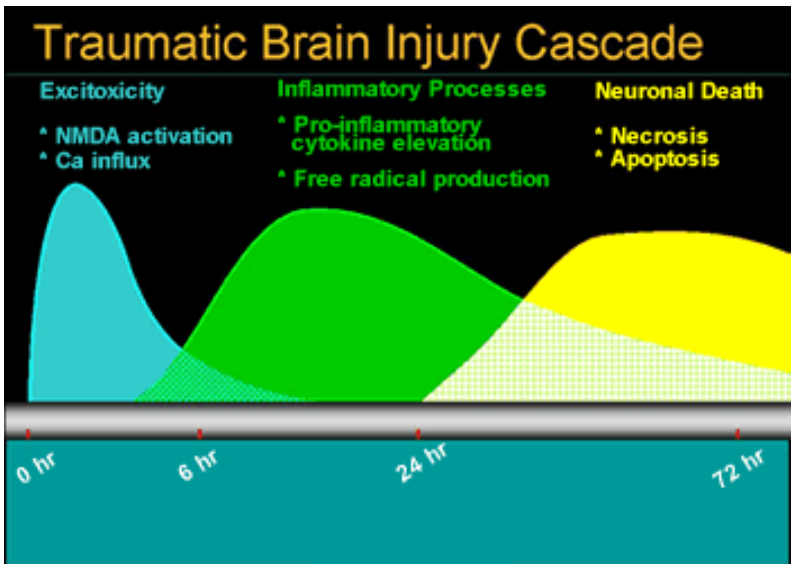
NNZ-2566 Development Overview
July 2009

NNZ-2566, Neuren's lead clinical stage asset, has a unique pedigree both in terms of its scientific origins and the development pathway that Neuren has planned. The Company believes — and this view has clearly been reinforced by scientific and financial support from the US Army — that NNZ-2566 is the most promising drug currently in development for acute traumatic brain injury (TBI). As a therapeutic target, TBI offers a special set of challenges but also a commensurate opportunity. At present, there are no drugs approved for the acute treatment of TBI despite numerous attempts and substantial expenditures. Neuren's strategy for development of NNZ-2566 is built on the lessons learned from previous TBI research programs as well as on recent scientific and medical findings that potentially change the landscape for development of a drug to treat this condition.

Scientific Background

NNZ-2566 is a synthetic analogue of a naturally occurring molecule produced by the brain in response to injury. That molecule, a small part of the insulin-like growth factor-1 (IGF-1) molecule, belongs to a group of molecules called neuropeptides. Neuropeptides are the subject of significant interest in research and drug development for a wide range of diseases and conditions from stroke to Alzheimer's disease. In contrast to most synthetic chemicals that focus on single, highly specific targets in well-known biological pathways, neuropeptides tend to exhibit a wider range of actions and to have fewer unwanted side effects. NNZ-2566 certainly offers these advantages.

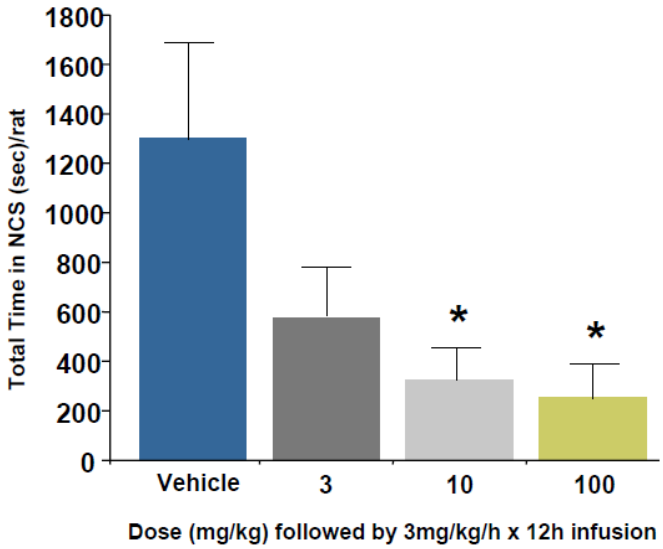
TBI results in a complex set of molecular, biochemical and cellular events — often referred to as a cascade — that begins immediately following injury. The figure below provides a somewhat simplified overview of the components comprising that cascade.



Almost immediately following injury, brain cells are exposed to excitotoxic forces that damage and destroy cells and interfere with their normal function. This leads to inflammatory processes which result in further functional disturbances and the subsequent death of brain cells. Most of the compounds previously evaluated in TBI target one or at most two components of this cascade. NNZ-2566 has been shown in animal models to reduce the magnitude and effects of all components. The Company believes that, to be effective, a drug for TBI must target all facets of the brain's response in the first few days following injury. In research conducted by the US Army, NNZ-2566 has been shown to reduce the level of expression of genes associated with inflammation, necrosis and apoptosis — key elements of the brain injury cascade — and to reduce the functional deficits induced by these phenomena. This ability to reduce the damage and consequences of brain injury is referred to as neuroprotection.

In addition to the molecular events associated with acute brain injury, a number of cellular events also occur. In particular, cells called microglia are activated. Microglia function as part of the brain's immune response to infection and injury and, among other functions, remove damaged or dying cells. However, activation of microglia following injury can result in an increased loss of brain cells as well as an increase in cytotoxic and inflammatory activity. In animal models NNZ-2566 significantly inhibits the activation of microglia, an action that is believed to be a key mechanism in reducing brain injury.

Besides the direct cellular and molecular effects of TBI, up to one-third or more of patients experience non-clinical or non-convulsive seizures (NCS) in the acute, post-injury period. NCS are associated with increased brain injury and long-lasting cognitive and neurological deficits. Identification of a therapy to prevent or reduce NCS is a major goal of the US Army's drug development strategy for TBI. In animal studies conducted by the US Army, NNZ-2566 has been shown to significantly reduce the number and duration of NCS following brain injury, even when NCS have emerged prior to treatment. The following chart shows the reduction in total NCS activity in a rat model of brain injury with increasing doses of NNZ-2566.



Neuren believes that the diverse and robust effects exhibited by NNZ-2566 following brain injury increases the likelihood that the drug will be effective. In addition, the range of effects observed has directly contributed to the logic and design of the upcoming Phase 2 clinical trial.

Clinical and Regulatory Strategy

As noted, there presently are no drugs approved for TBI. This and the serious and life-threatening nature of the condition supported the recent approval of NNZ-2566 for Fast Track designation by the US Food and Drug Administration (FDA) which can significantly accelerate the development and approval of a drug. Further, the lack of available therapies and the devastating nature of TBI mean that NNZ-2566 potentially can be approved following a single, pivotal trial with strongly positive results. Neuren's Phase 2 trial for NNZ-2566 takes this into consideration. The trial is large, complex and expensive and would only be possible with strong external support such as is being provided by the US Army. The trial is large enough and sufficiently comprehensive to support progressing to a pivotal trial following a positive outcome. It is designed to provide a definitive assessment of the ability of the drug to improve outcomes following TBI.

Most TBI trials have targeted severe TBI and, as a consequence, have relied on a small number of relatively insensitive endpoints focused on neurological outcomes. This approach makes it difficult to detect an effect of the drug unless that effect is very large. Because no drugs are

approved for this indication, neither the magnitude nor the nature of the effect has been pre-specified by FDA for Neuren's trial. A modest effect in any one of a number of endpoints built into the upcoming Phase 2 trial, with evidence of a clinically meaningful benefit in a functional outcome, is potentially approvable.

Neuren has elected to stratify the patient population 2:1 in favour of moderate (versus severe) TBI and to include a significant number of psychological, cognitive, biological and functional endpoints — many of which are more relevant to patients with moderate TBI and the consequences of TBI for these patients. We believe that this combination of patient selection and a range of endpoints with greater sensitivity will increase our ability to detect a small but clinically meaningful effect of the drug. This, of course, also is supported by the relatively large patient sample (260) made possible by the external funding from the US Army and by enrolment only at major trauma centres with investigators who are recognised experts in neurotrauma.

Economic Considerations

In the US alone, approximately 1 million TBI patients are either treated and released from the emergency department or admitted to the hospital each year. Of the approximately 300,000 admitted to the hospital, nearly half have mild or moderate TBI. With no drugs approved for this indication, TBI represents a significant market opportunity as well as a large unmet medical need. Based on conservative assumptions for reimbursement and modest assumptions on market penetration (25% for moderate to severe TBI patients and 20% for mild TBI patients admitted to hospital and 15% for patients treated and released from the emergency department), Neuren has estimated that total sales of NNZ-2566 in the first 10 years following approval would exceed US$2 billion in the US alone, with peak gross revenues of US$341 million. In our internal models, assuming drug registration success, this results in a net present value in excess of US$250 million.

With a successful result from the Phase 2 trial, we also believe that there would be multiple opportunities for a lucrative partnership with a larger pharmaceutical company. The funding committed by the US Army for development costs beyond the Phase 2 trial will both accelerate and reduce the risk and cost of completing clinical development of NNZ-2566 for Neuren or a partner and, as such, adds significant value to the program. Neuren has already invested in excess of US$10 million in the development of NNZ-2566 to this point. A relatively small additional investment by Neuren could result in a very substantial increase in shareholder value as well as a major contribution to the public health.