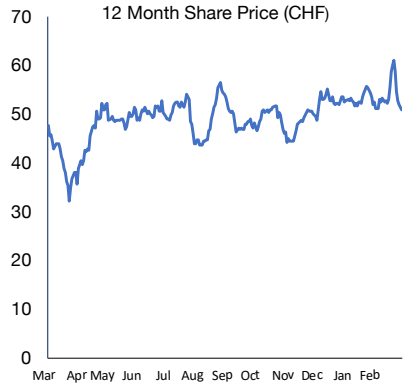
Basilea Pharmaceutica

22 February 2021

Share Price (CHF)	**50.8**
CP Fair Value (CHF)	**120**
Market Cap (CHFm)	605
Cash (CHFm)	167
EV (CHFm)	688
Country	Switzerland
Code	BSLN SW
Exchange	SIX



Source: Calvine Partners Research

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Lisavanbulin in focus

Inevitably, the emerging oncology franchise has been dominated by the outlook and expectations for lead programme derazantinib, particularly given the increasing competitive environment. However, although at an earlier stage, lisavanbulin, an inhibitor of the spindle assembly checkpoint (SAC), has made steady progress with highly encouraging clinical data in a small number of patients in the difficult to treat indication of glioblastoma (brain cancer). Furthermore, Basilea has identified a potential biomarker which, if successful, could help identify patients who should benefit most from this approach, allowing enrichment of clinical trials and increasing the probability of a positive outcome. Despite these encouraging data, lisavanbulin does not yet feature in our financial model or valuation of Basilea suggesting significant upside risk should clinical data prove to be supportive.

Targeting difficult to treat tumours

Basilea has invested heavily in establishing a meaningful and relevant oncology franchise. Lisavanbulin represents an important programme targeting the mitotic spindle. While historically microtubule targeting agents (MTAs) have been dominated by natural compounds and have proven to be some of the most effective chemotherapeutic agents available commercially, they have been associated with significant toxicity and the rapid emergence of resistance. Nevertheless, advances in our understanding of the importance of the microtubule in tumour biology has led to the development of new synthetic candidates like lisavanbulin, which are active against previously resistant cancers. Furthermore, Basilea is evaluating the employment of a likely predictive biomarker (EB1) which should enable identification of suitable patients, and reduce the risk of treatment failure.

Clinical data highly encouraging

As a synthetic small molecule, lisavanbulin, unlike natural compounds, can cross the blood brain barrier. Consequently, Basilea has chosen glioblastoma multiforme (GBM) as the lead indication for lisavanbulin. GBM is a particularly insidious cancer and, although rare, is highly malignant and incurable, with a median survival time of around 15-18 months. These stark statistics put into perspective the importance of the profound responses observed in two patients who received lisavanbulin while suffering from recurrent glioblastoma. All this bodes well for the ongoing Phase 2 study evaluating in patients with advanced EB1 positive glioblastoma, Interim results from this study are expected in H2 2021. (For Risks see Page 10).

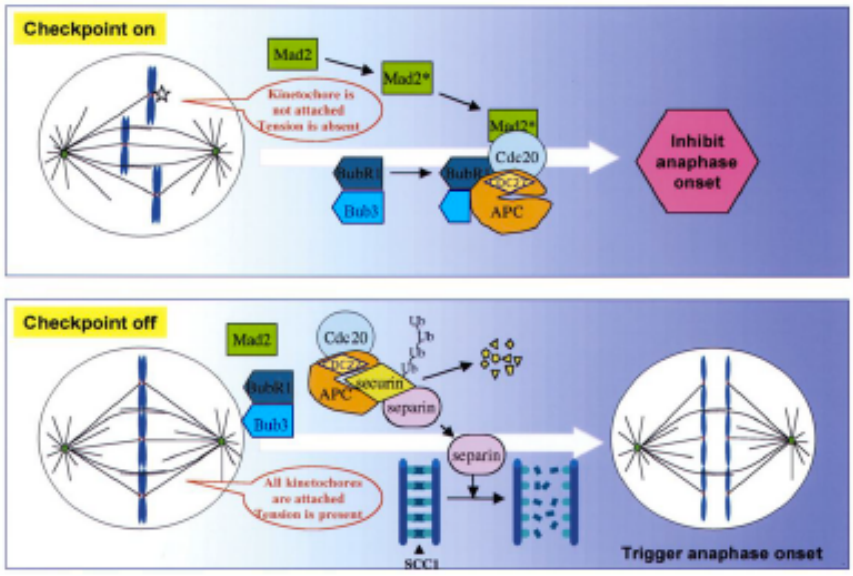
An underappreciated pipeline asset

Acting at the spindle assembly…

Lisavanbulin is a tumour checkpoint controller exerting its influence at the mitotic checkpoint or spindle assembly checkpoint (SAC). The mitotic checkpoint has long been an important target in cancer, since preventing cells (in this case tumour cells) from passing through mitosis (somatic cell division), rapidly leads to apoptosis and cell death. Unsurprisingly, mitosis has been identified as a particularly important step in cancer cell proliferation.

…arrests cell division and leads to cell death

Over time it has proven to be a successful therapeutic strategy to selectively eliminate actively growing cancer cells. The mitotic checkpoint is one of four checkpoints which ensure the fidelity of cell division, arresting cell division if each checkpoints condition is not successfully met. For the sake of completeness, in addition to the mitotic checkpoint the other three (G1/S, S & G2/S) are DNA damage checkpoints and lie beyond the scope of this note.



Adapted from Zhou J et al. J Cell Sci 2002;115:3547-3555

MTAs have been around for 50 years

An increased understanding of the mechanisms involved in cell cycle progression and division has facilitated pharmaceutical intervention to specifically interrupt tumour cell proliferation. The role of microtubules and their associated proteins in mitotic spindle assembly, chromosome segregation, and cell division, makes them attractive targets for cancer drug development resulting in the availability of numerous microtubule targeting agents (MTAs).

Despite their age, MTAs remain important in the fight against cancer

The MTAs have served as the mainstay of chemotherapy for many decades. The vinca alkaloids (microtubule de-stabilizing agents including colchicine), taxanes (microtubule stabilizing agents) and epothilones are all highly potent in multiple cancer types. Most well-established therapies target the paclitaxel or vinblastine sites and include paclitaxel, vinblastine, vincristine, vinorelbine and eribulin. Despite the introduction of targeted therapies, and more recently immunotherapy, microtubule agents remain first-line therapy for many cancers.

Historically, a class dominated by naturally derived therapeutics

Microtubules perform several important roles in cell division (mitosis and meiosis) as well as migration, hence they have proven to be fruitful targets for cancer chemotherapy. In dividing cells during mitosis, the microtubules forming the mitotic spindle ensure that genetic material (chromosomes) is shared equally between dividing cells and have proven to be very tractable to therapeutic intervention, although historically with natural compounds. With many existing microtubule therapies derived from natural sources, development of newer synthetic (or semi synthetic) microtubule agents has been slow, but represent an obvious route for development, particularly as they should be able to cross the blood brain barrier.

Dose limiting toxicities and rapid resistance have plagued the class

However, it is not all good news. While MTAs have proven to be highly effective in multiple solid and haematological cancers, side effects associated with their long and short-term use have limited their applicability and use. DNA damage and apoptosis are the main causes of drug-induced cytotoxicity, while neurological and haematological side effects are dose-limiting toxicities.

Other limitations have hampered optimisation efforts

Due to the essential role of microtubules in non-dividing cells, MTAs cause significant toxicity in normal cells. Specifically, microtubules play an important role in synaptic signalling in neuronal cells. Consequently, MTAs have been associated with neuronal damage resulting in serious peripheral neuropathy in cancer patients. For example, peripheral neuropathy is estimated to affect as many as 80% of patients receiving taxanes. There are also other challenges which include poor solubility and cumbersome synthesis/ manufacture which have served to hamper their optimisation.

Rapid resistance an inevitable occurrence

One of the most significant factors limiting the applicability of microtubule inhibitors has been the development of rapid resistance. This is perhaps unsurprising given the importance of microtubules to human life. Multiple mechanisms of drug resistance have been characterized, which typically involve efflux mechanisms and membrane-associated changes to prevent drug accumulation within tumour cells. Both taxanes

and vinca alkaloids are substrates of the (P-gp) efflux pump and represent the primary resistance mechanism. Tubulin variants (isotypes) are also involved in the induction of resistance. Attempts to overcome resistance using irreversible tubulin inhibitors have been disappointing largely due to significant toxicity.

The colchicine binding site holds significant promise, but development efforts yet to bear fruit

Apart from the taxane or vinca alkaloid binding sites, there are other binding sites on tubulin, including colchicine, laulimalide, maytansine and pironetin. Of these, the colchicine binding site targeted by lisavanbulin, should be a promising target given that most tubulin inhibitors which bind here are not substrates of MDR proteins. Additionally, these compounds often have a relatively simple structure and consequently, are relatively straightforward to manufacture. Historically, development of inhibitors (and colchicine itself) which bind to the colchicine binding site has been hampered by their low therapeutic index (CA-4 for example). Consequently, as far as we are aware, no inhibitors which bind to the colchicine site have yet been approved.

Alternative approaches to minimise resistance have been disappointing

Elsewhere, there have been significant endeavours to develop therapies which inhibit mitotic exit. Premature mitotic exit is postulated to be a major route through which cancer cells evade MTAs, so inhibitors of mitotic exit theoretically should have activity against resistant tumours. Additionally, with a limited effect on microtubules, these therapies should have a more acceptable toxicity profile, particularly with respect to peripheral neuropathy.

Aurora B kinase looks an interesting target

Furthermore, it appears that targeting mitotic exit could sensitise tumour cells to MTAs, suggesting a synergistic effect and the potential for future combinations. While there are several targets for therapeutic intervention, aurora B kinase has proven to be more tractable with several mid stage programmes ongoing, but it is fair to say that progress has been slow. AstraZeneca, for example, has been progressing its aurora B kinase inhibitor AZD2811 for some time, yet we await compelling clinical data in support of this approach. AZD2811 is currently in Phase 2 clinical development in solid tumours (small cell lung cancer) and haematological malignancies (AML).

Lisavanbulin to the rescue

Basilea's focus has been to overcome resistance issues and deliver a chemotherapy combining strong efficacy, a suitable therapeutic window, and reduced resistance issues. Lisavanbulin (formerly BAL101553) is a (highly soluble) prodrug of BAL27862, retaining its potency in human tumour models which are resistant to archetypal MTAs including the taxanes and the vinca alkaloids. BAL27862 binds to the colchicine site with distinct effects on microtubule organisation via a unique mechanism of action which importantly, is separate to that of other established MTAs. Preclinical studies have shown that BAL27862 activates the SAC arresting tumour cell proliferation in the G2/M phase of the cell cycle. As a result, it is an extremely potent inhibitor of tumour growth and a promoter of cell death.

Lisavanbulin targets to the colchicine binding site

As a small molecule, another key feature of lisavanbulin is its ability to cross the blood-brain barrier - unlike many commercially available MTAs which are natural compounds. The successful discovery and development of a small molecule inhibitor is no mean feat given that replicating the activity of natural compounds involves complex chemistry. Success here is a testament to Basilea's medicinal chemistry expertise and in-depth understanding of tumour biology. Additionally, lisavanbulin appears to possess a dual mechanism of action, inhibiting not only growth and viability of the tumour but also the vasculature feeding the tumour. While this led to some additional toxicities affecting the intravenous formulation, these have now been resolved.

Crossing the blood brain barrier important for GBM

Basilea has been assiduous in ensuring that lisavanbulin has an appropriate therapeutic window, balancing its antitumour effect with an acceptable side effect profile. Given that all commercially available MTAs are delivered parenterally, lisavanbulin may offer a potential convenience advantage given that it is orally bioavailable.

Oral administration a clear convenience advantage

Clinical development of lisavanbulin initially sought to optimise both intravenous as well as oral delivery. With early (vasculature) toxicity issues affecting the 2h infusion, Basilea has shown that delivering lisavanbulin by this route over 48h resolves this issue. Nevertheless, and despite IV administration being a well-trodden path for chemotherapy, the prospect of offering the first orally available MTA has resulted in future clinical development of lisavanbulin focussing on oral delivery. With approximately 80% of the bioavailability of the IV infusion, it is highly likely that oral

delivery of lisavanbulin should result in similar outcomes to the IV route.

Initial focus on highly aggressive brain cancer

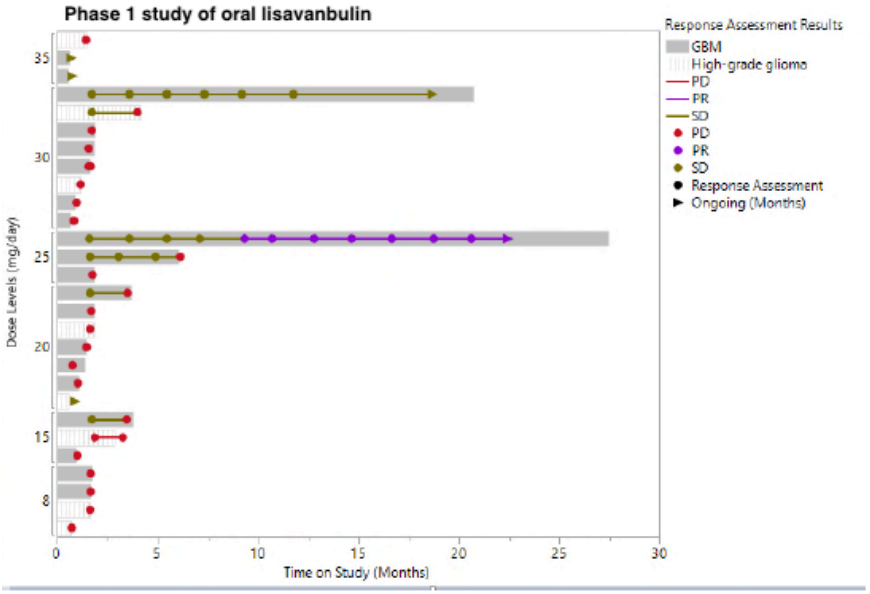
Basilea has been evaluating lisavanbulin in a broad range of cancer types, both in monotherapy as well as in combination, reflecting the applicability of the approach. With encouraging evidence of activity seen in solid tumours, the focus for now has been on the challenging glioblastoma indication. Glioblastoma multiforme is the most common malignant glioma, characterised by highly aggressive growth – its highly invasive nature accounting for the poor overall survival in sufferers. Post resection, current standard of care consists of radio-chemotherapy and concomitant temozolomide.

GBM has proven largely intractable to new treatment options

Temozolomide is a DNA alkylating agent discovered in the 1970s and approved by the FDA in 2005 to treat newly diagnosed brain cancer. However, treatment provides a disappointing median survival period of only 14.6 months, and new therapeutic approaches are urgently required. Development of novel therapies for glioblastoma has been disappointing. It will be interesting to see if Basilea's biomarker-based approach can successfully identify suitable patients. The generally received wisdom holds that the heterogeneity of the tumour itself has prevented successful drug development.

Profound responses in two patients remarkable

While our enthusiasm for lisavanbulin must be guarded given the early stage of development, the challenges of the target cancer and the limited data generated, we are highly encouraged by the data from the Phase 1 programme. Phase 1 evaluated both oral and IV administrations in glioblastoma patients (less so in ovarian cancer). These data are generated in a relatively small number of patients with the intention of identifying a suitable dose for the Phase 2 programme. Despite this we note the commentary surrounding two patients who experienced "profound" responses which were characterised as an 80% reduction in tumour size.



Source: ESMO poster 2020

Biomarker driven selection should improve probability of success

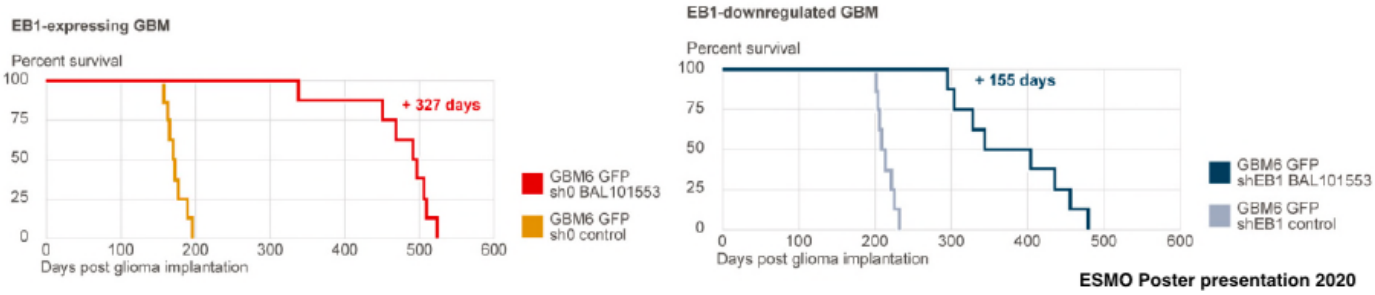
EB1 offers a realistic opportunity to identify suitable GBM patients

One of the more intriguing aspects of the lisavanbulin data is its association with a potential biomarker End-binding protein (EB1). The literature is supportive of such an approach given the importance of EB1 (along with its binding partners) in microtubule regulation. EB1 is a member of the plus-end-tracking protein family which serves to regulate microtubule dynamics. EB1 overexpression has been reported in lung, gastric, hepatic and oral cancers, suggesting that EB1 potentially serves as a biomarker for tumour progression. Its cancer-causing potential has also been noted in glioblastoma, oesophageal, and breast cancer cell lines. Consequently, there are good reasons to believe EB1 expression levels in glioblastoma are a predictive biomarker of MTA activity.

Preclinical data supportive

From Basilea's perspective, the preclinical data incorporating EB1 appears strong, with daily oral administration of lisavanbulin associated with a significant improvement in survival in an EB1 positive model of GBM. Median survival was extended by 327 days in EB1 proficient mice compared with 155 days in EB1 negative mice treated with lisavanbulin.

Effect of lisavanbulin on survival in mice with EB1 proficient or deficient GBM



ESMO Poster presentation 2020

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Clinical data although limited also supports this biomarker driven approach

In the clinic, one EB1 (highly) positive patient experienced a strong and durable response to lisavanbulin. Basilea provided additional information on this patient at ESMO 2020. This GBM patient's cancer had previously progressed rapidly after two prior lines of therapy. The patient experienced an 80% GBM area reduction and remains on study two years after start of lisavanbulin therapy. Development of therapies to treat GBM patients is clearly a challenging proposition. The use of a biomarker led strategy should help Basilea to enrich the patient population in the ongoing Phase 2 study, and hopefully increase the probability of delivering a positive result. From studies of GBM tumour biopsy tissue, EB1 positive patients represent circa 2-5% of GBM patients.

If Phase 2 trial successful clinical development risk should be lowered

Glioblastoma patients who express EB1 are presumably best placed to benefit from treatment. If EB1 turns out to be a valid biomarker in glioblastoma patients, the clinical development risk should be substantially lowered. This is an attractive proposition given the high failure rate for chemotherapy-based approaches in the treatment of glioblastoma.

Collaboration with ABTC helpful

Basilea's efforts are augmented by the collaboration with the Adult Brain Tumour Consortium (ABTC) which is funded by the US National Cancer Institute. Towards the end of 2017, the ABTC began evaluating lisavanbulin in combination with radiotherapy in newly diagnosed glioblastoma patients.

Efficacy hurdle relatively low

The data supporting the potential of lisavanbulin in glioblastoma are exciting, although in small numbers. Nevertheless, in a cancer setting where death is inevitable after a relatively short period (<5% survival after five years), the efficacy hurdle should be low, particularly where improved resistance is a potential benefit. Fortunately, it is a relatively rare cancer causing about 2% of all cancers and 17% of all brain tumours.

It has an incidence of 2-3 per 100,000 in the population, with almost 23,000 newly diagnosed patients per year in the US.

Currently too early to include in our valuation

Most patients will die within 15 months. Lisavanbulin currently sits outside our Basilea valuation, with not enough data to justify inclusion in our financial forecasts. Additionally, as things stand the use of EB1 as a companion diagnostic would suggest that approval would be reserved for the 2-5% of the GBM population where this is a relevant biomarker. Over time, we suspect that lisavanbulin could well be more relevant to a broader GBM patient population and/or to additional cancer setting where EB1 is also an important biomarker.

Long term plans for lisavanbulin will likely be based on the data

Although the use of MTAs has been overshadowed in recent years by the enthusiasm for immunotherapy-based approaches, we suspect that their use will remain for those patients where few treatment options exist. Consequently, although we have yet to see plans for lisavanbulin outside glioblastoma, assuming a positive outcome here, we would expect to see further clinical evaluation.

EB1 certainly a feature of other cancers

Importantly, EB1 is a general feature of microtubule formation generally suggesting that it may be relevant to many other cancers where previous MTAs have been effective, but their utility has been limited by emerging resistance. For example, in breast cancer cell lines, EB1 has been shown to increase the ability of MTAs (paclitaxel) to induce mitotic arrest, effectively sensitising tumour cells to this approach. EB1s own role in cancer progression remains a subject of significant scrutiny and we look forward to the results of Basliea's endeavours in the potential use of EB1 as a relevant biomarker for lisavanbulin.

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Risks

Basilea's currently marketed products are out-licensed to third parties, which suggests that the company has little influence with the end customer. That said, in the case of the anti-fungal Cresemba, Astellas and Pfizer bring market leading anti-fungal franchises with good execution to date.

While the antibiotic Zevtera is already marketed outside of the USA for the treatment of CAP and HAP (excluding VAP), we see the more significant market opportunity in the *Staph aureus* bacteraemia indication. While data from the four completed Phase III trials are supportive of this approach in those patients which suffered from a bacteraemia, there is uncertainty associated with this difficult to treat patient population. This uncertainty is reflected in the probability adjustment we have employed. At the same time, approval of ceftobiprole in the US requires a positive outcome for both the ABSSSI indication (already achieved) as well as the bacteraemia indication.

Our forecasts suggest that Basilea will self-commercialise ceftobiprole in the US and we have burdened the income statement with the costs associated with such an eventuality. On the other hand, Basilea has communicated that it will seek to attract a commercial partner for ceftobiprole in the US, and while there is a partnering risk associated with this strategy, we believe that Basilea has a successful track record in this regard.

The un-partnered oncology programmes are still relatively early stage, apart from the recently in-licensed derazantinib. While there is a risk that the oncology pipeline may be associated with development delays and potentially negative and/or inconclusive clinical trial results, the positive interim analysis in iCCA for derazantinib largely de-risks this programme in this indication. This is a highly competitive field, but we note Basilea's endeavours to differentiate derazantinib, and we would highlight the combination with the checkpoint inhibitors in urothelial cancer as a starting point.

Our financial forecasts suggest Basilea will experience several years of losses. With our expectation of a 2023 launch for ceftobiprole, our forecasts do not incorporate the expected upfront payment that the company would receive from a commercial partnership. Consequently, we are forecasting a negative cash position for several years but recognise that

there are many puts and takes to our forecasts. Additionally, we have effectively taxed the company on its first year of profits despite the observation that there are significant tax losses which will significantly reduce the tax burden in the near term.

Financial Model and Summary

Success for derazantinib relies on a differentiated profile

Basilea has invested heavily in pursuing its goal of delivering an oncology franchise, diversifying its historical reliance on anti-infectives. Clearly much relies in the near term on the future of derazantinib given it is a late-stage asset in a class (FGFR inhibitors) which has been validated by approvals elsewhere. Ultimately, success here will rely on the ability of Basilea to deliver a differentiated profile in an intensifying competitive environment.

Lisavanbulin data has been encouraging so far

Lisavanbulin represents an important endeavour, with the promise of delivering a chemotherapy with activity in glioblastoma multiforme, one of the most challenging of cancers. While the efficacy hurdle may be low, lisavanbulin needs to balance its potency with an effective therapeutic window from a class (the MTAs) which has been associated with significant toxicities. Data in a limited patient population has been highly encouraging and we look forward with keen anticipation to the results from the forthcoming Phase 2 trial. Importantly, Basilea appears to have identified a biomarker (EB1) which should help enrich the patient population and increase the probability of success, albeit this represents a modest proportion (2-5%) of the GBM population. A future path for lisavanbulin will clearly depend on the data and whether there is also applicability to a broader patient population with GBM and/or in additional cancers where EB1 has been shown to be a valid prognostic biomarker.

EB1 biomarker should increase the probability of success

Lisavanbulin could be relevant to a wider GBM population or to other cancers

Despite its promise, lisavanbulin currently sits outside of our financial model and valuation for Basilea. Clearly GBM is a challenging cancer with many approaches failing to provide any real benefit. At the same time, the use of EB1 should increase the probability of success. Ultimately, the market potential for lisavanbulin could be substantial should it be relevant to a wider patient population in GBM, or if the EB1 driven approach is relevant to a broader range of cancers.

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Income Statement (CHF '000s)

Year to December	2019A	2020A	2021E	2022E	2023E	2024E	2025E
Total revenue	**134381**	**127629**	**132817**	**139145**	**169090**	**238005**	**309933**
COGS	(18,868)	(24,054)	(25,235)	(22,263)	(21,982)	(30,941)	(40,291)
Gross profit	**115,513**	**103,575**	**107,581**	**116,882**	**147,108**	**207,065**	**269,642**
Gross margin	86.0%	81.2%	81.0%	84.0%	87.0%	87.0%	87.0%
R&D	(102,662)	(97,410)	(96,956)	(97,401)	(92,999)	(97,582)	(102,898)
SG&A	(30,051)	(29,422)	(31,876)	(32,003)	(40,582)	(45,221)	(55,788)
Total cost and operating expenses	(151,581)	(150,886)	(154,067)	(151,668)	(155,563)	(173,744)	(198,977)
Non-underlying items	0.00	15,035	(21,251)	(12,523)	13,527	64,261	110,956
Operating profit US GAAP	**(17,200)**	**(8,222)**	**(21,251)**	**(12,523)**	**13,527**	**64,261**	**110,956**
Finance income	28	104	0	161	167	119	(13)
Finance expense	(6,424)	(7,589)	(7,456)	(7,480)	(3,225)	(3,225)	(3,225)
Other financial income	1,583	2,057	0	0	0	0	0
Other financial expense	(369)	(1,017)	0	0	0	0	0
Underlying PBT	(22,382)	(29,702)	(28,707)	(19,842)	10,469	61,156	107,718
PBT IFRS	(22,382)	(14,667)	(28,707)	(19,842)	10,469	61,156	107,718
Loss before tax	(22,382)	(29,702)	(28,707)	(19,842)	10,469	61,156	107,718
Tax	(40)	(55)	(60)	(60)	(1,487)	(8,684)	(15,296)
Underlying net income	**(22,422)**	**(29,757)**	**(28,767)**	**(19,902)**	**8,983**	**52,472**	**92,422**
Net income US GAAP	**(22,422)**	**(14,722)**	**(28,767)**	**(19,902)**	**8,983**	**52,472**	**92,422**
EPS Basic (CHF)	(2.09)	(1.43)	(2.80)	(1.94)	0.87	5.10	8.99
EPS Diluted (CHF)	**(2.08)**	**(1.36)**	**(2.80)**	**(1.94)**	**0.87**	**5.10**	**8.99**

Source: Calvine Partners Research

Balance Sheet (CHF 000s)

Year to December	2019A	2020A	2021E	2022E	2023E	2024E
Non-current assets						
Tangible assets, net	5,162	2,627	6,246	6,871	7,558	8,313
Intangible assets, net	372	672	672	672	672	672
Long-term investments	30,000	0	0	0	0	0
Other non-current assets	1,073	2,967	2,967	2,967	2,967	2,967
Total non-current assets	36,607	6,266	9,885	10,510	11,197	11,952
Current Assets						
Cash and cash equivalents	109,024	60,749	93,858	(51,663)	(50,214)	(13,160)
Short-term investments	22,020	106,530	25,507	0	0	0
Accounts receivable	6,242	8,710	6,590	7,653	9,300	13,090
Other receivables	22,053	23,684	23,684	23,684	23,684	23,684
Inventories	18,569	21,192	23,963	23,182	28,170	39,652
Other current assets	6,952	2,663	2,663	2,663	2,663	2,663
Total current assets	184,860	223,528	176,265	5,519	13,604	65,929
Total assets	**221,467**	**229,794**	**186,150**	**16,028**	**24,800**	**77,881**
Current liabilities						
Accounts payable	6,765	13,151	5,499	5,385	5,174	5,784
Deferred revenue	32,873	2,556	0	0	0	0
Accruals and other current liabilities	35,856	34,454	34,454	34,454	34,454	34,454
Total current liabilities	75,494	50,161	39,953	39,839	39,628	40,238
Non-current liabilities						
Convertible senior unsecured bonds	197,740	239,668	249,340	99,234	99,234	99,234
Deferred revenue, less of current portion	16,471	13,158	0	0	0	0
Other non-current liabilities	24,722	28,853	28,853	28,853	28,853	28,853
Total non-current liabilities	238,933	281,679	278,193	128,087	128,087	128,087
Total liabilities	**314,427**	**331,840**	**318,146**	**167,926**	**167,715**	**168,325**
Shareholders equity (deficit)						
Share capital	11,882	11,922	11,922	11,922	11,922	11,922
Additional paid-in capital	927,342	982,438	982,438	982,438	982,438	982,438
Accumulated other comprehensive loss	(24,555)	(27,252)	(27,252)	(27,252)	(27,252)	(27,252)
Treasury shares held by a subsidiary	(5,963)	(52,766)	(52,766)	(52,766)	(52,766)	(52,766)
Loss carried forward	(979,244)	(1,001,666)	(1,016,388)	(1,045,155)	(1,065,057)	(1,056,074)
Net loss for the year	(22,422)	(14,722)	(28,767)	(19,902)	8,983	52,472
Total shareholders' equity (deficit)	(92,960)	(102,046)	(130,813)	(150,715)	(141,732)	(89,261)
Total liabilities and equity (deficit)	**221,467**	**229,794**	**187,333**	**17,211**	**25,983**	**79,064**

Source Calvine Partners Research

Disclosures

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