Press release November 18, 2016

Interim Report for Kancera AB (publ) Q3 2016

January 1 – September 30, 2016

The period January to September 2016 in brief

- R&D expenses for the period amounted to SEK 13.3m (SEK 12.1m) of which the third quarter constituted SEK 4.6m (SEK 3.2m).
- Operating income for the period amounted to SEK -15.8m (SEK -13.8m) of which the third quarter constituted SEK -5.2m (SEK -3.4m).
- Income after financial items for the period amounted to SEK -15.9m (SEK -13.7m) of which the third quarter constituted SEK -5.4m (SEK -3.4m).
- Earnings per share for the period were SEK -0.14 (SEK -0.14) of which the third quarter constituted SEK -0.04 (SEK -0.03).
- Cash flow from operating activities for the period amounted to SEK -17.4m (SEK -16.1m) of which the third quarter constituted SEK -8.2m (SEK -5.1m).
- Equity as of September 30, 2016 amounted to SEK 65.9m (SEK 27.8m) or SEK 0.50 (SEK 0.27) per share. The equity/assets ratio as of September 30, 2016 was 85 percent (74 percent).

  Cash and cash equivalents as of September 30, 2016 amounted to SEK 63.5m (SEK 20.2m).

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.

- 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.

- With the authorization of the extraordinary general meeting on 22 April 2016, Kancera AB carried out an issue of units with preferential rights for the shareholders, as well as an issue of units in the form of over-allotment space through a separate directed share issue without preferential rights. The rights issue, which was fully subscribed in May 2016, concerned 20,785,072 units and an over-allotment space of 4,000,000 units consisting of one share and one warrant at a price of SEK 2.50 per unit. On top of this compensation to underwriters and financial advisors was added. After registration of the issuance of the over-allotment option and compensation to underwriters and financial advisors, the number of shares in Kancera AB amounts to 131,486,720 and the number of warrants to 27 561 356. The new issue has brought Kancera AB approximately SEK 61.9 million before issue costs. 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 provided the following operational update of the Fractalkine and ROR projects:

    - the Fractalkine antagonist KAN0440567 is able to eliminate pain resulting from inflammation of the pancreas. This type of pain is similar to the pain resulting from cancer in the pancreas and therefore these results support the continued development of KAN0440567 towards clinical trials against cancer.

    - the ROR inhibitor KAN0439834 has been shown to effectively kill resistant cancer cells from the bone marrow of multiple myeloma (MM) patients. MM originates in the bone marrow and is an essentially incurable chronic disease today. 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.

- Kancera AB announced that VINNOVA has paid an additional SEK 358,451 to the HDAC6 project as part of the grant totaling SEK 2 million which has been designated by VINNOVA for the further development of Kancera's HDAC6 inhibitors against cancer. This payment was made following the approval of Kancera's third progress report for the project.

- Kancera AB announced that its subsidiary Kancera Förvaltning AB has been formed. The operations of the subsidiary include mainly financial management including Kancera's stock option plan.

- Kancera AB announced that the company is preparing for clinical trials by appointing Niclas Brynne to lead Kancera's clinical development projects. He will be part of the management team and report directly to the CEO.

- Kancera AB announced that the company within the framework of the EU research program Horizon 2020 has been awarded a research grant of approximately EUR 500,000 over three years for the financing of two Ph.D. students. They will explore how resistance of cancer cells arises, how it can be broken and how such findings can form the basis for new drug project against cancer. One of these Ph.D. students will focus on PFKFB3 as a target for the treatment of cancer.

- Kancera AB hereby announces that the anti-parasitic EU-funded project A-PARADDISE has started efficacy studies in a disease model for schistosomiasis in mouse. Thus, the consortium has taken a decisive step towards achieving the final goal of the A-PARADDISE project, which ends on January 31, 2017.

Significant events after the end of the reporting period

- Kancera AB reported on the preparations for a clinical study in the Fractalkine project. In the HDAC6 project it was reported on possibilities for structure-based drug design and effect on an immune check point protein in cancer cells:
    - The Fractalkine project has initiated the procurement of a clinical contract company that will conduct the first clinical study, as well as with contract manufacturing companies for the delivery of the formulation of KAN0440567 to be used in this study.
    - The HDAC6 project has shown that Kancera's substances are capable of down-regulating a protein that acts like a brake on the immune system in cancer cells which may contribute to that cancer cells escape the patient's immune system. Furthermore, the project has determined crystal structures that show how Kancera's substances bind to "Target 2", which gives information on how HDAC6 / Target 2 inhibitory drugs can be optimized.

- Kancera announced that Nasdaq has approved Kancera's application regarding listing on First North Premier. The first trading day for Kancera shares on this list is October 31, 2016. The Company's shares continue to be traded with the same short name and ISIN code.
- Kancera reported advances in the ROR project regarding
    - A possible broadened use of ROR inhibitors against the lymphoma disease "Richter's syndrome" as seen from analyses of tumor samples. The disease affects approximately 15% of patients with chronic lymphocytic leukemia (CLL) and there is currently no effective treatment available.
    - The production method for KAN0439834 has been further developed and is now implemented in a straightforward and efficient way. The company believes that this paves the way for the further preclinical and clinical development of the compound.

Statement from the CEO

With the start of the procurement of clinical contract companies for the clinical study of the Fractalkine receptor antagonist KAN0440567, we now proceed from plan to action in line with Kancera's prospectus from May 2016. Before applying for authorization of a clinical trial, we focus on the formulation (*i.e.* the product, *e.g.* capsule) of KAN0440567 that is to be given to humans. When this product is tested and ready we intend to submit the application for authorization of a clinical trial to the relevant authority (the Medical Products Agency in Sweden or equivalent authority in another EU country) and for the corresponding ethical permission.

Also in the ROR project the focus has been on the development of the product, since e.g. its purity when produced at large scale provides the foundation for both the toxicology studies and ultimately for a commercially viable production. The technological advances in the synthesis and purification of the ROR inhibitor KAN0439834 are therefore important for the entire development of the project. The recently reported findings that ROR1 is present as a target for drugs in an intractable condition called Richter's syndrome also strengthens the project's ability to position itself on the market as a strong complement to the new drugs for leukemia and lymphoma.

In the autumn, we have also seen significant progress in the HDAC6 project, partly in the form of a successful crystallization of "Target 2" which gives us access to a molecular design drawing that facilitates the final steps in the optimization of our unique dual action (HDAC6 / Target 2) substances before the selection of a candidate drug. That we also have been able to show that Kancera's HDAC6 inhibitors have the capacity to reduce the immunosuppressive protein PD-L1 in cancer cells confirm the published findings which support HDAC6 as a new promising immuno-oncology target for drug development.

In September, we were informed that the EU, through its research initiative Horizon2020, has chosen to give a total of about four million Euro (of which Kancera receives more than 10%, about 500,000 Euro) in grants to a leading international team of researchers that will recruit Ph.D. students in order to evaluate new approaches to combat cancer, especially with regard to how drugs can act to stop the DNA repair in cancer cells. Kancera is awarded two Ph.D. students and a grant to cover research costs, allowing us to work with cutting-edge talent in oncology to identify new project opportunities.

Thomas Olin

CEO Kancera AB

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. The number of shareholders was around 7700 as of July 29, 2016. FNCA Sweden AB is Kancera's Certified Adviser. Professor Carl-Henrik Heldin, Professor Håkan Mellstedt and Dr Charlotte Edenius are scientific advisors and board members in Kancera AB.

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)

Kancera Group (previous year, parent company Kancera AB)

	July-Sept		Jan-Sept		1 Jan-31 Dec
	2016	2015	2016	2015	2015
Net turnover	42	183	174	267	282
Operating expenses	-5 276	-3 578	-15 916	-14 000	-19 894
R&D expenses	-4 645	-3 249	-13 300	-12 055	-20 355
Operating Income	-5 246	-3 441	-15 785	-13 797	-19 686
Income after financial items	-5 360	-3 418	-15 870	-13 741	-19 612
Net income	-5 360	-3 418	-15 870	-13 741	-19 612
Cash-flow from operating activities	-8 249	-5 105	-17 404	-16 149	-20 658
Cash on hand at closing date	63 494	20 155	63 494	20 155	15 567
Equity at closing date	65 860	27 798	65 860	27 798	21 925
Key ratios					
Return on equity, %	neg	neg	neg	neg	neg
Return on capital employed, %	neg	neg	neg	neg	neg
Investments in intangible assets	-	-	-	-	
Investments in tangible assets	-		-	-366	-366
Solvency ratio	85%	74%	85%	74%	80%
No. of employees	13	12	13	12	10
Earnings per share, before dilution	-0,04	-0,03	-0,14	-0,14	-0,19
Earnings per share, after dilution	-0,04	-0,03	-0,14	-0,14	-0,19
Equity by share, kr	0,50	0,27	0,50	0,27	0,21
Cash-Flow by share, kr	-0,06	-0,05	-0,15	-0,16	-0,20

Comments on the financial development

Increased costs for the period compared with the corresponding period in 2015 are primarily attributable to increased research and development costs incurred due to that Kancera operates one additional project in an advanced preclinical phase. As a consequence of the acquisition of the subsidiary Kancera Förvaltning AB, the present Interim Report, Q2 2016, is prepared in accordance with IAS 34 and related parts of the Annual Accounts Act. Comparative figures from the previous year used below pertain to the parent company Kancera AB. The transition to the new accounting standards did not affect the income statement or balance sheet for the period January 1 - March 31, 2016 accounted for using the previous principles, or the comparison figures used below in the comments from the previous year regarding the parent company Kancera AB.

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 0.0m (SEK 0.0m). The company also receives financial support from the EU project A-PARADDISE where the support, following an approved final report (see Note 4), is offset against incurred costs for the period amounting to SEK 3.8m of consumables, performed months of work plus 60% overhead on the sum of these costs. The financial support from EU covers 75% of the project costs plus 60% overhead. The company has also received a grant of EUR 299,000 for another EU project called SYNTRAIN. This amount represents 60% of the total grant that Kancera will receive from the EU. The grant covers salary, research training, and overhead costs for two students for three years. Kancera has an obligation to cover the corresponding costs for one additional year. The company deducts the grant received against a unit cost per completed month of the Ph.D. education.

Expenses
Expenses in the third quarter amounted to SEK 5.3m (SEK 3.6m), which breaks down into costs of services sold of SEK 0.0m (SEK 0.0m), research and development expenses of SEK 4.6m (SEK 3.2m) and other sales and administrative expenses of SEK 0.6m (SEK 0.3m). Expenses during the period January 1 to September 30, 2016, amounted to SEK 15.9m (SEK 14.0m), which breaks down into costs of services sold of SEK 0.0m (SEK 0.1m), research and development expenses of SEK 13.3m (SEK 12.1m) and other sales and administrative expenses of SEK 2.6m (SEK 1.9m).

Earnings
Income after financial items for the third quarter amounted to SEK -5.4m (SEK -3.4m) or SEK -0.04 per share (SEK -0.03) and for the period SEK -15.9m (SEK -13.7m) or SEK -0.14 per share (SEK -0.14). The costs of the option program for employees and other senior executives during the third quarter amounted to SEK 0.1m (SEK 0.1m).

Cash flow and liquidity
Cash flow amounted to SEK -5.2m (SEK -5.2m) in the third quarter. Cash flow from operating activities for the third quarter amounted to SEK -8.2m (SEK -5.1m) or SEK -0.06 per share (SEK -0.05) and from financing activities amounted to SEK 3.1m (SEK 0.0m).

With the authorization of the extraordinary general meeting on 22 April 2016, Kancera AB carried out an issue of units consisting of one share and one warrant at a price of SEK 2.50 per unit. The rights issue, which was fully subscribed in May 2016, has brought Kancera AB approximately SEK 61.9 million before issue costs.

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. The European Union has so far paid approximately SEK 7.5m for the project. Ongoing work for the period amounting to SEK 3.8m 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 when the remaining grant of ca SEK 1.5m is paid to Kancera and the work will be registered as revenue and settled against accumulated costs.

Cash and cash equivalents as of September 30, 2016 totaled SEK 63.5m (SEK 20.2m).

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

Investments in intangible assets in the third 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 4.6m (SEK 3.2m) for the third quarter.

Equity and share data

Total equity as of September 30, 2016 was SEK 65.9m (SEK 27.8m).

Share capital as of September 30, 2016 amounted to SEK 10 957 227 spread over 131 486 720 shares with a quotient value (rounded off) of SEK 0.0833 per share.

Earnings per share for the third quarter, based on a weighted average of the number of outstanding shares, were SEK -0.04 (SEK -0.03).

The equity/assets ratio as of September 30, 2016 was 85 percent (74 percent). Total equity per share was SEK 0.50 (SEK 0.27) 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 as of December 31, 2015 amount to SEK 94.7m.

Personnel
Kancera AB had 13 full time employees (10) as of September 30, 2016 of which 9 are men and 4 are women.

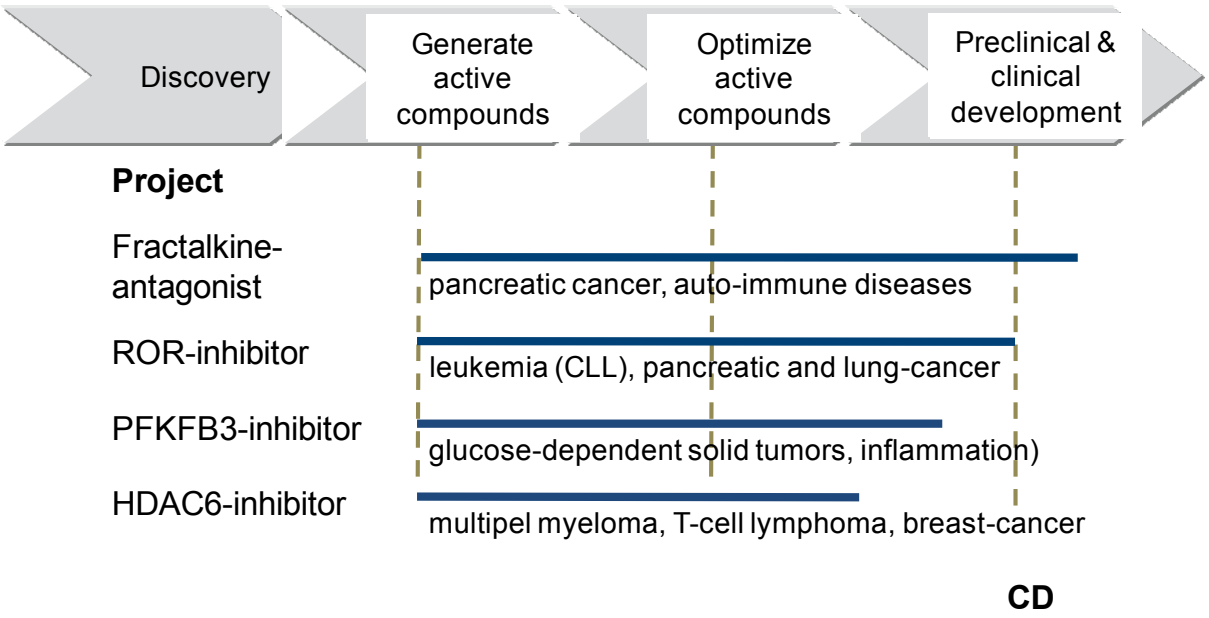
Pharmaceutical Development

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.

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 in connection with nerve damages and 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 (e.g. myeloma) by decreasing the cancer cell's ability to move and support the patient's immune system to identify and eliminate cancer 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



The goal of the development of Kancera AB's product portfolio over the next 18-24 months is to bring at least one of Kancera AB's drug candidates in the Fractalkine and ROR projects to clinical trials and thus first clinical use (chronic lymphocytic leukemia / pancreatic cancer). In parallel, we will complete the evaluations of broader use of the drug candidates from these projects in order to reduce the risk of the further product development and to demonstrate their full commercial potential. The operational objectives also include delivering drug candidates from the HDAC6 and PFKFB3 projects.

In the ROR project, the first drug candidate KAN0439834 will be tested continuously 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 with the purpose to broaden the use of ROR inhibitors to lymphoma and solid tumors. 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 KAN0440567 (AZD8797). The agreement with Acturum Life Science gives Kancera right to evaluate AZD8797 in preclinical studies and then to acquire the project. In light of demonstrated effects in disease models with relevance to the treatment of cancer pain, Kancera AB announced in April 2016 the decision to acquire the Fractalkine project following successful transfer of know-how and data from AstraZeneca and Acturum. For the Fractalkine project Acturum Life Science AB is paid in total 6 million Kancera shares divided into three tranches which can only be achieved if the project is successfully developed. This payment model means that the two companies share the risk in the product development up to when the first study has been conducted in man. 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.

The main part of the company´s resources is invested in the ROR, Fractalkine and 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. According to plan, this project will be completed and the final report will be submitted to EU in the first quarter of 2017.

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 until July 2017 for the further development of HDAC6 inhibitors. The HDAC6 project is within approximately 9 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 4.6m (SEK 3.2m) for the third quarter 2016 which have been recognized as costs in its entirety.

See page 22 for more information on market prospects for Kancera AB's products.

ROR technology – reprograms cancer to self-destruct

Product profile – ROR1 inhibitor

Property	Summary of "Target Product Profile" (TPP)
Primary indication	Chronic lymphocytic leukemia, other ROR1 driven hematologic cancer forms.
Secondary indication	Pancreatic, 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 receptor ROR1 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 control 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 receptor 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.

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.

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 blood cancer such as chronic lymphatic leukemia (CLL).

Multiple myeloma (MM) is manifested in the bone marrow and is an essentially 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.

Events after the end of the period

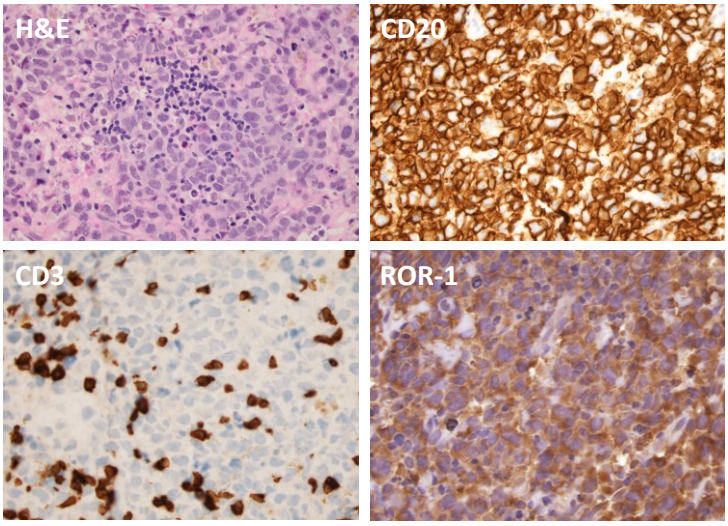
15% of patients with chronic lymphocytic leukemia (CLL) develop an intractable type of lymphoma, called Richter's syndrome. Neither standard treatments nor the newest drugs for CLL have the desired effect on Richter's syndrome, so there is a great medical need for new drugs against this disease.

Studies together with Dr. Georgios Rassidakis at the Karolinska Institute and MD Anderson Cancer Center (USA) have shown that tumor cells from Richter's syndrome carry ROR1 in a majority of patients examined. Kancera has also shown that its ROR1 inhibitors are active against cancer cells of the same type as in Richter's syndrome. Thus, the potential clinical use of ROR1 inhibitors may be broadened.

The further development of KAN0439834 depends on a large scale synthesis and purification (production) of the substance. Kancera has reported that the production method for KAN0439834 has been further developed and is now implemented in a straightforward and efficient way. The company believes that this paves the way for the further development of the substance towards clinical development and commercial production. Toxicology and safety studies under GLP still remain before decision on clinical trial.

Figure 2

ROR-1 Expression in Richter's transformation of CLL



An example of Richter's transformation of CLL: Large neoplastic lymphoma cells seen with routine staining (H & E), that are positive for the B-lymphocyte marker CD20 + and negative for the T-cell marker CD3. These lymphoma cells also stained positive for the presence of ROR-1 (mostly inside the cell).

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	Pancreatic cancer and metastasizing breast cancer.
Secondary indication	Cancer pain (pain related to pancreatic 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 KAN0440567 (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 KAN0440567 (AZD8797).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

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 one of 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. Today, close to 80% of these cancer patients have a disease that

has advanced too far to allow 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 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 pancreatic 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 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 better 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.* macrophages which is a type of immune cells. 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.

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.

Kancera AB reported that the Fractalkine antagonist KAN0440567 is able to eliminate pain resulting from inflammation of the pancreas. Kancera AB 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. Kancera reported 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.

Events after the end of the period

The company intends to bring at least one drug discovery project to a clinical study over the next 18-24 months.

Kancera has reported that the development of the Fractalkine project is making progress according to the project goals described in Kancera’s prospectus from May 2016. The procurement of clinical contract companies for the execution of the clinical trial has been initiated in parallel with the procurement of the manufacture and stability testing of the Fractalkine-blocking product to be used in this study. At the same time, an application for a clinical trial is prepared to the responsible authority (the Medical Products Agency in Sweden or equivalent authority in another EU country) and for the corresponding ethical permission.

The planned clinical study aims to map out the safety, tolerability and pharmacokinetics of KAN0440567 in healthy subjects. KAN0440567 will be administered orally in increasing doses, and thereafter in multiple doses. The study will include a part that aims to determine if food intake influences the absorption of KAN0440567. Also included are biological markers that will show how KAN0440567 affects the mechanism in the body that is expected to provide the desired pharmaceutical effect.

Before the timetable for the clinical study is publicly communicated, Kancera intends to complete the development of at least one of the products to be used in the clinical trial and await approval from the responsible authority and the ethical committee.

The PFKFB3 project –blocks glycolysis in solid tumors and weakens cancer cells

Product profile – PFKFB3-inhibitor

Property	Summary of "Target Product Profile" (TPP)
Primary indication	Solid tumors with high glucose consumption such as pancreatic 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 of these cover new PFKFB3 inhibitors (registered in 2010 and 2012) and one of these (registered in 2016) covers 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. 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.

Within the framework of the EU research program Horizon 2020, Kancera has been awarded a research grant of approximately EUR 500,000 over three years for the financing of two Ph.D. students in order to explore how resistance in cancer cells arises and how it can be broken. The two Ph.D. students' research will be performed in close collaboration with Prof. Thomas Helleday at Karolinska Institutet. One of these students will focus his/her research on PFKFB3.

Kancera is part of a consortium, along with nine internationally recognized research groups, that will explore the area DNA damage response (DDR) in tumor cells. DDR is one of the most promising research areas for the development of new drugs against currently incurable cancers.

Events after the end of the period

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

The HDAC6 project – acts 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	T-cell lymphoma, Breast cancer, Fibrotic diseases
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 ability of the cancer to check 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.

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 9 months. In a next step Kancera intends to evaluate how the new mechanism can be combined with inhibition of HDAC6 to combat 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.

VINNOVA has paid Kancera's HDAC6 project the third installment of the grant from the Strategic Innovation Agenda of common diseases (SWElife). The payment of SEK 358,451 followed VINNOVA's approval of Kancera's progress report for the project.

Kancera AB has evaluated a selection of the company's patent-pending HDAC6 inhibitors for properties that determine how efficiently the compound is taken up in the body and the concentration in the blood. These properties will normally determine the ability to achieve the desired treatment effect. In Kancera's studies, compounds have been given orally to mice after which the concentration of the substance has been determined in circulation. The experiments led to the identification of HDAC6 inhibitors that are significantly better with respect to these properties as compared to Acetylon's HDAC inhibitor ACY-1215.

Events after the end of the period

Laboratory studies at Kancera have shown further support to that a combined effect against HDAC6 and Target 2 may provide an advantage over other HDAC6 inhibitors in cancer treatment by stopping the division of cancer cells *via* a dual mechanism of action. Further, Kancera reported that its HDAC6 inhibitors in studies of multiple myeloma cells reduce the amount of PD-L1 (Programmed Death Ligand 1) more effectively than the competing substance ACY-1215 (see Figure 3). Cancer researchers have previously shown that a reduction in the amount of PD-L1, which can be achieved with HDAC6 inhibitors, has the potential to contribute to an increased immune response against cancer. An observed positive effect of treatment with antibodies against the receptor of PD-L1, PD1, in malignant melanoma, lung and kidney cancer supports this (see studies with nivolumab, *e.g.* Ther Adv Med Oncol. 7 (2): 85-96). Additional studies are needed to determine if also Kancera's HDAC6 inhibitors can achieve the desired immune stimulatory effect against cancer cells.

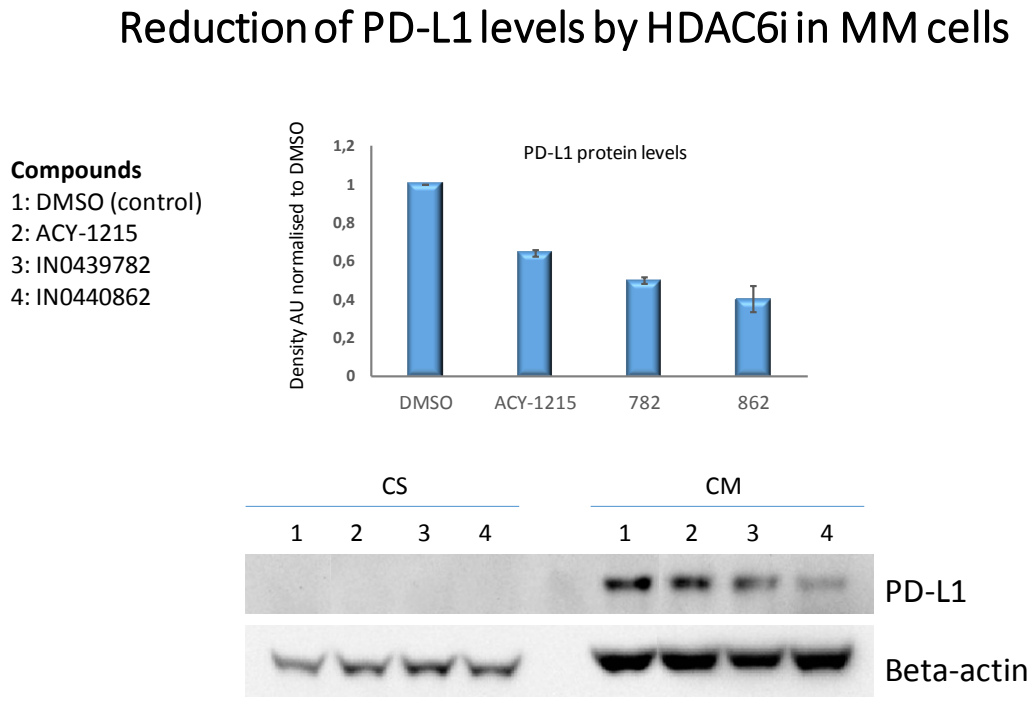


Figure 3. The Figure shows the level of the immunosuppressive protein PD-L1 after addition of various substances in the cells of the hematologic cancer multiple myeloma. The level of PD-L1 is measured as the degree of blackening (density) of this protein in a size exclusion analysis in relation to the reference protein beta-actin (shown at the bottom of the picture above) and described in a bar chart. CS represents the soluble fraction of the cancer cells and CM the cytoskeleton including the cell nucleus, where the immuno-active transmembrane fraction of PD-L1 is anchored to the cell skeleton. The results show that Kancera's HDAC6 inhibitors (782 and 862) reduce the amount of PD-L1 to a greater extent than the control treatment (DMSO) and the competing substance ACY-1215.

Kancera has also reported that the company in collaboration with SARomics Biostructures has succeeded to determine the crystal structure of Target 2 bound to Kancera's combined HDAC6 / Target 2 inhibitors. Through this structure determination at atomic level, Kancera obtains information on how drugs can be designed in a more optimal way to inhibit both HDAC6 and Target 2.

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.

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.

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. More than 150 new substances against parasitic target proteins have been synthesized since the start of the project. The academic groups in the consortium are continuously testing the effect of synthesized 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 target 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.

During the period, the project has commenced efficacy studies of a substance that has been developed by an academic partner within the consortium, for the treatment of schistosomiasis in a disease model in mice. Kancera has contributed with analyses of the drug properties of the substance, and established the formulation of the substance used in the present efficacy study. Thus, the consortium has taken a decisive step towards achieving the final goal of the A-PARADDISE project, which is to demonstrate the therapeutic efficacy of a substance developed within the project in a validated disease model for one of the world´s largest parasitic diseases.

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 USD 100 million 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 USD 1.7 billion. 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 2016, Acetylon has published clinical results showing that the company's HDAC inhibitor improves the treatment effect of Celgene's drug Pomalyst in an advanced stage of multiple myeloma. Despite these results, Celgene has chosen not to acquire Acetylon using the above referenced option agreement (according to a report from Xconomy commenting Celgene's notification in question to the American financial authority SEC), but will remain as shareholder of Acetylon.

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, the 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 SEK 3.5 to >10 billion 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, pancreatic cancer).

Consolidated Statement of Comprehensive Income

SEK 000´s (if otherwise not specified)	July-Sept		Jan-Sept		1 Jan-31 Dec
	2016	2015	2016	2015	2015
Kancera Group					
Revenues					
Net sales	**42**	**183**	**174**	**267**	**282**
Cost of sales & services	**-12**	**-46**	-43	-64	-74
Gross profit	**30**	**137**	**131**	**203**	**208**
Operating Expenses					
General & administrative expenses	**-616**	**-209**	-2 254	-1 488	-3 943
Selling expenses	**-15**	**-120**	-362	-457	-805
Research & development expenses	**-4 645**	**-3 249**	-13 300	-12 055	-20 355
					5 209
Total operating expenses	**-5 276**	**-3 578**	**-15 916**	**-14 000**	**-19 894**
Operating income	**-5 246**	**-3 441**	**-15 785**	**-13 797**	**-19 686**
Income from Financial Investments					
Financial net	**-114**	**23**	-85	56	74
Income after financial items	**-5 360**	**-3 418**	**-15 870**	**-13 741**	**-19 612**
Taxation	-	-	-	-	-
Net income	**-5 360**	**-3 418**	**-15 870**	**-13 741**	**-19 612**
Net income attributable to the shareholder´s of the parer	-5 360	-3 418	-15 870	-13 741	-19 612
Non-controlling interests	-	-	-	-	-
Average number of shares (thousands), before dilution	131 487	103 925	114 096	100 912	105 272
Average number of shares (thousands), after dilution	131 487	103 925	114 096	100 912	105 272
Number of shares at closing date (thousands)	131 487	103 925	131 487	103 925	103 925
Earnings per share, before and after dilution	-0,04	-0,03	-0,14	-0,14	-0,19

Comprehensive Income for the Period

SEK 000´s (if otherwise not specified)	July-Sept		Jan-Sept		1 Jan-31 Dec
	2016	2015	2016	2015	2015
Net income for the period	-5 360	-3 418	-15 870	-13 741	-19 612
Other comprehensive income, net before tax					
Total comprehensive income for the period	-5 360	-3 418	-15 870	-13 741	-19 612
Attributable to the shareholder´s of the parent company	-5 360	-3 418	-15 870	-13 741	-19 612
Non-controlling interests	-	-	-	-	-

Condensed Consolidated Statement of Financial Position

SEK 000´s (if otherwise not specified)

Kancera Group

	30 Sept		31 Dec
Assets	2016	2015	2015
Non-current Assets			
Intangible assets			
Capitalized R&D	6 000	6 000	6 000
Tangible assets			
Equipment and chemical library	2 307	3 425	3 145
Total non-current assets	**8 307**	**9 425**	**9 145**
Current Assets			
Work in progress	3 818	6 616	1 486
Trade receivables and other receiva	1 484	1 209	1 213
Cash and cash equivalents	63 494	20 155	15 567
Total current assets	**68 796**	**27 980**	**18 266**
TOTAL ASSETS	**77 103**	**37 405**	**27 411**
Equity and Liabilities			
Equity			
Equity	**65 860**	**27 798**	**21 925**
Provisions and Liabilities			
Long-term liabilities	2 898	1 500	1 500
Short-term liabilities	8 345	8 107	3 986
Total provisions and liabilities	**11 243**	**9 607**	**5 486**
TOTAL EQUITY and LIABILITIES	**77 103**	**37 405**	**27 411**

Consolidated Statement of Changes in Equity

SEK 000´s (if otherwise not specified)

Kancera Group	Sharecapital	Other capital contributions	Accumulated deficit	Total equity
Januari-June				
Opening balance 2015-01-01	8 212	35 056	-15 979	27 289
Comprehensive income				
Net income for the period			-10 323	-10 323
Transactions with shareholders				
Capital injection	448	13 594		14 042
Costs related to issue of shares		-255		-255
Employee stock option programme		403		403
Closing balance 2015-06-30	**8 660**	**48 798**	**-26 302**	**31 156**
Third quarter July-September				
Opening balance 2015-06-30	8 660	48 798	-26 302	31 156
Comprehensive income				
Net income for the period			-3 418	-3 418
Transactions with shareholders				
Employee stock option programme		60		60
Closing balance 2015-09-30	**8 660**	**48 858**	**-29 720**	**27 798**

Kancera Group, 1 January 2015-31 December 2015	Sharecapital	Other capital contributions	Accumulated deficit	Total equity
Opening balance 2015-01-01	8 212	35 056	-15 979	27 289
Comprehensive income				
Net income for the period			-19 612	-19 612
Transactions with shareholders				
Capital injections	448	13 594		14 042
Costs related to issue of shares		-425		-425
Employee stock option programme		631		631
Closing balance 2015-12-31	**8 660**	**48 856**	**-35 591**	**21 925**

Kancera Group, 1 January 2016-30 September 2016	Sharecapital	Other capital contributions	Accumulated deficit	Total equity
January-June				
Opening balance 2016-01-01	8 660	48 856	-35 591	21 925
Comprehensive income				0
Net income for the period			-10 510	-10 510
Transactions with shareholders				0
Capital injection	2 297	66 605		68 902
Costs related to issue of shares		-9 429		-9 429
Employee stock option programme		216		216
Closing balance 2016-06-30	**10 957**	**106 248**	**-46 101**	**71 104**
July-September				
Opening balance 2016-06-30	10 957	106 248	-46 101	71 104
Comprehensive income				0
Net income for the period			-5 360	-5 360
Transactions with shareholders				0
Capital injections		78		78
Employee stock option programme		38		38
Closing balance 2016-09-30	**10 957**	**106 364**	**-51 461**	**65 860**

Condensed Consolidated Statement of Cash-Flow

	July-Sept		Jan-Sept		1 Jan-31 Dec
SEK 000´s (if otherwise not specified)	2016	2015	2016	2015	2015
Kancera Group					
Cash-flow from operating activities					
Operating income after financial items	-5 360	-3 418	-15 870	-13 741	-19 612
Depreciation	279	393	838	809	1 088
Other non-cash-flow affecting items	38	151	361	554	631
Cash-flow from operating activities before working change	**-5 043**	**-2 874**	**-14 671**	**-12 378**	**-17 893**
Change in working capital	-3 206	-2 231	-2 733	-3 771	-2 765
Cash-flow from operating activities	**-8 249**	**-5 105**	**-17 404**	**-16 149**	**-20 658**
Investment activities					
Investment in tangible assets	0	0	0	-366	-366
Cash-flow from investment activities	**0**	**0**	**0**	**-366**	**-366**
FREE CASH-FLOW available to INVESTORS	**-8 249**	**-5 105**	**-17 404**	**-16 515**	**-21 024**
Financing activities					
Issue of shares/other capital infusions	78	-91	59 552	13 696	13 617
Financing from the EU/Vinnova	2 979	0	5 779	0	
Cash-flow from financing activities	**3 057**	**-91**	**65 331**	**13 696**	**13 617**
CASH-FLOW for the PERIOD	**-5 192**	**-5 196**	**47 927**	**-2 819**	**-7 407**
Cash and cash equivalents at the beginning of the	68 686	25 351	15 567	22 974	22 974
Cash and cash equivalents at the end of the perioc	63 494	20 155	63 494	20 155	15 567

Condensed Parent Company Income Statement

	July-Sept		Jan-Sept		1 Jan-31 Dec
SEK 000´s (if otherwise not specified)	2016	2015	2016	2015	2015
The Parent Company Kancera AB					
Revenues					
Net sales	**42**	**183**	**174**	**267**	**282**
Cost of sales & services	**-12**	**-46**	-43	-64	-74
Gross profit	**30**	**137**	**131**	**203**	**208**
Operating Expenses					
General & administrative expenses	**-616**	**-209**	-2 254	-1 488	-3 943
Selling expenses	**-15**	**-120**	-362	-457	-805
Research & development expenses	**-4 645**	**-3 249**	-13 300	-12 055	-20 355
	0	0	0	0	5 209
Total expenses	**-5 276**	**-3 578**	**-15 916**	**-14 000**	**-19 894**
Operating income	**-5 246**	**-3 441**	**-15 785**	**-13 797**	**-19 686**
Income from Financial Investments					
Financial net	**-114**	**23**	-85	56	**74**
Income after financial items	**-5 360**	**-3 418**	**-15 870**	**-13 741**	**-19 612**
Taxation	-	-	-	-	-
Net income	**-5 360**	**-3 418**	**-15 870**	**-13 741**	**-19 612**

Condensed Parent Company Balance Sheet

	30 Sept		31 Dec
SEK 000´s (if otherwise not specified)	2016	2015	2015
The Parent Company Kancera AB			
Assets			
Non-current Assets			
Intangible assets			
Capitalized R&D	6 000	6 000	6 000
Goodwill & Immateriella rättigheter			
Tangible assets			
Equipment and chemical library	2 307	3 425	3 145
Financial assets			
Shares in subsidiaries	50	0	0
Total non-current assets	**8 357**	**9 425**	**9 145**
Current Assets			
Work in progress	3 818	6 616	1 486
Trade receivables and other receivables	1 484	1 209	1 213
Cash and cash equivalents	63 444	20 155	15 567
Total current assets	**68 746**	**27 980**	**18 266**
TOTAL ASSETS	**77 103**	**37 405**	**27 411**
Equity and Liabilities			
Equity			
Restricted equity	10 957	8 660	8 660
Non-restricted equity	54 903	19 138	13 265
Total equity	**65 860**	**27 798**	**21 925**
Provisions and Liabilities			
Long-term liabilities	2 898	1 500	1 500
Intercompany payables	0	0	0
Short-term liabilities	8 345	8 107	3 986
Total provisions and liabilities	**11 243**	**9 607**	**5 486**
TOTAL EQUITY and LIABILITIES	**77 103**	**37 405**	**27 411**

Notes

Note 1. Accounting and valuation principles

As a consequence of the acquisition of the subsidiary Kancera Förvaltning AB prepared this Interim Report Q2 2016 in accordance with IAS 34 and the Annual Accounts Act. The consolidated financial statements have been prepared in accordance with International Financial Reporting Standards (IFRS) as adopted by the EU and the Swedish Annual Accounts Act. The accounting of the parent company has been prepared in accordance with the Annual Accounts Act and the Swedish Financial Reporting Board's recommendation RFR 2, including a number of new or revised standards, interpretations and improvements adopted by the EU and apply from January 1, 2016.

In the absence of regulations in K3, IAS 33 has been applied for calculating earnings per share.

The transition to IFRS / RFR 2 did not affect the income statement or balance sheet for the period January 1 - March 31, 2016 accounted for using the previous principles. Comparative figures used in the comments from the previous year refer to the parent company Kancera AB.

Balance Sheet **At balance date 2016-06-30**

The Kancera Group

SEK 000´s (if otherwise not specified)

	Accounting according to K3 Swedish GAAP	Adjustments	Accounting according to IFRS	
Assets				
Non-current Assets				
Intangible assets, activated R&D expenses	6 000	0	6 000	Capitalized R&D
Tangible assets				
Total fixed assets	2 586	0	2 586	Equipment and chemical library
Total non-current assets	**8 586**		**8 586**	**Total non-current assets**
Current Assets				
Work in progress	2 863	0	2 863	Work in progress
Receivables	2 154	0	2 154	Trade receivables and other receivables
Cash and cash equivalents	68 686	0	68 686	Cash and cash equivalents
Total current assets	**73 703**	0	**73 703**	**Total current assets**
TOTAL ASSETS	**82 289**	**0**	**82 289**	**TOTAL ASSETS**
Equity and Liabilities				
Equity				
Restricted equity	10 957			
Non-restricted equity	60 147			
Total equity	**71 104**	0	**71 104**	Equity
Provisions and liabilities				
Long-term liabilities	2 800	0	2 800	Long-term liabilities
Short-term liabilities	8 385	0	8 385	Short-term liabilities
Total provisions and liabilities	**11 185**	0	**11 185**	**Total provisions and liabilities**
TOTAL EQUITY and LIABILITIES	**82 289**	**0**	**82 289**	**TOTAL EQUITY and LIABILITIES**

Condensed Consolidated Statement of Financial Position

SEK 000´s (if otherwise not specified)

Kancera Group

Assets
Non-current Assets
Intangible assets
Tangible assets
Current Assets
Equity and Liabilities
Equity
Provisions and Liabilities

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 the previous 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 140,000 and SEK 75,000 to Allmora Life Science AB. Håkan Mellstedt, a Board member at Kancera, is the CEO and owner of F:a Mellstedt Medical. Charlotte Edenius, a Board member at Kancera, is the CEO and owner of Allmora Life Science AB. 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 counts on that these can be sold with income in the future.

In connection with the rights issue in June 2016 27,561,356 warrants were issued. Two (2) warrants of series TO2 entitles the holder to subscribe for one new share. The warrants are valid until April 30, 2018. During the period from October 1, 2016 - June 30, 2017 the exercise price is SEK 5 to subscribe for one share with the support of two warrants; during the remaining term until April 30, 2018 the strike price is SEK 6 to subscribe for one share, with the support of two warrants.

The dilution effect on the exercise of all warrants amounts to approximately 12.2%.

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	1 455	July 2017
EU PARADDISE	8 520*	7 487	March 2017
EU SYNTRAIN	4 462**	2 677	Sept. 2018
	10 520	**8 942**	

* Assuming an EUR exchange rate of SEK 8.95. The paid amount SEK 7,487,000 corresponds to 85% of the grant. An additional 15% of the grant is paid following an approved final report which will be submitted in March 2017.

** Assuming an EUR exchange rate of SEK 8.95. The paid amount SEK 2,677,000 corresponds to 60% of the grant. An additional 25% of the grant is paid following an approved report for period 1 which will be submitted in September 2018 and an additional 15% is paid following an approved final report which will be submitted in October 2020.

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, November 18, 2016

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

Financial calendar
- Press Release 2016: February 21, 2017
- Annual Report 2016: May 5, 2017
- Interim Report January 1 – March 31, 2017: May 19, 2017
- Interim Report January 1 – June 30, 2017: August 22, 2017
- Interim Report January 1 – September 30, 2017: November 17, 2017
- Press Release 2017: February 20, 2018

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