specific inhibitors. Predictive biomarkers might also identify subgroups of patients that are not likely to respond to specific drugs, as shown for KRAS mutations and anti-EGFR monoclonal antibodies in colorectal carcinoma. The discovery of novel driver molecular alterations and the availability of drugs capable to selectively block such oncogenic mechanisms are leading to a rapid increase in the number of putative biomarkers that need to be assessed in each single patient. In this respect, two different approaches are being developed to introduce a comprehensive molecular characterization in clinical practice: high throughput genotyping platforms, which allow the detection of

recognized genetic aberrations in clinical samples, and next generation sequencing that can provide information on all the different types of cancer-causing alterations. The introduction of these techniques in clinical practice will increase the possibility to identify molecular targets in each individual patient, and will also allow to follow the molecular evolution of the disease during the treatment. By using these approaches, the development of personalized medicine for patients with cancer will finally become possible.



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