EDISON

Selvita

Emerging biotech player

Partner & pipeline progress

Pharma & biotech

2016 could be a transformational year as in-house oncology drugs move into Phase I. A fresh Merck KGaA deal could add new candidates to the oncology pipeline. This and continued collaborations help maintain strong service revenues. SEL24 will enter Phase I in H216 (dosing expected to begin in July 2016) thereby bringing it closer to a potential deal. Our fair value has increased from PLN275m to PLN333m (from PLN21 to PLN25.4 per share) due to improved operational performance, internal programme advancement, addition of new partnering programmes and cash position.

Year end	Revenue (PLNm)	PBT* (PLNm)	EPS* (PLN)	DPS (PLN)	P/E (x)	Yield (%)
12/13	21.9	(2.4)	(0.23)	0.0	N/A	N/A
12/14	41.6	5.4	0.56	0.0	40.6	N/A
12/15e	55.4	6.8	0.51	0.0	44.2	N/A
12/16e	64.0	6.0	0.45	0.0	50.4	N/A

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

SEL24 and SEL120 moving closer to the clinic

Two lead cancer candidates have advanced. SEL24 is expected to start Phase I in acute myeloid leukaemia (AML) in H216. SEL120 is expected to begin IND-enabling studies in Q216 and move to Phase I in 2017. FLT3 inhibitors are 'hot' targets in AML and midostaurin, an FLT3 inhibitor, showed survival benefit of more than four years versus placebo in a Phase III study in AML (data at ASH 2015). This result should be viewed as a positive indicator for SEL24 and improves the partnering deal outlook for the dual PIM/FLT3 inhibitor.

New Merck deal and collaborations continue

A new milestone/royalty deal was announced in November to identify lead cancer drug candidates for Merck KGaA. Our model now assumes two oncology products from this initiative moving into Phase I by 2020. Collaboration success to date endorses the quality of research at Selvita and underpins confidence in the services and innovation businesses.

Valuation: Raised to PLN333m or PLN25.4 per share

The increase in our fair value from PLN275m to PLN333 is driven by a Services division showing better growth than previously forecast, increased valuation from the collaboration with Merck and advancement of the internal pipeline. This has led to a change in our valuation, including cash and allocating specific R&D costs to the internal programmes. Our fair value has progressed consistently over the year and is up 59% since initiation in November 2014.

9 December 2015

Price	**PLN22.7**
Market cap	**PLN298m**

Net cash (PLNm) at 30 September 2015	35.6
Shares in issue	13.1m
Free float	23%
Code	SLV
Primary exchange	WSE
Secondary exchange	N/A

Share price performance

%	1m	3m	12m
Abs	(5.4)	21.0	102.6
Rel (local)	5.3	42.5	171.5
52-week high/low	PLN25.25		PLN11.2

Business description

Selvita is a drug discovery services provider based in Poland. It employs 300 staff (30% PhDs) and operates two main business units: Innovations Platform (internal NME pipeline) and Research Services (medicinal chemistry/biology, biochemistry).

Next events

Q4/FY15 results	February 2016
Start SEL24 Phase I	H216
Start SEL120 IND studies	Q216

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Edison profile page

Selvita is a research client of Edison Investment Research Limited

Investment summary

Company description: Research and discovery

Selvita, founded in 2007, is already the largest research-based biotechnology company in Poland. It employs 300 staff (c 30% with PhDs) split approximately 40:60 between its Innovations Platform and Research Services divisions. The company operates out of a ~20,000ft^2 modern research facility in Krakow, Poland. The internal pipeline is close to Phase I and focused on novel small molecules for cancer. The research outsourcing business covers contract chemistry, biology and bioinformatics (clients from a range of big biopharma companies). Selvita listed on the Warsaw Stock Exchange NewConnect Market in July 2011 and upgraded its listing to the WSE Main Market in December 2014 with the successful offering and placing of 2.65m shares at PLN10.3 per share and gross proceeds of PLN27.3m. Selvita operates a capital-efficient financial model, although fresh funds may be sought to advance its internal pipeline.

Valuation: Consistently strengthening

Our fair value increases from PLN275m to PLN333m or from PLN21 to PLN25.4 per share. Services, pipeline products from collaborations and subsidies are valued at PLN198m (PLN15 per share), the new Merck collaboration adds just under PLN1 per share and cash the remainder. Since our initiation note in November 2014, our valuation has consistently risen on solid performance, collaboration and portfolio newsflow; an increase of 59% in one year. We now take the view that some of Selvita's cash will be required to develop SEL24 and SEL120 and we have therefore allocated some cash to R&D expenses for the SEL24 and SEL120 projects ($6m in total) to enable progress through Phase I and pre-Phase I respectively.

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 Innovations Platform. Contract research is a highly competitive and increasingly global field, which may put pressure on this side of its business achieving the rapid growth guidance. However, we feel this business is well placed and has sufficient flexibility in its business model to adapt. Since our initiation in November 2014, the innovations pipeline has progressed to Phase I with first clinical studies expected to commence in early 2016. 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 have a materially positive or negative impact on our valuation.

Financials: Strong revenues and profitability to be sustained

Selvita reached break-even in Q413 and we estimate that it will record a net profit of c PLN6.7m in 2015. Profitability should be sustained, although significant extra investment in its own pipeline will be required. Selvita has delivered consistently strong performance over the last seven quarters, with 2015 annual revenues forecast to grow 54% in its core Service business, 16% in its subsidies and 24% in Innovation. We anticipate continued strong growth from Innovation due to improving fees from innovations and an increasing number of contracts as the Service business expands its reach into the UK and US. The value of the 2015 order book, including commercial contracts signed, at September 2015 totalled an all-time high of PLN53.3m (Services PLN 24.6m, Innovation PLN14.3m, Grants PLN14.3m). It is too early to assess the value of the Bioinformatics business separately at this stage. Selvita's cash position is strong, with an estimated PLN36.5m at the end of 2015. There is sufficient cash to progress the internal drug candidates SEL24 and SEL120 to complete Phase I and pre-Phase I respectively. For these programmes to progress further, we would anticipate additional financing and/or a collaborative deal.

Outlook: Evolving into a biotech player

Selvita has established a good track record with its Services business, which has enabled it to also develop its own pipeline of novel small molecule programmes. Revenues from the Services division, research collaborations from eg Merck and H3 Biomedicine and grants/subsidies have propelled Selvita to profitability (since Q413). The Services business is expected to continue on a high-growth revenue trajectory (>20%) over the next two years, but the greater potential long-term upside could come from the development and licensing of a number of candidates from the internal pipeline. Key among these could be SEL24 and SEL120 in the near term, but Selvita has another five preclinical programmes, across oncology and autoimmune indications, which may also offer partnering potential. Selvita's operations are summarised in Exhibit 1.

Exhibit 1: Overview of Selvita's divisions

Division	Key features	Examples
Innovations Platform	Multiple wholly owned preclinical programmes in oncology	SEL24 (PIM/FLT3 inhibitor – AML); SEL120 (CDK8 inhibitor – CRC); SEL201 (MNK1/2 inhibitor – cancer); SEL303 (HO-1 inhibitor - cancer); SEL212 (NALP3 inflammasome – autoimmune); EPTHERON (Cancer epigenetics platform).
	Research collaborations with big pharma/biotech	Merck (cancer metabolism collaboration signed November 2015). Merck (cancer metabolism collaboration signed October 2013). H3 Biomedicine/Eisai (kinase discovery collaboration signed September 2013).
Research Services	Contract chemistry	Custom organic synthesis; medicinal chemistry; scale-up/process chemistry; industrial chemistry.
	Biology	Cell and molecular biology; biochemistry; analytical chemistry; ADME and pharmacokinetics; method validation.
	Bioinformatics* - 60% stake in Ardigen JV	LIMS systems; bioinformatics solutions; document management.

Source: Edison Investment Research, Selvita. Note: *Spun into Ardigen in October 2015.

Internal pipeline: Key partners are satisfied

Towards the end of 2013 Selvita's research capabilities received significant validation through the signing of two oncology research collaborations with H3 Biomedicine (a US research subsidiary of Eisai) and Merck KGaA.

Most recently a fresh three-year drug discovery deal was agreed with Merck to identify first-in-class small molecules as lead candidate drugs for multiple oncology indications. Under the terms of the new agreement, Merck will have an exclusive licence to the joint intellectual property and Selvita will receive milestone payments and royalties on successful development and commercialisation of products by Merck. The collaboration consists of a joint research phase up to lead identification, after which Merck will further research and develop the projects on its own. The scope of these collaborations is summarised in Exhibit 2.

Exhibit 2: Selvita's research collaborations

Partner	H3 Biomedicine (Eisai)	Merck KGaA	Merck KGaA
Deal signed	September 2013.	October 2013.	November 2015.
Initial contract period	2013-15, extended in October 2015.	2013-15.	2015-18.
Target	Validation of several kinase targets (2-4) in specific genetic contexts and generate multiple NMEs.	Discovery and development of multiple NMEs against selected protein targets (two projects on two targets) involved in cancer cell metabolism.	Discovery of first-in-class NMEs as lead candidates for multiple oncology indications.
Funding structure	Upfront payment, research funding, milestones and royalties to Selvita (first major research milestone met in May 2014).	PLN18.8m in research funding over 2015-18, with clinical candidates selected in 2019.	Milestone payments and royalties on successful development and commercialisation of products by Merck.
Funding terms	PLN5.8m funding guaranteed over 2013-15.	PLN10m funding guaranteed over 2013-15.	
Project target	Selection of a clinical candidate	Selection of a clinical candidate (2016-17).	

Source: Edison Investment Research, Selvita presentation (November 2015)

The H3 collaboration is focused on validating and developing compounds against new kinase targets, while the Merck deal will seek to inhibit targets associated with cancer metabolism. These

collaborations had an initial contract period of 2013-15 and, given the positive progress of the collaborative projects, both companies extended those programmes to the next phases of development. In addition, Merck has recently entered into a completely new collaboration which, if successful, is directly linked to developmental and sales milestones and royalties on sales for Selvita.

Primary focus on oncology

Selvita's current pipeline of novel drug programmes, often referred to as new molecular entities (NMEs), is displayed in Exhibit 3. The primary focus is on the anti-cancer programmes, particularly SEL24 and SEL120, which are the most advanced in terms of development and selection of a preclinical candidate.

Exhibit 3: Selvita's own pre-clinical stage pipeline

Project	Mechanism	Therapeutic area	Status
SEL24	Dual PIM/FLT3 kinase inhibitors	Oncology: leukaemias (AML), lymphomas, myeloma	■ Clinical candidate SEL24-B489 selected (GLP toxicity/CMC manufacturing planned). ■ IND filing Q216 and start Phase I July 2016. ■ Partner(s) sought.
SEL120	CDK8 kinase inhibitors	Oncology: colorectal, breast cancers	■ Clinical candidate SEL120-034 selected (further characterisation planned). ■ IND-enabling studies to complete in 2017. ■ Partner(s) sought.
SEL201	MNK1/2 kinase inhibitors	Oncology: solid tumours (prostate, NSCLC), lymphomas	■ Potent MNK1/2 inhibition established (POC in vitro data). ■ Lead optimisation. ■ Biomarker response identified.
SEL303	HO-1	Oncology	■ Anti-proliferative properties of heme oxygenase-1 (HO-1) inhibitors.
SEL212	NALP3 inflammasome modulators	Autoimmune disorders: FMF, psoriasis, rheumatoid arthritis	■ Assay development ongoing. ■ Clinical development 2016.
Kinase discovery platform	Protein phosphorylation	Cancer: solid tumours, oncology	■ Discovery platform.
Cancer metabolism platform	Small molecule inhibitors	Oncology	■ Progressing to lead stage.

Source: Selvita

SEL24: A potentially unique combination

SEL24 is Selvita's most advanced oncology programme, focused on developing a compound with a potential dual mechanism of action to target two kinases, namely PIM (1, 2 and 3 isoforms) and FLT3 mutants, which have been shown to be important in the development of acute myeloid leukaemia (AML) and other haematological malignancies.[1]

A significant unmet need in AML

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). AML is generally a disease of older people and the average age at initial diagnosis is 66 years. The five-year survival rate for all AML patients, irrespective of age or genetic status, is 23%. The standard-of-care treatment for AML has not changed significantly for many decades, primarily based on chemotherapy (cytarabine) 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

[1] Levis, M and Small, D (2003). FLT3: ITDoes matter in leukemia. Leukemia (2003) 17, 1738–1752.

recognised as the only curative treatment option, but is not always appropriate. To date there are no targeted agents approved for the treatment of AML, although many are in development.

PIM – A promising target upstream of FLT3

The PIM family of serine-threonine kinases play an important role in intracellular signalling. PIM kinases are induced by several pro-oncogenic proteins such as FLT3, PI3K kinases, JAK and NF-κB, and when overexpressed can mediate drug resistance. As such, targeting both PIM kinases and FLT3 mutants may improve response rates and reduce relapses.

Overexpression of PIM has been reported in a variety of haematological malignancies[2] (eg. AML, CLL, B-cell lymphoma), as well as some solid tumours (pancreatic, liver). In Exhibit 4 shows the most advanced candidates against PIM kinases and those against FLT3. AstraZeneca's AZD1208[3] completed a Phase I study, but terminated clinical development in Q314 due to efficacy and safety reasons (no detail provided). Novartis has launched a number of Phase I and Phase I/II studies with LGH447, so data over the next 12-18 months will provide greater insight into the viability of specifically targeting PIM kinase.

Exhibit 4: Competitive clinical pipeline of PIM and FLT3 kinase inhibitors

Pharma class	Product	Company	Lead indication	Status	Trial data
PIM inhibitors	LGH447	Novartis	Haematological malignancies (multiple myeloma/AML/MDS)	20-pt Phase I/II (multiple myeloma) 77-pt Phase I (multiple myeloma) 40-pt Phase I (AML/MDS)	H215 H216 H116
	AZD1208	AstraZeneca	Malignant lymphoma	43-pt Phase I	Discontinued Q314
	INCB053914	Incyte	Advanced malignancies	145-pt Phase I	H217
	TP-3654/SGI-9841	Tolero/Astex	Hematological malignancies, Prostate cancer, urothelial carcinomas	Preclinical	N/A
FLT3 inhibitors	Quizartinib (AC220)	Ambit Biosciences (Daiichi Sankyo)	Acute myeloid leukaemia (AML)	326-pt Phase III	H116
	Midostaurin (PKC412)	Novartis	AML	717-pt Phase III	Dec 2015 (ASH)
	Lestaurtinib (CEP-701)	Children's Oncology Group (NCI)	Acute lymphoblastic leukaemia (ALL)	242-pt Phase III	H218
	Gilteritinib (ASP2215)	Astellas Pharma	AML	369-pt Phase III	N/A
	Crenolanib	Arog Pharma	AML	70-Pt Phase II	H215
	E6201	Strategia Pharma	AML	62-Pt Phase II	H217
	TAK-659	Takeda	AML	81-Pt Phase II	H217
	AKN-028	Akinion	AML	55-Pt Phase II	H216

Source: Edison Investment Research, clinicaltrials.gov

FLT3 – a natural dual target

FLT3 (FMS-like tyrosine kinase-3) is a kinase receptor expressed on hematopoietic progenitor cells (immature blood cells) and plays a critical role in regulating their activation, growth, proliferation, survival and differentiation into mature blood cells. The specific FLT3-ITD mutation results in aggressive proliferation of immature, irregular blasts that lack the ability to differentiate into normal blood cells. Physicians, as a standard part of diagnosis, routinely test patients for the FLT3-ITD mutation, which is estimated to affect 25-35% of elderly AML patients. Patients who are FLT3-ITD positive have a significantly worse prognosis compared to FLT3-ITD negative patients.

As such, FLT3 presents an obvious molecular target for new therapies. While a number of multi-kinase inhibitors already on the market, including Bayer's Nexavar and Pfizer's Sutent, already act

[2] Alvarado Y, Giles FJ, Swords RT (2012). The PIM kinases in hematological cancers. Expert Rev Hematol. 2012 Feb; 5(1):81-96.

[3] Keeton EK, et al (2014). AZD1208, a potent and selective pan-Pim kinase inhibitor, demonstrates efficacy in preclinical models of acute myeloid leukemia. Blood. 2014 Feb 6; 123 (6): 905-13.

on FLT3 (as well as other receptors such as cRAF, VEGFR, c-KIT), compounds that specifically target FLT3 are still in clinical development (Exhibit 4).

Midostaurin survival benefit – a halo effect for SEL24?

Ambit Biosciences' quizartinib (AC220) and Novartis's midostaurin (PKC412) are the most advanced FLT3 inhibitors in AML. A recent abstract disclosure for the 2015 ASH meeting in December revealed event-free survival and overall survival in a 717-patient midostaurin study in AML patients with FLT3 mutations (Exhibit 5). This is the first large Phase III trial to confirm a therapeutic benefit of FLT3 inhibition in AML patients.

Exhibit 5: Midostaurin Phase III results

Endpoint	Arm	Median (months)	p-value	Five-year event rate (%)*	Hazard ratio
Overall survival	Midostaurin	74.7	0.007	50.8	0.77
	Placebo	26.0		43.1	
Event-free survival	Midostaurin	8.0	0.0044	26.7	0.80
	Placebo	3.0		19.1	

Source: ASH Abstracts 2015. Note: *Proportion of patients with an event defined as the earliest of death, relapse, or no complete response.

The midostaurin story had been quiet for a number of years and there may have been some uncertainty about any clinical benefit. However, the first survival results from a large Phase III study should help to reinvigorate interest in the FLT3 target in AML. Overall survival was increased from approximately two years to just over six years. This result could now greatly improve the chances of Selvita finding a suitable partner and move SEL24 through clinical development. FLT3 plays an important role in leukemogenesis and its presence is associated with poor prognosis in acute myeloid leukemia (AML). PIM-1 was found to be one of the most significantly downregulated genes on FLT3 inhibition. Midostaurin inhibits FLT3 but not PIM. SEL24 inhibits both, so could offer improved efficacy. It is too early to say based on clinical evidence to date, but the midostaurin data should help refocus the industry on SEL24.

Ambit should report Phase III data within the next six to eight months. Both these selective compounds, and others, have showed strong in vitro cytotoxicity to leukaemia cells, and activity in relapsed AML patients with FLT3 mutations in clinical trials, with improvements in response rates and overall survival, although this needs to be confirmed for AC220 in pivotal Phase III studies. In addition, the reductions of blasts in blood have often been transient.

We note that Ambit secured a co-development deal with Astellas Pharma in 2009 for quizartinib, when the drug was undergoing Phase II studies, with Astellas paying a $40m upfront fee as part of a $390m total deal value. Astellas subsequently returned rights to the compound in 2013, but Daiichi Sankyo recently agreed to purchase Ambit for $315m, with a further $95m payable depending on commercial milestones being achieved.

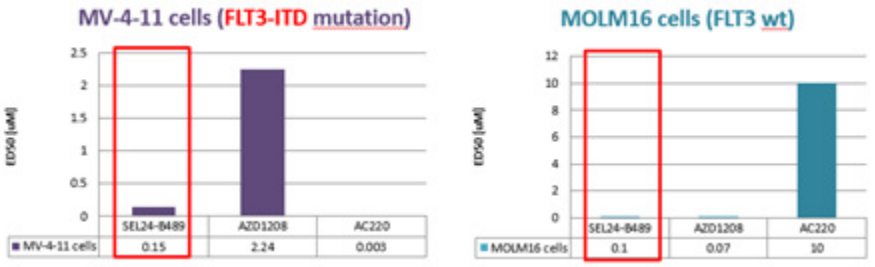
SEL24 moving into Phase I in H216

Selvita has developed SEL24 as a potentially first-in class dual PIM/FLT3 kinase inhibitor, with potential to treat AML and a range of other haematological malignancies (eg diffuse large B-cell lymphoma, multiple myeloma, follicular lymphoma). SEL24-B489 has been tested extensively in in vitro and in vivo models, which confirm the drug's dual mechanism of action and demonstrate a strong cytotoxic effect in multiple haematological models, as well as potential synergy with current standard low-cost chemotherapy (cytarabine). Current drugs in development target either PIM or FLT3 kinase enzymes, but SEL24 has the ability to inhibit both families of kinases at therapeutic doses.

SEL24 has shown superior activity in AML cell lines irrespective of the mutation background, in comparison to both AC220 and AZD1208. Exhibit 6 shows how B489 is effective against both FLT3-

ITD positive AML cells (MV-4-11) and FLT3-ITD negative AML cells (MOLM-16), which both express high levels of PIM kinases. AZD1208 appears less effective against FLT3 positive cells, while ACC220 is less efficacious against FLT3 negative cells.

Exhibit 6: Comparing the efficacy of B489, AC220 and AZD1208 in FLT3+ve/-ve AML cells



Source: Selvita

In terms of potential combinations with existing therapies, an important consideration for drug development, the use of B489 with cytarabine (current standard of care) produced tumour growth inhibition (TGI) rates that indicate a dose-dependent synergistic anti-tumour effect (Exhibit 6). Increased toxicity can be an issue when combining agents, yet no toxic effects of SEL24 were observed at the highest combination dosing schedule.

Next steps for the SEL24 programme

Selvita continues to work on preparing SEL24 for an IND filing, with the aim of starting a Phase I study in H216. Non-GLP toxicology studies have been conducted in dogs and rats, which revealed no issues at proposed therapeutic doses of SEL24-B489. IND-enabling studies are being carried out by Aptuit and the Phase I trial will be conducted by Theradex.

Selvita's goal is to secure a partner for the SEL24 programme, and the company has stated that it is in advanced partnering negotiations with multiple potential pharma and biotech partners.

SEL120: A potentially unique profile

SEL120 is a potential first-in-class selective CDK8 inhibitor. Cyclin-dependent kinases (CDKs) play an essential role in the control of cell cycle, proliferation and mRNA transcription. These kinases, as well as their regulators, are frequently deregulated in multiple tumour types, so compounds that interfere with these kinases hold therapeutic potential.[4] A number of CDK inhibitor candidates are in advanced stages of clinical trials, summarised in Exhibit 7.

Exhibit 7: Competitive clinical pipeline of CDK inhibitors (Phase III and II)

Product	Company	Mechanism	Lead indication	Status	Trial data
Palbociclib (PD-0332991)	Pfizer	CDK4/6 inhibitor	Breast cancer	Approved Feb 2015	
LEE011	Novartis	CDK4/6 inhibitor	Breast cancer	666-pt Phase III	2016
Abemaciclib (LY2835219)	Eli Lilly	CDK4/6 inhibitor	NSCLC/breast cancer	550-pt Phase III (NSCLC); 660-pt and 450-pt Phase III (breast)	H216 H217
Alvocidib (flavopiridol)	Tolero Pharmaceuticals	CDK9/cyclin-T inhibitor	Acute myeloid leukaemia	Phase III planned	
Milciclib (PHA-848125)	Nerviano Medical Sciences	CDK2/TRKA inhibitor	Thymic carcinoma	60-pt Phase II	H216
AT7519	Otsuka (Astex)	CDK1/2/9 inhibitor	Multiple myeloma	18-pt Phase I/II	Completed 2015

Source: Edison Investment Research, clinicaltrials.gov

A number of pan-CDK inhibitors have been developed, the majority of which target a range of CDK subtypes, such as CDK4/6 (eg palbociclib, abemaciclib, LEE011) and CDK1/2/5/9 (dinaciclib). Palbociclib (Pfizer) was granted accelerated approval by the FDA in February 2015 as a first-line

[4] Cicenas, J and Valius, M (2011). The CDK inhibitors in cancer research and therapy. J Cancer Res Clin Oncol. 2011 Oct;137 (10):1409-18.

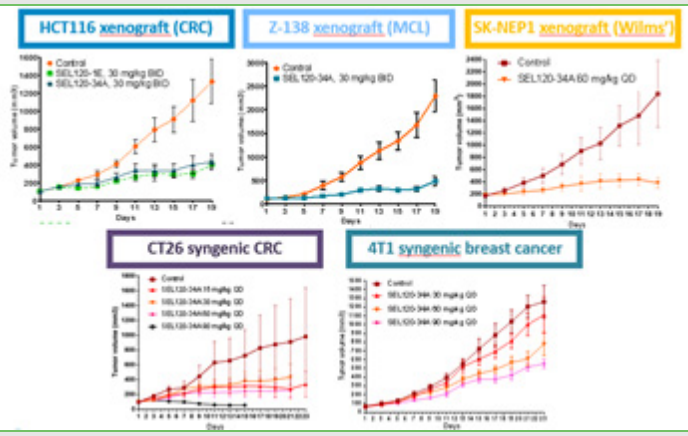
treatment (with letrozole) for ER+, HER2- advanced breast cancer, with consensus global annual sales of $3bn by 2020 (EvaluatePharma World Preview 2014).

However, Selvita's SEL120 programme appears to be the only selective CDK8 inhibitor in active development. Specific targeting of CDK8 may avoid some of the toxicity issues that pan-CDK inhibitors can cause, while CDK8 is known to be particularly overexpressed in colorectal cancer and in certain haematological malignancies, such as mantle cell lymphomas. Tumour cells that express elevated CDK8 levels are highly dependent on its expression for proliferation.[5] CDK8 is also thought to be involved in regulating the activity of natural killer cells (NK cells), which are important components of the innate immune response against cancer cells. Inhibition of CDK8 may therefore also promote natural immunosuppression mechanisms.

SEL120 – scheduled for Phase I in 2017

Selvita has identified SEL120 as a lead candidate from the programme to take forward into IND-enabling studies. SEL120 has been tested in in vitro and in vivo models, which show significant reductions in the viability of colorectal cancer and mantle cell lymphoma cell lines, with particularly good activity in cell lines overexpressing CDK8. A selection of tumour inhibition data from multiple in vivo models is displayed in Exhibit 8. Importantly, the drug's safety profile is encouraging, with no signs of neutropenia reported, which is characteristic of cell cycle modulators.

Exhibit 8: SEL120 inhibits tumour survival in multiple in vivo cancer models



Source: Selvita (Nov 2015). Note: HCT116=CRC, Z-138=MCL, CT26.WT=CRC, 4T1=breast Wilms = kidney (xenograft models).

Next steps for the SEL120 programme

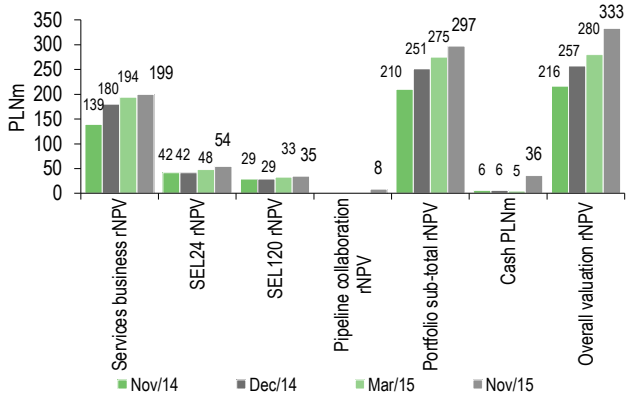
Selvita continues to work on fully characterising SEL120 and developing back-up compounds. Development of SEL120 is part-funded by the KIND-P1 research grant, as discussed above, for SLE24. Selvita is targeting an IND filing and the start of a Phase I study in 2017, but is also aiming to secure a partner for further development of the SEL120 programme. The company has stated that it is in advanced partnering negotiations with multiple pharma and biotech partners.

Valuation

Applying a standard DCF model for the Research Services business, and an rNPV for the SEL24 and SEL120 programmes, we derive an indicative value for Selvita of PLN333m (€70m), or PLN25.4 per share. The existing operations (services, collaborations, subsidies) are valued at PLN199m (PLN15 per share and the new Merck collaboration also adds just under PLN1 per share. Since initiation in November 2014 our valuation has consistently risen based on solid performance, collaboration and portfolio news flow; an increase of 59% in one year.

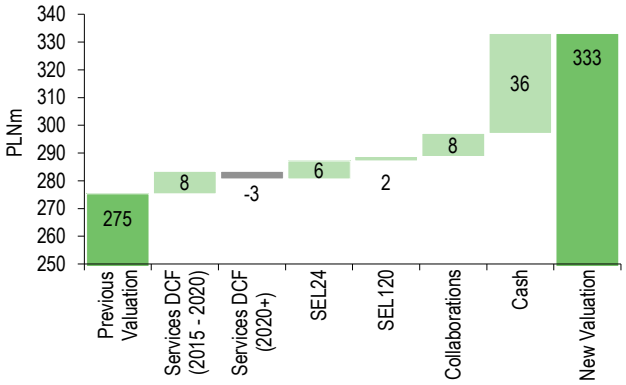
[5] Rzymski T, et al (2013). Development of selective CDK8 inhibitors for colorectal cancer and mantle cell lymphoma treatment. Cancer Research: April 15, 2013; Volume 73, Issue 8, Supplement 1.

Exhibit 9: Valuation progression since initiation



Source: Edison Investment Research

Exhibit 10: Adjustments to previous valuation



Source: Edison Investment Research

It is clear that there has been an improvement across all the valuation elements. The main adjustments to the valuation model reflect increased cash (capital increase of PLN25.4m), improving Research and Services performance, progress of SEL24 and SEL120 towards Phase I and the recent Merck collaboration announcement. Given the increase in cash after the capital increase of PLN25.4m and the further detail provided by the company of Phase I trials for SEL24 and SEL120, we now value cash separately, allocating some of it to specific research programmes. We now take the view that a small fraction of Selvita's cash will be required to develop SEL24 and SEL120, so we have allocated some cash to R&D expenses for those projects ($6m in total) to enable progress through Phase I and pre-Phase I respectively. The 2018 Services revenue forecast has increased to PLN41m vs PLN28m and Innovation to PLN20m vs PLN15m in our old model. Cash currently represents 11% of the valuation, whereas the Services business represents 60%, and 27% of our fair value corresponds to SEL24 and SEL120 combined. Our key assumptions and valuation metrics are summarised in Exhibit 11.

Exhibit 11: Selvita valuation model

Division	Metric	Value (PLNm)	Value per share (PLN)	Notes
Services/ research collaborations	DCF (2015-20)	67	5.12	Services: sliding scale pa growth 25-15% in 2015-20; research collaborations: +7.5% pa growth; subsidies: +2.5% pa growth; tax = 2%-9% sliding scale (2015-2020); 10% WACC.
	Terminal value	132	10.08	0.75% growth on 2020 FCF.
	Subtotal	**199**	**15.20**	
Internal pipeline	SEL24	54	4.15	$750m indicative peak sales (2028); launch in 2022; 5% royalty (preclinical); 7.5% probability of success (preclinical). Includes deal milestone estimates: $15m upfront after Phase I results in 2017 (60%); $10m on start of Phase II in 2017 (30%); $20m on start Phase III in 2019 (15%); $40m on NDA filing/approval in 2021 (7.5%). 12.5% WACC. Internal R&D Phase I costs of $5m in 2016.
	SEL120	35	2.66	$750m indicative peak sales (2029); launch in 2023; 5% royalty (preclinical); 5% probability of success (preclinical). Includes deal milestone estimates: $3m upfront in 2017 (60% probability); $5m on IND/Phase I start in 2017 (50%); $10m on start Phase II in 2018 (25%); $20m on start Phase III in 2020 (10%); $40m on NDA filing/approval in 2022 (5%). 12.5% WACC. Internal R&D pre-Phase I costs of $1m in 2016.
	Collaboration	8	0.63	New Merck KGaA collaboration as of October 2015, which allows for identification of five to six Phase I candidates over the next three years against cancer metabolism targets. We assume two in Phase I in 2020. Receive €1.47m annually for three years for services. Milestones related to candidate selection, commencement of Phase I, initiation of pivotal trials, launch in major regions and sales thresholds. We assume the following royalty rates: 0.5% on sales up to $500m, 1% on $500m-1bn and 2% on sales greater than $1bn. Probability of 5% to market.
	Subtotal	**98**	**7.45**	
Cash		36	2.74	
Selvita total		**333**	**25.4**	Based on 13.1m shares outstanding.

Source: Edison Investment Research

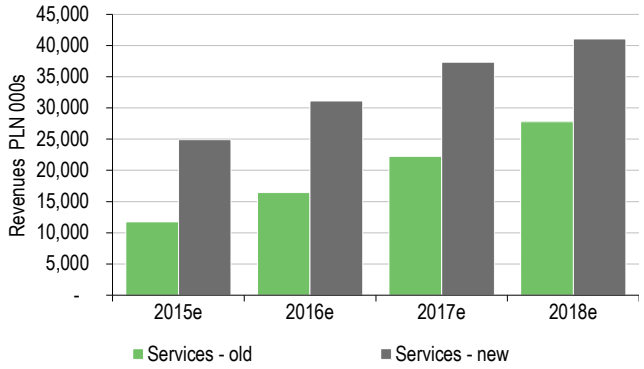
For the recent Merck collaboration we have assumed that five to six lead candidates are identified and that two of these proceed to Phase I with potential annual peak sales of $750m each. We

assume a 5% chance of these reaching the market by 2026 and milestones at various points during the clinical and market stages.

Financials

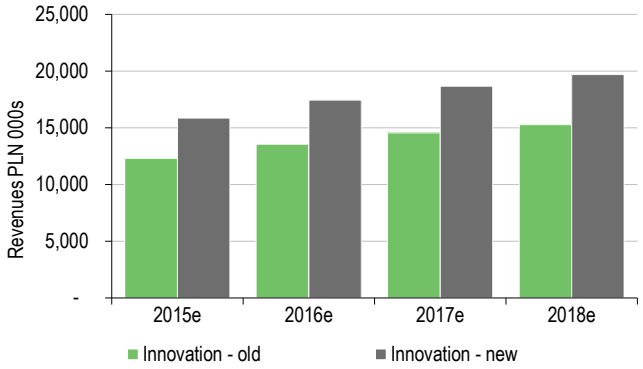
Exhibits 12 and 13 illustrate the significant upgrades to our forecast revenue lines for the Service and Innovation businesses. In 2018 we have revised Services up 48% and Innovation 30%.

Exhibit 12: Adjustments to Service business revenue forecasts



Source: Edison Investment Research

Exhibit 13: Adjustments to Innovation business revenue forecasts

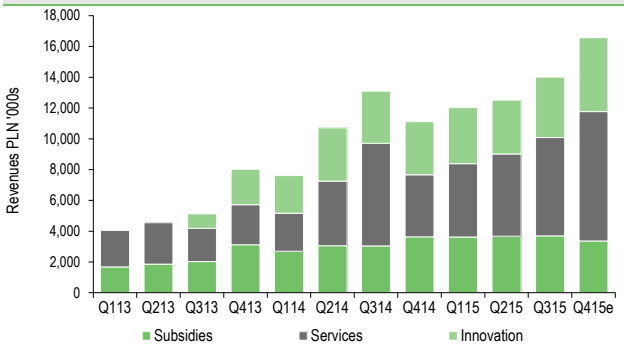


Source: Edison Investment Research

Selvita has demonstrated consistently strong performance over the last seven quarters, with 2015 annual revenues forecast to grow 54% in its core Service business, 16% in its subsidies and 24% in Innovation. The growth in the Innovation division was due to the increasing collaboration activities around oncology candidates, which may ultimately lead to several new chemical entities entering Phase I trials. As Exhibit 14 shows, we anticipate continued strong growth from Innovation and Services revenues in Q4 of this year due to improving fees from Innovations and an increasing number of contracts as the Service business expands its reach into the UK and US. The value of the 2015 order book, including commercial contracts signed at September 2015 totalled PLN53.3m (Services PLN24.6m, Innovation PLN14.3m, Grants PLN14.3m).

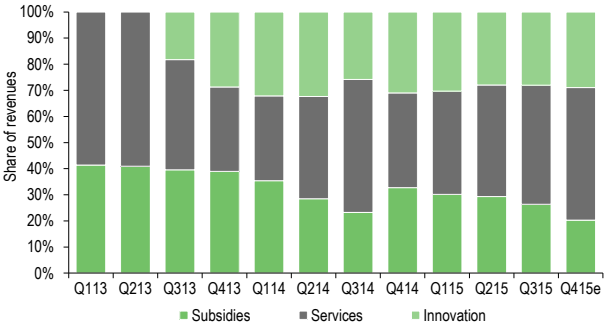
The bioinformatics business is focused on personalised medicine and has business with players such as Abbott Informatics, Microsoft, Oracle, IBM and Qiagen. Given its strong performance and expertise in the overall business, it was felt that the value from bioinformatics business would be better realised if it was operated as a separate entity. Consequently, the Bioinformatics part of the service group has been spun off into Ardigen, with Selvita retaining a 60% stake, which it expects to be marginally profitable. We have insufficient detail at this early stage to value it separately.

Exhibit 14: Quarterly revenue performance



Source: Edison Investment Research, Selvita

Exhibit 15: Share of revenue across business units



Source: Edison Investment Research, Selvita

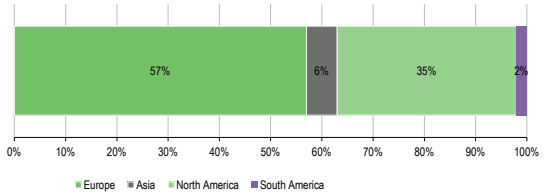
Selvita's cash position is strong, with an estimated PLN36.5m at the end of 2015. There is sufficient cash to progress the internal drug candidates SEL24 and SEL120 to complete Phase I and pre-

Phase I respectively. For these programmes to progress further, we would anticipate additional financing and/or a collaborative deal.

Exhibit 16: Financial summary

PLN000s	2013	2014	2015e	2016e	2017e
Year end 31 December	IFRS	IFRS	IFRS	IFRS	IFRS
PROFIT & LOSS					
Revenue	21,914	41,557	55,355	63,954	72,153
of which: Services (research outsourcing)	9,812	16,121	24,894	31,118	37,341
Innovation pipeline funding	3,241	12,744	15,871	17,458	18,680
Subsidies	8,688	12,430	14,360	15,078	15,832
Cost of Sales (External services; value of goods sold)	(4,469)	(6,503)	(9,044)	(10,891)	(13,069)
Gross Profit	17,445	35,054	46,311	53,062	59,084
EBITDA	(146)	7,626	10,441	9,285	11,789
Operating Profit (before GW and except.)	(2,228)	5,272	6,770	6,000	8,457
Intangible Amortisation	0	0	0	0	0
Exceptionals/Other	0	0	0	0	0
Operating Profit	(2,228)	5,272	6,770	6,000	8,457
Net Interest	(198)	155	18	18	21
Exceptionals/Other	0	0	0	0	0
Profit Before Tax (norm)	(2,427)	5,427	6,789	6,018	8,478
Profit Before Tax (FRS 3)	(2,427)	5,427	6,789	6,018	8,478
Tax	(19)	423	(53)	(120)	(254)
Deferred tax	0	0	0	0	0
Profit After Tax (norm)	(2,445)	5,850	6,736	5,898	8,223
Profit After Tax (FRS 3)	(2,445)	5,850	6,736	5,898	8,223
Average Number of Shares Outstanding (m)	10.5	10.5	13.1	13.1	13.1
EPS - normalised (PLN)	(0.23)	0.56	0.51	0.45	0.63
EPS - FRS 3 (PLN)	(0.23)	0.56	0.51	0.45	0.63
Dividend per share (PLN)	0.0	0.0	0.0	0.0	0.0
BALANCE SHEET					
Fixed Assets	7,067	9,494	11,556	12,064	12,619
Intangible Assets	282	331	312	312	312
Tangible Assets	4,932	6,845	7,823	8,330	8,886
Other	1,854	2,318	3,421	3,421	3,421
Current Assets	11,191	17,310	48,336	53,546	61,086
Stocks	391	706	806	795	784
Debtors	5,161	10,314	10,547	10,547	10,547
Cash	5,418	4,878	36,587	41,807	49,359
Other	221	1,411	396	396	396
Current Liabilities	(11,401)	(15,271)	(15,299)	(15,419)	(15,553)
Creditors	(11,239)	(15,180)	(15,240)	(15,360)	(15,494)
Short term borrowings	(161)	(91)	(59)	(59)	(59)
Long Term Liabilities	(3,454)	(2,278)	(2,386)	(2,386)	(2,386)
Long term borrowings	0	0	0	0	0
Other long term liabilities	(3,454)	(2,278)	(2,386)	(2,386)	(2,386)
Net Assets	3,403	9,254	42,207	47,804	55,767
CASH FLOW					
Operating Cash Flow	(7,198)	(4,902)	(6,857)	(6,024)	(4,272)
Net Interest	0	0	0	0	0
Tax	0	0	0	0	(120)
Capex	(2,167)	(3,611)	(3,701)	(3,793)	(3,888)
Acquisitions/disposals	0	0	0	0	0
Financing	0	0	27,314	0	0
Dividends	0	0	0	0	0
Other (subsidies)	9,642	8,548	14,360	15,078	15,832
Net Cash Flow	276	36	31,117	5,261	7,551
Opening net debt/(cash)	(5,192)	(5,257)	(4,787)	(36,528)	(41,748)
HP finance leases initiated	0	0	0	0	0
Exchange rate movements	0	0	0	0	0
Other	(212)	(506)	624	(40)	0
Closing net debt/(cash)	(5,257)	(4,787)	(36,528)	(41,748)	(49,300)

Source: Edison Investment Research, Selvita

Contact details	Revenue by geography (FY 2014)
Park Life Science ul. Bobrzyńskiego 14 30-348 Kraków Poland +48 12 297 47 00 www.selvita.com	

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 sub-section 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.

Director of bioinformatics department: Sebastian Kwaśny

Sebastian Kwaśny joined Selvita in 2011 and was appointed to the management board in 2012. From 1998 to 2010 Sebastian worked at Comarch, where roles included: engineer and department manager (1998-1999), director of the Center of Responsibility (2000-2006), and director of the IT infrastructure sub-sector (2006-2010). He holds an MSc Eng (telecommunications) and an MBA.

Principal shareholders	(%)
Paweł Przewięźlikowski (Co-founder, CEO)	42
Bogusław Sieczkowski (Co-founder, COO)	7
Management and Supervisory Board	18
Other shareholders	23

Companies named in this report

Merck KGaA; H3 Biomedicine (Eisai)

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