Neurolysin degrades neurotensin

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.
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Literature references

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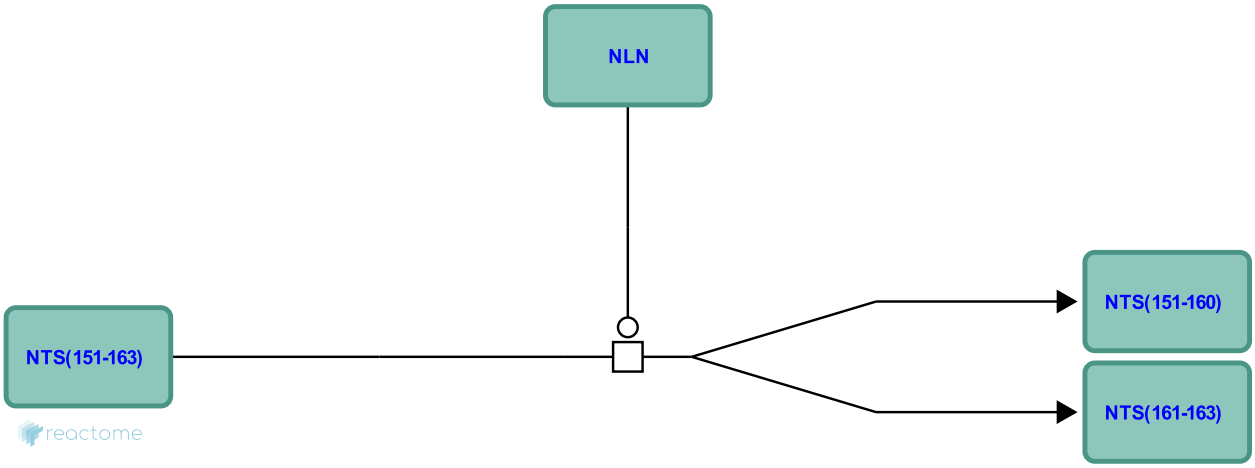
Stable identifier: R-HSA-8940959

Type: transition

Compartments: extracellular region



Neurolysin (NLN, EC 3.4.24.16) is a member of the thermolysin-like mammalian zinc endopeptidase family (Dauch et al. 1995). It is maximally active at neutral pH and responsible for hydrolytic processing of bioactive peptides in the extracellular environment (Shrimpton et al. 2002). It cleaves 3 residues from the C-teminal end of Neurotensin (Dauch et al. 1995). Other endogenous substrates of NLN include bradykinin, angiotensins I and II, substance P, hemopressin, dynorphin A(1–8), metorphamide, and somatostatin (Wangler et al. 2016). The functional significance of NLN is poorly understood (Checler 2014). In vivo studies have linked it to neurotensin-dependent nociception (Vincent et al. 1997), bradykinin-mediated hypotension, microvascular permeability and hyperalgesia (Gomez et al. 2011), and pathogenesis of stroke (Rashid et al. 2014).

Literature references

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Editions

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