

Initiation of Coverage

KANCERA AB

Since its founding in 2010, Kancera has worked with the development of small molecule drug candidates for inflammatory diseases and cancer. The company conducts clinical development in myocardial infarction.

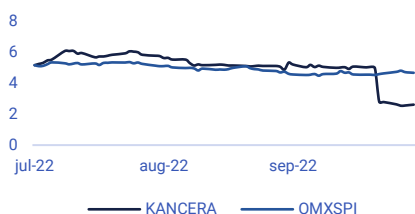
CEO: Thomas Olin
CoB: Erik Nerpin
www.kancera.com

Bloomberg: KAN:SS
Refinitiv Eikon: KANC.ST

Listing: Nasdaq OMX First North Premier

Last price: SEK 2.6
Market Cap: SEK 143m

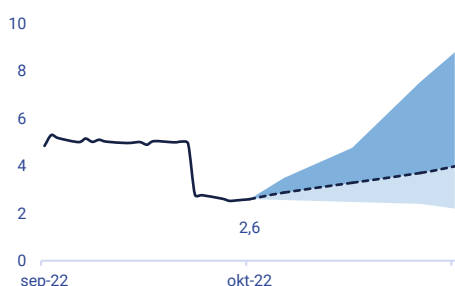
SHARE DEVELOPMENT



| | 12M | YTD | 6M | 1M |
|----------|-----|-----|-----|-----|
| Dev. (%) | -71 | -58 | -58 | -50 |

Source: S&P Capital IQ

VALUATION INTERVAL



| | BEAR | BAS | BULL |
|-------------------|------|-----|------|
| Share Price (SEK) | 2.2 | 4.1 | 8.8 |
| Up-/downside (%) | -15 | 58 | 238 |

Source: S&P Capital IQ and Carlsquare estimates

CARLSQUARE EQUITY RESEARCH

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Expands the clinical program

Carlsquare Equity Research begins coverage of Kancera. In the event of a positive outcome in the ongoing study in myocardial infarction patients (results are expected in the first half of 2023), we see great potential in the shares. At the same time, it is a challenging indication for clinical development, and we welcome the announced broadening of the clinical program to cancer, which reduces dependence on a single project. To finance the investment, Kancera is raising up to approximately SEK 135 million. We calculate a valuation range of around SEK 2 to 9 per share over a period of around nine months.

Readout from the first myocardial infarction study in H1, 2023

Kancera's first study in myocardial infarction patients is nearing its final stage and is expected to be fully recruited at the turn of the year. We see the fact that the study largely follows the schedule as a good sign. The study is controlled and, although the sample is limited, the outcome should first evidence regarding effect for important clinical parameters such as infarct size and heart function. There are no approved treatments for this indication, a market that could be worth at least a billion dollars. We assess that external clinical results provide some support for anti-inflammatory treatment in connection with today's standard treatment (angioplasty/PCI). We predict that topline results in Kancera's study may be reported towards the end of the first half of next year, and these will have a very large impact on the view of the company's fractalkine inhibitors.

Share issue for an expanded clinical program

Recently, Kancera has announced that it will begin clinical development with the fractalkine project in cancer next year. The focus is on ovarian cancer and combination with standard treatment to, if possible, overcome resistance to chemotherapy. Preclinical studies show that the company's candidate inhibits a mechanism for DNA repair, which makes it rational to combine it with chemotherapy to improve the effect. We see good potential in cancer and welcome the broadening of the clinical program. Kancera intends to run the cancer program up to and including a proof-of-concept study (phase IIa) and for that purpose the company is carrying out a rights issue of approximately SEK 90 million at subscription price of two SEK per share. At the same time, warrants are issued that can bring in up to approximately SEK 45 million when they expire in May 2023.

Valuation and news flow should support the share price

We assess that Kancera is valued lower than similar companies. With a relatively conservative assumption about the probability of success in myocardial infarction, in line with the average historical outcome for similar indications, our valuation model gives a risk-adjusted justified value of just over four SEK per share after separation of unit rights and fully diluted.

We believe that the share can multiply if the study in myocardial infarction shows promising results with a positive trend in clinical effect on infarct size and function. In return, we see further fall in the stock if the outcome is disappointing and does not support further development in myocardial infarction. The risk is mitigated to some extent by the fact that the clinical program is expected to be expanded to cancer. The expansion also provides the opportunity for a supportive news flow in the coming months if and when Kancera gets the go-ahead for a cancer study.

Key Ratios (SEKm)

| | 2020 | 2021 | 2022P | 2023P | 2024P | 2025P |
|-----------|-------|-------|-------|-------|-------|-------|
| Net Sales | 2.7 | 0.0 | 0.0 | 0.0 | 25.0 | 183.0 |
| Revenue | 5.4 | 1.9 | 0.5 | 0.0 | 25.0 | 183.0 |
| EBITDA | -38.4 | -44.9 | -53.1 | -59.2 | -42.5 | 171.5 |
| EBIT | -40.1 | -45.3 | -53.4 | -59.3 | -42.9 | 170.9 |
| EBT | -40.5 | -45.7 | -53.7 | -59.3 | -42.9 | 170.9 |
| EPS (SEK) | -1.3 | -0.9 | -0.9 | -0.5 | -0.4 | 1.5 |
| EV/Sales | 47.2x | NaN | NaN | NaN | 5.1x | 0.7x |
| EV/EBITDA | NM | NM | NM | NM | NM | 0.7x |
| EV/EBIT | NM | NM | NM | NM | NM | 0.7x |

Källa: Bolagsinformation och Carlsquare prognoser

Investment case

A positive outcome from the first phase II trial in myocardial infarction could lead to a rapid re-rating for Kancera shares in the next year, and vice versa. Expected start of a cancer study can provide supportive news flow before then. The announced rights issue of approximately SEK 90 million lends credibility to the strategy of broadening the clinical program to oncology. We calculate a valuation range of approximately SEK 2 to 9 per share over a period of approximately nine months. Our risk-adjusted valuation in the base case amounts to roughly four SEK per share after full dilution, which we believe is supported by a relative valuation.

Important milestone and expansion in sight

Since its founding in 2010, Kancera has worked with the development of small molecule drug candidates for inflammatory diseases and cancer. The company began clinical development with KAND567 in 2017 and since 2020 trials have been ongoing in patients. In 2021, a controlled study was initiated in ST-elevation myocardial infarction in patients undergoing percutaneous coronary intervention (PCI).

- **Significant clinical milestone ahead.** We assess that the recruitment to the phase II study in myocardial infarction is progressing largely according to plan (54 patients at the end of September). Kancera expects to include the last patient around the turn of the year. The plan is to present top-line results in the first half of 2023. This constitutes a very significant milestone where we expect the first results regarding clinical effect in this patient group to be presented.
- **Second generation fractalkine inhibitors approach clinic.** KAND567 has in previous studies been generally safe and tolerable but requires high dosage. Kancera has completed the preclinical development of the candidate KAND145, which has improved properties including increased uptake in tumors. In addition to improved formulation, KAND145 also entails extended patent protection if the patent application is approved, which should facilitate discussions with potential partners.
- **Broadened clinical program strengthens opportunities.** Recently, Kancera has announced that the company intends to start clinical development with the fractalkine project in cancer during the next year. The focus is on ovarian cancer and combination with standard treatment. Given that the authorities approve the arrangement, it means a welcome broadening and acceleration of the clinical program for fractalkine inhibitors.
- **Good scientific and commercial rationale in ovarian cancer.** Preclinical results suggest that Kancera's candidate can counteract tumor resistance to platinum-based chemotherapy by preventing DNA repair. Ovarian cancer is a challenging indication but the success of PARP inhibitors such as Astra Zeneca's multi-blockbuster Lynparza shows that blockade of DNA repair is a viable path. There is currently a lack of adequate treatments for platinum-resistant ovarian cancer, which underlines the potential.
- **Share issue to finance cancer trials.** Kancera has announced a rights issue of approximately SEK 90 million with a subscription period of 1 to 15 November and also has a relatively good initial financial position and limited fixed costs. If the issue is subscribed to a large extent, we

calculate that Kancera will have the financial endurance to at least reach crucial milestones and carry out clinical development up to and including phase II in both ovarian cancer and heart attack. Kancera themselves assess that the guaranteed amount (that is, 50 percent of the rights issue) is sufficient to carry out these two studies.

Intended development plan for Kancera

| | Discovery | Preclinical PoC | Preclinical tox | Phase I | Phase II | Phase III | NDA |
|---------|-----------|-----------------|-----------------|---------|----------|-----------|-----|
| KAND567 | → | | | | | | |
| KAND145 | → | | | 2023 | | | |
| PFKFB3 | → | | | | | | |
| ROR1 | → | | | | | | |

Source: Kancera, Carlsquare.

- Great potential but also high risk in heart study.** We assess that Kancera is valued somewhat lower than similar companies. We expect that upcoming top-line results in myocardial infarction will have great significance for the outlook for the shares and the company. In a positive scenario, we estimate that the shares can rise multi-fold from today's levels. We believe today's valuation reflects low expectations about the chance of success, which in turn is largely explained by the fact that the indication has historically been challenging for clinical development. Only roughly 20 percent of all phase II studies are successful in the indication cardiovascular diseases. In a negative scenario where development in cardiovascular disease is shut down, we see, on the other hand, a further fall, tentatively to just over two SEK per share.

Assumptions and forecasts

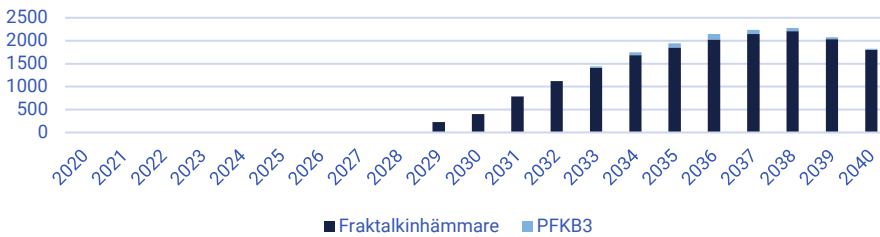
Targets key niche markets in major indications

Kancera develops drug candidates for serious inflammatory diseases and cancer. The company focuses on submarkets where there is a complete or partial lack of effective drug treatments. Although there is potential competition developing, we see good opportunities for high market shares.

We see a sales potential of around USD 1.2 billion for Kancera's portfolio based in the indications of myocardial infarction, ovarian cancer, and rectal cancer. So far, however, there is yet no evidence for clinical effect in these indications, which makes it difficult to assess the prospects.

Given that the planned studies in one or both of the clinical programs are successful, we see the possibility that the internal projects can be outlicensed in a few years' time. In our base scenario, we expect that this can happen from 2024 and that Kancera will then receive initial income in the form of upfront payment. We calculate with possible license deals with a value of up to between 200 and 700 MUSD based on a comparison with similar external projects. We assume that the company's future income will primarily consist of royalties from license agreements. Illustrated below is the expected royalty income from sales.

Estimated royalty income (SEK million) (not risk-adjusted)

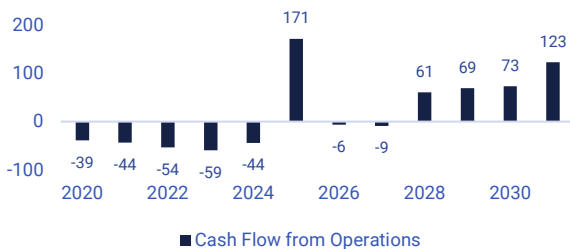


Source: Company information and Carlsquare forecasts

Increased clinical activity drives costs

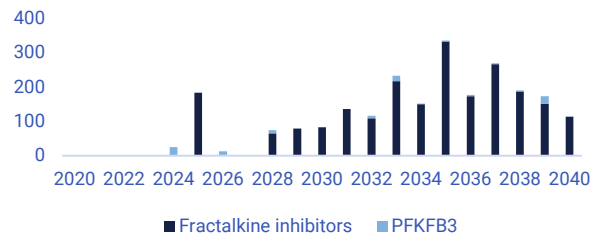
Kancera has a lean organization and low fixed costs. The company's ambition is to further develop and broaden the clinical program from next year, which means increased costs. However, we expect the business to be sufficiently financed to reach important milestones in the coming year. The announced rights issue should give Kancera financial scope to further develop the clinical programs up to and including phase II. If these studies are successful, we expect improved conditions for financing or partner collaborations.

Cash flow from current operations (SEK million), risk-adjusted



Source: Company information and Carlsquare forecasts

Risk-adjusted net revenue (SEK million)



Source: Carlsquare forecasts

Valuation

Phase II results and start of cancer study value drivers

We calculate an enterprise value of approximately SEK 290 million. Our valuation model is based on Kancera being able to find partners for both the fractalkine inhibitors and PFKFB3 after successful development. We have expected full dilution from the recently made public rights issue including warrants. All in all, the risk-adjusted justified value is just over four SEK per share in our base scenario (after full dilution).

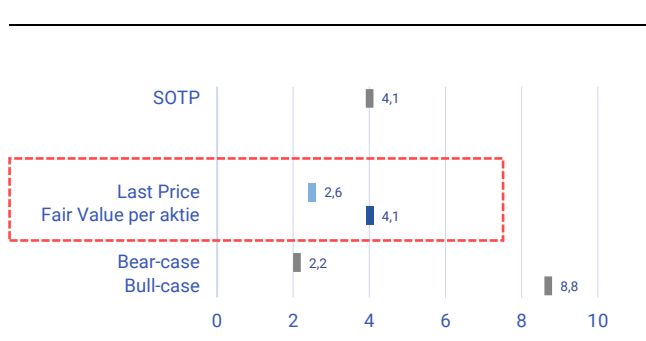
In our bull scenario of around SEK nine per share we pencil in that Phase IIa in myocardial infarction is completed with promising results and that we can thus raise the LOA to 27 percent from ten percent. Clinical studies in cancer with fractalkine inhibitors are also starting as planned. In a conservative Bear scenario, we expect results in myocardial infarction to be disappointing and set the value to zero for this indication. We assume that clinical studies in cancer begin according to plan. We calculate a value of just over two SEK per share after dilution.

Compilation Sum-of-the-parts valuation, base scenario, SEKm

| Project | Indication | LOA*, % | Royalty, % | Peak Sales, MUSD | Launch | rNPV, SEKm |
|--------------------------------------|----------------|---------|------------|------------------|--------|------------|
| KAND567 | STEMI | 10 | 25 | 470 | 2029 | 227 |
| KAND145 | Ovarian cancer | 7 | 20 | 550 | 2031 | 163 |
| Others | Rectal cancer | 3 | 5 | 230 | 2033 | 37 |
| Technology value before taxes | | | | | | 436 |
| Overhead and taxes | | | | | | -141 |
| EV | | | | | | 287 |
| Net cash (22'Q3E) | | | | | | 67 |
| Fair value | | | | | | 363 |
| # shares (m) | | | | | | 56.1 |
| Per share, SEK | | | | | | 6.3 |
| Assumed financing | | | | | | 121 |
| Shareholder value | | | | | | 476 |
| Shares fully diluted (m.) | | | | | | 116.0 |
| Fair value per share, SEK | | | | | | 4.1 |

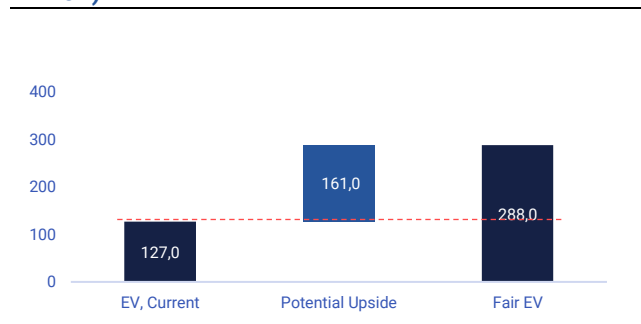
Source: Carlsquare *LOA: Likelihood of Approval

Fair value within a range, SEK



Source: Carlsquare forecasts

Visualization of market value, base scenario (SEK million)



Source: Carlsquare forecasts

Relative valuation

The reference group below is a heterogeneous group where we have taken indication, mechanism of action and clinical development into account in order to find reasonably relevant comparison objects. The valuation is clearly linked to the development phase and also external validation in the form of partner

collaborations and ownership. The comparison indicates that Kancera is valued lower than similar companies. The valuation is likely to be pressured, at least in the short term, by the recently announced rights issue. The relative valuation provides some support for our project valuation model based on risk-adjusted forecasted cash flows, in our opinion.

Valuation reference group (SEK million)

| Bolag | EV (SEKm) | Fas |
|--------------------------------------|------------|-----|
| Medivir AB (publ) | 306 | 1 |
| Modus Therapeutics Holding AB (publ) | 23 | 1 |
| Cantargia AB (publ) | 276 | 2 |
| Active Biotech AB (publ) | 248 | 2 |
| Faron Pharmaceuticals Oy | 1 292 | 2 |
| Quantum Genomics Société Anonyme | 1 224 | 3 |
| Median | 291 | |
| Kancera* | 127 | |

Source: S&P Capital IQ. * Assumed that Kancera's rights issue will be fully subscribed.

Risks and challenges

Challenging indications

Cancer and cardiovascular diseases are among the most challenging areas in clinical development with a relatively low probability of success. Clinical studies in cardiovascular diseases are extensive. Most likely Kancera will have to find a larger partner to carry out a possible phase III study. Although there is evidence for a biological effect in humans for Kancera's fractalkine inhibitors, it is not certain that it will translate into a relevant clinical effect in patient groups with acute myocardial infarction or advanced cancer.

Funding for clinical development

Kancera has announced a rights issue to finance clinical studies in oncology (ovarian cancer). It is a prerequisite for developing the project portfolio at the desired pace. There is a risk that the issue will not be fully subscribed. In addition, Kancera may be forced to rely on guarantors who are usually short-term investors. In a positive scenario, future financing can take place via share issues priced at a higher valuation than the current situation, or alternatively by a larger partner taking responsibility for the further development in a licensing agreement.

Competition

There is a clear medical need for treatments to improve outcomes in acute myocardial infarction. Although Kancera is a leader in the fractalkine blockers space, there are external projects in the pipeline with other mechanisms of action that are further along in clinical development than KAND567.

In cancer, there is a high level of clinical activity (for example, 4,700 clinical studies in immuno-oncology took place in 2021). Many companies and drug candidates compete for resources and to recruit patients for clinical studies.

Introduction to the company

Kancera develops small molecule treatments for cancer and inflammatory diseases. Small molecules have important advantages that allow them to be combined with other treatments. The company is a leader in fractalkine inhibitors, a new concept for blocking harmful inflammatory signals. The company is in clinical development in myocardial infarction where topline results from a phase II study are expected during the next year. Recently, Kancera has decided to start clinical development in cancer.

About Kancera

Kancera is a research company in the clinical phase that develops new small molecule treatments against severe inflammation and cancer diseases. The focus is on new treatment concepts that involve blocking harmful inflammatory signals in cardiovascular diseases and, in cancer treatment, inhibiting tumor cell metabolism and ability to repair DNA damage. These properties indicate that the company's drug candidates also have good potential as additions to today's standard treatment for cancer patients. Kancera's most advanced project is in the treatment of myocardial infarction, and the company expects that a phase II study currently conducted in Great Britain will be fully recruited around the turn of the year. The company's goal is to start a clinical study in patients with ovarian cancer during the first half of next year.

The company's leading projects originates from the external developers Astra Zeneca and Sprint Bioscience. Kancera has cooperated or is cooperating in pre-clinical and clinical development with leading researchers in the respective specialty area at, among others, Freeman University Hospital in Newcastle, Great Britain, and Karolinska Institutet.

Kancera pipeline

| Project | Target | Indication | Phase |
|---------|----------------------|------------|-------------|
| KAND567 | Fractalkine receptor | STEMI | II |
| KAND145 | Fractalkine receptor | Oncology | Preclinical |
| PFKFB3 | PFKFB3 | Oncology | Preclinical |
| ROR1 | ROR1 | Oncology | Preclinical |

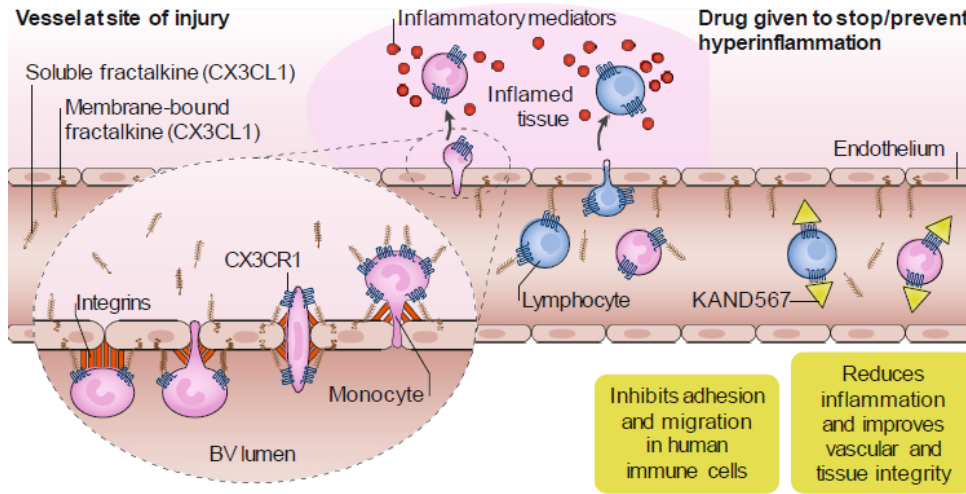
Source: Kancera

Leader in new immunological treatment concept

We assess that Kancera is a leader in drug development with the so-called fractalkine system as a treatment target. Fractalkine is an immunoregulatory factor, a so-called chemokine, which controls important functions of immune cells.

The drug candidate KAND567 blocks receptors (called CX3CR1) for this chemokine. Such receptors are expressed on immune cells (T cells, NK cells) and smooth muscle cells. By binding to these receptors, fractalkine makes it easier for circulating immune cells to be able to penetrate the walls of blood vessels and into tissue. There are also other molecular systems such as integrins and selectins that have a similar role. Fractalkine is found both in soluble form and in the endothelium (the inside of the blood vessels). In the latter case, the fractalkine is found on "stalks" of mucin (a common protein in the endothelium) which are attached to the blood vessel walls and which can trap cells carrying specific receptors. The levels of fractalkine molecules and CX3CR1 receptors have been shown to be elevated in several inflammatory diseases, in cancer and in chronic pain conditions.

KAND567 Mechanism of action



Source: Kancera. Immune cells ("Lymphocytes and Monocytes") invade tissues via blood vessels when receptors ("CX3CR1") on the surface of the immune cells bind to the immune-regulating protein fractalkine on the surface of the vessel wall. This allows the immune cell to pass through the vessel wall. KAND567 blocks the receptors on the immune cell and thus inhibits this invasion process. The aim is to stop or prevent harmful inflammatory processes in, e.g., heart infarction where immune cells become "culprits"

Fractalkine contributes to heart damage during infarction

Research has shown that CX3CR1 receptors are associated with atherosclerosis and that they are overexpressed in vascular plaques (Low, S., et al, "VHH antibody targeting the chemokine receptor CX3CR1 inhibits progression of Atherosclerosis", *MABS*, 2020). Researchers at Newcastle University have found in retrospective studies of around 1,400 heart attack patients that lymphopenia (low levels of white blood cells in the blood) after PCI was associated with poorer survival. One hypothesis is that the changed blood values are due to the fact that the immune cells in these patients migrated to the heart muscle with the help of the fractalkine system and there contributed to inflammation and ischemia/reperfusion damage.

Acute Myocardial Infarction is treated with surgery/angioplasty, or drugs/thrombolysis, to achieve reperfusion, i.e., restoration of blood flow in or around arteries after the blockage that caused the heart attack. Paradoxically, damage to tissue (for example the heart's musculature) that has occurred as a result of ischemia (strangled blood and oxygen supply) can worsen when blood flow is restored. This as a result of, among other things, an inflammatory reaction. In a smaller prospective study, it was shown that patients with the clearest signs of such reperfusion damage had a greater decline in T cells than others and that this decline was in turn positively correlated with expression of fractalkine receptors. Ischemic damage after PCI can account for up to 50 percent of the infarct size, according to animal studies. Despite this, studies have shown that the procedure still has advantages compared to thrombolysis in terms of mortality and morbidity in severe myocardial infarction. At the same time, it underlines a need to improve current treatment methods.

If the theory above is correct, fewer fractalkine-activated immune cells in tissues should in return reduce the risk of harmful inflammation. By blocking the fractalkine system, KAND567 reduces the ability of these immune cells to invade tissues from the bloodstream.

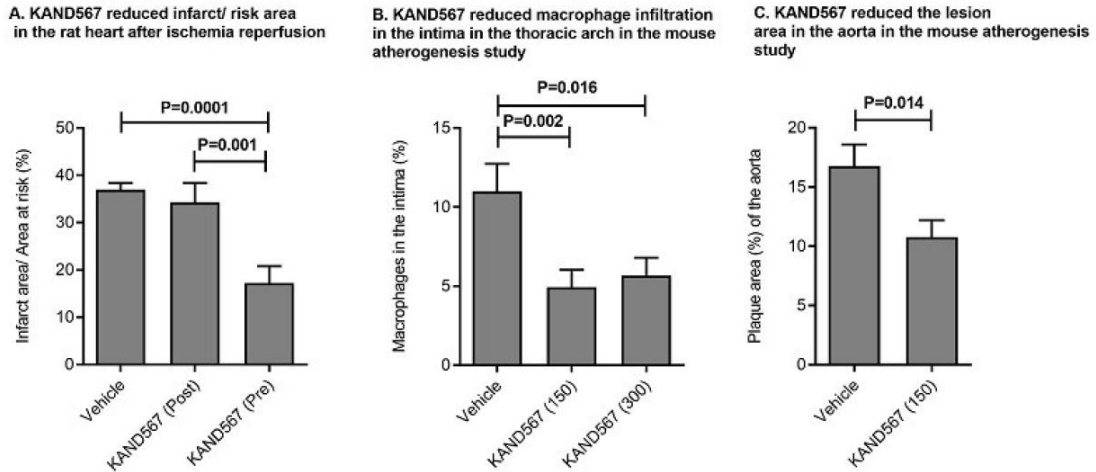
KAND567 is an allosteric modulator of the fractalkine receptor, that is, it affects CX3CR1 by binding to a different part of the receptor compared to the natural ligand fractalkine. The aim is to block the fractalkine system "upstream" at an early

stage, while the binding in theory enables a relatively wide therapeutic window compared to stronger antagonists.

Results in infarct model drew attention at international conference

Preclinical results regarding KAND567 in a model of myocardial infarction and atherosclerosis have shown that KAND567 reduces infiltration of immune cells into the heart and reduces infarct size by up to 50 percent in rats. The results have been presented at the European Society of Cardiology (ESC) Congress 2019.

Preclinical results for KAND567 support a cardioprotective effect

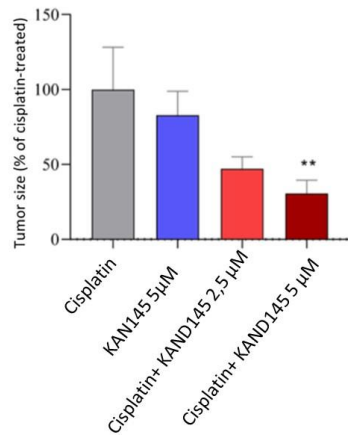


Source: Abdelmoaty, S. et al, "KAND567, the first selective small molecule CX3CR1 antagonist in clinical development, mediates anti-inflammatory cardioprotective effects in rodent models of atherosclerosis and myocardial infarction, abstract ESC 2019.

Fractalkine in cancer

The mechanism of action for fractalkine and fractalkine inhibitors is partly different in cancer compared to inflammatory and cardiovascular diseases. The hypothesis is that the fractalkine receptor has an important role in DNA repair, via a specific signaling pathway called Fanconi Anemia (FA). FA repairs so-called DNA crosslinking damage, which is the type of damage that platinum-based cytotoxics inflict on cancer cells. By blocking the fractalkine receptor, this signaling pathway and, thus, the repair process, is inhibited. This in turn reduces cancer cells' resistance to, for example, platinum-based chemotherapy agents. The mechanism of action is supported by the fact that people who lack FA genes have a poor ability to repair DNA damage and are sensitive to chemotherapy. In an in vitro model of cancer cell lines, researchers have shown that Kancera's fractalkine inhibitor improves response to treatment with platinum-based chemotherapy. Kancera has also announced promising in vivo results in a zebrafish model as well as in a mouse model where treatment-resistant tumors have been implanted. In these studies, Kancera's fractalkine blocker KAND145 in combination with platinum-based chemotherapy reduced the tumor volume.

KAND145 restores anti-tumor effect in model of platinum-resistant cancer



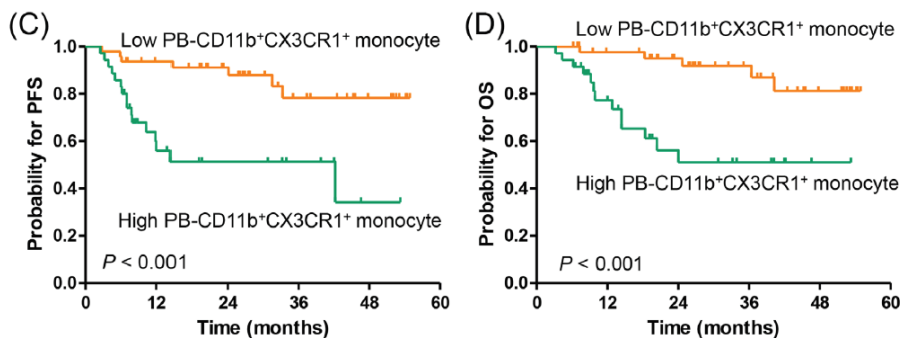
Source: Kancera

Fractalkine may have multiple roles in driving cancer

There is also a lot of external research that indicates that fractalkine has a role in several forms of cancer by facilitating the spread of tumors to surrounding tissue, by favoring the formation of new blood vessels by tumors and by having an immunosuppressive effect in the tumor microenvironment.

A prospective Korean study in lymphoma (89 participants) showed that patients with high expression of CX3CR1 receptors (about forty percent of patients) had worse survival when treated with the standard treatment R-CHOP than patients with low expression (Yhim, H., et al., "The prognostic significance of CD11b+CX3CR1+ monocytes in patients with newly diagnosed diffuse large B-cell lymphoma", *Oncotarget*, 2017).

Lymphoma patients with high expression of fractalkine receptors (green line) have worse survival with standard treatment



Source: Yhim et al (2017). PFS = Progression-free survival. OS = Overall Survival

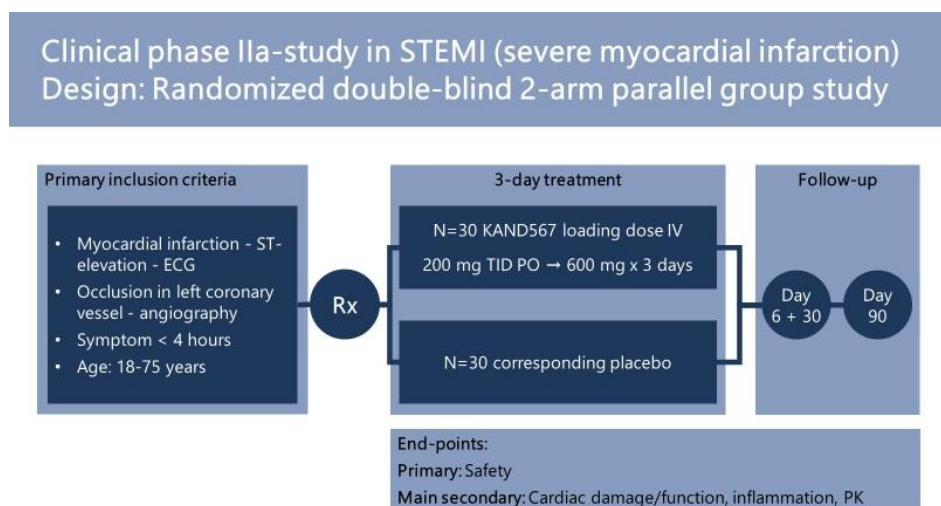
Recently, Kancera has also reported positive preclinical results in studies on cells from patients with Chronic Lymphatic Leukemia. In these studies, which were carried out in collaboration with Karolinska Institutet, increased cell death was observed in leukemia cells when treated with Kancera's fractalkine blocker. The studies show blockade of cancer-associated immune cells (monocytes) that contribute to stimulating the growth of leukemia cells. It indicates a potential for fractalkine blockers to improve efficacy for standard treatment.

Clinical development is ongoing in myocardial infarction

Currently, the phase IIa study FRACTAL is ongoing with a total of 60 patients who have suffered myocardial infarction in the anterior wall of the myocardium (STEMI). According to the updated timetable, the last patient is expected to be included around the turn of the year 2022/23, and topline data can be presented four to six months after that. At the end of September, Kancera stated that 54 patients had been recruited. According to our assessment, this suggests that the study is keeping to the planned pace. Kancera stated at the same time that the investigators are planning to take in another ten patients as complete samples have not been collected for all patients. We look positively on the efforts to ensure study quality. There are several examples of study quality being a challenge in clinical development during the pandemic, perhaps especially when it comes to follow-ups. We do not know if the expansion of the study is a sign of larger problems with the study, but we see a good recruitment rate as a good sign after all.

The study is placebo-controlled (1:1). The aim of the study is primarily the safety and tolerability of intravenous treatment with a bolus dose of 134 mg over six hours followed by oral treatment (a 200 mg tablet every eight hours for three days).

Study design myocardial infarction



Source: Kancera

For reference, a phase Ib study in 17 people showed that intravenous KAND567 for six hours was well tolerated. With longer infusions, inflammation in the blood vessels has been observed.

Oral administration has also been generally well tolerated. In 2020 to 2021, a study was conducted in Covid patients of which 15 received KAND567 250 mg twice a day for seven days. According to top-line results, this dose was well tolerated, and no treatment-related safety signals could be identified. In a phase I multi-dose study, treatment was well tolerated at doses up to 1000 mg per day (2 x 500 mg). At higher doses, dose-limiting toxicity occurred in the form of clinically relevant, but transient, effects on liver values.

Overall, KAND567 must be administered in relatively high doses but for a short time. Since similar dosage and treatment time have already been investigated, it suggests that even the ongoing study will show at least acceptable tolerability. However, the combination of intravenous and oral treatment has not yet been investigated in humans and, as in all drug development, it is not certain that all safety problems will be discovered already during the initial studies in humans.

Fraktalkin bidrar till ökad överlevnad för makrofager. Fractalkine contributes to increased survival of macrophages. Inhibition of the fractalkine system should therefore theoretically lead to a certain impairment of the immune system. So far, however, there are no reports of severe side effects in clinical trials. One caveat is that we do not know the background to the fact that a collaboration between Ablynx and Boehringer Ingelheim concerning fractalkine receptor blockers was apparently abandoned after a phase I study.

Outcomes will be scrutinized for trends in effect

Secondary objectives in the myocardial infarction study are biological and clinical outcome measures such as activated T cells, inflammation markers such as CRP and outcome measures assessed based on image diagnostics such as infarct size and heart function. We judge that the study, as it is relatively small, probably has too little statistical power for the differences between the groups to be statistically significant. However, we believe overall that the result will be interpreted positively if we see a clear positive and relevant trend in clinical efficacy measures, and vice versa. Achieving a reduction in infarct size is of utmost importance as there is a clear link to mortality.

In the Covid-19 study, the primary objective was to investigate the effect on oxygen restoration (ROX index) and respiration in patients with SARS-Cov-2 infection. According to top-line results, there was no difference in terms of the degree of lung damage, measured by CT scan, or oxygen absorption capacity. No results regarding the effect on inflammatory markers such as CRP or cytokines have been communicated. The study was small and there was an imbalance between the study arms in terms of inflammation status to the detriment of KAND567. However, according to Kancera, an effect was measured in the form of a significantly reduced activation of immune cells that carried the marker CD163. No detailed results have yet been published, which in combination with the limited sample overall makes it difficult for us to draw any conclusions for, for example, the chance of success in myocardial infarction.

External studies of anti-inflammatory treatments

To our knowledge, no other fractalkine blocker has been evaluated in myocardial infarction. However, there have been studies with a number of biological anti-inflammatory treatments with varying efficacy measures.

- An example is **anakinra** which is an IL-1 receptor antagonist marketed by SOBI. In a phase II study in STEMI, the systemic inflammatory response was significantly lower in the active arm and treatment with anakinra also resulted in a significantly lower risk of death or new infarction during twelve months of follow-up. However, no difference was demonstrated in the effect on heart function compared to placebo.
- The antibody **tocilizumab** (anti-IL6) has also been evaluated as an anti-inflammatory treatment in STEMI. In a placebo-controlled study in 200 patients, a statistically significant benefit was observed for tocilizumab regarding the myocardial salvage index, a measure of relative infarct size. However, the clinical significance of the difference was assessed as uncertain, and no impact on cardiac function was measured in this study compared to control.

Overall, we assess that the external studies above provide support that anti-inflammatory treatment can mitigate the extent of myocardial infarction and reduce the risk of death or new infarction, at least in the short term. However, the impact on heart function is still a question mark.

In a possible phase III study, we guess that reduction of mortality or heart failure will be the preferred primary objective of authorities. In addition to the fact that the requirements for clinical effect are high (probably around 30 percent relative

reduction is required), large studies with thousands of patients are also necessary to demonstrate statistical significance.

Clinical development in cancer

Kancera has announced that the company plans to start the clinical development in cancer with two parallel studies: A phase Ib/IIa where KAND567 is evaluated in cancer patients and a phase Ia where the sister candidate KAND145 is evaluated in healthy volunteers. The purpose of the arrangement is to speed up the development of KAND145, which is the preferred candidate in cancer.

Kancera will evaluate KAND567 in patients who have relapsed during treatment with platinum-based chemotherapy. It is a relatively broad inclusion criterion. One challenge is that there is probably a reluctance to give platinum-based treatment again after resistance, especially if there are alternatives. However, we assume that there will be seriously ill patients who have undergone several lines of treatment that will eventually be included because these are recommended to participate in clinical studies according to current guidelines. The patients will be treated with a combination of platinum-based chemotherapy and KAND567. The study program will begin with a dose escalation closely followed by a phase IIa part as a potential concept study in cancer.

The set-up makes it relevant to compare with standard treatment which is chemotherapy (paclitaxel, topotecan or PLD) with or without the addition of bevacizumab. In clinical studies, these treatments have demonstrated an objective response of twelve to 30 percent and a progression-free survival of three to seven months (better outcomes with bevacizumab). However, since bevacizumab is also indicated as adjuvant treatment, it limits the percentage of patients who can be treated with this antibody in later lines.

KAND567 and KAND145

The differences between KAND145 and KAND567 appear to be small but significant. According to the 2021 prospectus, '145 has properties that facilitate the formulation of an easily soluble and stable product for both oral and intravenous treatment. It makes it easier to give a high dose, for example, in a smaller capsule. It is stated that activation of the drug occurs through a process where '567 is released from '145. This suggests that for practical purposes KAND145 can be considered an improved formulation of KAND567. Examples of properties that Kancera hopes to improve are reduced liver toxicity and increased uptake (in tumors). Other positive effects with a new formulation are a potentially extended patent protection until 2038, and the possibility of price differentiation.

Preclinical portfolio

In addition to the fractalkine project, Kancera has three preclinical projects:

- An inhibitor of **ROR-1**, a growth factor found both on and in cancer cells and which is a treatment target to reprogram cancer cells to cell death. The development takes place in collaboration with independent academic research groups.
- The **HDAC6** project concerns a small-molecule inhibitor of HDAC6, an enzyme that controls how the cell's inner fibers function. In tumors, HDAC6 appears to enhance the ability to invade surrounding healthy tissue. Kancera continues to hold the patent application for the most promising chemical series.

Of the preclinical projects, however, PFKFB3 is prioritized. Kancera has selected KAND757 as a drug candidate for preclinical development. In 2021, Kancera reconsidered an earlier decision to wind down the project. The turnaround came in

the light of new studies (in vitro and in vivo (toxicity)) at the University Medical Center Göttingen that pointed to a tumor-killing effect on rectal cancer tumors.

KAND757 is a small molecule inhibitor of an enzyme called PFKFB3. Research has shown that cancer cells use up to 200 times as much sugar as healthy cells in their metabolism. The PFKFB3 enzyme acts here as an accelerator in the conversion of sugar into energy.

The mechanism of action is, through a specific inhibition of the PFKFB3 enzyme, to choke the metabolism of cancer cells and make them more sensitive to cell and radiation therapies. The company's hypothesis is also that inhibition of the enzyme reduces the ability of the cells to repair so-called double-strand breaks in DNA, which is the mechanism of action in radiation treatment of tumors.

Kancera, in collaboration with Professor Thomas Helleday's research group at SciLifeLab, has previously conducted studies that confirm that KAND757 weakens the cancer cell by blocking PFKFB3. In a later study published in *Nature Communications*, it has also been shown that KAND757 inhibits the cancer cell's ability to recover from DNA damage after radiation treatment without inhibiting the ability of healthy cells to repair damaged DNA. This lends support to the second type of mechanism of action.

Kancera intends to evaluate the effect in a larger tumor sample material and also develop a suitable technique for local delivery to the tumor. On an overall level, the project is somewhat reminiscent of KAND145 and, in addition, has apparently dual mechanisms of action in cancer.

History

Kancera has its roots in iNovacia AB, a consulting company in the early development of small molecule drug candidates that was spun off from Biovitrum in 2006. The research contributed to the development of several drug candidates, including the project that later led to the leukemia treatment Idhifa marketed by Bristol Myers Squibb. In 2008, the decision was made to start own drug development. In 2010, Kancera AB was formed by merging iNovacia with operations at Karolinska Institutet's cancer research center, with the help of a group of private investors. The company was formed through a combination of two cancer drug projects, the ROR and PFKFB3 projects. Kancera was listed on First North in 2011 at a pre-money valuation of approximately SEK 67 million. iNovacia operations were wound down in 2013 as external revenues did not justify further operations.

In 2014, development of the HDAC6 inhibitor began. In 2015, an agreement was concluded with Acturum Life Sciences AB to develop the project AZD8797 (immunological diseases with fractalkine as a treatment target) for pancreatic cancer. The following year, the entire project was acquired against payment in own files (a total of six million). In 2017, clinical development of the fractalkine inhibitor KAND567 began in healthy volunteers. In a strategic review in 2018, Kancera decided to focus on KAND567 and deprioritize the development of the preclinical portfolio. Nevertheless, in the same year, Kancera sold an option to the German drug company Grünenthal to license parts of the HDAC6 project for up to approximately EUR 33 million in milestone payments plus royalties on future sales (however, the option was never exercised and the HDAC6 project was returned). In 2019, the investment in the fractalkine area was intensified when a new candidate was developed at the same time as a new clinical study with intravenous administration of KAND567 was started with the aim of preparing for a phase II study in heart attack patients.

The pandemic that began in 2020 led to a greatly increased interest in anti-inflammatory treatments and KAND567 was evaluated in a phase II study in Covid-19 patients admitted to intensive care. The study was small, and no clinical effect could be demonstrated, but the treatment caused no serious side effects, and the investigators observed a positive biological effect on immune cells. In the fourth quarter of 2021, a clinical trial started in heart attack patients at Freeman Hospital and James Cook Hospital in the UK.

Share issue for cancer project

As of June 30, Kancera had approximately SEK 82 million in cash. In order to finance the clinical investment in fractalkine inhibitors in cancer, Kancera is carrying out a rights issue of approximately SEK 90 million. At the same time, up to around 15 million warrants (TO6) are issued, which can bring in up to SEK 45 million when fully subscribed (subscription period May 2023).

The proceeds are intended to finance a planned phase IIa study in ovarian cancer. Kancera has not communicated the study plan. However, we assess that Kancera, if the issue is fully subscribed, will have the necessary financing to carry out planned clinical development. A large part of the issue proceeds must also be used for the manufacture of pharmaceutical products.

In total, up to just under 60 million shares can be issued compared to the existing stock of around 56 million. The weak market climate has probably contributed to the large discount in the rights issue (issue price two SEK compared to the current share price of around five SEK before announcement). That view is supported by other financing rounds in recent times. The rights issue is guaranteed to 50 percent.

Share and valuation development

Like other biotechnology companies in the early development phase, Kancera has had a fluctuating price trend, probably partly attributable to a recurring need for financing. Larger price rises have mainly come in connection with progress in the development of the leading candidate KAND567. The stock benefited from great interest during the pandemic when this candidate was evaluated as a possible treatment for acute Covid-19. The proportion of foreign investors then clearly increased.

Kancera, share development (SEK)



Source: S&P Capital IQ and Carlsquare

Management, board, and ownership

Experienced organization

Kancera is a small organization, currently with seven employees, all with extensive experience in research, clinical development, or business development. The company's research activities were scaled back significantly in 2018 when Kancera chose to focus on clinical development. The new direction is underlined by the fact that the management has been strengthened in recent years with people responsible for clinical development and business development.

Management



Thomas Olin has been CEO and sat on the board since the company was founded in 2010. He has a Ph.D. in physiology and a M.Sc. in biology, chemistry, and geoscience. His background is in research and business development and management at Biovitrum. Thomas was responsible for the spin-off iNovacia from Biovitrum. He also has extensive experience in licensing of drug development projects. He owns 232,008 shares.



Peter Selin is Vice President, with responsibility for business development and partnerships, and took office in 2022. Peter has previously held various senior positions, working with business development and operational activities within Vivesto, Inceptua and SOBI. Peter owns 15,000 shares.



Martin Norin is the Chief Operating Officer since 2013. He has a Tekn.dr in bi-chemistry and a M.Sc. in chemistry from the Royal Institute of Technology in Stockholm. Among other things, he has been project manager and member of the committee for drug development in the preclinical phase at Pharmacia and Biovitrum. Together with Thomas Olin, he led the spin-off of iNovacia from Biovitrum in 2006. Martin owns 10,899 shares.



Torbjörn Lundström is since 2020 Chief Medical Officer. He is a doctor with cardiology and internal medicine as clinical specialties. Torbjörn Lundström most recently came from a position as Global Clinical Lead at AstraZeneca R&D. He has 20 years of experience in clinical development of products, mainly in the advanced development phase, within cardiovascular and metabolic diseases.

Source: Company information

The board

Several members have held positions on the board since the company was originally founded. The board is characterized by solid medical expertise in both cancer research and cardiology. There is a lack of a large external owner in the ownership picture and in the board. In general, this is something that can be seen as an advantage for other external owners and minority shareholders.

The board



Erik Nerpin is the company's chairman of the board and has been on the board since 2010. Nerpin is a lawyer and owner of Advokatfirman Nerpin. Nerpin's law practice is focused on stock market law, company law and corporate governance. He is also chairman of the board on Diamyd Medical AB and Blasieholmen Investment Group AB. He is also a board member on Niccocino Holding AB and Effnetplattformen AB. Nerpin owns 109,100 shares.



Håkan Mellstedt is Professor of oncological biotherapy at Karolinska Institutet since 1999. He is a MD. from Karolinska Institutet in Stockholm, honorary chairman of the board and member of Kancera's industrial council. Mellstedt has specialist competence in general internal medicine, hematology, and general oncology. He was during 1999–2010 director of the Karolinska Cancer Center at Karolinska University Hospital. Professor Mellstedt has published 375 scientific articles and 140 review articles. He owns 201,760 shares.



Charlotte Edenius has more than 20 years of experience from leading positions in the pharmaceutical industry, including positions such as Executive Vice President, R&D, at Medivir and Senior Vice President and Head of Research at Orexo. She owns 2,395 shares and has been on the board since 2016.



Professor **Carl-Henrik Heldin** was during 1986–2017 director of the Ludwig Institute for Cancer Research in Uppsala and since 1992 is a professor of molecular cell biology at Uppsala University. He is chairman of the board of the Nobel Foundation (since 2013), Science for Life Laboratory (since 2015) and the EMBL node Molecular Infection Biology (since 2009). He owns roughly 46,000 shares.

Source: Company information

The largest owners

Ownership is dispersed and Kancera has over 18,000 shareholders. The board and management together own 1.3 percent of the company. The absence of strong owners in a development company can be seen as a possible disadvantage. However, it is common for larger institutions or specialist investors to invest in biotech companies, through private placements, only after promising clinical results have been presented.

The ten largest owners

| Ägare | Share of capital | Share of votes | Verified |
|----------------------------|------------------|----------------|------------|
| Avanza Pension | 8.8% | 8.8% | 2022-09-28 |
| Sydbank A/S, W8IMY | 2.7% | 2.7% | 2022-09-30 |
| Nordnet Pensionsförsäkring | 1.9% | 1.9% | 2022-09-28 |
| Swedbank Försäkring | 1.1% | 1.1% | 2022-09-28 |
| Charlotte Rapp Hamrén | 0.8% | 0.8% | 2022-09-28 |
| Fredrik Rapp and family | 0.8% | 0.8% | 2022-09-28 |
| Susanne Rapp Nilsson | 0.8% | 0.8% | 2022-09-28 |
| JGM Invest AB | 0.7% | 0.7% | 2022-09-28 |
| Anders Tamsen | 0.7% | 0.7% | 2022-09-28 |
| Brunnby Rör Aktiebolag | 0.6% | 0.6% | 2022-09-28 |

Source: Holdings.se

Kancera is comparable with other pharmaceutical companies on First North in terms of liquidity and ownership. Looking at all pharmaceutical companies listed on First North, Kancera stands out with a relatively high liquidity. In terms of volume as a percentage of market capitalization, Kancera is on average 387 percent above the median when looking at all companies over the time horizons of 1 week, 1, 2, 3, 6 months and 1 year. At the three-year horizon, the company's turnover was 19 times higher than the median. This could be explained by the large proportion of foreign investors who bought in during the pandemic years 2020 to 2021. If you instead sort by oncology companies on the same stock exchange, the liquidity is not as impressive by comparison, but even here the company is on average 52 percent above Median.

This comparatively good liquidity could partly be explained by the large proportion of shares available to investors. Kancera ranks highest among comparable oncology companies when it comes to "free float" with a share of 99 percent, while the others average 74 percent.

Market and competition

Coronary artery bypass grafting (PCI) is the standard treatment for acute myocardial infarction, but the procedure is associated with increased damage when blood flow is restored. Antioxidants and anti-inflammatory treatments are being evaluated as countermeasures, but no drug is, yet, approved for this indication. Studies show that a high expression of fractalkine and fractalkine receptors is associated with worse outcomes in heart attacks. Something that provides a rationale for Kancera's candidates. Within ovarian cancer, a large percentage of patients are platinum-resistant and lack effective treatment. Antibody conjugates and new combinations of existing drugs are possible ways forward but probably not for all patients.

With the aim of reducing inflammatory damage of the heart

High mortality and damage frequency stemming from myocardial infarction

Cardiovascular diseases are the leading cause of death globally and myocardial infarction is an acute syndrome when calcifications (plaques) in one of the heart's coronary vessels rupture and the subsequent coagulation forms a clot that blocks said vessel. This in turn leads to heart muscle-cells being damaged as a result of the subsequent lack of oxygen. The damage becomes more severe the longer the blood flow to the heart muscle is obstructed. In Sweden alone, around 24,300 people suffer heart attacks annually (Source: The National Board of Health and Welfare, Year 2019). In the USA, there are an estimated 805,000 cases per year, of which undiagnosed silent heart attacks is estimated at around 170,000 cases. In the Western world, the incidence of heart attacks is decreasing. The National Board of Health and Welfare states that 5,200 died of acute heart attacks in Sweden, while the mortality in the USA was 104,300. We do not know the reason for the relatively worse outcome in Sweden, but an older population may be a contributing factor.

Acute coronary syndrome (ACS) is a collective name for coronary artery diseases such as unstable angina pectoris, ST-elevation myocardial infarction (STEMI, Q-wave infarction) and non-ST-elevation myocardial infarction (NSTEMI). ST elevation refers to an abnormal reading on the EKG. Approximately thirty percent of ACS are ST-elevation myocardial infarctions, which is the indication for which Kancera's KAND567 is currently being evaluated.

Symptoms of ischemia (lack of oxygen) are dull pains in the central chest that radiate outward to the left arm or other atypical pains in, for example, the abdomen. For diagnosis, ECG and (if possible) coronary X-rays are used.

The prognosis is highly dependent on which artery is blocked. In the majority of all deaths, the anterior descending branch (LAD) of the left coronary artery has been affected. Studies indicate that LAD, or more extensive localization of the heart attack, constitutes just over half of STEMI cases.

Patients with acute ST-elevation myocardial infarction must undergo PCI treatment (Percutaneous Coronary Intervention), i.e., coronary artery dilation with or without subsequent stenting. Stenting means that a metal mesh is inserted to keep the vessel open to prevent new structures in the same place. In the United States, 480,000 PCI procedures are performed annually. Based on these statistics, and a French study, we assume that PCI is performed in approximately 80 percent of cases. Alternative treatment in the form of thrombolysis is given primarily if the transport time to coronary care is more than two hours. The in-hospital mortality rate for STEMI was 6.4 percent, thus most deaths occur outside of hospitals.

Heart failure from myocardial infarction a difficult complication to treat

Heart attack treatment is complicated by the progression of the condition, where several factors increase the risk of heart failure. In case of coronary artery blockage, ischemic death in heart muscle cells begins within a few hours. Cell damage and cell death trigger release of pro-inflammatory cytokines, infiltration of neutrophils and mobilization of monocytes from the spleen. Opening the occluded coronary artery with coronary artery dilation does indeed improve recovery of the damaged heart muscle, but in the short term leads to an explosion of oxidative stress and further cell death. Even after reperfusion, blockage remains in the form of microvascular obstruction (MVO), caused by thrombosis, plaque debris and endothelial damage, in up to 50 percent of cases. In subsequent days, inflammation drives further infarct expansion. Cahill (2017)

Repair of the damaged left ventricle is characterized by the activation of fibroblasts to myofibroblasts, which deposit collagen matrix that in turn leads to scar formation. Remodeling of the damaged left ventricle then occurs over weeks to months, but cardiac function remains impaired during this time, and therefore systems that maintain blood pressure through vasoconstriction and fluid retention are activated. These mechanisms are initially compensatory but over time become harmful to the heart muscle and heart function can be further adversely affected.

Anti-inflammatory treatments have not made it all the way

The market for drugs used in the treatment of cardiovascular diseases is dominated by blood-pressure-lowering, blood-thinning and blood-lipid-lowering drugs. Inflammatory processes are considered to play a part in accelerating the course of the disease, and anti-inflammatory treatments have therefore been proposed as complementary therapies to, for example, blood thinners and statins (cholesterol-lowering agents). No treatments of this type have yet reached the market. The results of the large CANTOS study, in which the antibody treatment canakinumab was given as a treatment to prevent recurrent heart attacks, gave some hope and fueled new similar efforts. Novartis applied for approval in 2018 for canakinumab, but it was denied by the FDA. Probably at least one cause for the fumble was that the effect was not strong enough.

Antioxidant the clear competitor in the pipeline

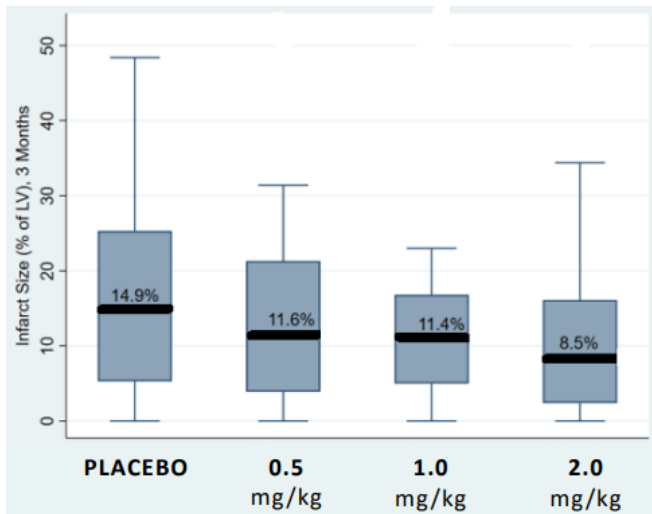
Blood thinning treatment is given for up to twelve months after coronary artery dilation. However, to our knowledge, there is still no approved drug specifically indicated for post-PCI myocardial infarction that targets the prevention of damage caused by reperfusion or inflammation.

As we previously mentioned, anti-inflammatory drugs approved for other indications, such as anakinra and tocilizumab, have been evaluated in clinical studies for the treatment of STEMI. However, there have been investigator-initiated studies and it is uncertain whether further development will be carried out. The Swedish company Athera Biotechnologies conducted a phase II study in heart attack patients with elevated ST with the antibody ATH3G10, which binds to oxidized phospholipids. The purpose was an anti-inflammatory action. We cannot find any detailed results from the above-mentioned study, but the company states that development has been stopped with reference to the fact that the treatment gave rise to unexpectedly high immunogenicity.

FDY-5301 is a formulation of sodium iodide that aims to reduce reactive oxygen radicals in order to reduce skeletal and heart damage in myocardial infarction. In a phase II study (n=120), a positive trend in effect was observed where infarct size was lower and cardiac function was better compared to the control group (placebo) three months after treatment. However, it should be noted that there was a comparatively low proportion of patients (34 percent) with myocardial infarction localized to the LAD in the sample, as previously mentioned a significant

risk factor. Recently, the developer Faraday Pharmaceuticals has started a phase III study with the goal of recruiting 2,300 patients.

The antiradical FDY-5301 reduces infarct size vs placebo



Source: Adlam, A. et al, "A Randomized, Double-Blind, Dose Ranging Clinical Trial Of Intravenous FDY-5301 In Acute STEMI Patients Undergoing Primary PCI", Presentation AHA December 2019

Below we have compiled the competitive landscape. We have also included fractalkine blockers that have not been specifically investigated in myocardial infarction.

Overview of selection of competitor projects and results in myocardial infarction

| Project | Phase | Mechanism of action | Dosing | Safety | Survival | Infarction | Function |
|-------------|------------|---|-----------------------------|-----------------------|----------------|---------------|---------------|
| KAND567 | II | Fractalkine receptor block | 134 mg + 600 mg/qd 3d | <1000 mg/day | Ongoing study | Ongoing study | Ongoing study |
| FDY-5301 | III | Anti-peroxidant Fractalkine receptor block (nanobody) | 1-2 mg/kg single dose | Similar to control | No information | Pos. trend | Pos. trend |
| BI-655088 | I | Fractalkine receptor block (nanobody) | Unknown | Unknown | Not tested | Not tested | Not tested |
| E6011 | II | Fractalkine receptor (mAB) | 100-400 mg every other week | Generally tolerable | Not tested | Not tested | Not tested |
| ATH3G10 | Terminated | Anti-oxPL (mAB) | Unknown | Immunogenicity | Unknown | Unknown | Unknown |
| Tocilizumab | II | IL-6 antibody | 280 mg single dose | Increased cholesterol | Not measurable | Significant | No |
| Anakinra | II | IL-1R antagonist | 100-200 mg/qd 2w | Well tolerated | Significant | Not tested | No |

Source: Company information

Among the other fractalkine blockers, BI 655088, a nano-antibody that blocks the fractalkine receptor, is being developed by Ablynx and Boehringer Ingelheim (B-I). Preclinical results showed a plaque-reducing effect in a mouse model of atherosclerosis. The candidate has been evaluated in a phase I study. We cannot trace any published results and the project no longer appears to be in B-I's official pipeline.

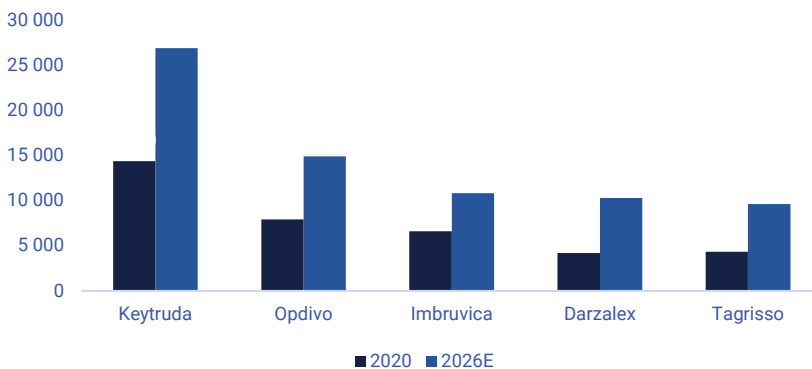
The antibody treatment E6011 (being developed by Eisai) which directly targets fractalkine is being investigated in autoimmune diseases. Early clinical studies have shown promising efficacy in patients with rheumatoid arthritis who do not respond to standard treatment. The treatment was generally tolerable, which could also be interpreted as positive for KAND567.

Remedies for treatment resistance are sought

Small molecule cancer treatments are proving themselves well

It is no exaggeration to claim that immuno-oncology has been responsible for the most important gene breakthrough for cancer treatment, and perhaps even for the pharmaceutical field at large, in the last decade. Sales of immuno-oncology drugs are growing at an average rate of 20 percent per year and are expected to reach \$100 billion by 2026 (Source: Evaluate Pharma). There has been an increased focus on (mainly antibody-based) biological treatments in oncology. According to Informa, almost 4,700 clinical trials in immuno-oncology were ongoing in July 2021.

The best-selling cancer treatments 2020E (million USD)



Source: Evaluate Pharma World Preview 2021

Nevertheless, small molecule treatments are seeing great success. A very important group are kinase inhibitors, which target protein kinases. These are catalysts for phosphorylation, i.e., reactions that activate e.g., enzymes. Protein kinases are often overactivated in cancers. There are around 60 approved cancer treatments that are based on kinase inhibitors. Examples of drugs are Imbruvica (ibrutinib) in blood cancer and Ibrance (palbociclib) in breast cancer.

One of the main advantages of antibodies over small molecules is the high probability of success in developing selective drugs against biological targets of interest. This limits, among other things, the risk of unwanted effects "off the target". However, antibodies also have important limitations as infusion is less practical than oral intake of small molecules. More importantly, immune-related adverse events (irAEs) have occurred in more than 57 percent of patients treated with checkpoint inhibitor (CTLA4 or PD1/PDL1) antibodies. Severe side effects of grade 3 or higher have been documented in between 10 and 27 percent of those treated. The long half-life makes it difficult to avoid irAEs from antibodies by fine-tuning the dosage, as it can take 1–2 months after the end of dosing before the symptoms disappear. Premedication with corticosteroids and other immunomodulatory agents to manage toxic effects may contribute to additional complications. Short half-lives enable a more precise calibration of treatments based on small molecules (Source: Offringa, R., et al "The expanding role for small molecules in immuno-oncology", Nature Reviews Drug Discovery, 2022). Unlike conventional antibodies, small molecule treatments can also be directed against targets inside cells.

Kancera's candidates in cancer, in spite of being small molecules, are still difficult to characterize. As mentioned, they are chemokine receptor inhibitors and as far as we know, there is only one small molecule drug of this type that is approved in cancer (Plerixafor).

Drug resistance a challenge in ovarian cancer

According to the National Cancer Institute, around 20,000 new cases of ovarian cancer are diagnosed every year in the United States. Globally the incidence is over 300,000 (Globocan). Unfortunately, this form of cancer is difficult to detect in the early stages and in about 60 percent of the cases, the cancer has already spread to other parts of the body such as the liver or lungs when diagnosed. Surgery is standard treatment even for patients with cancer that has spread to reduce the tumor burden. A large percentage of patients receive platinum-based chemotherapy. Bevacizumab (Avastin) is a so-called targeted antibody treatment that inhibits the ability of tumors to form blood vessels and is a common addition to chemotherapy.

Depending on treatment response, the cancer is usually divided into a platinum-sensitive (recurrence more than six months after treatment at the earliest), platinum-resistant (recurrence within six months) or platinum-refractory (no response to treatment) disease. The prognosis is worse for platinum-resistant/refractory cancer compared to platinum-sensitive, with about half the median survival rate using standard treatment (16 months compared to 33).

PARP-inhibitors driving the market

The main innovation in the field from the last ten years is PARP inhibitors. It is a class of cancer treatment drugs that inhibit enzymes of the poly-ADP-ribose polymerase (PARP) family. These enzymes have a role in repairing DNA. In particular, tumors with BRCA 1 and 2 mutations depend on PARP to repair DNA and avoid cell death. BRCA 1 and 2 ("breast cancer gene 1 and 2") are indeed dreaded mutations as they are associated with a significantly increased risk of developing cancer. However, tumors with these mutations become vulnerable to PARP inhibitors. PARP inhibitors are designed to enter the cell and compete for the coenzyme NAD⁺, after which PARP activity stops and repair no longer occurs.

BRCA 1 and 2 mutations have been considered rare and only account for 10 to 15 percent of all breast and ovarian cancer cases if genetic mutations are counted. However, it has been shown that somatic mutations are more common and that PARP inhibitors are also effective in a larger patient group.

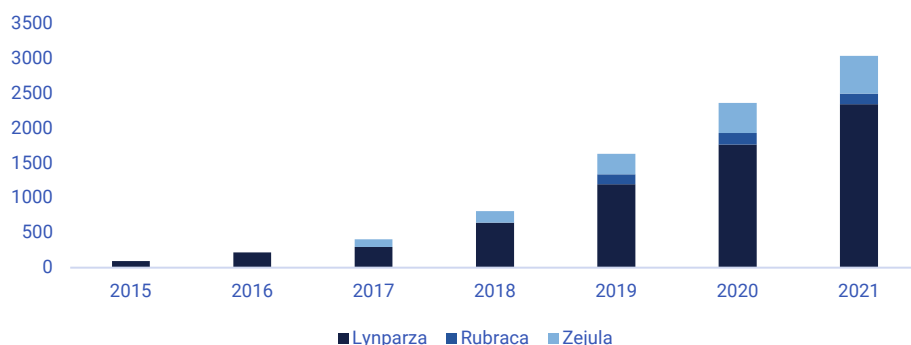
The best-selling PARP inhibitor olaparib (Lynparza) is given as maintenance treatment after chemotherapy to platinum-sensitive patients with a BRCA mutation in first line and second-line treatment (also platinum-sensitive after chemotherapy), regardless of mutation. Niraparib (Zejula) has an even broader indication in the first line as maintenance treatment regardless of mutation.

A large patient group lacking effective treatments

The PARP inhibitors have resulted in a clearly improved progression-free survival in particular for patients with a BRCA mutation. The effect on overall survival is unclear, but it is also a difficult measure as it is affected by later-line treatments. However, the medical need for more effective treatments for platinum-resistant cancer, where PARP inhibitors are not indicated, is still great.

The market for PARP inhibitors is around US\$3 billion (2021). In addition to ovarian cancer, this type of treatment is also indicated for adjuvant (BRCA+/HER2-) and advanced breast cancer, and prostate cancer.

Sales of PARP inhibitors (million USD)



Source: Company information

Immunotherapies of the checkpoint inhibitor type, which have been so successful in, for example, melanoma, lung cancer and kidney cancer, have not enjoyed any success against ovarian cancer. A phase III trial showed no benefit from adding atezolizumab (Tecentriq) to standard first-line therapy.

Antibody conjugates on the cusp of approval

Examples of new treatments in the pipeline are Mirvetuximab Soravtansine, an antibody drug conjugate directed against folate receptor alpha (FR α), which is often expressed in ovarian cancer. A cytotoxic compound (DM4) is linked to the antibody. The developer ImmunoGen has applied for approval under an accelerated approval procedure based on a relatively small (n=106), non-randomized trial as a monotherapy for patients who have received up to three lines of treatment and are platinum resistant. The primary endpoint was objective response, and, in the study, 32 percent was achieved. The PDUFA date is set for November 28, 2022.

Mirvetuximab is also being investigated as an addition for combination treatment in other treatment lines. In order to be eligible for treatment, it will likely be required that tumor biopsies from the patient test positive for a high expression of FR α . Around 40 percent are FR α -positive. A special circumstance is that mirvetuximab has already completed a phase III study where the primary endpoint – improved progression-free survival compared to chemotherapy – was not achieved. However, secondary endpoints were positive and, according to the investigators, the outcome was probably affected by a lack of screening methods and an incorrect selection of patients.

Generally speaking, there are good opportunities for accelerated approval in oncology as a treatment for advanced cancer for patients with limited treatment options. The catch is that this procedure most likely requires KAND145 to demonstrate clinically relevant efficacy as a monotherapy. This is difficult to assess seeing as preclinical trials indicate that the best effect is achieved with combination treatment with platinum-based cytotoxics. However, the Phase Ib study may provide important guidance on possibilities as monotherapy.

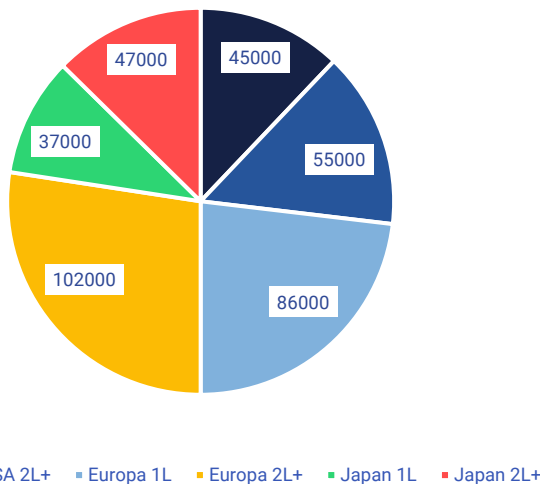
Rectal cancer

Colorectal cancer is one of the most common forms of cancer. In the United States, 151,000 new cases are diagnosed each year. Chemotherapy treatments such as FOLFOX or FOLFIRI are most often used as adjuvant treatment after surgery or in advanced cancer, but target-directed treatments such as the angiogenesis inhibitor bevacizumab are also often used in combination therapies.

As mentioned above, Kancera states that the company sees an opportunity for PFKFB3 in combination with radiation therapy against rectal cancer. Radiation therapy is used before surgery (neoadjuvant) to facilitate surgery and sometimes after surgery to prevent recurrence. Radiation therapy is more common for rectal cancer than colon cancer. Rectal cancer makes up approximately 30 percent of colorectal cancer. Based on statistics from the Swedish Colorectal Cancer Registry, we estimate that a number of patients corresponding to 30 percent of all cancer cases (incidence) receive preoperative radiotherapy. A combination treatment with neoadjuvant radiation therapy in rectal cancer could therefore roughly address up to approximately ten percent of all colorectal cancer patients.

It is probably challenging to incorporate a new drug treatment into the neoadjuvant cancer guidelines. Fairly large and long studies will likely be required to demonstrate a possible survival benefit in this patient group, which has a significantly better outlook than cases with cancer that has metastasized. An alternative is to pursue development in more advanced cancer. The market for advanced colorectal cancer is admittedly more competitive, but comparatively large and with a shorter development path. This requires a different approach, for example in combination with chemotherapy or targeted drugs.

Market for advanced (stage IV) colorectal cancer (drug-treated patients)



Source: Isofol Medical/Global Data, Carlsquare

Business development

Kancera's business model is based on the sale or out-licensing of pharmaceutical projects to the international pharmaceutical industry. It is however difficult to track down examples of licensing deals relevant to the myocardial infarction indication. Our limited sample suggests a reference value of up to around 200 MUSD.

Selection of licensing deals in the cardiovascular space

| Licensor | Partner | Project | Phase | Value (MUSD) | Upfront (MUSD) | Royalties | Date |
|---------------|-----------|--------------------------------|-------|--------------|----------------|--------------|--------|
| Idorsia | Janssen | Endothelin receptor antagonist | II | 230 | 230 | 20-35% | Dec-17 |
| Inotek | Genentech | PARP-inhibitor/INO-1001 | I | 200 | 0 | Yes, unknown | Jul-06 |
| Median | | | | 215 | 115 | | |

Source: Company information.

In the cancer space we believe that it is reasonable to use PARP inhibitors as a reference. Like Kancera's fractalkine inhibitors, they are small molecules that affect signaling pathways for DNA repair. PARP inhibitors have had their

breakthrough in ovarian cancer and breast cancer. In addition, the PARP-1 enzyme is also considered to have a role in worsening the course of, for example, strokes and heart attacks. It may be noted that the PARP inhibitor talazoparib has been evaluated as myocardial infarction treatment for STEMI patients who have undergone vasodilatation.

In 2006, Inotek licensed its PARP inhibitor to Genentech in a licensing deal worth up to USD 625m. Of this, USD 200m was related to possible milestones from development in the cardiovascular area.

Selection of licensing deals, PARP-inhibitors

| Licensor | Partner | Project | Phase | Value (MUSD) | Upfront (MUSD) | Royalties | Date |
|------------------|------------|-------------------------|-------|--------------|----------------|-------------------|--------|
| Nerviano Medical | Merck KgaA | PARP1-inhibitor | I | missing | 65 | Not specified | Sep-22 |
| Tesaro | Janssen | PARP-inhibitor | III | 500 | 35 | Not specified | Apr-16 |
| BioMarin | Medivation | talazoparib | III | 570 | 410 | Mid-Single digits | Aug-15 |
| Inotek | Genentech | PARP-inhibitor/INO-1001 | I | 425 | 20 | Not specified | Jul-06 |
| Median | | | | 500 | 50 | | |

Source: Company information.

Our compilation shows that the median value of a deal for PARP inhibitors in the cancer space is 500 million USD.

Kancera's preclinical portfolio consists of three projects that all have potential applications as cancer treatments. Our research provides an indicated median value for licensing deals involving projects in preclinical development in oncology of up to \$300M (based on ~130 deals).

Sector colleagues and companies in the reference group

Listed industry colleagues

Cardiovascular diseases and cancers are very large areas in drug development. For smaller biotech companies, the path to success may be to focus on sub-markets/indications with little competition and the possibility of rapid development paths.

Active Biotech

Active Biotech develops immunomodulating and immuno-oncology treatments for cancer and autoimmune diseases. The company's partner NeoTx is conducting phase II clinical development with naptumomab.

Cantargia

Like Kancera, Cantargia develops treatments based on the blockage of inflammatory signals. The company is developing antibodies against a coreceptor for IL-1 cytokines. Cantargia is developing a broad program in cancer and autoimmune diseases (including myocarditis) with phase II clinical trials ongoing.

Faron Pharmaceuticals

Faron Pharmaceuticals has two drug candidates undergoing clinical trials. Bexmarilimab is currently undergoing three phase I/II clinical trials for solid tumors as well as hematologic malignancies. The other candidate, traumakine (interferon beta), failed in a phase III study for the ARDS indication but is currently undergoing new preclinical trials.

Medivir

Medivir develops cancer treatments based on prodrugs of chemotherapy agents, HDAC inhibitors and IAP blockers. MIV-818 is being studied in phase Ib as a

possible combination treatment in liver cancer. Medivir has license agreements with, among others, the American IGM Biosciences.

Modus Therapeutics

Modus is developing a polysaccharide-based treatment against sepsis. The mechanism of action is based on an anti-inflammatory effect. A phase Ib study is currently being conducted in healthy volunteers.

Quantum Genomics

A French company that develops treatments for hypertension and heart failure based on new RAS blockers. Quantum is currently conducting phase III clinical development.

Valuations, reference group

The reference group above is a heterogeneous group where we have taken indication, mechanism of action and clinical development into account in order to find reasonably relevant objects of comparison. The valuation is clearly linked to the development phase in combination with external validation in the form of partner collaborations and ownership structure. The comparison indicates that Kancera is valued lower than similar companies. The valuation is likely to be pressured, at least in the short term, by the recently announced rights issue.

Valuations, reference group

| Company | EV (SEKm) | Phase |
|--------------------------------------|------------|-------|
| Medivir AB (publ) | 306 | 1 |
| Modus Therapeutics Holding AB (publ) | 23 | 1 |
| Cantargia AB (publ) | 276 | 2 |
| Active Biotech AB (publ) | 248 | 2 |
| Faron Pharmaceuticals Oy | 1 292 | 2 |
| Quantum Genomics Société Anonyme | 1 224 | 3 |
| Median | 291 | |
| Kancera* | 127 | |

Source: S&P Capital IQ

Forecasts

In recent years, Kancera has reduced fixed costs and directed more and more resources to clinical development. We see a good chance for revenue-generating partner collaborations from primarily the fractalkine project, but also the preclinical portfolio. Relatively limited competition speaks for high market shares if Kancera's candidates make it all the way. We see the greatest revenue potential in oncology, of which roughly USD 500 million is in ovarian cancer.

Revenue and profitability forecasts

Expanded clinical program

The focus of the operational activities is completing the ongoing phase IIa "FRACTAL" study in myocardial infarction and preparations for the first study in cancer patients including the application for approval.

Kancera indicated in the prospectus from 2021 a cost of just under SEK 60 million to reach important milestones in the development of a cancer treatment based on fractalkine inhibitors: 1) Conduct a phase Ib study up to and including dose escalation with KAND567 in combination with carboplatin in patients with platinum-resistant ovarian cancer 2) develop KAND145 through a phase Ia study.

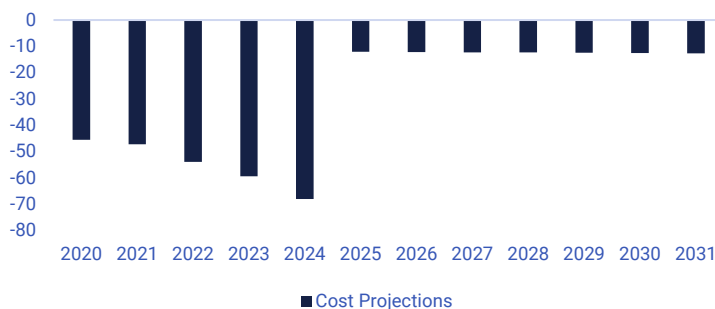
The recently announced rights issue will also finance further steps in the form of a phase IIa study. In addition, around 30 percent of the net cash of approximately SEK 76 million is earmarked for the manufacture of pharmaceutical products KAND145 and KAND567.

Furthermore, we expect that Kancera will start a phase IIb study in myocardial infarction in order to further evaluate effect using data from phase IIa to calibrate effect measures.

At the same time, we have also accounted for preclinical development to make PFKFB3 ready for a partnership.

Given that one or more of the planned clinical studies are successful, we see that both the fractalkine projects and a preclinical project (we assume PFKFB3) can be outlicensed in a few years' time. In our base scenario, we expect it to occur between 2024 and 2025. We assume that collaboration partners will then take over costs for further clinical development and commercialization.

Forecast of costs (SEKm) risk-adjusted



Source: Company information and Carlsquare estimates

Project assumptions

Fractalkine inhibitors in myocardial infarction

According to statistics from the American Heart Association, approximately 635,000 heart attacks are diagnosed annually in the United States. Statistics from the European Society of Cardiology indicate an incidence of over 900,000 in the EU and Great Britain. If we assume that ST-elevated myocardial infarction makes up 30 percent (Source: Swedeheart Annual Report 2021), this means around 460,000 cases in total for these geographical areas.

We further assume that on average 80 percent receive PCI treatment. The proportion varies however and is lower for patients over 80 but higher among younger patients. In the FRACTAL study, patients with infarction in the anterior descending branch of the left coronary artery (LAD) are included and we assume that this will be the indication even after market approval. We estimate that this is 50 percent of the population. With these assumptions, a fractalkine inhibitor would address a population corresponding to 12 percent of all heart attack cases.

An assumption about penetration or market share at this early stage is fraught with great uncertainty. FDY-5301 is a competitor that has a head start in clinical development and, it seems, easier administration with a single dose. In the absence of comparable data, we assume a "duopoly" and a 50 percent penetration in the US, EU, and UK.

Kancera has commissioned analysis using a pricing point between \$2,000 and \$9,000 per three-day treatment. Since there is no approved drug treatment for damage caused by heart attack, there is uncertainty as to how KAND567 could be priced. A number of anti-inflammatory treatments that are approved in other indications have also been tested in heart attack patients, for example anakinra and tocilizumab. In clinical studies, anakinra has been administered with high doses, which indirectly speaks for a very high cost/pricing, while in return relatively low doses have been used for, for example, tocilizumab, which in turn speaks for a significantly lower pricing point. After completed coronary artery dilation, patients are treated with blood-thinning treatments such as ticagrelor (a so-called P2Y12 receptor antagonist) for up to 12 months. Based on list prices in the USA, the cost of an annual treatment is approximately USD 5,500. Given a smaller target population, we use Kancera's market research as a benchmark for KAND567 and assume a net income of USD 8,000 in the US and USD 4,000 in Europe.

Assumptions regarding peak sales for myocardial infarctions

| 2038P | USA | Europe | Total |
|------------------------------|--------|--------|---------|
| Myocardial infarction | 690000 | 910000 | 1600000 |
| STEMI-myocardial infarctions | 30% | 30% | |
| Treated | 85% | 85% | |
| PCI-intervention | 80% | 80% | |
| LAD-localization | 50% | 50% | |
| Kancera share | 50% | 50% | |
| Kancera treated | 35190 | 46410 | 81600 |
| Net income per treated, USD | 8000 | 4000 | |
| Revenue, MUSD | 282 | 186 | 467 |

Source: Carlsquare

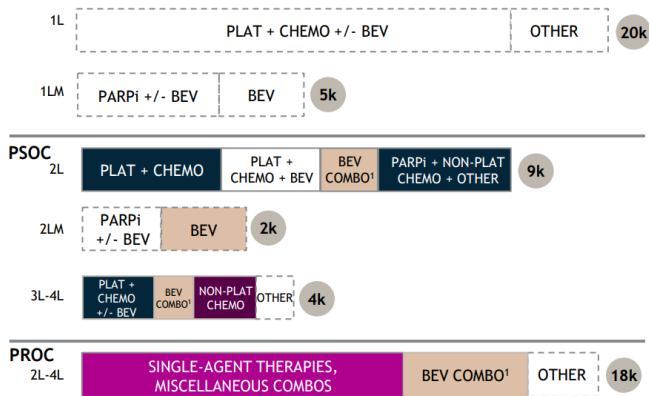
All in all, we have calculated possible peak sales amounting to 470 MUSD.

Fractalkine inhibitors for advanced ovarian cancer

As we described above, Kancera primarily aims to develop a fractalkine inhibitor for advanced platinum-resistant ovarian cancer. In a phase Ib study, KAND567 will be combined with carboplatin.

In the USA, the first-line drug market amounts to around 20,000 treated patients per year (Source: Roche, ImmunoGen). The number of treated patients with resistant cancer in later lines is estimated to be somewhat smaller.

US market for advanced ovarian cancer

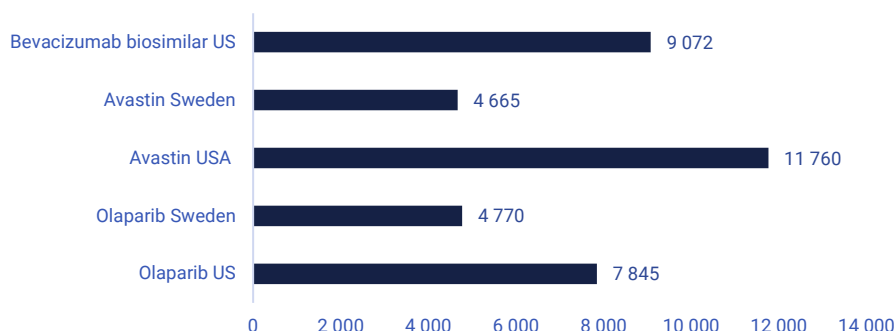


Source: ImmunoGen. PSOC: Platinum-sensitive ovarian cancer. PROC: Platinum-resistant/refractory ovarian cancer. xL: Treatment in line x. xLM: Maintenance treatment. PLAT: Platinum-based chemotherapy. BEV: Bevacizumab. PARPi: PARP inhibitor.

At this early stage, it is difficult to assess what the competitive landscape will look like. Simplistically, we assume that mirvetuximab or bevacizumab will be the main competitors. There is however a theoretical potential to also combine with, for example, bevacizumab, even if it is limited by the fact that the antibody treatment is already used in earlier lines. The PARP inhibitors on the other hand are primarily maintenance treatments and we therefore regard them as complementary. We consider that an optimistic but realistic assessment is approximately 30 percent penetration in resistant/refractory patients in the second line and later treatment of advanced ovarian cancer. We assume that this corresponds to approximately ten percent of the number of patients with advanced ovarian cancer who receive drug treatment.

For pricing we have used other targeted treatments in ovarian cancer for comparison, which consists of PARP inhibitors and bevacizumab. We estimate list prices for these drugs to be around USD 9,000 per month in the USA and a little over half that in Sweden. We make the simplified assumption that treatment is given for six months (which roughly corresponds to progression-free survival when treated with bevacizumab in combination with chemotherapy for platinum-resistant cancer).

Estimated cost per month of ovarian cancer drugs, (USD), list prices



Source: Company information, TLV, Carlsquare estimates

Overall, we assume peak sales of \$500 to \$600 million in ovarian cancer.

Assumptions regarding peak sales for ovarian cancer

| 2041P | USA | EU+UK+JP | Total |
|---------------------------------------|------------|------------|------------|
| Advanced ovarian cancer, incidence | 23409 | 42240 | |
| Platinum resistant, treated | 20065 | 36206 | |
| Kancera share | 30% | 30% | |
| Kancera treated | 6019 | 10862 | 16881 |
| Net revenue per treated patient (USD) | 48 000 | 24 000 | |
| Revenues (MUSD) | 289 | 261 | 550 |

Source: SEER, Roche, ImmunoGen, Carlsquare estimates

In the light of new preclinical results in models for rectal cancer, Kancera has also resurrected the PFKFB3 project. Below we present assumptions for calculating possible peak sales. A disadvantage is that a large part of the patent period for the original substance patents (which expire around 2032) is in risk of being used up when a possible future launch approaches. New patent applications, for example based on new formulations, may therefore be required to convince potential collaboration partners. We expect similar pricing as for the fractalkine inhibitors. However, we expect a short treatment period of two months before surgery, which reduces the net income per treated patient.

Peak sales assumptions, rectal cancer

| 2036P | USA | EU+UK+JP | Total |
|--------------------------------|------------|------------|------------|
| Neoadjuvant rectal cancer | 48119 | 86085 | |
| Surgery | 60% | 60% | |
| Preoperative radiation therapy | 54% | 54% | |
| Kancera share | 49% | 49% | |
| Kancera treated | 7600 | 13597 | 21197 |
| Price per treatment | 16 000 | 8 000 | |
| Revenues (MUSD) | 122 | 109 | 230 |

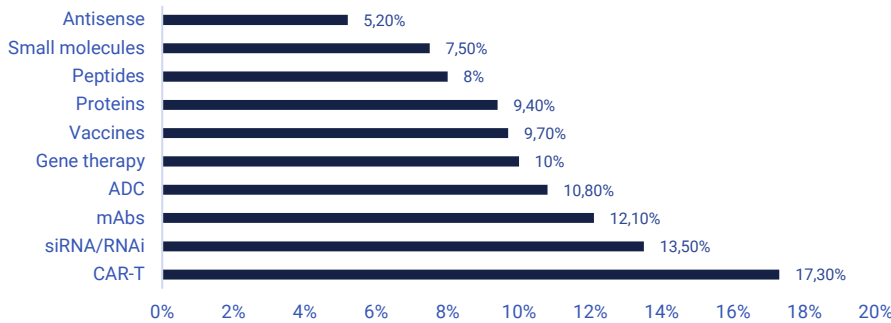
Source: Swedish colorectal cancer registry, Carlsquare estimates

Probability of success

Based on statistics on drug development, the probability of reaching the market is estimated to be 7.9 percent for an average drug project in phase I. For oncology, the corresponding probability is 5.4 percent ("Clinical Development Success Rates and Contributing Factors 2011–2020", Bio/ Informa Pharma/QLS). Even for

cardiovascular diseases, the window is narrow at 4.8 percent. Factors that influence the possibilities are indication, target molecule and modality.

Likelihood of approval from phase I, per modality



Source: Bio/Informa Pharma/QLS

We have used historical probabilities for each indication. We have adjusted the probability for KAND145 with regards to completed clinical studies with KAND567, which we believe justifies an assumption of a higher probability compared to other projects in the preclinical phase.

Assumptions for likelihood of approval

| Project | Indication | Precl. | Phase I | Phase II | Phase III | NDA | LOA |
|---------|-----------------------|--------|---------|----------|-----------|-----|-----|
| KAND567 | Myocardial infarction | 100% | 100% | 21% | 55% | 83% | 10% |
| PFKFB3 | Oncology | 50% | 50% | 25% | 50% | 92% | 3% |
| KAND145 | Oncology | 80% | 75% | 25% | 50% | 92% | 7% |

Source: Bio/Informa Pharma/QLS, Carlsquare LOA: Likelihood of approval

Our forecasts are predicated on licensing starting in 2024

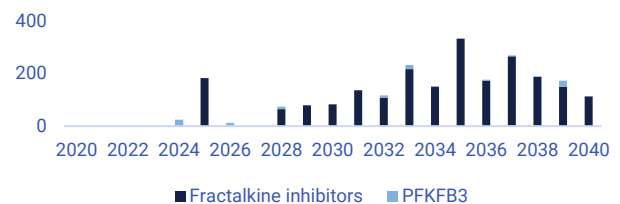
For our revenue assumptions, we assume that both PFKFB3 and the fractalkine project will be licensed after preclinical development and first proof of concept in humans, which we estimate will take place in 2024 and 2025 respectively.

Forecast, royalty income (SEKm)



Source: Company information and Carlsquare estimates

Forecast, net revenues, risk adjusted (SEKm)



Source: Company information and Carlsquare estimates

We have assumed licensing deals worth up to a total of \$700 million for the fractalkine project and \$250 million for PFKFB3. Since KAND567 and KAND145 are similar, we see it as logical that both projects are sequentially licensed out at the same time as we assume that a licensee would reasonably prefer control over all of Kancera's fractalkine inhibitors. However, that scenario is not decisive for our valuation. We expect an upfront payment corresponding to two percent of the possible total value for PFKFB3 and up to 50 MUSD for the fractalkine inhibitors. We have assumed varying royalty rates of five to 25 percent, see below. Our assumptions are based on the (admittedly limited) selection of license deals that we compiled above.

Valuation

Expanded clinical program gives upside

Large range of valuation depending on study results

Our valuation in a base case is predicated on the sales assumptions we described in the forecast section above. We have used a risk adjusted DCF valuation, as shown below. The risk adjustment is based on the development risks that we have mentioned, where we expect probabilities of reaching the market between three and ten percent with the largest risk adjustment for PFKFB3. In our model, we have used a discount rate of 14.3 percent. This is based on a risk-free interest rate of 1.9 percent, a beta value of 1.2 and a risk premium of 10.3 percent. The latter is based on PwC's Risk Premium Study 2021 and consists of a market risk premium of 6.7 percent and a size-based component of 3.6 percent.

We calculate an operating value of approximately SEK 290 million. Our valuation is based on Kancera being able to find partners for both the fractalkine inhibitors and PFKFB3 after successful development. To get there, we believe that financing is required, which the company addresses through the recently announced rights issue. We have therefore expected full dilution from the rights issue including warrants. All in all, the risk-adjusted justified value, also adjusted for dilution, is just over four SEK per share in our base scenario

Sum-of-the-parts valuation, base case, SEKm

| Project | Indication | LOA*, % | Royalty, % | Peak Sales, USDm | Launch | rNPV, SEKm |
|---|----------------|---------|------------|------------------|--------|------------|
| KAND567 | STEMI | 10% | 25% | 470 | 2029 | 227 |
| KAND145 | Ovarial cancer | 7% | 20% | 550 | 2031 | 163 |
| Other | Rectal cancer | 3% | 5% | 230 | 2033 | 37 |
| Technology value before taxes | | | | | | 436 |
| Overhead and taxes | | | | | | -141 |
| EV | | | | | | 287 |
| Net cash (22'Q3E) | | | | | | 67 |
| Fair value | | | | | | 363 |
| # shares | | | | | | 56.1 |
| Per share, SEK | | | | | | 6.3 |
| Expected financing | | | | | | 121 |
| Equity value after financing activities | | | | | | 476 |
| Number of shares after financial activities | | | | | | 116.0 |
| Fair value per share, SEK | | | | | | 4.1 |

Source: Carlsquare *LOA: Likelihood of approval

Valuation range

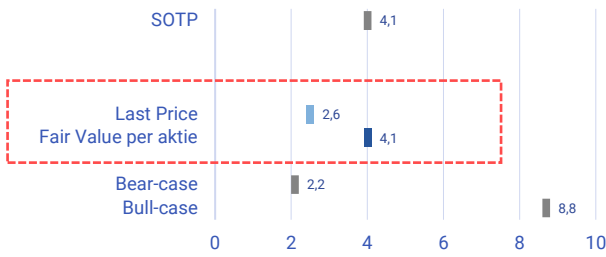
In an **optimistic Bull scenario**, we expect:

- Phase IIa in myocardial infarctions is completed with promising results and we raise the LOA to 27 percent from ten percent.
- Clinical studies in cancer with fractalkine inhibitors begin as planned

We calculate a justified operating value of approximately one billion Swedish crowns or around nine SEK per share after dilution.

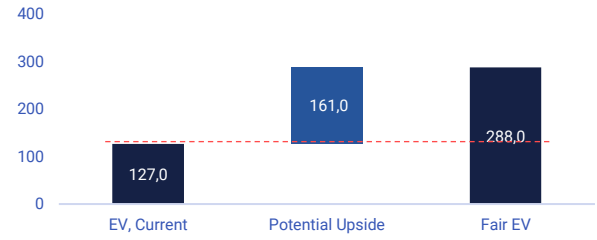
In a **cautious Bear** scenario, we expect the results in myocardial infarction to be disappointing and accordingly set the value to zero for this indication. We assume that clinical studies in cancer begin according to plan. We calculate a value of just over two SEK per share after dilution.

Fair value within a range, SEK



Source: Carlsquare forecasts

Visualization of market value, base scenario (SEK million)



Source: Carlsquare forecasts

Key figures and accounts

Income statement (SEKm), quarterly

| | Q1, 21 | Q2, 21 | Q3, 21 | Q4, 21 | Q1, 22 | Q2, 22 |
|---------------------|--------------|--------------|--------------|--------------|--------------|--------------|
| Net sales | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Total revenue | 0.6 | -0.3 | 0.4 | 1.1 | 0.3 | 0.2 |
| Gross profit | 0.6 | -0.3 | 0.4 | 1.1 | 0.3 | 0.2 |
| OPEX | -9.6 | -12.4 | -11.3 | -13.9 | -12.2 | -15.6 |
| EBIT | -9.0 | -12.6 | -10.9 | -12.7 | -11.9 | -15.4 |
| EBITDA | -8.9 | -12.6 | -10.8 | -12.7 | -11.8 | -15.3 |
| EBT | -9.1 | -12.7 | -11.0 | -13.0 | -12.0 | -15.6 |
| EPS (SEK) | -0.20 | -0.25 | -0.20 | -0.23 | -0.21 | -0.28 |

Source: Company information and Carlsquare estimates

Income statement (SEKm)

| | 2020A | 2021A | 2022E | 2023E | 2024E | 2025E |
|-----------------------|--------------|--------------|--------------|--------------|--------------|--------------|
| Net sales | 2.7 | 0.0 | 0.0 | 0.0 | 25.0 | 183.0 |
| Other revenue | 2.7 | 1.9 | 0.5 | 0.0 | 0.0 | 0.0 |
| Totala revenue | 5.4 | 1.9 | 0.5 | 0.0 | 25.0 | 183.0 |
| COGS | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Gross profit | 5.4 | 1.9 | 0.5 | 0.0 | 25.0 | 183.0 |
| Adj. Gross profit | 2.7 | 0.0 | 0.0 | 0.0 | 25.0 | 183.0 |
| R&D | -39.3 | -43.1 | -50.0 | -55.4 | -63.9 | -8.0 |
| SG&A | -6.2 | -4.0 | -3.8 | -3.9 | -4.0 | -4.1 |
| Dep. and amort. | -1.7 | -0.3 | -0.2 | -0.1 | -0.5 | -0.6 |
| Other costs | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Total operating costs | -45.5 | -47.1 | -53.9 | -59.3 | -67.9 | -12.1 |
| EBIT | -40.1 | -45.3 | -53.4 | -59.3 | -42.9 | 170.9 |
| EBITDA | -38.4 | -44.9 | -53.1 | -59.2 | -42.5 | 171.5 |
| Financial net | -0.3 | -0.4 | -0.3 | 0.0 | 0.0 | 0.0 |
| EBT | -40.5 | -45.7 | -53.7 | -59.3 | -42.9 | 170.9 |
| Taxes | 0.0 | 0.0 | 0.0 | 0.0 | -1.7 | 0.0 |
| Net profit | -40.5 | -45.7 | -53.7 | -59.3 | -44.6 | 170.9 |
| EPS (SEK) | -1.3 | -0.9 | -1.0 | -0.5 | -0.4 | 1.5 |

| Growth | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
|------------|------|------|------|------|------|------|
| Net sales | NA | NaN | NaN | NaN | NaN | 632% |
| EBIT | NA | -13% | -18% | -11% | 28% | 498% |
| EBITDA | NA | -17% | -18% | -11% | 28% | 504% |
| EBT | NA | -13% | -18% | -10% | 28% | 498% |
| Net profit | NA | -13% | -18% | -10% | 25% | 483% |
| EPS | NA | 31% | -9% | 40% | 29% | 483% |

| Margins | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
|--------------|---------|----------|-----------|------|---------|--------|
| Gross margin | 99.5% | 100.0% | 100.0% | N/A! | 100.0% | 100.0% |
| EBIT-margin | -744.9% | -2445.5% | -10545.2% | N/A | -171.6% | 93.4% |

Source: Company information and Carlsquare estimates

Balance sheet (SEKm)

| | 2020A | 2021A | 2022E | 2023E | 2024E | 2025E |
|------------------------------|-------------|--------------|-------------|--------------|--------------|--------------|
| ASSETS | | | | | | |
| Tot. intangible assets | 21.0 | 21.0 | 21.0 | 21.0 | 26.9 | 30.1 |
| Tot. tangible assets | 0.9 | 0.6 | 0.4 | 0.3 | 0.0 | -1.5 |
| Tot. other fixed assets | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Total fixed assets | 21.9 | 21.6 | 21.4 | 21.3 | 26.9 | 28.6 |
| Inventories | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Accounts Receivables | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Other current assets | 6.2 | 5.5 | 5.1 | 5.1 | 5.1 | 5.1 |
| Prepaid expenses | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Cash | 55.0 | 106.5 | 56.2 | 118.4 | 68.2 | 237.4 |
| Total current assets | 61.2 | 112.0 | 61.3 | 123.5 | 73.3 | 242.5 |
| Total assets | 83.1 | 133.6 | 82.7 | 144.8 | 100.2 | 271.1 |
| Shareholder equity | | | | | | |
| Total equity | 72.3 | 122.8 | 68.8 | 130.9 | 86.4 | 257.3 |
| DEBT | | | | | | |
| Debt to creditors | 1.0 | 0.4 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tot. long-term liabilities | 1.0 | 0.4 | 0.0 | 0.0 | 0.0 | 0.0 |
| Debt to creditors | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Accounts payable | 9.8 | 10.4 | 13.8 | 13.8 | 13.8 | 13.8 |
| Other short-term liabilities | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Accrued expenses | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Tot. short-term debt | 9.8 | 10.4 | 13.8 | 13.8 | 13.8 | 13.8 |
| Tot. equity and debt | 83.1 | 133.6 | 82.7 | 144.8 | 100.2 | 271.1 |
| Key Ratios | | | | | | |
| | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
| Current ratio | 6.2 | 10.7 | 10.2 | 8.9 | 5.3 | 17.5 |
| Cash ratio | 5.6 | 10.2 | 9.8 | 8.6 | 4.9 | 17.1 |
| | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
| Net debt (-)/Net cash (+) | 54.0 | 106.1 | 135.7 | 118.4 | 68.2 | 237.4 |
| Net debt/EBITDA | N.M. | N.M. | N.M. | N.M. | N.M. | N.M. |
| Net debt/Equity | N.M. | N.M. | N.M. | N.M. | N.M. | N.M. |
| Debt/Equity | 15% | 9% | 9% | 11% | 16% | 5% |
| Equity/Total assets | 87% | 92% | 91% | 90% | 86% | 95% |
| | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
| ROA | Neg. | Neg. | Neg. | Neg. | Neg. | 63.0% |
| ROE | Neg. | Neg. | Neg. | Neg. | Neg. | 66.4% |
| ROIC | Neg. | Neg. | Neg. | Neg. | Neg. | 50.1% |

Source: Company information and Carlsquare estimates

Cashflow (SEKm)

| | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
|--------------------------------------|-------|-------|-------|-------|-------|-------|
| CF operating activities | -36.1 | -45.3 | -53.8 | -59.2 | -44.1 | 171.5 |
| CF investing activities | 0.0 | 0.0 | 0.0 | 0.0 | -6.1 | -2.3 |
| CF financing activities | 82.1 | 95.2 | 79.0 | 41.9 | 0.0 | 0.0 |
| Cashflow | 43.2 | 51.5 | 25.2 | -17.3 | -50.2 | 169.2 |
| Cash, BoP | 2.7 | 45.8 | 97.3 | 122.6 | 105.3 | 55.1 |
| Cash, EoP | 45.8 | 97.3 | 122.6 | 105.3 | 55.1 | 224.3 |
| Key ratios | | | | | | |
| | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
| CF operating activities/Sales | -13.4 | NaN | NaN | NaN | -1.8 | 0.9 |
| CF operating activities/Total assets | -0.4 | -0.3 | -0.7 | -0.4 | -0.4 | 0.6 |
| DPS (SEK) | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |

Source: Company information and Carlsquare estimates

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