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ASIA EDITION

Cellmid gets positive data in midkine study

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Singapore:Ã?Â Cellmid has completed its first in-life diabetic nephropathy study with the company's anti-midkine antibodies (MKAb) in a mouse model of the disease. Two of Cellmid's proprietary MK-Ab's were tested. Both antibodies reduced kidney damage significantly, as assessed by functional and histological analysis, with kidney structure largely preserved in the treated animals.

This study provides important new information, as it is the first time the company has used its own MK-Ab's in a therapeutic setting in a kidney disease model.

Renal histological assessment showed that glomerular sclerosis was reduced from 48 percent in untreated animals to below 20 percent in both MK-Ab treated groups ($p < 0.01$). Interstitial volume was also significantly reduced, from 35 percent in untreated animals to 12 percent in both antibody groups ($p < 0.01$). MK-Ab treatment also maintained tubular cell height; untreated animals had mean cell heights below 2Ã?Â½Ã?Â¼m, compared to 4Ã?Â½Ã?Â¼m for treated animals ($p < 0.05$).

Kidney function was also preserved, with MK-Ab treated animals showing reduced protein leakage into the urine compared to untreated controls. Protein casts in the kidney, indicating damage, were also significantly reduced in antibody treated animals. Importantly, the MK-Ab treated animals showed healthy weight gain and reduced mortality compared to untreated controls; only 6.3 percent of treated animals died before the end of the study, compared to 25 percent of the untreated animals.

Midkine's role in kidney disease has been extensively studied in the past and is the subject of a dozen peer-reviewed publications. These studies show that MK is a key driver of inflammation and damage in a variety of kidney disease and injury settings.

The current study using Cellmid's MK-Ab's was conducted by scientists at the Centre for Transplantation and Renal Research (CTRR), based at the Westmead Millennium Institute and University of Sydney, Westmead Hospital, using an Adriamycin (AN)-induced mouse model of nephropathy. In this model, a single AN injection leads to kidney damage reminiscent of that

seen in human diabetic nephropathy.

Diabetic nephropathy is the leading cause of chronic kidney disease globally. It is also one of the most significant long-term complications in terms of morbidity and mortality for patients with diabetes. In the US alone, diabetes affects 26 million people, and the US Centre for Disease Control (CDC) estimates that as many as one in three adults could have diabetes by 2050 if current trends continue.

Currently, diabetic nephropathy is managed by keeping glucose levels under control, however many of the patients develop end stage renal disease (ESRD). It is estimated that 30-40 percent of all ESRD is caused by diabetic nephropathy.

ESRD requires the traumatic and costly interventions of kidney dialysis or transplant. A treatment that slowed or halted the progression of diabetic nephropathy into full-blown ESRD would have enormous benefits for the quality of life of diabetes sufferers in addition to reducing the massive costs associated with the treatment of ESRD.

A 2010 report by Kidney Health Australia estimated that dialysis costs between $53,000 and $79,000 per patient per year. The same report costed kidney transplants at $81,000, with nearly $12,000 per patient per year in ongoing costs.