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Competitor Analysis: KRAS Inhibitors

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Abstracts

Competitor Analysis: KRAS Inhibitors

This Competitive Intelligence report about KRAS Inhibitors evaluates the landscape of investigational small molecules, antibodies, cell therapeutics and vaccines targeting the Kirsten RAt Sarcoma virus oncogene (KRAS) for treatment of cancer as of October 2019.

KRAS is a gene that acts as an on/off switch in cell signalling. When it functions normally, it controls cell proliferation. When it is mutated, negative signalling is disrupted, allowing that cells can continuously proliferate, and often develop into cancer.

KRAS was first identified as an oncogene in Kirsten RAt Sarcoma virus. The viral oncogene was derived from cellular genome. Thus, KRAS gene in cellular genome is called a proto-oncogene.

The KRAS protein is a GTPase and is an early player in many signal transduction pathways. KRAS is usually tethered to cell membranes because of the presence of an isoprene group on its C-terminus. There are two protein products of the KRAS gene in mammalian cells that result from the use of alternative exon 4 (exon 4A and 4B, respectively): K-Ras4A and K-Ras4B, these proteins have different structure in their C-terminal region and use different mechanisms to localize to cellular membranes including the plasma membrane.

Somatic KRAS mutations are found at high rates in leukemias, colorectal cancer, pancreatic cancer and lung cancer. Driver mutations in KRAS underlie the pathogenesis of up to 20% of human cancers. Hence KRAS is an attractive drug target, however lack of obvious binding sites has hindered pharmaceutical development.

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Despite its well-recognized importance in cancer malignancy, continuous efforts in the past three decades failed to develop approved therapies for KRAS mutant cancer. KRAS has thus long been considered to be undruggable. Encouragingly, recent studies have aroused renewed interest in the development of KRAS inhibitors either directly towards mutant KRAS or against the crucial steps required for KRAS activation.

One fairly frequent driver mutation is KRASG12C which is adjacent a shallow binding site. This has allowed the development of electrophilic KRAS inhibitors that can form irreversible covalent bonds with nucleophilic sulfur atom of Cys-12 and hence selectively target KRASG12C and leave wild-type KRAS untouched.

The report includes a compilation of currently active projects in research and development of KRAS inhibitors for treatment of cancer. In addition, the report lists company-specific R&D pipelines of KRAS inhibitors. Competitor projects are listed in a tabular format providing information on:

Drug Codes,

Target/Mechanism of Action,

Class of Compound,

Company,

Product Category,

Indication,

R&D Stage and

additional comments with a hyperlink leading to the source of information.

About Competitor Analysis Series:

The Competitor Analysis Series delivers NO-FRILLS, but concise information about the pipeline of R&D projects for targets, diseases, technologies and companies. The information is provided in a tabular format and fully referenced.

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