Press release Stockholm 2016-05-18

Kancera provides operational update of the Fractalkine and ROR projects

Kancera reports that the Fractalkine antagonist KAN0440567 is able to eliminate pain resulting from inflammation of the pancreas and that the ROR inhibitor KAN0439834 has been shown to effectively kill resistant cancer cells from the bone marrow of multiple myeloma patients.

Kancera has previously announced that the company's goal for the Fractalkine antagonist KAN0440567 in cancer is to cause tumor regression and to relieve severe pain. Pain in pancreatic cancer is similar to the pain resulting from inflammation of the pancreas. For this reason, studies have been conducted to find out how effective KAN0440567 can alleviate pain in animal models of inflamed pancreas. Now Kancera reports that the researchers and surgeons Gueralp Ceyhan and Jan D'Haese, Klinikum rechts der Isar (University Hospital at the Munich Technical University), have conducted animal studies showing that the severe pain caused by an inflamed pancreas is eliminated by oral administration of KAN0440567. The study also shows that the pain signal activation through the spinal cord, which in cancer could be caused by the cancer itself or due to side effects of chemotherapy (e.g. following treatment with paclitaxel), is reduced by Kancera's Fractalkine antagonist . The results support Kancera's continued investment in KAN0440567 for clinical development. Further studies will be focused on determination of the minimum effective dose for the treatment of cancer pain, and based on that, assess the safety of the treatment.

In the ROR project, Kancera has previously announced that studies are undertaken to examine possible additional applications of the ROR inhibitors against cancer in addition to treatment of chronic lymphocytic leukemia (CLL). One of the cancer diseases studied is multiple myeloma (MM) which is manifested in the bone marrow and is an incurable chronic disease today. Cancer cells from both CLL and MM patients carry ROR1 and are driven by a cancer stimulating signaling called "Wnt". Kancera now reports that the company's ROR inhibitors block both the pathways that "Wnt" conveys in cancer cells. In line with these results, Kancera together with Professor Håkan Mellstedt at the Karolinska Institute and University Hospital, has also shown that resistant cells from the bone marrow of MM patients are effectively killed by Kancera's ROR inhibitor KAN0439834. Further studies are now focused on translating these findings to effects in animal models of MM which will provide a basis for decisions on future clinical trials evaluating Kancera's ROR inhibitors.

About the Fractalkine project
Fractalkine is an immune regulatory factor that sends signals via the CX3CR1 receptor, also called G-protein coupled receptor 13 (GPCR13). In the healthy individual, Fractalkine and its receptor regulate migration of immune cells from the blood capillary wall into areas where the immune system is needed. Animal studies show that Fractalkine and its receptor are not essential for survival and that important immune functions remain intact indicating that inhibition of the Fractalkine signaling by a drug probably will be tolerated without significant adverse effects. Fractalkine and its receptor have been linked to the growth and proliferation of pancreatic, breast and prostate cancer. Also, cancer cells that have the Fractalkine receptor on their surface migrate towards nerve ends that have Fractalkine on their surface. Thus, cancer cells are led to surround and apply pressure on nerves and thereby cancer pain may arise. Another proposed mechanism for how Fractalkine and its receptor affect the development of tumors is that they contribute to the transformation of the body's macrophages from being a threat against the cancer (the M1 form) to supporting the cancer (the M2 form). This mechanism is also suggested as a predictive factor for responsiveness to the new immuno-oncology drugs that act through PD-1 and PD-L1 such as nivolumab, pembrolizumab and pidilizumab. During 2014 and 2015 studies have been published

demonstrating that the absence of Fractalkine in tumor cells is a significant marker for how successful the immuno-oncology treatment is expected to be (see e.g. the publication in Nature on November 27, 2014, Vol. 515, pp 563). In the light of these observations, there are good reasons to further study if inhibition of the Fractalkine signaling with KAN0440567 (AZD8797) has the potential to increase the proportion of patients responding to the new immuno-oncology drugs that act through PD-1 and PD-L1.

About the ROR project
ROR is a family of receptors, ROR1 and ROR2. The ROR receptors mediate signals for growth and survival. Originally ROR was linked to fetal development, but it is now known that they also contribute to cancer cell development and proliferation. Professor Håkan Mellstedt, Kancera´s co-founder and professor at the Karolinska Institute, and his colleagues have shown that Kancera´s ROR inhibitors have the ability to kill cells from tumors in pancreas and leukemia cells. Professor Mellstedt and his colleagues as well as independent researchers have shown that ROR is also active as a target in prostate, breast, skin and lung cancer as well as multiple myeloma.

Because ROR primarily generates a survival and growth signal to tumor cells but is inactive in healthy cells in adults, there are good prospects that a drug directed against ROR hit the tumor much harder than the surrounding healthy cells. Kancera and Professor Mellstedt have shown that inhibition of ROR leads to that cancer cells eliminate themselves by cellular suicide. Against this background, there are reasons to anticipate that a ROR-targeted drug is both safer and more effective than several chemotherapies currently used to treat cancer.

About Kancera AB
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 share holders with more than 500 shares were 7900 as of March 31, 2016. Remium Nordic AB is Kancera's Certified Adviser. Professor Carl-Henrik Heldin and Professor Håkan Mellstedt are board members and Kancera´s scientific advisers.

For further information, please contact,
Thomas Olin, CEO: +46 (0) 735 20 40 01
Address:
Kancera AB (publ)
Karolinska Institutet Science Park, Banvaktsvägen 22
SE 171 48 Solna

Please visit the company website; http://www.kancera.com