DRUGGABLE ONCOGENIC FUSIONS

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BACKGROUND

Cancer patients frequently bear therapeutically relevant genome alterations. For instance, lung adenocarcinomas of patients that have never smoked carry genome alterations affecting kinases, such as EGFR mutations and translocations affecting ALK, ROS1, and RET genes. These patients can be effectively treated with an ever-growing number of kinase inhibitors.

PROBLEM

However, despite substantive cancer genome sequencing efforts a majority of tumors still lacks therapeutically tractable alterations.

SOLUTION

Scientists of the University of Cologne identified NRG1 gene fusions as ideal diagnostic and prognostic markers and targets for various tumors. The MTSS1-NRG1 fusion event has e.g. been detected in patients with small cell lung cancer and the gene fusion CD74-NRG1 has been shown to occur frequently in never smokers with invasive mucinous lung adenocarcinoma lacking KRAS mutation. The latter has been verified by several other groups.

CD74-NRG1 was found to signal through induction of ERBB2-ERBB3 heterodimers. In light of the multitude of available drugs or drugs in clinical trials targeting ERBB2, ERBB3 and their downstream pathways, the detection of CD74-NRG1 fusions may aid making a decision on the appropriate medical treatment e.g. for invasive mucinous lung adenocarcinomas.

Furthermore, the fusion itself represents a promising target for the development of medical interventions.

Thus, these findings position MTSS1-NRG1 and CD74-NRG1 as druggable oncogenic fusions.
ADVANTAGES

- CD74-NRG1 is a novel fusion gene which - by itself or its products - can be used both as a target for medical intervention and for diagnosis e.g. in invasive mucinous adenocarcinomas
- MTSS1-NRG1 as another druggable oncogenic NRG1 fusion gene
- Known signalling pathways # Enables stratification of patients for ERBB2 and/or ERBB3 inhibitor treatment

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PUBLICATIONS & LINKS


