NEUREN PHARMACEUTICALS LIMITED

Initiation of coverage

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Rosemary Cummins
Senior Research Analyst

New approach offers breakthrough

Neuren Pharmaceuticals is a biotechnology company that is targeting five neurodevelopmental disorders including Rett and Fragile X syndromes. Currently, there are no approved treatments for these conditions. Neuren's drug candidates, trofinetide and NNZ-2591, are optimised versions of two neural acting peptides, which play a key role in the body's nervous system.

Patients present with a wide range of profound symptoms including intellectual, speech and movement disabilities. To date, the complexity of the nervous system has limited care to symptomatic relief. As versions of normal body compounds, Neuren's drug candidates 'mimic' their roles and address the underlying pathological features. Studies to date support both drugs' ability to help restore neural form and function.

Strong pipeline with late stage asset

In 2018, Neuren licensed the North American (NAM) rights for its late stage drug candidate, trofinetide, to Acadia Pharmaceuticals (NASDAQ: ACAD). Acadia has commenced a Phase III trial in Rett syndrome. If the trial results confirm the efficacy and safety data of the Phase II trial, regulatory approval and market entry are likely in early CY22.

Under the Acadia agreement, Neuren may receive up to USD455m in milestone payments and double-digit percentage royalties based on NAM net sales revenues. Neuren has appointed an advisor to assist in the sale of the exNAM trofinetide rights.

Neuren's other drug candidate, NNZ-2591, is expected to commence clinical trials over CY20 in three neurodevelopmental disorders.

Potential Value Drivers

Three significant value-adding events should emerge over the next two years

- release of Phase III results of trofinetide in Rett syndrome
- release of Phase II results for NNZ-2591 in three conditions
- appointment of a commercial partner for trofinetide's ex North American (NAM) markets.

Neuren's licensing agreement with Acadia specifies developmental milestones of USD105m. A positive Phase III trial with US market entry in early CY22 are likely to trigger the Rett Syndrome portion of these milestone payments. Assuming the exNAM rights are acquired, milestone payment for approval and launch into the exNAM markets are likely to follow.

Valuation

A risk-adjusted discounted cash flow (DCF) presents a valuation of $436m, implying a share price of $4.25. The risk adjustment is based on industry data that measures probability of success at the different development stages. Current global markets data have been used to estimate sales, potential milestone payments and royalties for trofinetide and NNZ-2591. The valuation is supported by comparison to ASX-listed biotech companies Opthea (OPT.AX) A$753m Paradigm (PAR.AX) $581m, that are developing drugs at a similar stage.

neuren

Neuren Pharmaceuticals is an ASX listed biotechnology company developing two drugs, trofinetide and NNZ-2591.
Trofinetid has commenced Phase III trial in Rett Syndrome with results expected over CY21.

Acadia Pharmaceuticals has licensed the North American (NAM) rights to trofinetide.
A corporate advisor has been appointed to explore the sale of the ex NAM rights for trofinetide. There is a strong relationship between trofinetide and Neuren's second compound, NNZ-2591. Clinical success in trofinetide is likely to draw interest in NNZ-2591.

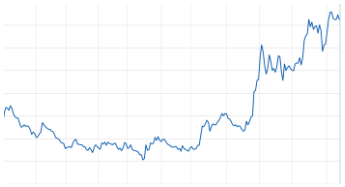
Trofinetide and NNZ-2591 are targeting five disorders. Their mechanism of action offers the potential to address a much wider range of neural diseases and trauma related injury.
Board and management are well credentialled with expertise in drug development and commercialisation.

Company Data	
Stock	NEU.AX
Price	$2.62
Market cap	$270.3m
Shares on issue	103.2m
Cash Sept CY19	$16.49m

Next steps

- Appoint partner for exNAM rights to market trofinetide;
- Commence Phase I & II clinical trials NNZ-2591

2019 Share price performance



Investment Thesis:

Mechanism of action offers wide application in difficult conditions

Neuren's (NEU.AX) two candidate drugs, trofinetide and NNZ-2591, target debilitating neurodevelopmental diseases for which there are currently no approved treatments. Patient care is limited to ad hoc management of symptoms. By addressing the underlying pathological processes, trofinetide and NNZ-2591 present the potential to modify brain impairment and improve function across multi system areas.

Well advanced asset

Value increases and risk decreases as candidate drugs progress along the development path. Neuren offers a well advanced pipeline with trofinetide currently in Phase III trial and NNZ-2591 planned to undertake its Phase I and II trials over CY20/21. The trofinetide trial is relatively short with results expected to emerge over CY21 with possible submission for approval of trofinetide in the same year. As an Orphan Drug, the New Drug Application (NDA) will qualify for a 6 month Priority Review by the US Food and Drug Administration (FDA), rather than the standard 10 months.

Strong support data

In terms of proof to date, trofinetide demonstrated a statistically significant benefit during a paediatric Phase II trial in Rett Syndrome. Positive trends in the adult patients were observed in an earlier trial. The Phase II trial results have been published in the medical journal, Neurology. The licensing agreement of the NAM rights for trofinetide to NASDAQ listed Acadia Pharmaceuticals presents further validation from peer review. Phase II data in Fragile X have also shown improvement in pre-specified criteria. Highly encouraging results in animal models supported the designation of Orphan Drug status for NNZ-2591 in each of its three targeted disorders.

Potential expedited approval

Preclinical study data support efficacy of NNZ-2591 in a range of neurological conditions. The current lack of effective treatments for these severely debilitating diseases may grant NNZ-2591 an expedited approval if strong data is elicited in the Phase II trial.

Further corporate interest

Corporate activity may emerge. Neuren has engaged Torreya, a life sciences investment bank, as an advisor to the possible sale of the exNAM rights for trofinetide.

Open market opportunity

The lack of treatments in neurodevelopmental disorders presents Neuren with an open market. The mechanisms of action (MOA) of both Trofinetide and NNZ-2591 target the underlying pathological features of brain impairment creating the potential for trofinetode and NNZ-2591 to treat a wide range of neurological conditions – developmental, degenerative and traumatic. Corporate interest in both drugs is likely to increase if proof of efficacy continues to emerge.

Potential Near-Term Catalysts

- CY19/20 Licensing/sale of exNAM rights of trofinetide
- CY20 Phase I clinical trial and commencement of Phase II clinical trial for NNZ-2591 in Phelan-McDermid, Angelman & Pitt Hopkins syndromes
- CY21 Release of the results for Phase III clinical trial of trofinetide in Rett Syndrome
- CY21 Release of results for Phase II clinical trials of NNZ-2591
- CY21 New Drug Application (NDA) submission to FDA for approval of trofinetide in Rett Syndrome
- CY21/22 Announcement of the Rett portion of the USD105m developmental milestone payments as per the Acadia Pharmaceuticals' licensing agreement and potential additional payments for exNAM rights
- CY22 FDA approval of trofinetide in Rett Syndrome with further developmental milestone payments

Risks and Sensitivities

Drugs in development carry approval and commercial risk. Early clinical data support trofinetide's potential efficacy and safety, however, pivotal trials to gain regulatory approval are still to be undertaken. NNZ-2591 is at an earlier stage so carries a lower probability of approval. From a financial perspective, Neuren must secure funding to progress NNZ-2591's development to market. Neuren may not be able to secure a licensing agreement to market trofinetide outside North America. Commercial performance, market size, pricing, patient populations, timing of regulatory approval are based on market averages so there is upside and downside risk in the valuation assumptions.

Neural complexity has thwarted treatment to date

Neuren's two drug candidates, trofinetide and NNZ-2591, are targeting neural disorders. The human nervous system is the body's wiring system acting via a relay of electric impulses that assist in controlling the body's different systems. Its role includes

- cognition, learning and memory
- movement – including both voluntary such as speech and walking and involuntary movements such as breathing and blinking
- autonomic functions - heart function, blood pressure, digestion, sweating and shivering.
- senses - sight, hearing, taste, touch and smell are all controlled by the nervous system.

Loss of function or efficiency in any component of the nervous system can have a profound effect on the body. The nervous system is complex and highly integrated with the other body systems, which poses challenges to treat any impairment or dysfunction. However, the commonality of how the nervous system functions and its widespread effect in the body's other systems opens the opportunity for therapeutics to have effect across many neural diseases.

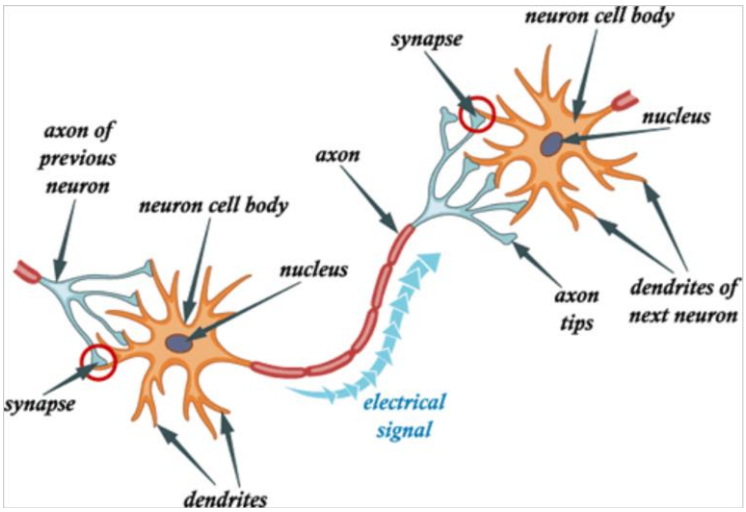


Exhibit 1- Diagram of a nerve cell pathway

Source -pulpbits.

The nervous system comprises neurons or nerve cells imbedded in different support cells. The neuron is a specialised cell with dendrites that receive stimuli from surrounding cells. The neuron's elongated tail, the axon, passes an electric signal to the neighbouring cells. The electric signal passes the gaps or synapses between the neurons via neurotransmitter fluids. A neuron may receive signals from many cells and then may pass it on to multiple cells. The signal continues until it reaches the target cell where it can induce the desired action – for example a muscle contraction to move or take a breath, establish a memory. A complex system of support cells and regulatory proteins coordinate with one another to control the nervous system. Disruption of any part of the nervous system can have widespread effect leading to profound symptoms affecting all the body's systems.

Replication of key proteins offers potential breakthrough

Insulin growth factor 1(IGF-1) is one of the key regulatory proteins in the brain. It is critical for brain development and bodily functions such as movement and cognition. IGF-1 promotes the normal transmission of signals along the neural pathways to the muscles, blood vessels, gastrointestinal tracts and other organs. It is also important in the body's response to disease, stress and injury.

IGF-1's availability is controlled by a complex loop of circulating body proteins or factors. These include the glycine-proline-glutamate or glypromate (GPE) and cyclic-glycine-proline (cGP) peptides. They are metabolites or by-products produced by IGF-1 as it is broken down. Their exact roles are still being explored but it is known that they act to normalise IGF-1 function and are therefore important in pathophysiological conditions. GPE helps regulate the microglia neural support cells that maintain the synapses. cGP is active in regulating the bioavailability of circulating IGF-1 in the brain and improves brain development by acting as a protective agent.

Neuren has developed two synthetic versions or analogues of GPE and cGP, trofinetide and NNZ-2591 respectively. They act to replicate the activity of the naturally occurring peptides. The analogues have been enhanced to provide clinical advantages. From a pharmacokinetic perspective, they are more stable and are given orally which is important in the treatment of children. In contrast to almost all other neural targeting drugs, they readily cross the BBB so can offer more effective treatment. The BBB acts a layer of protection for the brain whereby it restricts the passage of any compounds in the surrounding blood vessels from passing into the brain cells. Almost all drugs are unable to breach the barrier.

Shared pathological features offers wide ranging treatment

Studies have shown that different neural diseases and brain trauma often trigger the same pathological effects at the cellular and molecular level. The common pathological features include the following;

- inflammation -The release of cytokines in inflammation can result in a disruption of signal delivery, often leading to cell death and seizures in patients.
- dendrites changes - Alterations in the number, density, size, and shape of dendritic spines have been correlated with neuronal dysfunction in several disorders associated with intellectual disability, including Rett syndrome.
- impairment of support cells - Microglia, one type of neural support cells, have a central role in maintaining the dendrites. They are commonly affected in neurological disease, impairing the transmission of the electric signals. They also respond to disease by producing inflammatory cytokines which can further compound the damage to neuron signalling.
- reduced IGF-1 levels - Studies have shown depressed levels of IGF-1 in a wide range of neurological disorders.

The combined effects of these pathological features result in impairment of the neural signalling and can thereby negatively impact all brain activity with flow-on effects across all the body's systems. Cognition, memory, vascular regulation, voluntary and involuntary movement, growth and physical features can all be affected from impairment of neural signalling. In a research model of Rett syndrome, it has been shown that increasing the levels of IGF-1 corrects deficits in dendritic spines. Studies in isolated cells from human Rett syndrome patients, have shown that both IGF-1 and GPE were able to partially reverse the deficits in cellular function.

Neuren is targeting five neurodevelopmental conditions which include Rett and Fragile X syndromes. In Rett syndrome, the dendrites are sparse and immature while in Fragile X syndrome there is excessive dendritic branching with immature dendrites. Trofinetide, acts to normalise the underlying dendrite control mechanisms and thereby can be effective in treating two different conditions with seemingly opposite needs.

The close relationship of IGF-1, GPE and cGP presents the opportunity for trofinetide and NNZ-2591 to directly impact IGF-1. Trofinetide has demonstrated the ability to raise IGF-1 levels and improve neural signalling in Rett and Fragile X syndromes. Higher levels of IGF-1 are also believed to inhibit the production of inflammatory cytokines.

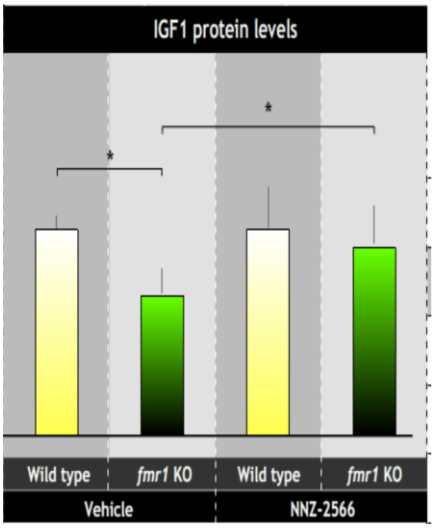
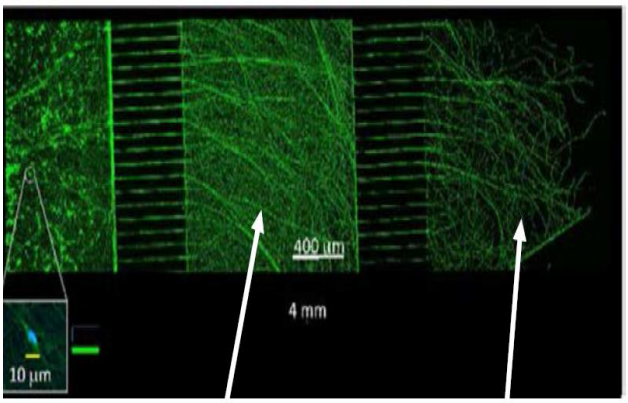


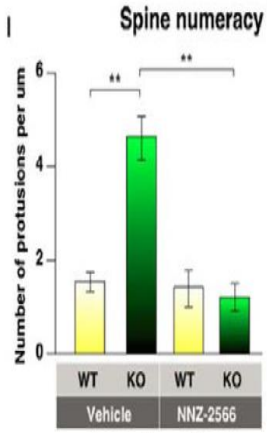
Exhibit 2 - Effect of trofinetide (NNZ-2566) on IGF-1 levels (Neuren Presentation)



Abnormal dendrites in shank3 knockout mice

Normalisation after treatment with NNZ-2591

Correction of abnormal dendritic spines in mouse models:
Left - Phelan-McDermid syndrome (*shank3*)
Right - Fragile X syndrome (*fmr1*)



Correction in fmr1 knockout mice after treatment with trofinetide (NNZ-2566)

Exhibit 3 - Effect of NNZ-2591 and trofinetide on dendrites (Neuren Presentation)

Initial disease targets

Neuren's pipeline includes the use of trofinetide in Rett and Fragile X syndromes and NNZ-2591 for Phelan McDermid, Angelman and Pitt Hopkins syndromes. Each disorder arises from a gene mutation that causes the loss of a protein that helps regulate the nervous system. All the targeted illnesses have an underlying impairment of the neural cell connections and signalling between neurons.

Rett syndrome

Rett syndrome is a progressive X-linked chromosomal disorder that is associated with severe disability. It mainly affects girls with an incidence estimated to be between 1:10,000 and 1:15,000 female births worldwide. It is significantly rarer in males. Symptoms present from 6-12 months of age with developmental regression resulting in the onset of a variety of effects,

including motor or movement impairment, loss of acquired language, intellectual disability, seizures, and anxiety. Autonomic dysfunction caused by Rett Syndrome affects breathing and can result in cardiovascular and gastrointestinal difficulties.

The majority of Rett-affected patients carry mutations in methyl CpG-binding protein 2 (MECP2) gene. More than 620 mutations in the MECP2 gene have been identified. The gene encodes the MECP2 protein. Through a process known as alternative splicing, MECP2 is believed to help modify other proteins that are important in the brain cell function and critical for normal communication between neurons.

In Rett syndrome, microglia and astrocytes, key neuron support cells, are overactive with exaggerated maintenance of existing synapses leading to underdeveloped and too few synapses.

Fragile X Syndrome

Fragile X syndrome, an inherited mutation of the X chromosome, causes a range of developmental problems including learning disabilities and cognitive impairment. It is more common and more severe in males with an incidence of 1:4,000 to 1:7,000 while the incidence in girls averages at 1:6,000 to 1:11,000 with about half of the girls suffering symptoms. It is the most common inherited cause of intellectual disabilities and highest cause of autism. Other symptoms include anxiety, mood instability, seizures, intellectual disability with delayed speech and language development. The patients also share common physical characteristics.

Fragile X involves the Fragile X mental retardation-1 (FMR1) gene which provides instructions for making a protein called FMRP. FMRP protein is present in many tissues, including the brain and is believed to play a role in the development of the synapses. 'Synaptic plasticity' allows synapses to change and adapt over time in response to experience therefore FMRP's role is believed to be important for learning and memory.

Phelan-McDermid (PMS) Syndrome

The prevalence of Phelan-McDermid Syndrome (PMS) has been estimated to be between 1:8,000 and 1:15,000 with both males and females equally affected. Symptoms usually appear in early childhood with patients displaying developmental delay, intellectual disability, and absent or severely delayed speech. Other common symptoms include autism, low muscle tone, breathing difficulties, poor motor control, seizures, and epilepsy. Some children present with heart or kidney defects. The patients share facial and body characteristics.

PMS results from the mutation of the SHANK3 gene. PMS is also referred to as 22q13 Deletion Syndrome which indicates the chromosomal position. The SHANK3 gene provides instructions for making the SHANK3 protein which is most abundant in the brain. Its neural role is related to directing cell-to-cell communication particularly between the synapses as it is involved in the formation and maturation of dendritic spines. Within synapses, the SHANK3 protein acts as a scaffold that supports the connections between neurons, optimising the transmission of signals from one neuron to another.

Angelman Syndrome

Angelman syndrome (AS) is estimated to affect between 1:12,000 and 1:24,000 people. It is a complex genetic disorder caused by a mutation of the ubiquitin protein ligase E3A (UBE3A) gene that results in a loss or impairment of its corresponding protein, UBE3A. The loss of UBE3A leads to an array of symptoms that primarily affect the nervous system. Patients present with delayed development, intellectual impairment, speech and balance problems, seizures and ataxic movement. Common physical characteristics include a small head.

Studies suggest that UBE3A protein's role is critical to the normal development and function of the nervous system by helping to regulate proteostasis, the balance of protein synthesis and degradation at the synapses. It also is believed to have a role in synaptic plasticity. The protein is abundant in the hippocampus and cerebellum areas of the brain which are important for memory and movement.

Pitt Hopkins Syndrome

Pitt Hopkins syndrome (PTHS) is estimated to affect between 1:11,000 and 1:41,000 people, both males and females. It arises from mutation of the transcription factor 4 (TCF4) gene. At least 50 different mutations in the TCF4 gene have been found to result in Pitt-Hopkins syndrome. The TCF4 gene encodes for TCF4 protein which is important in controlling the development of specialised cells. It is found in the brain, muscles, lungs and heart. PTHS is characterized by severe intellectual disability and development delay with poor or no speech and distinctive facial features with a small head. Breathing problems, epilepsy and autism are common. It also plays a role in the maturation of cells to carry out specific functions (cell differentiation).

Development and commercialisation of Neuren's pipeline

In August 2018, Acadia Pharmaceuticals Inc, a US based biopharmaceutical company (NASDAQ: ACAD), acquired the North American rights to trofinetide for all indications, including Rett and Fragile X syndromes. Acadia specialises in the development and commercialization of innovative medicines used to treat central nervous system disorders. Under the terms of its license agreement with Acadia, Neuren received an upfront USD10 million plus potential milestone payments of up to USD455 million. The potential payments consist of USD105 million subject to the achievement of development milestones for Rett and Fragile X syndromes and up to USD350 million subject to the achievement of thresholds of annual total net sales of trofinetide in North America.

In addition, Neuren is eligible to receive tiered, escalating, double-digit percentage royalties on net sales of trofinetide in North America. Acadia will fund and execute the remaining development for trofinetide in the treatment of Rett syndrome in North America. Neuren has free access and rights to use all the technical, clinical and regulatory data developed by Acadia in the US to seek approval and commercialise the drug in countries outside North America.

The agreement also provides for Neuren to receive one third of the market value of a Rare Paediatric Disease Priority Review Voucher, if awarded by the FDA upon market approval of trofinetide in the treatment of Rett Syndrome. Under this program, once a drug is approved for a 'rare paediatric disease' the sponsor qualifies for a voucher that can be redeemed to receive a priority review of a subsequent marketing application for a different product. The vouchers can be sold. Review of recent sales shows an average voucher price of around USD100m.

Neuren retains the rights to the rest of world (ROW) markets which include EU and Japan. Registration for the Japanese market usually requires additional clinical data through a small pharmacokinetic trial in a Japanese population. In February 2019, Neuren announced the appointment of Torreya, a specialist global life-sciences investment bank as a corporate advisor. It will evaluate all potential corporate transactions. Acadia may express an interest.

Clinical Trial program

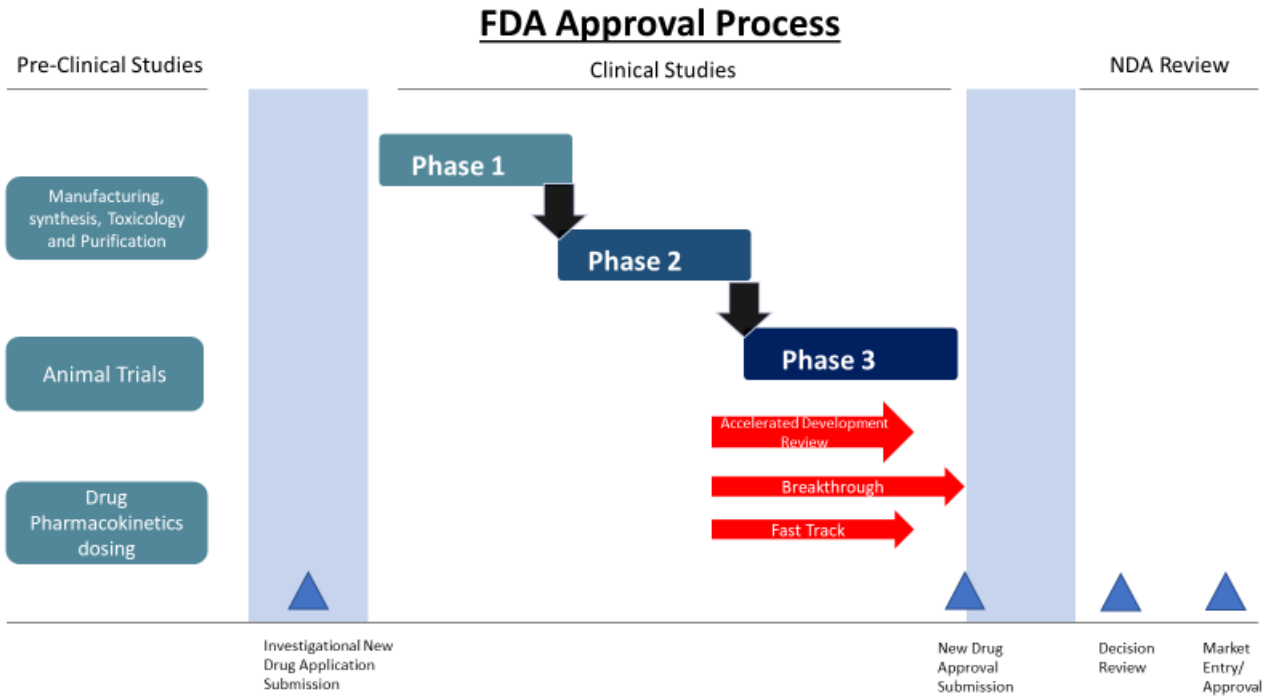


Exhibit 4 FDA Approval Process (Source FDA)

The clinical trial program typically consists of three phases. The Phase I trial is the first-in-person trial, so it is predominantly designed to confirm the safety of the drug. The participants of a Phase I trial usually comprise healthy volunteers. Phase II trials are designed to elicit first signs of efficacy. Efficacy is usually determined by the comparison of the trial drug to the current standard of care (SOC) for the target disorders. However, as there are no approved treatments for these conditions, efficacy is likely to be based on improvement in a series of neurobehavioural measures assessed by the caregivers and health professionals.

As the US represents the largest pharmaceutical market, many drugs are trialled under FDA requirements to gain access. Agreements between the different regulatory regions allows the registration data from the FDA trials to support registration in markets such as the EU and Japan. There may be a requirement to supply additional data. As an example, registration in Japan may require the clinical trial patients to include a defined percentage of Japanese patients or an additional trial that includes Japanese patients.

Neuren Drug Development Pipeline					
		Stage of Development			
		Value →			
		Risk ←			
Drug	**Syndrome**	**Preclinical**	**Phase I**	**Phase II**	**Phase III**
Trofinetide					
	Rett				
	Fragile X				
NNZ-2591					
	Phelan McDermid		X		
	Angelman		X		
	Pitt Hopkins		X		
X = planned to commence in CY20					

Exhibit 5 - Planned Neuren Development pipeline (Neuren presentation)

Trofinetide Development Program

Rett Syndrome

Trofinetide Development Program		
Lavender	**Lilac**	**Lilac 2**
Phase III Pivotal trial Double blind Week 1- 12	Follow on Open Label Weeks 12 - 52	Folllow on Open label On going

Exhibit 6- Planned Phase III Trial and follow on trials for trofinetide in Rett Syndrome (Neuren Acadia announcements)

Pivotal Phase III Trial

The Phase III Lavender trial commenced in October 2019. The trial is a randomized, double-blind placebo-controlled study that entails 12-weeks of treatment. The design of the trial is structured to have two groups, one for trofinetide and one for a placebo drug. Comparison of the two groups forms the basis of whether the drug shows a benefit. To prevent any bias, patient allocation to the groups is randomised and both the patient and clinician are 'blinded' or unaware of the allocation.

The trial will include approximately 180 girls aged 5 -20 years with Rett Syndrome. The co-primary endpoints are improvement in the patient's baseline assessments as measured by the Rett Syndrome Behaviour Questionnaire (RSBQ), a caregiver assessment, and, by the Clinical Global Impression of Improvement (CGI-I), a physician assessment. Positive trial results are expected to enable Acadia to submit a New Drug Application (NDA) to the FDA during CY21. The NDA submission and subsequent FDA approval are likely to trigger milestone payments.

From a risk perspective, the Lavender trial has a number of important features in comparison to Neuren's paediatric Phase II trial. The Phase II trial showed statistical significance in both the RSBQ and CGI-I assessments.

- The Phase III trial will include 180 girls which is more than 3 times larger than the Phase II trial. Therefore, it offers a significantly higher statistical ability to detect the superiority of trofinetide over placebo or non-treatment drug.
- The treatment period is 12 weeks in comparison to 6 weeks in the Phase II trial. The positive effect in the Phase II trial was continuing to increase when the trial ended, suggesting that a longer treatment period may produce a larger effect.

- Finally, the dosing regimen for the active group in the Phase III trial has been optimised based on analyses of the clinical effect of the different dosing regimens used in the Phase II trial.

Follow on studies

The 'Lilac' and 'Lilac 2' studies are planned to follow the Lavender trial and would allow eligible patients to continue treatment. The purpose of the Lilac and Lilac 2 studies is twofold. Firstly, the studies will allow examination of longer-term safety data. Secondly, the continuation of treatment will present the opportunity to assess if prolonged treatment continues to provide improvement. The nervous system is dynamic, showing adaptation and learning from ongoing stimuli and experience. The improvement from trofinetide may result in additional benefits as treatment may allow the nervous system to mature and develop correctly. Confirmation of the continued improvement observed in the Phase II trial, may support a recommendation for on-going therapy. The 'Lilac 2' study will allow continued treatment for eligible patients before marketing approval is achieved.

Proof to date – Results of Phase II trials

Neuren conducted a Phase II double-blind placebo-controlled dose ranging study in girls with Rett syndrome who were aged between 5 to 15 years old. The study demonstrated a statistically significant and clinically meaningful improvement in the RSBQ, the CGI-I and RTT DSC. The RSBQ, CGI-I and RTTDSC are completed by the participants' caregivers and the attending clinicians and measure function, behaviour and quality of life of the patients before and after initiating the study medication. Due to the wide range of symptoms that the patients can experience, the assessments are designed to cover both condition-specific and patient-specific changes.

Overall, the RSBQ showed ~16% mean improvement from treatment baseline. The higher dose of trofinetide showed statistically significant and clinically relevant improvements in comparison to the results measured for the placebo drug in the RSBQ, RTT DSC (Visual Analog Scale) and CGI-I assessments. Clinician-based CGI-I reported that 22% of the patients treated with trofinetide had a 'much improved' rating versus 4% of the placebo group. Trofinetide was well tolerated and no safety concerns were identified.

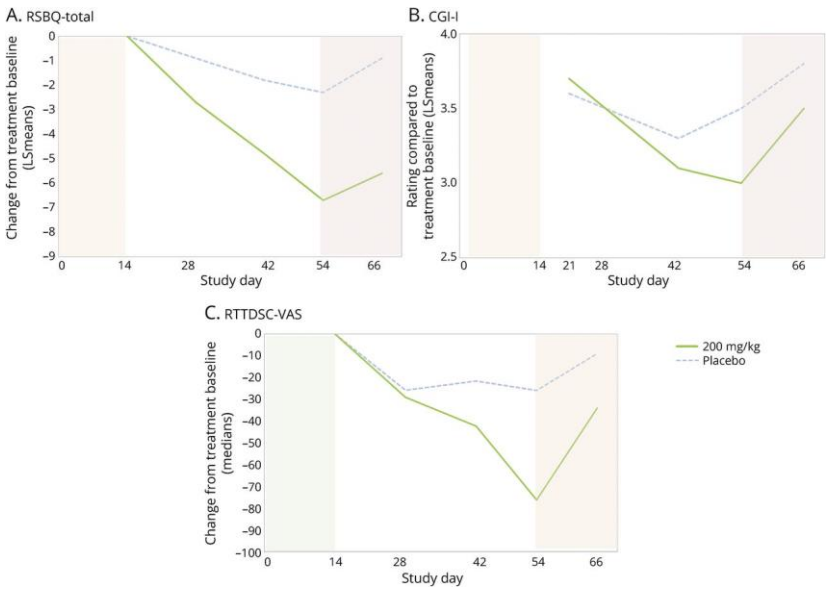


Exhibit 7 Improvement with trofinetide in Rett Syndrome CGI-I, RSBQ and RTTDSC at 200mg/kg dose versus placebo (Neuren presentation)

This trial followed positive trends observed in an earlier Phase II trial in adolescents and adults aged 16 to 45 years with Rett syndrome.

Fragile X Syndrome

Clinical trial program

Acadia has licensed the development and commercialisation rights of trofinetide in North America. Acadia is yet to confirm a schedule for the further development program of trofinetide. The Phase III trial for Rett Syndrome is likely to facilitate the planning of clinical trial program for Fragile X syndrome and therefore its timing may be linked to the Rett Phase III trial.

Supporting data - Results of Phase II trials

In terms of proof to date, a double-blind, placebo-controlled Phase II trial in 70 subjects aged 12 to 45 years demonstrated consistent trends in clinical improvement as measured by clinician and caregiver assessments. Assessment is performed in three parts, incorporating measures developed by clinicians looking at changes in both general syndrome symptoms and patient-specific measures. The Aberrant Behaviour Checklist, Fragile X-specific showed that participants reported higher tolerance of sensory input, reduced anxiety, better self-regulation and more social engagement. Trofinetide was well tolerated and no safety concerns were identified.

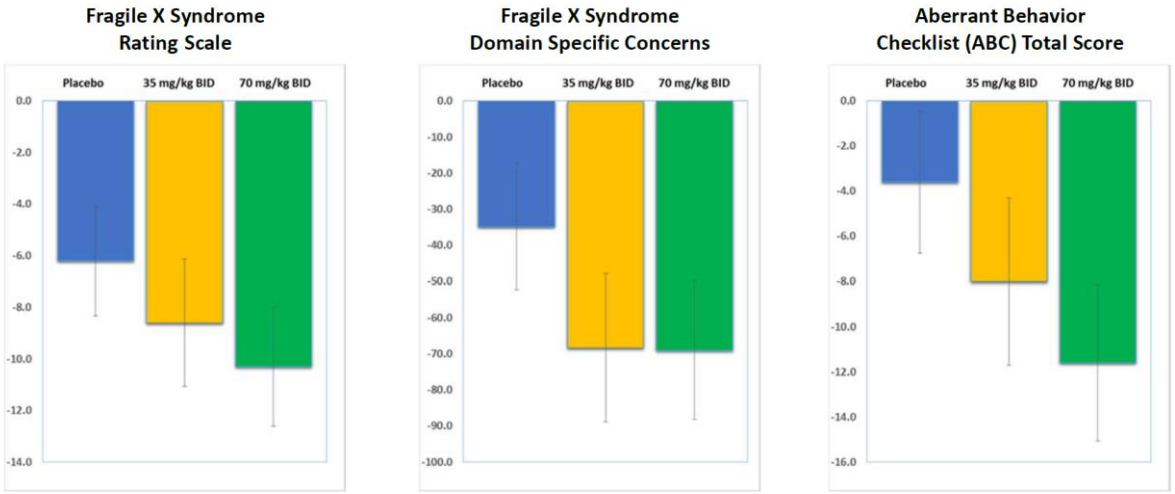


Exhibit 8 Reduction in symptoms - Three assessments (Rating Scale, Domain Specific Concerns and Checklist (ABC) Total Score) formed the basis of comparing patients who received trofinetide and those who received a placebo drug. Both groups who received trofinetide (35mg and 70mg doses) were reported as having a higher reduction in symptoms to those who received the placebo drug.

NNZ-2591 Development Program

Initial targets

Neuren's initial targets for NNZ-2591 are three neurodevelopmental conditions, Phelan McDermid, Angelman and Pitt Hopkins syndromes. The data required to support Neuren's application to commence clinical trials are being generated. It includes toxicology studies, manufacturing optimisation and pharmacokinetic parameters. Neuren is expected to submit an Investigational New Drug application (IND) to the FDA in CY20 to obtain approval to commence clinical trial. A Phase I trial will be conducted in Australia in which demonstration of safety will be the primary purpose. As NNZ-2591 is an analogue of the body's cGP peptide, safety is not viewed as high a risk as in other drugs with new chemical entities (NCE) or novel drug compounds. As the Phase I trial is likely to enrol healthy volunteers, the first signs of efficacy are not expected to emerge until the Phase II trial.

Phase II trials are expected to commence in H2CY20. The Phase II trials are likely to entail 12 weeks of treatment and may include patients from all three targeted conditions, Phelan McDermid, Angelman and Pitt Hopkins syndromes. Neuren may first undertake an open label trial to confirm the optimum dose. Some indications of safety and efficacy may be seen over the Phase II trial. Children will be included in the cohort as their central nervous systems are regarded as being more plastic or adaptable and therefore may elicit clearer signals of NNZ-2591's efficacy. First results are expected in CY21. A subsequent pivotal trial of

the optimum dose versus placebo is likely to be required to support a New Drug Application (NDA) to gain approval. However, the trial may potentially be a Phase II/III trial which would significantly reduce the development time required for market approval.

Supporting data in animal model studies

In terms of proof to date, several preclinical studies support NNZ-2591's use in the specified target conditions. The studies included comparison of shank3 model or 'Phelan McDermid Syndrome model' mice (mice in which PMS was induced) with normal 'wild type' mice before and after treatment with NNZ-2591. Changes in behavioural habits such as sniffing, burying marbles, and nest building were assessed. The studies demonstrated that NNZ-2591 produced a 60% reduction in seizure rates and normalised four behavioural and two movement deficits. No effect was seen in the normal wide type mice.

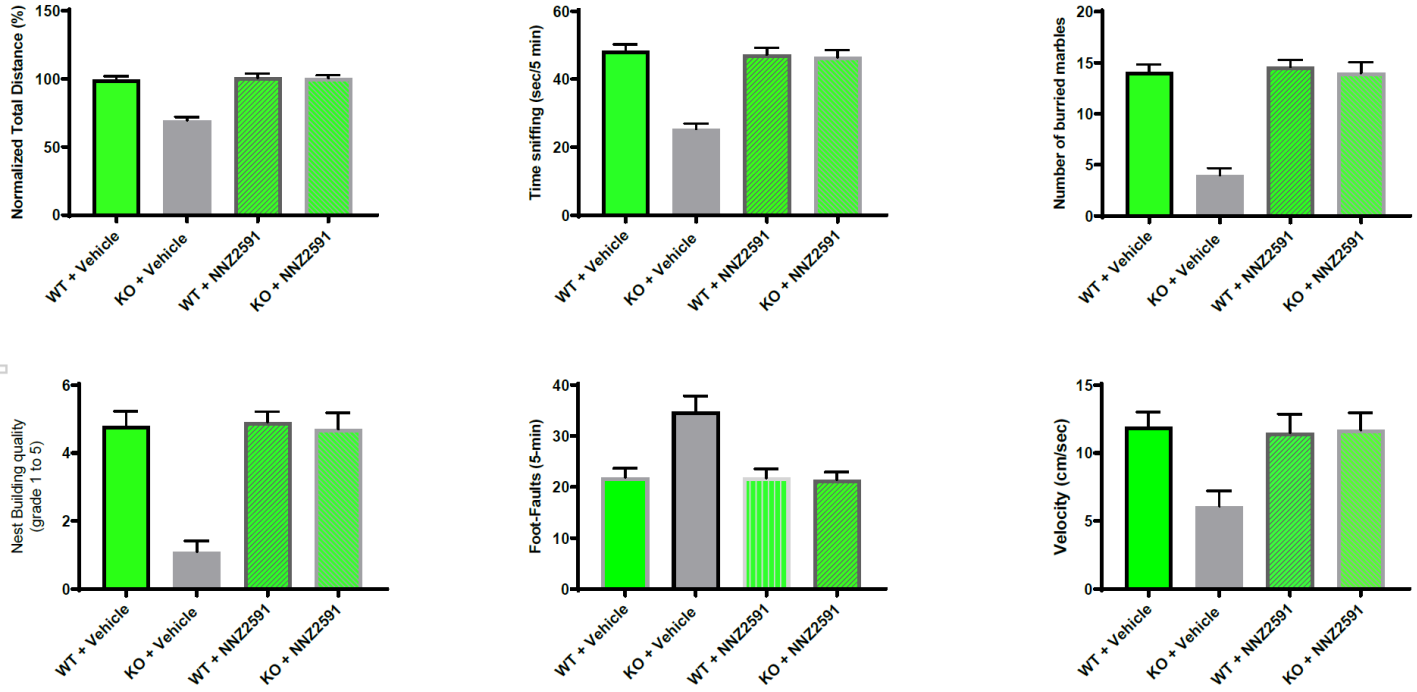


Exhibit 9 Reduction in behavioural symptoms NNZ-2591 treatment in PMS model mice

The graphs compare the two mice groups - WT – wild type or normal mice and KO – the Phelan-McDermid mice with SHANK3 gene 'knocked out'. The first two bars on each graph demonstrate the difference in a social or movement skill. The second two bars of each graph show the impact of NNZ-2591. In each case administration of NNZ-2591 resulted in the Phelan McDermid mouse improvement, scoring the same as the 'normal' mouse.

Similarly, in Angelman Syndrome, preclinical studies have been conducted to compare the effect of NNZ-2591 on the behaviour of ube3a knockout mice (induced Angelman mice) and 'normal' mice. Administration of NNZ-2591 normalised all behavioural and movement deficits. Importantly NNZ-2591 eliminated all seizures.

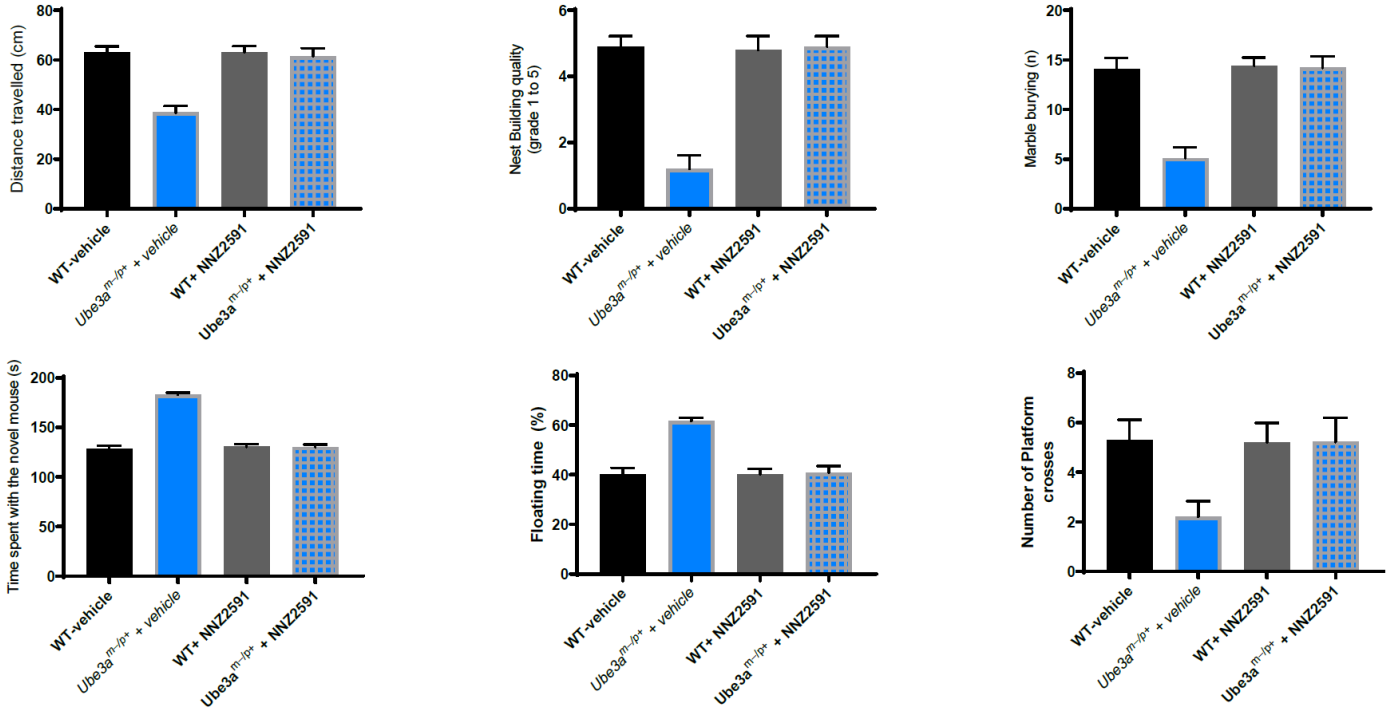


Exhibit 10 Reduction in behavioural symptoms in Angelman syndrome with NNZ-2591 therapy

The WT(Wild Type) mice represent the normal group and the Ube3A are the Pitt Hopkins induced mice cohort. Both groups were assessed in a range of functional and social skills. The Pitt Hopkins mice displayed different behaviour in all measures (first two bars on graph). Administration of NNZ-2591 resulted in the Pitt Hopkins mice performing in keeping with the normal mice (last two bars on graph).

Studies were undertaken to observe the use of NNZ-2591 in tcf4 gene mouse (induced Pitt Hopkins Syndrome model). NNZ-2591 normalised all deficits which included hyperactivity, learning and memory impairments, sociability and motor performance. There was no effect on the 'normal' wild type mice.

Competitor Drugs

There are currently no approved treatments for any of the neurodevelopmental conditions that Neuren is targeting. Patient care is generally based on ad hoc treatment of the presenting symptoms, such as anticonvulsants for seizures, physiotherapy for movement impairment and speech therapy for language delay.

In terms of potential new competitor therapies, Newron Pharmaceuticals is developing Sarizotan for the treatment of Rett syndrome patients with respiratory dysfunction. Patient enrolment in its Phase II/III has been completed, with top line results expected in the last quarter of 2019. Sarizotan has been developed to target patients with significant impaired respiratory function. Respiratory distress occurs in about 70% of patients. Trofinetide has a different MOA, targeting the underlying pathological deficits thereby has the potential to impact all the symptoms. There may be an opportunity to combine the therapies if both are approved.

The targeted neurodevelopmental disorders are caused by gene mutations, which suggests that gene therapy could be a possible treatment. While there is exploration of its use, research is still in early stages of development and therefore is not believed to offer a short to medium term 'cure' or treatment.

Intellectual Property

The patents relating to composition of matter expire in 2022 for trofinetide and in 2024 for NNZ-2591. Method of treatment patents and applications for trofinetide's use in treating autism spectrum disorders have been issued in Europe, Japan and Australia. They extend to 2032. Patents for method of treatment are pending in Canada, Israel and Brazil. NNZ-2591 has been

granted patents for treatment of neurodevelopmental disorders in the US and Japan and are pending in Europe. They expire in 2034.

Under legislation in the US, EU and Japan, New Chemical Entities (NCEs) attract additional coverage. In relation to an NCE, one nominated patent may receive an extension of protection for 5 years. The selection of the patent will be determined on approval.

The FDA and European Medicines Agency have granted orphan drug status to trofinetide in the treatment of Rett and Fragile X syndromes. The FDA has also granted orphan drug status to NNZ-2591 in the treatment of Phelan McDermid syndrome, Angelman syndrome and Pitt Hopkins syndrome.

Orphan drug status confers several advantages, including protection from generic drug competition. The length of the protection varies across the key markets. The protection lasts for 7 years in the US, 10 years in the EU, and 10 years in Japan. For drugs that treat paediatric disorders the US grants a 6-month extension, while the EU offers a 2-year extension.

Valuation

Key Valuation Assumptions		
Probability of Approval		
Trofinetide	Rett	60%
	Fragile X	25%
NNZ-2591	Phelan McDermid	19%
	Angelman	19%
	Pitt Hopkins	19%
Milestone Payments		
	Trofinetide	USD730m
	NNZ-2591	USD370m
Market Entry		
US	Rett	CY22
	Fragile X	CY25
	NNZ-2591	CY26
ROW		
	Rett	CY23
	Fragile X	CY26
	NNZ-2591	CY27
Pricing		
	US	USD160k
	EU	USD96k
	ROW	USD112k

The valuation of Neuren has been derived from a risk adjusted discounted cash flow (DCF). Drugs in development face two key areas of risk – regulatory approval to enter the market and commercial performance.

With respect to approval risk, both drugs are in developmental stage and as yet to undergo the clinical testing required to enter the key markets of US, EU and Japan. The overall probability of a drug being approved increases as it passes through each stage of development. Industry data have been used to provide a guide to the probability of each drug candidate receiving approval. Review of the data shows different areas of disease carry different rates of success. The model uses a blended rate to derive the risk adjustment probability.

Trofinetide in Rett Syndrome carries the highest level of probability of being approved as it is in Phase III trial having demonstrated a statistically significant improvement in its Phase II trial and no safety concerns. Trofinetide in Fragile X has undertaken some clinical testing in Fragile X patients. The valuation assumes a Phase II probability of approval. NNZ-2591 has not commenced clinical trials and carries the lowest probability.

In terms of development costs, the model assumes that trofinetide is licensed for the treatment of Rett and Fragile X syndromes in exNAM markets with all related costs such as registration and marketing passing to the acquiror. Similarly, NNZ-259 is assumed to be licensed for all markets after Phase II trial with further development and commercial costs passing to the acquiror.

In terms of commercial performance, a number of parameters have been assumed. These include pricing, timing of market entry, market penetration, royalty and commercial milestone payments. Current market trends have been used to form the estimates. Pricing dynamics vary significantly across the different markets. The US commands a significant premium to the EU and Rest of World (ROW). The valuation model takes into account the pricing differences. A newly emerged risk involves the US lead review of the drug pricing differences across the key global markets. The US may seek an international benchmarking system. Industry feedback suggests that any measures to introduce equalisation of drug pricing is likely to trigger price increases in the EU and other non-US markets with little net gain or loss.

Neuren has announced it has secured an advisor to commercialise the exNAM rights to trofinetide. The model assumes that the terms of an agreement will similar in royalty percentages with the milestone payments reflective the different market dynamics. Similarly in NNZ-2591, the licensing agreement terms will be reflective of the stage of development and market size.

Market size has been based on the relevant disease prevalence as it is assumed that the patients will require ongoing treatment. Neuren's drugs do not address the underlying gene mutation but rather act as a 'supplement' and therefore there an ongoing need is expected. Diagnosis may present some challenge. For the neurodevelopmental disorders that Neuren is targeting there is an array of symptoms that are common to a number of neurodevelopemental conditions. The difficulty in obtaining an accurate diagnosis may slow market uptake and genetic testing to confirm the diagnosis may be a requirement of approval.

Neurological diseases are usually difficult to treat because of the protective function of BBB which prevents or limits the drug's passage to the targeted brain cells. Both trofinetide and NNZ-2591 are based on naturally occurring molecules and have shown good uptake to date. The model assumes that limitation of uptake across the BBB is not a high risk.

The DCF valuation of $4.26 per share is driven by two factors – timing and revenue. With respect to timing, Phase III trial is the final hurdle before seeking regulatory approval. These trials commonly average three years in duration. The relative shortness of the Rett Syndrome Phase III trial (12 weeks of treatment) brings the timing of possible approval one year sooner than most Phase III trials. Trofinetide has Fast Track Status and Orphan Drug Designation for Rett syndrome in the US which will expedite the regulatory review process by possibly four months.

In terms of revenue, there are a number of significant inflows expected over the next 2-3 years, on the assumption the trials are positive.

- The development milestone schedule under the Acadia agreement amounts to a total of USD105m revenues for both Rett and Fragile X syndromes. Development milestones are triggered by events such as positive trial data, regulatory approval and market entry. If the trial is positive, the timeline could see an NDA submission to the FDA for approval in late CY21 to be followed by market entry in early CY22, triggering the Rett developmental milestone payments over that period.
- Additional milestone payments are also likely as Neuren is currently seeking a partner for its ROW rights for trofinetide. Development milestones are likely to form part of the agreement.
- Market entry would result in the commencement of double-digit net sales royalties, potentially in US from CY22 and the EU and Japan in CY23.

The risk adjusted DCF valuation is supported by comparable ASX listed biotechnology companies. Opthea (OPT.AX) and Paradigm (PAR.AX) are at a similar stage of development with the lead compound preparing for Phase III trial. Their market capitalisations are around AUD753m and AUD581m respectively.

Risks and Sensitivities

The key assumptions of the valuation model present some of the potential risk and sensitivities. Valuation at this stage is based on industry averages. Drug efficacy and safety, market size and penetration will only be confirmed as the drug candidates progress through the development/commercialisation pathway. Reimbursement presents another risk.

The timing of the trials is also a key risk. It may differ significantly from the expected trial periods, presenting upside and downside risk. Factors such as patient recruitment, regulatory approvals, funding may result in delay or abandonment of the project/s.

The financial model assumes that additional funding will be required to commercialise Neuren's R&D program. It assumes a capital raising. However, Neuren may choose to delay the NNZ-2591 trials until trofinetide milestone payments are received or seek a partner earlier which may change the forecast milestone/royalty payments.

The clinical trials to date have not shown any signs of serious side effects. However, one of the purposes of the larger Phase III trials is to look more closely for potential adverse effects. Serious complications from the therapy could result in the trial being abandoned.

There is upside risk. Neuren is targeting diseases for which there are no approved treatments. Clear signs of patient benefit may result in expedited approval. Neuren is currently targeting five syndromes. The MOA of both trofinetide and NNZ-2591 act to 'normalise' the underlying pathological features of the diseases and therefore both trofinetide and NNZ-2591 are likely to be applicable to more neurodevelopmental conditions. Formal approval may be undertaken and/or the drugs may be used 'off label' as clinicians try to relieve shared symptoms with other neurological conditions. No value has been assigned to these potential additional applications.

Neuren Pharmaceuticals

Statement of Comprehensive Income

Year end 31 December

A$ 000's	**2018a**	**2019e**	**2020e**	**2021e**	**2022e**	**2023e**	**2024e**
Revenue							
Revenue from Licence	13,544	0	14,500	29,000	58,820	52,087	33,364
Australian R&D tax incentive	446	300	800	300	0	0	0
Gross profit	13,098	300	15,300	29,300	58,820	52,087	33,364
Expenses							
R&D	-6,101	-10,986	-7,250	-29,000	0	0	0
Administration	-2,074	-2,000	-2,000	-2,000	-2,000	-2,000	-2,000
Other -	-3,921	0	0	0	0	0	0
Amortisation of intangibles	-72	-72	-72	-72	-72	-72	-72
Depreciation	-6	-6	-6	-6	-6	-6	-6
Operating profit (loss)	1,002	-12,686	5,972	-1,778	56,742	50,009	31,286
Interest received	218	400	384	411	821	1,207	1,759
Interest paid	0	0	0	0	0	0	0
Net interest received	218	400	384	411	821	1,207	1,759
Profit (loss)before income tax	3,073	-12,286	6,356	-1,367	57,563	51,217	33,045
Tax	0	0	0	0	0	0	0
Profit after income tax	3,073	-12,286	6,356	-1,367	57,563	51,217	33,045
Total comprehensive profit (loss) attribu	3,073	-12,286	6,356	-1,367	57,563	51,217	33,045
Income tax expense	0	0	0	0	0	0	0
Marginal tax rate (%)							
Profit after tax	3,073	-12,286	6,356	-1,367	57,563	51,217	33,045

Statement of Financial Position

A$ 000's	**2018a**	**2019e**	**2020e**	**2021e**	**2022e**	**2023e**	**2024e**
Trade and other receivables	942	942	942	942	942	942	942
Cash and cash equivalents	23,576	11,426	32,840	31,551	89,192	140,487	173,610
Other (Financial assets measured at fa	2,121	2,121	2,121	2,121	2,121	2,121	2,121
Total current assets	26,639	14,489	35,903	34,614	92,255	143,550	176,673
Non-current assets							
Property, plant and equipment	2	2	2	2	2	2	2
Intangible assets	1	1	1	1	1	1	1
Total non-current assets	3	3	3	3	3	3	3
Total assets	**26,642**	**14,492**	**35,906**	**34,617**	**92,258**	**143,553**	**176,676**
Current liabilities							
Trade and other payables	1,973	1,973	1,500	1,500	1,500	1,500	1,500
Total current liabilities	1,973	1,973	1,500	1,500	1,500	1,500	1,500
Non-current liabilities							
Total liabilities	1,973	1,973	1,500	1,500	1,500	1,500	1,500
Net assets	**24,669**	**12,519**	**34,406**	**33,117**	**90,758**	**142,053**	**175,176**
Minority interest							
Net assets attributable	24,669	12,519	34,406	33,117	90,758	142,053	175,176
Equity	126,426	126,426	141,426	141,426	141,426	141,426	141,426
Other reserves	-8,497	-8,361	-7,830	-7,752	-7,674	-7,596	-7,518
Accumulated deficit	-93,260	-105,546	-99,190	-100,557	-42,994	8,223	41,268
Total Equity	**24,669**	**12,519**	**34,406**	**33,117**	**90,758**	**142,053**	**175,176**

Statement of Cash Flows

A$ 000's	**2018a**	**2019e**	**2020e**	**2021e**	**2022e**	**2023e**	**2024e**
Receipts from licence agreement	13,544	0	14,500	29,000	58,820	52,087	33,364
Receipts from Australian R&D Tax Incer	631	450	800	300	0	0	0
Interest Received	165	400	384	411	821	1,207	1,759
GST refunded	95	0	0	0	0	0	0
Payments for employees and directors	-1,909	-2,000	-2,000	-2,000	-2,000	-2,000	-2,000
R&D and other payments	-6,118	-11,000	-7,250	-29,000			
Net Cash flow (to) /from operating ac	6,408	-12,150	6,434	-1,289	57,641	51,295	33,123
Cash flows from (to) investing activit	0	0	0	0	0	0	0
Net Cash flow (to) /from investing ac	0	0	0	0	0	0	0
Cash flows from financing activities							
Proceeds from issue of shares	11,730	0	15,000				
Payments of shares issue expenses	-17	0	-20				
Net Cash provided frim financing act	11,713	0					
Net increase/decrease in cash	18,122	-12,150	21,414	-1,289	57,641	51,295	33,123
Cash equivalents at beginning of year	4,706	23,576	11,426	32,840	31,551	89,192	140,487
Cash and cash equivalents at end of ye	23,576	11,426	32,840	31,551	89,192	140,487	173,610

Exhibit 11 - Financial forecast to CY/FY 2024. The financial summary has been prepared under the assumption of regulatory approval therefore non-risk adjusted revenue estimates have been included.

Board and Management

The Board members, togther with senior management, offer wide experience across the different areas of drug development. The skills in both preclinical and clinical research, clinical trials, regulatory affiars and commercialisation are valuable and provide good oversight as the development of trofinetide and NNZ-2591 progress through clinical trials to commercilisation.

Board Members

Dr Richard Treagus, BScMed, MBChB, MPharmMed, MBA

Executive Chairman

Richard joined the Neuren Board as Executive Chairman in January 2013. He is a physician, with more than 20 years' experience in all aspects of the international biopharmaceutical industry. Richard served as Chief Executive of the ASX-listed company Acrux Limited from 2006 to 2012. He has held senior roles in large pharmaceutical organisations in the US, EU and Asia. Under his leadership as CEO of ASX litsed Acrux, the company gained FDA approval for three drug products and concluded a product licensing transaction with Eli Lilly worth US$335m plus royalties. Richard is Chairman of BTC Health Limited, which is listed on the ASX.

Dr Trevor Scott, MNZM, LLD (Hon), BCom, FCA, FNZIM, DF Inst D

Non-Executive Director

Trevor joined the Neuren Board in March 2002. He is the founder of T.D. Scott and Co., an accountancy and consulting firm, which he formed in 1988. He is an experienced advisor to companies across a variety of industries. Trevor serves on numerous corporate boards and is chairman of several other companies.

Dianne Angus BSc (Hons), Master of Biotechnology, IPTA

Non-Executive Director - Appointed 1 July 2018

With over twenty-five years' experience in senior executive and non-executive director roles in biotechnology, biopharmaceutical and agri-tech industries, Diane has expertise in business development, capital raising, investor relations, regulatory affairs and intellectual property. She has created numerous global partnerships with companies including Prana Biotechnology, Florigene, Suntory and Monsanto. Dianne holds a Masters degree in biotechnology and is a registered patent attorney.

Patrick Davies B EC, MBA

Non-Executive Director - Appointed 1 July 2018

Patrick has held executive management roles in the Australian and New Zealand healthcare industry for over twenty five years having performed successfully in senior roles across many industry sectors including pharmacy, primary care, pharmaceutical and consumer products. During his ten year period as Chief Executive Officer of EBOS Group Limited (and previously Symbion), the enterprise value of the group achieved compound annual growth in enterprise value of +20% (from circa $450M to in excess of $3.1B).

Dr Jenny Harry BSc (Hons), PhD (Non-Executive Director)

Non-Executive Director - Appointed 7 July 2018

Jenny has 20 years' experience in executive management of companies in the biotechnology and biopharmaceutical sectors. She is currently the Managing Director of Ondek Pty Ltd, an Australian biopharmaceutical company developing new treatments for paediatric allergy. In her previous role, as CEO and Managing Director of Tyrian Diagnostics, Jenny transformed the company from an R&D business to a diagnostics company and oversaw development of the company's first products through to commercialisation and early revenue generation. She is a graduate of the Harvard Business School General Manager Program and the Australian Institute of Company Directors. Jenny is currently Chair of QUT Enterprise Holdings and a non-executive director on the boards of QUTbluebox and Creative Enterprise Australia.

Senior Management

Larry Glass BSc

Chief Science Officer

Larry joined Neuren in 2004 and was an Executive Director from May 2012. He has more than 30 years' experience in the life sciences industry, including clinical trials, basic and applied research, epidemiologic studies, diagnostics and pharmaceutical product development.

Jon Pilcher BSc (Hons), FCA

CFO Company Secretary

Jon joined Neuren in 2014 after 11 years in the leadership team at ASX-listed company Acrux Limited. During this time, Acrux underwent an IPO with ASX, the developed three FDA approved novel pharmaceutical products and negotiated a licensing deal with Eli Lily. Jon is a Chartered Accountant and holds a degree in Biotechnology. He is a non executive Director of BTC Health Limited.

Exhibit 12 Neuren Board Members and Senior Management (Neuren Annual Report)

Key Share Register Holdings

Key Share Register Holdings			
1	LANGLEY WALKER	18,267,119	18.2%
2	CAMERON RICHARD PTY LTD	5,689,992	5.7%
3	CITICORP NOMINEES PTY LIMITED	4,534,774	4.5%
4	DR TREVOR SCOTT	3,989,784	4.0%
5	STUART ANDREW PTY LTD	2,903,666	2.9%
6	LINWIERIK SUPER PTY LTD	2,515,000	2.5%
7	DR RICHARD SPENCER TREAGUS	2,068,680	2.1%
8	HSBC CUSTODY NOMINEES (AUSTRALIA) LIMITED	1,998,600	2.0%
9	SMITHLEY SUPER PTY LTD	1,950,000	1.9%
10	INVESTMENT CUSTODIAL SERVICES LIMITED	1,480,587	1.5%
11	MXB INVESTMENTS LLC	1,330,000	1.3%
12	DR ROBIN LANCE CONGREVE	991,637	1.0%
13	ROXTRUS PTY LIMITED	716,000	0.7%
14	J P MORGAN NOMINEES AUSTRALIA PTY LIMITED	693,964	0.7%
15	FIRST COLBYCO PTY LTD	624,649	0.6%
16	NAMARONG INVESTMENTS PTY LTD	555,556	0.6%
17	MR HE ZHAO	550,000	0.5%
18	SUNTANEOUS PTY LTD	446,195	0.4%
19	BNP PARIBAS NOMS PTY LTD	419,776	0.4%

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