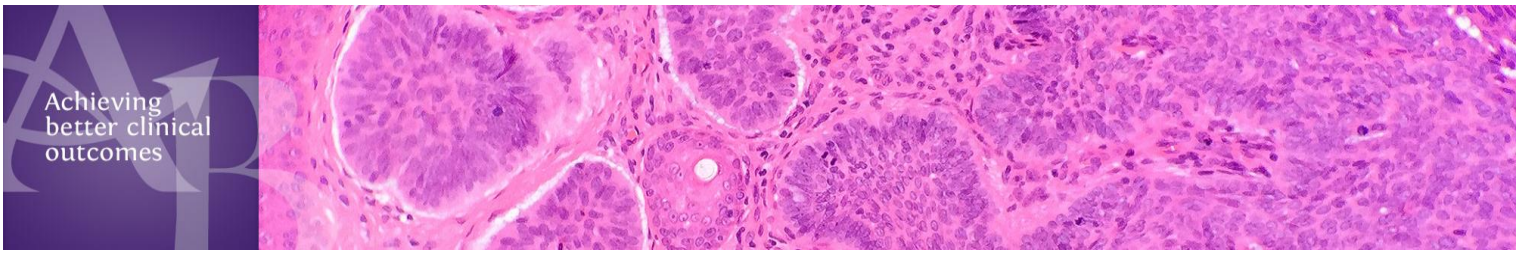



## Corporate Overview

Ascend is a cancer immunotherapy company developing medicines to treat primary, recurrent and metastatic cancers. Cancers are made up of many different cell types that can influence and regulate the local tumour microenvironment to promote tumour growth. Ascend's strategy focuses on targeting these key cells and altering the host microenvironment from one that supports tumour growth to one that results in cancer eradication.

The company applies a number of different technologies and approaches utilizing viral vectors, and affinity agents that can be conjugated to deliver biologicals and small molecule (immune modulators, kinase inhibitors or chemotherapeutics) payloads. We believe that applying a combination chemotherapy and immunotherapy (chemo-immunotherapy) approach has the potential to materially improve therapeutic outcomes.

## Pipeline

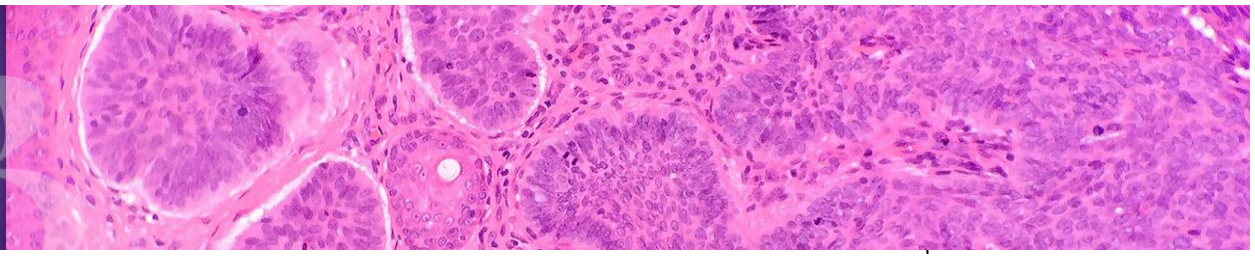
| Product | Composition | Indication | Preclinical | Phase 1 | Phase 2 | Phase 3 |
|---|---|---|---|---|---|---|
| ASN-002 | ASN-002 Monotherapy | Cutaneous BCL | | | | Completed |
| ASN-002 | ASN-002 Monotherapy | Nodular BCC | | | | H1, 2017 complete |
| | ASN-002 Chemo-immuno therapy | Head & Neck SCC | | | | H2 2017 start |
| | ASN-002 Chemo-immuno therapy | Recurrent OC | | | | H2, 2017/2018 start |
| | ASN-002 Chemo-immuno therapy | Nevoid BCC | | | | H2, 2017/2018 start |
| ASN-006 | Mannan-genetic adjuvant | Peritoneal Cancer | | | | H2, 2017 |
| ASN-008 | Replicon vector - RCD genes* | Refractory cancers | | | | on-going |
| | Replicon vector - Neoantigens | Refractory cancers | | | | on-going |

* Preclinical work performed in collaboration with Fox Chase Cancer Centre in Philadelphia, USA

## ASN-002

- Basal cell carcinoma (BCC) is the most common form of skin cancer, and the most common form of cancer in the US, Australia and Europe.
- Surgery is common for BCC, but surgery is not possible or optimal for an estimated 500,000 patients.
- Non-surgical alternatives are lacking for nodular BCC.
- ASN-002 is directly injected into the BCC tumour to stimulate an immune response which destroys cancer cells.
- Phase 2 clinical trial evaluating ASN002 in nBCC is currently on-going and in expected to be completed by 1H, 2017
- Pivotal studies are planned for nBCC and BCC nevoid syndrome in 2H, 2017/2018
- Additional follow-on indications evaluating ASN002 in Head and Neck Cutaneous Squamous Cell Carcinoma and Recurrent Ovarian Cancer are planned for 2017

ASCEND BIOPHARMACEUTICALS




## ASN-006 – Overview: Targeting Myeloid Derived Suppressor Cells

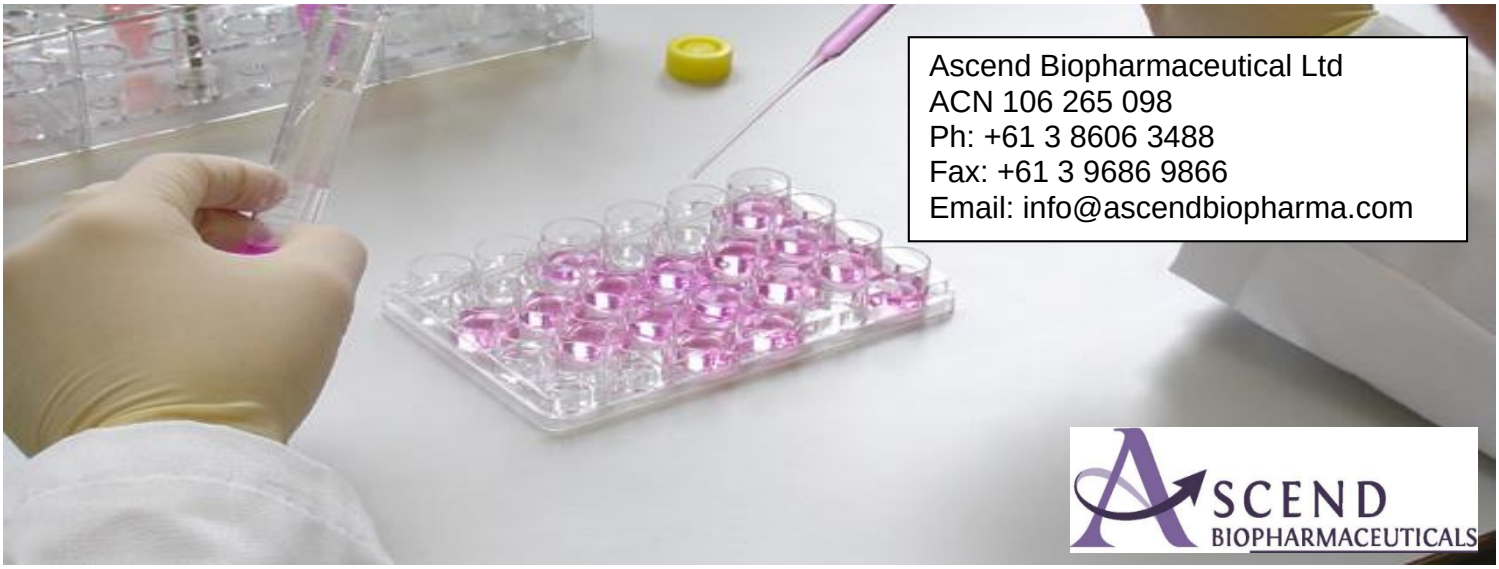
ASN-006 consists of a mannose polymer that can preferentially deliver RNA payloads into myeloid cells that can arise within the tumour microenvironment. The mannose polymer is conjugated to replicon RNA encoding Interleukin-12 and Interlukin-15. The replicon RNA is a self-amplifying RNA and enables persistent gene expression.

There is increasing evidence that many cancers are immunogenic and that the quantity, quality and location of the immune infiltrate within the tumour microenvironment correlates with beneficial clinical outcomes. There is also evidence that in many cancers there is a correlation between elevated myeloid derived suppressor cell numbers with worst patient prognosis. Preclinical studies are being performed to develop an optimized therapeutic candidate combining the selective delivery capability of the mannose polymer with the efficient gene expression of the kunjin replicon RNA. Studies are being performed to evaluate the therapeutic benefit of delivering Interleukin-12 and Interleukin-15 replicon RNA into myeloid cells within the tumor microenvironment.

## ASN-008 – Induction of immunogenic and regulated cell death pathways

ASN-008 is designed for intralesional delivery into cancers. Earlier work with ASN-002 plus a number of chemotherapeutic agents demonstrated that multiple cell death pathways can be evoked through the combination use of a viral DNA/RNA, pro-cell death cytokine and certain cytotoxic small molecule drugs. This chemo-immunotherapy combination resulted in significant lymphocytic infiltration and abscopal regression. Early gene expression also showed intralesional induction of chemokines that recruited various dendritic cell population including cross-presenting dendritic cells into the tumor microenvironment. These combination chemo-immunotherapy also led to enhanced regulated cell death. Recently it has been reported that cell death that is triggered via the RIPK3 pathway can result in immunogenic cell death.

The current lead optimization of ASN-008 builds upon earlier ASN-002 chemotherapy combination studies. The aim of this program is to develop a purely genetic approach of triggering a broader range of cell death pathways. ASN-008 is based upon the SRIP platform.



Ascend Biopharmaceutical Ltd
ACN 106 265 098
Ph: +61 3 8606 3488
Fax: +61 3 9686 9866
Email: info@ascendbiopharma.com

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