Press release February 19, 2016

Interim Report for Kancera AB (publ) Q4 2015

January 1 – December 31, 2015

The period January to December 2015 and the fourth quarter 2015 in brief

- R&D expenses for the period amounted to SEK 20.4m (SEK 13.7m) of which the fourth quarter constituted SEK 8.3m (SEK 4.1m). Following EU's approval of Kancera's mid-term report for the A PARADISE project, the project's revenues and expenses have been taken up which means that the reported R & D expenses and revenues for the period increased by SEK 4.8m. Thus, excluding the EU-project the R & D expenses for the period amounted to SEK 15.6m (SEK 13.7m), of which the fourth quarter constituted SEK 3.5m (SEK 4.1m).
- Operating income for the period amounted to SEK -19.7m (SEK -16.1m) of which the fourth quarter constituted SEK -5.9m (SEK -5.1m).
- Income after financial items for the period amounted to SEK -19.6m (SEK -16.0m) of which the fourth quarter constituted SEK -5.9m (SEK -5.0m).
- Earnings per share for the period were SEK -0.19 (SEK -0.18) of which the fourth quarter constituted SEK -0.06 (SEK -0.05).
- Cash flow from operating activities for the period amounted to SEK -20.7m (SEK -19.1m) of which the fourth quarter constituted SEK -4.5m (SEK -7.5m).
- Equity as of December 31, 2015 amounted to SEK 21.9m (SEK 27.3m) or SEK 0.21 (SEK 0.28) per share. The equity/assets ratio as of December 31, 2015 was 80 percent (75 percent).
- Cash and cash equivalents as of December 31, 2015 amounted to SEK 15.6m (SEK 23.0m). Cash and cash equivalents exclude the contribution of SEK 2.8m paid by the EU in January 2016.

Significant events during the period

- Kancera reported that a second efficacy study of the drug candidate KAN0439834 has been completed in an animal model of an advanced stage of chronic lymphocytic leukemia characterized by a genetic change which makes the disease more difficult to treat. The results show that KAN0439834 reduces the number of ROR expressing leukemia cells in the lymphatic system (spleen) after 14 days of treatment. Further, Kancera reported that a second patent application EP15153394.0 has been filed covering small-molecule ROR inhibitors, including the drug candidate KAN0439834.

- Kancera reported that the patent WO 2011/079902 concerning monoclonal antibodies against ROR1 has been approved in China. Kancera has acquired partial rights to this patent from Bioinvent under an agreement that does not involve any financial burden for Kancera (except patent expenses) before revenues are generated. Kancera through the company's co-founder Professor Håkan Mellstedt has been involved in the development of these antibodies. These antibodies have mainly been used to identify and validate new indications for a future ROR-inhibiting drug. Any further development of the ROR-targeted monoclonal antibodies for therapeutic purposes will only be done in a partnership that provides funding and access to expertise in development of antibody-based drugs.

- Kancera reported an operational update of the cancer projects ROR, PFKFB3, and HDAC6.

    - The ROR project reported that Kancera's candidate drug KAN0439834 is effective against both leukemic cells circulating in the blood and leukemic cells that have invaded the lymph nodes in humans.

    - Recent studies of clinical samples from leukemia patients underscore that ROR inhibitors mainly target the white blood cells causing cancer while the healthy white blood cells, including T cells, are spared. These results are of significance for the possibility to combine ROR inhibitors with the new generation of immuno-stimulating cancer drugs that have been developed since the effect of those requires functional T-cells.

    - A new generation of ROR inhibitors is being developed against solid tumors.

- The PFKFB3 project reported a new discovery showing that Kancera's PFKFB3 inhibitor KAN0438757 kills cancer cells by preventing them to repair their DNA. The discovery indicates that KAN0438757 could be an efficient complement to radiation for the treatment of advanced cancer.

- The HDAC6 project reported that Kancera's HDAC6 inhibitors counteract the migration of cancer-associated fibroblast cells and that an international patent application was filed in May.

- Kancera's Annual General Meeting on May 28, 2015 decided to re-elect the current Board of Directors and auditor (Ernst & Young). The General Meeting also decided to authorize the Board, on one or more occasions until the next Annual General Meeting, to issue new shares. A new share issue may be made with or without preferential rights and against cash payment and / or in kind or set-off. The purpose of the authorization and the reason for the deviation from shareholders' preferential rights is to enable the acquisition of capital for corporate acquisitions and the company's operation. If the share issue is made against cash payment and without preferential rights for the shareholders, the number of shares issued may not exceed ten percent of the total number of shares outstanding at the time the authorization is exercised.

- Kancera announced that a new share issue, with the authorization of the Annual General Meeting in 2014, was closed on May 27, 2015. The issue comprised a maximum of 4,927,386 shares. In total 25,926,793 shares were signed, of which 4,644,304 with preferential rights (with the support of subscription rights) and 21,282,489 without preferential rights. The share issue was thus oversubscribed to about 500 percent. This issue raised Kancera AB approximately SEK 12.3m before issue costs.

- Kancera announced that the first subscription period for the exercise of the employee warrants was closed in June 2015. In total 450,246 new shares were signed giving Kancera SEK 1.7m before issue costs. There remain 2,349,754 warrants, of which 560,000 are held by Kancera to cover social security costs that are part of the employee warrants program.

- Kancera announced that the company's HDAC6 project has been awarded a grant totaling SEK 2m from the Swedish innovation agency VINNOVA. The grant is directed to projects that can develop into new strong innovations in a range of common diseases, including cancer. The grant is paid on four occasions during the two-year project. The project will be implemented in collaboration with the Cancer Center Karolinska (CCK), and is also planned to involve Swedish companies such as SARomics Biostructures, MetaSafe and Adlego Biomedical.

- Kancera announced that the company has entered into an agreement with Acturum Life Science AB in order to evaluate and further develop the unique Fractalkine receptor antagonist AZD8797. Based on published research that supports that the Fractalkine receptor antagonist may have a central role in different cancer forms, Kancera will evaluate how efficiently the Fractalkine receptor antagonist AZD8797 may stop tumor growth and relieve severe cancer pain. The agreement with Acturum Life Science gives Kancera right to evaluate AZD8797 in preclinical studies and then to acquire the project. This agreement entails no expenses for Kancera apart from investments in the patent portfolio and in the scientific evaluation. If Kancera chooses to acquire the Fractalkine project, following the preclinical evaluation phase, the total payment to Acturum will consist of 6 million Kancera shares divided into three tranches, which are due at pre-defined success-milestones.

- Kancera provided an operational update on the ROR and Fractalkine projects:

  - In the ROR project, Kancera reported that follow-up studies of the pharmaceutical properties of KAN0439834 show that they probably are better than previously assumed with respect to uptake and penetration of the substance to the cancer. The new studies indicate that dosing 2-3 times a day at 65-300 mg gives a concentration in the body that may be sufficient to exert an effect on solid tumors. Against this background, ROR inhibitors will be tested in animal models of solid tumors. It was further reported that ROR inhibitors have shown effect against leukemic cells from bone marrow which is a capacity wanted since the existing drugs are not sufficiently effective against cancer cells in the bone marrow.

  - In the Fractalkine project, Kancera reported that a network of leading cancer and pain scientists that has been established that will evaluate the drug candidate KAN0440567 (AZD8797) in an advanced animal model closely resembling the human form of pancreatic cancer. Kancera has synthesized and quality controlled the salt form of the drug candidate that will be used in this study and has conducted a successful peroral dosing study in mice.

- Kancera provided an operational update on the PFKFB3 and HDAC6 projects as well as the EU-funded and epigenetically targeted anti-parasitic project A-PARADDISE:

- From the collaboration with Prof. Thomas Helleday, Kancera reported that Kancera's PFKFB3 inhibitor significantly reduces the size of a tumor formed by aggressive human breast cancer cells (so-called triple negative breast cancer) transplanted in zebrafish. The results from the study support that Kancera's PFKFB3 inhibitor is effective against these aggressive cancer cells if the substance reaches the tumor in sufficient concentration, which is easier to achieve in zebrafish than *e.g.* in mice.

- Kancera has developed several chemical families of potent and selective HDAC6 inhibitors based on a common scaffold, and Kancera reported the decision to withdraw the original patent application from 2014 in order to postpone the publication of the structures at least 12 months. This is done in order to prevent Kancera's existing patent application to become an obstacle to a new patent application covering the recently developed HDAC6 inhibitors.

- Vinnova announced in June 2015 that Kancera has been awarded a grant to support the further development of HDAC6 inhibitors against cancer. The first part of the grant was paid in July. VINNOVA decided to bring forward the second payment (SEK 750, 000) to the HDAC6 project to December 2015.

- In February 2014 Kancera received an initial payment from the EU amounting to € 523,655 (about SEK 4.6m) for the execution of the A-PARADDISE project. The project has now delivered a midterm report which has been approved by the EU. This means that a second installment of the grant was paid to Kancera at year-end according to plan. This payment amounted to € 300,000 (about SEK 2.8m).

Significant events after the end of the reporting 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.

    - The patent application covering ROR inhibitors filed in February 2015 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.

Statement from the CEO

2016 began as usual with an intense biotech week in San Francisco where Kancera and most of the Pharmaceutical and Biotech industry gathered to discuss cooperation opportunities in individual meetings. During the week in San Francisco, Thomson Reuters also presented its retrospection of 2015 which was a strong year for the industry with more and larger acquisitions than ever and a strong interest in preclinical drug development projects. The generally high interest from major pharmaceutical and biotech companies to acquire pharmaceutical project continues, which is reflected partly in the increasing level of payment upon signature of the acquisition or licensing agreements and partly in increasing total price tags for pharmaceutical projects. Cancer, by virtue of the great medical need, continues to be the therapeutic area in which most agreements are reached (for more information, see the Market Outlook section).

In the third quarterly report in 2015, I described the challenge in the ROR-project to achieve a sufficiently high concentration of the drug candidate in the blood to enable studies of efficacy against more cancer diseases, in addition to chronic lymphocytic leukemia, such as solid tumors. During the fourth quarter we have succeeded to develop a new series of compounds in the ROR project that can be maintained in an active concentration in the blood for 10 hours in mice. This can be compared with the approximately 2.5 hours shown by Kancera's first drug candidate KAN0439834 in the same type of measurement. This progress now provides us with new opportunities to test the effect of ROR inhibitors in several preclinical models of severe human cancers.

During the fourth quarter we have also been able to show that oral administration of the Fractalkine receptor antagonist KAN0440567 to mice effectively blocks the function of the Fractalkine receptor. This is a first step in the ongoing studies to examine the effect of this substance against cancer of the pancreas in a preclinical model of the disease.

Furthermore, in January we reported several reinforcements of Kancera's portfolio of patents and patent applications, including a new application from the HDAC6 project, a granted patent from the PFKFB3 project and a completion of the international patent application from the ROR project including new substances showing up to 20 times higher effect against leukemia cells compared to Kancera's first drug candidate.

Overall, we see progress in Kancera's entire project portfolio which further strengthens the company's competitiveness and business development efforts.

Thomas Olin

CEO Kancera

About Kancera AB (publ)
Kancera develops the basis for new therapeutics, starting with new treatment concepts and ending with the sale of a drug candidate to international pharmaceutical companies. Kancera's operations are based in the Karolinska Institutet Science Park in Stockholm and the company employs around 13 people. The Kancera shares are traded on NASDAQ OMX First North and the number of shareholders was around 7300 as of December 16, 2016. Remium Nordic AB is Kancera's Certified Adviser. Professor Carl-Henrik Heldin and Professor Håkan Mellstedt are board members and Kancera´s scientific advisors.

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

Financial development, summary

Financial development, summary

SEK 000´s (if otherwise not specified)

Kancera AB	1 Oct-31 Dec		1 Jan-31 Dec	
tkr (om ej annat anges)	2015	2014	2015	2014
Net turnover	15	50	282	470
Contributions from the EU/Vinnova	5 209	-	5 209	-
R&D expenses	-8 300	-4 129	-20 355	-13 692
Operating Income	-5 889	-5 079	-19 686	-16 095
Income after financial items	-5 871	-5 046	-19 612	-15 979
Net income	-5 871	-5 046	-19 612	-15 979
Cash-flow from operating activities	-4 509	-7 547	-20 658	-19 105
Earnings per share, before and after dilution	-0,06	-0,05	-0,19	-0,18
Cash on hand at closing date	15 567	22 974	15 567	22 974
Solvency ratio	80%	75%	80%	75%
Key ratios				
Return on equity, %	neg	neg	neg	neg
Return on capital employed, %	neg	neg	neg	neg
No. of employees	13	10	13	10
Earnings per share, before dilution	-0,06	-0,05	-0,19	-0,18
Earnings per share, after dilution	-0,06	-0,05	-0,19	-0,18
Equity by share, kr	0,21	0,28	0,21	0,28
Cash-Flow by share, kr	-0,04	-0,05	-0,07	0,10

Comments on the financial development

The increased R&D costs for the period compared to the corresponding period in 2014 can mainly be attributed to that more projects now are run in parallel with three additional employees and that costs for out-sourcing have increased in connection with the advancement of the product development in the ROR project and the HDAC6 project.

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 0.3 m (SEK 0.5m). The company also receives financial support from the EU project A-PARADDISE where the support is offset against incurred costs for the period amounting to SEK 4.8m of consumables, performed months of work plus 60% overhead on the sum of these costs as was summarized in a midterm report. The financial support from EU covers 75% of the project costs plus 60% overhead.

Expenses

Expenses in the fourth quarter amounted to SEK 11.1m (SEK 5.1m), which breaks down into costs of services sold of SEK 0.0m (SEK 0.1m), research and development expenses of SEK 8.3m (SEK 4.1m) and other sales and administrative expenses of SEK 2.8m (SEK 1.0m). Expenses during the period January 1 to December 31 amounted to SEK 25.2m (SEK 16.6m), which breaks down into costs of services sold of SEK 0.1m (SEK 0.3m), research and development expenses of SEK 20.4m (SEK 13.7m) and other sales and administrative expenses of SEK 4.7m (SEK 2.6m).

Following EU's approval of Kancera's mid-term report for the A PARADISE project, the project's revenues and expenses have been taken up which means that the reported R & D expenses and revenues for the period increased by SEK 4.8m. Thus, excluding the EU-project the R & D expenses for the period amounted to SEK 15.6m (SEK 13.7m), of

which the fourth quarter constituted SEK 3.5m (SEK 4.1m).

Earnings
Income after financial items for the fourth quarter amounted to SEK -5.9m (SEK -5.0m) and for the period SEK -19.6m (-16.0m).

As reported in the Annual Report 2014, the operating earnings decreased compared with the press release 2014 due to a revaluation of an option program for Board members and staff. The effect on earnings for the third quarter 2014 was SEK 618,000 and for the fourth quarter 2014 SEK 286,000, thus the sum effect for the entire 2014 is SEK 904,000. The effect of the adjustments on the quarterly reports as of September 30, and December 31, 2014 are reported in the comparative figures for 2015. The cost of the program during the fourth quarter amounted to SEK 77,000 (SEK 286,000).

Cash flow and liquidity
Cash flow amounted to SEK -4.6m (SEK -4.5m) in the fourth quarter. Cash flow from operating activities for the fourth quarter amounted to SEK -4.5m (SEK -7.5m). Cash flow from financing activities for the third quarter amounted to SEK -0.1m (SEK 3.1m).

Cash flow during the period totaled SEK -7.4m (SEK 8.9m). Cash flow from operating activities during the period amounted to SEK -20.7m (SEK -19.1m). Cash flow from financing activities during the period amounted to SEK 13.6m (SEK 28.6m).

In the first quarter 2014, Kancera was awarded a grant of € 523,655 (which represents the first installment and 55% of the total awarded grant) from the European Union's 7th Framework Program for the A-PARADDISE project that targets parasitic diseases. Ongoing work for the period amounting to SEK 1.5m 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, but due to the EU approval of the midterm report, it has been registered as revenue and settled against accumulated costs.

Cash and cash equivalents as of December 31, 2015 totaled SEK 15.6m (SEK 23.0m). Cash and cash equivalents exclude the grant of SEK 2.8m that was paid by EU in January 2016.

Investments
Investments in fixed assets in the fourth quarter totaled SEK 0.0m (SEK 0.1m) and for the period net SEK 0.4m (SEK 0.6m).

No investments in intangible assets were made in the period.

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 8.3m (SEK 4.1m) for the fourth quarter.

Equity and share data

Total equity as of December 31, 2015 was SEK 21.9m (SEK 27.3m).

Share capital as of December 31, 2015 amounted to SEK 8 660 446,97 spread over 103 925 364 shares with a quotient value (rounded off) of SEK 0.0833 per share.

Earnings per share for the fourth quarter, based on a weighted average of the number of outstanding shares, were SEK -0.06 (SEK -0.05).

The equity/assets ratio as of December 31, 2015 was 80 percent (75 percent). Total equity per share was SEK 0.21 (SEK 0.28) based on total equity divided with the number of shares on the balance sheet day at the end of the quarter.

The Board and the CEO propose that no dividend be paid.

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 for the income year 2014 amount to SEK 75.2m.

Personnel
Kancera AB had 13 full time employees (13) as of December 30, 2015 of which 9 are men and 4 are women.

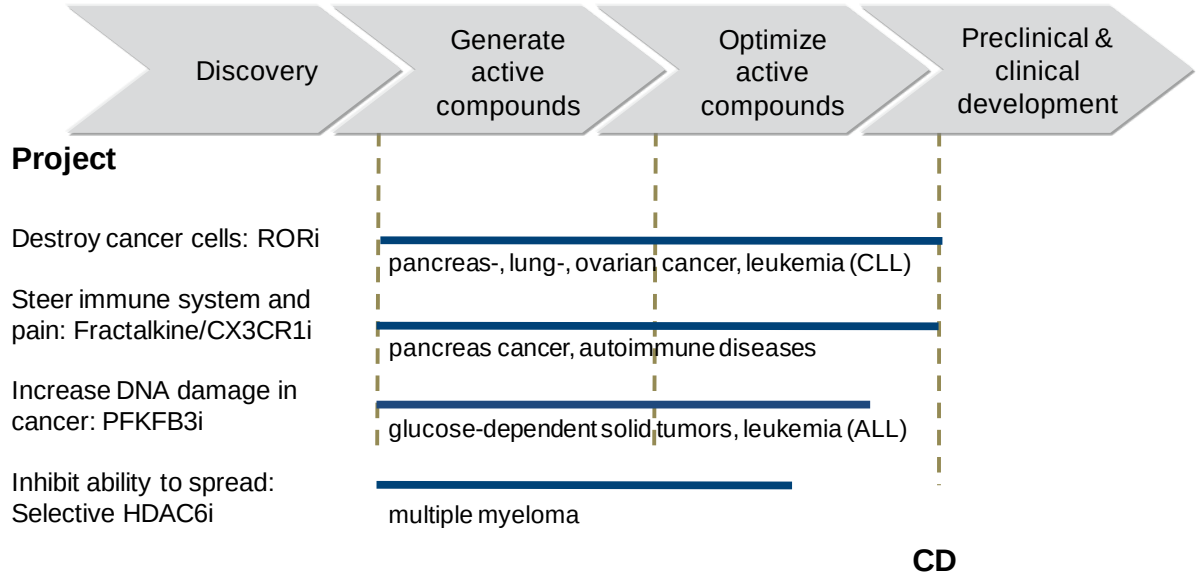
Pharmaceutical Development

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

The company has five drug development projects in the portfolio.

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

Figure 1. Kancera´s cancer project portfolio



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

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

In parallel, KAN0439834 will be tested in new efficacy and safety models. Kancera's research shows that there is an opportunity to create additional value in the project for the small-molecule ROR inhibitors why new formulations of KAN0439834 and analogues of this substance have been successfully developed. However, the road towards commercialization is still risky since increasingly advanced safety- and efficacy studies are performed in order to clarify the product's commercial value and to meet the requirements for clinical trials. A successful commercialization may mean that the risk and cost of these studies are shared with a partner and that Kancera obtains a stepwise

compensation at signing of the agreement and when the project reaches milestones. However, Kancera has not established a timeline for the commercialization of the ROR project.

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

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

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

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

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

R & D costs amounted to SEK 8.3m* (SEK 4.1m) for the fourth quarter 2015 which has been recognized as costs in its entirety.

* Following approval of the mid-term report for the A PARADDISE project, the project's revenues and expenses have been recognized, which means that the reported R & D costs and revenues for the period increased by SEK 4.8m. Thus, R & D expenses for the fourth quarter was SEK 3.5 million (SEK 4.1 million).if the EU project is excluded.

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

Since ROR is present in higher amounts in cancer cells from refractory patients and is selectively found in cancer cells and not in the surrounding healthy tissue, the Kancera project offers good possibilities to develop effective drugs with fewer side effects that may contribute to increased quality of life for patients and lower costs for society.

Kancera develops synthetic compounds that enter the tumor cell and work on the part of the ROR receptor that is inside the tumor cell, with the aim of blocking the cancer cell's survival signal and thus re-program the cancer cells so that they destroy themselves.

A comparative study has been performed with four successful drugs (Dasatinib, Gefitinib, Sorafinib, Sunitinib) in order to examine the competitiveness of ROR inhibitors. The results show that these four drugs are unable to efficiently inhibit ROR1 and that they kill cancer cells from leukemia patients less selectively compared to ROR inhibitors. Further, the study shows that these drugs also kill healthy white blood cells, which cause the patient to become more susceptible to infections and that a high enough dose of the drug can be administered to obtain optimal effect on the tumor.

Kancera´s ROR inhibitors have been shown to be more effective and more selective when killing cancer cells from leukemia patients than comparable classes of reversible cancer drugs that inhibit the kinases BTK, PI3K and Syk. In collaboration with Professor Håkan Mellstedt and his research group at Karolinska Institutet, Kancera studied how effective these competing candidate drugs kill cancer cells derived from patients with chronic lymphocytic leukemia (CLL, the most common form of leukemia in adults) whose cancer is no longer sensitive to one of today's most widely used small molecule drug (Fludarabine). This study included leukemia cells from 7patients and compared the killing effect of Kancera´s ROR inhibitor KAN0439363 with the effect of four newly developed drugs including Ibrutinib (PCI-32765). The competing kinase inhibitors studied reached maximum ca 15-50% killed cancer cells at a concentration of about 5 µM while Kancera´s ROR inhibitor show higher effect at a lower concentration. Kancera's drug candidate, KAN0439834, kills 70% of the cancer cells at a more than ten-fold lower concentration, 300 nM. It should, however, be emphasized that the study does not indicate whether the competing substances have an improved effect over a longer time course, but Kancera´s negative result for Ibrutinib agrees with published findings showing that the cancer can develop resistance against Ibrutinib (Chang et al. ASCO 2013). In this therapy situation, Kancera's ROR-inhibiting drug may have a place in the treatment of resistant disease. Independent of Kancera, Professor Thomas Kipps at the University of California San Diego has showed that ROR-inhibition may become an important treatment of the severe cancer form acute myeloid leukemia (AML). Together with Kancera´s own studies, this shows that ROR inhibiting substances have the potential to combat both the most common chronic and the acute form of blood cancers (CLL and AML, respectively).

Kancera reported that KAN0439834 was selected as a first candidate drug in the ROR project. The candidate drug was selected on the basis of results from *in vivo* studies of both efficacy and safety of treatment with KAN0439834. The evaluation of this efficacy study is based on analysis of leukemia cells using flow cytometry and protein analysis. In addition, an analysis of possible side effects was performed. The results show that the number of human leukemia cells and ROR-bearing cells has been reduced by approximately 75% after seven days of daily oral administration of 40 mg/kg of KAN0439834. Protein analysis was carried out with the help of markers of ROR1-activation in cancer cells as well as apoptosis (cellular self-destruction). The results of the protein analysis show that the animals that were treated with 40 mg/kg of KAN0439834 orally per day have reduced ROR1 activity and an increase of apoptosis. Tolerance studies show that healthy cells from the spleen are not affected by treatment with KAN0439834 at the dose used, supporting that the effect of this substance is mainly directed against cancer cells. A clinical chemistry analysis of 17 markers in the blood of treated animals shows an indication from one marker of some side effect on the liver. However, this indicated effect on the liver can be avoided by further development of the formulation of KAN0439834. See “Events during the period”.

Kancera has applied for intellectual property protection for small-molecule ROR inhibitors by two patent applications: EP13180941.0 and EP15153394.0.

International research shows that many types of solid tumor cells can be ROR dependent. Kancera, in collaboration with Professor Håkan Mellstedt´s and Professor Matthias Löhr´s research groups at Karolinska Institutet, has found that Kancera´s substances effectively kill pancreatic cancer cells. Pancreatic cancer affects more than 100 000 patients anually in Europe and USA. The survival rate among these patients is less than two per cent five years after diagnosis. As with leukemia it has been demonstrated also for pancreatic cancer that ROR1 levels increase in tumor cells of patients with progressive (aggressive) cancer.

In parallel, independent researchers from the U.S. and Japan have shown that ROR is a promising target for development of drugs also against breast cancer and lung cancer (Yamaguchi et al, Cancer Cell 2012, Zhang et al, PLoS One 2012), indicating a potentially wide range of use for a future ROR inhibiting drug. This is supported by results published by Karachaliou *et al.* (Karachaliou *et al*., Translational Lung Cancer Research Vol 3, No 3, June 2014) showing that the drug Erlotinib™ is able to slow down lung cancer progression significantly better if the tumor contains low levels of ROR1 while high levels of ROR1 is associated with a more rapid disease progression. The results imply that there is reasonable to examine whether Erlotinib can cooperate with Kancera's ROR inhibitor to slow down or kill resistant lung cancer cells.

Kancera has also evaluated the possibilities of developing a vaccine against the ROR receptor. Studies have shown that certain peptide sequences found on the outside of the ROR protein gave an immune response in rat that selectively killed leukemic cells from patients. However, the effect of this immune response against leukemic cells was significantly weaker than the effect of Kancera's small molecules. Further studies have shown that leukemia patients have the ability to immunologically react against ROR by generating antibodies against the parts of the ROR protein (ROR peptides) which Kancera previously identified as potential starting points for vaccine development. Thus, these patients' immune system reacts spontaneously to peptide sequences overlapping with Kancera's selected vaccine candidates. These

observations prompted Kancera to start a new vaccine study to test methods to enhance the immune response to selected ROR-peptides in order to evaluate whether a sufficiently strong immune response can be generated against cancer or if small molecules ROR inhibitors are to be preferred. 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 also developed a first generation of diagnostic antibodies that allow the identification of patients who may benefit from Kancera´s future cancer treatment directed against ROR. This will guide future clinical studies and demonstrate the commercial value of the ROR-inhibiting drug.

By an agreement with Bioinvent AB, Kancera has secured rights to both human monoclonal (exclusive rights to the patent application WO 2012/076727) and mouse monoclonal (partial rights to the patent application WO 2011/079902) antibodies against ROR1. The acquisition of the patent rights is based on an agreement with Bioinvent that does not involve any financial burden for Kancera (except future patent expenses) before revenues are generated. Kancera, through the company's co-founder Professor Håkan Mellstedt, has been involved in the development of these human monoclonal antibodies directed against ROR. These antibodies are currently used primarily to identify and validate new indications for future ROR-inhibiting drugs. Any further development of the ROR-targeted monoclonal antibodies for therapeutic purposes will only be done in a partnership that provides funding and access to expertise in development of antibody-based drugs.

Events during the period

Kancera reported that a second efficacy study of the drug candidate KAN0439834 (small molecule inhibitor of ROR1) has been completed in an animal model of an advanced stage of chronic lymphocytic leukemia. The completed animal study is based on a cancer model in which human cells from an aggressive form of chronic lymphocytic leukemia were introduced to immune-deficient mice. This animal model is considered by leading scientists to be of clinical relevance and therefore suitable for evaluation of new drugs for the treatment of chronic lymphocytic leukemia, despite the inherent limitations and variations seen in the model (Bertilaccio et al : Xenograft models of chronic lymphocytic leukemia: problems, pitfalls and future directions. Leukemia 27:534-540, 2013).

After 14 days of treatment with KAN0439834 the number of leukemia cells was reduced by an average of 50% in the treated animals as compared to the control group that did not receive treatment. This is a statistically significant effect. A newly developed crystalline formulation of KAN0439834 was orally administered to the animals once daily resulting in a blood drug concentration that was sufficient for a significant reduction in number of leukemia cells. A toxicological evaluation of the new formulation was conducted by histopathology on 10 organs. The results from this evaluation indicated a possible mild side effect in the kidney but no tissue damage in the examined organs. The analyses of the liver histology and blood markers of liver function show that the indications of effects on the liver that were observed in November 2014 when using a different formulation, are not present with the new crystalline formulation of KAN0439834. Protein analyses showed that the amount of activated ROR1 was significantly reduced, suggesting that also in this study, treatment with KAN0439834 has had the desired effect on leukemia cells with ROR1 as target.

Overall, Kancera's report from November 3, 2014 shows that a seven-day treatment with KAN0439834 significantly reduces leukemia cells in the lymphatic system in an animal model of a progressive phase of chronic lymphocytic leukemia. The present study, which was reported on February 3, 2015, shows that KAN0439834 is active also in a phase of chronic lymphocytic leukemia characterized by a genetic change (17p deletion), which makes the disease more difficult to treat. Moreover, the same study shows that a newly developed formulation of crystalline KAN0439834 has improved properties both in terms of absorption in the body as safety compared to the previously used formulation.

The submitted national patent application (EP15153394.0), which includes newly invented small molecule ROR inhibitors, will be converted to an international patent application after 12 months. In parallel with the submission of this new patent application, Kancera has managed to postpone the publication of the company's first patent application EP13180941.0. The purpose of this is to extend the time when it is possible to broaden the scope of the patent applications and thus increase their commercial value.

The progress in the ROR-project continues in 2015 with studies in several animal species which have made it possible to calculate how Kancera's drug candidate KAN0439834 may function in the body of a patient. The calculations show that Kancera's drug candidate is likely to possess characteristics that provide a desired effect against cancer in humans

with an oral therapy 2-3 times per day.

Kancera also developed a new group of small molecule ROR inhibitors that are chemically similar to the drug candidate KAN0439834 but are smaller in size and show a three-fold higher killing effect *in vitro* against cancer cells from solid tumors such as pancreatic cancer. Thereby Kancera now takes steps towards addressing one of the cancers that are most difficult to treat.

Animal studies, performed in cooperation with Professor Håkan Mellstedt's research group at the Karolinska Institute, support that Kancera's candidate drug is effective against both leukemic cells circulating in the blood and leukemic cells that have invaded the lymph nodes and are more difficult to treat. These results are supported by studies of lymph node, blood and bone marrow samples from patients. The results show that Kancera's ROR inhibitors are efficient against cancer cells from all three studied tissues and thus show an effect profile that is different from currently marketed drugs such as Ibrutinib and Idelalisib. Kancera has shown that the ROR inhibitors mainly target cancer cells while the healthy immune cells are spared. These results are of significance for the ability of patients to counteract infections and also open up the possibility to combine ROR inhibitors with the new generation of immuno-stimulating cancer drugs that are currently being developed since the effect of these drugs requires healthy white blood cells.

A summary of the effect profile of the small molecule ROR inhibitors was presented by Professor Håkan Mellstedt at the ASCO (American Society for Clinical Oncology) meeting in Chicago in June, 2015.

Further analyses were undertaken to evaluate the killing effect of Kancera's new generation of ROR inhibitors on pancreas cancer cells that exhibit resistance to the company's drug candidate KAN0439834. The results from these studies show that the effect of the ROR inhibitors against solid tumor cells has been increased significantly. Ongoing work is directed towards improving the properties that determine how effective the new ROR substances are taken up and distributed in the body. This will be followed by efficacy studies.

Kancera reported that follow-up studies of the pharmaceutical properties of KAN0439834 show that they probably are better than previously assumed with respect to uptake and penetration of the substance to the cancer. The new studies indicate that dosing 2-3 times a day at 65-300 mg gives a concentration in the body that may be sufficient to exert an effect on solid tumors. However, the effect of KAN0439834 on solid tumors cannot be demonstrated in mice since the substance is metabolized too quickly in that species. Against this background, KAN0439834 and similar substances will be tested against human solid tumors that are developed in an established model for tumor growth and metastazing – the zebra fish. In this way it is possible to study the effect at concentrations expected to be achieved in humans.

Furthermore, the effect of KAN0439834 and three new drugs have been evaluated on tumor cells isolated from blood, lymph and bone marrow from 9 patients. The background to the study is that new drugs such as Ibrutinib and Idelalisib give effect in 70-80% of the patients with chronic lymphocytic leukemia. However, so-called complete remission (the symptoms have disappeared) has only been reached in a small number of these patients. This lack of effect is particularly evident in the bone marrow. 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. In the present study, conducted by Prof. Håkan Mellstedt's group at the Karolinska Institute, it is shown that Kancera's ROR inhibitor is also effective in killing CLL cells from bone marrow which is a characteristic sought as a complement to today's registered drugs against CLL

Events after the end of the period

In February 2015, 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 a new series of compounds have been developed which exhibit high efficacy against primary cells from patients with chronic lymphocytic leukemia, as well as improved pharmaceutical properties which allow Kancera to assess the effect of ROR inhibitors in several preclinical models of severe human cancers.

Table 1 below presents the properties of the new compound series in comparison with Kancera's first drug candidate KAN0439834. The new compound series a) is more potent against leukemia cells and affects healthy blood cells to a lesser extent, b) exhibits a higher metabolic stability in liver cells from both mouse and human, and c) remains available

in the blood circulation four times longer compared to KAN0439834. Both KAN0439834 and substances in the new series exhibit good oral bioavailability, indicating that they can be developed to be given in the form of pills.

Table 1

Compound	a) Effect in cells (IC_{50}*) CLL cells/ healthy cells	b) Metabolic stability in liver cells (CLINT** for mouse/human)	c) Concentration in blood ($t_{1/2}$*** in mice)
KAN0439834	250 nM/ 18 µM	20/ 15	2.5 hours
New compound series	100-150 nM/ >20 µM	10/ <<10	10 hours

** IC_{50} = the compound concentration giving 50% killing of cells.*
*** Clint = the speed of degradation of a compound expressed as µl/minute/one million liver cells.*
**** t1/2 = time required to decrease the compound concentration in the blood by half.*

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.

The Fractalkine project – a candidate that control the immune system in inflammation and cancer

Published research points to that Fractalkine signaling probably contributes to the growth and spread of tumors and the pain that often affects cancer patients. In addition, the presence of Fractalkine has been proposed to be associated with a lack of efficacy of immuno-oncology drugs. Taken together, these findings provide a new perspective of Fractalkine signaling as a target for cancer drug development. Kancera will now evaluate how efficiently the Fractalkine receptor antagonist AZD8797 may stop tumor growth and relieve severe pain.

Originally, the candidate drug AZD8797 was successfully developed by AstraZeneca in Södertälje as an effective inhibitor of Fractalkine signaling. The present documentation of AZD8797 includes drug properties, safety, toxicology, and production. Kancera's assessment is that this documentation is likely to meet requirements for an application to undertake clinical trials against cancer. AstraZeneca originally developed AZD8797 against multiple sclerosis and showed effect of AZD8797 in a preclinical model of the disease (see the publication in PNAS April 8, 2014 vol. 111, no. 14, p 5409). Acturum Life Science acquired the rights to the Fractalkine project from AstraZeneca as part of Acturum's acquisition of the research facility in Södertälje. However, AstraZeneca has retained the rights to develop Fractalkine receptor antagonists against respiratory diseases.

Since AZD8797 already meets the pharmaceutical properties Kancera considers necessary for the biological evaluation of the effect against cancer, the project can be run without significantly affecting the resource allocation to Kancera's other projects.

Events during the period

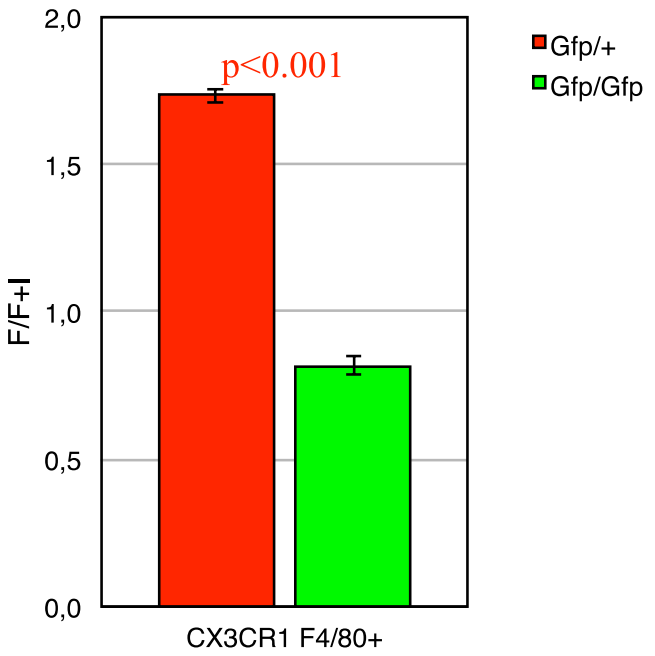
Since AZD8797 already meets the pharmaceutical properties Kancera considers necessary for the biological evaluation of the effect against cancer, the project can be run without significantly affecting the resource allocation to Kancera's other projects.

Events after the end of the period

In a first step in a series of planned studies which will examine whether Kancera's Fractalkine receptor antagonist can be an anticancer drug, Kancera conducted a study in transgenic mice. The results presented in Figure 2 show that KAN0440567 in a selective and effective way blocks the effect of Fractalkine signaling on *e.g.* immune cells called macrophages. Independent research has shown that Fractalkine signaling in cancer contributes to the reprogramming

of macrophages from being a threat to the tumor to aid the tumor. Thus, it may be desirable to block the effect of the Fractalkine signaling in cancer.

Figure 2. The figure displays two bars representing the blockade of the effect that Fractalkine provides on macrophages (cells that have the marker F4/80). In this case, the effect is measured as the macrophage's ability to move towards Fractalkine which has been administered to the animal. The study was conducted both on mice that lack the receptor mediating the effect of Fractalkine (CX3CR1) and on mice that have the functional receptor for Fractalkine. The group represented by the green bar (GFP/CFP) lacks CX3CR1 and the group represented by the red bar has functional CX3CR1. Both groups of mice were treated with Fractalkine and KAN0440567 (F+I). The larger the difference from 1.0, the more different the group is compared to a control group only treated with Fractalkine (F). The results show that KAN0440567 blocks the macrophage's reaction to Fracalkine only if CX3CR1 is available on the macrophage. The effect of KAN0440567 is thus significant and selective.



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

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. Through extensive crystallography studies Kancera has been established as an international leader in structure-based design of drugs targeting the PFKFB family of enzymes. Kancera has also reported a synergistic inhibitory effect on cancer cells of PFKFB3 inhibitors in combination with cisplatin (a commonly used cytostatic) in the laboratory and reported an inhibitory effect of Kancera´s PFKFB3 inhibitors on tumor growth in an animal study of pancreatic cancer. Two independent patent applications are registered in order to protect Kancera´s PFKFB3 inhibitors. The next step in the project is to improve the ability of the PFKFB3 inhibitors to penetrate the tumor.

During 2013 Kancera initiated a collaboration with Professor Thomas Helleday and his research group at Karolinska Institutet and the Science for Life Laboratory (SciLifeLab). In the collaboration Professor Helleday and Kancera combine their strengths in research on disease mechanisms and product development in order to investigate the functions of PFKFB3 in the cell nucleus in processes such as DNA replication and DNA repair. This information will be important both to identify biomarkers that can predict which patients will respond to the treatment and also to improve understanding of how inhibitors can best be combined with existing or new cancer treatments.

The partnership means that Kancera contribute know-how and drug-like PFKFB3 inhibitors while Professor Helleday´s research team invest their own resources in the project to investigate the best combination with other drugs, mechanisms of how PFKFB3 inhibitors act, as well as markers that show how and when a future drug is best used. In a future out-licensing or sale of the project Kancera shall compensate the scientists in proportion to the work performed. Within the collaboration Kancera retains exclusive ownership of its PFKFB inhibitors. An agreement has been reached between Kancera and the researchers providing Kancera exclusive rights to acquire inventions that may arise within the framework of the collaboration.

Kancera reported that a large-scale laboratory evaluation of synergistic effects between Kancera's PFKFB3 inhibitors and a large number of approved drugs has been performed as a part of the collaboration between Kancera and Professor Thomas Helleday´s research team. The results show that a synergistic effect against cancer cells can be

achieved by combining PFKFB3 inhibitors and some defined classes of approved drugs.

Kancera has also announced that the collaboration project with the Science for Life Laboratory (SciLifeLab) around the PFKFB3 protein has been awarded a grant of 436 561 SEK from Vinnova. The grant is coordinated by the Innovation Office at Karolinska Institutet and funds research conducted by Professor Thomas Helledays research team at Karolinska Institutet and SciLifeLab.

Events during the period

Kancera reported that the collaboration with Professor Thomas Helleday's research group at SciLifeLab has led to a surprising discovery showing that Kancera's PFKFB3 inhibitor KAN0438757 prevents cancer cells to repair the DNA following treatment with *e.g.* radiation. When the cancer cell is unable to repair its DNA it will die. This opens up for a new type of treatment of radiation resistant cancer, which combines the currently available DNA-damaging treatments (chemotherapy or radiation) with a PFKFB3 inhibitor. This new treatment concept is supported by studies in cancer cells showing that PFKFB3 contributes to the ability of the cancer to resist treatment. Thus, a PFKFB3 inhibitor could have the function to amplify the effect of cancer treatments such as radiation treatment. Additional basic research studies are necessary in order to fully understand the capabilities and limitations of a cancer treatment that combines Kancera's PFKFB3 inhibitors and radiation. However, it is clear that this new discovery provides strong reasons to examine the possibilities of an improved cancer treatment.

The results from the study of Kancera's PFKFB3 inhibitors was presented in June 2015 by Dr. Nina Sheppard at the scientific meeting Tomas Lindahl Conference on DNA Repair in Oslo with the title "Inhibition of the glycolytic enzyme PFKFB3 kills cancer cells by modulating DNA repair".

Results from the collaboration between Kancera and Prof. Thomas Helleday show that Kancera's PFKFB3 inhibitor significantly reduces the size of a tumor formed by aggressive human breast cancer cells (so-called triple negative breast cancer cells) transplanted into zebra fish. The results from the study support that Kancera's PFKFB3 inhibitor is effective against these aggressive cancer cells provided that the substance reaches the tumor in sufficient concentration, which is easier to achieve in zebra fish than *e.g.* in mice.

Besides investments in the national phase of patent applications covering PFKFB inhibitors for the time being there will be no further investments of significance in the chemistry development part of the PFKFB3 project until adequate funding has been secured.

Events after the end of the period

Kancera reported that a patent covering small molecule PFKFB3 inhibitors has been approved in the USA. To complement the patent approved in the United States (patent number US9233946), Kancera intends during the spring to file a divisional application for the use of anti-cancer PFKFB3 inhibitors by counteracting the ability of cancer cells to repair their DNA.

The HDAC6 project - a candidate acting against cancer by controlling the cancer cell's genome and mobility

Histone deacetylases (HDACs) are primarily involved in removing the acetyl groups from the so-called histones that are an essential part of how our genome is stored in the cell nucleus. Some HDACs also affect cell function outside the cell nucleus. HDAC6 belongs to that group of HDACs with its major biological role as regulator of the cytoskeleton and mechanical properties of the cell which are closely linked to the formation of tumors and metastases.

The link to tumor formation is partly explained by the fact that several so-called "oncogenes" such as "Ras" are dependent on a functional HDAC6 which allows the cancer cell to divide freely without being part of a tissue. Active HDAC6 also affects the tumor's ability to invade surrounding healthy tissue and metastasize. Larger amounts of active HDAC6 lead to an increased division of the cancer cells and increased metastasis. This property of HDAC6 is attributed partly to that the enzyme contributes to the growth of circulating cancer cells in e.g. blood, and partly to that high HDAC6 activity increases the cancer cell´s ability to move and to resist mechanical stress. HDAC6 has also been shown to be a valuable marker indicating how difficult the cancer in an individual patient will be 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 and therefore is an attractive target for development of new effective drugs against cancer.

The use of HDAC inhibitors in the treatment of cancer patients has so far shown promising results, but has been limited due to severe side effects. For this reason, the pharmaceutical industry is now looking for HDAC inhibitors with a higher level of selectivity within this family of enzymes. Kancera´s discovery of selective HDAC6 inhibitors may provide a

solution to how physicians could take advantage of HDAC inhibitors in the treatment of cancer without causing the patient severe side effects.

There are currently five HDAC inhibitors on the market for the treatment of various forms of T-cell lymphoma, 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. stomach and intestine. Also, the risk of significant negative impact on cardiac function is considered to be large. Selective inhibition of HDAC6 is expected to reduce these side effects, while activity against cancer cells is maintained.

During the second quarter 2014 Kancera commenced chemical synthesis in order to further develop the company´s HDAC6 inhibitors with the goal of delivering a competitive candidate drug. The development has led to inventions claimed in the patent application EP14167988.6. Kancera developed new HDAC6 inhibitors that exhibit an approximately 10-fold higher potency to kill tumor cells from multiple myeloma compared with the previous HDAC6 inhibitors from Kancera. Furthermore, these compounds showed higher potency and selectivity in vitro against cancer cells from multiple myeloma compared to Acetylon´s corresponding inhibitor ACY-1215.

In order to enable the development of a unique pharmaceutical drug Kancera has investigated possible mechanisms behind the high level of potency and selectivity of the company's HDAC6 inhibitors against cancer cells. In a first step, Kancera's compounds were evaluated in an in-vitro toxicology study against about 50 known risk factors. In this study Kancera´s HDAC6 inhibitors exhibited a remarkably high level of selectivity since no significant effect was detected against any of these risk factors. In a second step of the investigation it was examined whether Kancera´s HDAC6 inhibitors affect any of about 100 selected molecular mechanisms of action. Results show that Kancera 's HDAC6 inhibitors exhibit a significant effect on only one of the studied mechanisms of action in addition to HDAC6. During 2014 this particular mechanism of action has attracted attention as a new promising opportunity to treat cancer by blocking the formation of new cancer cells. Kancera does not communicate the identity of this new mechanism of action since the results indicate that the discovery can be utilized to further enhance the competitive edge of the HDAC6 project and to form the basis for a new proprietary cancer project. With adequate resources allocated to the HDAC6 project the company predicts that a candidate drug can be delivered in approximately 15-21 months. The next step is now to evaluate how the new mechanism can be combined with inhibition of HDAC6 to fight intractable cancer.

Events during the period

In December 2014 Kancera reported that the company's HDAC6 inhibitors act selectively through an additional mechanism *via* a not yet disclosed target protein (Target 2), which may contribute to the inhibition of cancer cell survival. In order to evaluate this potential united action, Kancera has now designed and synthesized compounds that only inhibit HDAC6 and compounds that inhibit both HDAC6 and Target 2. When the patent application for the HDAC6 inhibitor that was filed last year enters the international phase in May 2015 it will provide protection for these new compounds.

In 2015, Kancera, in collaboration with Dr Li-Sophie Zhao Rathje at the Karolinska Institute, has performed laboratory studies demonstrating that Kancera's HDAC6 inhibitors selectively counteract the migration of the cells that normally surround tumors (so-called cancer-associated fibroblasts). This finding indicates that Kancera's HDAC6 inhibitors could make it more difficult for these cells to migrate to the tumor and create a surrounding protection against medical treatment and the body's immune system.

Kancera has successfully developed several chemical families of potent and selective HDAC6 inhibitors based on a common scaffold, and it was reported that Kancera decided to withdraw the original patent from 2014 in order to postpone the publication of the structures at least 12 months. This is done in order to allow the preparation of a new supplementary patent application that will strengthen the company's IP position.

In June 2015, Vinnova announced that a grant was awarded to Kancera to support the further development of HDAC6 inhibitors against cancer.

Events after the end of 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. Figure 3 shows that 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

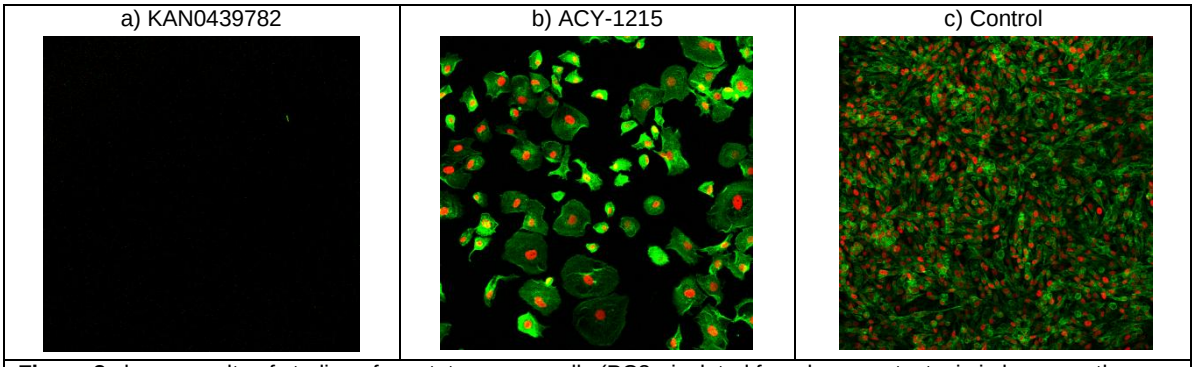


Figure 3 shows results of studies of prostate cancer cells (PC3 - isolated from bone metastasis in humans, these cells readily spread to surrounding tissues). The PC3 cells have been exposed for a week to a) Kancera's HDAC6 inhibitor KAN0439782 (10uM concentration), b) Acetylon's HDAC6 inhibitor ACY-1215 (10uM concentration) which is now tested in clinical trials against cancer, and c) a control containing no active ingredient. The PC3 cells have been colored so that the nucleus is visible in red and the cytoskeleton in green. Picture a) shows that Kancera's KAN0439782 makes the PC3 cells detach and die while Picture b) shows that some PC3 cells treated with AC-1215 survive and spread out over the surface. Picture c) shows the number and appearance of PC3 cells without treatment.

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

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

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

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

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

Events during the period

The EU consortium has qualified a new target protein for drug development against parasites. Kancera has developed a method of analysis in order to prepare for a high-throughput screen (HTS) in order to identify attractive starting points for drug development. This HTS was conducted during the period against a target protein from the schistosome-

parasite in accordance with the midterm goal of the EU project. The midterm report was submitted to the EU in the third quarter of 2015. This report constituted the basis for EU's decision to grant the next payment to the project by the end of the year 2015.

Further, Kancera has developed chemical substances that inhibit HDAC8 in parasites with the objective to increase the uptake of these drugs into the parasite.

In February 2014 Kancera received an initial payment from the EU amounting to € 523,655 for the execution of the A-PARADDISE project. The project has now issued an interim report which has been approved by the EU. This means that a further installment of the grant will be paid to Kancera at year-end according to plan. This installment amounts to € 300,000 (about SEK 2.8m).

Events after the end of the period

After the end of the period, the project's mid-term report was approved by the EU and a second payment of € 300,000 (about SEK 2.8m) was paid to Kancera.

Market outlook for Kancera's development projects

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 therapeutical area with most agreements between biotechs and pharmaceutical companies (35% of license and option agreements). It is also welcome to see that the proportion of options that are really used to acquire projects in the period 2010-2013 is 40% and the duration is 2-3 years, suggesting that many collaborations relatively quickly leads to the decision to continue product development, (Thomson Reuters Life Sciences Report 2015).

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

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

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

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

There are several examples of license sales in the oncology area in preclinical phase amounting to several hundred million USD. Two of the most influential deals between biotech companies and pharmaceutical companies during the period 2010-2011 were made by companies whose projects had been partially developed by Kancera´s former subsidiary iNovacia AB, including Agios Inc. contracts with Celgene which included a payment upon signature of 130

million USD (however, this deal is regarded as an exception with respect to the size of the payment). Since the start, the cooperation between the two companies has been extended for a total of two years to allow delivery of Agios´ first Phase 1 project. This was announced on June 13, 2014 when Celgene decided to make use of the right to acquire Agios´ candidate drug AG-221 which attacks hematologic cancers through inhibition of the enzyme IDH to thereby disrupt the cancer metabolism. Celgene paid USD 120 million plus royalties for this early clinical project.

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

In April 2012 an agreement was announced between Boston-based Epizyme and Celgene regarding a preclinical drug development project directed against epigenetic targets in cancer, i.e. drugs active against the same target group as Kancera´s HDAC inhibitors. The agreement involved an upfront payment of 90 million USD including equity. Epizyme is a biotech company that has been a frontrunner for a new cancer treatment concept and has managed to close a series of preclinical deals in the cancer area since early 2011 with GSK and Esai.

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

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

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

The trend is towards

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

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

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

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

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

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

These cancer indications each represent a world market in the range of 3.5 to >10 billion SEK annually (Source : GlobalData). According to the Dental and Pharmaceutical Benefits Agency (TLV) in Sweden the society is willing to pay for drugs that treat life-threatening and other serious diseases up to SEK 1 million per year of life with full quality of life (so-called quality adjusted life year, QALY. Two extra years survival with an estimated 50% level of full quality of life corresponds to one QALY). Although there are no definitive requirements to show prolonged survival of new drugs, TLV means that in practice it will be difficult to justify subsidization of new drugs that prolong survival less than 6 months* since this level of prolongation of survival implies a low pricing to cope with the cost per QALY. There exist similar principles for society's willingness to pay in the rest of the world. For example, in England drugs with a cost per QALY in excess of £ 30,000 are not subsidized. However, exceptions are made for life-threatening conditions where the boundary is moved up to £ 50,000 in accordance with the Agency's (NICE) "end-of-life criteria".

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).

*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.

Chronic lymphocytic leukemia (CLL) annually affects approximately 30 000 patients in Europe and the U.S., which makes CLL to the most common chronic form of leukemia. The traditional treatment of cancers such as CLL is currently not sufficiently effective and selective. The most common type of treatment of CLL is a combination of the antibody Rituximab and chemotherapy such as Fludarabine and Cyclophosphamid. This combination of drugs is used in 19% of the treatments in the seven countries that represent the largest pharmaceutical markets. Following the initial treatment

of patients approximately 85% are symptom free, but already after four years clear symptoms of cancer disease had returned for 80% of the patients. New and better treatments are required in this phase of the disease. New drugs with other effects on refractory CLL is now being introduced, such as ibrutinib and idelalisib. Ibrutinib and Idelalisib have clearly improved the treatment of CCL, and give effect in 70-80% of the patients with this disease. However, so-called complete remission (the symptoms have disappeared) has only been reached in a small number of these patients. Since complete remission in cancer is generally linked to a longer survival, there is a need for drugs that work in a new way. Kancera has previously shown that the candidate drug KAN0439834 effectively kills CLL cells from blood and lymph taken from patients *in-vitro* and also in animal models of the human disease. Also, Kancera in collaboration with Prof. Håkan Mellstedt's group at the Karolinska Institute, has demonstrated that Kancera's ROR inhibitor is also effectively killing CLL cells from bone marrow which is a characteristic sought as a complement to today's registered drugs against CLL.

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

Income Statement

SEK 000´s (if otherwise not specified)	1 Oct-31 Dec 2015	1 Oct-31 Dec 2014	1 Jan-31 Dec 2015	1 Jan-31 Dec 2014
Kancera AB				
Revenues				
Net sales	**15**	**50**	**282**	**470**
Contributions from the EU/Vinnova	5 209	0	5 209	
Cost of sales & services	-10	-32	-74	-306
Gross profit	**5 214**	**18**	**5 417**	**164**
Operating Expenses				
General & administrative expenses	-2 455	-863	-3 943	-1 911
Selling expenses	-348	-105	-805	-656
Research & development expenses	-8 300	-4 129	-20 355	-13 692
Total expenses	**-11 103**	**-5 097**	**-25 103**	**-16 259**
Operating income	**-5 889**	**-5 079**	**-19 686**	**-16 095**
Income from Financial Investments				
Financial income	18	36	84	187
Financial expenses		-3	-10	-71
Financial net	18	33	74	116
Income after financial items	**-5 871**	**-5 046**	**-19 612**	**-15 979**
Taxation	**-**	**-**	**-**	**-**
Net income	**-5 871**	**-5 046**	**-19 612**	**-15 979**
Earnings per share, before and after dilution	-0,06	-0,05	-0,19	-0,18

Balance Sheet

SEK 000´s (if otherwise not specified)	30 Sept 2015	30 Sept 2014	31 Dec 2015	31 Dec 2014
Kancera AB				
Assets				
Non-current Assets				
Intangible assets, activated R&D expenses	6 000	6 000	6 000	6 000
Tangible assets	3 425	4 107	3 145	3 868
Total non-current assets	**9 425**	**10 107**	**9 145**	**9 868**
Current Assets				
Work in progress	6 616	2 792	1 486	2 706
Receivables	1 209	748	1 126	872
Cash and cash equivalents	20 155	27 492	15 567	22 974
Total current asstes	**27 980**	**31 032**	**18 179**	**26 552**
TOTAL ASSETS	**37 405**	**41 139**	**27 324**	**36 420**
Equity and Liabilities				
Equity				
Restricted equity	8 660	8 212	8 660	8 212
Non-restricted equity	19 138	23 409	13 265	19 077
Total equity	**27 798**	**31 621**	**21 925**	**27 289**
Provisions and liabilities				
Long-term liabilities	1 500	3 822	1 500	1 500
Short-term liabilities	8 107	5 696	3 899	7 631
Total provisions and liabilities	**9 607**	**9 518**	**5 399**	**9 131**
TOTAL EQUITY and LIABILITIES	**37 405**	**41 139**	**27 324**	**36 420**

Statement of Changes in Equity

SEK 000´s (if otherwise not specified)

Kancera AB

	2015		**2014**
Total equity, opening balance on the 1st of Jan 2015	**27 289**	**Total equity, opening balance on the 1st of Jan 2014**	**18 956**
Optionprogram	219	Issue of shares	7 489
Q1 net income	-4 918	Q1 net income	-4 173
Total equity, closing balance on the 31st of March 2015	**22 590**	**Total equity, closing balance on the 31st of March 2014**	**22 272**
Issue of shares	14 042	Proceeds on issue of shares	16 583
Costs related to issue of shares	-255	Costs related to issue of shares	-1 262
Optionprogram	184	Q2 net income	-3 752
Q2 net income	-5 405	**Total equity, closing balance on the 30th of June 2014**	**33 841**
Total equity, closing balance on the 30th of June 2015	**31 156**	Q3 net income	-3 008
Q3 net income	-3 418	Optionprogram	300
Optionprogram	151	Adjustment of costs related to issue of shares	488
Adjustment of costs related to issue of shares	-91	**Total equity, closing balance on the 30th of Sept 2014**	**31 621**
Total equity, closing balance on the 30th of Sept 2015	**27 798**	Q4 net income	-5 046
Q4 net income	-5 871	Adjustment of costs related to issue of shares	26
Optionprogram	77	Optionprogram	688
Adjustment of costs related to issue of shares	-79	**Utgående balans 2014-12-31**	**27 289**
Total equity, closing balance on the 31st of Dec 2015	**21 925**		

Cash-Flow Statement	1 Oct-31 Dec		1 Jan-31 Dec	
SEK 000´s (if otherwise not specified)	2015	2014	2015	2014
Kancera AB				
Cash-flow from operating activities				
Operating income after financial items	-5 871	-5 046	-19 612	-15 979
Depreciation	279	263	1 088	1 024
Other non-cash-flow affecting items	77	-	631	-
Cash-flow from operating activities before working capit change	**-5 515**	**-4 783**	**-17 893**	**-14 955**
Change in working capital	1 006	-2 764	-2 765	-4 150
Cash-flow from operating activities	**-4 509**	**-7 547**	**-20 658**	**-19 105**
Investment activities				
Investment in tangible assets	0	-101	-366	-601
Cash-flow from investment activities	**0**	**-101**	**-366**	**-601**
FREE CASH-FLOW available to INVESTORS	**-4 509**	**-7 648**	**-21 024**	**-19 706**
Financing activities				
Issue of shares/other capital infusions	-79	766	13 617	23 876
Financing from the EU/Vinnova	0	2 364		4 686
Cash-flow from financing activities	**-79**	**3 130**	**13 617**	**28 562**
CASH-FLOW for the PERIOD	**-4 588**	**-4 518**	**-7 407**	**8 856**
Cash and cash equivalents at the beginning of the perioc	20 155	27 492	22 974	14 118
Cash and cash equivalents at the end of the period	15 567	22 974	15 567	22 974

Notes

Note 1. Accounting and valuation principles

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

The accounting principles of the company are described in the latest published Annual Report (2014).

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

Note 2. Related party disclosures

During the period, Kancera paid compensation to the law firm Nerpin AB for legal services in connection with the new share issue at an amount of 7 kSEK, F:a Mellstedt Medical for scientific consulting and scientific marketing services at an amount of SEK 176 kSEK, and Carl-Henrik Heldin for scientific consulting at an amount 12 kSEK. Erik Nerpin, the Chairman of the Board at Kancera, owns the law firm Nerpin AB. Håkan Mellstedt, a Board member at Kancera, is the Managing Director and owner of F:a Mellstedt Medical. Carl-Henrik Heldin is a Board member at Kancera. No other remuneration was paid to

related parties with the exception of Board fees.

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 June 30 and has been admitted to SEK 0 in the balance sheet. The company management count on that these can be sold with income in the future.

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

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

* Final report

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

***The sum includes EU:s payment of ca SEK 2.8m in January 2016 which means that it cannot be compared to the reported cash flow from financing activities in 2015

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 2014.

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, February 19, 2016

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

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

Financial calendar

• Year End Report 2015	May 5, 2016
• Interim Report January-March 2016	May 20, 2016
• Annual General Meeting	May 26, 2016
• Interim Report January-June 2016	August 19, 2016
• Interim Report January-September 2016	November 18, 2016

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