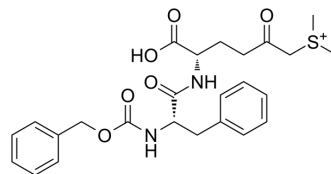


NTU281

Cat. No.:	HY-100446
CAS No.:	815619-12-6
Molecular Formula:	C ₂₅ H ₃₁ N ₂ O ₆ S ⁺
Molecular Weight:	487.59
Target:	Glutaminase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	NTU281 is a potent transglutaminase-2 inhibitor. NTU281 can reduce the increases in serum creatinine and albuminuria in diabetic rats. NTU281 can also reduce glomerular collagen I accumulation, Hic-5 and α -SMA expression, and apoptosis. NTU281 can be used for researching glomerulosclerosis caused by diabetes ^{[1][2]} .																
In Vivo	<p>NTU281 (2.5 μl/h of 50 mM; cannulate to deliver into kidneys) reduces glomerular collagen I overexpression as well as the increases in glomerular Hic-5 and α-SMA expression; also decreases serum creatinine, albuminuria, glomerulosclerosis and tubulointerstitial scarring in diabetic rats^{[1][2]}.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>Male Wistar Han rats [subjected to right uninephrectomy, then induced hyperglycemia by tail vein injection of streptozotocin (35 mg/kg in citrate buffer pH 4)]^[1]</td> </tr> <tr> <td>Dosage:</td> <td>2.5 μl/h of 50 mM</td> </tr> <tr> <td>Administration:</td> <td>Cannulated to deliver into kidneys</td> </tr> <tr> <td>Result:</td> <td>Reduced glomerular collagen I overexpression by ~50%; reduced the increases in glomerular Hic-5 expression; reduced diabetic nephropathy-induced α-SMA expression.</td> </tr> </table> <table border="1"> <tr> <td>Animal Model:</td> <td>Male Wistar Han rats [subjected to right uninephrectomy, then induced hyperglycemia by tail vein injection of streptozotocin (35 mg/kg in citrate buffer pH 4)]^[2]</td> </tr> <tr> <td>Dosage:</td> <td>Various concentration</td> </tr> <tr> <td>Administration:</td> <td>Cannulated to deliver into kidneys</td> </tr> <tr> <td>Result:</td> <td>Significantly reduced the increases in serum creatinine (-68%) and albuminuria (-80%) in diabetic rats during eight-month experimental period; reduced in glomerulosclerosis (-76.6%) and tubulointerstitial scarring (-68.2%) as a result of lowered accumulation of collagen I, III and IV; and reduced numbers of myofibroblasts present.</td> </tr> </table>	Animal Model:	Male Wistar Han rats [subjected to right uninephrectomy, then induced hyperglycemia by tail vein injection of streptozotocin (35 mg/kg in citrate buffer pH 4)] ^[1]	Dosage:	2.5 μ l/h of 50 mM	Administration:	Cannulated to deliver into kidneys	Result:	Reduced glomerular collagen I overexpression by ~50%; reduced the increases in glomerular Hic-5 expression; reduced diabetic nephropathy-induced α -SMA expression.	Animal Model:	Male Wistar Han rats [subjected to right uninephrectomy, then induced hyperglycemia by tail vein injection of streptozotocin (35 mg/kg in citrate buffer pH 4)] ^[2]	Dosage:	Various concentration	Administration:	Cannulated to deliver into kidneys	Result:	Significantly reduced the increases in serum creatinine (-68%) and albuminuria (-80%) in diabetic rats during eight-month experimental period; reduced in glomerulosclerosis (-76.6%) and tubulointerstitial scarring (-68.2%) as a result of lowered accumulation of collagen I, III and IV; and reduced numbers of myofibroblasts present.
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REFERENCES

[1]. Hornigold N, et al. Inhibition of collagen I accumulation reduces glomerulosclerosis by a Hic-5-dependent mechanism in experimental diabetic nephropathy. *Lab Invest.* 2013 May;93(5):553-65.

[2]. Huang L, et al. Do changes in transglutaminase activity alter latent transforming growth factor beta activation in experimental diabetic nephropathy? *Nephrol Dial Transplant.* 2010 Dec;25(12):3897-910.

Caution: Product has not been fully validated for medical applications. For research use only.

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