

Press release February 20, 2015

Full Year Report for Kancera AB (publ) Q4 2014

January 1 – December 31, 2014

In 2013 Kancera changed from the RFR2 regulations, applicable to companies in groups, to BFN's complementary regulation K3. The full year report and consolidated accounts fulfill the requirements of Nasdaq First North for the accounting of Kancera AB. The transition to K3 did not significantly affect the income statement or the balance sheet for 2013. The result for the period January 1, 2013 - December 31, 2013 and the balance sheet as of December 31, 2013 correspond to those accounted for according to earlier accounting principles.

The period January to December 2014 and the fourth quarter 2014 in brief

- R&D expenses for the period totaled SEK 13.1m (SEK 7.5m) of which the fourth quarter constitute SEK 3.6m (SEK 2.1m).
- Operating income for the period totaled SEK -15.2m (SEK -7.4m) of which the fourth quarter constitute SEK -4.1m (SEK -2.1m).
- Income after financial items for the period totaled SEK -15.1m (SEK -7.4m) of which the fourth quarter constitute SEK -4.1m (SEK -2.1m).
- Earnings per share for the period were SEK -0.17 (SEK -0.22) of which the fourth quarter constitute SEK -0.04 (SEK -0.05).
- Cash flow from operating activities for the period totaled SEK -18.9m (SEK -6.6m) of which the fourth quarter constitute SEK -4.5m (SEK 0.4m).
- Equity as of December 31, 2014 totaled SEK 27.5m (SEK 19.0m) or SEK 0.28 (SEK 0.56) per share. The equity/assets ratio as of December 31, 2014 was 76 percent (74 percent).
- Cash and cash equivalents as of December 31, 2014 totaled SEK 23.0m (SEK 14.1m).

Significant events during the period

- Kancera reports that the company is initiating the development of a vaccine directed against ROR. This initiative is motivated by the residual disease in the form of a small number of cancer cells that remain in some patients despite treatment. These cancer cells are difficult to detect and are expected to contribute to relapse of cancer disease. In the most common form of leukemia (chronic lymphocytic leukemia) these remaining cancer cells often express ROR. A vaccine can teach the patient's own immune system to recognize and destroy these ROR-expressing cancer cells. Thus it is expected that a vaccine will add to the suppression of the disease leading to a longer and healthier life for the patient compared to what is possible today. Kancera's strategy is to use its future small-molecule ROR inhibitors as a first line treatment for the disease to remove the main part of the tumor and the symptoms, and thereafter follow with a prophylactic ROR vaccine to prevent relapse. Thus, there are possible synergies between Kancera's small molecule products and the vaccine against ROR.
- Kancera announced that the company has received a first payment from the EU of € 523,655 for the execution of the A-PARADDISE project and that the project thus has started. In August 2013 Kancera announced that the company together with international research groups in the project A-PARADDISE has been awarded a grant from the European Union Seventh Framework Programme to develop drugs to combat severe parasitic diseases including malaria, schistosomiasis, leishmaniasis and Chagas disease. The total three-year project budget is 6 M€ where the Kancera part of about € 950,000 is the largest.
- Kancera has reported results from the collaboration on PFKFB3 inhibitors with Professor Thomas Helleday at the Science for Life Laboratory which was initiated in 2013. Within the framework of the collaboration a large-scale laboratory evaluation of synergistic effects between Kancera's PFKFB3 inhibitors and a large number of approved drugs has been performed. The results show that synergistic effect against cancer cells can be achieved by combining PFKFB3 inhibitors and some defined classes of approved drugs. In light of the present results, new

experiments are planned using preclinical disease models to verify whether PFKFB3 inhibitors can improve the treatment of advanced lung cancer and metastatic breast cancer.

- Kancera reports that the company has registered a patent application (EP14167988.6) for new compounds against cancer that selectively inhibit the enzyme HDAC6. The new patent application is based on the ability of HDAC6 inhibitors to influence mechanisms both inside and outside of the cell nucleus. It has been shown that the major biological role of HDAC6 is in the regulation of the cancer cell's ability to migrate and form metastases.
- Kancera's Annual General Meeting on May 26, 2014 decided to implement an incentive program for the employees and corresponding executives and board members (for further information, see Note 3). Further, the Annual General Meeting authorized the Board to issue new shares, on one or several occasions until the next Annual General Meeting. New shares may be issued with or without preferential rights and payment in cash and/or in kind or set-off. If a new 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.
- In accordance with the decision of the Board of Kancera AB (publ) November 7, 2013, and pursuant to the authorization of the Extraordinary General Meeting October 30, 2013, there was a share issue through the exercise of warrants TO 1 2013 for the subscription of new shares. A total of 21,603,424 shares were subscribed. This share issue was therefore subscribed to around 98 percent and brought Kancera AB approximately SEK 16.2m before issue costs.
- Kancera announced that the ROR project was awarded a grant for the last phase of a project co-funded by Vinnova. For the project Kancera has in total received SEK 1.5m from the grant which is directed to young innovative companies with growth potential.
- Kancera announced that animal studies are proceeding as planned and that the results so far support that an effective concentration of the ROR inhibitor can be achieved in cancer cells for a time sufficient to reach the desired anti-cancer effect.
- Kancera announced that the development of the HDAC6 inhibitors are progressing faster than previously estimated in the second quarter when HDAC6 inhibitors were developed that are more potent against cancer cells than Acetylon's ACY-1215 and also better tolerated by healthy human blood cells. Kancera also announced that the development of an active immunotherapy against ROR in the form of a cancer vaccine has now reached a milestone since Kancera's first series of vaccines results in an immune response in animals with antibodies that bind to ROR.
- Kancera announced that the co-operation 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.
- Kancera reported results from a detailed analysis of the preclinical efficacy study that was completed during the third quarter, as reported in a press release on October 3, 2014. The results confirm that the number of leukemic cells is significantly reduced in an animal model of chronic lymphocytic leukemia after 7 days of oral treatment with KAN0439834. The results of the efficacy and tolerance studies support the selection of KAN0439834 as the first drug candidate in the project, and also points to opportunities to further improve the efficacy profile by developing the technology for the delivery of the product.
- Kancera reports that an investigation of the mechanism of action of the company's patent-pending HDAC6 inhibitors has demonstrated a unique profile that could strengthen the competitiveness of the project. 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. The investigation showed that Kancera's HDAC6 inhibitors exhibited a remarkably high level of selectivity since no effect was detected against any of ca 50 known risk factors, but show a significant effect on one mechanism of action, in addition to HDAC6, which probably can be utilized to further enhance the competitive edge of the project in oncology. With adequate resources allocated to the HDAC6 project the company predicts that a candidate drug can be delivered in 18-24 months.

Significant events after the end of the reporting 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 reports 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.

Statement from the CEO

The biotech industry started the year with an intense January week in San Francisco where a large number of pharmaceutical and biotech companies gathered to kick-off a promising 2015 (the JP Morgan and Biotech Showcase conferences). The confidence in the industry, especially in the US, was high during the previous year which is reflected in a high level of investments in pharmaceutical projects, a rising valuation of the biotech companies and an increasing number of new drugs approved by the FDA and EMA. A clear trend in 2014 was the increased number of orphan drugs approved for market. Also, the interest in investing in new orphan products shows an upward trend which can be partly attributed to clinical studies that are focused on smaller and well-defined diseases (so-called orphan Indications), which in turn means smaller and less expensive studies, and partly to that the companies, in exchange for improved efficacy and safety, can count on a pricing of the product that creates good profitability.

In 2014, Kancera's ROR project has made significant steps forward from displaying a potent effect in vitro on treatment-resistant cancer cells from severely ill patients to a product that is efficiently absorbed into an animal model of human cancer and reaches the cancer cells in the lymphatic system to eliminate these. The driving force behind this development is Kancera's interdisciplinary team that designed and developed a new generation of ROR inhibitors for which a patent application was filed in February 2015 and a newly developed crystalline formulation of the drug candidate KAN0439834. The first two efficacy trials of the drug candidate in animal models targeted human chronic lymphocytic leukemia (CLL). In the autumn 2014, treatment with KAN0439834 gave good effects both in a disease model of progressive CLL and later in a model for an advanced phase of CLL that is refractory to treatment with the currently available drugs. New effective drugs to treat the today incurable disease chronic lymphocytic leukemia would qualify as an orphan drug.

Next in 2015 our work is directed towards assessing the ROR inhibitors for treatment of additional cancer forms, and advancing our still early but promising HDAC6 project to better understand how the HDAC6 inhibitors' dual mechanism of action best can be utilized against intractable cancer. Also, the business development efforts progresses and is primarily aimed at finding the right partner for the ROR project. In 2015, in addition to company meetings in San Francisco, we have met with pharmaceutical companies in Zürich and presented the ROR project at the annual meeting of the Global CLL Research Foundation in Houston. Next in line are presentations of the company and new business meetings at the BIOEurope conference in Paris.

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 is currently developing drugs for the treatment of leukemia and solid tumors, based partly on blocking survival signals in the cancer cell and partly on metabolic strangulation.

Kancera's operations are based in the Karolinska Institutet Science Park in Stockholm and the company employs around 10 people. The Kancera shares are traded on NASDAQ OMX First North and the number of share holders is ca 5400 as of January 31, 2014. Remium Nordic AB is Kancera's Certified Adviser. Professor Carl-Henrik Heldin and Professor Håkan Mellstedt are 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 PFKFB-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				
<i>SEK 000's (if otherwise not specified)</i>				
Kancera AB	1 Oct-31 Dec		1 Jan-31 Dec	
	2014	2013	2014	2013
Net turnover	50	1 354	470	1 813
R&D expenses	-3 555	-2 148	-13 118	-7 533
Operating Income	-4 174	-2 083	-15 190	-10 404
Income after financial items	-4 141	-2 096	-15 074	-7 418
Net income	-4 141	-2 096	-15 074	-7 418
Cash-flow from operating activities	-4 520	363	-18 853	-6 638
Cash-flow from financing activities	-	11 702	28 310	17 649
Earnings per share, before and after dilution	-0,04	-0,05	-0,17	-0,22
Cash on hand at closing date	22 974	14 118	22 974	14 118
Solvency ratio	76%	74%	76%	74%
Key ratios				
Return on equity, %	neg	neg	neg	neg
Return on capital employed, %	neg	neg	neg	neg
Solvency ratio	76%	74%	76%	74%
Investments in tangible assets	24	-	601	2 000
No. of employees	10	7,5	10	7,5
Earnings per share, before dilution	-0,04	-0,05	-0,17	-0,22
Earnings per share, after dilution	-0,04	-0,05	-0,17	-0,22
Equity by share, kr	0,28	0,56	0,28	0,56
Cash-Flow by share, kr	-0,05	0,31	0,10	0,27

Comments on the financial development

The increased cash flow and the enhanced liquidity for the period compared to the corresponding period in 2013 can be attributed to new share issues during the fourth quarter in 2013 and the following exercise of TO1 during the second quarter 2014. The increased R&D costs for the period compared to the corresponding period in 2013 can be attributed to that more projects now are run in parallel and that costs for out-sourcing have increased in connection with the evaluation of the drug candidate that was selected in the ROR project in November 2014. The lower income after financial items and earnings per share for the period compared to the corresponding period in 2013 can mainly be attributed to a capital gain that of SEK 3m which arose in 2013 in connection with an acquisition of a claim.

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 0.5m (SEK 1.8m). The company also receives financial support from the EU project A-Paradise where the support is offset against incurred costs for the period amounting to SEK 2.7m (SEK 0.0m) of consumables, performed months of work plus 60% overhead on the sum of these costs as will be summarized in an interim report. The financial support from EU covers 75% of the project costs plus 60% overhead.

Expenses

Expenses in the fourth quarter totaled SEK 4.2m (SEK 3.4m), which breaks down into costs of services sold of SEK 0.0m (SEK 0.2m), research and development expenses of SEK 3.6m (SEK 2.1m) and other sales and administrative expenses of SEK 0.6m (SEK 1.1m). Expenses during the period January 1 to December 31 2014 amounted to SEK 15.6m (SEK 12.2m) which breaks down into costs of services sold of SEK 0.3m (SEK 0.5m), research and development expenses of SEK 13.1m (SEK 7.5m) and other sales and administrative expenses of SEK 2.2m (SEK 4.2m). The higher administrative costs in 2013 compared to the same period in 2014 is attributable to costs incurred in connection with preparations for the share issue in 2013.

Earnings

Income after financial items for the fourth quarter totaled SEK -4.2m (SEK -2.1m) and for the period SEK -15.1m (SEK -7.4m). When taking the capital gain in the previous year into account, the result for the period is SEK 2.6m lower compared to the same period in 2013.

Cash flow and liquidity

Cash flow totaled SEK -4.5m (SEK 12.1m) in the fourth quarter. Cash flow from operating activities for the fourth quarter amounted to SEK -4.5m (SEK 0.4m). Cash flow from financing activities for the fourth quarter amounted to SEK 0.0m (SEK 11.7m).

Cash flow during the period totaled SEK 8.9m (SEK 9.0m). Cash flow from operating activities during the period amounted to SEK -18.9m (SEK -6.6m). Cash flow from financing activities during the period amounted to SEK 28.3m (SEK 17.6m) which mainly can be attributed to the share issue, the exercise of warrants TO1 2013, and the EU support received.

Kancera has been 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-Paradise project that targets parasitic diseases. The grant is accounted for as a recognized liability until the project's interim report has been approved by the EU 20 months after the project start after which it settled against accumulated costs. This is expected to happen in the fourth quarter 2015..

In accordance with the decision of the Board of Kancera AB (publ) on November 7, 2013 and pursuant to the authorization from the Extraordinary General Meeting on October 30, 2013, Kancera solves warrants TO 1 2013 during the period May 1-31, 2014, which raised SEK 16 202 568 before issue expenses. Also, Kancera closed an incentive program for the employees and corresponding executives and board members in accordance with the decision on the Annual General Meeting 2011, which raised SEK 1 034 669 before issue expenses.

Ongoing work for the period amounting to SEK 2.7m is attributable to the work performed within the framework of the EU project A Paradise. Ongoing work is offset against grants received following an approved mid-term report for the project. The mid-term report will be submitted to the EU in Q3 2015.

Kancera's cash and cash equivalents as of December 31, 2014 totaled SEK 23.0m (SEK 14.1m).

Investments

Investments in fixed assets in the third quarter totaled SEK 0.0m (SEK 0.0m) and for the period net SEK 0.6m (SEK 2.0m).

Investments in intangible assets in the fourth quarter 2014 totaled SEK 0.0m (SEK 0.0m) and for the period SEK 0.0m (SEK 0.0m).

The company continuously invests in research projects that increase the company's technology knowledge, and where also a patent application covering the technology can be included. In the accounts these investments including patent costs, are entered as costs since the time of activation for projects is based on the time when the project will be commercialized and that time point has not yet occurred. R & D costs, which therefore are entered as R & D, amounted to SEK 3.6m (SEK 2.1m) for the third quarter.

During the period Kancera acquired instruments previously leased by the previous subsidiary iNovacia AB from Handelsbanken Finans AB for SEK 500,000 considered by the company to be an estimated market price.

Equity and share data

Total equity as of December 31, 2014 was SEK 27.5m (SEK 19.0m).

Share capital as of December 31, 2014 amounted to SEK 8 212 310,97 spread over 98 547 732 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 -0.04 (SEK -0.05). In connection with the share issue in December 2013 a bonus element was identified, which means that the weighted average number of shares used to calculate earnings per share has been adjusted. Prior periods have been recalculated to reflect the bonus element.

The equity/assets ratio as of December 31, 2014 was 76 percent (74 percent). Total equity per share was SEK 0.28 (SEK 0.56) based on total equity divided with the number of shares on the balance sheet day at the end of the quarter.

Deficits for tax purposes

Kancera's present operations are expected to initially result in negative earnings and deficits for tax purposes. There are no sufficiently convincing evidence at present that tax surpluses will exist in the future that may justify capitalization of the value of the deficit, and no deferred tax claim has therefore been reported. In the event a drug candidate is sold, profits will be reported which may be offset for tax purposes against the deficits. This signifies a low tax burden for the company when a project is sold. The determined tax losses amount to SEK 75.0m as of December 31, 2014.

Personnel

Kancera AB had 10 full time employees (7) as of December 31, 2014 of which 6 are men and 4 are women.

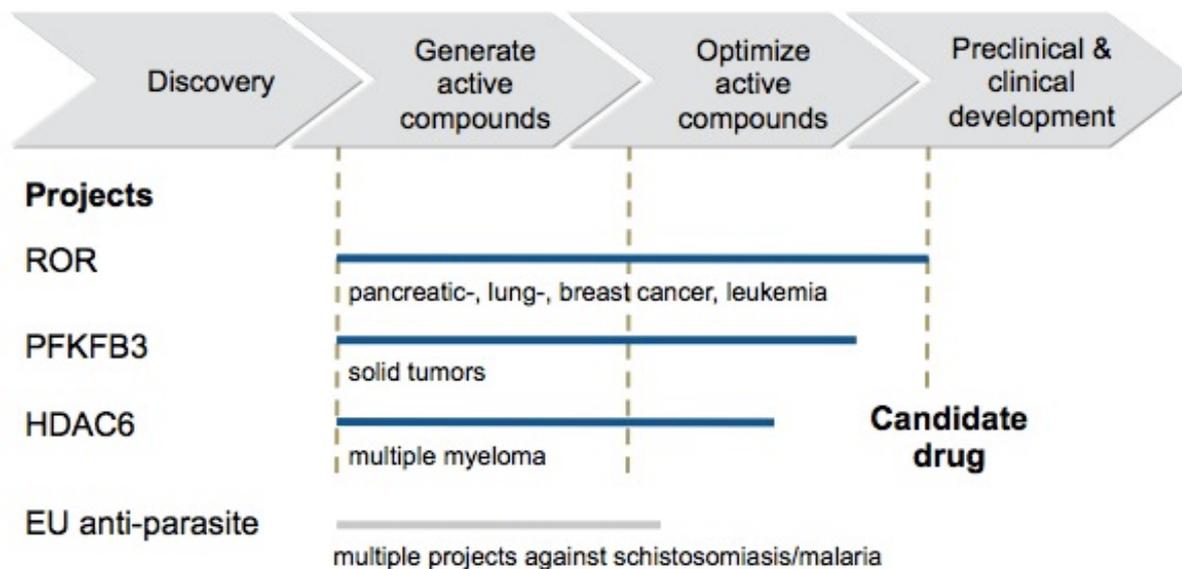
Pharmaceutical Development

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 four 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. Kancera has also initiated a project aiming to develop a vaccine against ROR.
- **Small molecule PFKFB3 inhibitors** that strangle the energy supply from glucose to solid tumors, thereby increasing tumor sensitivity to other anticancer drugs.
- **Small molecule HDAC6 inhibitors** that primarily aim to neutralize blood cancer by controlling the cancer cell genome and ability to move.
- **Small molecule inhibitors of epigenetic processes in parasites** to develop new treatments against e.g. malaria and schistosomiasis (snail fever)

Figure 1. Kancera's product 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. 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 are 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.

The main part of the company's resources is invested in the ROR project and the HDAC6 project, 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 costs plus 60% overhead 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 efficiently develop epigenetically acting drugs against cancer, including HDAC6 inhibitors, since a similar technical expertise and capacity are needed for both epigenetic projects. The HDAC6 project has developed at a faster pace than previously estimated, which means that it is possible to select a candidate drug within 18-24 months.

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 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.

Kancera's Board of Directors has decided not to communicate financial goals for the pharmaceutical development because Kancera's projects are in the early phases of development, which means the risk is high and the overall financial goals are difficult to assess.

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-1 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. In addition, Kancera develops a vaccine based on the part of ROR situated on the outside of the cancer cell. Vaccines are able to stimulate the patient's own immune system to recognize cancer cells and destroy them by means of antibodies and white blood cells.

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. According to the study Kancera's ROR inhibitors spare the healthy white blood cells. Thus a future patient receiving this drug may withstand severe infections better compared to those receiving today's medications.

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 7 patients 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 μ M while Kancera's ROR inhibitor show higher effect at a lower concentration (70% killing of cancer cells at about 3 μ M). The maximum killing effect on cancer cells is negligible after 24 hours for the BTK inhibitor (Ibrutinib) and the PI3K inhibitor. 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 recently 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 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 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 annually 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.

Kancera has 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

In the period June to September 2014, research groups independent of Kancera published results that support that future ROR1 targeted drugs have the potential to help cancer patients and to be established as pioneering drugs on the market. In June a study was published (Karachaliou Others, 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. In September, Professor Thomas Kipps together with the US company Celgene published a press release announcing the initiation of clinical development of an antibody directed against ROR1 that is proposed to target the cancer cells that are most important for tumor development (so called cancer stem cells).

During the period Kancera designed and synthesized new ROR inhibitors that show an increased efficacy against cancer cells, which further strengthens the possibility of creating an effective drug with an improved efficacy profile compared to the newest anticancer drugs such as Ibrutinib and Idelalisib. Results showed that the company's small molecule ROR inhibitor KAN0439834 is more potent than previously assumed since it inactivates ROR1 already after 15 minutes at a low concentration (at 25 nM) and kills cancer cells from patients with leukemia at 300 nM concentration. Kancera also reported that laboratory studies showed that it is sufficient to inhibit ROR1 for six hours in order for cancer cells to complete self-destruction after 24 hours.

Kancera reported that the company is initiating the development of a vaccine directed against ROR. A successful development of a ROR-directed vaccine would be able to teach the patient's own immune system to recognize and destroy ROR-expressing cancer cells. Thus it is expected that the disease will be suppressed for a longer time leading to a longer and healthier life for the patient than what is possible today.

The development of the vaccine concept against ROR is accelerated by Kancera's existing knowledge of ROR and the close collaboration with Professor Håkan Mellstedt, at the Karolinska Institute, who is an internationally recognized expert in the development of cancer vaccines. The principle to use a ROR vaccine for treatment is also supported by a preclinical study published by Professor Thomas Kipps at the University of California, San Diego.

During the period, vaccine candidates have been synthesized and animal studies have been started with the aims to demonstrate the immune stimulating performance of the vaccine candidates and to test their therapeutic effect. The results from a first vaccination study showed that a couple of the company's ROR1-directed vaccine candidates teach the immune system in rats and rabbits to recognize important parts of ROR1 and after 24 hours kill cancer cells from patients while leaving blood cells from healthy subjects unaffected in the same period of time. The results show that Kancera's vaccine project progresses according to plan. However, it remains to be shown that a vaccine can compete with small-molecule inhibitors of ROR and antibodies against ROR in terms of efficacy to kill cancer cells. The continued development is directed at improving the vaccine characteristics and to examine its safety. The vaccine development costs during 2014 are accommodated within the existing budget, due to the synergies between the company's development of small molecules and vaccines.

During the period, Kancera reported that KAN0439834 has been selected as the 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 as reported as an event after the period.

Events after the end of 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*.

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 (see Figure 2).

The recently 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.

* Reference: Bertilaccio et al.: Xenograft models of chronic lymphocytic leukemia: problems, pitfalls and future directions. *Leukemia* 27:534-540.2013

Figure 2. The new formulation of KAN0439834 and the effect of 14 days of treatment with this formulation on the amount of human treatment-resistant cancer cells in an animal model of chronic lymphocytic leukemia.

2 a) Crystalline formulation of the drug candidate KAN0439834

A new formulation of the drug candidate KAN0439834 has been developed by crystallization of the compound followed by a micronization of the crystals to a size of less than one micrometer. Subsequently, these sub-micrometer-sized crystals have been prepared in solution as a stable suspension. The suspension (60-80 mg/kg per day) is administered orally and gives a progressive dissolution in the gastrointestinal tract resulting in a balanced uptake in the blood which transports the ROR inhibitor to the cancer cells in a dose that is well tolerated by the body's healthy cells. The picture below shows crystals of KAN0439834 before micronization.



Figure 2 b) shows selected results from a study of how Kancera's ROR inhibitor KAN0439834 affect human leukemia cells that have infiltrated the lymphatic system of mice which in the analysis is represented by the spleen. Human leukemia cells have been transferred to the animals on Day 0. During day 8-21 (14 days) the animals are treated either with a dummy control (C) or with 60 mg/kg (PO Low) or with 80 mg/kg for 7 days + 60 mg/kg for 7 days (PO High) of Kancera's ROR inhibitor KAN0439834.

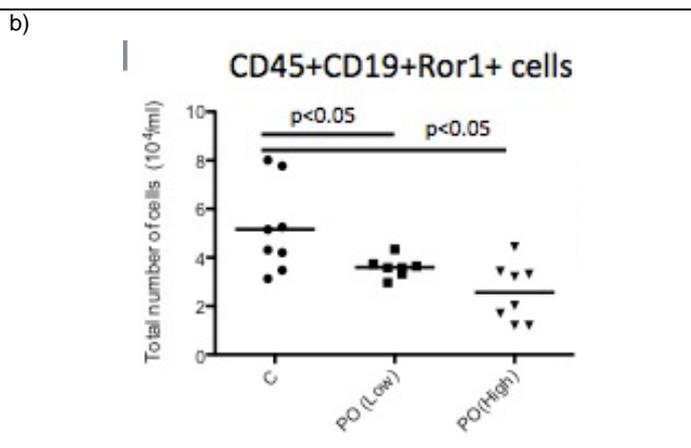
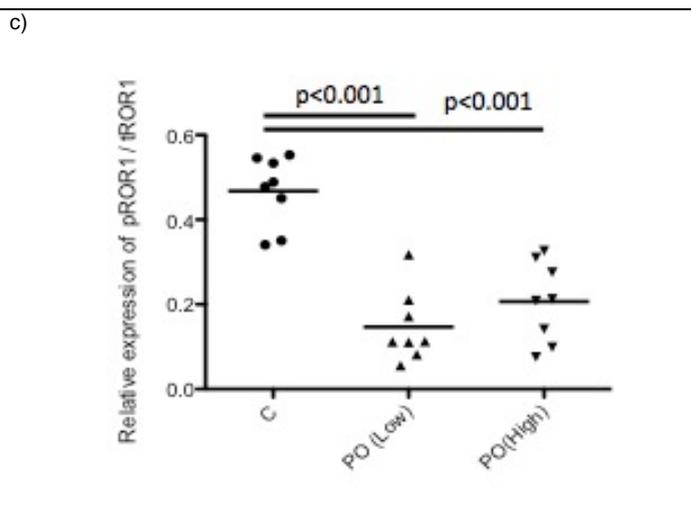


Figure 2 c) shows that in the same study shown in Figure 2b), the amount of activated ROR1 is reduced by treatment with KAN0439834. The analysis is performed with the immune method Western blot.



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 PFKFB3 project – a candidate that blocks glycolysis in solid tumors

The project aims to develop PFKFB3 enzyme inhibitors to strangle 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 has initiated a collaboration with Professor Thomas Helleday and his research group at Karolinska Institutet and the Science for Life Laboratory (SciLifeLab) in order to advance unique research on energy metabolism in cancer and Kancera's PFKFB3 project. During the collaboration Professor Helleday and Kancera combine their strengths in research on disease mechanisms and product development in order to deliver a new treatment against cancer with the goal to break down the resistance of the cancer to existing drugs. 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 PFKFB3 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.

Events during the period

Within the framework of the collaboration 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 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. In light of the present results, new experiments are planned using preclinical disease models to verify whether PFKFB3 inhibitors can improve the treatment of advanced lung cancer and metastatic breast cancer.

Kancera has 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.

The project combines the strengths of professor Helleday and Kancera in research on disease driving mechanisms and product development, respectively. The project investigates 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 project is part of the SciLife Innovation project between Karolinska Institutet Innovation office and Uppsala University Innovation which aims to develop a partnership model, based on mutual benefit, between academia and industry in Life Science. The partnership should be given the opportunity to grow into a more comprehensive partnership program with interactions at multiple levels, such as training, seminars, and utilization of research.

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

No significant news regarding the PFKFB3 project have been reported after the end of the period.

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 two HDAC inhibitors on the market for the treatment of various forms of T-cell lymphoma. 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.

Laboratory tests have shown that Kancera's substances are able to kill cancer cells and they have a higher level of selectivity against the HDAC6 enzyme as compared to a competing inhibitor, ACY-1215, developed by the Boston based Acetylon Pharmaceuticals.

In collaboration with Professor Håkan Mellstedt's group at Karolinska Institutet, Kancera has demonstrated lethal effect of Kancera's HDAC6 inhibitors on cells from three different cancer forms: multiple myeloma, osteosarcoma and pancreatic cancer.

Events during the period

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.

During the period, new HDAC6 inhibitors have been developed 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 18-24 months. The next step is now to evaluate how the new mechanism can be combined with inhibition of HDAC6 to fight intractable cancer.

Events after the end of the period

No significant news regarding the HDAC6 project have been reported after the end of the period.

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

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

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

Kancera is the only pharmaceutical development company in the A PARADDISE consortium and is well positioned to commercialize the drug candidates that the company develops and owns together with its partners. For clinical development and commercialization of drugs for neglected diseases, it is likely that Kancera will seek cooperation with internationally established pharmaceutical companies and nonprofit organizations that have chosen to take social responsibility by investing in the development therapies against diseases that primarily affect poor countries in tropical and subtropical areas.

In addition to parasitic diseases, analyses at Kancera show that some of the lead substances now being developed against targets in the parasite also inhibit similar human target proteins that are linked to cancer.

Overall, the project's potential application in cancer and the fact that countries that currently suffer from serious parasitic diseases have an increasing financial capacity to invest in drugs, show that the project's future drug candidates have a good commercial potential.

Events during the period

In February 2014, Kancera together with international research teams in the project A-PARADDISE (Anti-Parasitic Drug Discovery in Epigenetics), have launched the next phase in the development of these drugs, which will run for three years and result in one or more lead substances and drug candidates. The project has commenced with the start of optimization of the anti-parasitic substances that Kancera successfully initiated during the completed EU funded project Settrend. 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 (Malaria (*Plasmodium falciparum*), Schistosomiasis (*Schistosoma mansoni*), Leishmaniasis (*Leishmania*) and Chagas disease (*Trypanosoma cruzi*).

In 2014, the project work mainly concerned on the further development of anti-parasitic compounds that the company have previously developed. The primary focus of this ongoing work is to modify the substances in a manner that provides a higher exposure of the substances in the parasite. In 2014, about 70 substances were synthesized within the project. The academic groups in the consortium are currently testing the effect of these compounds against various types of parasites.

Events after the end of the period

No significant news regarding the Antiparasite-project have been reported the after the end of the period.

Market outlook for Kancera's development projects

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 2014, the European Medicines Agency approved 82 new drugs of which about 20 percent were orphan drugs against several cancer diseases. A full 50% of the 82 drugs that were approved were based on completely new drug compounds that have never been part of a product before, i.e. the type of drug substances Kancera's projects are aimed at. The US Food and Drug Administration (FDA), that approved 27 drugs in 2013, approved 35 new drugs in 2014. Of these, as many as 40% were orphan drugs. Further, in the United States the number of requests increases for accelerated assessment of new drugs called "Break-through therapy" which are directed towards very serious and life threatening diseases. The number of applications to the FDA in this category has exceeded all expectations from the authority. The group of diseases that has dominated the number of approvals in the category "Break-through therapy" is cancer with Ibrutinib from Pharmacyclics as a good example (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.

Deals in preclinical development dominated over deals in the clinical phase in 2012 and represented 46% of global partnering agreements regarding rights related to pharmaceuticals according to the analyst Burrill & Company (Source: Burrill & Company). Thus it can be concluded that the trend in 2009-2011, with a significant number of deals in the same early phase as the Kancera projects, continues.

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 pays 120 million USD 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 150 million USD to Pharmacocyclics for a BTK inhibitor Ibrutinib in clinical phase II, in addition to future installments of 825 million USD.

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 Ib for a potential treatment of leukemia.

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, PFKFB and HDAC6 in

- Solid tumors in the pancreas, lung, bowel and breast. The three first mentioned forms of cancer are among the four types of cancer that causes most deaths in both men and women. Breast cancer is with the exception of lung cancer the form of cancer that causes most deaths in women.

- 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). A drug able to contribute to a 6-months prolonged life at a cost of less than about 1 million SEK is today regarded by the price authorities such as TVL to represent a significant value for patients and society.

Kancera's own published results, as well as publications from independent research groups in the ROR and PFKFB area (see sources in each project section) support that future drugs acting through ROR and PFKFB 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. The European Medicines Agency EMA has steadily increased the number of approved drugs for the treatment of rare diseases from four approved products in 2011 to eight in 2012 and eleven in 2013. Kancera's projects have in preclinical studies been shown to be a possible way to treat several forms of cancer that meet the requirements for designation as an Orphan disease (in the U.S. fewer than 200,000 affected 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 781 million USD and the expected growth was -4 to +8% in 2017, (Source : Global Data Healthcare).

Chronic lymphocytic leukemia (CLL) annually affects approximately 30 000 patients in Europe and the U.S., which makes CLL to the most common chronic form of leukemia. The traditional treatment of cancers such as CLL is currently not sufficiently effective and selective. The most common type of treatment of CLL is a combination of the antibody Rituximab and chemotherapy such as Fludarabine and Cyclophosphamid. This combination of drugs is used in 19 percent of the treatments in the seven countries that represent the largest pharmaceutical markets. Following the initial treatment of patients approximately 50 percent are symptom free, but already after four years about 80 percent regained clear symptoms of cancer disease. New, increasingly tougher treatments are required in this phase of the disease, but the treatment results become progressively worse. New drugs with other effects on refractory CLL is now being introduced, such as ibrutinib and idelalisib. 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.

Income Statement	1 Oct-31 Dec		1 Jan-31 Dec	
<i>SEK 000's (if otherwise not specified)</i>	2014	2013	2014	2013
Kancera AB				
<i>Revenues</i>				
Net sales	50	1 354	470	1 813
Cost of sales & services	-32	-231	-306	-530
Gross profit	18	1 123	164	1 283
<i>Operating Expenses</i>				
General & administrative expenses	-532	-806	-1 580	-3 375
Selling expenses	-105	-252	-656	-779
Research & development expenses	-3 555	-2 148	-13 118	-7 533
	-	-	-	-
Total expenses	-4 192	-3 206	-15 354	-11 687
Operating income	-4 174	-2 083	-15 190	-10 404
<i>Income from Financial Investments</i>				
Financial income	36	0	187	3 001
Financial expenses	-3	-13	-71	-15
Financial net	33	-13	116	2 986
Income after financial items	-4 141	-2 096	-15 074	-7 418
Taxation	-	-	-	-
Net income	-4 141	-2 096	-15 074	-7 418
Earnings per share, before and after dilution	-0,04	-0,05	-0,17	-0,22

Balance Sheet	30 Sept		31 Dec	
<i>SEK 000's (if otherwise not specified)</i>	2014	2013	2014	2013
Kancera AB				
<i>Assets</i>				
<i>Non-current Assets</i>				
Intangible assets, activated R&D expenses	6 000	6 000	6 000	6 000
Tangible assets	4 107	4 499	3 868	4 291
Total fixed assets	10 107	10 499	9 868	10 291
<i>Current Assets</i>				
Work in progress	2 792	-	2 706	-
Receivables	748	507	872	1 240
Cash and cash equivalents	27 492	2 053	22 974	14 118
Total current assets	31 032	2 560	26 552	15 358
TOTAL ASSETS	41 139	13 059	36 420	25 649
<i>Equity and Liabilities</i>				
<i>Equity</i>				
Restricted equity	8 212	2 689	8 212	17 989
Non-restricted equity	23 409	6 661	19 294	967
Total equity	31 621	9 350	27 506	18 956
Long-term liabilities	3 822	1 500	1 500	1 500
Short-term liabilities	5 696	2 209	7 414	5 193
Total provisions and liabilities	9 518	3 709	8 914	6 693
TOTAL EQUITY and LIABILITIES	41 139	13 059	36 420	25 649

Statement of Changes in Equity

SEK 000's (if otherwise not specified)

Kancera AB

	2014	2013
Total equity, opening balance on the 1st of Jan 201	18 956	10 225
Proceeds on issue of shares	7 489	4 834
Costs related to issue of shares	-	-387
Q1 net income	<u>-4 173</u>	<u>690</u>
Total equity, closing balance on the 31st of March :	22 272	15 362
Proceeds on issue of shares	16 583	<u>-3 274</u>
Costs related to issue of shares	-1 262	
Q2 net income	<u>-3 752</u>	
Total equity, closing balance on the 30th of June 2014 :	33 841	12 088
Proceeds on issue of warrants	300	<u>-2 738</u>
Adjustment of costs related to issue of shares	488	
Q3 net income	<u>-3 008</u>	
Total equity, closing balance on the 30th of Sept 2014 :	31 621	9 350
Adjustment of costs related to issue of shares	26	15 300
Q4 net income	<u>-4 141</u>	-3 598
Total equity, closing balance on the 31st of Dec 2014 :	27 506	18 956
		<u>-2 096</u>

Cash-Flow Statement

SEK 000's (if otherwise not specified)

Kancera AB

	1 Oct-31 Dec		1 Jan-31 Dec	
	2014	2013	2014	2013
<i>Cash-flow from operating activities</i>				
Operating income after financial items	-4 141	-2 096	-15 074	-7 418
Depreciation	263	429	1 024	709
Other non-cash-flow affecting items	-	-	-	-3 000
Cash-flow from operating activities before working capital change	-3 878	-1 667	-14 050	-9 709
Change in working capital	<u>-642</u>	<u>2 030</u>	<u>-4 803</u>	<u>3 071</u>
Cash-flow from operating activities	-4 520	363	-18 853	-6 638
<i>Investment activities</i>				
Investment in tangible assets	-24	-	-601	-2 000
Cash-flow from investment activities	-24	-	-601	-2 000
FREE CASH-FLOW available to INVESTORS	-4 544	363	-19 454	-8 638
<i>Financing activities</i>				
Issue of shares/other capital infusions	26	11 702	23 624	16 149
Financing from the EU/Vinnova	-	-	4 686	-
New loans	-	-	-	1 500
Cash-flow from financing activities	26	11 702	28 310	17 649
CASH-FLOW for the YEAR	-4 518	12 065	8 856	9 011
Cash and cash equivalents at the beginning of the year	<u>27 492</u>	<u>2 053</u>	<u>14 118</u>	<u>5 107</u>
Cash and cash equivalents at the end of the year	22 974	14 118	22 974	14 118

In 2013 an extraordinary net income of SEK 3 million occurred in connection with the acquisition of a preferential claim from SOBI AB in 2013.

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 (2013).

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

Note 2. Related party disclosures

During the period, Kancera paid compensation to F:a Mellstedt Medical for scientific consulting and scientific marketing services at an amount of SEK 97 100. Håkan Mellstedt, a Board member at Kancera, is the Managing Director and owner of F:a Mellstedt Medical. During the period Kancera also paid compensation to Kilpatrick Townsend & Stockton Lawyer KB for services in connection with the share issues and option schemes by an amount of 169 000 SEK. Erik Nerpin, Chairman of the Board at Kancera was a Partner at Kilpatrick Townsend & Stockton Lawyer KB until the second quarter of 2014. During the period, Kancera paid compensation to Carl-Henrik Heldin for scientific consulting at an amount of SEK 7 000. No other remuneration was paid to related parties with the exception of Board fees.

Note 3. Incentive schemes

Following a resolution passed by the Annual General Meeting on May 26, 2014 Kancera introduced an incentive scheme for employees of the company and corresponding executives and Board members. The incentive scheme involves the issue of maximum 2 800 000 warrants. Of these, 2 200 000 will be 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 $\frac{1}{4}$ of the number of granted options after one and two years, respectively, leaving, in this example, $\frac{1}{2}$ 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.

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.

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

Funded by	Amount granted, kSEK	Amount paid, kSEK	Reporting date
Vinnova	500	500	Dec 2014*
EU	8520**	4 686	Aug 2015 and March 2017*
	9020	5 186	

* final report

** Assuming an EUR exchange rate of 8.95 SEK. 30% of the grant is paid following an approved interim report which will be submitted in August 2015, and the remaining 15% of the grant is paid following an approved final report which will be submitted in March 2017.

Note 5. 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 less non-interest bearing liabilities.

Equity/assets ratio

Equity as a percentage of total assets.

The company's operations and risk factors

The Board of Directors and the CEO give an assurance 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.

In assessing Kancera's 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 earnings and financial position to varying degrees. For further information regarding company risks, see the company's Annual Report 2013.

Stockholm, February 20, 2015

Erik Nerpin
Chairman of the Board

Håkan Mellstedt
Director

Bernt Magnusson
Director

Carl-Henrik Heldin
Director

Thomas Olin
CEO/Director

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

Financial calendar

- | | |
|---|-------------------|
| • Annual Report 2014 | May 7, 2015 |
| • Interim Report January-March 2015 | May 22, 2015 |
| • Annual General Meeting | May 28, 2015 |
| • Interim Report January-June 2015 | August 21, 2015 |
| • Interim Report January-September 2015 | November 20, 2015 |

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