EDISON

Selvita

Company outlook

SEL120 to enter clinical development in 2019

Pharma & biotech

The data readout from the SEL24 Phase I/II trial in relapsed/refractory AML and the second lead asset, SEL120, moving into clinical development next year are milestones in Selvita's internal drug R&D. This should accelerate, as the company's plans to focus on innovation were endorsed by shareholders during the fund-raise earlier in 2018. R&D progress across the earlier-stage pipeline has been reported in several publications in recent months. The Innovations Platform continues to receive support from Selvita's rapidly growing drug discovery services business. Our valuation is PLN1.24bn or PLN77.6/share.

Year end	Revenue (PLNm)	PBT* (PLNm)	EPS* (PLN)	DPS (PLN)	P/E (x)	Yield (%)
12/16	66.7	4.6	0.64	0.0	N/M	N/A
12/17	105.9	10.2	0.51	0.0	N/M	N/A
12/18e	104.7	5.9	0.12	0.0	N/M	N/A
12/19e	119.9	(15.8)	(0.99)	0.0	N/M	N/A

Note: *PBT and EPS are normalised, excluding amortisation of acquired intangibles, exceptional items and share-based payments.

SEL24 to deliver data as SEL120 moves into clinic

The most advanced asset, SEL24, a dual PIM/FLT3 inhibitor out-licensed to Menarini Group, is in a Phase I/II trial with relapsed/refractory AML patients, with results expected in early 2019. Several specific FLT3 inhibitors developed by other companies performed well in clinical trials, two of which received FDA approval (midostaurin, Novartis, and gilteritinib, Astellas) and one of which is under priority review (quizartinib, Ambit Biosciences/Daiichi Sankyo), demonstrating the success of this class of drugs. With its dual mechanism of action, Selvita's SEL24 (or MEN1703 under Menarini) is differentiated and *in vivo* data show potentially broader activity. Following completion of the SEL24 handover process to Menarini, Selvita can fully focus on the rest of its pipeline with SEL120, a selective CDK8 inhibitor, now the most advanced asset moving into Phase I likely in H119.

Financials: Drug discovery services grow fast

Selvita's reported total 9M18 revenues of PLN77.2m (-4% y-o-y) were largely in line with our expectations. The slight decline y-o-y is a result of a large income recognised in Q117 after the company out-licensed SEL24 to Menarini Group. Total commercial 9M18 revenues, which exclude subsidies and the out-licensing upfront, were PLN56.6m (up 17% y-o-y). Operating profit of -PLN4.7m was somewhat below our expectations, prompting us to lower our estimates. However, overall this was in line with Selvita's guidance that the main focus following the successful fund-raise earlier in 2018 is now on creating value through R&D research, and R&D spending should therefore pick up going forward.

Valuation: PLN1.24bn or PLN77.6/share

Our valuation of Selvita is slightly lower at PLN1.24bn or PLN77.6/share versus PLN1.30bn or PLN81.2/sh previously. The revision of the operating estimates resulted in a slightly lower DCF valuation, while rolling the model forward had a positive effect on the rNPV values of the R&D projects. Results from the SEL24 Phase I/II trial in 2019 is the main R&D catalysts in the near term.

18 January 2019

Price	**PLN54.40**
Market cap	**PLN870m**

Net cash (PLNm) at end-Q318	148.6
Shares in issue	16.0m
Free float	57%
Code	SLV
Primary exchange	WSE
Secondary exchange	N/A

Share price performance

%	1m	3m	12m
Abs	4.6	0.7	(9.3)
Rel (local)	1.9	(5.5)	(0.6)
52-week high/low	PLN64.3	PLN45.0	

Business description

Selvita is an R&D and drug discovery services company. It operates three business segments: Innovations Platform (internal R&D pipeline), Research Services (medicinal chemistry/biology, biochemistry) and Ardigen (a spin-out bioinformatics company, 52%-owned). Lead R&D asset SEL24 was out-licensed to Menarini Group and is currently in a Phase I/I trial for AML. Selvita is focusing on its other projects in a broad pipeline dedicated to oncology.

Next events

SEL24 Phase I/II data readout	2019
IND for SEL120 Phase I study filed	H119

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Edison profile page

Investment summary

Company description: Focus on innovative R&D increased

Selvita, founded in 2007, has become one of the largest independent drug discovery companies in Europe with a broad R&D pipeline of novel small molecules for oncology. The company is listed on the Warsaw Stock Exchange Main Market and operates in three business segments: Innovation, Services and Bioinformatics. Innovation is responsible for the progress of Selvita's R&D pipeline, which can be broadly described by three platforms: targeted therapies, cancer metabolism and immunometabolism, and immunology and immunooncology. The remaining two segments provide drug discovery (contract chemistry, biology) and bioinformatics services (Ardigen also has an internal R&D programme). Selvita employs more than 550 staff and operates out of research facilities in Krakow and Poznan, Poland. Recently, Selvita announced 10-year expansion plans, which could substantially expand its laboratory and office space and double the employee count from the current level. Selvita continues executing its multi-year strategy to substantially boost R&D research following a successful share issue in March 2018, which raised PLN134m.

Valuation: PLN1.24bn or PLN77.6/share

Our valuation is slightly lower at PLN1.24bn or PLN77.6/share vs PLN1.30bn or PLN81.2/share previously. We maintain our valuation approach and the assumptions listed in Exhibit 11 in accordance with Selvita's updated R&D strategy. We use risk-adjusted NPV models with a discount rate of 12.5% for R&D projects in various stages. Separately, we use DCF-based calculations with a discount rate of 10% to value the core drug discovery services business and research collaborations.

Financials: Strong organic growth continues; ramping up R&D

Selvita reports in three business segments: Services, Innovation (income from the SEL24 out-licensing was included in this segment) and Bioinformatics. Selvita's commercial revenues include external income from customers allocated to all three segments, while subsidies are allocated to each of the segments. In 9M18, commercial income was PLN56.6m (up 17%, adjusted for the SEL24 out-licensing) and subsidies were PLN20.1m (up 73% y-o-y). Operating profit of -PLN4.7m was somewhat lower than we expected, and we have therefore revised our operating profit estimates from -PLN6.6m to -PLN16.3m for 2018 and from -PLN4.6m to -PLN15.8m for 2019. Selvita is in the capacity expansion phase, which we have included in our model previously, and the current revision affected mainly near-term estimates, with minimal impact on long-term operating margin, which is now 32% (35% previously). Selvita reported cash of PLN153m at end-Q318, which includes proceeds of PLN134m gross from the share issue in Q118. It had PLN4.4m in debt and, according to our model, we do not currently envisage the need for further fund-raises.

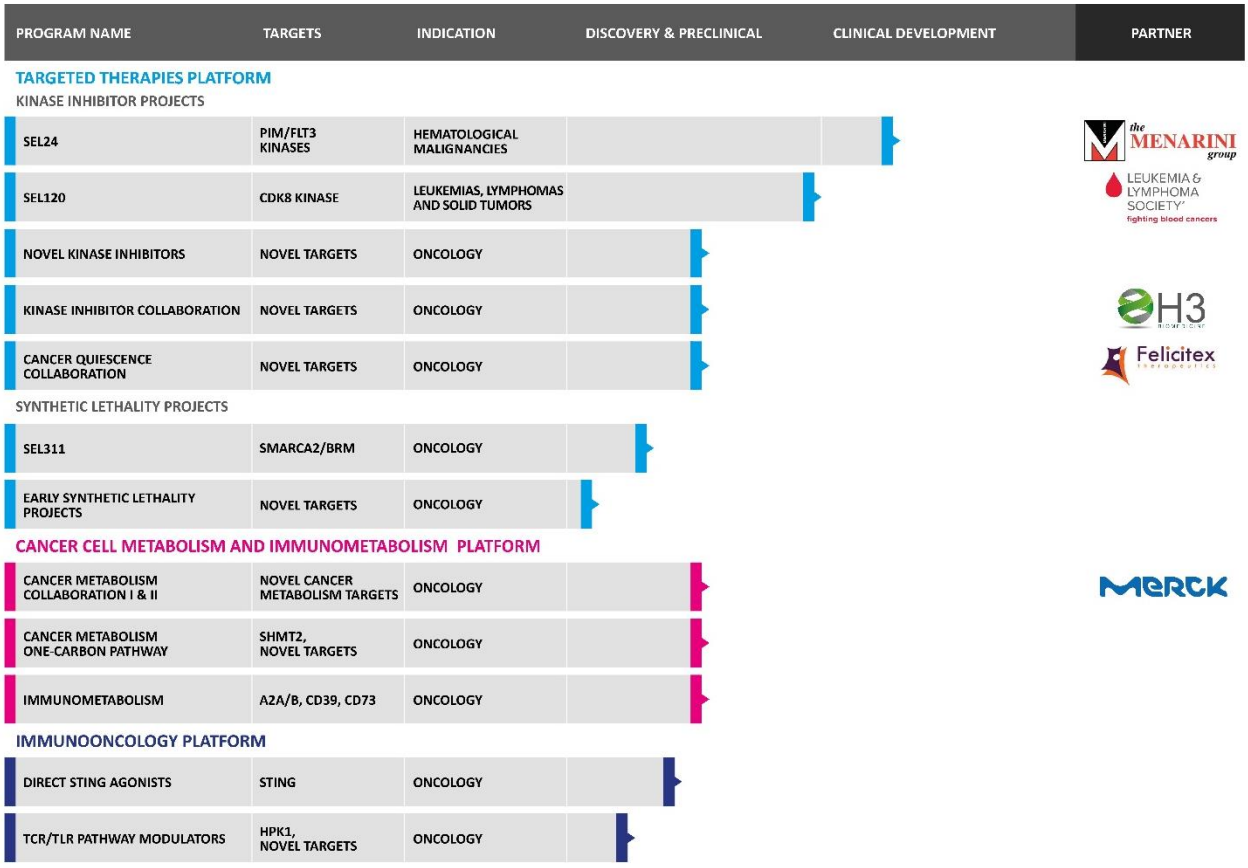
Sensitivities: Service/drug discovery mix lowers risk

Selvita operates a hybrid business model, with a largely de-risked research services business and higher-risk drug development in its innovation platform. Contract research is a highly competitive and increasingly global field, which may put pressure on this side of its business. Since our initiation in November 2014, assets from the innovations pipeline have progressed to the clinical stage, where the results can cause greater volatility in the share price. We have included estimates for deal metrics that Selvita could secure for SEL24 and SEL120, but these are indicative only, so the actual terms secured could differ materially.

Outlook: R&D acceleration ongoing

Selvita's Innovation segment can be described broadly as three discovery platforms focused on small molecules: targeted therapeutics, cancer metabolism and immunometabolism, and immunooncology. The company also expects to build biologic drug discovery capabilities in addition to its existing expertise in small molecules. Currently Selvita continues executing its multi-year strategy following a successful share issue in March 2018 that raised PLN134m. Additional funds from sales, subsidies and loans are expected to form a total budget of c PLN390m, which will help to accelerate the R&D strategy and expansion of other business areas over the next few years.

Exhibit 1: Selvita's R&D pipeline



Source: Selvita

Targeted therapeutics platform

Selvita's targeted therapeutics platform can be broadly divided into two types of projects: kinase inhibitors and synthetic lethality projects. The **kinase inhibitor** platform includes:

- Clinical-stage SEL24 in Phase I for relapsed/refractory AML is out-licensed to Menarini.
- Second lead project, SEL120, partnered with the Leukemia & Lymphoma Society, is undergoing regulatory toxicity studies and the Phase I could start in H119.
- Established in September 2013, the collaboration with H3 Biomedicine (Eisai) involves novel small molecule compounds for new kinase targets associated with cancer development.
- The collaboration with Felicitex Therapeutics was established in November 2014. It focuses on the phenomenon of cancer quiescence, a novel approach with expected efficacy against

quiescent cancer cells that are not actively proliferating and are therefore less susceptible to classical chemotherapy drugs.

The other strategic area of focus in this platform is **synthetic lethality.** One of the disclosed targets is BRM/SMARCA2. A preclinical candidate BRM/SMARCA2 inhibitor is expected to be identified in 2020.

SEL24 – Phase I/II trial readout in 2019

The most advanced asset, SEL24, or MEN1703 under Menarini, a dual inhibitor of PIM and FLT3 kinases, is undergoing a Phase I/II trial in relapsed/refractory AML patients, with results expected in 2019. The asset was out-licensed in March 2017 to Berlin-Chemie (part of the Menarini Group) for an upfront payment of €4.8m, a total of €89.1m in potential milestone payments, and non-specified single- to low double-digit royalties and cost sharing. Initially, Selvita continued to manage the Phase I/II trial, but the handover process was completed in June 2018, and the company's resources and capacity can therefore now be dedicated to other projects in the pipeline. The Phase I/II study is an open-label, dose-escalation study. Part 1 of the trial aimed to establish the recommended dose, which is being evaluated in Part 2 for safety, but also for initial efficacy in several cohorts.

SEL24: Unique mechanism of action

SEL24 specifically inhibits PIM- and FLT3-related pathways and exhibits broader anti-tumour activity in AML compared to selective FLT3-ITD or PIM inhibitors. FMS-like tyrosine kinase receptor-3 gene internal tandem duplication (FLT3-ITD mutation) is one of the most common genetic lesions in AML (around 25% of newly diagnosed AML cases) and, although its inhibition has been shown to be effective in clinical trials, resistance to treatment develops rapidly. PIM kinases are major oncogenes and downstream targets with expression triggered by FLT3-induced STAT5 activity. The expression of PIM kinases amplifies FLT3's oncogenic potential in addition to other pro-oncogenic signalling, thus presenting a rationale for a dual FLT3/PIM inhibition. Previously, third-party data have shown PIM expression increases cancer resistance to FLT3-ITD inhibitors, while PIM inhibition would restore cancer sensitivity to FLT3 inhibitors. Selvita's SEL24, a first-in-class dual inhibitor of PIM and FLT3 kinases, can simultaneously inhibit both kinases and provides a novel treatment strategy.

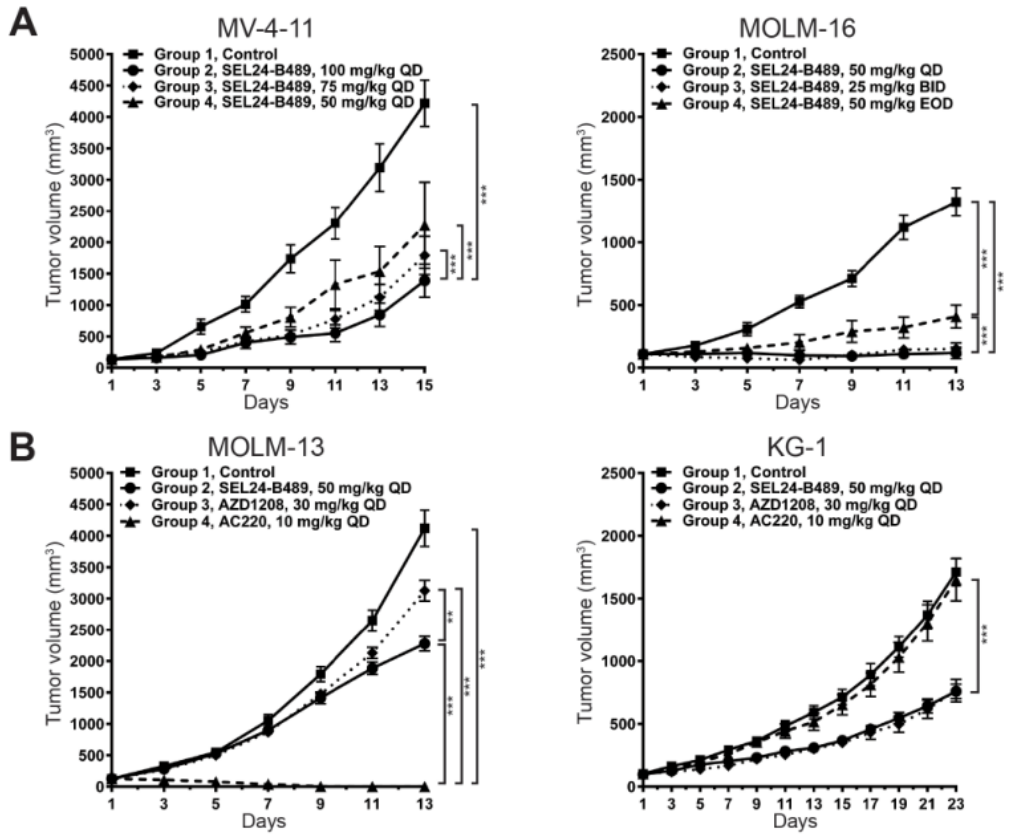
The rationale of SEL24 mechanism of action has been tested by Selvita in various *in vitro* and *in vivo* studies comparing it to control or active treatment with standalone PIM (AZD1208, AstraZeneca) or FLT3-ITD (AC220/quizartinib, Daiichi Sankyo) inhibitors and recently published in an article in Oncotarget:

- **Synergistic effect of dual inhibition**. SEL24 has been described as a potent, dual inhibitor of PIM and FLT3 kinases and has anti-proliferative activity in AML cell lines, which was broader than that of selective PIM inhibitors or FLT3 inhibitors. The combination of AZD1208 and quizartinib also demonstrated a synergistic effect further supporting a dual specificity treatment rationale.
- **Efficacy *in vivo* animal models** (Exhibit 2). SEL24 was tested in several *in vivo* xenograft models.
  - In a single-agent test in mice bearing FLT3-ITD tumours (MV-4-11) a dose dependent reduction in tumour volume by 67-82% has been observed (Exhibit 2A). In the FLT3-WT (wild-type, ie not mutated) model (MOML-16) c 100% tumour growth inhibition was observed at certain doses.
  - To compare the efficacy of SEL24 versus PIM inhibitor AZD1208 and FLT3-ITD inhibitor AC220, the drugs were used in two models – MOLM-13 (FLT3-ITD) and KG-1 (FLT3-WT). SEL24 reduced tumour growth by c 50% in both models. AZD1208 had a variable effect with 20% response in FLT3-ITD and 60% in FLT3-WT. AC220 had no effect in FLT3-WT

mice, but reduced the volume growth in the FLT3-ITD tumour by 100% surpassing the effect of SEL24 (Exhibits 2B, A). Researchers stipulated that this could have been caused by SEL24's relatively short half-life in mice. Previously, other in-house data (not included in the article) showed higher tumour growth inhibition by SEL24 with twice-daily dosing (the current study used a once-daily regime) (Exhibit 2B).

- Of particular note is that SEL24 had a largely similar effect in both the FLT3-WT and FLT3-ITD models (although in the FLT3-ITD model there is the potential to observe even higher efficacy with different dose regimens as explained above). PIM kinases are known to be expressed also in FLT3-WT AML cells. Therefore SEL24 could, theoretically, potentially be used to treat AML patients even with FLT3-WT status.

Exhibit 2: *In vivo* models with SEL24, AZD1208 (PIM inhibitor) and AC220 (FLT3 inhibitor)



Source: W Czardybon et al. MOLM-16, MV-4-11, KG-1 and MOLM-13 cell lines were implanted subcutaneously in immunodeficient mice.

AML still an unmet need; changing treatment paradigm

AML normally originates in the bone marrow (where new blood cells are made), but often quickly moves into the blood, resulting in uncontrolled growth and accumulation of malignant white blood cells, which fail to function normally and interfere with the production of normal blood cells. AML is the most common type of acute leukaemia in adults and affects nearly 40,000 patients in the EU and US (new cases per year). The five-year survival rate for all AML patients, irrespective of age or genetic status, is around 23%. The standard-of-care treatment for AML has not changed significantly for many decades, primarily based on chemotherapy (cytarabine with anthracycline or mitoxantrone) and followed by a stem cell transplant where appropriate. The goal of treatment is to reduce the blasts in the bone marrow to below 5% and return the blood cell counts to normal levels. A bone marrow transplant is generally recognised as the only curative treatment option, but is not always appropriate.

Rydapt (midostaurin, Novartis) was the first approved drug that specifically targets FLT3 for the treatment of adults with newly diagnosed FLT3-ITD AML in combination with standard of care

chemotherapy. This was the first large Phase III RATIFY trial to confirm a therapeutic benefit of FLT-ITD inhibition in AML patients. Overall survival was increased from approximately two years to just over six years and there was a 23% reduction in risk of death compared to the placebo arm (hazard ratio 0.77, p=0.0074). The FDA granted breakthrough therapy designation and a priority review into midostaurin's NDA application and ultimately approved in April 2017. Consensus sales forecast Rydapt sales of $374m in 2024 (EvaluatePharma).

On 28 November 2018, the FDA approved gilteritinib (Xospata, Astellas) as monotherapy for adults with FLT3-positive AML in a relapsed or refractory setting. Approval was based on results of the Phase III ADMIRAL trial. Treatment with gilteritinib resulted in complete remissions (CRs), or CRs with partial haematologic recovery in 21% of patients (95% CI 14.5%–28.8%). Consensus forecast Xospata sales of $451m in 2024.

The latest significant achievement in the FLT3 inhibitor development area was Daiichi Sankyo's results presentation from its Phase III QuANTUM-R study at the European Hematology Association (EHA), on 14-17 June in Stockholm, Sweden. The trial tested quizartinib as a salvage therapy in patients with relapsed/refractory AML with FLT3-ITD mutations after first-line treatment with or without haematopoietic stem cell transplantation. The results demonstrated that quizartinib significantly prolonged overall survival (OS) compared to standard-of-care salvage chemotherapy (27% versus 20% respectively, at week 52). The FDA granted priority review to quizartinib. Consensus sales forecast Rydapt sales of $374m in 2024 (still in the rapid growth phase).

Outside the FLT3 inhibitor space, the FDA approved two other novel drugs – glasdegib (Daurismo, Pfizer) in November 2018 and venetoclax (Venclexta, AbbVie/Roche). Daurismo, a hedgehog pathway inhibitor, was approved for use in combination with low-dose cytarabine for newly diagnosed, frail AML patients (aged 75 years or older and ineligible for intensive chemotherapy). Venclexta, a BCL-2 inhibitor, was approved for a similar group of newly-diagnosed, frail AML patients in combination with azacitidine or decitabine or low-dose cytarabine. Venclexta has received breakthrough designation from the FDA. While both novel drugs add to treatment options for AML, they target a specific group of patients, who are ineligible for more aggressive therapies. Even with this restriction, the consensus sales estimates for Daurismo are at $413m and at $812m for Venclexta in AML by 2024.

SEL120

SEL120 is a first-in-class selective CDK8 inhibitor. Depending on subtypes, cyclin-dependent kinases (CDKs) play varied roles in the control of cell cycle, proliferation and mRNA transcription. Specifically, CDK8 is uniquely differentiated and is a part of a multi-protein complex that regulates gene expression. So far, preclinical studies point to potential efficacy in haematological malignancies, which should be further explored in Phase I. Preclinical efficacy has also been established in solid tumours, such as colorectal cancer or triple-negative breast cancer, and in combination therapies with immunooncology products, which are all potential indications for expansion in later trials. Preparations for the clinical development of SEL120 are ongoing and Selvita plans to file an IND application in Q119, with a Phase I trial following subsequently.

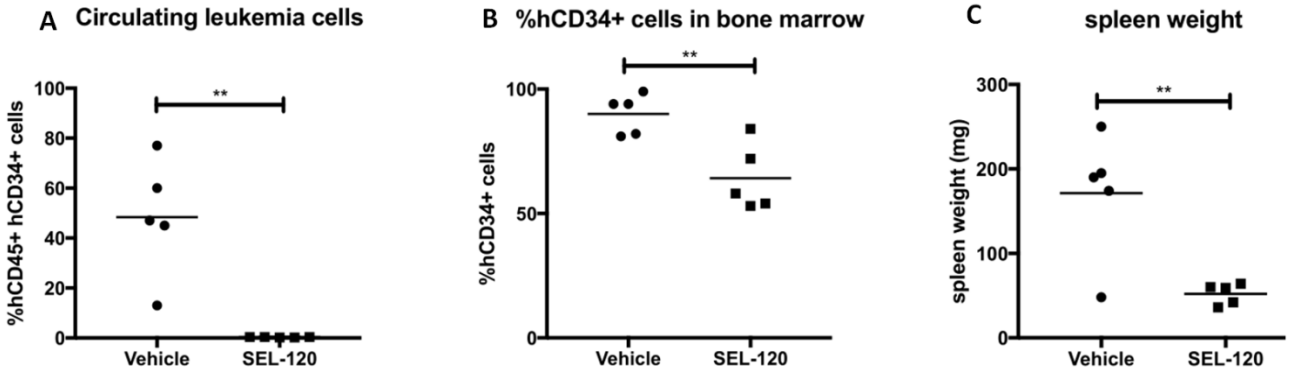
SEL120 development supported by Leukemia & Lymphoma Society

In August 2017, Selvita announced a partnership agreement with the **Leukemia & Lymphoma Society** (LLS) to co-fund further preclinical development of SEL120 for AML patients. The rationale for this indication is that SEL120's unique mechanism of action does not overlap with existing therapies and may allow the development of synergistic combination treatments. According to the deal terms, LLS will provide up to $3.25m in funding over the next four years. This should allow SEL120 to progress though IND-enabling studies through to Phase I in AML. Founded in 1949, LLS is headquartered in the US and has invested over $1bn in various projects so far.

CDK8 inhibitor mechanism of action

This year Selvita presented new preclinical data at multiple conferences, while previous data have been discussed in our past outlook reports. The results of Selvita's collaboration with Lund University were presented during the annual **American Society of Hematology (ASH) meeting** in December 2018, one of the highest-profile research conferences in haematology-oncology. The presentation included *in vivo* results from murine AML model and human patient-derived xenograft (PDX) AML model. In the first instant, the treatment of leukemic mice with SEL120 resulted in reduced leukemia burden in bone marrow and blood. In the PDX model SEL120 completely eliminated circulating primitive CD45+/CD34+ leukemic cells in comparison to control animals (Exhibit 3A). Fewer CD34+ cells were observed in bone marrow in SEL120 treated group than control (Exhibit 3B) and spleen weight was also significantly lower in SEL120 group (spleen is enlarged in AML) (Exhibit 3C). These *in vivo* results demonstrate strong anti-leukaemic activity.

Exhibit 3: SEL120 demonstrated an anti-leukaemic effect in an AML PDX mouse model

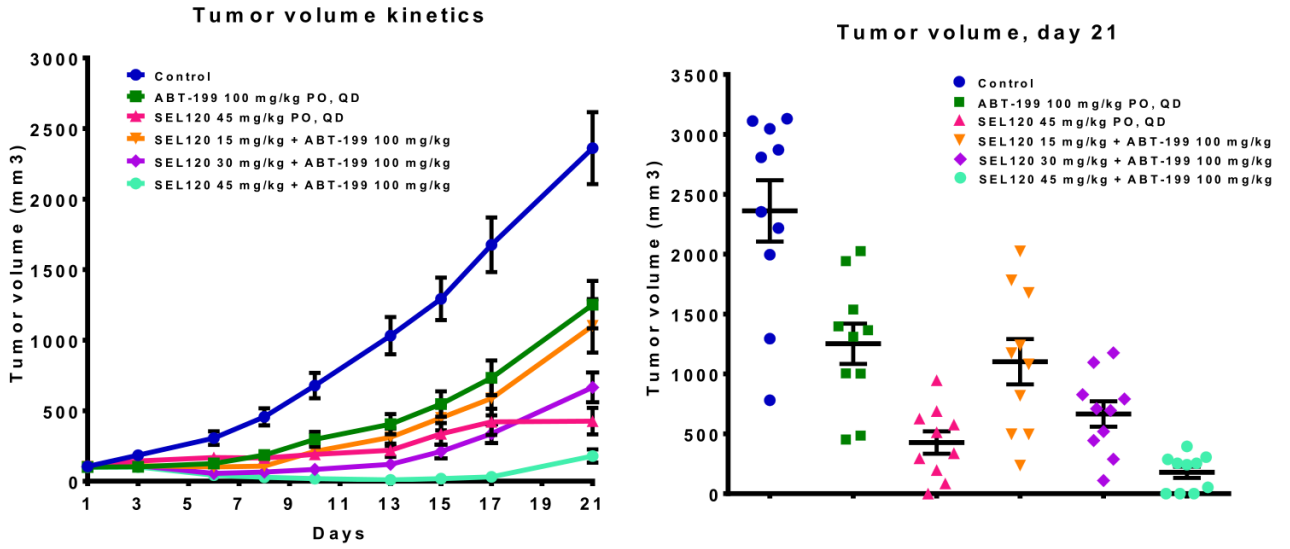


Source: Selvita; M. Chapellier et al, presentation at the ASH annual meeting, December 2018.

New preclinical data was also presented at **the EORTC-NCI-AACR Molecular Targets and Cancer Therapeutics Symposium**, 13–16 November 2018, Dublin, Ireland. The presentation included an overview of the recent preclinical data accumulated with SEL120 in both AML and acute lymphoblastic leukaemia (ALL) as a standalone therapy or in combinations. Some of the highlights include:

- SEL120 was effective with nanomolar activity in cell lines with activated STAT signalling and CD34 expression, which are features of leukaemia stem cells (LSCs). LSCs contribute to AML relapse through treatment-resistant clones. SEL120 induced cell differentiation, ie immature leukaemic cells turn into normal mature blood cells (tested in LSC-like TEX cell line).
- In an *in vivo* of model where immunodeficient mice were injected subcutaneously with AML cell lines, a significant synergistic therapeutic effect has been observed in animals that received both SEL120 and venetoclax (ABT-119) (Exhibit 4). As described in the section entitled ***AML still an unmet need; changing treatment paradigm*** above, venetoclax is now approved as frontline therapy for frail, elderly AML patients.
- *In vitro* results also demonstrated SEL120 is active in acute T-cell lymphoblastic leukaemia. For example, in DND-41 cell line SEL120 demonstrated sub-micromolar activity. These cells have activating Notch pathway mutation, which are present in around 60% of human T-cell ALL cases.

Exhibit 4: SEL120 is active in combination therapy with recently approved for AML venetoclax (ABT-199)



Source: Selvita; M. Mazan et al, presentation at the EORTC-NCI-AACR, November 2018. Note: cell line-derived mouse model (BALB/c Nude + MV4-11)

Selvita will continue testing SEL120 in preclinical proof of concept *in vivo* models to better inform clinical efficacy trials with humans. In plans are tests whether SEL120 treatment extends survival of mice with AML in syngeneic and patient-derived xenograft models. One of the more notable news in the CDK8 inhibitor area was Merck & Co licensing agreement for a selective CDK8/CDK19 inhibitor from Harvard University in March 2016. The deal included an upfront payment of $20m and tiered royalties, which was the largest licence fee for technology developed at the university. The agreement involved compounds derived from a natural compound, cortistatin A, and recently published articles showing anti-leukaemic *in vitro* and *in vivo* efficacy, which adds to Selvita's preclinical data.

SMARCA2/SMARCA4 and synthetic lethality

Primary indication for synthetic lethality projects are cancers bearing recurrent mutations in the SWI/SNF complex. The so called switch sucrose nonfermentable (SWI/SNF) complex is a large complex of proteins involved in chromatin (compact 'strand' of the DNA) remodelling.[1] SMARCA2 (BRM) and SMARCA4 (BRG1) are two catalytic subunits essential for the function of the complex. An increasing number of cancers have been found to have inactivating mutations in the SMARCA4/ SMARCA2 genes. In malignant cells with the mutated SMARCA4 gene, non-mutated SMARCA2 becomes essential.[2] Therefore the inhibition of SMARCA2 causes cell death if there is an oncogenic mutation in the SMARCA4 gene. This concept of a 'biological genetic flaw' complemented by an intervention with a drug, which results in cell death, is known as synthetic lethality. This approach is very specific and potentially offers better safety profile.

Selvita is developing a first-in-class, selective SMARCA2 small molecule inhibitor, has established *in vitro* proof-of-concept, has identified hit compounds and is currently in a hit-to lead optimisation process. The company believes that the advantage of its SMARCA2 inhibitor programme is that it targets ATPase domain of SMARCA2, as opposed to bromodomain, which is the target of other

1 Herpel et al. SMARCA4 and SMARCA2 deficiency in non–small cell lung cancer: immunohistochemical survey of 316 consecutive specimens. *Annals of Diagnostic Pathology 26 (2017) 47–51.*

2 Wilson et al. Residual Complexes Containing SMARCA2 (BRM) Underlie the Oncogenic Drive of SMARCA4 (BRG1) Mutation. *Molecular and Cellular Biology p. 1136–1144.*

SMARCA2 inhibitors. For example, both bromodomain-targeting agents from Pfizer (PFI-3) and from Genentech failed to inhibit cell growth *in vitro* studies.[3]

When it comes to market opportunity, Selvita has indicated that the non-small cell lung cancer (NSCLC) patient population could be the first clearly defined target as SMARCA4 is mutated in around 6-8% of cases. This would correspond to more than 10,000 new patients in the US alone. Other solid tumours with inactivated SMARCA4 have also been identified. There is also the likelihood that SMARCA2 inhibition would show efficacy in tumours with mutations in other proteins from the SWI/SNF complex, which is 20% of all tumours, thus significantly expanding the market potential. In NSCLC the top 10 drugs, which include Keytruda (pembrolizumab, Merck & Co), Opdivo (nivolumab, Bristol-Myers Squibb) and Avastin (bevacizumab, Roche) among others, are expected by the consensus to bring in sales of $21.6bn in 2022. The significant upside is, however, in other indications as discussed.

Cancer metabolism and immunometabolism

The cancer metabolism and immunometabolism platform focuses on deregulated cancer cell metabolism due to oncogenic tumour mutations or the effect of small molecule metabolites on tumour microenvironment. Major disclosed targets from this platform are SHMT2 (serine catabolism) and A2A/A2B, CD73/CD39 (both with a role in adenosine immunosuppression). Selvita also has long-standing drug discovery collaboration with Merck KGaA within this platform.

A2A/A2B antagonists

Natural molecule adenosine is a key element in immune regulation and many cancers have the ability to accumulate it, which allows them to escape detection by the immune system. Antagonising adenosine receptors (A2A/A2B) or inhibiting the enzymes of the adenosine synthesis pathway (CD39/CD73, another project in Selvita's portfolio) was shown to restore the adenosine-suppressed anti-tumour response of the immune system. Selvita has discovered novel A2A/A2B receptor antagonists with activity in picomolar concentrations, which makes them the most active compound known currently, according to the company. With its Q318 update Selvita indicated that pilot *in vivo* studies had been completed (data yet to be published), demonstrating that its A2A/A2B antagonist inhibited tumour growth in a dose-dependent manner and increased infiltration of immune cells (eg CD8+ T-cells). Earlier projects in this area include inhibitors of enzymes of extracellular adenosine synthesis pathway (CD39 and CD73).

While this type of technology is still mostly in early clinical development, over the past three years there have been multiple deals in the industry with values reaching $500m as large pharma in-license assets targeting this pathway. Earlier in 2018 Arcus Biosciences, a US-based biotech company, underwent a successful IPO, raising $120m. The company is running Phase I/II trials with its lead drug candidates AB928, a dual adenosine receptor antagonist, and AB122, a PD-1 antibody, and has an option agreement with Taiho for regional rights in Asia (excluding China), according to which Arcus will receive $35m over three years and could get $275m for each drug programme that Taiho in-licenses. Further news in this area came from a private Belgian biotech iTeos Therapeutics (A2A antagonist), which raised €75m from a consortium of investors in June 2018.

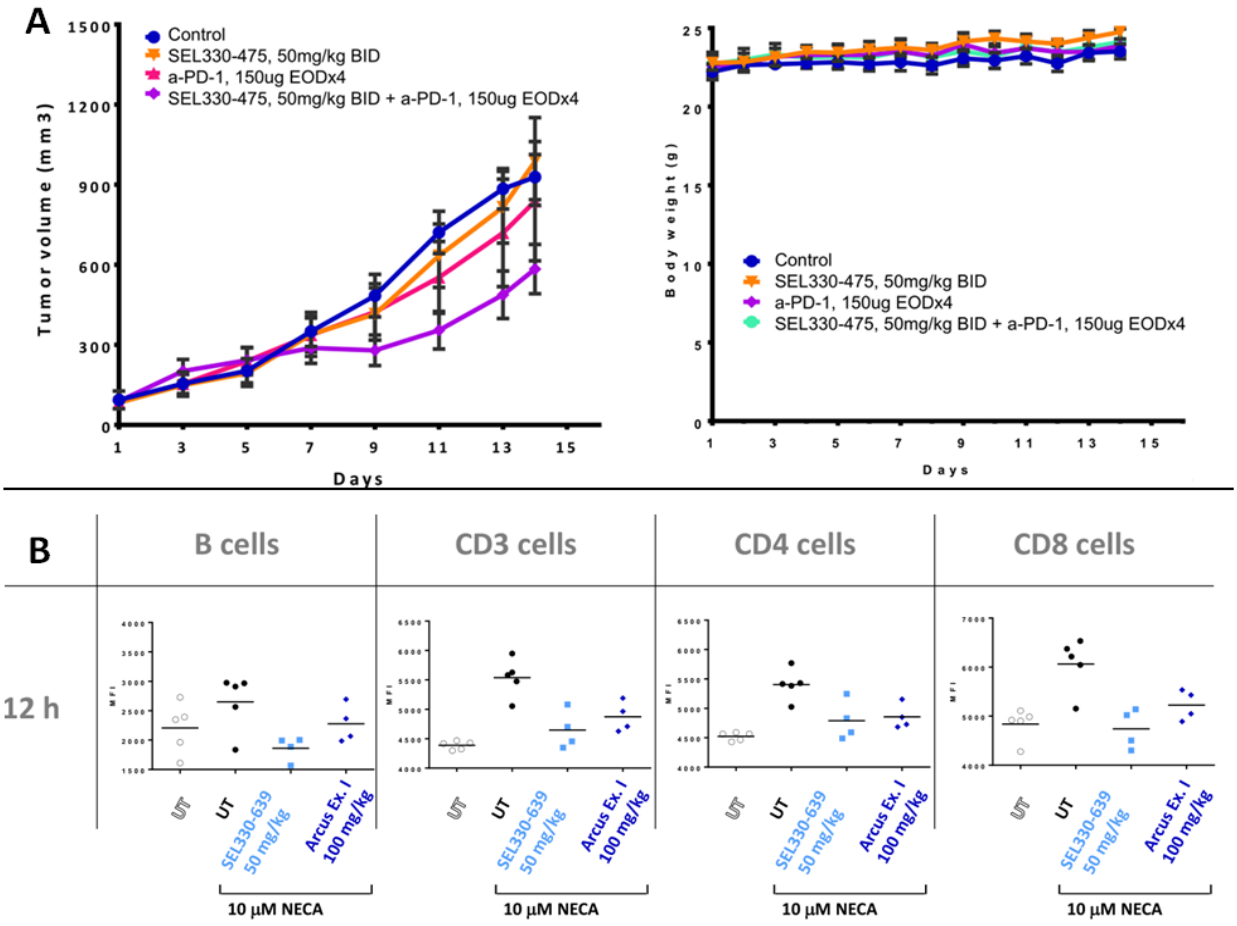
Selvita's asset (SEL330) stands out, in our view, because of its dual A2A/A2B inhibition activity, while other technologies in the area mainly are selective A2A inhibitors. Selvita believes the

3 B. Vangamudi. The SMARCA2/4 ATPase Domain Surpasses the Bromodomain as a Drug Target in SWI/SNF-Mutant Cancers: Insights from cDNA Rescue and PFI-3 Inhibitor Studies. *Cancer Res. 2015 Sep 15;75(18):3865-3878.*

relevant target populations could be broad, with the immunosuppressive environment prevalent in around 50% of all cancers. There is also a strong rationale for synergistic potential with checkpoint inhibitors, therefore Selvita is conducting various preclinical studies to establish which combinations would be optimal for the clinical trials. Selvita's poster presentations at the AACR meeting in April and at the Society for Immunotherapy of Cancer (SITC) meeting in November 2018 included data describing the discovery process and some *in vivo* data. Main conclusions include:

- SEL330 demonstrated dose-dependent activity to restore the adenosine agonist-impaired functionality of CD4+ and CD3+ human T-lymphocytes and rescue adenosine-suppressed cytotoxicity of NK cells. This indicates the potential to restore the anti-tumour response of the patient's immune system.
- In a surrogate *in vivo* assay SEL330 effect was compared against the dual A2A/A2B inhibitor from Arcus Biosciences (Exhibit 5B). NECA, which is a potent pan-adenosine agonist, was used to stimulate adenosine receptors in mice model in such a way, simulating a high adenosine environment, which can be measured by biomarker CREB (transcription factor) phosphorylation. Selvita's and Arcus's antagonists were administered to mice after NECA and CREB phosphorylation inhibition in different cells was consistently better in SEL330 arms than in Arcus.
- SEL330 demonstrated synergistic activity with PD-1 inhibitor in syngeneic mouse cancer model (Exhibit 5A).

Exhibit 5: SEL330 is synergistic with anti-PD-1 in mouse models and versus competitor in CREB phosphorylation (*in vivo*)

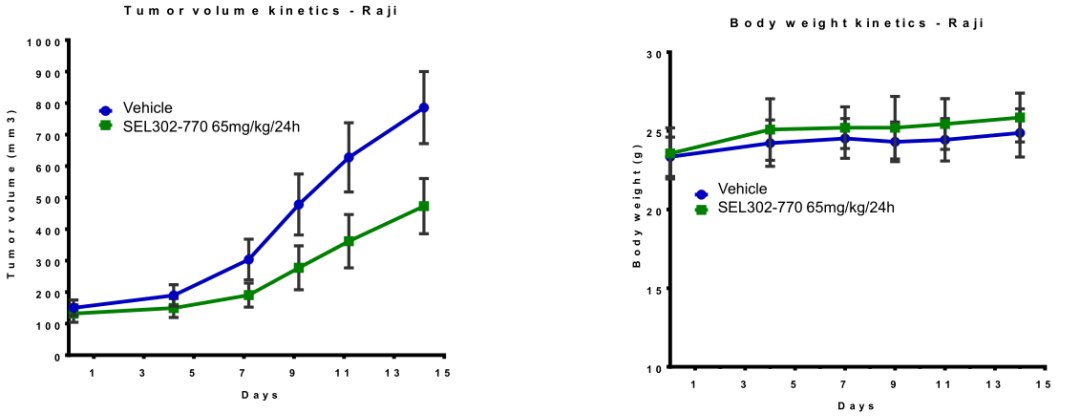


Source: Selvita poster presentations. P. Węgrzyn, M. Gałęzowski et al., AACR meeting, April 2018; STIC meeting, November 2018. NECA – potent pan-adenosine agonist. Arcus Example I – dual A2A/A2B inhibitor from Arcus Biosciences. UT – untreated.

SHMT2 inhibitor

Over-activation of the serine synthesis pathway and upregulation of Serine hydroxymethyltransferase (SHMT) makes cancer cells highly dependent on serine and has been described in over 20% of solid tumours. In cellular models, Selvita's SHMT2 inhibitor SEL302 has shown specificity and efficacy at nanomolar levels. In the latest update together with Q318 results, the company indicated that the first promising *in vivo* proof-of-concept studies have been completed in oncological and non-oncological models. Research results were summarised during the recent poster presentations at the conference Mechanisms to Therapies: Innovations in Cancer Metabolism, 9–11 October 2018, Bilbao, Spain.

Exhibit 6: SHMT1/2 targeting in non-Hodgkin lymphoma model

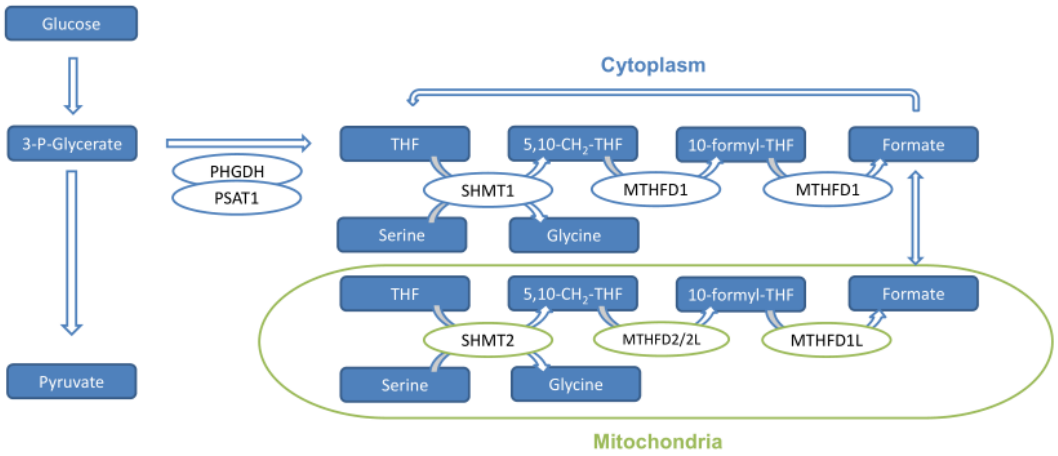


Source: A Bartosik et al. Discovery of novel SHMT1/2 small molecule inhibitors for cancer treatment. Poster at 30th EORTC-NCI-AACR Molecular Targets and Cancer Therapeutics Symposium. November 2018.

Serine hydroxymethyltransferase (SHMT) plays a key role in a so-called one-carbon pathway, a group of biochemical reactions involved in amino acid metabolism (Exhibit 7). SHMT catalyses the conversion of serine to glycine and also plays a role in the folate (vitamin B9) cycle. Selvita's research focuses on the discovery of specific inhibitors of SHMT2, which is located in mitochondria (SHMT1 is found in cytoplasm). Over-activation of the serine synthesis pathway and upregulation of SHMT has been described in over 20% of solid tumours (eg breast, lung, colorectal cancers). Such cancer cells are highly dependent on serine.[4] The target population for SHMT inhibition can be well defined by using oncogene c-Myc as a biomarker. c-Myc protein is a transcription factor involved in numerous cell process and c-Myc positive tumours have been correlated with poor prognosis.

4 Ch. F. Labuschagne et al. Serine, but not glycine, supports one-carbon metabolism and proliferation of cancer cells. *Cell Reports, Volume 7, Issue 4, p1248–1258, 22 May 2014.*

Exhibit 7: Serine synthesis and one-carbon pathway



Source: T. Rzymski et al. Small molecule inhibitors of SHMT1/2 validate serine metabolism as a target in the treatment of c-Myc positive solid tumours. Poster presentation, AACR meeting, 2017.

As mentioned above, SHMT is also important in the folate cycle. Antagonists of folate metabolism or antifolates are an established chemotherapy in certain cancers. Folate antagonism disrupts cell division, DNA/RNA synthesis and protein synthesis. Pemetrexed (for non-small cell lung carcinoma, mesothelioma) and methotreaxate (for autoimmune conditions like rheumatoid arthritis and certain cancers) are two well established and effective antifolates. The main drawback with antifolates in cancer treatment, however, is the development of resistance. Therefore, Selvita sees potential synergism of SHMT inhibitor in combination with antifolates.

Immunooncology platform

The immunooncology platform aims to provide novel immunotherapies mobilizing the immune system to attack tumours. This approach transforms 'cold' or resistant tumours into 'hot'. The disclosed lead programme in this platform is **STING pathway modulators**. More recently, Selvita introduced a **HPK1 kinase inhibitor** project. HPK1 (MAP4K1) is one of the major proteins involved in the signalling cascade triggered by T-cell receptor (TCR) activation. Inhibition of HPK1 stimulates dendritic cells to antigen presentation and enhances the activation and proliferation of T-cells, which leads to an immune response directed against the cancerous cells.

STING inhibitors

The most advanced immunooncology project in this platform is STING (stimulator of interferon genes) pathway modulators. A STING receptor is a known mediator of the immune system, which when activated induces expression of type I interferon and other T-cell recruitment factors. This results in the activation of dendritic cells, which act as antigen presenting cells. The ultimate outcome is the specific immune response with 'trained' CD8+ T-cells attacking the cancer. The strategic opportunity for STING agonists could be patients not responding to checkpoint inhibitors (CPIs), but also there is potential for use in combination with CPIs. The strong rationale for combinations is based on the fact that CPIs act late in the immunity cycle (makes the tumour 'visible' to T-cells), while the STING pathway appears to prime the production of cancer-specific T-cells, so both technologies are potentially synergistic. Selvita identified a potentially first-in-class small molecule of the STING agonist, a direct protein binder. This unique structure and optimised ADME properties distinguish Selvita's compounds from the competitors that develop derivatives of nucleic acid which, due to their chemical nature, can mainly be used for inconvenient intratumoural

injections. Selvita is optimising the lead series of molecules and has conducted initial *in vivo* proof-of-concept studies.

Nodthera

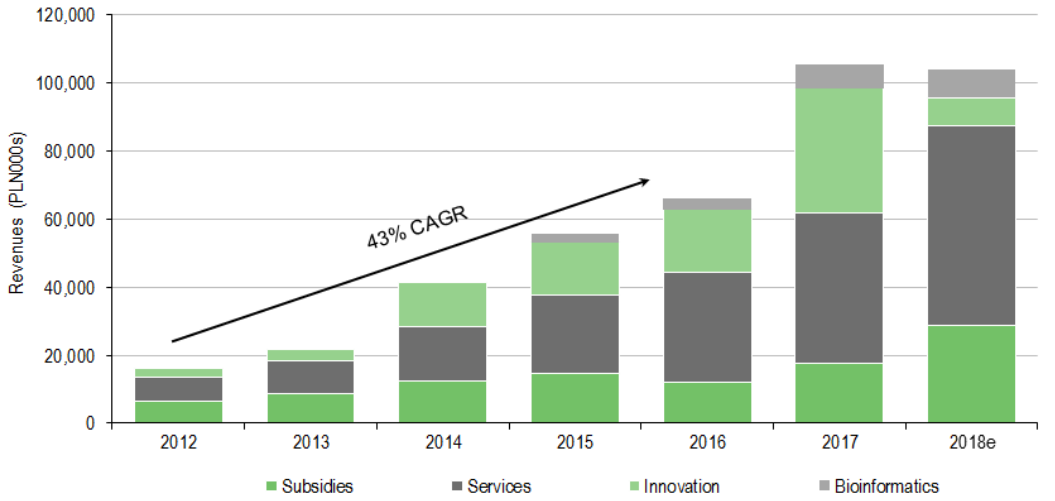
A project in the inflammation field, NLRP3 inflammasome inhibitors, was spun out to NodThera and seeded together with Epidarex Capital in 2016 (detailed overview). In June 2018, NodThera announced a £28m fund-raise from a consortium of investors co-led by Sofinnova Partners and 5AM Ventures with participation from Epidarex Capital and F-Prime Capital Partners. Selvita's last reported ownership is 18.4% on a fully diluted basis. According to the recent newsflow, NodThera is now expanding its offices to Boston, MA and has hired an experienced CEO Adam Keeney, PhD. Dr Keeney has over 20 years' experience in the pharmaceutical industry and joined NodThera from Sanofi Genzyme, where he served as global head of Sanofi Genzyme business development.

NodThera centres on NLRP3 inflammasome inhibitors, a first-in-class technology, based on the scientific programme originated and developed at Selvita since 2012. Inflammasomes have been identified as the molecular mechanism behind the activation cascade of interleukin (IL)-1. IL-1 is a family of pro-inflammatory cytokines that have been widely implicated in pain, inflammation and autoimmune conditions and more recently in cancer. Therefore, NodThera's focus is currently rather broad as the asset is in a preclinical stage. This will be narrowed down once past the proof-of-concept clinical trials, for which the new funds should be sufficient, according to the company.

Drug discovery services and bioinformatics

Since incorporation Selvita has seen rapid growth of its drug discovery business: 43% CAGR 2012-2016, while income in 2017 was boosted by the outlicensing of SEL24. We expect the **Services business**s to continue on a high-growth revenue trajectory, but in our view the **Innovation segment** offers greater long-term potential upside, which could come from the development and licensing of a number of candidates from the diverse internal pipeline. **Ardigen** is recognized as a third business segment. The bioinformatics company was spun out in October 2015 (currently Selvita holds 52% of shares). The logic behind its spin out was to increase the range of services within the bioinformatics and IT solutions offering, but also to expand into new areas rapidly evolving with the global precision medicine trends. Ardigen now has stable existing business, which provides revenues and can explore expansion opportunities.

Exhibit 8: Selvita's growth



Source: Edison Investment Research, Selvita

Going forward we find both macro trends and company specific characteristics as beneficial for Selvita to maintain the rapid organic growth. Main macro trends that support outsourcing in drug discovery include:

- **Efficiency, expertise of the provider, access to novel technologies**. A specialised service provider accumulates expertise in specific research stages and becomes expert in certain therapeutic areas. To maintain the competitive advantage, the provider has to employ cutting-edge technologies, which may not be owned by outsourcing companies. Established providers can also attract top talent in the industry.
- **Need for R&D cost reduction and increasing comfort with outsourcing**. The need to control rising costs is a compelling incentive to look for ways of outsourcing at least part of the R&D activities. This has become especially acute with the decreasing efficiency of the in-house R&D efforts at large pharma companies. Biotech and pharma started outsourcing a range of different activities with varying degrees of complexity and, with increasing complexity, the quality of the service provider becomes crucial.
- **Flexibility with different outsourcing models**, which range from typical fee-for-service contracts (eg outsourcing of synthesis of a library of compounds) to fully integrated drug discovery projects, through different FTE-based partnership structures involving research fees, milestones and royalties (margins increase accordingly).
- **Scalability**. Outsourcing allows for fixed costs to be converted to variable costs. The extent of the work can be rapidly increased or decreased, while accomplishing this in-house would involve hiring, reassigning or laying off personnel. This is especially true when the outsourcing company is of smaller scale or so called 'virtual', in which case all the R&D activities are outsourced.

Company-specific trends: Growing faster than the market

Selvita's Services division operates out of a research facility in Krakow and Poznan, Poland. The division is split into two main units – Chemistry and Biology – each run as separate businesses within the group. Selvita's client list for its services is extensive, ranging from big pharma, to generics, to biotech companies. A range of contract options are offered – fixed price, to FTE, to integrated – which generate increasing revenues, costs and complexity. Fixed price contracts normally involve fierce price competition from lower-cost Asian countries, while FTE contracts and integrated projects are more of a value proposition and compete with Western drug discovery services providers such as Evotec, Charles River Laboratories and Covance. In line with its

strategy of moving away from fixed-priced contracts, Selvita has been increasing the percentage of FTE contracts signed. One of Selvita's significant competitive edges is its lower cost base, while at the same time the country's large population means that there should be enough MSc and PhD life sciences professionals to join the company during the expansion stage.

Financials

Selvita's reported total 9M18 revenues of PLN77.2m (-4% y-o-y) were largely in line with our expectations. The slight decline y-o-y is a result of a large income recognised in Q117 after the company out-licensed SEL24 to Menarini Group. Selvita reports in three business segments: Services, Innovation (income from SEL24 was included in this segment) and Bioinformatics. Selvita's commercial revenues include external income from customers allocated to all three segments, while subsidies are allocated to each of the segments. Total commercial 9M18 revenues, which exclude subsidies and the out-licensing upfront, were PLN56.6m (up 17% y-o-y) and subsidies were PLN20.1m (up 73% y-o-y).

- The **Services segment** generated sales of PLN43.0m (excluding subsidies) in 9M18, an increase of 41% y-o-y, reflecting continuing solid organic growth.
- Commercial revenues from the **Innovation segment** were PLN8.0m in 9M18 vs PLN32.7m in 9M17, with the out-licensing of SEL24 to the Menarini Group in March 2017 being the main reason for the strong performance in 2017. Commercial revenues from this segment came from payments related to different partnerships, such as milestone payments from drug discovery collaborations, and therefore tend to be volatile from quarter to quarter, but offer potentially higher margins. In the longer term, the Innovation segment includes Selvita's own R&D pipeline activities, so developing it is a strategic goal that will be supported by substantial investments, according to plans.
- The **Bioinformatics (Ardigen)** segment recorded income of PLN5.7m versus PLN5.3m a year ago. This business was spun out from Selvita (which currently holds 52% of the shares) in October 2015 and FY17 sales were PLN6.9m (a 101% y-o-y increase), so while still a small operation for Selvita, the growth rate is encouraging.

We have made some modest revisions to our revenue estimates as shown in Exhibit 9 (overall 9M18 revenues were close to our expectations), with a small positive effect on our top-line forecasts for 2018. Operating profit of -PLN4.7m was below our expectations, but in line with Selvita guidance that the main focus following the successful fund-raise earlier in 2018 is creating value through R&D research, and R&D spending should therefore pick up. We have therefore revised our operating profit estimates from -PLN6.6m to -PLN16.3m for 2018 and from -PLN4.6m to -PLN15.8m for 2019. Selvita is in a capacity expansion phase, which we have included in our model previously, and the current revision affected mainly near-term estimates. One of the more notable cost drivers in 2019 will be the Phase I trial with SEL120, the results of which also represent a catalyst for the share price. Our projected long-term operating margin is c 32% (35% previously).

Selvita reported cash of PLN153m, which includes proceeds of PLN134m gross from the share issue in Q118. It had PLN4.4m in debt and, according to our model, we do not envisage the need for further fund-raises to run the existing R&D programmes we include in our model. Our financial forecasts do not include any subsequent milestone payments from Menarini Group.

Exhibit 9: Key changes to our financial forecasts and introduction of FY19 forecasts

PLN000s	FY17	FY18e			FY19e		
	Actual	Old	New	Change (%)	Old	New	Change (%)
Revenue	105,872	101,294	104,700	+3%	116,816	119,905	+3%
--- *Services*	44,208	55,260	58,360	+6%	67,417	67,417	+0%
--- *Innovation*	36,727	8,000	8,400	+5%	22,207	22,207	+0%
--- *Bioinformatics*	6,885	7,574	8,400	+11%	8,331	9,240	+11%
--- *Subsidies*	17,591	30,000	28,900	-4%	18,400	20,400	+11%
Operating profit/loss (norm)	13,222	(6,554)	(16,251)	+148%	(4,580)	(15,824)	+246%
Profit/loss before tax (norm)*	10,183	(6,536)	(14,905)	+128%	(4,505)	(15,782)	+250%
Profit/loss after tax (norm)*	7,315	(7,249)	(19,060)	+163%	(4,190)	(15,782)	+277%
EPS (norm) (PLN)*	0.51	(0.49)	(1.28)	+163%	(0.26)	(0.99)	+277%

Source: Selvita accounts, Edison Investment Research. Profit before tax, net profit and EPS are adjusted for the change in accounting method of Nodthera's shares from equity method to fair value (PLN20.8m booked in 2018).

Valuation

Our valuation of Selvita is slightly lower at PLN1.24bn or PLN77.6/share versus PLN1.30bn or PLN81.2/share previously. The revision of the operating estimates resulted in a slightly lower DCF valuation, while rolling the model forward had a small positive effect on the rNPV values of the R&D projects. We maintain our valuation approach and assumptions listed in Exhibit 10 and discussed in detail in our last outlook report where we revised our R&D model in accordance with Selvita's updated R&D plans. We use risk-adjusted NPV models with a discount rate of 12.5% for Selvita's R&D projects in various stages. Separately, we use DCF-based calculations with a discount rate of 10% to value the core drug discovery services business and research collaborations.

Exhibit 10: Sum-of-the-parts Selvita valuation

Product	Launch	Peak sales ($m)	NPV (PLNm)	NPV/share (PLN)	Probability	rNPV (PLNm)	rNPV/share (PLN)
Innovation							
SEL24	2023	750	744.5	46.6	15%	147.4	9.2
SEL120	2025	1,500	1,563.7	97.9	10%	181.7	11.4
SMARCA2 inhibitor	2030	1,000	708.1	44.3	2%	114.7	7.2
A2A/A2B antagonist	2030	1,000	786.9	49.3	2%	109.9	6.9
SHMT2 inhibitor	2031	1,000	479.2	30.0	2%	76.8	4.8
Merck collaborations	2026	2,000	50.7	3.2	5%	8.3	0.5
Services (including Ardigen)	Market			DCF (Q119-2027)	100%	82.7	5.2
				Terminal value	100%	368.4	23.1
Net cash (end-Q318)					100%	148.6	9.3
Valuation			**4,333.1**	**271.3**		**1,238.8**	**77.6**

Source: Edison Investment Research. Note: WACC = 12.5% for product valuations, WACC = 10% for Services segment.

Exhibit 11: Assumptions for R&D projects and services business

Product/stage/ indication	Comments
Targeted therapeutics platform	
SEL24 *- Phase I/II* *- r/r AML*	Market potential: $750m indicative peak sales. AML treatment currently dominated largely by traditional chemotherapy drugs. Two novel drugs were approved in 2017 and the consensus expects rapid market growth to $2.9bn in 2022 as more novel drugs are expected to be approved in coming years. R&D costs and timelines: according to the agreement, Menarini is due to take over the development by the end of 2017 and Selvita will not incur any R&D costs afterwards. We assume launch in 2023 with peak sales reached in six years. Licensing terms: Menarini deal terms include an upfront payment of €4.8m, a total of €89.1m in potential milestone payments and non-specified single- to low-double-digit royalties (we assume up to 10%). Menarini took over the trial in June 2018. Market protection: until mid-to-late 2030.
SEL120 *- IND studies* *- Cancer*	Market potential: $1.5bn indicative peak sales. While SEL120 (CDK8 inhibitor) is not directly comparable to CKD4/6 inhibitors, we nevertheless view the strong the strong performance of the first CDK4/6 inhibitors (Ibrance, Kisqali and Verzenio) as indicative of a large potential within the CDK family, especially since they can be developed for a variety of solid and haematological cancers. R&D costs and timelines: $19m in R&D costs for Selvita (partly funded by LLS, see below) to develop the drug to Phase II in 2020. Then out-licensing. The partner continues the development and launches in 2027 with peak sales reached in six years. Licensing terms: According to the deal terms, LLS will provide up to $3.25m in funding over the next four years. This should allow Selvita to progress SEL120 through IND-enabling studies to Phase I in AML. In Phase II (2020) we assume a licensing deal with terms similar to Novartis/Astex deal. Upfront was undisclosed, we use 5% of the milestone value $26m. We assume up to 15% royalty rates. Market protection: until mid-to-late 2030.
BRM/SMARCA2 *- Identification of lead compounds* *- Various cancer, primary indication could be NSCLC*	Market potential: Selvita has identified NSCLC with mutated SMARCA4 as clear initial target. Average sales of the top 10 drugs in NSCLC are estimated to reach $2.16bn. The target population of NSCLC is only around 8% with SMARCA4 mutations; however, there is potential that SMARCA2 inhibition would show efficacy in tumours with mutations in other proteins from SWI/SNF complex, which is 20% of all tumours thus significantly expanding the market potential. To account for the overall potential, but erring on the conservative side we use an assumption of $1bn in peak sales for Selvita's SMARCA2 inhibitor. R&D costs and timelines: Selvita indicated that a clinical candidate could be identified in 2019, we therefore assume Phase I to start in 2021 and an out-licensing deal the same year, as the company indicated that an early partnership will be needed. R&D cost of $3m to reach Phase I. The partner continues the development and launches in 2030 with peak sales reached in six years. Licensing terms: $40m upfront and $485m in milestones split over clinical development and commercialisation. We use AbbVie/Dong-A ST deal as a benchmark from. We assume 7–10% royalty rates. Market protection: we assume market protection until late 2030.
Cancer metabolism and immunometabolism platform	
Merck deals #1 and #2 *- Preclinical* *- cancer*	Technology remains undisclosed. Assume two projects in Phase I in 2020 with launch in 2028 and peak sales of $1bn in each project in 2034. Licensing fee €0.2m; total milestone payments could add up to €16.5m in each deal. Royalties have not been disclosed, we assume up to 2%.
A2A/A2B antagonist *- Identification and optimization of lead compound* *- Various cancers*	Market potential: Selvita believes the relevant target populations could be broad with the immunosuppressive environment prevalent in around 50% of all cancers. There is also a strong rationale for synergistic potential with checkpoint inhibitors. Because checkpoint inhibitors have gained widespread recognition, but suffer from non-responder issues, combination treatments are likely the future of immunooncology. Because of the lack of detail about potential specific indications, but to reflect the broad potential, we use $1bn as our peak sales assumption. R&D costs and timelines: We assume that a clinical candidate could be identified in 2018 and partnered in 2019. We assume an R&D cost of $2m for Selvita to develop the asset to partnership deal. Phase I to start in 2021 and the partner continues the development and launches in 2030 with peak sales reached in six years. Licensing terms: We use the Heptares/AstraZeneca deal as a benchmark. $10m upfront and $500m in milestones split over clinical development and commercialisation. We assume 7-10% royalty rates. Market protection: we assume market protection until late 2030.
SHMT2 inhibitor *- Identification and optimization of lead compound* *- Various cancers*	Market potential: Overactivation of serine synthesis pathway and upregulation of SHMT has been described in over 20% of solid tumours therefore, while it is still not clear what could be primary indications, the potential seems wide. As in the case of A2A/A2B antagonist, we assume $1bn in peak sales. R&D costs and timelines: We assume that a clinical candidate could be identified in 2018/19 and partnered in 2020. We assume the R&D cost for Selvita to develop the asset to partnership deal is $3m. Phase I to start in 2022 and the partner continues the development and launches in 2031 with peak sales reached in six years. Licensing terms: We use the value of Agios/Celgene deals involving three separate products, but all focusing on isocitrate dehydrogenase mutant (IDH) mutant inhibitors. $10m upfront and $310m in milestone payments split over clinical development and commercialisation. We assume 7-10% royalty rates. Market protection: we assume market protection until late 2030.
Drug discovery services collaborations	Services: sliding scale pa growth from c 25% in 2018 to 12% in 2027; research collaborations: +10-5% pa growth to 2027; subsidies: +5-3% pa growth; tax = 2-11% sliding scale (2018-27); 10% WACC. For terminal value calculation we use 0.75% growth on 2027 FCF.

Source: Edison Investment Research.

Exhibit 12: Financial summary

PLN'000s	2016	2017	2018e	2019e
Year end 31 December	IFRS	IFRS	IFRS	IFRS
PROFIT & LOSS				
Revenue	66,721	105,872	104,700	119,905
of which: Services (research outsourcing)	32,404	44,208	58,360	67,417
Innovation platform	18,353	36,727	8,400	22,207
Subsidies	12,067	17,591	28,900	20,400
Bioinformatics	3,431	6,885	8,400	9,240
EBITDA	8,264	18,462	(8,751)	(11,376)
Operating Profit (before amort. and except.)	4,646	13,222	(16,251)	(15,824)
Intangible Amortisation	0	0	0	0
Exceptionals/Other*	(5,860)	(583)	0	0
Operating Profit	(1,214)	12,639	(16,251)	(15,824)
Net Interest	947	(1,956)	1,346	46
Share in profit/(loss) of assocs. and JVs**	(1,016)	(1,082)	20,787	0
Other	0	0	0	0
Profit Before Tax (norm)	4,577	10,183	5,883	(15,778)
Profit Before Tax (reported)	(1,283)	9,600	5,883	(15,778)
Tax	0	(345)	(4,155)	0
Deferred tax	3,968	(2,523)	0	0
Profit After Tax (norm)	8,545	7,315	1,727	(15,778)
Profit After Tax (reported)	2,685	6,732	1,727	(15,778)
Average Number of Shares Outstanding (m)	13.4	13.8	14.9	16.0
EPS - normalised (PLN)	0.64	0.51	0.12	(0.99)
EPS - reported (PLN)	0.20	0.47	0.12	(0.99)
Dividend per share (PLN)	0.0	0.0	0.0	0.0
BALANCE SHEET				
Fixed Assets	41,451	43,701	76,989	137,541
Intangible Assets	6,640	2,638	2,638	2,638
Tangible Assets	21,833	31,377	43,877	104,429
Other	12,979	9,686	30,474	30,474
Current Assets	47,669	59,873	174,553	97,980
Stocks	1,403	1,591	1,569	1,548
Debtors	16,320	19,226	30,000	30,000
Cash	29,095	36,124	140,687	64,136
Other	851	2,932	2,296	2,296
Current Liabilities	(18,933)	(26,752)	(22,627)	(22,427)
Creditors	(7,883)	(10,873)	(10,873)	(10,873)
Provisions	(3,600)	(5,150)	(5,150)	(5,150)
Deferred revenues	(5,469)	(8,451)	(4,200)	(4,200)
Short term borrowings	(859)	(912)	(912)	(912)
Other	(1,122)	(1,366)	(1,491)	(1,291)
Long Term Liabilities	(14,477)	(12,826)	(33,110)	(33,110)
Long term borrowings	(4,792)	(3,982)	(4,182)	(4,182)
Deferred revenues	(6,382)	(4,233)	(12,500)	(12,500)
Other long term liabilities	(3,303)	(4,611)	(16,429)	(16,429)
Net Assets	55,711	63,996	195,805	179,984
CASH FLOW				
Operating Cash Flow	(6,280)	10,265	(34,588)	(31,751)
Net Interest	0	0	0	0
Tax	0	717	(74)	(200)
Capex	(21,210)	(21,558)	(20,000)	(65,000)
Acquisitions/disposals	0	10	0	0
Financing	303	715	130,125	0
Dividends	0	0	0	0
Other (incl. subsidies)	21,859	19,174	28,900	20,400
Net Cash Flow	(5,329)	9,323	104,363	(76,551)
Opening net debt/(cash)	(28,773)	(23,445)	(31,230)	(135,593)
HP finance leases initiated	0	0	0	0
Exchange rate movements	0	0	0	0
Other	0	(1,537)	0	0
Closing net debt/(cash)	(23,445)	(31,230)	(135,593)	(59,042)

Source: Edison Investment Research, Selvita accounts. Note: *Non-cash cost related to the employee stock options programme. **Profit and loss from 2016 include share in Nodthera's earnings according to an equity method valuation.

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Management team

Chief executive officer (co-founder): Paweł Przewięźlikowski

Paweł Przewięźlikowski co-founded Selvita in 2007. From 1994 to 2007 he worked at Comarch, a Polish information technology company, becoming VP on the management board in 1996. While at Comarch, he was also the co-founder and the first CEO of Interia.pl, the third largest portal in Poland. He holds an MBA and MSc in information technology.

Chief operating officer (co-founder): Bogusław Sieczkowski

Bogusław Sieczkowski co-founded Selvita in 2007. From 2001 to 2007 he was VP and subsection director at Comarch. Previously he was IT manager at Bahlsen Polska (1995-99). He holds an MBA and MSc in information technology.

Chief scientific officer: Krzysztof Brzózka

Krzysztof Brzózka joined Selvita in 2007, became project manager (oncology compound) in 2009 and was appointed CSO in 2012. From 2003 to 2007 Krzysztof worked on a broad immunology research programme at Ludwig Maximillian University (Munich). He holds a PhD (molecular biology), an MSc and an MBA.

Director of chemistry department: Mirosława Zydroń

Mirosława Zydroń joined Selvita in 2009 and was appointed to the management board in 2013. From 2005 to 2009 Mirosława held various roles at Pliva (now Teva), including head of the R&D laboratory. She holds a PhD (analytical chemistry), an MSc and an MBA.

Director of biology department: Miłosz Gruca, PhD

Miłosz Gruca was appointed director of biology in 2010 (appointed to the management board in 2012), having worked at Selvita and BioCentrum (a Selvita subsidiary) since 2007, responsible for the introduction of complex biological and analytical services at Selvita. He holds a PhD (biochemistry), an MSc and an MBA.

Business Development Director: Edyta Jaworska

Edyta Jaworska is responsible for the development of Selvita integrated drug discovery services. Before she joined Selvita, she worked for several years as a manager in various healthcare companies, including OneMed Group and International Nephrology Centers. Mrs Jaworska holds an MSc in Economics and an MBA.

Chief Medical Officer: Steffen Heeger

Dr Steffen Heeger is responsible for all clinical, medical affairs and regulatory functions for Selvita's oncology pipeline. He is an oncologist with prior experience working as Vice President, Head of Clinical Development and Head of Clinical Operations at Morphosys, haematology and oncology products. Previously he also was Head of Medical Affairs at Merck Serono and led the Global Clinical Development Team for Erbitux.

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