EDISON

IRLAB Therapeutics

Focused on novel Parkinson's disease treatments

IRLAB Therapeutics is focused on developing novel, potential first-in-class treatments for the symptoms of Parkinson's disease (PD). PD is a complex and progressive neurodegenerative condition with huge unmet need. IRLAB has two lead assets in Phase II trials: mesdopetam (D3 antagonist) and pirepemat (PFC enhancer). Both assets have a unique MOA and were developed through its proprietary technology ISP platform to address a wide spectrum of symptoms such as levodopa-induced dyskinesias (PD-LIDs), psychosis and PD-Falls. Top-line data from the mesdopetam US/EU Phase IIb/III trial in PD-LIDs, expected in H122, will define the pivotal trials required for approval. We value IRLAB at SEK4.8bn.

Year end	Revenue (SEKm)	PBT* (SEKm)	EPS* (SEK)	DPS (SEK)	P/E (x)	Yield (%)
12/19	0.4	(95.1)	(2.34)	0.0	N/A	N/A
12/20	0.4	(91.4)	(1.92)	0.0	N/A	N/A
12/21e	0.4	(107.6)	(2.08)	0.0	N/A	N/A
12/22e	0.4	(158.5)	(3.06)	0.0	N/A	N/A

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

PD treatment represents a high unmet need

PD is characterised by a triad of cardinal motor symptoms, although non-motor symptoms (eg psychosis, dementia and cognitive impairment) are as debilitating and remain undertreated. Despite substantial efforts to develop disease-modifying approaches in PD, symptomatic treatment remains the mainstay. Standard-of-care, L-DOPA improves motor symptoms but is associated with troublesome side effects including PD-LIDs. IRLAB's in-house R&D pipeline holds early promise to bring about a paradigm shift in the identified treatment priorities in PD, eg mesdopetam could reduce dyskinesias and thus extend daily time with good and controlled mobility, so-called 'good ON-time', and potentially maximise L-DOPA therapy.

Portfolio approach ISP platform

IRLAB's proprietary drug discovery ISP platform is designed to select high-quality drug candidates rapidly for an array of tough-to-drug disease targets in neurodegenerative conditions. We expect ongoing discovery work to focus on bringing further novel drugs into clinical development in PD and potentially for Alzheimer's disease. In the near term, progress of key assets serves as validation of its platform. IRLAB is funded to key inflection points in 2022 of proof-of-concept data for both mesdopetam and pirepemat. After this we expect partnering deals to facilitate Phase III development and potential commercialisation strategies.
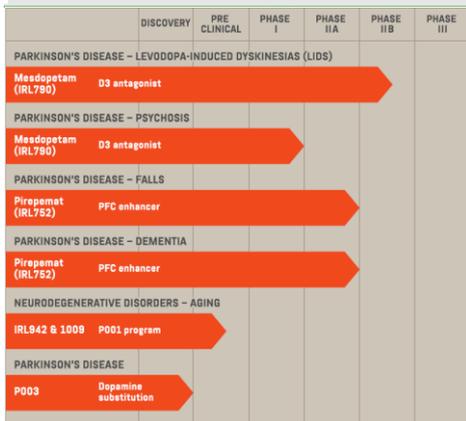
Valuation: SEK4.8bn or SEK93.3/share

Our valuation of SEK4.8bn or SEK93.3/share is based on a risk-adjusted NPV model for mesdopetam in PD-LIDs (SEK45.9/share) and PD-Psychosis (SEK13.2/share), and pirepemat in PD-Falls (SEK29.3/share). We forecast US/EU launches in 2026 and 2027 respectively; China represents an additional opportunity not reflected in our forecasts. Additionally, we do not include the early-stage portfolio (preclinical assets IRL942 and IRL1009), nor the proprietary ISP platform technology in our valuation. We include net cash of SEK253.9m at 31 March 2021.

Share price performance

%	1m	3m	12m
Abs	(5.0)	6.1	26.0
Rel (local)	(7.1)	(4.8)	(12.8)

52-week high/low	SEK55.8	SEK25.8

Business description

IRLAB Therapeutics is a Scandinavia-based biotechnology company focused on developing novel drugs for the treatment of neurodegenerative diseases utilising its ISP technology platform. Its two lead assets are in late-stage clinical trials for the symptomatic treatment of Parkinson's disease: mesdopetam (D3 antagonist) and pirepemat (PFC enhancer).

Next events

Pirepemat Phase IIb trial initiation	H121
Top-line data from mesdopetam US/EU Phase IIb/III PD-LIDs trial	H122
Potential partnering deal for mesdopetam	2022

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Edison profile page

IRLAB Therapeutics is a research client of Edison Investment Research Limited

Identifying the multiple unmet needs in PD

Founded in 2013, IRLAB Therapeutics is a Scandinavia-based biotechnology company focused on developing novel drug targets for the treatment of neurodegenerative diseases. IRLAB's history is entrenched in neurodegenerative conditions and key personnel have worked extensively with Arvid Carlsson (awarded a Nobel Prize for his contributions to seminal work on the neurotransmitter dopamine and pathophysiology in PD) at Gothenburg university. This has been instrumental in the development of the Integrative Screening Process (ISP, see page 13), IRLAB's proprietary drug discovery platform. The platform has been validated by the progress of two novel mechanisms of action (MOAs), potential first-in-class drugs into Phase II trials for PD. IRLAB is targeting several identified treatment priorities with the hope of improving on the current class of PD treatment modalities with a portfolio of novel mechanism drugs (some of which are at the preclinical/discovery stage), Exhibit 1. Mesdopetam (IRL790) is being developed for the treatment of PD-LIDs and PD-Psychosis. Pirepemat (IRL752) is being developed to treat postural dysfunction (impaired balance and falls) in PD (PD-Falls). Exhibit 2 outlines the key milestones and expected near-term catalysts.

Exhibit 1: IRLAB R&D pipeline	Exhibit 2: Milestones and catalysts 2020–26e
Source: IRLAB corporate presentation	Source: IRLAB corporate presentation

Funded to proof-of-concept data on both lead assets

We forecast sufficient cash runway through to proof-of-concept readouts from both the mesdopetam and pirepemat trials in 2022. Beyond that, operating costs will be dependent on Phase III registration trial requirements for both assets. We expect IRLAB will look to partner the asset following favourable Phase IIb data, with the partner assuming responsibility for completing the Phase III study and commercialisation. This would allow IRLAB to focus on its core competency of research and development utilising its ISP platform. Additionally, an upfront payment from any partnering deal could be used to progress IRLAB's other assets into and through clinical development. We note that preclinical assets include IRL942 and IRL1009, which could be developed for psychiatric, cognitive and motor symptoms associated with neurodegenerative diseases and ageing.

PD is a substantial commercial opportunity

Parkinson's disease (PD) is a complex, heterogenous, multifactorial neurodegenerative condition affecting nine million people globally in 2020, and this number is projected to more than double by 2040, driven by an increase in the ageing population and by increasing industrialisation. PD is characterised by the progressive and irreversible loss of dopaminergic neurons. Despite intensive research efforts in the last few decades, the current mainstay of drug treatment is limited to oral

therapies such as levodopa (L-DOPA), dopamine agonists (eg pramipexole, apomorphine), monoamine oxidase B inhibitors (eg selegiline, safinamide) and COMT inhibitors (eg entacapone), drugs that aim to increase or substitute for dopamine. While existing medications do alleviate the motor symptoms of PD, they have no impact on non-motor symptoms and are not believed to halt disease progression. Major areas of unmet need include L-DOPA-induced dyskinesias (LIDs) and treatments for the broad range of non-motor symptoms. Currently there are no approved disease-modifying drugs (DMDs) for this progressive disease that slow or alter its advancement. At the point of diagnosis, the average patient has lost 80% of the dopaminergic neurons. IQVIA estimates the global value of the PD market in 2029 to be $5.2bn (4% CAGR from 2019), driven by novel formulations of existing products with little contribution from DMDs.

Complex biology presents hurdles and opportunities

PD is generally an idiopathic condition, although 10–15% of patients have a genetic predisposition through key gene mutations. PD treatment in the future is likely to become fractionated, with only a subset of patients eligible for DMDs or gene therapies (assuming successful clinical development and regulatory approval), leaving a significant commercial opportunity for symptomatic treatments. PD-related M&A and in-licensing deals during 2020 focused on DMDs. However, many high-profile failures have highlighted the high risk that accompanies drug development for neurodegenerative conditions, relating to the complexity of the biology of the central nervous system (CNS) and the fact that at diagnosis most of these patients have moderate to severe disease with underlying pathophysiology at an advanced stage. This means that in the absence of any reliable biomarkers, the ability to diagnose PD at an early stage represents a major practical issue. Treatments that halt disease progression, or even reverse some of the degeneration, could transform the course of these conditions. However, they would ideally need to be administered at early onset, which represents a significant additional hurdle. In the interim, there is a huge need for new drugs to treat the symptoms of PD.

IRLAB looking to transform symptomatic PD treatment algorithm

IRLAB's strategy is to develop novel MOA drugs against validated targets in the pathophysiology of PD specifically for several identified treatment priorities. Mesdopetam has the potential to transform the current treatment algorithm by being added to L-DOPA in mid-stage and late-stage PD. Standard-of-care L-DOPA initially provides motor benefit, but long-term treatment inevitably results in LIDs in the vast majority of patients, limiting the dose they can tolerate and, ultimately, its benefits. Mesdopetam is being developed for the treatment of PD-LIDs (troublesome involuntary movements). The aim is to reduce troublesome dyskinesias and thus extend the daily time with good and controlled mobility, so-called 'good ON-time', not changing the total daily ON/OFF-time. If successful, future studies could assess its ability to enable optimisation of L-DOPA therapy (use of higher doses), which could increase total daily ON-time. Mesdopetam may have utility as a prophylactic and warrant use in early-stage disease as a preventive treatment for LIDs, which would transform the PD treatment algorithm (Exhibit 3).

Exhibit 3: Mesdopetam's positioning in the current PD treatment algorithm

|	Early-stage Parkinson's	Mid-stage Parkinson's	Late-stage Parkinson's
Standard of Care	• Levodopa • DA agonist • MAO-B inhibitors	• Levodopa, • DA agonists, • MAO-B inhibitors • COMT inhibitors	• Levodopa, DA agonists • MAO-B inhibitors tapered • Advanced therapies: Duopa, DBS, Apo infusion
Challenges in Parkinson's	• Acceptable symptomatic control • Low societal costs	• Occurrence of dyskinesia • Wearing-off • Levodopa dosing low leading to undertreatment	• Dyskinesia • Motor fluctuations • Non-motor symptoms: hallucinations, orthostasis, cognitive impairment • Increased disability • High societal costs
Mesdopetam's role		✓ Prevention and treatment of *dyskinesias* ✓ Enabling levodopa treatment optimization of motor symptoms	✓ Prevention and treatment of *dyskinesia and hallucinations* ✓ Enabling optimization treatment of motor and non-motor symptoms
The new algorithm		• **Mesdopetam +** • **Levodopa**	• **Mesdopetam +** • **Levodopa**

Source: IRLAB corporate presentation

Mesdopetam novel MOA for PD-LIDs

Mesdopetam (IRL790) is an oral D3 antagonist currently in Phase IIb/III development for the treatment of PD-LIDs, a significant unmet medical need. It is an innovative new chemical entity (NCE) discovered by IRLAB's ISP and developed in house. Based on its completely novel MOA and structure, the World Health Organization (WHO) proposed the new international non-proprietary name (INN), mesdopetam (generic name), recognising that it represents an entirely new class of CNS compounds. Preclinical studies have highlighted mesdopetam's potential in psychosis and procognitive effects at the same therapeutic dose that it is administered for LIDs. Both symptoms present in patients with mid- to late-stage disease. Mesdopetam's potential to combine both antipsychotic and anti-dyskinetic benefits, which contribute to motor function, could lead to a shift in the treatment paradigm, replacing the need for multiple different drugs and providing a significant improvement in patient outcomes. Mesdopetam is wholly owned by IRLAB and composition of matter patents cover all major markets including Europe, the United States and China until 2037. This is exceptionally long protection for a drug at mesdopetam's late stage of development and recently filed applications could extend this into the 2040s. We note that as an NCE it will enjoy market exclusivity for at least 10 years after approval in Europe and five years in the US.

First-in-class D3 antagonist

The D3 antagonist mesdopetam belongs to a new class of CNS agents called psychomotor stabilisers, which modify activity depending on its initial level. The asset was designed as a new type of dopaminergic antagonist that exhibits an agonist-like binding mode with finely balanced physicochemical properties that allow it to penetrate the blood brain barrier efficiently, while maintaining its high selectivity and not being completely cleared by first-pass metabolism (which reduces the amount of oral drug that reaches systemic circulation) before reaching its target. Its agonist-like structure allows it to delicately modulate endogenous neurotransmission in such a way that normal dopaminergic functions are unaltered, while alleviating aberrant signalling that causes motor and psychiatric disturbances. Mesdopetam is highly selective for the dopamine D3 receptor, which has recently emerged as a promising target for treating PD-LIDs, and IRLAB has shown that patients treated with mesdopetam have maintained the therapeutic benefits of L-DOPA on motor function thus far. IRLAB is advancing scientific knowledge in identifying the potential of this receptor and as a result estimates mesdopetam is ~four to five years ahead of the nearest competition.

Exhibit 4: Mesdopetam mechanism of action
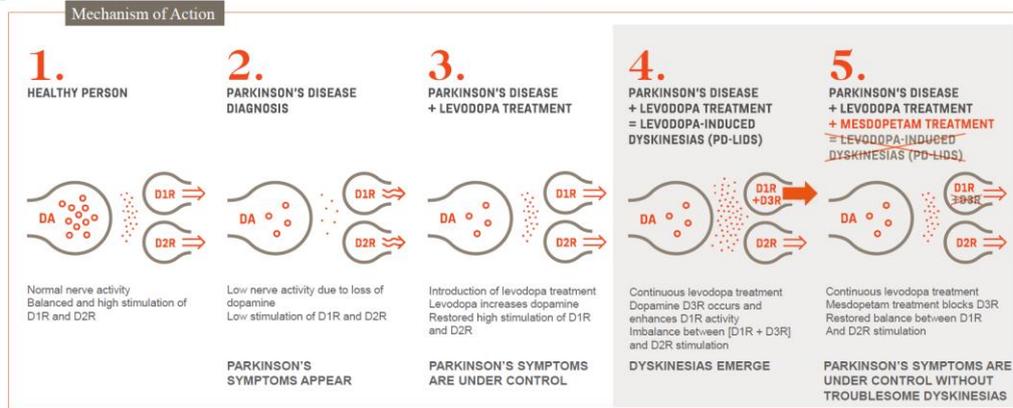
Source: IRLAB corporate presentation

PD-LIDs treatment could lead to optimisation of L-DOPA treatment

Standard-of-care L-DOPA initially provides motor benefit, but long-term treatment inevitably results in LIDs in the vast majority of patients. These involuntary movements are troublesome, difficult to treat and often overshadow the benefits of L-DOPA treatment. It is not fully understood how LIDs develop. However, it is believed that a number of neurotransmitters, including dopamine, serotonin and glutamate and their respective neuroreceptors and signalling pathways, are involved. More than 30% of PD patients develop such dyskinesias within five years of treatment, with the rates rising to 60% within 10 years and almost all patients within 15 years. Patients experiencing dyskinesias often have great difficulty optimising their treatment with L-DOPA and, as a result, the vast majority of PD patients are actually undertreated with L-DOPA (receive a lower than optimal daily dose) due to the fear of developing LIDs. Adamas Pharmaceuticals' Gocovri, an oral long-acting formulation of amantadine, is currently the only FDA-approved pharmacotherapy for the treatment of PD-LIDs. However, although efficacious, debilitating side effects have limited its use. There are currently no drugs for PD-LIDs approved outside the US.

Early signs of efficacy from Phase IIa in tough-to-treat patients

Mesdopetam has shown good safety and tolerability at all doses and early signs of clinical efficacy in Phase I and IIa studies, alleviating LIDs without compromising the therapeutic benefits of L-DOPA on motor function. In the Phase IIa study in PD-LIDs (n=75), mesdopetam was taken orally twice daily (at three dose levels: 5mg, 7.5mg and 10mg) for 28 days as an add-on to patients' regular medication for PD. An in-depth data analysis (including all dose levels) showed that mesdopetam had a significant and clinically meaningful improvement on dyskinesias in two out of the three assessment methods. The Hauser standardised patient-reported diaries showed that mesdopetam reduced patients' time with troublesome dyskinesias by 1.6 hours daily vs placebo, which increased significantly to 2.7 hours for patients that had not receive concomitant amantadine. The Unified Parkinson's Disease Rating Scale (MDS-UPDRS) dyskinesia assessment also found that mesdopetam reduced troublesome dyskinesias (0.9 hours daily vs placebo, increasing to 1.3 hours for amantadine-naïve patients), highlighting the potential benefits of mesdopetam versus approved PD-LIDs treatment amantadine.

However, no improvement was observed using the Unified Dyskinesia Rating Scale (UDysRS), which was the primary endpoint for the study, despite the significant effects on dyskinesia observed by the other assessment methods. IRLAB's shares fell sharply on the announcement, although an in-depth analysis of the data highlighted the technical shortcomings of this assessment method

(many patients were not in the required ON-state during assessment) and indicated that it was not relevant for assessment of ON-state dyskinesias in this study.

Mesdopetam has a particularly wide therapeutic dosing window for a CNS drug and peak efficacy was observed with the 7.5mg dose, with no added benefit from higher doses, Exhibit 5. This dose increased good ON-time without dyskinesias by 5.6 hours daily vs one hour with placebo, a significant improvement for patients who, prior to treatment, had only approximately 6.3 hours of good ON-time per day.

Exhibit 5: Increase in good ON-time through reduction in dyskinesias
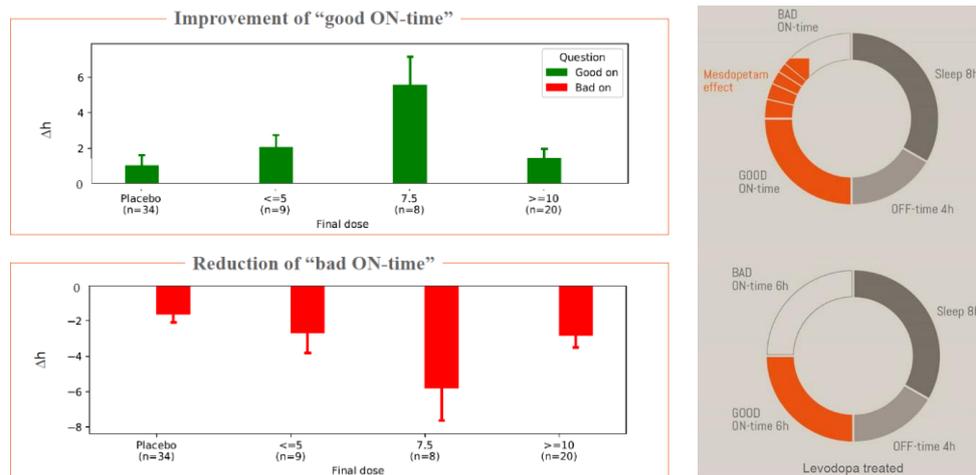
Source: IRLAB corporate presentation

Importantly, assessment of other motor symptoms of PD (using MDS-UPDRS parts II+III, MDS-UPDRS OFF-time, and 24-hour patient diaries) showed that patients treated with mesdopetam maintained the therapeutic benefits of L-DOPA on motor function, with no change in total daily ON/OFF-time. As mesdopetam successfully reduced the side effects currently limiting L-DOPA dosing, it may enable the use of higher doses of L-DOPA, providing further improvements in motor function. Looking at the broader picture, this could reduce the amount of concomitant treatments PD patients have to take (MAO-B inhibitors, dopamine agonists, amantadine, etc). Additionally, preclinical studies suggest that mesdopetam may also prevent the development of LIDs, and future clinical studies could focus on its use in early-stage PD as a prophylactic treatment for LIDs, potentially increasing its commercial potential and the addressable patient population. In clinical studies so far, mesdopetam has shown good safety and tolerability at all doses, as well as a favourable pharmacokinetic profile with no long-term accumulation. Notably, it has shown no cardiovascular side effects to date.

Phase IIb/III study will determine route to regulatory approval

Mesdopetam is currently in a Phase IIb/III study in PD-LIDs as an add-on therapy to current standard of care, which includes L-DOPA. The Phase IIb/III study is assessing three dose of mesdopetam (7.5mg, 5mg and 2.5mg twice daily) and the primary endpoint is change in daily hours of good ON-time without troublesome dyskinesia based on Hauser standardised 24-hour patient-reported diaries over three months. Key secondary endpoints include dyskinesia assessments under UDysRS and MDS-UPDRS. The study will enrol c 140 patients at sites in the EU and US, and top-line data are expected in H122. IRLAB believes this study could be supportive of an NDA filing, meaning only one additional Phase III pivotal study would be required before regulatory filings. This means a potential pivotal Phase III study could start in 2023, leading to approval and launch in 2026. Additionally, the Phase III study could include an arm in which patients received higher doses of L-DOPA with or without other concomitant PD treatments, providing another important differentiating factor versus other treatment options.

PD clinical trial pitfalls

The historically high attrition rate of PD clinical trials can be attributed to several factors. Firstly, the heterogeneity of PD is increasingly being recognised; with marked genetic variability, environmental effects and patients taking multiple different treatments, there can be substantial variability in the patient population at baseline. Furthermore, there is also a well-documented placebo effect that is commonly observed in PD studies, which raises the bar for statistical significance even further. Increasing the size of clinical trials is a common strategy to somewhat average out these differences.

There are also many challenges in measuring endpoints to assess improvements in PD and LIDs in clinical trials. Patient- and clinician-completed rating scales such as UDysRS, MDS-UPDRS and patient diaries are somewhat subjective in comparison to the hard clinical endpoints used in oncology trials (such as overall survival and progression-free survival), and can be less accurate and subject to recall bias. Furthermore, an actual reduction in LIDs may not reflect a patient's perception of the severity of dyskinesia or their level of disability. Using multiple clinical trial endpoints maximises the number of shots on goal, as different endpoints will be more relevant to specific patient populations.

Psychosis and Tardive dyskinesia additional opportunities

Mesdopetam is also being developed for psychosis in Parkinson's disease (PD-Psychosis), a condition characterised by hallucinations and sometimes delusions. IRLAB estimates that c 35% of PD patients develop symptoms which can occur both as a result of the underlying disease or as a consequence of treatment with certain drugs. This is a significant unmet medical need that can be debilitating and cause patients to discontinue treatments that may have procognitive effects. Acadia Pharmaceuticals' 5-HT2A antagonist Nuplazid (pimavanserin) is the only FDA-approved treatment for PD-Psychosis, although it does carry a black box warning for increased mortality in elderly patients with dementia-related psychosis. Net product sales of Nuplazid were $442m in 2020. There are currently no approved treatments for PD-Psychosis in Europe and patients are commonly treated with off-label anti-psychotic drugs that are approved for schizophrenia.

Preclinical studies have highlighted mesdopetam's potential in psychosis and procognitive effects at the same therapeutic dose at which it is administered for LIDs. IRLAB expects to initiate a Phase II study in PD-Psychosis in 2022–23. Mesdopetam's unique MOA suggests it may also have potential as a treatment for tardive dyskinesia (TD), which affects patients with psychosis and is caused by long-term treatment with antipsychotics. IRLAB estimates that approximately 25% of patients treated with antipsychotic drugs develop TD following long-term treatment with current drugs. IRLAB expects to initiate a Phase IIa study in TD in 2022–23, contingent on funding. This indication represents further upside to our current valuation.

D3 receptor a neglected target in PD?

The exact pathophysiological mechanism by which LIDs manifest in PD has yet to be fully elucidated. However, recent scientific publications highlight the potential key role the dopamine D3 receptor plays. Dopaminergic signalling controls movement through two pathways – the direct pathway and the indirect pathway. Dopamine exerts opposing effects on each pathway, stimulating signal transduction through D1 receptors predominately found on the postsynaptic membrane of the direct pathway, while inhibiting signal transduction through D2 receptors predominately found on the postsynaptic membrane of the indirect pathway. The fine balance between these two signalling pathways and the dopamine D1 and D2 receptors controls smooth movement. Chronic treatment with L-DOPA causes abnormal pulsative stimulation that results in increased expression of sensitised D1 receptors, as well as a colocalised increased expression of the D3 receptor. The dopamine D3 receptor has been shown to enhance aberrant D1 receptor signalling, potentially

through the formation of heteromers, causing an imbalance between the direct (D1 receptor) and indirect (D2 receptor) signalling pathways, which has been implicated in the manifestation of LIDs. Strategies to normalise the activity of the upregulated D3 receptor through either partial agonism or antagonism are expected to alleviate LIDs. Upregulation of the D3 receptor in PD-LIDs patients has been confirmed using PET scans. The D3 receptor possesses the highest affinity for dopamine compared to D1 and D2 receptors, which exhibit much higher expression, therefore upregulation of the D3 receptor may significantly modify dopaminergic signalling.

Pirepemat first-in-class for PD-related falls

Pirepemat (IRL752) is a first-in-class oral small molecule currently in development for the treatment of postural dysfunction (impaired balance) and falls in PD (PD-Falls). Impaired balance and increased risk of falls have been strongly linked to cognitive decline in PD. These motor symptoms are incredibly debilitating for patients and represent a significant unmet medical need as they are highly prevalent and not addressed by current PD treatments. Pirepemat is an innovative NCE discovered by IRLAB's ISP and developed in-house. It also represents an entirely new class of CNS compounds and the WHO proposed the INN pirepemat, recognising its completely novel MOA and structure. Pirepemat is wholly owned by IRLAB and composition of matter patents cover all major markets including Europe, the US and China until 2037; recently filed applications could extend this into the 2040s. We note that as an NCE, it will benefit from market exclusivity for at least 10 years after approval in Europe and five years in the US.

Differentiated mechanism of action

Pirepemat is primarily an antagonist of the 5-HT7 and alpha-2 receptors, which potentially has cognitive-enhancing effects and can restore the normal function of the cerebral cortex by selectively increasing extracellular levels of the neurotransmitters, norepinephrine and dopamine, Exhibit 6. Pirepemat also activates specific genes involved in the function of nerve cell synapses that have precognitive effects. It therefore has the potential to ameliorate deficits of cortical function, such as cognitive impairment and postural/axial motor dysfunction. Although pirepemat is only moderately potent in targeting any single neuroreceptor, its ability to regio-selectively modulate multiple pathways (a common approach that usually requires combinations of multiple different drugs) may result in superior therapeutic characteristics. Importantly, preclinical studies have shown that it does not affect any negative feedback mechanisms on neurotransmission in monoaminergic projection areas. To the best of our knowledge, there are no other treatments in development with a similar MOA and IRLAB estimates that it is four to five years ahead of potentially similar competitors.

Exhibit 6: Pirepemat mechanism of action
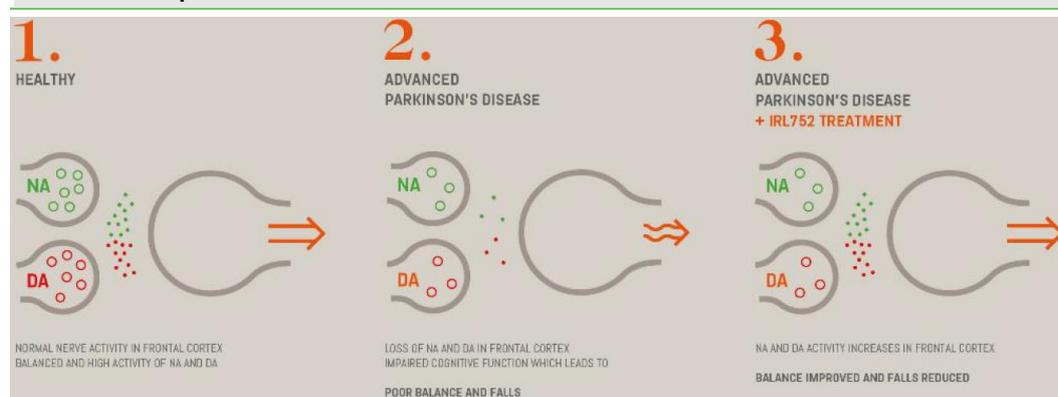
Source: IRLAB corporate presentation

Prevalent falls in PD represent untapped market

Postural dysfunction is a debilitating motor symptom of PD associated with increased risk of falls and subsequent complications such as fractures, decreased mobility and lower quality of life. Approximately 60% of PD patients fall every year, with around 70% of these patients falling more than once. While postural dysfunction is also generally associated with old age, the risk of falling for PD patients is two to three times higher. More than 70% of PD-Falls require some form of medical care and approximately one-third result in fractures such that fall-related injuries are the dominant cause of hospitalisation for PD patients. The US Centers for Disease Control and Prevention (CDC) estimates the average hospital cost is c $30,000 per fall (for patients over 65). Thus PD-Falls represent a significant burden on healthcare systems that is expected to continue to grow due to an ageing population and increased prevalence of PD. From a health economic perspective, a successful balance-improving treatment has significant market potential and could command favourable pricing. We note that an expert panel of PD specialists (780 clinicians) recently concluded that a 25% relative reduction in annual falls would be viewed as clinically meaningful.

Patients with PD have dysfunctional reactive postural responses, which lead to lagging adjustments in balance control. Maintaining balance when standing is critically dependent on the function of the prefrontal cortex (PFC), and postural instability and increased risk of falls have been strongly linked to cognitive decline in PD. The Hoehn and Yahr Scale is an established methodology used to describe the progression of PD symptoms and the level of disability (Exhibit 7). The key feature as patients transition from stage 2 to 3 is the emergence of postural instability, loss of balance and falls. This coincides with a significant increase in the level of disability, impact on quality of life and requirement for assistance. Approximately 50% of PD patients are at stage 3 and above.

Exhibit 7: Hoehn and Yahr Scale

|			50% of the patient population		
Hoehn & Yahr stage	1	2	3	4	5
	Unilateral involvement only	Bilateral or midline involvement without impairment of balance	Bilateral disease: Mild to moderate disability with *impaired postural reflexes (poor balance)*	Severely disabling disease; still able to walk or stand unassisted	Confinement to bed or wheelchair unless aided
Challenge			**Transition from stage 2 to stage 3:** The emergence of postural instability, loss of balance and falls	The advent of cognitive impairment is associated with additional impairment of postural reflexes and leads to falls.	
Treatment		Levodopa, DA agonists, MAO-B inhibitors, COMT inhibitors: Treatment response usually acceptable with mild residual disability. May lead to motor complications.	**Pirepemat introduced into the algoritm: Potential to provide** • **Reduced falls** • **Improved cognitive functions and** • **Reduced apathy**		

Source: IRLAB corporate presentation

The exact underlying mechanism of PD-Falls has not been fully elucidated. However, deficits in cortical catecholaminergic (dopamine and norepinephrine) and cholinergic (acetylcholine) signal transmission have been implicated. Additionally, subcortical Lewy body-related neuropathology is thought to be associated with cognitive decline in PD. There are currently no treatments for PD-Falls and the causative cognitive impairment. A successful treatment would represent a significant advancement in the armament of symptomatic treatments for PD (see Exhibit 7).

Early signs of efficacy in PD-Falls

Pirepemat has demonstrated preliminary signs of efficacy in a Phase IIa study (n=32) in patients with PD (50% reduction in fall frequency) and dementia and appears to be well tolerated at clinically relevant doses (600mg). An exploratory efficacy analysis of axial motor functions (using the UPDRS assessment) showed that pirepemat led to a significant improvement in postural stability/balance and a numerical reduction in the frequency of falls (Exhibit 8). Cognitive and neuropsychiatric tests showed a significantly reduced apathy (75% versus 33% for placebo by the NPI-12 scale) and a trend towards improvement in cognitive impairment (memory and thinking ability). Treatment was well tolerated at clinically relevant doses and adverse reactions were mainly related to the CNS, gastrointestinal tract and infections. A moderate and transient increase in liver enzymes was observed for three patients that normalised following discontinuation of treatment at follow-up. Importantly, pirepemat had no negative effects on cardiovascular function, thus its tolerable safety profile makes it ideal for use in combination with other PD treatments.

Exhibit 8: Pirepemat Phase IIa data – improvement of postural dysfunction and falls
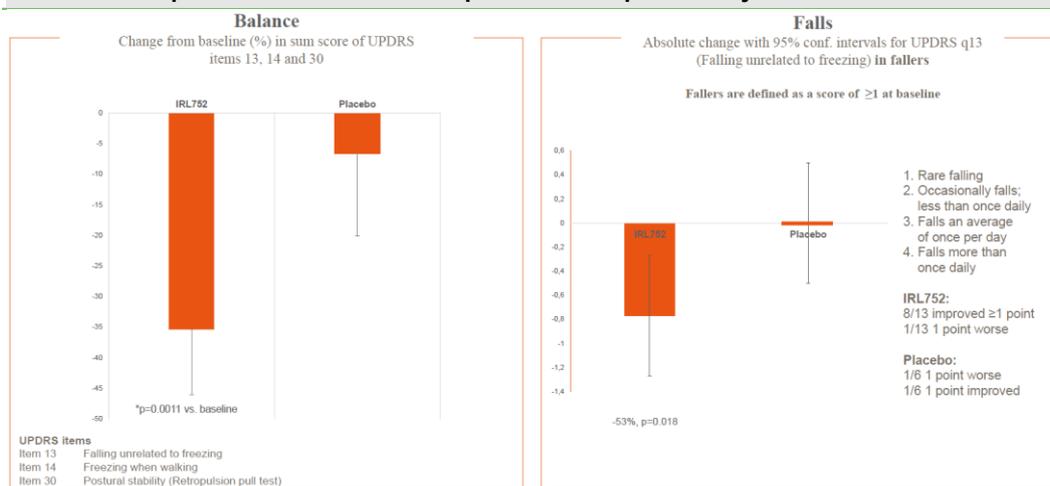
Source: IRLAB corporate presentation

Phase IIb study with first-in-class treatment to start in H121

A Phase IIb study in PD-Falls is expected to start in H121 and will enrol c 150 patients at sites in the EU and US. Pirepemat will be given orally once daily as an add-on therapy at one of two dose levels for 12 weeks. The objective primary endpoint is change in frequency of falls versus placebo. Top-line data are expected in H222. This means a pivotal Phase III study could start in 2024, with potential approval and launch in 2027.

PD-related dementia an additional opportunity

Pirepemat also has the potential to be developed for the treatment of dementia in PD. Almost 80% of PD patients develop dementia, suggesting that it is in fact a manifestation of the disease. While IRLAB does not have any imminent plans to initiate a Phase IIb/III study in dementia, pirepemat has shown early signs of efficacy in a Phase IIa study in PD-dementia as it decreased apathy (lack of motivation and ability to take initiative) and improved results in cognitive tests (memory and thinking ability). We expect IRLAB will also look to out-license pirepemat following favourable Phase IIb data in PD-Falls. Development in dementia represents an interesting opportunity to further maximise the potential value of this asset.

Competitive landscape

L-DOPA and the dopamine agonists are the mainstay of pharmacological treatments. In the main these drugs are available as generics with limited new treatment options, with only one drug approved in PD in the last decade (Newron's MAO-B inhibitor Xadago, approved March 2017). Non-drug treatment options include deep brain stimulation (DBS), a surgical technique (FDA approved in 1997) reserved for very advanced PD patients who have an unstable L-DOPA response. However, DBS does not address the progressive nature of PD and, due to the cost, lack of accessibility, invasiveness and side effect profile, it has seen limited use. There are multiple drugs in development that fall into two groups: those that treat the symptoms of PD, and DMDs that aim to slow down or halt disease progression. However, PD remains a significant unmet medical need. Studies have demonstrated a wide spectrum of associated costs for PD and in the US alone the burden on society (including hospital, medical and care giver costs, loss of productivity and work days) from PD is estimated to be $51,800 per year per patient.

Disease-modifying treatments the holy grail in PD

PD is generally an idiopathic condition, although 10–15% of patients have a genetic predisposition (eg mutations in GBA1, alpha-synuclein, LRRK2, VPS35 and PINK1 genes). Recent research efforts have focused on developing DMDs for these subsets of patients, and PD-related M&A and in-licensing deals during 2020 focused on alpha-synuclein, immunomodulators and gene therapies. However, there are currently no DMDs approved for PD and high-profile failures include Biogen's cinpanemab and Sanofi's venglustat, which failed to meet efficacy endpoints in late-stage trials.

Due to the heterogeneity of PD, we hypothesise that the current DMDs in development will likely target, or be efficacious for, only a specific subset of patients, thus even if one is successfully approved, we expect that a significant portion of patients will still be reliant on symptomatic treatments. There are five main potential disease-modifying strategies currently in clinical trials, including decreasing aggregation of alpha-synuclein, using GLP-1 agonists to decrease neuroinflammation, enhancing lysosomal activity to compensate for GBA mutations, inhibition of LRRK2 and inhibition of c-Abl.

Decreasing aggregation of alpha-synuclein

There are multiple small molecule drugs and antibodies in development targeting alpha-synuclein, a key component of Lewy bodies (aggregates of protein deposits in the brain) believed to play a role in the pathogenesis of PD. There have been several high-profile disappointments in this area despite several billion invested in M&A and licensing deals. Biogen discontinued development of cinpanemab (BIIB054), following failure to meet the primary endpoint (change from start in total MDS-UPDRS) and secondary endpoints in the Phase II SPARK trial. Roche/Prothena's prasinezumab (PRX002/RG7935) also failed to meet its primary endpoint (slowdown in progression of both motor and non-motor symptoms) in the Phase II PASADENA study, but did significantly reduce motor function decline and improved disease biomarkers. A Phase IIb study (PADOVA) in early-stage PD is now recruiting. In December 2020, UCB initiated a Phase IIa study evaluating UCB0599 in early PD, completion expected in late 2023.

Reduction of inflammation using GLP-1 agonists

Several studies suggest that type II diabetes is associated with an increased risk of developing PD. Glucagon-like peptide-1 (GLP-1) agonists are a leading class of diabetes drugs that are currently being assessed for a disease-modifying benefit in PD. The hypothesis is that these products exert neuroprotective effects in several different ways. Currently, there are five GLP-1 agonists in development for PD following encouraging results from a Phase II clinical trial evaluating

AstraZeneca's Bydureon (exenatide-extended release) in PD. The most advanced are Bydureon, which is now in Phase III (data expected in 2023) and Novo Nordisk's Victoza (liraglutide) in Phase II (data expected this year).

Mutations in glucocerebrosidase (GBA1)

Recently discovered genes implicated in PD include mutations in glucocerebrosidase gene 1 (GBA1), the gene which codes for the lysosomal enzyme glucocerebrosidase. The mechanisms underlying this association are not fully known, but theories include enhanced protein aggregation, alterations in lipid levels and lysosomal dysfunction. In February 2021, Sanofi discontinued development of venglustat, an oral glucosylceramide synthase inhibitor (hypothesised to reduce toxic alpha-synuclein conversion), following failure to demonstrate efficacy in the Phase II MOVES-PD study. Other approaches include SPARC's BCR-ABL1 tyrosine kinase inhibitor vodobatinib (K0706/SCC-138), with the Phase II PROSEEK trial ongoing (data expected in 2023).

Inhibition of LRRK2

Denali Therapeutics has a portfolio of PD assets in preclinical and clinical development led by DNL151, which targets leucine-rich repeat kinase 2 (LRRK2), and late-stage trials are expected to initiate in 2021. In October 2020, Biogen entered a co-development/co-commercialisation deal with Denali covering DNL151 plus options for rights to two other programmes for $560m upfront plus $465m equity investment plus potential milestones, profit sharing and royalties.

Symptomatic treatments

This includes gene therapies and other agents targeting existing pathways and novel targets. There are currently more than 140 ongoing clinical trials across the PD space. We focus here on gene therapies and areas relevant to IRLAB (PD-LIDs, PD-Psychosis and PD-Falls).

Gene therapies: Mixed results to date

Gene therapy is an increasingly hot area, as evidenced by recent deals in the space that include Eli Lilly's acquisition of Prevail Therapeutics in December 2020 for $880m upfront to gain access to its pipeline of products including PR001, an AAV9 gene therapy (which aims to transfer the GBA1 gene that encodes the enzyme beta-glucocerebrosidase) for PD patients with GBA1 mutations (~7–10% of PD patients carry at least one GBA mutation). Voyager/Neurocrine's Phase II study with AAV-based gene therapy candidate VY-AADC was placed on hold following reports of MRI abnormalities in some patients.

Sio Gene Therapies is currently assessing AXO-Lenti-PD, a lentiviral vector therapy (aiming to restore dopamine levels via the delivery of three genes needed for dopamine synthesis) in a Phase II clinical trial. The aim is to stabilise disease and reduce the amount of L-DOPA medication. To date, AXO-Lenti-PD data show that it is well tolerated, with encouraging preliminary efficacy signals in the ongoing SUNRISE-PD study, albeit in very small patient numbers. AXO-Lenti-PD is administered intrathecally, which means its use will be limited to younger PD patients who are fit enough for neurosurgery (such as the DBS patient population).

PD-LIDs and PD-Psychosis

Adamas's Gocovri (amantadine ER) is the only approved treatment for PD-LIDs limited to the US market. Approval was based on two Phase III studies (EASE LID and EASE LID3), which demonstrated that once-daily amantadine significantly reduced dyskinesias versus placebo based on the UDysRS total score at week 12 (37% versus 12% and 46% versus 16%). Gocovri is associated with multiple disabling side effects including peripheral edema, falls, suicidality and depression. Hallucination affects c 30% of patients and physicians often find it hard to balance improvements in motor symptoms without inducing symptoms of psychosis. Although efficacious, side effects have limited its use, such that, priced at $28,500 per year, sales of Gocovri have been

lacklustre, at $71m in 2020. A number of Phase II assets are in development for the symptomatic treatment of PD-LIDs (Exhibit 9), and Newron/Zambon's Xadago is the most advanced asset in Phase III. Mesdopetam's potential to enable the use of higher doses of L-DOPA and prevent the development of LIDs are important potential differentiating factors vs competitors.

The only approved treatment for PD-Psychosis is Acadia's 5-HT2A antagonist Nuplazid (pimavanserin) in the US. Sunovion has two assets in development for PD-Psychosis: SEP-363856, a novel MOA TAAR1 and 5-HT1A agonist, recently reported encouraging data from a Phase II study and DSP-6745, a dual 5-HT2A and 5-HT2C antagonist, is in a Phase I study.

Exhibit 9: Selection of late-stage assets in development for PD-LIDs

Company	Drug candidate	Therapeutic class	Notes
Newron/Zambon	Safinamide	MAO-B inhibitor	Phase III*
Addex Therapeutics	Dipraglurant	mGluR5 allosteric modulator	Phase II/III
VistaGen Therapeutics	AV-101	NMDA glutamate receptor modulator	Phase II
Bukwang Pharmaceutical Co	JM-010	Combination of 5-HT1A and 5-HT1B/5-HT1D receptor agonists	Phase II
Neurolixis	NLX-112	5-HT1A receptor agonist	Phase II*

Source: IRLAB Therapeutics, Edison Investment Research. Note: *Expected to start shortly.

PD-related falls

There is little in late-stage development for PD-Falls. The generic cholinesterase inhibitor rivastigmine (transdermal patch) is in a Phase III study with top-line data expected by end 2021. Takeda Pharmaceuticals' TAK-071 (muscarinic M1-selective positive allosteric modulator) is in a Phase II study evaluating its ability to improve gait and balance in patients with PD. Results from this study are expected towards the end of 2022. Lundbeck's Northera (droxidopa), a prodrug of norepinephrine, received FDA approval in 2014 for neurogenic orthostatic hypertension (sudden drop in blood pressure when changing positions that causes dizziness, light-headedness and falls) related to multiple causes including PD.

Integrative Screening Process (ISP) research platform

IRLAB's unique, in-house developed Integrative Screening Process (ISP) research platform is at the heart of its drug discovery engine and enables the discovery of novel drugs for CNS-related diseases. The proprietary platform is based on a unique combination of a systems biology approach (phenotypic screening) and efficient AI-based machine learning methods. The platform contains a growing database of almost 1,400 CNS drug-like compounds developed over 25 years. Each compound is profiled using a series of highly optimised assays and in vivo animal models to capture dose-dependent data on a range of responses that include movements, behavioural patterns, biochemical markers and gene expression. Data on pharmacokinetics, receptor binding, chemical properties and safety are also collected. Using machine learning methods, IRLAB has developed a comprehensive map of the CNS drug property space which it can leverage to generate high-quality clinical candidates. We note that numerous other biotech and pharmaceutical companies have developed screening platforms, but the ISP platform is unique in combining measurements of both neurochemistry and behaviour through the use of AI-based analytics. So far, IRLAB's strategy has focused on developing its own drug candidates. The ISP platform could be out-licensed to partners for the discovery of novel candidates.

Industry-leading approach to developing novel treatments

The early use of in vivo studies and phenotypic screening is of particular importance and is a competitive advantage for IRLAB for several reasons. It takes into account holistic effects such as synergies arising from interactions with multiple targets and downstream effects, which reduces the risk of discounting promising compounds that may be overlooked using classical target-based

screening (the dominant method used in the pharmaceutical industry). It also provides an early insight into safety and the compound's ability to cross the blood-brain barrier (which is notoriously hard to predict using facile in vitro assays). Thus, key development risks are managed at an early stage. Additionally, as the exact pathogenesis underlying the majority of CNS disorders has not been fully clarified, the use of a systems biology approach is far superior as it allows the discovery of novel drug candidates with new mechanisms of action (such as mesdopetam and pirepemat). The ISP platform offers significantly improved cost and time efficiencies for the development of clinical candidates versus historical industry averages (a more than fourfold cost saving and less than half the development time). IRLAB also believes that it generates candidates with improved chances of clinical success. This is only possible as the brain's monoaminergic systems (such as dopamine) are highly conserved between species and IRLAB has seen good translation between preclinical observations in the ISP platform and clinical effects in humans. IRLAB continues to upgrade the ISP platform through collaborations with experts and scientific advisors.

Sensitivities

IRLAB is subject to the usual risks associated with drug development including clinical development delays or failures, regulatory risks, competitor successes, partnering setbacks and financing risks. Value crystallisation in the future will depend on successful R&D progress and any potential partnering activities. The biggest near-term development sensitivity is related to mesdopetam and pirepemat. The science for both assets' MOA in particular is still nascent and validation will in part be led by IRLAB's Phase IIb/III clinical trial findings as well as ongoing work in academia. IRLAB has funding through to proof-of-concept for both assets, but further funds or partnering deals will be needed for additional trials required for regulatory filings.

Valuation

Our valuation of SEK4.8bn or SEK93.3/share including net cash of SEK253.9m (at 31 March 2021) is based on a risk-adjusted NPV model for mesdopetam in PD-LIDs (SEK45.9/share) and in PD-Psychosis (SEK13.2/share), and pirepemat in PD-Falls (SEK29.3/share). The breakdown of our NPV valuation, which uses a 12.5% discount rate, is shown in Exhibit 10. We do not include the early-stage portfolio (preclinical assets IRL942 and IRL1009), nor the proprietary ISP platform technology. We see uplift as assets move into clinical development.

Exhibit 10: IRLAB sum-of-the-parts valuation

Product	Indication	Launch	Peak sales ($m)	Value (SEKm)	Probability	rNPV (SEKm)	rNPV/share (SEK)
Mesdopetam	PD-LIDs	2026	1,207	4,793.5	50%	2,375.6	45.9
Mesdopetam	PD-Psychosis	2027	688	2,356.5	30%	683.6	13.2
Pirepemat	PD-Falls (postural dysfunction)	2027	1,036	5,190.8	30%	1,515.5	29.3
Net cash at 31 March 2021				253.9	100%	253.9	4.9
Valuation				**12,594.7**		**4,828.6**	**93.3**

Source: Edison Investment Research

We model mesdopetam in PD-LIDs and PD-Psychosis in the US and EU5 only; China and Japan represent additional opportunities which we will include once registrational trials are initiated. We forecast launch in 2026 and peak sales of $1.2bn in the US/EU5 in 2032 based on a conservative peak penetration rate of 10% of the 200,000 US patients and 230,000 EU patients with PD-LIDs. If mesdopetam shows a significant clinical benefit and achieves a penetration rate of 20%, peak sales increase to $2.4bn. We will revisit our assumptions as data emerge.

We forecast launch in 2027 and peak sales of $688m in the US/EU5 in 2033 for PD-Psychosis. This is based on a peak penetration rate of 7.5%; we assume 50% of the estimated patients with

psychosis (US: 306,000, EU5: 322,000) are the eligible patient population, taking into account the invariable overlap of patients with PD-LIDs. We assume a US price of $28,500 pa (in line with Gocovri) and assume a 50% discount to arrive at an EU price. We note that both mesdopetam and pirepemat composition of matter patents provide protection until 2037 and recently filed applications could extend this into the 2040s.

We model pirepemat for the treatment of PD-Falls in the US and EU5 only, and forecast peak sales of $1.0bn in 2033 assuming launch in 2027. The population at risk of PD-related recurrent falls in the US is 330,000 and 395,000 in Europe. We assume 100% of these patients are eligible for pirepemat, but apply a conservative peak penetration rate of 5%. We assume pricing on a par with mesdopetam, in the absence of any direct comparator. For both assets, we assume an out-licensing deal post Phase IIb/III data and a blended tiered royalty rate of 30%, which takes into account any potential milestone value.

Financials

In the near term, IRLAB will continue to be cash consumptive and operate as a non-revenue generating biotech. In FY20, it reported an operating loss of SEK91.5m, slightly down from SEK95.8m in FY19. We forecast the operating loss to increase to SEK107.4m in FY21 and SEK158.3m in FY22 as the Phase IIb/III studies in PD-LIDs and PD-Falls conclude and the Phase II study in PD-Psychosis initiates. IRLAB has a strong debt-free balance sheet and is funded through to proof-of-concept data based on net cash of SEK277.0m at 31 December 2020 and current cash burn. However, any increases in clinical trial costs or head count could reduce our forecast cash runway. We expect IRLAB will look to partner the assets following favourable Phase IIb/III data, with the partner assuming responsibility for completing the Phase III registrational studies and commercialisation. Additionally, an upfront payment from any partnering deal could be used to progress IRLAB's other assets (IRL942 and IRL1009) into and through clinical development. Research and development expenses amounted to SEK76.0m in FY20 (versus SEK79.4m in FY19), representing c 83% of the total operating expense. We forecast research and development expenses to grow to SEK89.7m in FY21 and SEK132.1m in FY22, while we expect operating expenses (including personnel costs) to remain relatively flat over the coming years.

Q121 results were in line with expectations and IRLAB reported R&D expenses of SEK16.5m (versus SEK16.0m in Q120) and an operating loss of SEK20.0m (versus SEK19.1m in Q120). Net cash was SEK253.9m at 31 March 2021. While we forecast a partnering deal, if IRLAB chooses to pursue development itself, we expect it will need to raise significant funds ($50m) to cover the potential Phase III studies.

Exhibit 11: Financial summary

Accounts: IFRS, year-end: 31 December, SEK000s			2018	2019	2020	2021e	2022e
PROFIT & LOSS							
Total revenues			196	448	404	413	413
Cost of sales			0	0	0	0	0
Gross profit			196	448	404	413	413
Total operating expenses			(74,093)	(96,296)	(91,862)	(107,854)	(158,694)
Research and development expenses			(58,927)	(79,381)	(75,989)	(89,746)	(132,119)
EBITDA (reported)			(72,565)	(92,916)	(89,202)	(104,093)	(154,541)
Operating income (reported)			(73,897)	(95,848)	(91,458)	(107,441)	(158,281)
Operating margin %			N/A	N/A	N/A	N/A	N/A
Finance income/(expense)			(202)	(272)	(195)	(446)	(446)
Exceptionals and adjustments			0	0	0	0	0
Profit before tax (reported)			(74,099)	(96,120)	(91,653)	(107,887)	(158,727)
Profit before tax (normalised)			(73,359)	(95,121)	(91,394)	(107,628)	(158,468)
Income tax expense (includes exceptionals)			0	0	0	0	0
Net income (reported)			(74,099)	(96,120)	(91,653)	(107,887)	(158,727)
Net income (normalised)			(73,359)	(95,121)	(91,394)	(107,628)	(158,468)
Basic average number of shares, m			38.2	40.6	47.7	51.7	51.7
Basic EPS (SEK)			(1.94)	(2.37)	(1.92)	(2.08)	(3.07)
Adjusted EPS (SEK)			(1.92)	(2.34)	(1.92)	(2.08)	(3.06)
Dividend per share (SEK)			0.00	0.00	0.00	0.00	0.00
BALANCE SHEET							
Tangible assets			1,197	5,919	4,317	10,282	10,295
Intangible assets			83,269	82,270	82,011	81,751	81,492
Other non-current assets			0	0	0	0	0
Total non-current assets			84,466	88,189	86,328	92,033	91,787
Cash and equivalents			134,442	110,527	277,009	170,028	13,620
Inventories			0	0	0	0	0
Trade and other receivables			6,028	9,351	6,732	6,732	6,732
Other current assets			0	0	0	0	0
Total current assets			140,470	119,878	283,741	176,760	20,352
Non-current loans and borrowings			0	0	0	0	0
Non-current lease liabilities			0	2,900	1,270	7,270	7,270
Other non-current liabilities			0	0	0	0	0
Total non-current liabilities			0	2,900	1,270	7,270	7,270
Accounts payable			5,997	8,438	3,683	4,295	6,368
Non-current loans and borrowings			0	0	0	0	0
Current lease liabilities			0	1,643	1,657	1,657	1,657
Other current liabilities			6,463	13,259	15,578	15,578	15,578
Total current liabilities			12,460	23,340	20,918	21,530	23,603
Equity attributable to company			212,476	181,826	347,879	239,992	81,265
CASH FLOW STATEMENT							
Operating income			(73,897)	(95,848)	(91,458)	(107,441)	(158,281)
Depreciation and amortisation			1,332	2,932	2,256	3,349	3,740
Share based payments			0	0	0	0	0
Other adjustments			(202)	(244)	(195)	(446)	(446)
Movements in working capital			1,977	1,959	183	612	2,073
Cash from operations (CFO)			(70,790)	(91,201)	(89,214)	(103,927)	(152,914)
Capex			(1,052)	(137)	(394)	(266)	(330)
Acquisitions & disposals net			0	0	0	0	0
Other investing activities			0	0	0	0	0
Cash used in investing activities (CFIA)			(1,052)	(137)	(394)	(266)	(330)
Net proceeds from issue of shares			131,575	68,970	257,706	0	0
Movements in debt			0	(1,547)	(1,616)	0	0
Other financing activities			0	0	0	(2,789)	(3,164)
Cash from financing activities (CFF)			131,575	67,423	256,090	(2,789)	(3,164)
Cash and equivalents at beginning of period			74,709	134,442	110,527	277,009	170,028
Increase/(decrease) in cash and equivalents			59,733	(23,915)	166,482	(106,981)	(156,408)
Effect of FX on cash and equivalents			0	0	0	0	0
Cash and equivalents at end of period			134,442	110,527	277,009	170,028	13,620
Net (debt)/cash			134,442	110,527	277,009	170,028	13,620

Source: IRLAB company accounts, Edison Investment Research

Contact details	Revenue by geography
Arvid Wallgrens Backe 20 413 46 Gothenburg Sweden +46 (0)31 757 38 00 www.irlab.se	N/A

Management team

CEO: Dr Nicholas Waters

Dr Nicholas Waters worked as a PhD student in Nobel Laureate Professor Arvid Carlsson's research group at the Department of Pharmacology, Gothenburg University (1987–95). Between 1995 and 2000, he managed research collaborations between the university and pharma industry partners. In 1998, he co-founded Carlsson Research. In 2002, he was appointed chief scientific officer at Carlsson Research and in 2006 he was appointed CEO. In 2010, he was appointed executive VP, Research at NeuroSearch. Nicholas continued to serve as CEO of NeuroSearch Sweden until 2012. In 2013, he co-founded IRLAB. In 2007–10, he served as a board member of SwedenBIO, the Swedish biotech industry association. Nicholas holds a PhD in Pharmacology.

CFO: Viktor Siewertz

Viktor Siewertz started his career as an auditor and consultant at Deloitte. In 1999, he moved to the venture capital firm Speed Ventures, where he assisted portfolio companies in business development and financing. From 2001, he worked at HSH Nordbank's corporate finance department, Handelsbanken and Consensus Asset Management, with mid-sized companies in M&A and capital procurement. In 2013, he started a consultancy firm and began working with IRLAB as consultant. He was appointed COO in 2016 and CFO in 2017. Viktor holds an LLM and an MSc in Accounting and Finance from the Gothenburg School of Business, Economics and Law.

CMO: Dr Joakim Tedroff

Dr Joakim Tedroff is a practising neurology specialist in neurodegenerative disorders. He is also an associate professor of neurology at the Department of Clinical Neuroscience at Karolinska Institutet. Joakim has more than 15 years' experience in the pharmaceutical industry. He was co-founder of Carlsson Research and IRLAB. He previously held a position as scientific VP at NeuroSearch. He is currently engaged as a board member of Stockholm Brain Institute at Karolinska Institutet and as chairman of Abera Bioscience. Joakim has served as a consultant for a number of pharmaceutical companies in the neurology field including Allergan, Orion, Pfizer and Lundbeck, as well as for venture capital firms in life science projects.

CSO: Dr Clas Sonesson

Dr Clas Sonesson started his career at AkzoNobel. In 1989, he was appointed to the position of medicinal chemist in Nobel Laureate Professor Arvid Carlsson's research group at the Department of Pharmacology, Gothenburg University. In 1998, he co-founded Carlsson Research and between 1998 and 2002 he served as a member of the board of directors at Carlsson Research. In 2000, he was appointed head of medicinal chemistry and in 2002 director of chemistry and IP (2002–06). From 2006 to 2009, Clas was director of chemistry and IP at NeuroSearch Sweden. In 2009, he was appointed head of discovery (NeuroSearch Sweden 2009–11) and thereafter served as VP, Chemistry & IP (NeuroSearch, 2011–12). In 2013, Clas co-founded IRLAB. He holds a PhD in Medicinal Chemistry.

Principal shareholders	(%)
Försäkringsaktiebolaget, Avanza Pension	7.9
Ancoria Insurance Public	7.4
FV Group	7.1
Fjärde AP-fonden	5.9
Daniel Johnsson	5.2
Tredje AP-fonden	3.6
Futur Pension	3.5
Philip Diklev	3.1
Nordnet Pensionsförsäkring	3.0

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