Press release May 3, 2016

Interim Report for Kancera AB (publ) Q1 2016

January 1 – March 31, 2016

The period January to March 2016 in brief

- R&D expenses for the period amounted to SEK 4.2m (SEK 4.2m).
- Operating income for the period amounted to SEK -5.1m (SEK 4.9m).
- Income after financial items for the period amounted to SEK -5.0m (SEK -4.9m).
- Earnings per share for the period were SEK -0.05 (SEK -0.05).
- Cash flow from operating activities for the period amounted to SEK -6.3m (SEK -5.1m).
- Equity as of March 31, 2016 amounted to SEK 17.0m (SEK 22.6m) or SEK 0.16 (SEK 0.22) per share. The equity/assets ratio as of March 31, 2016 was 70 percent (71 percent).
- Cash and cash equivalents as of March 31, 2016 amounted to SEK 12.1m (SEK 17.8m).

Significant events during the period

- Kancera has from the 1st of January 2016 extended the lease of the company's laboratories within the Karolinska Science Park for three years through an agreement with Humlegården Fastigheter.
- Kancera has provided an update of the small molecule patent portfolio.
  - A patent covering small molecule PFKFB3 inhibitors has been approved in the USA.
  - A patent application covering new chemical series in the HDAC6 project has been filed.
  - An international patent application covering ROR inhibitors has been strengthened by adding examples of additional highly potent ROR inhibitors.
- Kancera reported that the company has developed a new series of ROR inhibitors that show improved pharmaceutical properties which will allow preclinical studies of their effect on *e.g.* solid tumors. These results have prompted Kancera to concentrate the investments in the ROR project to small molecule inhibitors and terminate the product development of a ROR-based vaccine. Furthermore, Kancera reported results from the Fractalkine project showing that KAN0440567 after oral administration to mice effectively blocks the function of the Fractalkine receptor.
- Kancera announced that the company according to plan has received another payment of about SEK 2.8 million in January, 2016 from the EU for the A-PARADDISE project, which aims to develop drugs against parasitic diseases.

Significant events after the end of the reporting period

- Kancera announced that the Extraordinary General Meeting of Kancera AB approved the Board's decision of 6 April 2016 to issue new shares and warrants in the form of units. The decision includes a preferential rights issue of shares and warrants (units) ("New Share Issue"), which upon full subscription brings Kancera about SEK 52 million before issue expenses and with a full exercise of the warrants brings Kancera an additional SEK 52-62 million. The issue assets will be used for Kancera's drug development, clinical studies and the further development of the Company's capacity to commercialize products. The majority of Kancera's resources are now concentrated on taking at least one of Kancera's drug candidates in the ROR and Fractalkine projects to clinical trial for chronic lymphocytic leukemia and pancreatic cancer, respectively. In parallel, the Company intends to validate a broader use of the drug candidates from these projects in order to demonstrate their full commercial potential.
- Kancera reported that ROR inhibitors have been tested against human triple negative breast cancer transferred to zebra fish. The experiments showed that Kancera's small molecule ROR inhibitors are able to both reduce tumor size and metastases (spread) of this aggressive tumor form. Further, Kancera reported that the company´s

PFKFB3 inhibitors are active in the same model of triple negative breast cancer and that a patent application has been filed covering the discovery that PFKFB3 inhibitors enhance the effect of radiation treatment

- Kancera reported that the Company due to positive efficacy data in disease models of cancer and pain has decided to exercise the exclusive option to acquire the Fractalkine project. The acquisition will be carried out in connection with the completion of the ongoing transfer of results and know-how from Acturum and AstraZeneca to Kancera. Payment for the project to Acturum Life Science AB will be made into three steps by a total of 6 million shares, of which the first payment is due at the submission of the application for authorization of a clinical trial after an approval by Kancera´s shareholders. In parallel, the company intends to validate a broader use of the drug candidate (KAN0440567) in order to demonstrate its full commercial potential.

Statement from the CEO

In 2016, we have reported progress in the development of a new generation of ROR inhibitors with properties that we expect will enable effect against both lymphoma and solid tumors. A first study where human triple negative breast cancer was transplanted into zebra fish, shows that ROR inhibitors are able to both inhibit tumor growth and prevent spread. Further, we have been able to validate that Kancera PFKFB3 inhibitor is able to inhibit tumor growth in the same model.

On the patent side, we have completed an international patent application in the ROR project with the addition of approximately 100 selected potent compounds. Also, a patent application covering PFKFB3 substances has been approved in the United States and a new application covering the discovery that Kancera's PFKFB3 inhibitor counteract cancer by enhancing the effect of radiation treatment has been submitted. An additional patent application has been filed in the HDAC6 project covering a new class of active substances which together with the first patent application in the project, broadens the protection around our unique selective and effective compounds against myeloma.

Furthermore, due to positive efficacy data in disease models of cancer and pain, the Kancera Board has decided to exercise the exclusive option to acquire the Fractalkine project.

The goal for the next 18-24 months is to take at least one of Kancera's drug candidates from the Fractalkine and ROR projects to clinical trials and thus first clinical use (chronic lymphocytic leukemia/ pancreas cancer). In parallel, we also complete the evaluation of a broader use of the drug candidates from these projects in order to reduce the risk in the product development and to identify full commercial potential of the projects. The operational objectives also include delivering drug candidates from the HDAC6 and PFKFB3 projects.

Given these ambitious goals, Kancera's Board supported by the decision of the Extraordinary General Meeting on April 22, 2016, decided to launch a new share issue. The decision includes a preferential rights issue of shares and warrants (units) which upon full subscription brings Kancera about SEK 52 million before issue costs.

With eight patent families, which together make up the intellectual property protection for four small-molecule cancer projects, we expect that Kancera has good opportunities to translate scientific results into commercially attractive new drugs against cancer. However, the development of new drugs is biologically and technically risky and challenged by international competition.

This share issue aims to push Kancera's cancer project from a promising development phase to completion in order to be tested in cancer patients. I am convinced that we will succeed in this work.

Thomas Olin

CEO Kancera

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's operations are based in the Karolinska Institutet Science Park in Stockholm and the company employs around 13 people. The Kancera shares are traded on NASDAQ OMX First North and the number of shareholders was around 7300 as of December 16, 2016. Remium Nordic AB is Kancera's Certified Adviser. Professor Carl-Henrik Heldin and Professor Håkan Mellstedt are board members and Kancera´s scientific advisors.

Kancera's history
In 2006, Pharmacia's and Biovitrum's unit for the development of drug candidates was spun-out to create iNovacia AB. In 2008, iNovacia started the development of the ROR project in collaboration with the Karolinska Institute. In May 2010, Kancera AB was formed by scientists from Cancer Center Karolinska, iNovacia AB and a group of private investors through capital contributions and two developed drug projects focusing on cancer: the ROR project and the PFKFB3 project, the latter had been initiated by Biovitrum AB. NASDAQ OMX approved Kancera's listing on First North with the first day of trading being February 25, 2011. In March 2013 Kancera acquired a complete drug development laboratory from its former subsidiary iNovacia AB and the drug development is since then performed within Kancera AB at the Karolinska Institutet Science Park, Stockholm.

Financial development, summary

Financial development, summary

SEK 000´s (if otherwise not specified)

	1 Jan-31 Mar 2016	1 Jan-31 Mar 2015	1 Jan-31 Dec 2015
Net turnover	114	10	282
Contributions from the EU/Vinnova	-4 249	-4 164	-20 355
R&D expenses	-5 055	-4 937	-19 686
Operating Income	-5 035	-4 918	-19 612
Income after financial items	-5 035	-4 918	-19 612
Net income	-6 284	-5 133	-20 658
Cash-flow from operating activities	-0,05	-0,05	-0,19
Earnings per share, before and after dilution			
Cash on hand at closing date	12 083	17 809	15 567
Key ratios	neg	neg	neg
Return on equity, %	neg	neg	neg
Return on capital employed, %	70%	71%	80%
No. of employees	10	10	10
Earnings per share, before dilution	-0,05	-0,05	-0,19
Earnings per share, after dilution	-0,05	-0,05	-0,19
Equity by share, kr	0,16	0,22	0,21
Cash-Flow by share, kr	-0,03	-0,05	-0,07

Comments on the financial development

Net sales
Kancera´s activities have mainly covered internal drug development projects alongside smaller consultancy projects which raised net sales during the period of SEK 0.14m (SEK 0.0m). The company also receives financial support from the EU project A-PARADDISE where the support is offset against incurred costs for the period amounting to SEK 0.9m of consumables, performed months of work plus 60% overhead on the sum of these costs as was summarized in a midterm report. The financial support from EU covers 75% of the project costs plus 60% overhead.

Expenses
Expenses in the first quarter amounted to SEK 5.2m (SEK 4.9m), which breaks down into costs of services sold of SEK 0.0m (SEK 0.0m), research and development expenses of SEK 4.2m (SEK 4.2m) and other sales and administrative expenses of SEK 1.0m (SEK 0.7m).

Earnings
Income after financial items for the first quarter amounted to SEK -5.0m (SEK -4.9m).
The cost of the option program for employees and other senior executives during the first quarter amounted to SEK 115,000 (SEK 219,000).

Cash flow and liquidity
Cash flow amounted to SEK -3.5m (SEK -5.2m) in the first quarter. Cash flow from operating activities for the first quarter amounted to SEK -6.3m (SEK -5.1m). Cash flow from financing activities amounted to SEK 2.8m (SEK 0.0m).

In the first quarter 2016, Kancera was awarded a grant of SEK 2.8m from the European Union's 7th Framework Program for the A-PARADDISE project which targets parasitic diseases. Ongoing work for the period amounting to SEK 2.2m is attributable to the work performed within the framework of the EU project A-PARADDISE. The grant has been accounted for as a current liability until approval of the final report which is expected in the third quarter 2017. Thereafter the work will be registered as revenue and settled against accumulated costs.

Cash and cash equivalents as of March 31, 2015 totaled SEK 12.1m (SEK 17.8m).

Investments
Investments in fixed assets in the first quarter totaled SEK 0.0m (SEK 0.0m).

Investments in intangible assets in the first quarter totaled SEK 0.0m (SEK 0.0m).

The company continuously invests in research projects that increase the company's technology knowledge, and where also a patent application covering the technology can be included. In the accounts these investments including patent costs, are entered as costs since the time of activation for projects is based on the time when the project will be commercialized and that time point has not yet occurred. R & D costs, which therefore are entered as R & D costs, amounted to SEK 4.2m (SEK 4.2m) for the first quarter.

Equity and share data

Total equity as of March 31, 2016 was SEK 17.0m (SEK 22.6m).

Share capital as of March 31, 2016 amounted to SEK 8 660 446,97 spread over 103 925 364 shares with a quotient value (rounded off) of SEK 0.0833 per share.

Earnings per share for the fourth quarter, based on a weighted average of the number of outstanding shares, were SEK -0.05 (SEK -0.05).

The equity/assets ratio as of March 31, 2016 was 70 percent (71 percent). Total equity per share was SEK 0.16 (SEK 0.22) based on total equity divided with the number of shares on the balance sheet day at the end of the quarter.

Deficits for tax purposes
Kancera's present operations are expected to initially result in negative earnings and deficits for tax purposes. There are no sufficiently convincing evidence at present that tax surpluses will exist in the future that may justify capitalization of the value of the deficit, and no deferred tax claim has therefore been reported. In the event a drug candidate is sold, profits will be reported which may be offset for tax purposes against the deficits. This signifies a low tax burden for the company when a project is sold. The determined tax deficits amount to SEK 94.7m.

Personnel

Kancera AB had 13 full time employees (10) as of March 31, 2016 of which 9 are men and 4 are women.

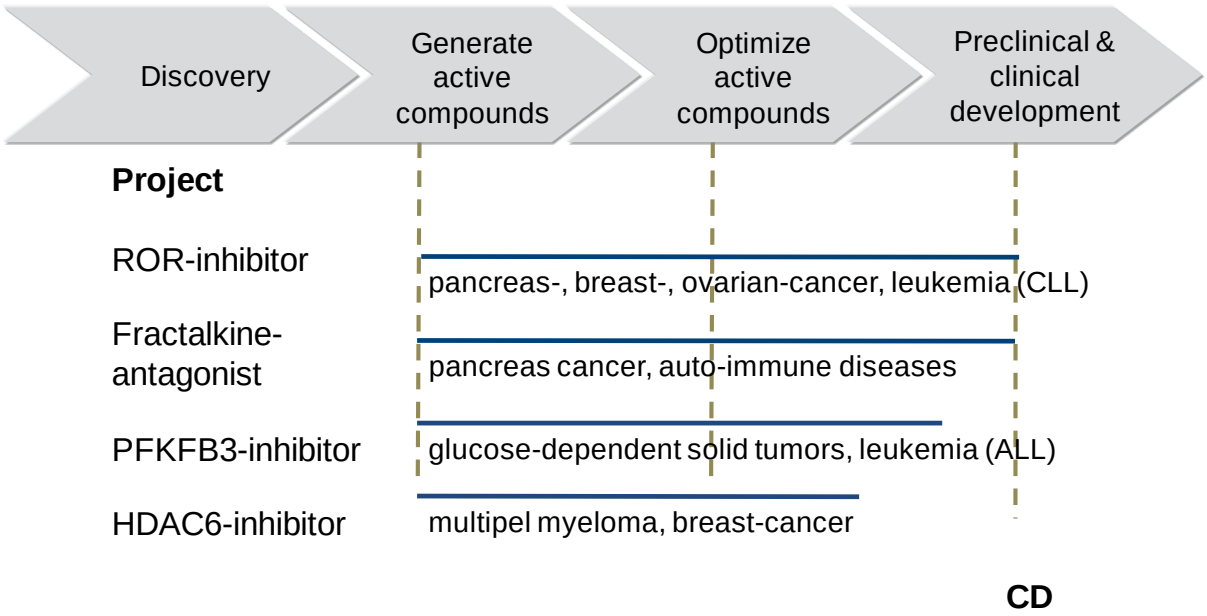
Pharmaceutical Development

Kancera develops cancer drugs, starting with a new treatment concept and ending with a patent-pending drug candidate that is offered for sale to larger pharmaceutical and biotech companies before it has reached the clinical phase in the product development chain.

The company has five drug development projects in the portfolio.

- ***Small molecule ROR inhibitors*** that reprogram the cancer cells so that they destroy themselves. In the laboratory, the ROR technology has been shown to work in both solid tumors and leukemia.
- ***Small-molecule antagonists of the Fractalkine receptor CX3CR1*** that control cancer cells and the immune system to counter tumor growth and spread as well as to counter cancer pain and tissue damages and pain during inflammation.
- ***Small molecule PFKFB3 inhibitors*** that strangle the energy supply from glucose to solid tumors and decrease the ability of the cancer cells to repair their DNA, which together may increase the tumor's sensitivity to other cancer therapies.
- ***Small molecule HDAC6 inhibitors*** that primarily aim to neutralize blood cancer (primarily myeloma) by decreasing the cancer cell's ability to move and support the patient's immune system (and thereby causing death of tumor cells).
- ***Small molecule inhibitors of epigenetic processes in parasites*** to develop new treatments against *e.g.* malaria and schistosomiasis (snail fever)

Figure 1. Kancera´s cancer project portfolio



In the fourth quarter, the product development in the ROR project has delivered KAN0439834 as a first drug candidate with the potential to treat refractory solid cancers (as seen in laboratory studies) as well as blood cancers (as seen in completed animal studies). This means that initial discussions with potential commercial partners have been initiated.

See page 17 for more information on the market potential for Kancera's products.

In parallel, KAN0439834 will be tested in new efficacy and safety models. Kancera's research shows that there is an

opportunity to create additional value in the project for the small-molecule ROR inhibitors why new formulations of KAN0439834 and analogues of this substance have been successfully developed. However, the road towards commercialization is still risky since increasingly advanced safety- and efficacy studies are performed in order to clarify the product's commercial value and to meet the requirements for clinical trials. A successful commercialization may mean that the risk and cost of these studies are shared with a partner and that Kancera obtains a stepwise compensation at signing of the agreement and when the project reaches milestones. However, Kancera has not established a timeline for the commercialization of the ROR project.

Kancera has entered into an agreement with Acturum Life Science AB in order to evaluate and further develop the unique Fractalkine receptor antagonist AZD8797. The agreement with Acturum Life Science gives Kancera right to evaluate AZD8797 in preclinical studies and then to acquire the project. This agreement entails no expenses for Kancera apart from investments in the patent portfolio and in the scientific evaluation.

If Kancera chooses to acquire the Fractalkine project, following the preclinical evaluation phase, the total payment to Acturum will consist of 6 million Kancera shares divided into three tranches, which are due at pre-defined success-milestones. Accordingly, the two companies share the risk in the product development through the first study in man. Kancera intends to apply for orphan drug designation, covering the Fractalkine inhibitor, in order to ensure at least 10 years of exclusivity on the market in Europe and 7 years in the United States.

The main part of the company´s resources is invested in the ROR, Fractalkine and the HDAC6 projects, while the epigenetically directed anti-parasite project is mainly financed by the EU. For the EU-project, Kancera has been awarded funding of € 950,000 for research and product development. This funding covers 75% of the project's personnel and material costs. In addition, EU covers overhead costs corresponding to 60% of the project's personnel and material costs, which means that the project also bears a part of Kancera´s administrative costs.

The company's product development of epigenetically acting drugs against parasites also makes it possible for Kancera to efficiciently develop epigenetically acting drugs against cancer, including HDAC6 inhibitors, since a similar technical expertise and capacity are needed for both epigenetic projects. Currently, Kancera receives a grant from Vinnova totaling SEK 2m over two years (from July 2015) for the further development of HDAC6 inhibitors. The HDAC6 project is within 9-15 months from selection of a candidate drug.

Kancera has developed inhibitors of PFKFB3 which in the laboratory have been shown to potentiate other cancer treatments and single-handedly slow the growth of pancreatic cancer in an experimental model. The PFKFB3 project is now developed in collaboration with Professor Thomas Helleday´s research group at the Science for Life Laboratory at the Karolinska Institute. The goal of this collaboration is to identify how Kancera´s PFKFB3 inhibitors most effectively can be combined with other drugs and radiation to achieve the best clinical outcome. Based on the results from this research Kancera will decide how the further optimization of the company´s PFKFB3 inhibitors towards the selection of a candidate drug is to be done. This product development depends on that adequate funding for the project is secured. The PFKFB3 project has been valued to SEK 3m in the balance sheet which was the original purchase value of the project. It is the opinion of the Board that the value, based on the currently known results of Kancera´s research, can be defended on the basis of currently prevailing prices of comparable projects and the potential to further develop the project in the future.

R & D costs amounted to SEK 4.2m (SEK 4.2m) for the first quarter 2016 which has been recognized as costs in its entirety.

ROR technology – candidate drug is developed for the treatment of leukemia and solid tumors

Product profile – ROR1 inhibitor

Property	Summary of "Target Product Profile" (TPP)
Primary indication	Chronic lymphocytic leukemia, other ROR1 driven hematologic cancer forms.
Secondary indication	Pancreas-, breast-, and ovarian cancer
Treatment regime	Mono-therapy or in combination with other drugs, one to two times per day.
Administration	Peroral/IV/SC
Biomarker	ROR1 antibody recognizing active ROR1.
Product differentiation	*Effect:* Induction of cancer selective cytotoxicity in blood, bone marrow and lymph as well as in solid tumor provides opportunities for complete remission. *Safety:* ROR1 is mainly found in cancer cells why a ROR1 targeted treatment should give a lower level of side effects compared to broad-acting drugs. *New mechanism of action:* Adds effect to existing drugs.

When healthy cells suffer genetic damage that is not repaired, a cellular suicide is normally initiated in order to eliminate the threat that these injuries constitute for the surrounding healthy parts of the body. Cancer cells, by contrast, have developed a resistance to signals that should lead to cellular suicide when serious injuries occur in the genome. In fact, the genomic errors in the cancer cells are a prerequisite for the aggressive and life threatening characteristics of the cancer.

Kancera has shown that if the growth factor ROR-1 is present in the tumor then anti-ROR drugs can be developed that reprogram cancer cells to destroy themselves through cellular suicide. This fact is the basis for the development of Kancera's drug candidate.

Kanceras first drug candidate in the ROR project is directed against lymphocytic leukemia.

After decades of stagnation in the development of drugs against this disease, several new drugs have been approved such as Imbruvica from Pharmacyclics/J&J/Abbvie and Zydelig from Gilead. The introduction of these drugs has brought great progress especially in the treatment of patients with advanced and refractory disease. For these patients, the disease can now be stabilized for an additional two to three years, compared with the traditional treatment. The clinical experience shows that significant medical need persists despite these advances.

Thus, a drug against chronic lymphatic leukemia which causes a long term retreat of the disease (give complete remission) without posing a threat to the patient's organs that are function normally is still missing. Kancera's inhibitors of the cancer-selective growth factor ROR1 has the potential to become such a drug since the company and independent researchers have demonstrated that blocking of ROR1 leads cancer cells, even the most refractory, to destroy themselves. Also, ROR1 is selectively found in cancer cells and not in the surrounding healthy tissue and a drug that acts with a high selectivity against ROR1 has the potential to give the patient possibilities to live a normal life with limited side effects of the treatment.

Kancera's ROR inhibitors act quickly and efficiently to treatment resistant cells from patients with refractory chronic lymphocytic leukemia. This has been demonstrated in the laboratory against isolated cancer cells and in animal studies in which human disease has been recreated in mice. Preliminary animal studies support that ROR inhibitors are well tolerated by the animals which has been studied in ten selected organs from treated animals. These studies on chronic lymphocytic leukemia were completed in early 2015. Since then Kancera's goal has been to develop a new generation of ROR inhibitors that through an extended residence time in the blood circulation is expected to provide efficacy against several cancers. Independent research groups have demonstrated that ROR1 is involved in blood cancer forms such as

acute myeloid leukemia (AML) and multiple myeloma (MM) as well as certain refractory solid cancers like pancreatic cancer, ovarian cancer and triple negative breast cancer (an especially intractable form of breast cancer that lacks three common targets of cancer drugs, hence “triple negative”).

A first goal in this work has been achieved since a second generation of ROR inhibitors have been developed that exhibit an improved effect against cancer cells (lower dose required to achieve the same killing effect). In addition, these ROR inhibitors are maintained in the blood circulation for a time long enough to have the potential to be efficient against lymphoma and solid tumors. Recent results show that this new generation of ROR inhibitors are effective in a first disease model for solid tumor in which triple negative breast cancer in humans has been implanted and treated in a zebra fish. In this study, the ROR inhibitors reduce both tumor size and metastasis (spread).

The assessment is that Kancera is a world leader in the development of synthetic drugs against the cancer-specific growth factor ROR. If ROR1 is blocked, then e.g. leukemia cells are reprogrammed to destroy themselves. There are competing groups that develop antibodies and modified immune cells directed against ROR1. In contrast to these, Kancera’s ROR inhibitors have the ability to penetrate into cancer cells and kill these even if ROR1 is not present on the surface of the cancer cells. Neither antibodies nor modified immune cells are able to do this.

In February 2015 Kancera reported that a patent application (EP15153394.0) had been registered containing examples of approximately 100 small-molecule ROR inhibitors, including the drug candidate KAN0439834. When this application entered the international stage, the application was strengthened by additional examples of approximately 300 substances. The application will then comprise substances which have been found to have more than 20 times higher potency than KAN0439834 against cancer cells from CLL patients.

Events during the period

Kancera reported that a patent application (EP15153394.0) was registered containing examples of approximately 100 small-molecule ROR inhibitors, including the drug candidate KAN0439834. This application has now entered the international stage and Kancera has strengthened the application by adding examples of a further approximately 300 substances, including substances that have shown to be more than 20 times more potent than KAN0439834 against cancer cells from CLL patients. Kancera also reported that the new series of compounds a) shows higher potency against leukemia cells and affects healthy blood cells to a lower degree, b) shows a higher metabolic stability in liver cells from both mouse and human, and c) remains in circulation during four times longer time compared to KAN0439834. Both KAN0439834 and compounds in the new series shows good oral availability indicating that both can be developed to be given in the form of pills.

The results of the evaluation of peptide sequences for vaccine development have confirmed that they do not generate an immune response that is effective enough against leukemia cells in comparison to that achieved with Kancera’s small molecules. Against this background, Kancera has now chosen to terminate the vaccine product development and bring back the vaccine project to academic research. Thus, Kancera will concentrate the ROR-project investments to small molecule inhibitors.

Events after the end of the period

Kancera has previously reported that a new generation of ROR inhibitors (*e.g.* the compound KAN0440550) have been developed and these show a high level of efficacy and selectivity against cancer cells compared with healthy cells at the same time as they reach a concentration in the blood after oral administration that is expected to be sufficient to achieve efficacy against several cancers such as lymphoma and solid tumors. Kancera has now examined the effect of a representative of this new generation of ROR inhibitors against solid tumor in a disease model based on a human triple negative breast cancer implanted and studied in zebra fish. The results show that a three day treatment with a ROR inhibitor results in both reduced tumor growth and reduced metastasis (spread). The study also shows that KAN0440550 is well tolerated at the effective concentration of the compound. KAN0440550 and related ROR inhibitors are now being tested against solid cancers and lymphomas in preclinical disease models for the selection of a candidate drug complementary to KAN0439834 which is a compound that is more suited for effect against leukemia.

Figure 2. The ROR1 inhibitor KAN0440550 decreases metastasizing of human triple negative breast cancer transplanted into zebra fish.

a)

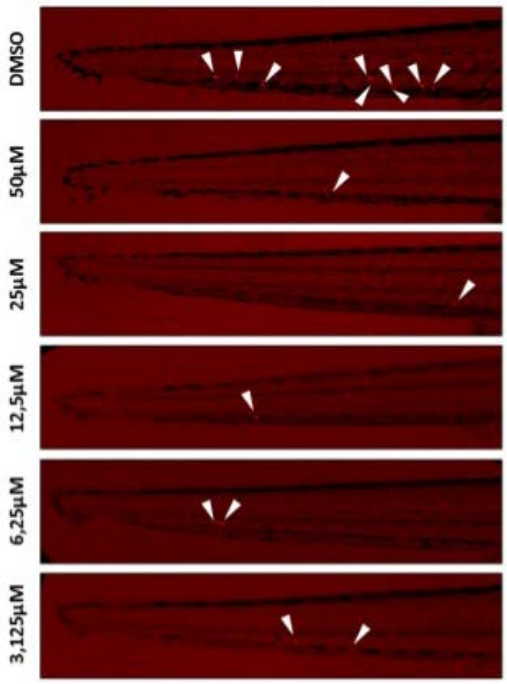


Figure 2a) shows microscopy images of zebra fish embryos transplanted with a triple negative breast cancer tumor. Three days following the tumor transplantation it is determined how many tumor cells that have metastasized (spread) into the body of the zebra fish carrying the transplanted human tumor. Each arrow denotes a cancer cell. The image marked DMSO represents a control group not treated with the active substance. The images below are representative of the animals that have been treated with KAN440550 in decreasing concentration of 50 µM to 3.125 µM. All the animals were exposed to DMSO (placebo) or KAN0440550 during three days before the degree of metastasis were determined.

b)

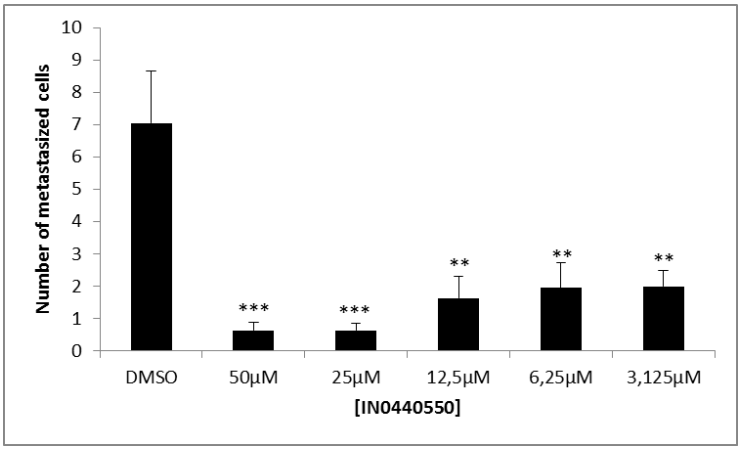


Figure 2b shows a bar graph of the results obtained in the study described in Figure 2a. The degree of significant effect calculated using the statistical method T-test are marked with ** or *** each of which represents a statistically significant difference.

The Fractalkine project – controls the immune system in cancer and alleviates severe pain

Product profile – Fractalkine-antagonist

Property	Summary of "Target Product Profile" (TPP)
Primary indication	Pancreas cancer and metastasizing breast cancer.
Secondary indication	Cancer pain (pain related to pancreas cancer, pain caused by metastases in the skeleton, and pain caused by chemotherapy treatment.
Treatment regime	Mono-therapy or in combination with other drugs, one to two times per day.
Administration	Peroral
Biomarker	Presence of receptor and/or ligand in biopsy or blood (circulating tumor cells).
Product differentiation	*Effect:* a) Increased survival, b) Prevention of renewed metastasis (so-called re-seeding), c) Efficient pain relief when opiates are not efficient. *Safety:* Low level of mechanism related side effects is expected. Therapeutic window compared to limiting kidney effects by metabolites is under investigation. *New mechanism of action:* Expected to be the first small molecule antagonist of the Fractalkine receptor.

Based on new research results that support that this antagonist may be of central importance in different cancer forms, Kancera performs studies to learn how efficiently the Fractalkine receptor antagonist AZD8797 stops tumor growth and relieves severe pain.

The project has now generated positive results in multiple disease models of cancer and pain. The results show desired treatment effects that are important for Kancera´s further product development and commercialization of the project. The results will be published at a later date, in collaboration with the academic partners involved. In light of the positive results Kancera Board has decided to acquire the Fractalkine project.

In 2015, Kancera signed an agreement with Acturum Life Science AB in order to evaluate and further develop the unique Fractalkine receptor antagonist AZD8797 (KAN0440567).The acquisition of the project will be carried out in connection with the completion of the ongoing transfer of results and know-how. For the Fractalkine project Acturum Life Science AB is paid in total 6 million Kancera shares divided into three steps which can only be achieved if the project is successfully developed. This payment model means that the two companies share the risk in product development up to when the first study has been conducted in humans. Kancera intends to strengthen the protection of the Fractalkine antagonist through an application for registration as an orphan drug ("Orphan Drug Designation") with the goal of ensuring at least 10 years of market exclusivity in Europe and 7 years in the United States.

Independent research groups have studied Fractalkine signaling and its biological and clinical role have reported data supporting that an antagonist of the Fractalkine-receptor may:

- facilitate for the immune system to attack the cancer
- prevent cancer cells from spreading to nerves and bone marrow
- reduce cancer pain caused by the tumor itself and side effects of chemotherapy such as paclitaxel

The focus of the ongoing R & D work in the Fractalkine project at Kancera is directed against pancreatic cancer. Although pancreatic cancer is a relatively rare disease (300 000 new cases in 2012), the disease is the fifth leading cause of cancer deaths in Europe and the fourth in the United States. Thus, pancreatic cancer is the cancer disease

with the worst prognosis why the medical need for new drugs that prolong or save the life of these patients is a major and urgent challenge for the whole society. Close to 80% of cancer patients today has a disease that has advanced too far for allowing surgery to be performed. In that phase of the disease there is currently no drug therapy providing a long-lasting effect.

Pancreatic cancer is characterized by that the tumor is surrounded and infiltrated by fibrous tissue and immune cells that have been made passive by the tumor, including a type of immune cells that have migrated to the tumor from the bone marrow and suppresses the anti-tumor immune response. This type of suppressing immune cells has probably migrated to the tumor through nearby blood vessels by binding to Fractalkine. The complex composition of cells that make up the tumor is believed to contribute to the drug resistance of pancreatic cancer.

Pancreatic cancer also represents a threat to other tissues in the body such as the surrounding nerves, liver and lung. Fractalkine that is released from surrounding nerves sends a signal to the cancer cells that make them migrate from the primary tumor to instead surround nerves. This spread contributes to both the recurrence of the disease after completion of treatment, and to severe pain that about 50% of these cancer patients experience. Cancer pain, which significantly reduces the quality of life for these seriously ill patients, is treated mainly with opiates today. The analgesic effect of opiates is gradually decreased and they are associated with undesirable side effects, *e.g.* on oxygenation. In addition to the pain that the disease itself causes, also the best chemotherapy Abraxane (containing paclitaxel) leads to nerve damage that causes pain in 70% of patients and severe pain in 10%. Pain can also prevent the drug to be administered in a high enough dose to produce a good effect. The pain caused by Paclitaxel has in part been shown to be mediated by an increasing amount of the CX3CR1 receptor that sends out signals when it binds to Fractalkine (Neurochemistry Research 2016).

KAN0440567 is a drug candidate that has undergone toxicological evaluation according to GLP and with a production method that has been proven in kg scale. The next step in the drug development will be to evaluate whether a sufficient therapeutic effect safely can be achieved for treatment of pancreatic cancer and, based on these results, prepare for a clinical phase I study. In parallel, preclinical studies will continue in order to better understand the mechanism of action and possibilities to broaden the indication area outside pancreas cancer.

Although the drug candidate KAN0440567 was originally developed by AstraZeneca more than 10 years ago, the compound is still the leading small molecule antagonist of the Fractalkine receptor CX3CR1. There are other projects that develop small molecule drug candidates against CX3CR1. Kerberos Biopharma (USA) develops small molecule antagonists of the CX3CR1 and their candidate JMS-17-2 has shown interesting effects against breast cancer metastasis in animal models (AACR; Cancer Res 2015; 75 (15 Suppl): Abstract No.4116. doi: 10.1158/1538-7445. AM2015-4116). However, public information indicates that JMS 17-2 does not have the desired pharmaceutical properties, which is supported by the fact that the compound was administered to the animal model by injection in the abdomen. The pharmaceutical company Eisai Co. Ltd. develops a monoclonal antibody that captures Fractalkine and makes Fractalkine unavailable to its receptor CX3CR1. This antibody is currently being studied in clinical phase I for rheumatoid arthritis (Clinicaltrials.gov).

Our assessment is that a small molecule antagonist of the Fractalkine receptor CX3CR1 has the potential to be a significantly stronger anti-cancer drug compared to an antibody that captures Fractalkine. This assessment is based on the fact that it is more difficult for antibodies to penetrate and influence a solid tumor compared to a small molecule compound and that CX3CR1 may affect cancer and immune cells independently of Fractalkine. A third aspect is that a small molecule compound usually is cheaper to produce than an antibody which may lead to a broader use of the small molecule than the antibody if it otherwise meets the requirements for efficacy and safety.

Kancera has reported that a network of leading cancer and pain researcher has been established as a collaboration project to evaluate the drug candidate KAN0440567 (AZD8797) in an advanced animal model, which closely resembles the human form of pancreatic cancer.

Events during the period

Kancera reported results from a first of a series of planned studies that will examine whether Kancera's Fractalkine receptor antagonist can become an anticancer drug. In this collaborative study with Professor Mia Phillipson (Department of Medical Cell Biology, Uppsala University), KAN0440567 was tested in transgenic mice lacking CX3CR1, *i.e.* the target for KAN0440567. The results show that KAN0440567 selectively and effectively blocks the effect of Fractalkine signaling on *e.g.* the immune cells called macrophages. Independent research has shown that Fractalkine signaling in cancer contributes to the reprogramming of macrophages from being a threat to the tumor to aid the tumor. Thus, it may be desirable to block the effect of the Fractalkine signaling in cancer.

Events after the end of the period

Kancera has previously announced that the company owns an option to acquire exclusive rights to the Fractalkine project (excluding the therapeutic area respiratiory diseases) during an evaluation period of 24 months (from September 2015). The project has now generated positive results in multiple disease models of cancer and pain. The results show desired treatment effects that are important for Kancera´s further product development and commercialization of the project. The results will be published at a later date, in collaboration with the academic partners involved. In light of the positive results Kancera Board has decided to acquire the Fractalkine project. The acquisition will be carried out in connection with the completion of the ongoing transfer of results and know-how from Acturum and AstraZeneca to Kancera. Payment for the project to Acturum Life Science AB will be made into three steps by a total of 6,000,000 shares, of which the first payment is due at the submission of the application for authorization of a clinical trial after an approval by Kancera´s shareholders. In parallel, the company intends to validate a broader use of the drug candidate (KAN0440567) in order to demonstrate its full commercial potential.

The PFKFB3 project – a candidate that blocks glycolysis in solid tumors

Product profile – PFKFB3-inhibitor

Property	Summary of "Target Product Profile" (TPP)
Primary indication	Solid tumors with high glucose consumption such as pancreas- and colorectal cancer.
Secondary indication	Acute lymphocytic leukemia
Treatment regime	Mono-therapy or in combination with other drugs, one to two times per day.
Administration	Peroral/IV/SC
Biomarker	PET-scanning with 2FDG to identify glucose consuming tumors.
Product differentiation	*Effect:* Synergistic effect with radiation or DNA damaging chemotherapy. *Safety:* PFKFB3 is mainly found in hypoxic tissue and in cancer cells why a PFKFB3 selective drug can be expected to give a low level of side effects. *New mechanism of action:* Adds effect to existing drugs.

The project aims to develop PFKFB3 enzyme inhibitors to strangulate the energy metabolism in cancer cells, thereby rendering the cancer cells more sensitive to chemotherapy and radiotherapy. Kancera has, together with Professor Thomas Helleday and his research group at Karolinska Institutet, made a surprising discovery that shows how Kancera's PFKFB3 inhibitor enters the cancer cell's nucleus and enhances the effect of a recently given radiation dose. This discovery has been claimed in a US patent application owned by Kancera.

The background to this invention is the unique metabolism of cancer. Cancer cells consume *e.g*. up to 200 times more sugar compared to a healthy cells. This fact is already used in clinical practice to detect tumors in patients with a so called PET camera. In recent years, both academic researchers and pharmaceutical companies have paid attention to that the altered metabolism contributes to that cancer cells can survive with very little oxygen available, creating an environment where aggressive cancer cells develop. By strangulating the special metabolism that cancer cells need to resist both chemotherapy and radiation, the tumor becomes weakened. Healthy cells, on the other hand, are not affected by the treatment in the same way since they have a different metabolism than the cancer cells. Thus, a new strategy for fighting cancer has emerged.

Kancera's drug discovery project directed against cancer metabolism targets PFKFB3 which is an enzyme acting like an accelerator in the metabolism of sugar to energy. Kancera has already developed a compound that inhibits PFKFB3 and shown that this slows the growth of pancreatic cancer in an animal study. Although this cancer is very difficult to

treat, the assessment was that the effect of the PFKFB3 inhibitor was not strong enough to proceed with the selected compound as a mono-therapy. Instead Kancera started a collaboration with Professor Thomas Helleday's group at Karolinska Institutet to better understand how PFKFB3 inhibitors are to be used to achieve maximum effect against cancer.

The collaboration with Professor Helleday and Karolinska Institutet has now led to the discovery that PFKFB3 not only regulates the metabolism of sugar to energy but also migrates into the cancer cell's nucleus where PFKFB3 contributes to the cell's ability to repair genetic material (DNA). As can be expected from this discovery, Kancera's patent pending compound KAN0438757 increases the damage that radiation causes cancer cells. These results, together with the knowledge that patients suffering from radiation resistant acute leukemia (ALL) have an elevated level of PFKFB3, support that Kancera continues the work to develop a drug candidate against PFKFB3 and test it in combination with radiation treatment to combat resistant cancers.

Radiation therapy is one of the most effective methods to treat cancer. In total, about 50% of cancer patients are treated with radiation. However, radiation therapy is challenged by the fact that cancer cells exhibit resistance and due to the adverse side effects of the radiation itself. To improve the therapeutic effect and reduce the side effects it is desirable to make cancer cells more sensitive to radiation. One of the most attractive ways to achieve this is to make it difficult for cancer cells to repair the genetic damage produced by radiation preferably without hindering healthy cells to repair their DNA. Healthy cells are exposed to external factors that cause single-strand DNA breaks, e.g. by sunlight. However, gamma radiation is stronger and causes, in addition to single-strand breaks, also double-strand breaks in the DNA. A drug that blocks repair of double-strand breaks but allows the repair of single-strand breaks could thus do more damage to cancer cells exposed to gamma radiation (and chemotherapy) compared to the healthy cell that has been exposed to sunlight. The discovery by Kancera together with Prof. Thomas Helleday's research group points to that Kancera's PFKFB3 inhibitor meets the requirements of a therapy that increases sensitivity to gamma radiation in a cancer-selective manner.

There are various possibilities to attack the metabolism of the cancer, and inhibition of PFKFB3 has attracted several pharmaceutical companies. However, the development of drugs against PFKFB3 is technically challenging, which is likely to have contributed to that no drug against this enzyme has been tested in clinical efficacy studies (Phase 2) yet. This also means that the area is not yet dominated by any company. Examples of companies working with PFKFB3 are AstraZeneca and the American biotech company Advanced Cancer Therapeutics. In comparison with AstraZeneca's compounds, Kancera's PFKFB3 inhibitors may have the advantage to be more cancer-selective due to another mechanism of action, as compared to the compounds that AstraZeneca have published. Regarding the PFKFB3 inhibitors from Advanced Cancers Therapeutics, Kancera has not been able to demonstrate that they have the desired effect on DNA repair which Kancera's PFKFB3 inhibitor shows.

Kancera has three patent applications (one granted in the US) in the PFKFB3 project. Two patent applications cover new PFKFB3 inhibitors, registered in 2010 and 2012, and one patent application cover the combination therapy with PFKFB3 inhibitors and radiation.

Events during the period

Kancera reported that a patent for small molecule inhibitors of PFKFB3 has been approved in the USA.

Events after the end of the period

Kancera´s PFKFB3 inhibitor (KAN0438757) has previously been shown to be effective against triple negative breast cancer. An additional zebrafish study verified the effect of Kancera's PFKFB3 inhibitor in monotherapy (treatment with substance without combining it with another therapy). Kancera's PFKFB3 inhibitor was well tolerated at the active concentration of the compound. Kancera has previously reported a discovery, made together with Professor Thomas Helleday's research team at the Karolinska Institute, that treatment with Kancera´s PFKFB3 inhibitor enhances the effect of radiation on cancer cells in laboratory studies. This discovery has now been claimed in the United States by complementing the company's earlier patent application which protects the PFKFB3-inhibiting compounds. Kancera is the owner also of this new patent application.

The HDAC6 project - a candidate acting against cancer by controlling the cancer cell's ability to spread

Product profile – HDAC6-inhibitor

Property	Summary of "Target Product Profile" (TPP)
Primary indication	Multipel myeloma
Secondary indication	Breast cancer
Treatment regime	Mono-therapy or in combination with other drugs, one to two times per day.
Administration	Peroral/IV/SC
Biomarker	Remains to be identified in biopsy and circulating tumor cells.
Product differentiation	*Effekt:* a) an increased effect on the ability of the cancer cell to divide. b) under investigation: immuno-stimulating effect against cancer by small molecule. *Safety:* Due to high selectivity for HDAC6, a lower degree of gastro-intestinal effects is expected compared to the less selective HDAC inhibitors that are currently in clinical development. *New mechanism of action:* Combination of effect on HDAC6 and Kancera's "Target 2".

HDAC6 is an enzyme that controls the interior cell fibers, a type of cell skeleton, functions and thereby how cells can move in the body. Active HDAC6 affects the tumor's ability to invade surrounding healthy tissue and form metastases. HDAC6 has also been shown to be a useful marker providing an indication on how difficult the cancer is to treat. Taken together, these observations point to that HDAC6 contributes to cell changes that lead to tumor formation and invasion of tumor cells into healthy tissue making HDAC6 an attractive target for the development of new effective drugs against cancer.

Recent research also shows that HDAC6 inhibitors can help the patient's immune system to recognize and attack cancer cells. The HDAC6 inhibitors relieve a molecular brake, called PD-L1, which is applied on the immune cells by the cancer. Thus, HDAC6 inhibitors may constitute an effective small molecule replacement of the new PD-L1 antibodies which are in clinical use today, with the advantages that the small molecule drug can be taken in pill form instead of via syringe and will be cheaper to produce, which can make the drug available to more patients. However, it remains for Kancera to show how effectively the company's compounds can counteract the cancer brake on the patient's immune system.

There are currently five HDAC inhibitors on the market for the treatment of various forms of T-cell lymphomas, AML and multiple myeloma. These inhibitors are active against several members of the HDAC family of enzymes leading to severe side effects on e.g. the gastrointestinal tract. Also, the risk of significant negative impact on cardiac function is considered to be high. Kancera's discovery of selective HDAC6 inhibitors may provide a solution to how the health care can take advantage of the HDAC inhibitor's effect on cancer without causing the patient severe side effects.

Kancera's HDAC6 inhibitors are covered by two patent applications submitted in 2014 and 2015. These compounds are more potent and selective in vitro against cancer cells from multiple myeloma than the furthest developed competing HDAC6 inhibitor ACY-1215, which is developed by Celgene.

Kancera has also discovered that the company's HDAC6 inhibitors can be designed to operate also through an additional mechanism which has not been described publicly for competitive reasons. Kancera's results show that a combined effect against HDAC6 and Target 2 in a more efficient manner stops the cancer cell's ability to proliferate.

The company estimates the project with adequate resources could deliver a drug candidate in approximately 12-18 months. In a next step Kancera intends to evaluate how the new mechanism can be combined with inhibition of HDAC6 to fight intractable cancer.

In June 2015, VINNOVA announced that SEK 2 million had been granted Kancera to support the further development of

HDAC6 inhibitors against cancer.

Events during the period

Kancera reported that new series of potent compounds that only inhibit HDAC6 have been developed and a patent application has been filed.

Kancera has previously reported that the company's substance KAN0439782, which acts by inhibiting both HDAC6 and "Target 2" (the identity of the target is not published), selectively prevents the ability of cells that normally surrounds and helps tumors (so-called cancer-associated fibroblasts) to migrate by disrupting the cytoskeleton (the cytoskeleton is composed of protein fibers that affect the cell's ability to *e.g.* divide, send hormone signals, invade and migrate to other locations in the body).

These studies have now been extended to cancer cells which have a strong ability to use the cytoskeleton to adhere to surrounding healthy tissues, invade and metastasize. KAN0439782 can affect the cytoskeleton so that aggressive prostate cancer cells detach and die while treatment with the competing HDAC6 inhibitor ACY-1215 allows a portion of the cancer cells to survive and spread out on the surface

Events after the end of the period

Kancera has not reported any significant events for this project.after the end of the period.

Anti Parasite Project - an EU-funded international cooperation against deadly diseases

The EU-financed project (A-PARADDISE (Anti-Parasitic Drug Discovery in Epigenetics) is coordinated by the Institut Pasteur and includes collaborations with epigenetic experts from Germany, France, UK, Italy, Australia and Brazil. Kancera´s primary focus during the first phase of the project is to optimize the pharmaceutical properties of the anti-parasitic substances.

The project focus on target proteins in the following diseases (parasites): Malaria (*Plasmodium falciparum*), Schistosomiasis (*Schistosoma mansoni*), Leishmaniasis (*Leishmania*) and Chagas disease (*Trypanosoma cruzi*).

Kancera is the only pharmaceutical development company in the A PARADDISE consortium and is well positioned to commercialize the drug candidates that the company develops and owns together with its partners. For clinical development and commercialization of drugs for neglected diseases, it is likely that Kancera will seek cooperation with internationally established pharmaceutical companies and nonprofit organizations that have chosen to take social responsibility by investing in the development therapies against diseases that primarily affect poor countries in tropical and subtropical areas. Since countries that currently suffer from serious parasitic diseases have an increasing financial capacity to invest in drugs, the project´s future drug candidates may also have a commercial potential.

In 2014, Kancera has continued the optimization of anti-parasitic compounds which Kancera successfully initiated during the completed EU funded project Settrend. The project work mainly focused on the further development of anti-parasitic compounds that the company previously developed. 70 new substances have been synthesized with the goal to increase the exposure of the substances in the parasite. The academic groups in the consortium are currently testing the effect of these compounds against various types of parasites. Further, Kancera together with partners in the consortium have established an experimental plan for the selection of antiparasitic drug candidates that can come from Kancera´s chemistry development or from other partners in the consortium. Exchange of substances has been initiated in order to identify the epigenetic mechanisms that are appropriate to attack in the four studied parasitic diseases.

Events during the period

In January,300 000 € (ca SEK 2.8m) was paid to Kancera from EU for the continuing operation of the project until January 31, 2017.

Events after the end of the period

Kancera has not reported any significant events for this project after the end of the period.

Patent portfolio and intellectual property rights

The basis for the commercial potential of new drugs is a broad patent protection. Patent work is an important and integral part of Kancera's operations, especially in the early preclinical phases. Kancera's management has extensive experience in establishing patent strategies and build competitive patent portfolios even in highly competitive fields. For Kancera's projects, patent strategies and patent portfolios are developed together with internationally established patent law firms with which Kancera's management has a long lasting relationship. Timelines for the first patent application is determined from case to case depending e.g. on competitor activity. When Kancera sells drug candidates, there is a negotiation whether the Company's patents or patent applications are to be licensed or sold, directly or through option. Kancera currently owns eight patent families of small molecules (including the exclusive option to acquire the patents from the Fractalkine project), one for ROR inhibitors, two for Fractalkine receptor antagonists, three for PFKFB3 inhibitors and two for HDAC6 inhibitors. In addition to these Kancera owns two patent families covering antibodies against ROR1. However, these are not commercially developed at the moment.

Project/ Patent family	**Description**	**Status**	**Application-/ Patent number**	**Filing date** (YYYY-MM-DD)
Fractalkine*	Substance patent 1	Approved international patent	US 7947693	2006-04-03
Fractalkine*	Substance patent 2	Approved international patent	US 7960395	2007-09-27
ROR1	Substance patent	International application	15201842.2	2016-02-01
ROR1	Antibodies from mouse	International application, approved in the US and China	US 9150647	2010-12-10
ROR1	Human antibodies	International application, granted in the US and China	US 9266952	2011-12-12
PFKFB3	Patent family "Sulphoneamide compounds"	International application, granted in the US	9233946	2011-09-19
PFKFB3	Patent family "Biarylsulfoneamides"	International application, granted in South Africa	2014/03652	2012-12-21
PFKFB3	Patent family "Biarylsulfoneamides". Divisional application in the United States. Combination of biarylsulfoneamides with radiation therapy.	Divisional application in the US	15/078502	2016-03-23
HDAD6	Substance patent 1	International application	PCT/EP2015/0603293	2015-05-11
HDAC6	Substance patent 2	International application	PCT/EP2015/052091	2015-12-22

* Kancera has an exclusive option to acquire these patents by Acturum AB.

Market outlook for Kancera's products

2015 was a strong year for the industry with more and larger acquisitions than ever (Thomson Reuters Life Sciences Report 2015). Preclinical agreements continue to represent a significant proportion of the total number of agreements in the preclinical and clinical phases (53%). Also the numbers and sizes of license agreements and collaborations surpassed previous years. When it comes to license and option agreements, the comparison for the period 2010-2015 shows both increasing levels of payment upon signature of the agreement and increasing overall price tags for pharmaceutical projects. The median payment for preclinical license agreements upon signature of the contract is now USD 10 million. Cancer continues to be the therapeutic area with most agreements between biotech and pharmaceutical companies (35% of license and option agreements). It is also welcome to see that the proportion of options that are really used to acquire projects in the period 2010-2013 is 40% and the duration is 2-3 years, suggesting that many collaborations relatively quickly leads to the decision to continue product development, (Thomson Reuters Life Sciences Report 2015).

IMS Health reports that the forecast for the use of drugs and the society's investment in the use of drugs will increase by 4-7% per year until 2018, which is an increased rate compared to the previous five years. The driving factors behind this growth is the increased availability of good new proprietary specialty pharmaceuticals (such as cancer drugs) for an increasing number of patients and that a growing proportion of the world population is over 65 years.

In 2015, the European Medicines Agency (EMA) approved 93 new drugs of which about 42% constituted a new class and 20% were orphan drugs. The US Food and Drug Administration (FDA), approved 45 new drugs in 2015 which is significantly higher than the average 28 new drugs per year during the period 2006 to 2014. Of these 45 new drugs, 36% constituted a new class and 47% were orphan drugs. The number of new applications to FDA for approval of drugs has remained at the same level during 2006-2015, indicating a higher fraction of approvals during 2015. Of the 45 drugs approved in the USA in 2015, the proportion that acquired the status of so-called "Break-through therapy" was 22%, which may mean a quicker way to trial and possible approval. Both in the USA and in Europe, cancer was the disease with most new registered drugs (11 new cancer drugs approved in the USA and 14 in Europe) (Source: EMA and FDA).

Kancera's primary market is based on business-to-business sales of drug candidates for further clinical development and marketing by internationally established pharmaceutical companies.

The prioritized deal is based on an option model where Kancera signs agreements in the preclinical phase, before regulatory studies have been initiated, with a selected international partner possessing the resources and capacity for effective clinical development and marketing internationally. The option model provides Kancera with a cash flow during the more expensive parts of the project's development, and at the same time the cooperation gives partners the opportunity to influence the direction of the project during the critical phase between preclinical and clinic. This also increases the possibility of a rapid start of a clinical program. A quick and successful transition from Kancera´s preclinical to the partner´s further clinical development also increases the likelihood that the schedule for milestone payments to Kancera is kept.

There are several examples of license sales in the oncology area in preclinical phase amounting to several hundred million USD. Two of the most influential deals between biotech companies and pharmaceutical companies during the period 2010-2011 were made by companies whose projects had been partially developed by Kancera´s former subsidiary iNovacia AB, including Agios Inc. contracts with Celgene which included a payment upon signature of 130 million USD (however, this deal is regarded as an exception with respect to the size of the payment). Since the start, the cooperation between the two companies has been extended for a total of two years to allow delivery of Agios´ first Phase 1 project. This was announced on June 13, 2014 when Celgene decided to make use of the right to acquire Agios´ candidate drug AG-221 which attacks hematologic cancers through inhibition of the enzyme IDH to thereby disrupt the cancer metabolism. Celgene paid USD 120 million plus royalties for this early clinical project.

Another example is AstraZeneca´s subsidiary MedImmune´s acquisition of Amplimmune, a company with preparations in late preclinical phase, for the initial purchase price of 225 million USD, which may be increased later. J & J paid USD 150 million to Pharmacyclics for a BTK inhibitor Ibrutinib in clinical phase II, in addition to future installments of USD 825 million. The success of Pharmacyclicss in developing Ibrutinib from a drug candidate in 2008 to one of the strongest new drugs on the market to treat chronic lymphocytic leukemia led to that the company was acquired by Abbvie in March 2015 for USD 21 billion with the aim to further develop the full potential of Ibrutinib in both cancer and autoimmune diseases.

In April 2012 an agreement was announced between Boston-based Epizyme and Celgene regarding a preclinical drug development project directed against epigenetic targets in cancer, i.e. drugs active against the same target group as Kancera´s HDAC inhibitors. The agreement involved an upfront payment of 90 million USD including equity. Epizyme is a biotech company that has been a frontrunner for a new cancer treatment concept and has managed to close a series

of preclinical deals in the cancer area since early 2011 with GSK and Esai.

Another example of the interest in this type of inhibitors is that Celgene in July 2013 for 100 million USD in cash acquired an option to purchase the Boston-based Acetylon Pharmaceuticals. The other conditions for the option mean that a completion of the deal gives the sellers a minimum of 1.7 billion USD. Acetylon´s leading drug candidate is an HDAC6 inhibitor and the most advanced project is in Phase II for a potential treatment of leukemia. In July 2015 Sprint Bioscience AB signed a license agreement with Bayer HealthCare concerning a preclinical project targeted at cancer metabolism via the enzyme MTH1. MTH1 as a target in cancer was previously published in 2015 in the journal Nature by a Swedish research team led by Prof Thomas Helleday at SciLifeLab and the Karolinska Institute in Solna. The agreement between Sprint Bioscience and Bayer Heathcare includes payments of up to approximately EUR 190 million if all milestones are achieved, and in addition, royalties on sold product. Taken together, this shows that Swedish pharmaceutical R & D is in the international forefront both when it comes to academic biological research and product development in the preclinical phase.

There are several reasons for preclinical projects to be met with increased interest from large pharmaceutical companies. The development departments at pharmaceutical companies want to influence the selection and design of an active substance themselves. It could be disastrous if a substance that has reached phase II or phase III proves to be suboptimal or insufficiently suited to its task. Time and money will be lost if a clinical trial needs to be redone from the beginning. Historically, there are many examples of projects that need to be corrected and where the clinical trial needs to be repeated from the start. Sometimes pharmaceutical companies also choose to run several parallel phase I and phase II studies to ensure that they cover several different patient populations and diseases, as well as schedules for treatment, and thereby position the product optimally for the costly phase III clinical trials.

The underlying demand for Kancera´s drug candidates is driven by the medical need to make the combat against cancer more efficient.

The trend is towards

- diagnostic methods that provide genetic information about exactly what factors in the individual patient's cancer drive the disease and whether there are mutations that render a traditional drug inactive

- drugs that attack the driving mechanisms of the cancer, that overcome causes of resistance and act selectively against cancer to reduce the side effects that would otherwise contribute to increased mortality and high medical costs

Consequently, more patients will be offered a personalized cancer treatment resulting in a longer and better life. The number of drug development projects within the cancer area has steadily increased, but many of them follow the same path as others (Source : lifescivc.com/2012/06/cancer-drug-targets-the-march-of-the-lemmings /) why pharmaceutical companies now focus their search for drug candidates that distinguish themselves from the mainstream and have the potential to fundamentally change the conditions for the treatment of life-threatening diseases. Drugs targeting ROR1 qualify for such an interest from the pharmaceutical industry and Kancera as a biotech company leads this development.

Kancera´s focus is on target molecules in the cancer that opens opportunities to break the resilience of life-threatening cancer forms as well as the development of diagnostics that allow early identification of patients who benefit from the new treatment.

Currently Kancera evaluates applications of future drugs against ROR, Fractalkine-receptor, PFKFB3, and HDAC6 in

- Solid tumors in the pancreas, ovary, lung, bowel and breast. These forms of cancer are among the types of cancer that causes most deaths.
- Chronic lymphocytic leukemia (CLL) and acute myeloid leukemia (AML), which are the most common chronic and acute form of leukemia respectively in adults, as well as multiple myeloma (MM).

These cancer indications each represent a world market in the range of 3.5 to >10 billion SEK annually (Source : GlobalData). According to the Dental and Pharmaceutical Benefits Agency (TLV) in Sweden the society is willing to pay for drugs that treat life-threatening and other serious diseases up to SEK 1 million per year of life with full quality of life (so-called quality adjusted life year, QALY. Two extra years survival with an estimated 50% level of full quality of life corresponds to one QALY). Although there are no definitive requirements to show prolonged survival of new drugs, TLV means that in practice it will be difficult to justify subsidization of new drugs that prolong survival less than 6 months* since this level of prolongation of survival implies a low pricing to cope with the cost per QALY. There exist

similar principles for society's willingness to pay in the rest of the world. For example, in England drugs with a cost per QALY in excess of £ 30,000 are not subsidized. However, exceptions are made for life-threatening conditions where the boundary is moved up to £ 50,000 in accordance with the Agency's (NICE) "end-of-life criteria".

*at the time of registration of the drug results in terms of overall survival is often lacking, so it is assumed that a longer period of stable disease translates into equally long prolongation in survival.

Kancera´s own published results, as well as publications from independent research groups in the ROR and PFKFB3 area (see sources in each project section) support that future drugs acting through ROR and PFKFB3 have the potential to improve treatment of the aforementioned cancers. How well this potential can be translated into clinical practice remains to be proven in clinical studies.

In addition, the industry's interest in rare diseases, so-called Orphan diseases, has increased in recent time given that they represent significant unmet medical need and that the patient group often is clearly defined thus facilitating clinical studies. This has led the authorities to facilitate the development of, and the protection of products against these diseases. New approved drugs by both the European Medicines Agency EMA and the American FDA include a high proportion of drugs to treat rare diseases (see the introduction for 2015 statistics). Kancera's projects have in preclinical studies been shown to be a possible way to treat several forms of cancer that precisely meet the requirements for designation as an Orphan disease (the definition of Orphan disease in the United States is diseases affecting fewer than 200,000 individuals).

The need for improved treatments is exemplified below for two of the cancer forms that Kancera addresses with its drug projects and that qualify as Orphan diseases.

Cancer of the pancreas annually affects more than 100 000 patients in Europe and the U.S. The survival of these patients is less than two percent five years after diagnosis. A combination of chemotherapy and radiotherapy is used to enable removal of the tumor by surgery. The life sustaining drug treatment mainly consists of various types of cell poisons (Gemcitabine and FOLFIRINOX which contain combinations of Fluorouracil, Irinotecan, and Oxaliplatin). Today, there is no recommended drug targeting pancreatic cancer. In recent years, more specific enzyme-inhibiting drugs have been approved for the treatment of pancreatic cancer, such as erlotinib (EGFR inhibitor mainly) and Sutent (a broad-acting inhibitor of many kinase enzymes, including VEGF, PDGF and SCF (Kit)). However, these drugs have shown limited therapeutic efficacy why the medical need for new drugs against this disease remains very high. The market for pancreatic cancer in the United States in 2009 totaled USD 781 million and the expected growth was -4 to +8% in 2017, (Source : Global Data Healthcare).

Chronic lymphocytic leukemia (CLL) annually affects approximately 30 000 patients in Europe and the U.S., which makes CLL to the most common chronic form of leukemia. The traditional treatment of cancers such as CLL is currently not sufficiently effective and selective. The most common type of treatment of CLL is a combination of the antibody Rituximab and chemotherapy such as Fludarabine and Cyclophosphamid. This combination of drugs is used in 19% of the treatments in the seven countries that represent the largest pharmaceutical markets. Following the initial treatment of patients approximately 85% are symptom free, but already after four years clear symptoms of cancer disease had returned for 80% of the patients. New and better treatments are required in this phase of the disease. New drugs with other effects on refractory CLL is now being introduced, such as ibrutinib and idelalisib. Ibrutinib and Idelalisib have clearly improved the treatment of CCL, and give effect in 70-80% of the patients with this disease. However, so-called complete remission (the symptoms have disappeared) has only been reached in a small number of these patients. Since complete remission in cancer is generally linked to a longer survival, there is a need for drugs that work in a new way. Kancera has previously shown that the candidate drug KAN0439834 effectively kills CLL cells from blood and lymph taken from patients *in-vitro* and also in animal models of the human disease. Also, Kancera in collaboration with Prof. Håkan Mellstedt's group at the Karolinska Institute, has demonstrated that Kancera's ROR inhibitor is also effectively killing CLL cells from bone marrow which is a characteristic sought as a complement to today's registered drugs against CLL.

The market for CLL is estimated at 800 million USD in 2017 (Source: Global Data Healthcare 2013). Kancera also expects that there are good opportunities to expand into other cancers, given that ROR-1 is found in at least eight other blood cancers and several solid tumors (ovarian cancer, lung cancer, breast cancer, pancreas cancer).

Income Statement

SEK 000´s (if otherwise not specified)	1 Jan-31 Mar 2016	1 Jan-31 Mar 2015	1 Jan-31 Dec 2015
Revenues			
Net sales	**114**	**10**	**282**
Cost of sales & services	-19	-7	-74
Gross profit	**95**	**3**	**208**
Operating Expenses			
General & administrative expenses	-672	-587	-3 943
Selling expenses	-229	-189	-805
Research & development expenses	-4 249	-4 164	-20 355
Contributions from the EU/Vinnova	-	-	5 209
Total expenses	**-5 150**	**-4 940**	**-19 894**
Operating income	**-5 055**	**-4 937**	**-19 686**
Income from Financial Investments			
Financial income	25	23	85
Financial expenses	-5	-4	-11
Financial net	20	19	74
Income after financial items	**-5 035**	**-4 918**	**-19 612**
Taxation	**-**	**-**	-
Net income	**-5 035**	**-4 918**	**-19 612**
Earnings per share, before and after dilution	-0,05	-0,05	-0,19

Balance Sheet

SEK 000´s (if otherwise not specified)	31 Mar 2016	31 Mar 2015	31 Dec 2015
Assets			
Non-current Assets			
Intangible assets, activated R&D expenses	6 000	6 000	6 000
Tangible assets	2 866	3 667	3 145
Total non-current assets	**8 866**	**9 667**	**9 145**
Current Assets			
Work in progress	2 155	3 501	1 486
Receivables	1 277	899	1 213
Cash and cash equivalents	12 083	17 809	15 567
Total current asstes	**15 515**	**22 209**	**18 266**
TOTAL ASSETS	**24 381**	**31 876**	**27 411**
Equity and Liabilities			
Equity			
Restricted equity	8 660	8 212	8 660
Non-restricted equity	8 345	14 378	13 265
Total equity	**17 005**	**22 590**	**21 925**
Provisions and liabilities			
Long-term liabilities	2 800	1 500	1 500
Short-term liabilities	4 576	7 786	3 986
Total provisions and liabilities	**7 376**	**9 286**	**5 486**
TOTAL EQUITY and LIABILITIES	**24 381**	**31 876**	**27 411**

Statement of Changes in Equity

SEK 000´s (if otherwise not specified)

	2016		2015
Total equity, opening balance on the 1st of Jan 201(	**21 925**	**Total equity, opening balance on the 1st of Jan 2015**	**27 289**
Optionprogram	115	Optionprogram	219
Q1 net income	-5 035	Q1 net income	-4 918
Total equity, closing balance on the 31st of March 2	**17 005**	**Total equity, closing balance on the 31st of March 2(**	**22 590**

Cash-Flow Statement

SEK 000´s (if otherwise not specified)	1 Jan-31 Mar 2016	1 Jan-31 Mar 2015	1 Jan-31 Dec 2015
Cash-flow from operating activities			
Operating income after financial items	-5 035	-4 918	-19 612
Depreciation	279	233	1 088
Other non-cash-flow affecting items	8	219	631
Cash-flow from operating activities before working capital change	**-4 748**	**-4 466**	**-17 893**
Change in working capital	-1 536	-667	-2 765
Cash-flow from operating activities	**-6 284**	**-5 133**	**-20 658**
Investment activities			
Investment in tangible assets	0	-32	-366
Cash-flow from investment activities	**0**	**-32**	**-366**
FREE CASH-FLOW available to INVESTORS	**-6 284**	**-5 165**	**-21 024**
Financing activities			
Issue of shares/other capital infusions	-	-	13 617
Financing from the EU/Vinnova	2 800	-	-
Cash-flow from financing activities	**2 800**	**-**	**13 617**
CASH-FLOW for the PERIOD	**-3 484**	**-5 165**	**-7 407**
Cash and cash equivalents at the beginning of the period	15 567	22 974	22 974
Cash and cash equivalents at the end of the period	12 083	17 809	15 567

Notes

Note 1. Accounting and valuation principles

This interim report has been prepared in accordance with BFNAR 2007:1, Voluntary interim reporting and adheres to the listing requirements of First North. From 2013 Kancera applies the Swedish Annual Accounts Act and BFN:s supplementary regulations BFNAR 2012:1 Annual Report and consolidated accounts (K3).

The accounting principles of the company are described in the latest published Annual Report (2015).

Unless otherwise indicated, amounts are reported in Swedish kronor (SEK) and rounded off to the nearest thousand. As a result of the rounding off to the nearest thousand kronor, adding up the amounts stated may not correspond exactly to the total given. Amounts and figures in parentheses are comparison figures for the same period last year.

Note 2. Related party disclosures

During the period, Kancera paid compensation to F:a Mellstedt Medical for scientific consulting and scientific marketing services at an amount of SEK 47 kSEK. Håkan Mellstedt, a Board member at Kancera, is the Managing Director and owner of F:a Mellstedt Medical. No other remuneration was paid to related parties with the exception of Board fees and outlays for expenses.

Note 3. Incentive schemes

The Annual General Meeting on May 26, 2014 decided to introduce an incentive scheme for employees of the company and corresponding executives and Board members. The inventive scheme involves the issue of maximum 2 800 000 warrants. Of these, 2 200 000 will form the base for the issue of maximum 1 650 000 warrants for the employees. Each warrant will entitle the holder to acquire one share for a price corresponding to 130 percent of the volume weighted trading price of the company's shares on NASDAQ OMX First North during the period May 27 to June 13, 2014. The warrants shall have a term of three years. During the period, the staff may choose to exercise ¼ of the number of granted options after one and two years, respectively, leaving, in this example, ½ of the number of options to exercise after three years.

The remaining 600 000 warrants are issued to the Board members Bernt Magnusson, Håkan Mellstedt and Carl-Henrik Heldin. Each warrant shall have a term of three years. The price of the warrants is a market price determined by the Black & Scholes valuation model. The warrants to staff and contractors are issued without charge. At full subscription and full exercise of all warrants, the share capital increases with SEK 233 333,33. If all warrants are exercised to subscribe for 2 800 000 shares, the dilution of the share capital will amount to about 2.8 percent.

The first period for exercising the options was closed in June 2015. In total 450 246 new shares were signed. There now remains 2,349,754 warrants,

Warrants in the company's treasury amounted to 560 000 as of December 30 and has been admitted to SEK 0 in the balance sheet. The company management count on that these can be sold with income in the future.

Note 4. Current grants to be accounted for at a later date

Funded by	Amount granted, kSEK	Amount paid, kSEK	Reporting date
Vinnova	2 000	1 097	June 2016 and July 2017
EU	8 520**	7 487	March 2017*
	10 520	**8 586**	

* Final report

** Assuming an EUR exchange rate of 8.95 SEK. The paid amount SEK 4,686,000 corresponds to 55% of the grant. An additional 30% of the grant is paid following an approved midterm report which will be submitted in September 2015, and the remaining 15% of the grant is paid following an approved final report which will be submitted in March 2017.

Note 5. The company's operations and risk factors

The Board of Directors and the CEO certify that the interim report provides a true and fair overview of the company's operations, financial position and results, and describes the significant risks and uncertainties faced by the company.

When assessing Kancera future development, it is important to consider risk factors alongside potential growth in earnings. Kancera's operations are affected by a number of risks that may affect Kancera's results and financial position to varying degrees. For a description of the risks associated with the Company, see the company's Annual Report 2015.

Note 6. Definitions

Return on equity (ROE)
Net profit for the period as a percentage of average equity.

Return on capital employed (ROCE)
Profit before tax plus financial expenses as a percentage of average capital employed.

Equity per share
Equity divided by the number of shares on the reporting date.

Cash flow per share
Cash flow from operating activities divided by the average number of shares.

Option-based deal
Agreement between two parties, giving one party the right through prepayment to later acquire sole rights to the asset concerned.

Earnings per share
Profit for the period divided by average number of shares.

Capital employed
Total assets reduced with non-interest bearing liabilities.

Equity/assets ratio
Equity as a percentage of total assets.

Stockholm, May 3, 2016

Erik Nerpin *Chairman of the Board*	Håkan Mellstedt *Director*	Bernt Magnusson *Director*
Carl-Henrik Heldin *Director*	Thomas Olin *CEO/Director*	

This Interim Report has not been reviewed by the company's auditors.

Financial calendar

- Annual General Meeting — May 26, 2016
- Interim Report January-June 2016 — August 19, 2016
- Interim Report January-September 2016 — November 18, 2016

For further information, please contact:

- Thomas Olin, CEO: +46 735 20 40 01
- Erik Nerpin, Chairman of the Board and Election Committee: +46 70 620 73 59

Kancera AB (publ)
Karolinska Institutet Science Park
Banvaktsvägen 22
SE-171 48 Solna

Please visit the company's website www.kancera.com