Herantis Pharma

Reason: In-depth research

Company-sponsored research

Not rated

Taking a closer look at the new R&D strategy

- Review of new intranasal delivery and clinical programme
- Growing industry interest in neurology & proteostasis
- Preview for AdeLE readout in Q1'21

Simplifying delivery of CDNF in new R&D programme

We take a closer look at the rationale behind Herantis' pivot away from intracerebral delivery of CDNF and toward intra-nasal and subcutaneous delivery, and map out the projected near-term development timelines for CDNF, Lymfactin and xCDNF. Although it means taking a step back in the short-term, it may pay off in the longer term, with CDNF now looking like a more patient-friendly and partnerable asset if it proves efficacious.

The right time and place

A review of the biotech funding landscape shows an increasing interest in neurology with neurodegenerative diseases leading the pack. This is a clear break from the historic cautiousness towards neurology, with the shift driven by the discovery of several causative and disease-associated genes in e.g. Parkinson's disease, allowing more targeted treatments, and developments in enabling technologies, particularly around delivery across the CNS with nanoformulations. Herantis' strategy of pharmacologically enhancing the ability of neurons to deal with misfolded and abnormal proteins (proteostasis) is being pursued by several other well-regarded players that are beginning to enter into the clinic, putting Herantis in good company.

Lymfactin topline results expected in Q1'21

Topline results from the Ph II AdeLE study of Lymfactin in breast cancer-associated lymphedema (BCAL) are expected in Q1'21. Given the small study size, showing statistical significance may prove difficult in this trial but we preview what trends to look out for when the results come.

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Estimate changes (%)

	2020e	2021e	2022e
Sales	0.0%	0.0%	0.0%
EBIT (rep)	0.0%	0.0%	0.0%
EPS (rep)	0.0%	0.0%	0.0%

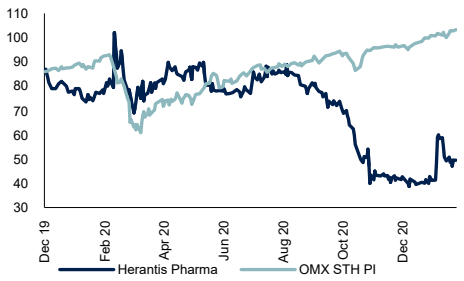
Source: ABG Sundal Collier

Share price (SEK)	08/02/2021	49.5
Fair value range (per share)		na

Healthcare, Sweden
HRNTS.ST/HRNTS SS

MCap (SEKm)	331
MCap (EURm)	33
Net debt (EURm)	5
No. of shares (m)	6.7
Free float (%)	*55*
Av. daily volume (k)	0
Next event	Q4 report: 26 Feb

Performance



	1m	3m	12m
Absolute (%)	*19.3*	*-2.9*	*-36.1*

Source: FactSet

EURm	2018	2019	2020e	2021e	2022e
Sales	0	0	0	36	14
EBITDA	-4	-5	-6	30	8
EBITDA margin (%)	*-1,594.1*	*-2,155.6*	*nm*	*83.4*	*54.3*
EBIT adj	-5	-6	-6	29	7
EBIT adj margin (%)	*-2,116.7*	*-2,655.2*	*nm*	*81.7*	*50.9*
Pretax profit	-4	-7	-6	29	7
EPS rep	-0.85	-1.13	-0.95	4.21	1.02
EPS adj	-0.85	-1.13	-0.95	4.21	1.02
Sales growth (%)	*2.2*	*-2.2*	*-100.0*	*na*	*-60.6*
EPS growth (%)	*-69.7*	*-32.9*	*15.7*	*542.7*	*-75.7*

Source: ABG Sundal Collier, Company data

	2020e	2021e	2022e
P/E (x)	-5.1	1.2	4.8
P/E adj (x)	-5.1	1.2	4.8
P/BVPS (x)	-9.77	1.20	1.00
EV/EBITDA (x)	-6.9	0.3	0.3
EV/EBIT adj (x)	-6.0	0.3	0.3
EV/sales (x)	nm	0.25	0.15
ROE adj (%)	*51,230.4*	*234.6*	*22.8*
Dividend yield (%)	*0*	*0*	*0*
FCF yield (%)	*-15.7*	*96.3*	*16.1*
Lease adj. FCF yld (%)	*-15.7*	*96.3*	*16.1*
Net IB debt/EBITDA	-0.9	-0.8	-4.0
Lease adj. ND/EBITDA	-0.9	-0.8	-4.0

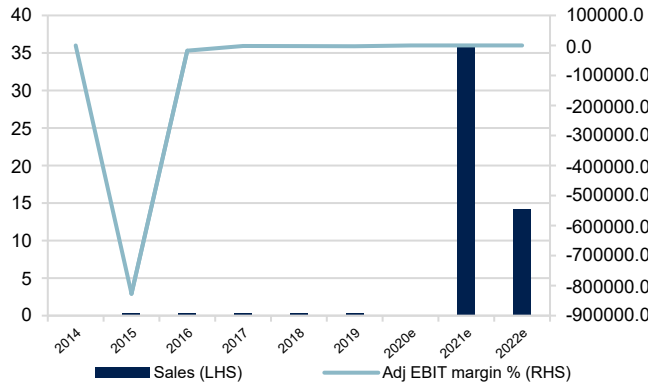
Company description

Herantis Pharma is a publicly listed (Finland, HRTIS and Sweden, HRNTS) clinical stage company aiming to revolutionize the treatment of diseases with unmet clinical needs. Based on leading academic research published in high-impact journals including Nature and Science, two ongoing clinical development programs explore the potential of its novel drug candidates in Parkinson's disease and secondary lymphedema. Both Parkinson's disease and lymphedema remain conditions in which current treatments only address symptoms and therefore do not enable long-term improvement for patients.

Risks

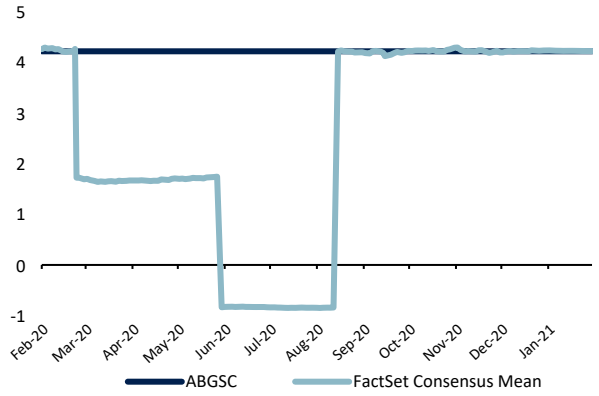
The key risks for Herantis are related to clinical and development risks of its candidates in terms of failed or delayed studies. Liquidity and financing risks are also important to consider, as Herantis is currently a development-stage company with negative cash flow. The competitive landscape could also be significantly different at the time when Herantis' drug candidates could be approved.

Annual sales and adj. EBIT margin



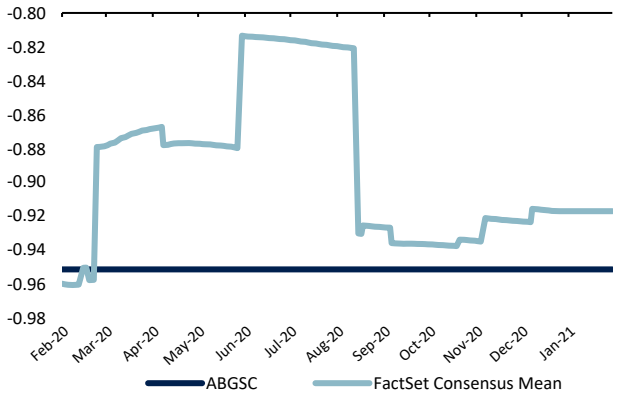
Source: ABG Sundal Collier, Company data

EPS estimate changes, 2021e, EUR



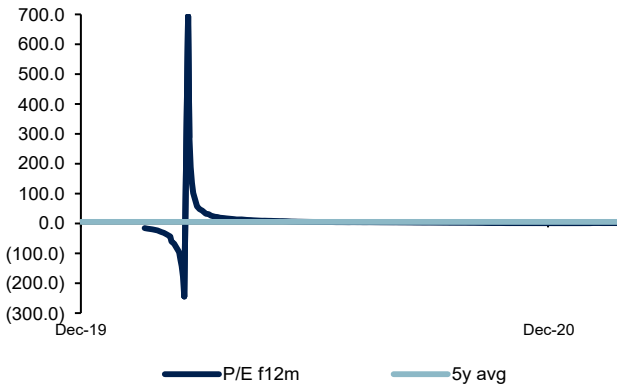
Source: ABG Sundal Collier, FactSet

EPS estimate changes, 2020e, EUR



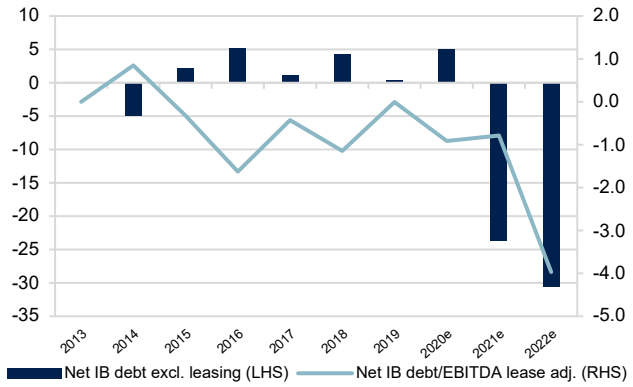
Source: ABG Sundal Collier, FactSet

12-month forward-looking P/E



Source: ABG Sundal Collier, Company data

Net debt and ND/EBITDA adj.



Source: ABG Sundal Collier, Company data

Getting a grip on the new development plan in Parkinson's

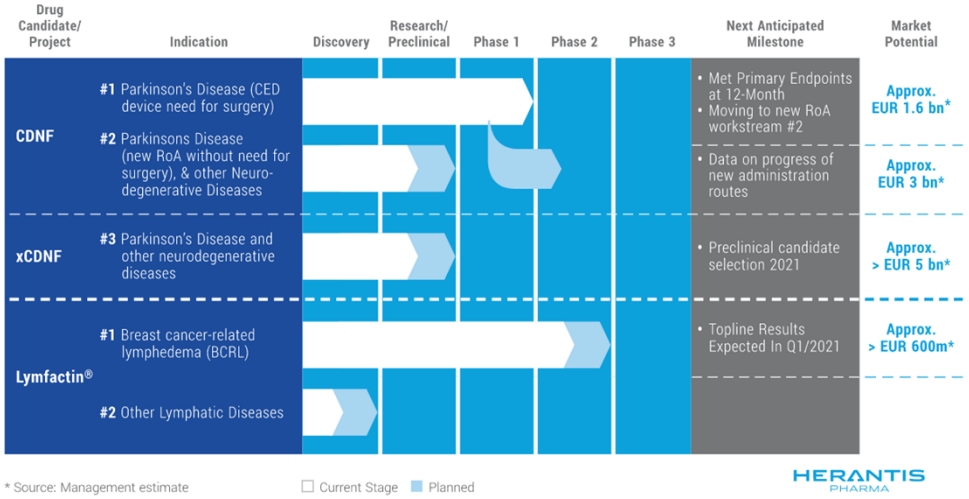
Herantis recently announced a new R&D strategy centred on the exploration of alternative delivery approaches for CDNF in Parkinson's disease. This means leaving direct intra-cerebral delivery behind, and instead working on intranasal and subcutaneous formulations. Although it represents a loss of short-term momentum as Herantis now moves to re-formulate CDNF for optimal intra-nasal delivery, this may prove to be the right decision, as a more patient-, physician-, and regulator-friendly delivery method could accelerate subsequent clinical development and make CDNF a more commercially attractive product for future partnering. We delve deeper into the rationale behind this, and what it means for Herantis' development plan.

In 2020, Herantis reported six- and 12-month data for its Ph I/II clinical study with Cerebral Dopaminergic Neurotrophic factor (CDNF) in patients with Parkinson's disease (PD). The study was initially troubled by concerns stemming from two serious adverse events (SAEs) due to infusion-related infections, but this later appeared manageable with improvements in aseptic procedures and other study treatment-related procedures. Although designed to assess safety and tolerability, some efficacy observations were seen on clinical assessment by UPDRS and DAT PET imaging, suggesting that CDNF may be having an effect despite the study being run in patients with more advanced disease (as required by regulators), and thus severe dopaminergic neuron loss, than could perhaps be expected to really benefit from the treatment.

Growing unease with the tolerability and acceptability of the original administration method and device, along with ongoing internal work on less invasive routes such as intranasal or subcutaneous delivery, resulted in Herantis announcing an update to its R&D strategy in November 2020. This revealed a pivot of the CDNF programme away from invasive intracerebral delivery using the Renishaw device and towards alternative delivery routes such as intranasal and subcutaneous. For CDNF in the lead indication of Parkinson's disease, nose-to-brain administration with uptake through the olfactory epithelium is now being explored.

This programme is proceeding on two tracks, with the main work stream being micro-formulation with Herantis' original CDMO. In parallel, the collaboration with nano-formulation experts Nanoform announced in December 2020 means that Herantis is increasing the likelihood of finding a good formulation by proceeding on these two tracks. In H2'21, we expect the winning formulation of CDNF to be decided, with pre-clinical testing and supplementation of safety and toxicology data for the new formulation and drug-device combination to follow, meaning we could potentially expect to see CDNF back in the clinic in a more patient- and physician-friendly form around H2'22-H1'23. Alongside this, lead candidate selection for xCDNF is expected in Q1'21, with pre-clinical testing proceeding for the rest of the year, followed by IND-enabling studies during 2022.

New pipeline overview for Herantis



Source: Herantis data

Rationale for updated R&D strategy

While the Renishaw device solved the problem of getting across the blood-brain barrier (BBB) and hastened the potential for clinical proof-of-concept data to be generated, it also came with a host of added complications. Beyond the predictable issues associated with requiring a neurosurgical procedure and using a device not yet approved for use beyond clinical trials, further concerns arose over the long-term safety and tolerability of the treatment following two cases of serious infections in the study. Coupled with Herantis' own pre-clinical data on the feasibility of nose-to-brain delivery, as well as developments in the state-of-the-art for intranasal delivery approaches, meant that Herantis had a viable alternative.

While the switch to intra-nasal delivery does mean a loss of momentum and having to return to re-working the formulation and pre-clinical testing, it may prove a sensible decision in the long-term. If proven effective this more patient- and physician-friendly administration approach will allow the treatment of patients with more early stage disease where more dopaminergic neurons remain for CDNF to act on, translating into a larger target population. Additionally, it may not mean a full re-start, as regulators may be able to rely in part on the safety data already generated for CDNF.

The less invasive drug-device combination now being explored will undoubtedly also allow a swifter development process, with more rapid patient enrolment into clinical trials, shorter trials, and less extensive safety parameters for regulators to review. On the commercial side, a CDNF-nasal spray drug device combination is also likely a more partnerable programme.

xCDNF – more than a hedge in Parkinson's disease

xCDNF is a second parallel programme in neurodegenerative disease being pursued by the company. Although seemingly very similar, there are some important differences in the CDNF and xCDNF programmes. Firstly, CDNF is a clinical stage asset and is thus some way ahead of xCDNF, a research-stage asset where lead candidate selection is expected to be completed in Q1'21 prior to entering a pre-clinical programme. Secondly, although designed to retain the pharmacological activity of CDNF, xCDNF has yet to generate the same pre-clinical or clinical data supporting its efficacy. Additionally, they also have different characteristics, as described in our initiation report. The fact that CDNF is a large protein classified as a biological drug while xCDNF is a significantly smaller

synthetic peptide able to pass across the BBB also means that it may have a different regulatory path from CDNF with e.g. other CMC requirements.

While it is possible that both CDNF and xCDNF could be shown to be effective in Parkinson's disease, this potential overlap is mitigated by the possibility of expanding either candidate into other chronic neurodegenerative diseases like ALS or frontotemporal dementia (FTL), or acute indications like reperfusion injury (RPI) following a stroke. For CDNF, in addition to the intranasal programme for chronic neurodegenerative conditions such as Parkinson's disease, the hope in acute indications is that the disruption of the BBB due to the acute insult to the brain may allow CDNF to be administered by subcutaneous injection as well. For xCDNF, which due to its engineered properties is able to cross the BBB, subcutaneous injection is the default mode of delivery.

It is therefore possible that CDNF and xCDNF could be found to be more suitable in different indications, and while being separate programmes, they of course benefit from the learnings between the two due to their common mechanism of action and objectives of crossing the blood brain barrier in order to treat the underlying pathology of neurodegenerative diseases.

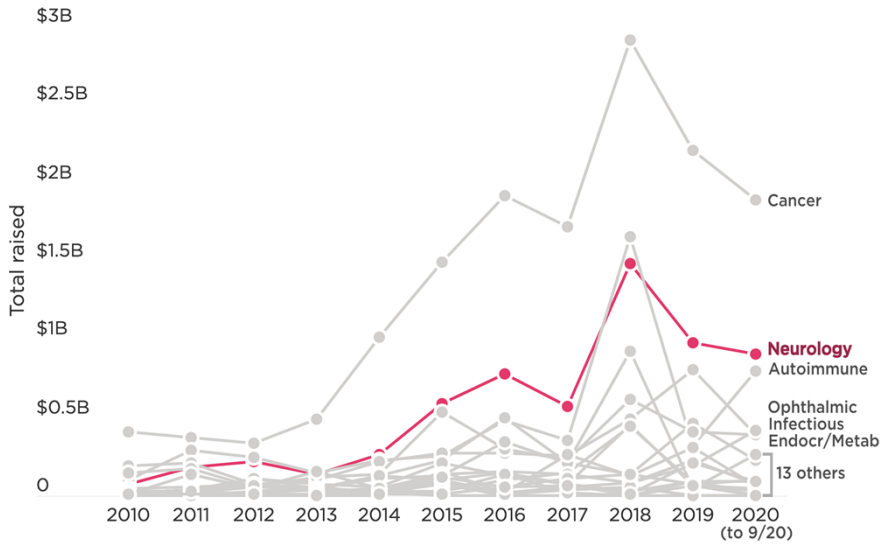
Developments in Parkinson's disease

There have been a few major developments in Parkinson's disease since our initiation report. We review these developments, paying particular attention to the developments in the area of proteostasis, in which Herantis is active. The trend of increasing investor interest in neurology from specialist healthcare investors in early rounds appears stable, with the focus primarily on neurodegenerative diseases like Parkinson's disease. Some big deals have taken place, most notably the Biogen-Denali deal, which we consider in more detail below, and view as a positive signal that Big Biotech and Big Pharma are once again seriously engaging with what has historically been a tricky area to develop drugs in. Additionally, the deal, which is centred on proteostasis candidate DNL151 for Parkinson's disease, can be seen as validating the concept of targeting disturbed cellular functioning and protein handling, which can be seen as a positive read-across for Herantis' CDNF.

Investors and industry are getting over their fear of neurology

Over the past three years, approx. USD 1b annually has been raised in seed and series A funding for neurology companies, moving neurology up into second place (behind cancer) for early stage financing and ahead of other "hot" areas like autoimmunity and inflammation. This is a break in the traditional view of neurology as being too risky for investment, which according to funding data already began in 2015. This perception was based on the fact that neurobiology is complex and still incompletely understood, and that greater heterogeneity between different patient populations is seen in these diseases, which makes running clinical trials difficult. Furthermore, difficulties delivering drugs to the brain, a greater reliance on subjective clinical metrics than in other indications, and a strong placebo effect – especially problematic in PD as the placebo effect is partly driven by dopamine secretion (the missing neurotransmitter in PD) – all meant that the bar for showing clinical efficacy was felt to be higher in neurology than in many other indications

Neurology now second indication by seed/series A funding

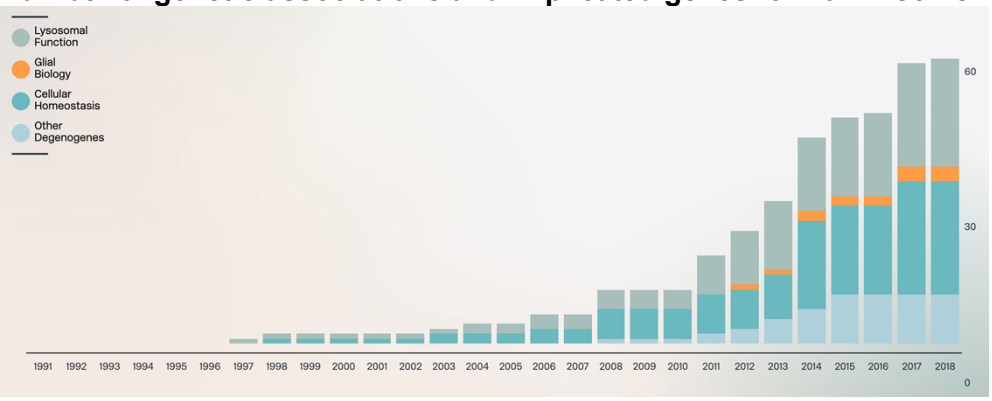


Source: ABG Sundal Collier, BioCentury (2020)

However, developments in neurology have spurred new confidence in the area, as reflected in the increased early stage funding. We believe the funding trend will likely continue to feed through into more late-stage partnering and M&A, which we saw a lot of for neurology and Parkinson's disease in particular last year.

These developments have primarily occurred in two areas. First, the discovery of rare disease-causing mutations have allowed the development of new targeted investigational treatments; Spinraza and Zolgensma for spinal muscular dystrophy (SMA) caused by SMN1 mutations are two early, high-profile examples. In Parkinson's disease, where an estimated 10-15% of patients are thought to have a genetic form of the disease, this has been reflected in the increasing number of programmes tailoring therapies for patients with particular mutations, such as mutations in GBA, a gene coding for the enzyme glucocerebrosidase. At least three companies – Prevail Therapeutics (recently acquired by Lilly), Lysosomal Therapeutics, and Gain Therapeutics – are targeting this area in early clinical or pre-clinical testing for PD. This is only one of many recently discovered genes associated with, or implicated in, the pathogenesis of PD, with drugs being developed to modify or rectify disturbed pathways arising as a result (more on this later). The second major development has been in improved technologies for accessing the central nervous system (CNS). Crossing the blood-brain barrier (BBB) and accessing the CNS has been especially important for Herantis' recent R&D strategy update.

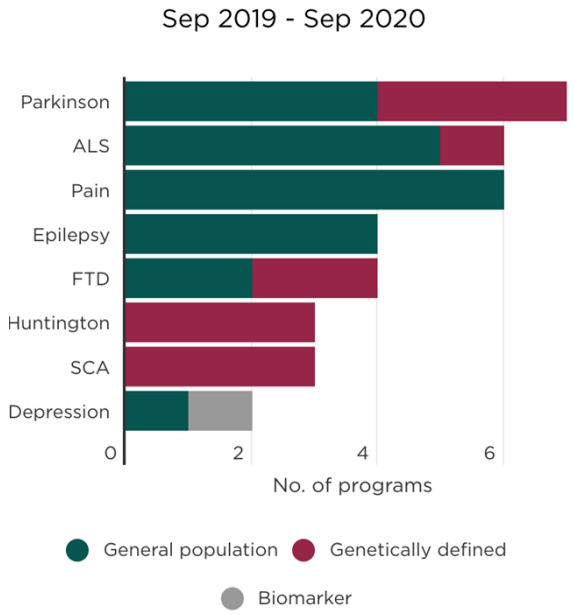
Number of genetic associations and implicated genes for Parkinson's



Source: ABG Sundal Collier, Denali

Along with improved understanding of pathology and cell biology, these developments help to explain the reinvigorated interest in neurology, where the majority of early stage investments are going into neurodegenerative disease, which Herantis is targeting with Parkinson's disease as the lead indication. Other neurodegenerative diseases like ALS, frontotemporal dementia (FTD), Huntington's disease and spinocerebellar ataxia (SCA) also feature prominently. With disturbed proteostasis playing a role in all of these disease pathologies, Herantis is in an interesting space and looks to be in the right place at the right time.

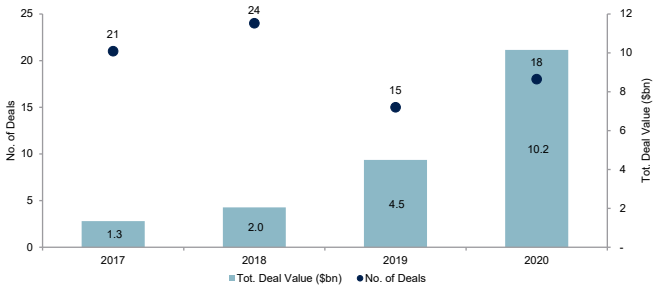
Newcos raising seed/series A rounds



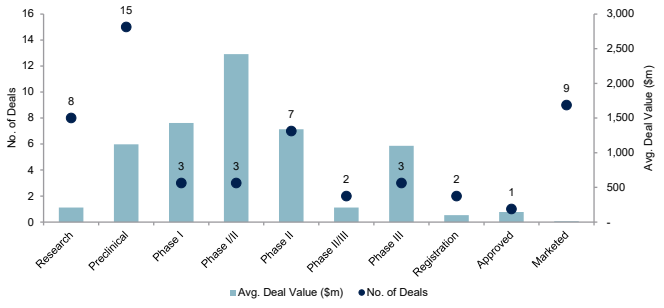
Source: ABG Sundal Collier, BioCentury (2020)

2020 was also a record year for deal-making in Parkinson's disease with 15 deals completed with a combined total value of over USD 10bn. This is a continuation of a rising trend in deal value in PD since 2017, with increasing deal size reflected in the number of individual deals remaining largely static year over year. In 2020, the two most significant deals both involved Biogen, which is likely looking to compensate for a possible pending rejection of aducanumab in Alzheimer's disease. Biogen acquired the exclusive rights to several of Sangamo's neurology gene therapies for a total deal value of USD 2.7bn, and also partnered with Denali Therapeutics to develop and commercialise its LRRK2 programme for a total deal value of USD 1bn (more on this below). Interestingly, outside of pre-clinical stage deals, Ph I/II and II are the stages where the most deals are taking place.

Parkinson's disease deal trends 2017-2020



Parkinson's disease deals by stage



Source: ABG Sundal Collier, company data

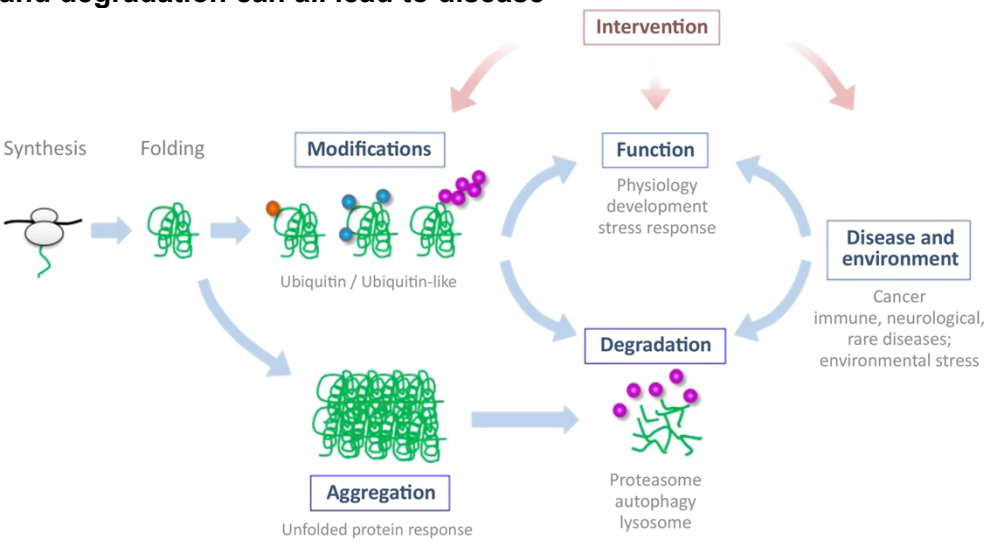
Proteostasis at the forefront

What is it?

Protein homeostasis, or proteostasis, is the process of regulating proteins within the cell in order to maintain the health of individual cells, tissues and whole organisms. Proteins are the body's building blocks, and disruptions of their function underlies many diseases. A hallmark of many age-related diseases is the dysfunction in

proteostasis, leading to the accumulation of protein aggregates. In healthy cells, a complex network of cellular machineries and regulators works to ensure the maintenance of proteostasis by regulating the synthesis, folding, and degradation of proteins. The external and endogenous stresses that accumulate during ageing can make maintaining proteostasis impossible, resulting in the accumulation of misfolded and aggregated proteins. This particularly affects post-mitotic cells like neurones, where effects of broken-down proteostasis manifest as neurodegenerative diseases like Parkinson's and Alzheimer's disease. Pharmacologically augmenting the capacity of proteostasis networks to deal with these stresses is viewed as a very promising field in drug development, and could delay or prevent the onset or progression of disease[1].

Dysfunction of protein synthesis, folding, modification, aggregation, and degradation can all lead to disease



Source: ABG Sundal Collier, European Cooperation in Science & Technology

Cells naturally try to prevent the formation of toxic oligomers (protein clusters) through so-called protein chaperones, but once they have been allowed to form, a number of strategies are employed to counteract the accumulation of these oligomers into toxic protein aggregates like Lewy bodies, a central pathology of Parkinson's disease. This includes disaggregating and remodelling abnormal proteins, shielding the cell from the abnormal proteins, or degrading them. The growing understanding of these processes has opened up the ability to target them with drugs in order to enhance their effects, as well as to try to prevent the formation of toxic oligomers by enhancing the activity of the proteostasis network.

While some companies like Denali are developing highly specific drugs that modify the activity of a particular component of the proteostatic network, Herantis' lead candidate CDNF is a molecule with a broader mechanism of action. It seeks to adjust cellular responses to support protein synthesis and functionality of cells across the key stages of proteostasis – the synthesis, folding, and degradation of proteins – as well as to reduce pro-inflammatory responses and promote the resolution of inflammation and tissue repair. While a more targeted treatment could be seen as having a higher likelihood of success given that its mechanism of action is more clearly understood and its effects therefore more predictable, CDNF's broader mechanism of action of could be regarded as providing some redundancy, i.e. if a particular effect proves clinically irrelevant, one of its other effects may cover for this. Furthermore, the broader mechanism of action could mean that CDNF may

[1] Nature Reviews Molecular Cell Biology. 20; 421–435 (2019)

work across more than one neurodegenerative disease, opening the possibility of a platform drug.

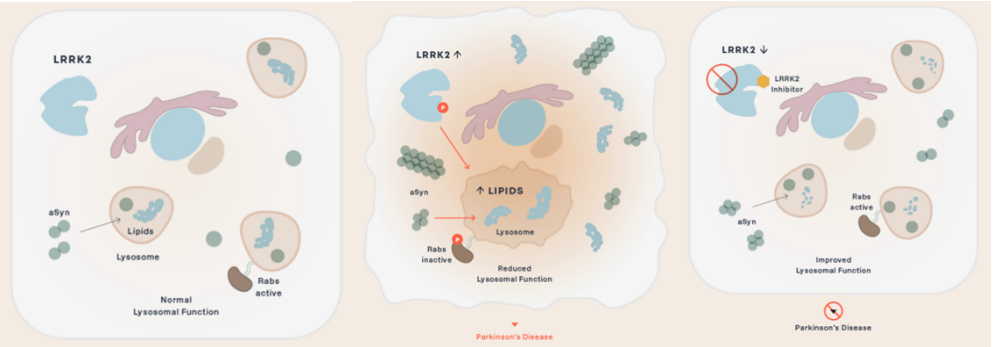
Mounting interest in proteostasis in Parkinson's disease

There are a growing number of companies targeting proteostasis across a host of indications. We focus on four companies that are relevant specifically within PD and provide an overview of their development and potential positive read-across to Herantis.

Denali Therapeutics – market cap USD 8.86bn

Denali has a broad pipeline of candidate drugs in several neurodegenerative diseases. These are mainly in the drug discovery stage, but some are also in clinical testing, including PD candidate DNL151, which targets LRRK2. LRRK2 is amongst the most common genetic risk factors for Parkinson's disease, and codes for a protein called dardarin, which is involved in maintaining a healthy cellular environment by regulating lysosomal function. Lysosomes act as the cell's waste disposal system by breaking down different biomolecules such as proteins and lipids. Increased levels of LRRK2 leads to lysosomal dysfunction, which is thought to allow the accumulation of alpha-synuclein and other waste products that contribute to neurodegeneration and the formation of Lewy bodies. DNL151 is a small molecule inhibitor of LRRK2 capable of crossing the BBB; it is currently in Ph I clinical testing and is expected to begin first clinical proof-of-concept studies in 2021.

Mechanism of action of DNL151



Source: ABG Sundal Collier, Denali

In additional to DNL151, DNL343 is another candidate drug in Ph I testing in healthy volunteers initially aimed at patients with ALS and FTD. It is a small molecule designed to restore normal function of EIF2B, a regulator of cellular stress, which modulates protein synthesis and down-regulates stress protein expression when allowed to function, and thus also fits the bill of a proteostasis drug.

In August 2020, a deal was struck between Denali and Biogen, amounting to >USD 1bn in the form of an USD 560m upfront payment and an equity investment of USD 465m, with another USD 1.125bn in milestones centred on DNL151. This could not only be viewed as a positive indication of the interest from 'Big Biotech' for neurodegenerative diseases and Parkinson's disease in particular, but also as an important validation of the therapeutic goal of targeting proteostasis. This deal followed from the USD 350m upfront deal Biogen struck with Sangamo earlier in 2020 for a host of pre-clinical neurology-focused gene-regulating therapies using its zinc-finger platform, including ST-502 for synucleinopathies including Parkinson's disease.

Neuron23 – not listed

This San Francisco-based start-up (located a stone's throw away from Denali) was launched as recently as December, with a USD 133 million combined Series A and Series B round with big name investors like Westlake, Kleiner Perkins and Redmile Group leading the rounds. It has acquired a molecule tackling LRRK2 from German AI-driven small molecule discoverer Origenis; this is the same target that Denali's DNL151 is aimed at. Neuron23 is not only looking to treat the 3% or so of PD patients with the LRRK2 mutation, but also other patients who have a highly active wild-type (normal) version and hope to move into clinic in 2021. Neuron23 can also be seen as an example of the mounting interest and belief in PD treatments targeting aspects of proteostasis.

Prothena / Roche

α-synuclein-targeting prasinezumab (RG7935), in development by Prothena and Roche, is among the more advanced stage disease-modifying treatments. Its Ph II study PASADENA, however, missed its primary endpoint in April 2020, but it did show signs of efficacy and is still being taken forward, showing that clinical setbacks are nothing new in drug development for Parkinson's disease, and may not spell the end for clinical programmes. It is one of five clinical stage antibodies targeting α-synuclein, with Biogen's BIIB054 (cinpanemab) the only other in Ph II development.

Yumanity Therapeutics – market cap USD 197m

Yumanity, a company focused on drug discovery for neurodegenerative diseases caused by protein misfolding and focusing synucleinopathies such as Parkinson's disease and Lewy body dementias (LBDs), completed a reverse merger with fellow proteostasis player Proteostasis Therapeutics in December 2020, forming a combined company with considerable know-how and resources around protein misfolding. Yumanity's lead candidate is YTX-7739, an SCD1 inhibitor that reduces levels of desaturated fatty acids, which appears to decrease α-synuclein association with membranes. YTX-7739 is in Phase I testing for Parkinson's disease, with a Phase II trial set to begin in 2022. The combined company has a cash runway into 2020 following a June deal with Merck & Co, which exclusively licensed rights to two programmes from Yumanity to treat ALS and frontotemporal lobar dementia FTLD for an undisclosed upfront payment and up to $500 million in milestones, plus royalties. Yumanity and Merck & Co will collaborate on preclinical testing, after which Merck will be responsible for clinical development and commercialisation.

Yumanity set to finish Ph I development in 2021 and start Ph II in 2022

Source: ABG Sundal Collier, Yumanity Therapeutics

Other recent key developments in PD

Voyager's lead gene therapy programme in PD was halted by the US Food & Drug Administration in December 2020 after MRI abnormalities appeared in some patients in its ongoing Ph II trial. This was only one of several gene therapy setbacks seen recently; Voyager's programme in Huntington's disease also put on clinical hold only two months prior. The RESTORE-1 is a Phase II clinical trial assessing NBIb-1817 (VY-AADC), an intracerebral AAV-based investigational gene therapy as a potential treatment for PD. NBIb-1817 is designed to help produce the aromatic L-amino acid decarboxylase (AADC) enzyme in brain cells of Parkinson's patients, where it can convert levodopa to dopamine.

Despite this setback, interest in novel therapies for neurodegenerative diseases remains high, with Lilly acquiring Prevail Therapeutics and its pipeline of Ph I/II and pre-clinical gene therapy candidates in PD, Gaucher disease and FTD for USD 1.04bn in December 2020. PR001, an AAV9 gene therapy for patients with Parkinson's disease with GBA1mutations and neuronopathic Gaucher disease (nGD) is currently recruiting patients for a Ph I/II clinical trial.

In January 2021, Cortexyme announced that it would begin testing its Alzheimer's disease candidate, atuzaginstat, in Parkinson's disease as well, with a Ph II PEAK trial planned to start enrolling patients in Q3'21. This oral bacterial protease inhibitor takes a new approach to treating neurodegenerative disease by targeting the toxic proteases called gingipains (enzymes that breakdown proteins) produced by certain gum bacteria that have been found to be present in patients with Alzheimer's disease.

Medtronic, a leader within DBS (deep brain stimulation), has launched a clinical trial to assess the safety and efficacy of adaptive deep brain stimulation, or aDBS, an investigational feature of its Percept PC Neurostimulator device. This device was approved in 2020 and become the first DBS system on the market able to sense and record patient brain signals during therapy sessions. Unlike conventional continuous deep brain stimulation (cDBS), in which electrical signals used for brain stimulation must be manually adjusted to suit the needs of each patient, aDBS allows these signals to be automatically adjusted to ease patients' symptoms based on their individual responses during therapy sessions. The ADAPT-PD trial aims to enrol 36 patients, and if it proves able to successfully prolong the duration of "on" time periods, it could allow aDBS to be permanently programmed into patients' devices, which would not only benefit patients but also reduce the burden of manual adjustments for clinicians.

In terms of approvals, Neurocrine's Ongentys was approved in April 2020, but is not expected to have a significant impact on the treatment of Parkinson's disease. It belongs to the COMT inhibitors class, within which approved drugs already exist and are used as add-ons to levodopa / DOPA decarboxylase inhibitors (DDCI).

Herantis is well-positioned

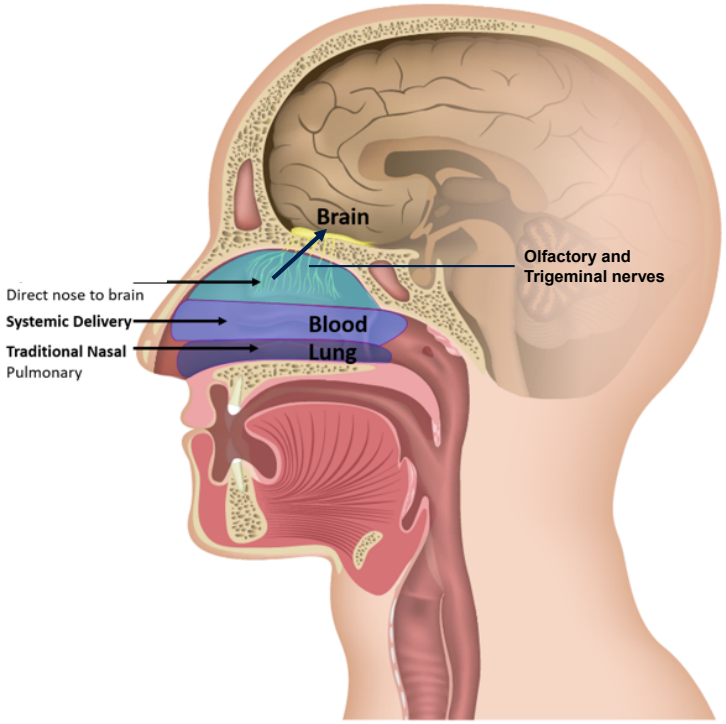
The bearish interpretation is that development within PD is fierce and that even within therapies targeting aspects of proteostasis, other well-funded companies are beginning to enter into early clinical testing. The bullish interpretation, however, is that interest in neurodegenerative diseases has not been this high among Big Pharma and Biotech in a long time, leaving Herantis in good company alongside other players pursuing treatments based on the same fundamental strategy of pharmacologically enhancing neurons' ability to deal with misfolded and abnormal proteins, although the pathways targeted obviously vary. With the right formulation and method of administration allowing testing in the right patient population (earlier stage disease), CDNF may prove itself a worthy competitor, and could be in a position to make good on the rising interest in neurodegenerative diseases.

Intranasal delivery and BBB penetration

Part of the rationale for the recent pivot away from direct invasive surgical intra-cerebral delivery using the Renishaw device to less invasive and more patient-and regulator-friendly administration routes has been internal pre-clinical work showing the feasibility of nose-to-brain delivery of CDNF. Although this has not yet been shared publicly, we note that a lot of work is underway in nose-to-brain (N2B) delivery, and progress has been made in recent years.

Nose-to-brain delivery is based on the conducive anatomical architecture between the nasal cavity and the CNS, primarily via the olfactory and trigeminal nerve pathways at the cribriform plate. The difficulties with nose-to-brain delivery centre on avoiding degrading enzymes in the nasal cavity and nasal ciliary clearance (tiny hairs whipping away foreign matter). Together, these factors can reduce the time that a drug stays in contact with the olfactory epithelium, and thus its ability to cross into the CNS. Much research has been done on how to optimally formulate drugs so that they can overcome these challenges, as well as on designing delivery devices that ensure as much of the drug load as possible gets into the upper portion of the nasal cavity. Due to their small size, shape and surface characteristics, as well as their low biotoxicity and good biocompatibility, nanoparticles have been explored extensively in this area[2].

Direct nose-to-brain delivery across the olfactory epithelium



Source: ABG Sundal Collier, SipNose Technologies

[2] Pharmaceutics. 12(2): 138 (2020)

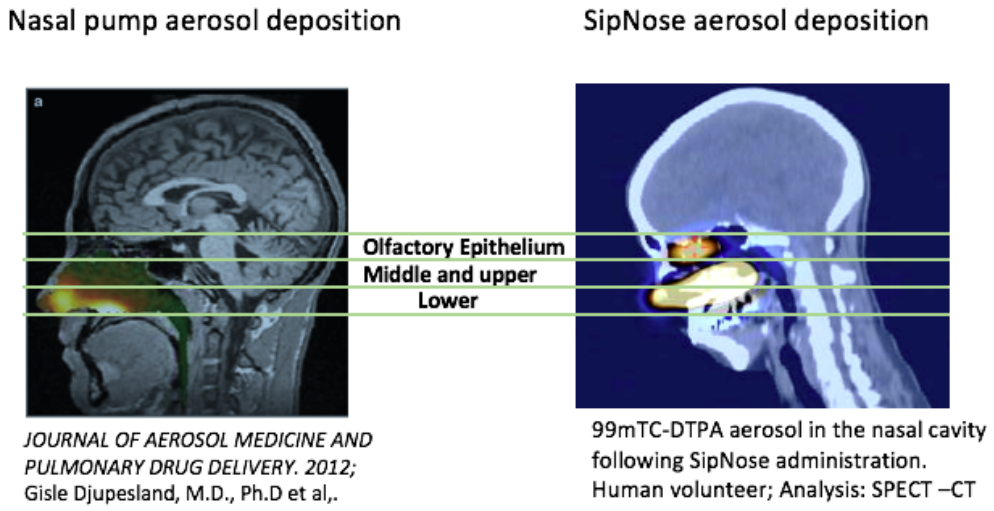
Herantis proceeding down dual formulation tracks

Herantis is evaluating two intranasal formulations. The original mainstream programme with Herantis' original CDMO is for a micro-formulation. The second programme is the recently announced collaboration with nano-formulation experts Nanoform, who are working on re-formulating CDNF and possibly its excipients into a nanoparticle formulation to evaluate whether this could further enhance the delivery of CDNF with intra-nasal delivery directly into the brain. Much of the work in terms of optimising the lipophilicity and size of the drug has already been completed, with the remaining work centred on optimising the formulation and delivery device.

Herantis is hoping to achieve high single-digit drug penetration into the brain parenchyma from the low single-digit penetration currently achieved with the existing formulation in pre-clinical testing. Intranasal insulin has been investigated elsewhere as a potential treatment for Alzheimer's disease and has been found to be feasible, with uptake into the CNS taking place, although it remains ineffective for treating AD. CDNF at 18.5 kD is approx. three times the size of insulin, suggesting that it could be more challenging, but it is still within the limits of what could feasibly cross the BBB. As part of its collaboration with Nanoform, the Finnish firm will carry out two proof-of-concept formulation studies, with the first being for CDNF with intranasal delivery. The second may also be in CDNF or possibly in xCDNF. Although modest in size, the fact that the deal with Nanoform also included Nanoform taking a €1.6m equity stake in Herantis ensures Nanoform has more skin in the game, which could be seen as a positive.

We expect that work on the two formulations will be completed in H2'21, at which point Herantis plans to compare the respective merits of the two formulations and decide on the formulation and device combination to take forward. A final pre-clinical study in primates would then likely follow after this, together with a full IND enabling programme before the nose-to-brain formulation of CDNF could potentially move back into the clinic.

Delivery devices can make a significant difference in the proportion of drug that makes it to the olfactory epithelium



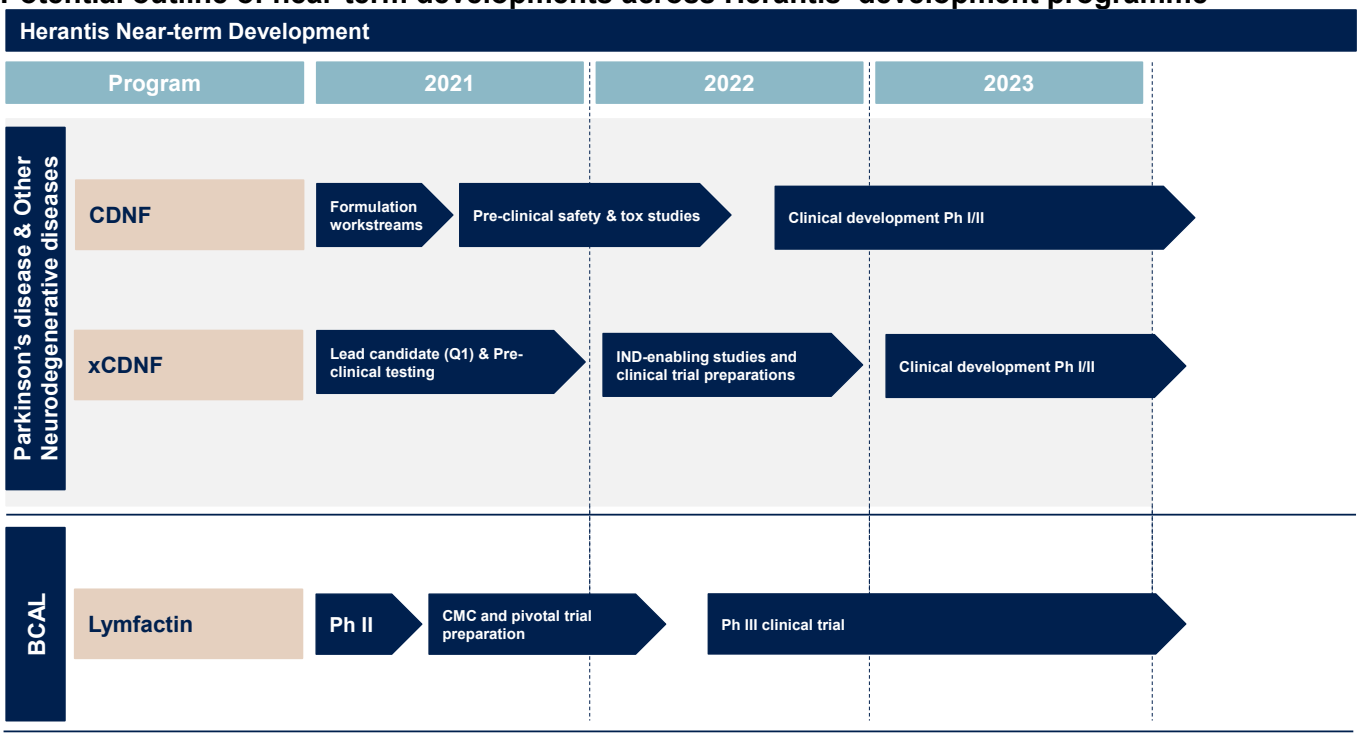
Source: ABG Sundal Collier, SipNose Technologies

Looking ahead

There are a number of key milestones in the near term for Herantis, with readout from the Ph II AdeLE study in Q1'21 being the most significant. Additionally, lead candidate selection for xCDNF is expected in Q1'21. Beyond these communicated milestones, we draw up a potential development timeline with future anticipated milestones and their approximate timing. We expect a final formulation for CDNF to be decided upon around Q3'21, with pre-clinical testing and generation of safety and toxicology data for the new drug-device combination to follow into 2022 in order to meet regulators' demands. Following this, clinical development could potentially be resumed around H2'22-H1'23, in our estimation, with deviations in either direction of course possible.

For xCDNF, IND-enabling studies are expected in 2022 following final pre-clinical testing during 2021 once a lead candidate has been selected. Early clinical development for xCDNF could then begin around 2023 in our estimation, although this of course highly dependent on the progress of the pre-clinical programme, regulatory interactions and future commercial considerations. Assuming a positive readout from the Ph II AdeLE study, we view a Ph III clinical start date in 2022 as feasible, with time given for CMC work to scale up manufacturing and for interactions with regulators over the design of the final pivotal trial.

Potential outline of near-term developments across Herantis' development programme

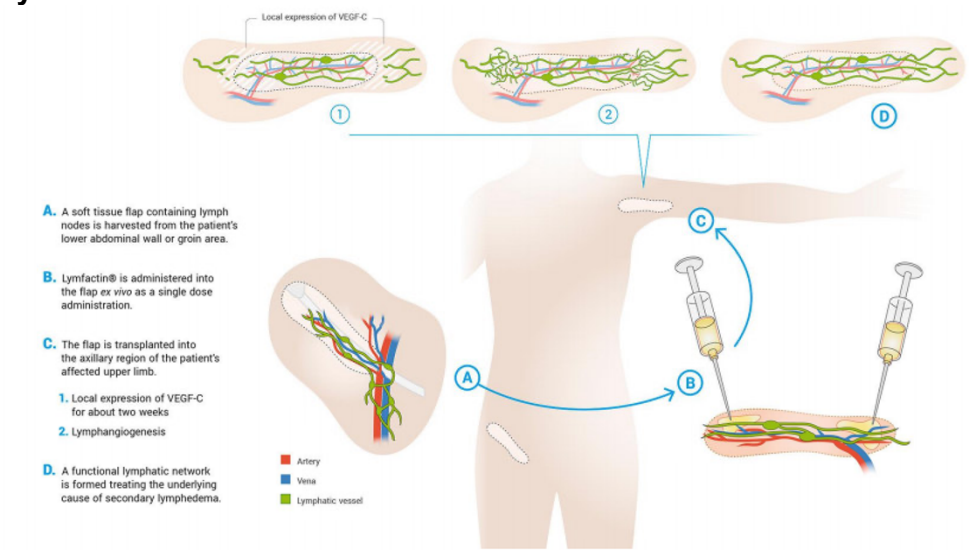


Source: ABG Sundal Collier

Lymfactin programme

Herantis' third programme is Lymfactin, a VEGF-C gene therapy being developed for breast cancer-associated lymphoedema (BCAL) with the intended effect of promoting the formation of new lymphatic vessels. This condition, described in more detail in our initiation report, occurs following breast cancer surgery where disruption of the lymphatic system leads to an accumulation of lymph fluid in the arm of the patient with resulting swelling, pain and reduced mobility as a result. Lymfactin is administered together with lymph node transfer surgery as a single-dose treatment to promote the reconstitution of a functional lymphatic system. In the completed Ph I study, Lymfactin was found to be safe and tolerable throughout the extended follow-up period of 24 months with observations of clinical benefit, although the lack of a control arm in this safety study prevents a firm read on efficacy.

Lymfactin mechanism of action



VEGF-C = Vascular endothelial growth factor C ; Lymphangiogenesis = The growth of lymphatic vessels
Source: Herantis

AdeLE readout

The Ph II AdeLE study is set to read-out during Q1'21 after 12 months of blinded follow-up, and will provide the first look at efficacy relative to a placebo arm. It's a relatively small study carried out in only 39 participants, which means that the effect of Lymfactin would need to be quite considerable for a statistically significant difference to be found. This means that a qualitative assessment of the degree of clinical significance seen in the lymfactin arm compared to the placebo arm will likely be important. There are three primary endpoints that look at change in the volume of the affected arm, lymphatic flow, and quality of life (QoL) in patients receiving lymfactin compared to patients receiving placebo. To determine the clinical significance of potential improvements with lymfactin one will need to look at two parameters: the number of patients in each group that respond to treatment by showing signs of improvement, and the degree of actual improvement for each endpoint.

Finally, an assessment of whether there are any patient-, disease- or treatment-related factors that appear to correlate with better outcomes will be relevant to consider for Herantis when it receives the data, although the size of the trial does limit this to an extent. Potentially relevant findings here could help optimise the design of the subsequent study where showing not only clinical but also statistical significance will be vital. This type of analysis could prove especially relevant in the AdeLE study given the significant variability in reported outcomes in different lymph node transfer studies (as seen below and discussed in our initiation report), suggesting that the number of variables influencing outcomes is substantial. An additional effect of this variability in outcomes is that the risk of a type II error (false negative) is increased as the chance of a spuriously high effect in the placebo arm is raised, which again could make showing statistically significant results in this study harder.

Outcome data for lymph node transfers is variable and scarce

Authors	No. of Flaps	Recipient site	Reduction rate (%)	Donor site complications	Recipient site complications	Follow-up (months, range)
Becker et al (2006)	24	Axilla, elbow	n.a.	Lymphorrhea (8)	None	99.6
Saaristo et al (2012)	10	Axilla	32.2 ± 30.9	Seroma (n = 1)	Delayed wound healing (2)	6
Vignes et al (2013)	34 flaps in 26 pts	Axilla (n = 14) Groin (n = 12)	n.a.	Iatrogenic Lymphedema (n = 6) Lymphocele (n = 3) Donor site pain (n = 3)	None	40 (14-72)
Dancey et al (2013)	18	Axilla	n.a.	Seroma (n = 2)	Fat necrosis (n = 1)	14 (4-22)
Granzow et al (2014)	8	Axilla	88.9%	None	Delayed healing (n = 1)	12
Nguyen et al (2015)	29	Axilla	10%	Delayed wound healing (n = 1) Abdominal hernia (n = 1) Venous thrombosis (n = 1)	Delayed healing (n = 1)	11
Barreiro et al (2014)	7	Axilla and Shoulder (n = 6) Dorsum Foot (n = 1)	n.a.	Prolonged edema (n = 1), Dehiscence (n = 1)	Prolonged flap edema (n = 1)	n.a.

Note: Reduction rate refers to the overall reduction in swelling of the affected limb
Source: ABG Sundal Collier

Financials & Business outlook

As of June 30, 2020, Herantis had a cash position of €9.3m with cash flow from operating activities for the six-month period (i.e. H1'20) of €-4.8m. In order to maintain its current development pace, Herantis carried out a private placement in December 2020, which raised €8m through the issuance of 2,162,163 placing shares at €3.70 per share (a 10% discount to the last close at the time), which represent approx. 28.5% of the issued shares in Herantis prior to the placing and approx. 22.2% following the placing.

We estimate that Herantis has a cash runway through 2021, covering the company over the anticipated readout of the Ph II AdeLE study for Lymfactin in Q1'21 and the 2021 developments in the CDNF and xCDNF programmes outlined earlier in this report. Following this, Herantis will likely look to raise further funding to sustain its clinical development programmes. Given the significant costs of later stage drug development, especially in PD where studies are run over a longer period of time, Herantis will likely look to partner with a pharma or biotech company able to carry the costs of further clinical development. In exchange, the typical model means Herantis would give up commercial rights in exchange for milestone and royalty payments on potential future sales.

Income Statement (EURm)	2013	2014	2015	2016	2017	2018	2019	2020e	2021e	2022e
Sales	0	0	0	0	0	0	0	0	36	14
COGS	0	0	0	0	0	0	0	0	0	0
Gross profit	0	0	0	0	0	0	0	0	36	14
Other operating items	0	-6	-7	-3	-3	-4	-5	-6	-6	-6
EBITDA	**0**	**-6**	**-7**	**-3**	**-3**	**-4**	**-5**	**-6**	**30**	**8**
Depreciation and amortisation	0	-2	-9	-1	-1	-1	-1	-1	-1	-0
Of which leasing depreciation	0	0	0	0	0	0	0	0	0	0
EBITA	**0**	**-8**	**-16**	**-4**	**-4**	**-5**	**-6**	**-6**	**29**	**7**
EO items	0	0	0	0	0	0	0	0	0	0
Impairment and PPA amortisation	0	0	0	0	0	0	0	0	0	0
EBIT	**0**	**-8**	**-16**	**-4**	**-4**	**-5**	**-6**	**-6**	**29**	**7**
Net financial items	0	-1	0	-0	2	1	-1	-0	-0	-0
Pretax profit	**0**	**-8**	**-16**	**-4**	**-2**	**-4**	**-7**	**-6**	**29**	**7**
Tax	0	0	0	0	0	0	0	0	-1	-0
Net profit	**0**	**-8**	**-16**	**-4**	**-2**	**-4**	**-7**	**-6**	**28**	**7**
Minority interest	0	0	0	0	0	0	0	0	0	0
Net profit discontinued	0	0	0	0	0	0	0	0	0	0
Net profit to shareholders	**0**	**-8**	**-16**	**-4**	**-2**	**-4**	**-7**	**-6**	**28**	**7**
EPS	0	-3.21	-3.94	-1.07	-0.50	-0.85	-1.13	-0.95	4.21	1.02
EPS Adj	*0*	*-3.21*	*-3.94*	*-1.07*	*-0.50*	*-0.85*	*-1.13*	*-0.95*	*4.21*	*1.02*
Total extraordinary items after tax	0	0	0	0	0	0	0	0	0	0
Leasing payments	0	0	0	0	0	0	0	0	0	0
Tax rate (%)	*ns*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*4.0*	*4.0*
Gross margin (%)	*nm*	*nm*	*100.0*	*100.0*	*100.0*	*100.0*	*100.0*	*nm*	*100.0*	*100.0*
EBITDA margin (%)	*nm*	*nm*	*nm*	*-12,598.9*	*-1,211.3*	*-1,594.1*	*-2,155.6*	*nm*	*83.4*	*54.3*
EBITA margin (%)	*nm*	*nm*	*nm*	*-17,349.6*	*-1,752.2*	*-2,116.7*	*-2,655.2*	*nm*	*81.7*	*50.9*
EBIT margin (%)	*nm*	*nm*	*nm*	*-17,349.6*	*-1,752.2*	*-2,116.7*	*-2,655.2*	*nm*	*81.7*	*50.9*
Pretax margin (%)	*nm*	*nm*	*nm*	*-17,366.7*	*-961.4*	*-1,816.5*	*-2,910.3*	*nm*	*81.4*	*50.2*
Net margin (%)	*nm*	*nm*	*nm*	*-17,366.7*	*-961.4*	*-1,816.5*	*-2,910.3*	*nm*	*78.2*	*48.2*
Growth rates Y/Y	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
Sales growth (%)	*na*	*na*	*na*	*1,195.2*	*789.1*	*2.2*	*-2.2*	*-100.0*	*na*	*-60.6*
EBITDA growth (%)	*na*	*high*	*-16.8*	*52.7*	*14.5*	*-34.5*	*-32.3*	*-13.5*	*645.2*	*-74.3*
EBIT growth (%)	*na*	*high*	*-111.1*	*72.8*	*10.2*	*-23.5*	*-22.7*	*-4.6*	*570.7*	*-75.4*
Net profit growth (%)	*na*	*high*	*-92.1*	*72.6*	*50.8*	*-93.1*	*-56.7*	*2.9*	*542.7*	*-75.7*
EPS growth (%)	*na*	*high*	*-22.8*	*72.9*	*53.1*	*-69.7*	*-32.9*	*15.7*	*542.7*	*-75.7*
Profitability	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
ROE (%)	*nm*	*-77.0*	*-115.9*	*-116.1*	*-76.4*	*-208.9*	*-404.9*	*51,230.4*	*234.6*	*22.8*
ROE Adj (%)	*nm*	*-77.0*	*-115.9*	*-116.1*	*-76.4*	*-208.9*	*-404.9*	*51,230.4*	*234.6*	*22.8*
ROCE (%)	*nm*	*-52.6*	*-76.1*	*-37.0*	*-18.3*	*-48.2*	*-71.4*	*-89.4*	*154.8*	*19.5*
ROCE Adj(%)	*nm*	*-52.6*	*-76.1*	*-37.0*	*-18.3*	*-48.2*	*-71.4*	*-89.4*	*154.8*	*19.5*
ROIC (%)	*na*	*-91.0*	*-129.3*	*-58.8*	*-65.6*	*-104.0*	*-160.2*	*-248.6*	*1,043.2*	*238.8*
ROIC Adj (%)	*na*	*-91.0*	*-129.3*	*-58.8*	*-65.6*	*-104.0*	*-160.2*	*-248.6*	*1,043.2*	*238.8*
Adj earnings numbers	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
EBITDA Adj	0	-6	-7	-3	-3	-4	-5	-6	30	8
EBITDA Adj margin (%)	*nm*	*nm*	*nm*	*-12,598.9*	*-1,211.3*	*-1,594.1*	*-2,155.6*	*nm*	*83.4*	*54.3*
EBITDA lease Adj	0	-6	-7	-3	-3	-4	-5	-6	30	8
EBITDA lease Adj margin (%)	*nm*	*nm*	*nm*	*-12,598.9*	*-1,211.3*	*-1,594.1*	*-2,155.6*	*nm*	*83.4*	*54.3*
EBITA Adj	0	-8	-16	-4	-4	-5	-6	-6	29	7
EBITA Adj margin (%)	*nm*	*nm*	*nm*	*-17,349.6*	*-1,752.2*	*-2,116.7*	*-2,655.2*	*nm*	*81.7*	*50.9*
EBIT Adj	0	-8	-16	-4	-4	-5	-6	-6	29	7
EBIT Adj margin (%)	*nm*	*nm*	*nm*	*-17,349.6*	*-1,752.2*	*-2,116.7*	*-2,655.2*	*nm*	*81.7*	*50.9*
Pretax profit Adj	0	-8	-16	-4	-2	-4	-7	-6	29	7
Net profit Adj	0	-8	-16	-4	-2	-4	-7	-6	28	7
Net profit to shareholders Adj	0	-8	-16	-4	-2	-4	-7	-6	28	7
Net Adj margin (%)	nm	nm	nm	-17,366.7	-961.4	-1,816.5	-2,910.3	nm	78.2	48.2

Source: ABG Sundal Collier, Company data

Cash Flow Statement (EURm)	2013	2014	2015	2016	2017	2018	2019	2020e	2021e	2022e
EBITDA	0	-6	-7	-3	-3	-4	-5	-6	30	8
Net financial items	0	-1	0	-0	2	1	-1	-0	-0	-0
Paid tax	0	0	0	0	0	0	0	0	-1	-0
Non-cash items	0	1	0	0	-2	0	0	0	0	0
Cash flow before change in WC	0	-5	-7	-3	-3	-3	-5	-6	29	7
Change in WC	0	1	-1	-0	1	-1	0	0	3	-2
Operating cash flow	**0**	**-4**	**-7**	**-3**	**-3**	**-4**	**-5**	**-5**	**32**	**5**
CAPEX tangible fixed assets	0	0	0	-0	0	0	0	0	0	0
CAPEX intangible fixed assets	0	-0	-0	-0	0	0	0	0	0	0
Acquisitions and disposals	0	0	0	0	0	0	0	0	0	0
Free cash flow	**0**	**-4**	**-7**	**-3**	**-3**	**-4**	**-5**	**-5**	**32**	**5**
Dividend paid	0	0	0	0	0	0	0	0	0	0
Share issues and buybacks	0	15	0	0	5	0	10	0	0	0
Lease liability amortisation	0	0	0	0	0	0	0	0	0	0
Other non cash items	0	-6	0	-0	2	1	-0	0	-3	2
Balance Sheet (EURm)	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
Goodwill	0	0	0	0	0	0	0	0	0	0
Other intangible assets	0	18	8	7	6	5	4	3	2	2
Tangible fixed assets	0	0	0	0	0	0	0	0	0	0
Right-of-use asset	0	0	0	0	0	0	0	0	0	0
Total other fixed assets	0	0	0	0	0	0	0	0	3	1
Fixed assets	0	18	8	7	6	5	4	3	5	3
Inventories	0	0	0	0	0	0	0	0	0	0
Receivables	0	0	0	0	0	0	0	0	2	1
Other current assets	0	0	0	0	0	0	0	0	3	1
Cash and liquid assets	0	11	6	3	5	2	7	2	31	38
Total assets	**0**	**29**	**14**	**10**	**12**	**7**	**11**	**5**	**40**	**42**
Shareholders equity	0	22	6	2	4	-0	3	-3	27	33
Minority	0	0	0	0	0	0	0	0	0	0
Total equity	**0**	**22**	**6**	**2**	**4**	**-0**	**3**	**-3**	**27**	**33**
Long-term debt	0	6	8	8	6	6	7	7	7	7
Pension debt	0	0	0	0	0	0	0	0	0	0
Convertible debt	0	0	0	0	0	0	0	0	0	0
Leasing liability	0	0	0	0	0	0	0	0	0	0
Total other long-term liabilities	0	0	0	0	0	0	0	0	0	0
Short-term debt	0	0	0	0	1	1	1	1	1	1
Accounts payable	0	1	0	0	0	0	0	0	5	1
Other current liabilities	0	0	0	0	1	1	1	1	1	1
Total liabilities and equity	**0**	**29**	**14**	**10**	**12**	**7**	**11**	**5**	**40**	**42**
Net IB debt	0	-5	2	5	1	4	0	5	-24	-31
Net IB debt excl. pension debt	0	-5	2	5	1	4	0	5	-24	-31
Net IB debt excl. leasing	0	-5	2	5	1	4	0	5	-24	-31
Capital invested	0	17	8	7	5	4	3	2	4	2
Working capital	0	-1	-0	-0	-1	-1	-1	-1	-1	-1
EV breakdown	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
Market cap. diluted (m)	na	na	na	na	na	na	52	33	33	33
Net IB debt Adj	0	-5	2	5	1	4	0	5	-24	-31
Market value of minority	0	0	0	0	0	0	0	0	0	0
Reversal of shares and participations	0	0	0	0	0	0	0	0	0	0
Reversal of conv. debt assumed equity	0	0	0	0	0	0	0	0	0	0
EV	**na**	**na**	**na**	**na**	**na**	**na**	**52**	**38**	**9**	**2**
Capital efficiency	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
Total assets turnover (%)	*nm*	*0*	*0.0*	*0.2*	*2.1*	*2.5*	*2.5*	*0*	*159.1*	*34.3*
Working capital/sales (%)	*nm*	*nm*	*-33,071.9*	*-1,391.0*	*-277.2*	*-336.7*	*-326.5*	*nm*	*-3.5*	*-7.5*
Financial risk and debt service	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
Net debt/equity	*nm*	*-0.23*	*0.36*	*3.30*	*0.29*	*-47.04*	*0.01*	*-1.50*	*-0.86*	*-0.94*
Net debt/market cap	*na*	*na*	*na*	*na*	*na*	*na*	*0.00*	*0.11*	*-0.72*	*-0.94*
Equity ratio (%)	*nm*	*73.6*	*42.6*	*15.6*	*35.3*	*-1.2*	*29.7*	*-67.1*	*67.9*	*77.0*
Net IB debt adj./equity	*nm*	*-0.23*	*0.36*	*3.30*	*0.29*	*-47.04*	*0.01*	*-1.50*	*-0.86*	*-0.94*
Current ratio	*nm*	*7.79*	*9.77*	*4.72*	*3.78*	*1.69*	*5.24*	*1.08*	*5.51*	*12.13*
EBITDA/net interest	*na*	*-8.25*	*-61.97*	*-736.76*	*-1.53*	*-5.31*	*-8.45*	*-52.18*	*284.48*	*73.07*
Net IB debt/EBITDA	*nm*	*0.85*	*-0.32*	*-1.63*	*-0.43*	*-1.15*	*-0.00*	*-0.91*	*-0.79*	*-3.97*
Net IB debt/EBITDA lease Adj	*nm*	*0.85*	*-0.32*	*-1.63*	*-0.43*	*-1.15*	*-0.00*	*-0.91*	*-0.79*	*-3.97*
Interest cover	*nm*	*-8.02*	*-189.61*	*-52.28*	*-6.00*	*-45.17*	*-9.71*	*-59.24*	*278.83*	*68.55*

Source: ABG Sundal Collier, Company data

Valuation and Ratios (EURm)	2013	2014	2015	2016	2017	2018	2019	2020e	2021e	2022e
Shares outstanding adj.	0	4	4	4	5	5	7	7	7	7
Fully diluted shares Adj	0	4	4	4	5	5	7	7	7	7
EPS	0	-3.21	-3.94	-1.07	-0.50	-0.85	-1.13	-0.95	4.21	1.02
Dividend per share Adj	0	0	0	0	0	0	0	0	0	0
EPS Adj	0	-3.21	-3.94	-1.07	-0.50	-0.85	-1.13	-0.95	4.21	1.02
BVPS	0	5.35	1.47	0.38	0.83	-0.02	0.50	-0.50	4.09	4.89
BVPS Adj	0	0.95	-0.60	-1.37	-0.40	-1.00	-0.11	-0.95	3.74	4.61
Net IB debt / share	na	-1.2	0.5	1.3	0.2	0.9	0.0	0.8	-3.5	-4.6
Share price	na	na	na	na	na	na	7.76	4.90	4.90	4.90
Market cap. (m)	na	na	na	na	na	na	52	33	33	33
Valuation	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
P/E	na	na	na	na	na	na	-6.9	-5.1	1.2	4.8
EV/sales	na	na	na	na	na	na	230.39	nm	0.25	0.15
EV/EBITDA	na	na	na	na	na	na	-10.7	-6.9	0.3	0.3
EV/EBITA	na	na	na	na	na	na	-8.7	-6.0	0.3	0.3
EV/EBIT	na	na	na	na	na	na	-8.7	-6.0	0.3	0.3
Dividend yield (%)	*na*	*na*	*na*	*na*	*na*	*na*	*0*	*0*	*0*	*0*
FCF yield (%)	*na*	*na*	*na*	*na*	*na*	*na*	*-12.1*	*-15.7*	*96.3*	*16.1*
Lease adj. FCF yield (%)	*na*	*na*	*na*	*na*	*na*	*na*	*-12.1*	*-15.7*	*96.3*	*16.1*
P/BVPS	na	na	na	na	na	na	15.59	-9.77	1.20	1.00
P/BVPS Adj	na	na	na	na	na	na	-69.73	-5.17	1.31	1.06
P/E Adj	na	na	na	na	na	na	-6.9	-5.1	1.2	4.8
EV/EBITDA Adj	na	na	na	na	na	na	-10.7	-6.9	0.3	0.3
EV/EBITA Adj	na	na	na	na	na	na	-8.7	-6.0	0.3	0.3
EV/EBIT Adj	na	na	na	na	na	na	-8.7	-6.0	0.3	0.3
EV/cap. employed	na	na	na	na	na	na	5.0	10.3	0.3	0.1
Investment ratios	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020e**	**2021e**	**2022e**
Capex/sales	nm	nm	314.7	281.7	0	-3.1	0	nm	0	0
Capex/depreciation	nm	0.1	0.1	5.9	0	-0.6	0	0	0	0
Capex tangibles/tangible fixed assets	nm	0	0	118.6	0	0	0	0	0	0
Capex intangibles/definite intangibles	nm	0.0	0.1	0.8	0	-0.1	0	0	0	0
Depreciation on intangibles/definite inta	nm	10.6	111.6	16.7	20.1	24.8	27.6	25.0	25.0	25.0
Depreciation on tangibles/tangibles	nm	0	0	0	0	0	0	0	0	0

Source: ABG Sundal Collier, Company data

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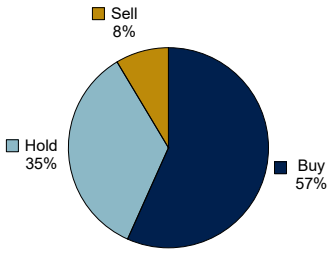

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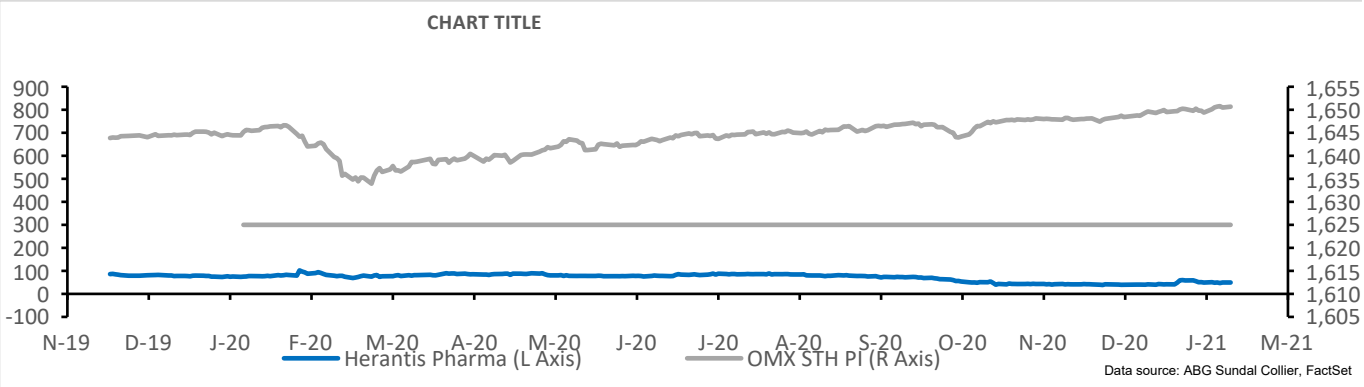
Company: Herantis Pharma **Currency:** SEK **Current Recommendation** COMPANY SPONSORED

Date: 08/02/2021 **Current Target price:**

Current Share price: 49.5



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