



*Market Sector:
Clinical diagnostics, Drug development*

Novel gene for classifying cancer

Summary of invention

This invention describes a novel gene, FANCI, that functions in the Fanconi anemia pathway of genomic maintenance. Maintenance of genomic stability is a major mechanism against tumor development, and tools that allow identification of Fanconi status have great impact on tumor classification and prediction of drug sensitivity.

Applications

1. Predicting drug sensitivity: With the discovery of this new Fanconi gene, tumors can be classified as being defective in this gene or not. In case a defect is found, this result predicts higher sensitivity to cross-linking agent (such as cisplatinum, cyclophosphamide) for chemotherapy. It is known that tumors with Fanconi pathway defects are oversensitive to cross-linking chemotherapeutics.
2. Drug development: A high-throughput screen for small molecule inhibitors of FANCI may result in the identification of agents that specifically block FANCI. Such agents, when specifically targeted to tumor cells, may result in substantial improvement of cure rates by cross-linking agents.

Commercial partner

- Available for licensing
- Specific area of activity of the partner: oncology, diagnostics or drug development

Remarks

This opportunity is being developed as part of a large Fanconi pathway program conducted at the VU medical centre by the group of Prof. Dr Hans Joenje. Additional information is offered upon execution of a Confidentiality Agreement.

KEYWORDS

Fanconi pathway;
Genomic instability;
Chemo-sensitivity;
Predicting anti-tumor response

KEY BENEFITS

Improved prediction of response to chemotherapy.

Improved tumor classification.

PATENT / IP STATUS

Patent application filed in March 2007

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OPPORTUNITY

Background

Cancer results from alteration in the genome of somatic cells. These alterations can accumulate due to a genomic instability that is thought to exist in the premalignant phase of tumor development. This state of genomic instability is caused by a somatically acquired defect in a genomic maintenance mechanism. Many different maintenance mechanisms have been described. One such mechanism is the "Fanconi anemia (FA) pathway of genomic maintenance". This pathway is controlled by at least 13 different proteins, each of which is equally essential for the pathway to function. Twelve genes/proteins have already been identified: FANCA, FANCB, FANCC, FANCD1/BRCA2, FANCD2, FANCE, FANCF, FANCG, FANCI/BRIP1, FANCL, FANCM and FANCN/PALB2. A thirteenth, FANCI has now been identified by us, and is subject of this licensing opportunity.

Two important features are associated with the FA pathway (and with defects thereof), which lead to possible applicability in cancer therapy.

First, cells with a FA pathway defect possess an unstable genome and as a result will accumulate genomic alterations at accelerated speed, leading to full-blown tumor cells with high probability.

Second, cells with a FA pathway defect are highly sensitive to a specific class of chemotherapeutic agents known as "polyfunctional alkylating" or "cross-linking" agents (examples: cisplatin, mitomycin C, cyclophosphamide). Tumors that have resulted from a FA defective premalignant cell are thus likely to be responsive to treatment with cross-linking agents. There is evidence for a proportion of common cancers to show such a FA pathway defect as a result of mutations in or silencing of one of the FA genes.

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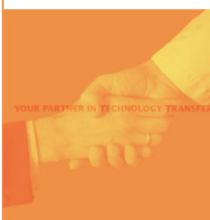
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