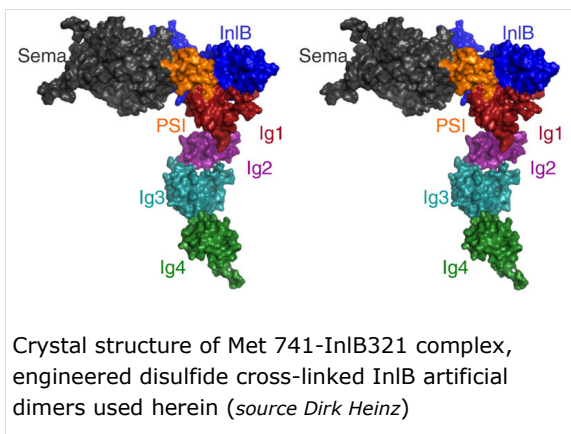


c-Met Agonists: Use for the Treatment of Tissue Damages

Reference Number: TO 02-00263

Challenge

Approximately eight million surgical procedures are performed annually only in the U.S. to treat tissue damage resulting from accidents, birth defects, hereditary disorders, and other diseases. As traditional tissue repair procedures are inefficient, potentially painful to the patient, and costly to perform, there is a continued demand for alternative efficient approaches to treat the damage of internal organs or skin.



Technology

The tyrosine kinase Met, the product of the c-met proto-oncogene and the receptor for hepatocyte growth factor/scatter factor (HGF/SF), mediates signals critical for cell survival and migration. The human pathogen *Listeria monocytogenes* exploits Met signaling for invasion of host cells via its surface

protein internalin B (InlB). The present technology discloses an isolated recombinant dimer derived from internalin B that binds to c-Met with very high specificity and activates the c-Met dependent signalling pathway. The dimer acts as an agonist of c-Met signalling and could support the development of a pharmaceutical composition useful for wound healing or tissue regeneration after tissue damage, in particular, skin or mucosal tissue damages but also damages or injuries of liver, lung or heart muscle, which may occur e.g. in the course of progressing diabetes. Additionally, the dimer could be developed as a proliferating agent stimulating or enhancing cell proliferation in particular for tissue engineering techniques.

Commercial Opportunity

The technology is offered for co-development of various tissue regeneration approaches or in-licensing.

Developmental Status

The status of the development is still research. The scattering potency and the induction of cell proliferation could be shown in different cell culture assay formats.

Patent Situation

European application filed in June 2008 (EP08011562.9), international application (PCT) filed June 2009.

Further Reading

Nieman et al. J. Mol. Biol. (2008) 377: 489-500.

Nieman et al. Cell (2007) Jul 27;130(2): 235-46

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