CELLMID

**ASX ANNOUNCEMENT**

# 4th MIDKINE SYMPOSIUM OUTLINES ADAPTIVE CLINICAL PATH FOR MIDKINE

- **Clinicians and scientists from nine countries presented data on midkine biology and its role in disease**
- **Novel findings confirmed clinical strategy for subsidiaries Lyramid and Kinera**
- **Symposium expected to contribute to adaptive clinical development path**

**SYDNEY, Wednesday, 11 MAY 2016: Cellmid Limited (ASX: CDY)** is pleased to report on its **4th Midkine Symposium**, which resulted in presentations of new data by twenty scientists from nine countries on the therapeutic potential of midkine (MK) and MK inhibitors.

Importantly, consensus was reached during the Symposium that an adaptive pathway for clinical validation may be available for MK antibodies with the clear understanding of MK's role in disease mechanisms, collaborations with academia and major pharma, and via the development of surrogate markers for efficacy.

The Symposium, a preeminent biennial meeting on MK previously held in Sydney in 2010, Istanbul in 2012 and Kyoto in 2014, was held in Budapest between 28 and 30 April 2016. Several of Cellmid's existing scientific and commercial collaborators attended the Symposium in addition to those new to the meeting, strengthening the Company's position and intellectual reach within the MK scientific community. The presentations and discussions held during the Symposium were made under confidentiality.

Significantly, further evidence emerged during the Symposium that MK is an important molecule in inter-organ signalling in a number of diseases. This means that targeting MK is one of the few novel approaches in the management of complex metabolic and cardiovascular diseases including chronic kidney disease and vascular calcification.

Studies on MK biology, mechanism of action and clinical utility were also presented, with promising data on the therapeutic potential of Cellmid's own drug candidates. Substantial unpublished and commercially sensitive confidential data were shared during the Symposium. It is anticipated that some of the work may be published after going through a peer review process, however, PowerPoint presentations and posters forming part of the scientific component of the conference will not be made public at this stage.

Some of the key messages of the meeting included the following:

- Cellmid's C and N terminal binding MK antibodies were assessed in a mouse model of myocarditis for their ability to attenuate disease by Dr Ulrich Grabmaier from Ludwig Maximilians University in Munich. N terminal binding MK antibodies showed marked efficacy in the model not only pointing to a novel potential clinical application but also demonstrating the difference in efficacy between the two MK antibodies. This important information on MK biology is instructive for future studies and further collaboration is expected with Dr Grabmaier's Munich based group.

- Two of Cellmid's MK antibodies have shown tumour supressing ability in glioblastoma cell lines resistant to cannabinoid treatment in studies conducted at the labs of Professor Guillermo Velasco of Complutense University in Madrid. Further work is

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ongoing on Cellmid's C and N terminal binding antibodies in animal models of the disease to assess efficacy.

- Cellmid's N terminal binding MK antibody enhanced bone fracture healing in ovariectomised mice *in vivo* in studies conducted by Dr Astrid Liedert from the University of Ulm in Germany. The model mimics the biology of osteoporosis in post-menopausal women and representative of the delayed bone healing that occurs in this population. Further *in vivo* work is expected to uncover the mechanism by which MK contributes to delayed bone healing.

- New insights were presented into the structure of MK's binding with glycosaminoglycans (GAGs) and how it may effect biological function. These findings were presented by Professor Xu Wang from Arizona State University and Dr Pedro Nieto of the University of Seville, Spain. Further collaboration is expected with both groups to ascertain the binding characteristics of MK in biological systems.

- MK human diagnostic work by three separate groups included further understanding on urinary MK in prostate and bladder cancers, as well as in patients with acute and chronic kidney disease. Cellmid's clinical adviser, Dr Victoria Campbell presented early evidence that MK may be an important marker of chronic kidney disease. This work is ongoing and involves several clinical centres in Australia.

- MK clinical development plans with two and five years' time horizons were discussed with key outcomes around uncovering mechanism of action in disease states, additional academic and commercial collaborations, engaging with key opinion leaders overseas and surrogate markers of efficacy identified as key milestones in an adaptive plan, in addition to the traditional drug development milestones of manufacture, pharmacodynamics, pharmacokinetics, safety and efficacy.

End

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**Cellmid Limited (ASX: CDY)**
Cellmid is an Australian life sciences accelerator with lead programs in multiple disease indications. The Company, through its wholly owned subsidiaries, is developing innovative novel therapies and diagnostic tests for fibrotic diseases, cancer, ischemic diseases of the heart and hair loss. Cellmid holds the largest and most comprehensive portfolio of intellectual property relating to the novel targets midkine (MK) and FGF5 globally. For further information, please see www.cellmid.com.au and www.evolisproducts.com.au.

**Midkine (MK)**
MK is a growth factor that is highly expressed during embryonic development. Midkine modulates many important biological interactions such as cell growth, cell migration and cellular adherence. Midkine is thought to be a non-redundant, key inter-organ signalling molecule with functions relevant to cancer, inflammation, autoimmunity, ischemia, nerve growth/repair and wound healing.