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Kancera provides an operational update of the PFKFB3 project.

Kancera reports that PFKFB3-inhibitor KAN0438757 increases the effect of a promising class of drugs for intractable cancer. Kancera has strengthened their development of drugs directed against PFKFB3 and DNA repair through a new recruitment. The European and US patent authorities have announced that a patent will be granted for the PFKFB- inhibitor KAN0438757 and analogues of this substance.

- Kancera, together with the Thomas Helleday group at the Science for Life laboratory, have previously shown that Kancera's PFKFB3 inhibitor increases the effect of DNA-damaging treatment such as chemotherapy and radiation by antagonizing cell repair functions. This treatment concept has grown strongly with the demonstration that PARP inhibitors, which act by this type of mechanism, have proven successful in the treatment of ovarian cancer, breast cancer and castration-resistant prostate cancer.

  PARP-inhibitors, such as Veliparib, are effective against cancers that are defective in their ability to repair DNA (recombination-defective), while cancer that retains this ability resists the same treatment.

- Studies now show that cancer cells that are resistant to Veliparib become sensitive to treatment when Veliparib is combined with KAN0438757. This means that Kancera's PFKFB3 inhibitor in combination with PARP inhibitors can combat more treatment-recalcitrant cancers than PARP inhibitors alone.

  The reason Kancera PFKFB3 inhibitors increase the effect of Veliparib is that they block cancer cells' ability to repair DNA.

  Since normal, healthy cells are neither recombination-defective nor do they depend on PFKFB3 for their DNA repair function, it is possible that such a combination could be well tolerated with few side effects.

- Kancera AB (publ) has employed Nina Gustafsson PhD as expert in the biology of DNA repair. Nina will divide her time between Kancera and Thomas Helleday's Laboratory at the Karolinska Institute, with main responsibility in the latter as project and team leader. Nina holds a PhD from the Karolinska Institute of Medical Sciences and has received numerous research grants for her work on cancer metabolism and its relationship to DNA repair.

  "With the recruitment of Nina Gustafsson we strengthen both Kancera's expertise in DNA repair and our successful collaboration with the Karolinska Institute on the PFKFB3 project," says Thomas Olin, CEO of Kancera.

- Kancera has received advance notice from the patent authorities in the EU and the United States that claimed around PFKFB3 inhibitor KAN0438757 will be granted.

About the PFKFB3 project
The project aims to develop inhibitors of the enzyme PFKFB3 so as to strangle cancer cell energy metabolism and thus render them vulnerable to chemotherapy and radiotherapy. Kancera AB has, together with Professor Thomas Helleday and his research group at Karolinska Institutet, made a surprising discovery that shows how the company inhibitor of

PFKFB3 enters the cancer cell's nucleus and enhances the effects of a newly given dose of radiation.

About Kancera AB (publ)
Kancera develops the basis for new therapeutics, starting with new treatment concepts and ending with the sale of a drug candidate to international pharmaceutical companies. Kancera is currently developing drugs for the treatment of leukemia and solid tumors, based on blocking survival signals in the cancer cell and on addressing cancer metabolism. Kancera's operations are based in the Karolinska Institutet Science Park in Stockholm and the company employs around 15 people. Kancera shares are traded on NASDAQ First North and the number of shareholders were more than 7700 as of January 13th, 2017. FNCA is Kancera's Certified Adviser. Professor Carl-Henrik Heldin, Professor Håkan Mellstedt, and MD PhD Charlotte Edenius are board members and Kancera´s scientific advisers.

For further information contact,
Thomas Olin, CEO: +46-735-20 40 01
Address:
Kancera AB (publ)
Karolinska Institutet Science Park
Banvaktsvägen 22
SE 171 48 Solna

Visit our home page at: http://www.kancera.com.