EDISON

e-Therapeutics

Business update

Nearing prime time validation

Pharma & biotech

e-Therapeutics' (ETX) Network-Driven Drug Discovery (NDD) platform has begun to deliver encouraging data that should support out-licensing of its immuno-oncology and hedgehog (Hh) projects with potential partners. Securing deals on the pipeline and the NDD platform are clear priorities for the company and having strong data in important new therapeutic areas will raise its profile. Although these are early-stage data, the company is moving in the right direction and meeting its targets. Our comparative venture capital (VC) methodology suggests an indicative value of £41.9m.

Year end	Revenue (£m)	PBT* (£m)	EPS* (p)	DPS (p)	P/E (x)	Yield (%)
01/16	0.0	(11.1)	(3.3)	0.0	N/A	N/A
01/17	0.0	(13.4)	(3.9)	0.0	N/A	N/A
01/18e	0.0	(7.2)	(2.1)	0.0	N/A	N/A
01/19e	0.0	(7.2)	(2.1)	0.0	N/A	N/A

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

Validating Network-Driven Drug Discovery (NDD)

e-Therapeutics has accumulated substantial data packages in its hedgehog project and two other immuno-oncology (I/O) programmes in checkpoint signalling modulation and tryptophan catabolism. In our opinion the initial data, and the generation of lead-like compounds, begin to validate the NDD platform as a highly efficient and productive discovery system and should support partnering/out-licensing. ETX has strategically focused on areas of significant commercial interest so that further elucidation of mechanisms of action and strengthening of the data packages could allow any of these programmes to be out-licensed. The pharmaceutical industry also recognises that *in silico* technologies can improve the efficiency and productivity of the discovery process. ETX's technologies are thus also well positioned for platform-related deals in specific areas of biology.

Waiting for the right deal

In the six months since the strategic review, the portfolio and activities have been rationalised and the company is focused on its key I/O programmes. ETX's internal assets and leads, albeit at an *early discovery stage*, are in 'hot' new therapeutic areas where bigger pharmaceutical partners are currently licensing. Business development efforts are actively underway by the new management and we expect these will bear fruit in 2018. The company clearly wants to do the right deal rather than just any deal.

Valuation: Made easier by comparison

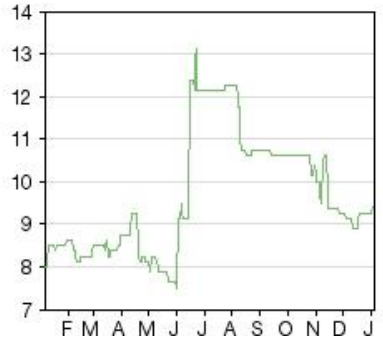
The valuations of loss-making preclinical platform companies are always difficult. We have used a later-stage NDD comparator from the US, Merrimack Pharmaceuticals. While both companies are public, we have estimated the clinical development time needed for ETX to reach the pipeline maturity of Merrimack at its US IPO and to date. We have used a discount rate of 12% and Lerner's VC valuation method, which results in an indicative valuation of £41.9m.

5 January 2018

Price	**9.40p**
Market cap	**£25m**

Net cash (£m) at 31 July 2017	12.4
Shares in issue	268.5m
Free float	74%
Code	ETX
Primary exchange	AIM
Secondary exchange	N/A

Share price performance



%	1m	3m	12m
Abs	3.0	(11.5)	17.5
Rel (local)	(1.6)	(14.3)	8.3
52-week high/low		13.1p	7.5p

Business description

e-Therapeutics is a UK-based drug discovery company that has developed an in silico network-driven drug discovery platform. Following a strategic review by the new CEO, the focus is now on commercialisation: securing partners for its platform, discovery and development projects.

Next events

Further preclinical data	2018
Out-licensing of I/O assets	2018
Deals on NDD platform	2018

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Edison profile page

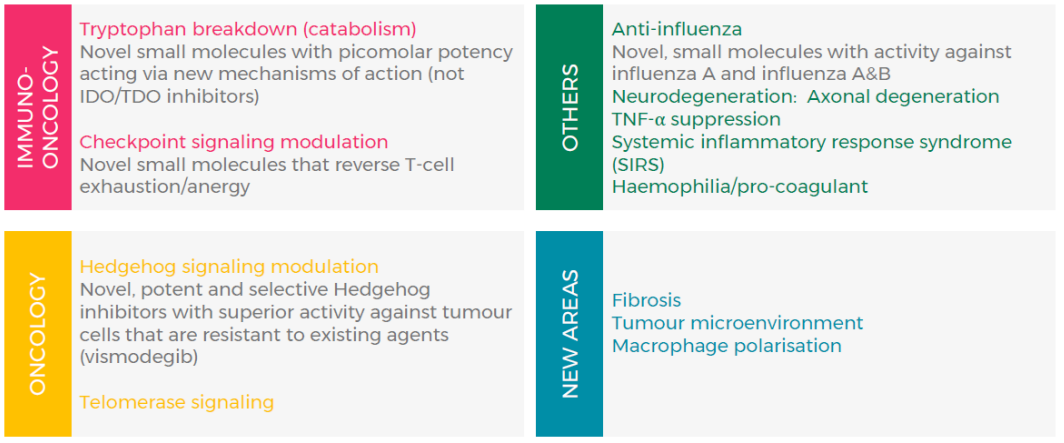
Company description

e-Therapeutics is a UK-based drug discovery company built around a proprietary network-driven *in silico* drug discovery platform, which has to date generated 17 different discovery programmes. The company was founded in 2003 as a technology spin-out from Newcastle University and listed on AIM in 2007, but the current incarnation of the platform has been in operation since 2014. Secondary offerings in 2011 and 2013 totalling £57m facilitated the development, strengthening and industrialisation of its network analysis platform from prototype to a "fully operational, highly engineered and efficient discovery engine". A dedicated drug discovery hub opened in 2012 in Oxford to expand its drug discovery capabilities; e-Therapeutics employs around 20 staff. Dr Raymond Barlow, formerly of Amgen, was appointed CEO in April 2017 and carried out a full strategic/operational review of the business, which focused the company on the most advanced and commercially interesting discovery assets (see below for more detail). This refocus should maximise the company's chances of securing commercial deals with pharma partners (for the platform and product leads), which would then progress these assets into the clinic and beyond.

Technology validated; portfolio refocus

Dr Barlow undertook a root and branch review of the business in July 2017 and enlisted the views of a panel of independent industry experts from big pharma and biotech companies. The review confirmed the utility of the NDD platform approach in terms of the potential for shorter times from concept to hits and the number/quality of output molecules. Financial and commercial imperatives resulted in a rationalisation of the portfolio. Internal resources were focused on the two immune-oncology (I/O) programmes, further investment in the NDD platform and on the application of the platform in areas of high unmet commercial need such as fibrosis.

Exhibit 1: e-Therapeutics areas of interest



Source: e-Therapeutics presentation

Six months on from the review, e-Therapeutics has generated extensive data packages on the two lead preclinical I/O programmes in checkpoint signalling modulation and tryptophan catabolism, which it hopes to out-license. A further hedgehog signalling programme has been used as an exemplar to validate the NDD platform and provide proof that the platform can generate novel, potent leads with activity superior to current agents.

What is the Network-Driven Drug Discovery platform?

At the core of e-Therapeutics is an *in silico* discovery engine consisting of **a battery of computational tools** and large-scale proprietary biological databases (enhanced by artificial intelligence (AI)/machine learning) that enable construction and analysis of biological networks.

ETX develops **proprietary models of complex disease processes** that it wants to disrupt, and uses its discovery engine to identify vulnerable points in the underlying protein interaction networks. It then matches these vulnerabilities to the direct and indirect biological activities of small molecules to find those compounds that will have the greatest impact.

The platform incorporates **multiple data sources** including protein-protein interactions, pan-omics information (from genomics, proteomics, metabolomics and transcriptomics), compound and protein structural data and compound-protein binding data.

Using its **AI-enhanced bioactivity footprints** ETX is able to screen and rank millions of individual compounds *in silico* for their impact on disease network integrity. At the compound triage stage, the most disruptive compounds with the best biological footprint are further screened for desirable 'hit-like' pharmacokinetic and pharmacological characteristics (eg low cytotoxicity, stability). In ETX's experience, this has typically resulted in a final deck of 300-1,700 compounds per programme, falling into 4-20 chemical groups, or chemotypes. The final deck undergoes phenotypic screening for activity in physiologically relevant *in vitro* cellular assays. In the hit-to-lead stage/lead optimisation stage, the phenotypic hits undergo medicinal chemistry optimisation to generate leads.

Advantages of the NDD approach

Disease process driven not target driven: the approach employs an understanding of disease network biology and hypotheses surrounding the disease process. It avoids the need to assume a drug target at the outset and can generate hits with a novel mode of action (for example the checkpoint signalling and tryptophan catabolism programmes have led to novel targets). ETX has validated the approach across 12 disease/biology areas to date, demonstrating its versatility.

Speed: the *in silico* and disease-focused nature of the platform enables it to generate quality hits in under nine months from hypothesis to compound testing (as demonstrated by the tryptophan catabolism programme). This is combined with a **high hit rate**: between 2% and 11% of compounds discovered have activity of <10μM across multiple, parallel phenotypic screens.

Intellectual property benefits are likely because the generation of diverse chemotypes (molecules in related chemical series) and the discovery of first-in-class drugs with a novel mechanism of action increase the likelihood of unique IP within the output.

In reality, this means that a partner could come to ETX with a disease phenotype that it wants to address and ETX could use its disease network models and *in silico* engine to identify therapeutic compounds with potentially novel structure(s) and mechanism(s) of action compared to currently available drugs.

Exhibit 2: NDD platform demonstrates high hit rate

Project	Compounds screened	% 'hits' in phenotypic screens	Number of chemotypes in hits
Telomerase signalling	393	4.3%	6
Hedgehog pathway	1,146	5.5%	20
TNF-a release	356	7.3%	12
Influenza replication	1,048	2.2%	4
Tryptophan catabolism	273	11.0%	4
Systemic inflammatory response syndrome	292	11.0%	4
Axonal degeneration	1,696	3.4%	9
Reversal of T-cell exhaustion	483	ongoing	-

Source: ETX data 2017

Hedgehog pathway project

Current hedgehog pathway inhibitors plagued by resistance

The hedgehog (Hh) pathway is a major regulator of many processes in embryonic development including stem cell maintenance, cell differentiation and cell proliferation. Dysfunction of the pathway underlies certain cancers (particularly basal cell carcinoma [BCC] and medulloblastoma) and targeting the Hh signalling pathway provides a new therapeutic option. To date efforts have focused on targeting the Smoothened (SMO) transmembrane receptor protein and the most advanced agents are Roche's Erivedge (vismodegib) approved for locally advanced or metastatic BCC in 2012 and Novartis's Odomzo (sonidegib), approved in 2015. The main issue with current SMO inhibitors is the development of mutations and resistance by tumours.

Potent leads identified with no SMO involvement

ETX's goal was therefore to identify hedgehog inhibitors with a reduced tendency to induce SMO resistance and demonstrate activity in tumours known to be resistant to vismodegib/sonidegib.

The NDD approach, followed by synthesis of novel compounds using standard chemistry, has generated two lead compounds whose activity *in vitro* is equivalent to the approved agents. In assays using vismodegib-resistant tumour lines, the ETX compounds demonstrated greater potency than vismodegib, and *in vivo*, their activity was equivalent or superior to sonidegib.

Primarily the data package generated for the hedgehog pathway signalling inhibitor programme demonstrates that the NDD platform is an effective discovery platform. However, it has generated at least two novel candidates for which patents have now been filed and which may have the potential to be out-licensed. ETX is not currently investing more in the programme but is exploring all avenues, including seeking external sources of funding or out-licensing.

Tryptophan catabolism programme

Rationale for targeting tryptophan breakdown

The amino acid tryptophan plays a key role in regulating normal immune tolerance, such as seen in the placenta, and in suppressing excessive inflammatory responses. Tryptophan breakdown (catabolism) to kynurenine is regulated by two enzymes: indoleamine-2,3-dioxygenase (IDO), and tryptophan-2,3- dioxygenase (TDO). Tryptophan depletion induces signalling events via the GCN2 pathway that lead to T-cell anergy and apoptosis. The accumulation of kynurenine also leads to T-cell anergy as well as increased survival and motility of tumour cells.

Cancer cells are thought to hijack this process by overexpressing IDO and/or TDO and creating an immunosuppressive microenvironment. Several inhibitors of IDO/TDO are being investigated as cancer therapies in combination with immune checkpoint inhibitors against CTLA-4, PD-1 or PD-L1.

IDO inhibitors: Most advanced is epacadostat in Phase III

The most advanced IDO inhibitor is Incyte's epacadostat. It has been studied in combination with approved immune checkpoint inhibitors (CTLA-4 inhibitor ipilimumab/Yervoy, the PD-1 inhibitors pembrolizumab/Keytruda and nivolumab/Opdivo, or PD-L1 inhibitor durvalumab/Imfinzi) and shown proof-of-concept across a range of solid tumour types, with improved response rates compared with the checkpoint inhibitors alone. Epacadostat has started Phase III trials in melanoma with pembrolizumab and will enter Phase III in non-small cell lung cancer (NSCLC), renal, bladder, and head and neck cancers. Less promising results were seen in triple-negative breast cancer and ovarian cancer, and these indications are not being advanced.

Newlink Genetics has an IDO-1 inhibitor programme headed by indoximod. It has completed Phase II trials in melanoma, acute myeloid leukaemia, pancreatic and prostate cancer. Phase III (enrolling by the end of 2018) will evaluate indoximod in combination with pembrolizumab and nivolumab in advanced melanoma. NLG 802, a prodrug of indoximod, is in a small Phase I trial in solid tumours reporting in 2018. NLG 919 was discontinued in 2017 by Roche, which is instead focusing on a preclinical dual IDO/TDO inhibitor licensed from Curadev in 2015.

Bristol-Myers Squibb's BMS 986205 has just started a Phase III trial (NCT03329846) with nivolumab in advanced melanoma.

ETX has generated novel hits as potent as epacadostat

ETX's goal was to look for small molecule modulators of tryptophan catabolism with a novel mechanism of action and potentially with superiority to current IDO inhibitors. Any compounds would almost certainly be used in combination with existing agents, such as the immune checkpoint inhibitors, and will need to demonstrate synergistic anti-tumour effects without increasing side effects.

By way of validating its discovery pathway, ETX compared its network analysis approach with the standard structural homology approach (generating 2D and 3D homologues of current agents). Starting with 14 hypothetical networks of tryptophan catabolism, ETX generated 273 compounds, of which 29 (10.6%) were positive hits in phenotypic assays. This compared favourably with 8/282 (2.8%) positive hits using structural homology. The most potent compounds in the series were refined over nine months and the results were superior to epacadostat and BMS 986205 in *in vitro* assays (with inhibition of tryptophan catabolism in cellular assays by some compounds at sub-nanomolar levels). *In vivo* activity in mouse models of plasma kynurenine inhibition was equivalent to that seen with epacadostat.

As a result, ETX now has two potent compounds with lead-like characteristics, which will continue to undergo lead optimisation and medicinal chemistry refinement. The mechanism of action of these compounds is being investigated but appears novel (not direct IDO-1 inhibition like epacadostat) and therefore offers potential IP advantages.

Checkpoint signalling modulation programme

A paradigm shift in immunotherapy: Immune checkpoint inhibitors

The regulation of T-cell mediated immunity involves many co-stimulatory and co-inhibitory receptors. Co-inhibitory receptors, referred to as immune checkpoints, act via complex signalling pathways as an "off switch" to downregulate T-cell activity – by inducing T-cell exhaustion, apoptosis (programmed cell death) or anergy, and thereby suppressing normal T-cell functions (proliferation, cytokine production and T-cell driven killing of tumour cells). This is a desirable process for the elimination of self-reactive T-cell clones and for limiting the extent of a normal immune response.

By expressing particular ligands or receptors, tumours utilise immune checkpoint pathways to induce T-cell dysfunction and evade the immune response. Antibodies that block these checkpoints have revolutionised cancer immunotherapy in recent years with the success of inhibitors of CTLA-4 and PD-1 in melanoma, NSCLC, head and neck cancer, renal cell carcinoma, and Hodgkin's lymphoma (Exhibit 3). Other checkpoint targets, including OX40, LAG-3, TIGIT, TIM-3, and BLTA, are in early-stage investigation (Exhibit 4).

Checkpoint inhibition is competitive, but room for improved agents

Although the field is crowded, there is room for improvement: clinical response rates range from 10-60% and there are significant side effect issues with some of the antibody therapies. Frequent immune system-related adverse events associated with the current CTLA-4 and PD-1 inhibitors include pruritus, extreme skin rash, nausea, diarrhoea and thyroid disorders. In addition to aiming for improved tolerability, there would be substantial interest in small molecule therapies that could pass through the blood-brain barrier (unlike conventional antibodies).

Exhibit 3: FDA approved immune checkpoint inhibitors

Drug	Checkpoint target	Indication (date of approval)
ipilimumab Yervoy (BMS)	CTLA-4	Unresectable metastatic melanoma (2011) Stage III melanoma as adjuvant (2015) BRAF melanoma, in combination with nivolumab (2015) Advanced/unresectable melanoma (2014) Metastatic NSCLC (2015)
pembrolizumab Keytruda (MRK)	PD-1	Recurrent SCCHN (2016) Hodgkin's lymphoma (2017) First-line treatment of metastatic NSCLC (2017) Urothelial cancer (2017) Microsatellite instability-high cancer (2017) Gastric cancer (2017) In Phase II for multiple myeloma
nivolumab Opdivo (BMS)	PD-1	Unresectable or metastatic melanoma (2014) Metastatic NSCLC (2015) Metastatic RCC (2015) BRAF melanoma, in combination with ipilimumab (2015) Hodgkin's lymphoma (2016) Metastatic SCCHN (2016) Urothelial cancer (2017) Metastatic colorectal cancer (2017) Hepatocellular carcinoma (2017)
atezolizumab Tecentriq (Roche/Genentech)	PD1	Metastatic NSCLC (2015) Urothelial cancer (2016) In Phase II for lung, Phase III ovarian

Source: Edison Investment Research, company data

Exhibit 4: New immune checkpoint inhibitors in development (selected)

Drug	Checkpoint target	Development information
pidilizumab (Medivation acquired by PFE in 2016)	Possibly PD-1 (unclear)	N/K
MGA012 (MacroGenics)	PD--1	In Phase I, licensed to Incyte October 2017
MGD013 (MacroGenics)	both PD-1 and LAG-3 (DART)	Started Phase I September 2017
tremelimumab (AZN)	CTLA 4	Colon (Ph I), Hepatocellular carcinoma (Ph I), Melanoma (Ph I), Breast (Ph II). Failed Ph IIb in mesothelioma, promising Ph Ib data in NSCLC with durvalumab
enoblituzumab/MGA271 (MacroGenics)	B7-H3	In Ph I in combo with ipilimumab in several cancers (completes 2018) and pembrolizumab (completes 2020)
MGD009 (MacroGenics)	B7-H3	In Phase I
IMP321 (Prima BioMed)	LAG-3	Phase IIb data in metastatic breast cancer
BMS-986016 (BMS)	LAG-3	Phase I in glioblastoma (in combination with nivolumab)
TSR-022 (Tesaro)	TIM-3	Phase I in advanced solid tumours
MBG453 (Novartis)	TIM-3	Phase Ib/II alone and in combination with anti-PD-1 in advanced cancers

Source: Edison Investment Research, company data. Note: LAG-3: Lymphocyte activation gene-3 (CD223); DART: Dual-Affinity Re-Targeting molecule.

Multiple network models identify hits with novel mode of action

ETX's aim was to find small molecule modulators able to reverse T-cell exhaustion, stimulate cytokine production and drive CD8 killing of tumour cells *in vitro*. The complex, and incompletely elucidated, signalling pathways associated with anergy and tolerance in T-cells provide an ideal substrate for the NDD engine. ETX constructed multiple network models *in silico* of the T-cell 'braking system' using known and predicted checkpoint signalling data, and corresponding models of the regulation of ligands expressed on tumour cells, each model acting as a separate

'experiment'. The bioactivity footprints of known checkpoint modulators were also used as data inputs.

The project is ongoing but after screening around 500 compounds, positive hits in several distinct chemical series have been generated to date, of which two are being progressed currently. These compounds activated T-cell killing of tumour cells *in vitro* at a level equivalent/superior to Keytruda plus Yervoy and gave positive results in assays of T-cell exhaustion and cytokine production. ETX has evidence for two distinct mechanisms of action, which are now being explored more fully in medicinal chemistry. Future development will include hit-to-lead generation, and the further elucidation of mechanism of action (MoA) and the targets.

Next steps: Deals, data, deals

Supported by a growing package of data and development of the platform, management will continue to be fully focused on business development and commercialisation of its assets throughout 2018.

ETX intends to develop the leads from its two I/O programmes (tryptophan catabolism and checkpoint inhibition) to late preclinical stage itself, but will look for an out-licensing partner to fund the more costly clinical development going forwards. Both programmes continue to generate data that will be published once commercially protected. Whether or not the hedgehog programme is out-licensed remains to be seen and we do not assume any revenue from this in our models.

Other opportunities being explored include deals involving the NDD platform whereby a partner defines a disease phenotype (but not necessarily targets) and biological output of interest, enabling ETX to develop network models and apply its computational tools to generate hits.

Work is also ongoing to develop the platform, to expand use of AI and incorporate patient segmentation data to enable segment-specific network analysis. Development of additional disease networks is also underway, including models for fibrosis, and aspects of tumour microenvironment and neurodegeneration.

Indicative valuation

For early-stage preclinical platform companies, the valuation is always going to have a healthy dose of subjectivity until the first product is licensed and investors get a sense of the external validation of the potential for the platform. However, we believe US-based Merrimack Pharmaceuticals is a reasonable comparator in determining an indicative valuation for ETX using a VC valuation approach as a first step. Both Merrimack and ETX are public companies in the systems biology and oncology therapeutic spaces.

Early-stage assets leads to a VC valuation method

Venture capitalists often have to value early-stage preclinical platform companies to determine a valuation at which they are comfortable investing. The Harvard academic Josh Lerner has published on this method[1] and we have used this method to value ETX.

- We have taken the January 2018 $145.4m valuation of a close comparator, Merrimack Pharmaceuticals, from the point of its IPO and then discounted to date. Merrimack Pharmaceuticals was founded by scientists from MIT and Harvard University in 2000. It develops novel cancer therapeutics based on knowledge of cancer systems biology and

[1] Venture Capital & Private Equity. A Casebook. Volume 2, Josh Lerner and Felda Hardymon. John Wiley & Sons, 2002, page 216.

systems pharmacology. It currently has three products in early clinical trials and several preclinical assets. Clinical trials are designed around biomarker-driven rationales.

- At the point of its IPO in 2012, Merrimack had three compounds in clinical development and a collaboration with Sanofi. In order to 'align' ETX to Merrimack's stage of development at the time of its IPO, we have estimated the time for ETX to have a similar development portfolio and then further discounted back that value to today. ETX may be able to start clinical development at in 2019, so as a starting point in Lerner's valuation formula we assume it would take ETX seven years to reach a similar stage as Merrimack at IPO. We can vary this time parameter, as shown in our sensitivity table.
- We have used a discount rate of 12% in the valuation formula since ETX is a public company and typically VCs use a much higher rate as a target rate of return.
- Like ETX, Merrimack has restructured since its IPO and has changed its CEO but, unlike ETX, Merrimack has seen a Phase III failure. We have included an additional 20% discount in our valuation to take into account the difference in market listings as Merrimack is listed on the much more liquid NASDAQ market and ETX on AIM.

VC valuation methods needs few inputs

Our valuation of ETX uses Merrimack's valuation at the time of its US IPO, a discount rate of 12% (adjusted by a further 20% to reflect Merrimack's listing on the more liquid NASDAQ exchange) and assumes seven years for ETX to reach the same stage as Merrimack at its IPO in 2012, and then discount by a further five years to Merrimack's current valuation. This results in a valuation of £41.9m. The inputs of time to 'equivalent portfolio maturity' and the discount rate are key drivers in the VC valuation, but are clearly subjective, so we have conducted a sensitivity analysis, shown in Exhibit 5 below. While MMAK is a fairly reasonable comparator to ETX, it is also worth noting that the valuation time point input is highly dependent on market conditions and investor sentiment towards the sector at the time, but represents a starting point. It is further important to note that Merrimack has a significantly higher valuation at its IPO than it does in January 2018.

Business development efforts could change everything

While investors are keenly waiting for the results of ETX's business development efforts, which would validate the NDD platform, the first deal would also change the valuation of ETX. As well as the value of any upfronts and milestones, the buy-in by a big pharma company could reduce the time for ETX to reach the stage that Merrimack was at its IPO and increase our valuation.

Sensitivities

We have used the key valuation drivers in Lerner's VC valuation methodology to flex the valuation of ETX. Should the time to 'equivalent portfolio maturity' be reduced to nine years, or extended to 15 years, our valuation would change to £54.05m or £32.42m, respectively.

We have used a reasonably aggressive discount of 12% for ETX as a public company. VCs use much higher rates, but with the objective of realising investments at a specific target rate of return. Should ETX's business development efforts result in a licensing deal, we may revisit the discount rate. In our sensitivity analysis, discount rates of 10% and 16% result in valuations of £49.34m and £30.34m, respectively.

Exhibit 5: Sensitivity of valuation to time to maturity and discount rate

		Target rate of return (discount rate)									
		6%	8%	10%	12%	14%	16%	18%	20%	22%	24%
Years	8	82.14	73.38	65.67	58.86	52.82	47.48	42.74	38.52	34.76	31.42
	9	78.65	69.28	61.14	54.05	47.86	42.45	37.70	33.54	29.89	26.67
	10	75.30	65.41	56.92	49.64	43.36	37.95	33.27	29.22	25.70	22.65
	11	72.10	61.74	53.00	45.58	39.29	33.93	29.36	25.45	22.10	19.23
	12	69.02	58.30	49.34	41.86	35.60	30.34	25.90	22.17	19.01	16.33
	13	66.09	55.03	45.94	38.45	32.25	27.12	22.86	19.30	16.34	13.86
	14	63.28	51.95	42.77	35.30	29.22	24.25	20.17	16.82	14.05	11.77
	15	60.58	49.05	39.82	32.42	26.47	21.68	17.79	14.65	12.08	9.992

Source: Edison Investment Research

Financials: Continued cost control

ETX's management is focused on tight cost control of the rationalised pipeline (and winding down a small legacy clinical study), plus continued investment into the NDD platform. We have made slight changes to our numbers following the interims in October 2017 to reflect the tighter cost control. We now forecast total spend in FY18 of £7.4m (previous forecast £7.9m) and an operating loss of £7.4m (previously £7.9m). With cash burn for FY18 estimated at £5.2m (previously £5.7m), and year-end cash of £8.8m, we believe ETX should have sufficient funds to operate into 2019.

Exhibit 6: Financial summary

£'000s	2016	2017	2018e	2019e
Year ending 31 January	IFRS	IFRS	IFRS	IFRS
PROFIT & LOSS				
Revenue	0	0	0	0
R&D	(9,965)	(10,911)	(5,488)	(5,500)
G&A	(1,375)	(2,614)	(1,800)	(1,700)
EBITDA	(11,267)	(13,469)	(7,243)	(7,164)
Operating profit (before amort. and except.)	(11,340)	(13,525)	(7,288)	(7,200)
Share-based payment	(215)	(99)	(120)	(100)
Operating profit	(11,555)	(16,429)	(7,408)	(7,300)
Net interest	271	132	50	40
Profit before tax (adjusted)	(11,069)	(13,393)	(7,238)	(7,160)
Profit before tax (as reported FRS3)	(11,284)	(16,297)	(7,358)	(7,260)
Tax	2,464	3,073	1,500	1,400
Profit after tax (norm.)	(8,605)	(10,320)	(5,738)	(5,760)
Profit after tax (as reported)	(8,820)	(13,224)	(5,858)	(5,860)
Average number of shares outstanding (m)	264.4	267.1	268.4	268.4
EPS - adj. (p)	(3.3)	(3.9)	(2.1)	(2.1)
EPS - as reported (p)	(3.3)	(5.0)	(2.2)	(2.2)
Dividend per share (p)	0.0	0.0	0.0	0.0
EBITDA margin (%)	N/A	N/A	N/A	N/A
Operating margin (before GW and except) (%)	N/A	N/A	N/A	N/A
BALANCE SHEET				
Fixed assets	804	207	247	296
Intangible assets	740	156	159	166
Tangible assets	64	51	89	131
Current assets	28,783	17,724	10,593	4,889
Stocks	0	0	0	0
Debtors	3,941	3,749	1,800	1,800
Cash	24,842	13,975	8,793	3,089
Other	0	0	0	0
Current liabilities	(1,156)	(1,951)	(915)	(915)
Creditors	(1,156)	(1,951)	(915)	(915)
Other creditors	0	0	0	0
Short-term borrowings	0	0	0	0
Long-term liabilities	0	0	0	0
Long-term borrowings	0	0	0	0
Deferred taxation	0	0	0	0
Other long-term liabilities	0	0	0	0
Net assets	28,431	15,980	9,925	4,270
CASH FLOW				
Operating cash flow	(11,204)	(11,711)	(8,156)	(7,164)
Net interest	329	194	91	45
Tax	2,027	2,570	2,968	1,500
Capex	(6)	(22)	(60)	(60)
Purchase of intangibles	(138)	(143)	(25)	(25)
Acquisitions/disposals	0	(1,768)	0	0
Financing	12	13	0	0
Dividends	0	0	0	0
Other	0	0	0	0
Net cash flow	(8,980)	(10,867)	(5,182)	(5,704)
Opening net debt/(cash)	(33,822)	(24,842)	(13,975)	(8,793)
HP finance leases initiated	0	0	0	0
Other	0	0	(0)	0
Closing net debt/(cash)	(24,842)	(13,975)	(8,793)	(3,089)

Source: Company data, Edison Investment Research

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