

Press release August 22, 2014

# Interim Report for Kancera AB (publ) Q2 2014

## January 1 – June 30, 2014

All figures from the first quarter 2013 relate only to Kancera AB as a consequence of the liquidation of the subsidiary iNovacia AB in the beginning of 2013. In connection with this Kancera has passed 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 OMX First North for the accounting of Kancera AB. The transition to K3 did not affect the income statement or the balance sheet for 2013 or 2012. 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. Comparative figures from the preceding year relate to the mother company Kancera AB.

### The second quarter 2014 in brief

- R&D expenses for the period totaled SEK 6.9m (SEK 3.6m) of which the second quarter constitute SEK 3.4m (SEK 2.1m).
- Operating income for the period totaled SEK -8.0m (SEK -5.6m) of which the second quarter constitute SEK -3.8m (SEK -3.3m).
- Income after financial items for the period totaled SEK -7.9m (SEK -2.6m) of which the second quarter constitute SEK -3.8m (SEK -3.3m).
- Earnings per share for the period were SEK -0.10 (SEK -0.08) of which the second quarter constitute SEK -0.05 (SEK -0.10).
- Exercise of warrants TO1 2013 and the directed share issue to employees brought in SEK 17.2m during the period before issue expenses.
- Cash flow from operating activities for the period totaled SEK -7.2m (SEK -5.1m) of which the second quarter constitute SEK -4.0m (SEK -2.8m).
- Equity as of June 30, 2014 totaled SEK 33.8m (SEK 12.1m) or SEK 0.44 (SEK 0.37) per share. The equity/assets ratio as of June 30, 2014 was 77 percent (78 percent).
- Cash and cash equivalents as of June 30, 2014 totaled SEK 31.1m (SEK 4.0m).

### 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 (CLL) 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 Euro 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.

#### **Significant events after the end of the reporting period**

- 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 hereby announces that animal studies are proceeding according to plan towards the selection of a candidate drug in Q3. The results so far support that an effective concentration of the ROR inhibitor can be achieved in cancer cells for long enough to reach the desired anti-cancer effect. Animal studies are currently being conducted in order to assess efficacy and safety, why risks in the development of the drug candidate in the ROR project remains.
- Kancera hereby announces 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 announces 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.

## Statement from the CEO

During the second quarter, the ROR project has been presented at scientific annual meetings of the American Society of Clinical Oncology, the American Association for Cancer Research and the European Haematology Association, which has generated new contacts with companies that may become Kancera future partners.

In the ROR project studies that show how the concentration of Kancera's small molecule inhibitors in the body is linked to effect. At present, the studies progress according to plan towards the selection of a candidate drug. The results so far support that we can reach the desired anti-cancer effect. However, efficacy and safety studies to show this are currently being conducted, why risks in the development of the drug candidate in the ROR project remains.

The development of an active immunotherapy against ROR has reached a milestone since we can show that Kancera's first series of cancer vaccines results in an immune response in animals with antibodies that recognize ROR. The continued work in the autumn is now directed towards examining whether the animal's immune response also leads to the production of antibodies that have the ability to stop cancer cells, and if so, how this effect can be optimized. The HDAC6 project has developed at a faster pace than expected, which is reflected in the recent Kancera HDAC6 inhibitors that are more potent and more selective against Multiple Myeloma cells than the competitor Acetylon's most advanced substance ACY-1215. However, the HDAC6 field moves rapidly forward, which means that better substances are constantly developed. With Kancera's recent successes, we see good opportunities to be competitive.

In the PFKFB3 project promising biological discoveries justify a continued collaboration with Professor Helleday's group at the Science for Life Laboratory. In the next step of this cooperation we will test if inhibition of PFKFB3 constitutes an effective attack on a number of the cancers that are most difficult to treat.

While the second-quarter external presentations mainly took place at scientific meetings, the marketing in the third quarter will focus more towards drug development and commercial partners. Next we meet pharmaceutical companies at the Nordic Life Science Days in Stockholm September 7-9 in Stockholm and thereafter we participate in "Cancer Immunotherapy Partnering Mission 2014" which is a concentration of businesses and researchers from Scandinavia that during a week in September will meet large pharmaceutical companies and institutions (including NIH) in Boston, New York, Baltimore and Washington DC.

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

<b>Financial development, summary</b>							
<i>SEK 000's (if otherwise not specified)</i>							
<b>Kancera AB</b>	1 Jan-31 March		1 April-30 June		1 Jan-30 June		1 Jan-31 Dec
	2014	2013	2014	2013	2014	2013	2013
Net turnover	212	-	153	218	365	218	1 813
R&D expenses	-3 468	-1 492	-3 435	-2 071	-6 903	-3 563	-7 533
Operating Income	-4 149	-2 311	-3 803	-3 274	-7 952	-5 585	-10 404
Income after financial items	-4 173	690	-3 752	-3 274	-7 925	-2 584	-7 418
Net income	-4 173	690	-3 752	-3 274	-7 925	-2 584	-7 418
Cash-flow from operating activities	-3 173	-2 236	-3 991	-2 844	-7 164	-5 080	-6 638
Cash-flow from financing activities	9 311	4 445	15 321	1 500	24 632	5 945	17 649
Earnings per share, before and after dilution	-0,06	0,04	-0,05	-0,10	-0,10	-0,08	-0,22
Cash on hand at closing date	19 756	5 316	31 086	3 972	31 086	3 972	14 118
Solvency ratio	72%	93%	77%	78%	77%	78%	74%
<b>Key ratios</b>							
Return on equity, %	neg	neg	neg	neg	neg	neg	neg
Return on capital employed, %	neg	neg	neg	neg	neg	neg	neg
Solvency ratio	72%	93%	77%	78%	77%	78%	74%
Investments in tangible assets	500	2 000	-	-	500	2 000	2 000
No. of employees	10	7	10	7	10	7	7,5
Earnings per share, before dilution	-0,06	0,04	-0,05	-0,10	-0,10	-0,08	-0,22
Earnings per share, after dilution	-0,06	0,04	-0,05	-0,10	-0,10	-0,08	-0,22
Equity by share, kr	0,31	1,01	0,44	0,37	0,44	0,37	0,56
Cash-Flow by share, kr	0,08	0,01	0,15	-0,04	0,22	-0,04	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. The lower income after financial items and earnings per share for the period compared to the corresponding period in 2013 can be attributed to a capital gain that arose in 2013 in connection with a claim in the former subsidiary. Comparative figures from the preceding year relate to the parent company Kancera AB.

### Net sales

Kancera's activities have mainly covered internal drug development projects alongside smaller consultancy projects which raised net sales during the period of SEK 0.4m (SEK 0,2m). The turnover excludes financial support from the EU project A-Paradise where the support is offset against incurred costs for the period amounting to SEK 1.3m (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.

### Expenses

Expenses in the second quarter totaled SEK 4.0m (SEK 3.5m), which breaks down into costs of services sold of SEK 0.1m (SEK 0.1m), research and development expenses of SEK 3.4m (SEK 2.1m) and other sales and administrative expenses of SEK 0.5m (SEK 1.3m). Expenses during the period January 1 to June 30 2014 amounted to SEK 8.2m (SEK 5.8m) which breaks down into costs of services sold of SEK 0.2m (SEK 0.1m), research and development expenses of SEK 6.9m (SEK 3.6m) and other sales and administrative expenses of SEK 1.2m (SEK 2.1m).

**Earnings**

Income after financial items for the second quarter totaled SEK -3.8m (SEK -3.3m) and for the period SEK 7.9m (SEK -2.6m).

**Cash flow and liquidity**

Cash flow totaled SEK 11.3m (SEK -1.3m) in the second quarter. Cash flow from operating activities for the second quarter amounted to SEK -4.0m (SEK -2.8m). Cash flow from financing activities for the second quarter amounted to SEK 15.3m (SEK 1.5m).

Cash flow during the period totaled SEK 17.0m (SEK -1.1m). Cash flow from operating activities during the period amounted to SEK -7.2m (SEK -5.1m). Cash flow from financing activities during the period amounted to SEK 24.6m (SEK 5.9m) 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 Euros from the European Union's 7th Framework Programme 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.

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. 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.

Kancera's cash and cash equivalents as of June 30, 2014 totaled SEK 31.1m (SEK 4.0m).

**Investments**

Investments in fixed assets in the second quarter totaled SEK 0.0m (SEK 0.0m) and for the period net SEK 0.5m (SEK 2.0m).

Investments in intangible assets in the second 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 3.4m (SEK 2.1m) for the second 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 June 30, 2014 was SEK 33.8m (SEK 12.1m).

Share capital as of June 30, 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 second quarter, based on a weighted average of the number of outstanding shares, were SEK -0.05 (SEK 0.10). In connection with the share issue 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 June 30, 2014 was 77 percent (78 percent). Total equity per share was SEK 0.44 (SEK 0.37) 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 63.4m.

**Personnel**

Kancera AB had 10 full time employees (7) as of June 30, 2014 of which 7 are men and 3 are women.

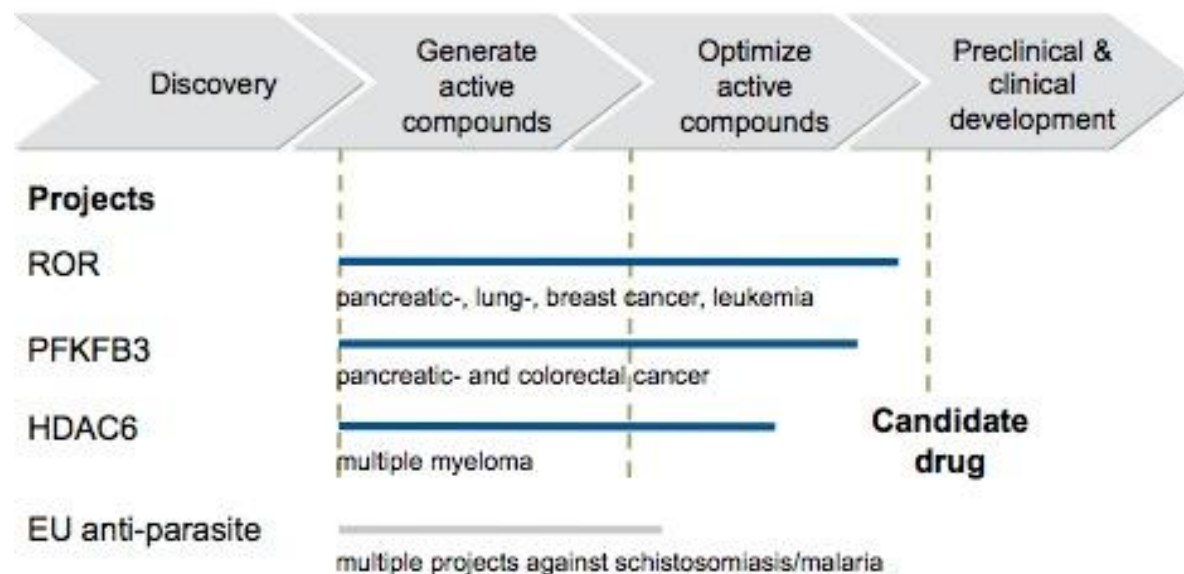
## Pharmaceutical Development

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 so that it destroys itself. 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 PFKB 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



The product development in the ROR project aims to deliver a drug candidate during the third quarter with the potential to treat refractory solid cancers as well as hematologic cancers. This means that also the commercialization of this project will commence in 2014. However, the road towards the selection of a candidate drug is still risky since necessary safety- and efficacy studies are currently ongoing. A successful commercialization may involve a sale probably with a stepwise compensation at signing of the agreement and when the project reaches milestones, or a partnership that yields net income and funding of a continued drug development.

In line with the Board's goal to increase the financial flexibility of the company and at the same time keep sufficient capacity to deliver a drug candidate it was decided to mainly focus the activities on the ROR project and the EU-financed epigenetically directed anti-parasite project.

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, including "overhead" such as rent and administration 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 types of epigenetic projects. The HDAC6 project has been developed at a faster pace than previously estimated, which means that it is possible to select a candidate drug within 2 years.

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 cell's survival signal and thus re-program the cancer so that it destroys itself. 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 (for more information about the ROR vaccine, see below under Events during the period).

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 ROR1 inhibitors have been shown to be more effective and more selective when killing cancer cells from leukemia patients than two 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 CLL patients whose cancer is no longer sensitive to 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 reach 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 (70% killing of cancer cells at about 3 µM). 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). The results thus point to that Kancera's ROR-inhibiting drug will have a clear and important place in the treatment of severely ill cancer patients. 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 respective AML).

Kancera's ROR inhibiting substance KAN043936 is effective against cancer cells from patients and shows good

metabolic stability in human liver cells and blood and thus meets the requirements that the company places on a candidate drug in these respects. In addition, in vitro laboratory methods have shown that KAN0439365 meet the company's requirements regarding a low risk for adverse drug-drug interaction (CYP inhibition) and cardiac side effects (hERG activation). After 24 hours in vitro, KAN0439365 shows a significantly higher killing effect against cancer cells from treatment-resistant patients than the new and groundbreaking drug Ibrutinib which is now on the market.

Kancera has also developed ROR inhibitors that meet the company's requirements for stability in the liver of both humans and the type of rodent that is studied in human cancer models. Thus, pharmacokinetic studies and tolerance studies have started to select the appropriate ROR inhibitor for efficacy studies and selection of a candidate drug.

Kancera has applied for intellectual property protection for small-molecule ROR inhibitors by the patent application EP13180941.0.

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 ROR. 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 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 (CLL) 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.

Kancera considers it possible that, by the means of a vaccine, act in synergy with pharmaceutical intervention, surgery and radiation to create a long lasting effect of the treatment given initially. It is Kancera's strategy to use its future small-molecule ROR inhibitors as a first line treatment for the disease 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.

Research on ROR has led to the discovery of surface elements on the ROR molecule suitable for the development of an effective vaccine. Kancera now takes these findings further to develop a proprietary product for prophylactic treatment that will improve the situation for patients with a cancer known to relapse. The development of this product 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.

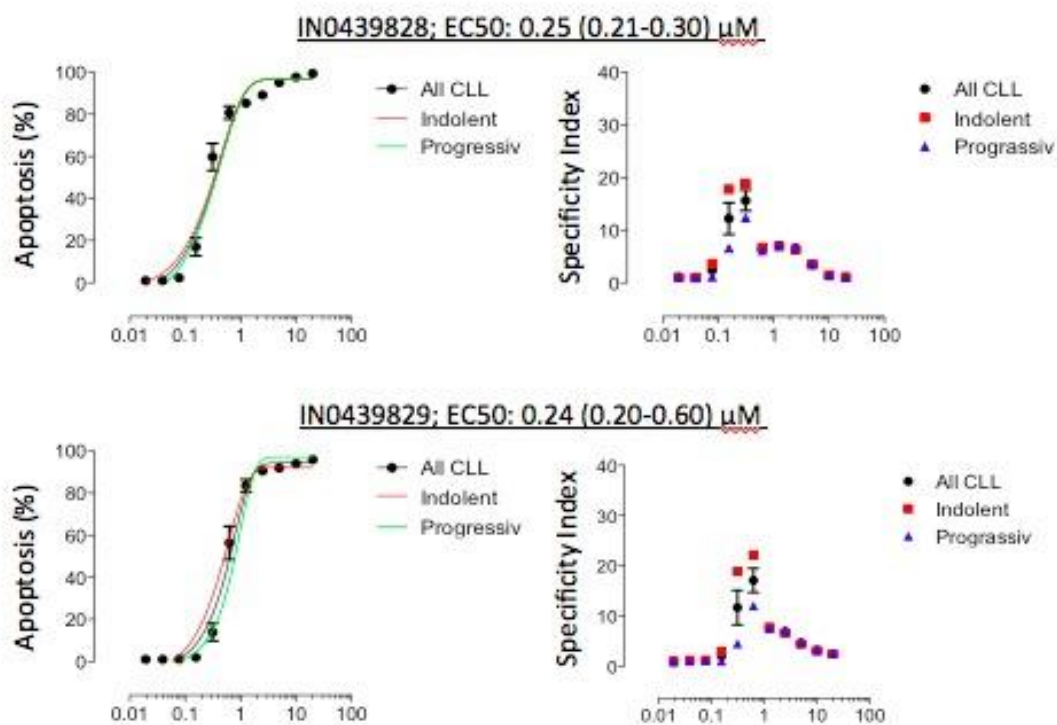
The principle to use a ROR vaccine for treatment is also supported by a preclinical study published by Professor



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 aim to demonstrate both the immune stimulating performance of the vaccine candidates and their therapeutic effect. This work is a step in the development of an optimized vaccine candidate and is expected to continue throughout in 2014. 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 has designed and synthesized new ROR inhibitors which according to Figure 2 show an increased efficacy against cancer cells. This 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. Meanwhile, the size of the ROR inhibitors has been reduced by approximately 20%, which may contribute to an improved uptake of the drug in the tumor. These advances have also led to new patentable structures and that a new security profiling is required. Overall, the results support the continued development of a small molecule drug candidate against ROR.



**Figure 2.** Results from studies of two of Kancera's recently developed ROR inhibitors KAN0439828 and KAN0439829. The graphs in the left column shows the percentage killed cancer cells (Apoptosis%; Y-axis) after 24 hours incubation with each ROR inhibitor at a logarithmically increasing concentration of the inhibitor. The right column shows the effect on healthy cells in the form of a "Specificity Index" that represents how many cancer cells are killed per healthy cell. In comparison with KAN0439365 (EC50: 1.5-2  $\mu\text{M}$ ), Kancera has succeeded to increase the potency of these ROR inhibitors at least six times. At the same time, the specificity against healthy cells has remained at a level of >10 times within the range of concentrations referred to be used therapeutically.

#### Events after the end of the period

Studies have been conducted to determine how high concentration of ROR inhibitor is needed outside the cancer cell in order for a sufficient amount to enter the cancer cell and start the process that leads to cancer cell death. Furthermore, studies have been conducted to determine how long time the ROR inhibitor needs to reside in the cancer cell in order to achieve a killing effect. These studies give support to that Kancera's substance KAN0439834 can achieve the desired effect in the ongoing animal studies.

Results from ongoing immunization experiments show that Kancera's vaccine candidates generate antibodies in animals that bind to ROR1. Further studies will be carried out in the autumn in order to detect whether the immune response results in antibodies that are able to stop cancer cells.

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

The project aims to develop PFKFB3 enzyme inhibitors to stragulate 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 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.

#### **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. 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.

#### **Events after the end of the period**

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.

#### **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 first quarter 2014 Kancera has 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 second quarter HDAC6 inhibitor have been developed that are more potent against cancer cells than Acetylon's ACY-1215 and also better tolerated by healthy blood cells from humans. For more information, see Table 1 below.

Table 1

a)			
Cells/ Compound	KAN0439221 IC50/ $\mu\text{M}$	KAN0439782 IC50/ $\mu\text{M}$	ACY-1215 IC50/ $\mu\text{M}$
RPMI-8226	0.44	0.63	8.1
AMO-1	0.94	1.0	4.6
OPM-2	0.46	>20	7.6
PBMC (healthy donor #6102)	>20 (18% inh @ 20 $\mu\text{M}$ )	>20 (13% inh @ 20 $\mu\text{M}$ )	5.2
b)			
Cells/ Compound	KAN0439221 IC50/ $\mu\text{M}$	KAN0439782 IC50/ $\mu\text{M}$	ACY-1215 IC50/ $\mu\text{M}$
RPMI-8226	0.45	0.32	9.4
AMO-1	0.79	0.81	5.0
OPM-2	0.64	0.84	3.9
PBMC (healthy donor #6102)	>20 (32% inh @ 20 $\mu\text{M}$ )	>20 (9% inh @ 20 $\mu\text{M}$ )	3.4

Table 1a and 1b show the cell killing effect (IC50 /  $\mu\text{M}$  = concentration of the substance expressed in micromolar at which 50% of full effect) of HDAC6 inhibitor at three different cell lines isolated from patients with disease multiple myeloma (RPMI 8226, AMO-1, and OPM-2) and on blood cells from a healthy donor (PBMCs). After both 48 hours (a) and 72 hours (b) the Kancera HDAC6 inhibitors KAN0439221 and KAN439782) exhibit a higher potency against multiple myeloma cells and a desired lower potency against healthy cells compared to Acetylon's ACY-1215.

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

The project 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*)).

**Market outlook for Kancera's development projects**

In 2013, the European Medicines Agency EMA approved 38 new drugs which represent a steady increase in numbers from the 10 new approved drugs in 2010. EMA now approves more new drugs than the corresponding American authority (FDA) which approved 27 new drugs in 2013. Of these, the vast majority were synthetic drugs, which is Kancera's focus, while only two were biologics. The cancer indication still dominates by constituting 37% of these new approved drugs (Source: EMA and FDA).

Kancera 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: <http://www.burrillandco.com/>). 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 recent 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/](http://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 CSF (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.



<b>Income Statement</b>	1 Jan-31 March		1 April-30 June		1 Jan-30 June		1 Jan-31 Dec
<i>SEK 000's (if otherwise not specified)</i>	2014	2013	2014	2013	2014	2013	2013
<b>Kancera AB</b>							
<i>Revenues</i>							
<b>Net sales</b>	<b>212</b>	<b>-</b>	<b>153</b>	<b>218</b>	<b>365</b>	<b>218</b>	<b>1 813</b>
Cost of sales & services	-106	-	-99	-142	-205	-142	-530
<b>Gross profit</b>	<b>106</b>	<b>-</b>	<b>54</b>	<b>76</b>	<b>160</b>	<b>76</b>	<b>1 283</b>
<i>Operating Expenses</i>							
General & administrative expenses	-579	-704	-155	-985	-734	-1 689	-3 375
Selling expenses	-208	-115	-267	-294	-475	-409	-779
Research & development expenses	-3 468	-1 492	-3 435	-2 071	-6 903	-3 563	-7 533
	-	-	-	-	-	-	-
<b>Total expenses</b>	<b>-4 255</b>	<b>-2 311</b>	<b>-3 857</b>	<b>-3 350</b>	<b>-8 112</b>	<b>-5 661</b>	<b>-11 687</b>
<b>Operating income</b>	<b>-4 149</b>	<b>-2 311</b>	<b>-3 803</b>	<b>-3 274</b>	<b>-7 952</b>	<b>-5 585</b>	<b>-10 404</b>
<i>Income from Financial Investments</i>							
Financial income	34	3 001	53	0	87	3 001	3 001
Financial expenses	-58		-2		-60		-15
<b>Financial net</b>	<b>-24</b>	<b>3 001</b>	<b>51</b>	<b>0</b>	<b>27</b>	<b>3 001</b>	<b>2 986</b>
<b>Income after financial items</b>	<b>-4 173</b>	<b>690</b>	<b>-3 752</b>	<b>-3 274</b>	<b>-7 925</b>	<b>-2 584</b>	<b>-7 418</b>
Taxation	-	-	-	-	-	-	-
<b>Net income</b>	<b>-4 173</b>	<b>690</b>	<b>-3 752</b>	<b>-3 274</b>	<b>-7 925</b>	<b>-2 584</b>	<b>-7 418</b>
Earnings per share, before and after dilution	-0,06	0,04	-0,05	-0,10	-0,10	-0,08	-0,22

<b>Balance Sheet</b>	31 March		30 June		31 Dec
	2014	2013	2014	2013	2013
<i>SEK 000's (if otherwise not specified)</i>					
<b>Kancera AB</b>					
<i>Assets</i>					
<i>Non-current Assets</i>					
Intangible assets, activated R&D expenses	6 000	6 000	6 000	6 000	6 000
Tangible assets	4 550	5 000	4 367	4 750	4 291
<b>Total fixed assets</b>	<b>10 550</b>	<b>11 000</b>	<b>10 367</b>	<b>10 750</b>	<b>10 291</b>
<i>Current Assets</i>					
Receivables	525	137	1 081	781	1 240
Cash and cash equivalents	19 756	5 316	31 086	3 972	14 118
<b>Total current assets</b>	<b>20 281</b>	<b>5 453</b>	<b>33 483</b>	<b>4 753</b>	<b>15 358</b>
<b>TOTAL ASSETS</b>	<b>30 831</b>	<b>16 453</b>	<b>43 850</b>	<b>15 503</b>	<b>25 649</b>
<i>Equity and Liabilities</i>					
<i>Equity</i>					
Restricted equity	6 378	2 689	8 212	2 689	17 989
Non-restricted equity	15 894	12 671	25 629	9 397	967
<b>Total equity</b>	<b>22 272</b>	<b>15 360</b>	<b>33 841</b>	<b>12 086</b>	<b>18 956</b>
Long-term liabilities	3 322	-	3 322	1 500	1 500
Short-term liabilities	5 237	1 093	6 687	1 917	5 193
<b>Total provisions and liabilities</b>	<b>8 559</b>	<b>1 093</b>	<b>10 009</b>	<b>3 417</b>	<b>6 693</b>
<b>TOTAL EQUITY and LIABILITIES</b>	<b>30 831</b>	<b>16 453</b>	<b>43 850</b>	<b>15 503</b>	<b>25 649</b>

<b>Statement of Changes in Equity</b>			
<i>SEK 000's (if otherwise not specified)</i>			
<b>Kancera AB</b>			
	2014		2013
<b>Total equity, opening balance on the 1st of Jan 2014</b>	<b>18 956</b>	<b>Total equity, opening balance on the 1st of Jan 2013</b>	<b>10 225</b>
Proceeds on issue of shares	7 489	Proceeds on issue of shares	4 834
Costs related to issue of shares	-	Costs related to issue of shares	-389
Q1 net income	-4 173	Q1 net income	690
<b>Total equity, closing balance on the 31st of March 2014</b>	<b>22 272</b>	<b>Total equity, closing balance on the 31st of March 2013</b>	<b>15 360</b>
Proceeds on issue of shares	16 583	Q2 net income	-3 274
Costs related to issue of shares	-1 262	<b>Total equity, closing balance on the 30th of June 2013</b>	<b>12 086</b>
Q2 net income	-3 752		
<b>Total equity, closing balance on the 30th of June 2014</b>	<b>33 841</b>		

<b>Cash-Flow Statement</b>	1 Jan-31 March		1 April-30 June		1 Jan-30 June		1 Jan-31 Dec
<i>SEK 000's (if otherwise not specified)</i>	2014	2013	2014	2013	2014	2013	2013
<b>Kancera AB</b>							
<i>Cash-flow from operating activities</i>							
Operating income after financial items	-4 173	690	-3 752	-3 274	-7 925	-2 584	-7 418
Depreciation	241	-	259	-	500	-	709
Other non-cash-flow affecting items	-	-3 000	-	-	-	-3 000	-3 000
<b>Cash-flow from operating activities before working capital change</b>	<b>-3 932</b>	<b>-2 310</b>	<b>-3 493</b>	<b>-3 274</b>	<b>-7 425</b>	<b>-5 584</b>	<b>-9 709</b>
Change in working capital	759	74	-498	430	261	504	3 071
<b>Cash-flow from operating activities</b>	<b>-3 173</b>	<b>-2 236</b>	<b>-3 991</b>	<b>-2 844</b>	<b>-7 164</b>	<b>-5 080</b>	<b>-6 638</b>
<i>Investment activities</i>							
Investment in tangible assets	-500	-2 000	-	-	-500	-2 000	-2 000
<b>Cash-flow from investment activities</b>	<b>-500</b>	<b>-2 000</b>	<b>-</b>	<b>-</b>	<b>-500</b>	<b>-2 000</b>	<b>-2 000</b>
<b>FREE CASH-FLOW available to INVESTORS</b>	<b>-3 673</b>	<b>-4 236</b>	<b>-3 991</b>	<b>-2 844</b>	<b>-7 664</b>	<b>-7 080</b>	<b>-8 638</b>
<i>Financing activities</i>							
Issue of shares	7 489	4 445	15 321	-	22 810	4 445	16 149
Financing from the EU	1 822	-	-	-	1 822	-	-
New loans	-	-	-	1 500	-	1 500	1 500
<b>Cash-flow from financing activities</b>	<b>9 311</b>	<b>4 445</b>	<b>15 321</b>	<b>1 500</b>	<b>24 632</b>	<b>5 945</b>	<b>17 649</b>
<b>CASH-FLOW for the YEAR</b>	<b>5 638</b>	<b>209</b>	<b>11 330</b>	<b>-1 344</b>	<b>16 968</b>	<b>-1 135</b>	<b>9 011</b>
Cash and cash equivalents at the beginning of the year	14 118	5 107	19 756	5 316	14 118	5 107	5 107
Cash and cash equivalents at the end of the year	19 756	5 316	31 086	3 972	31 086	3 972	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

No 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.

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. 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 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, August 22, 2014

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

- Interim report January-September 2014      November 21, 2014
- Year End Report 2014      February 20, 2015

For further information, please contact:

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

### Kancera AB (publ)

Karolinska Institutet Science Park  
Banvaktsvägen 22  
SE-171 48 Solna

Please visit the company's website [www.kancera.com](http://www.kancera.com)