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    <p><b>Structural and Functional Characterization of Conotoxins from
Conus achatinus Targeting NMDAR.</b></p>
    <p>Mar Drugs. 2020 Feb 26;18(3):</p>
    <p>Authors: Liu X, Yao G, Wang K, Liu Y, Wan X, Jiang H</p>
    <p>Abstract<br/>
    Conotoxin-Ac1 and its variant conotoxin-Ac1-06P, were isolated from
the venom duct of Conus achatinus, a fish-hunting cone snail species
collected in the Sea of Hainan, China. Conotoxin-Ac1 is linear peptide that
contain 15 amino acids. In the present study, we synthesized and
structurally and functionally characterized conotoxin-Ac1 as well as 19
variants. Electrophysiological results showed that conotoxin-Ac1 inhibited
N-methyl-D-aspartate receptor subunit 2B (NR2B) with an IC50 of 8.22 ± 0.022
µM. Further structure-activity studies of conotoxin-Ac demonstrated that
polar amino acid residues were important for modulating its active, and the
replacement of N1, O9, E10, and S12 by Ala resulted in a significant
decrease in potency to NR2B. °Furthermore, conotoxin-Ac1 and conotoxin-Ac1-
06P were tested in hot-plate and tail-flick assays to measure the potential
analgesic activity to an acute thermal stimulus in a dose-dependent manner.
Subsequently, the analgesic activity of conotoxin-Ac1 mutants was analyzed

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by the hot-plate method. The results show that N1, Y2, Y3, E10, N11, S12, and T15 play an important role in the analgesic activity of conotoxin-Ac1. N1 and S12 have significant effects on conotoxin-Ac1 in inhibiting NR2B and analgesic activity. In conclusion, we have discovered that conotoxin-Ac1 is an inhibitor of NMDAR and displays antinociceptive activity.

</p><p>PMID: 32111068 [PubMed - in process]</p>

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<author> Liu X, Yao G, Wang K, Liu Y, Wan X, Jiang H</author>

<category>Mar Drugs</category>

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<title>Dimerization of α -conotoxins as a strategy to enhance the inhibition of the human $\alpha 7$ and $\alpha 9\alpha 10$ nicotinic acetylcholine receptors.</title>

<link>https://www.ncbi.nlm.nih.gov/pubmed/32101438?dopt=Abstract</link>

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<p>Dimerization of α -conotoxins as a strategy to enhance the inhibition of the human $\alpha 7$ and $\alpha 9\alpha 10$ nicotinic acetylcholine receptors.</p>

<p>J Med Chem. 2020 Feb 26;:</p>

<p>Authors: Liang J, Tae HS, Xu X, Jiang T, Adams DJ, Yu R</p>

<p>Abstract

The affinity of α -conotoxins, a class of nicotinic acetylcholine receptor (nAChR) peptide inhibitors, can be enhanced by dendrimerization. It has been hypothesized that this improvement arose from simultaneous binding of the α -conotoxins to several spatially adjacent sites. We here engineered several α -conotoxin dimers using a linker length compatible between neighboring binding sites on the same receptor. Remarkably, the dimer of α -conotoxin PeIA compared to the monomer displayed an increase in potency by 11-fold ($IC_{50} = 1.9$ nM) for the human $\alpha 9\alpha 10$ nAChR. The dimerization of α -conotoxin, RgIA# resulted in a dual inhibitor that targets both $\alpha 9\alpha 10$ and $\alpha 7$ nAChR subtypes with an $IC_{50} = \sim 50$ nM. The RgIA# dimer is therapeutically interesting because it is the first dual inhibitor that potently and selectively inhibits these two nAChR subtypes, which are both involved in the aetiology of several cancers. We propose that the dimerization of α -conotoxins is a simpler and efficient alternative strategy to dendrimers for enhancing the activity of α -conotoxins.

</p><p>PMID: 32101438 [PubMed - as supplied by publisher]</p>

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<author> Liang J, Tae HS, Xu X, Jiang T, Adams DJ, Yu R</author>

<category>J Med Chem</category>

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    <p><b>11-Keto-β-Boswellic Acid Attenuates Glutamate Release and
  Kainic Acid-Induced Excitotoxicity in the Rat Hippocampus.</b></p>
    <p>Planta Med. 2020 Feb 25;:</p>
    <p>Authors: Lu CW, Lin TY, Wang SJ</p>
    <p>Abstract<br/>
    Excessive glutamate concentration induces neuronal death in acute
  brain injuries and chronic neurodegenerative diseases. Natural compounds
  from medicinal plants have attracted considerable attention for their use in
  the prevention and treatment of neurological disorders. 11-Keto-β-boswellic
  acid, a triterpenoid found in the medicinal plant Boswellia serrata, has
  neuroprotective potential. The present study investigated the effect of 11-
  keto-β-boswellic acid on glutamate release in vitro and kainic acid-induced
  glutamate excitotoxicity in vivo in the rat hippocampus. In rat hippocampal
  nerve terminals (synaptosomes), 11-keto-β-boswellic acid dose-dependently
  inhibited 4-aminopyridine-stimulated glutamate release. This effect was
  dependent on extracellular calcium, persisted in the presence of the
  glutamate transporter inhibitor DL-threo-β-benzyloxyaspartate, and was
  blocked by the vesicular transporter inhibitor bafilomycin A1. In addition,
  11-keto-β-boswellic acid reduced the 4-aminopyridine-induced increase in
  intrasynaptosomal Ca2+ levels. The N- and P/Q-type channel blocker ω-
  conotoxin MVIIC and the protein kinase A inhibitor H89 significantly
  suppressed the 11-keto-β-boswellic acid-mediated inhibition of glutamate
  release, whereas the intracellular Ca2+-releasing inhibitors dantrolene,
  CGP37157, and xestospongin C, mitogen-activated protein kinase inhibitor
  PD98059, as well as protein kinase C inhibitor calphostin C had no effect.
  In a rat model of excitotoxicity induced by intraperitoneal kainic acid
  injection (15 mg/kg), intraperitoneal 11-keto-β-boswellic acid
  administration (10 or 50 mg/kg) 30 min before kainic acid injection
  considerably ameliorated kainic acid-induced glutamate concentration
  elevation and CA3 neuronal death. These data suggested that 11-keto-β-
  boswellic acid inhibits glutamate release from the rat hippocampal
  synaptosomes by suppressing N- and P/Q-type Ca2+ channels and protein kinase
  A activity, as well as exerts protective effects against kainic acid-induced
  excitotoxicity in vivo.<br/>
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  <author> Lu CW, Lin TY, Wang SJ</author>

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    <p><b>Synthesis of disulfide surrogate peptides incorporating large-
span surrogate bridges through a native chemical ligation-assisted
diaminodiacid strategy.</b></p>
    <p>Angew Chem Int Ed Engl. 2020 Feb 14;:</p>
    <p>Authors: Qu Q, Gao S, Wu F, Zhang MG, Li Y, Zhang LH, Tian C,
Zheng JS, Liu L</p>
    <p>Abstract<br/>
    The use of synthetic bridges as surrogates for disulfide bonds has
emerged as a practical strategy to obviate the poor stability of some
disulfide-containing peptides. However, peptides incorporating large-span
synthetic bridges are still beyond the reach of existing methods. Here we
report a native chemical ligation (NCL)-assisted diaminodiacid (DADA)
strategy that enables the robust generation of disulfide surrogate peptides
incorporating surrogate bridges up to 50 amino acids in length. This
strategy provides access, for this first time, to some highly desirable but
otherwise impossible-to-obtain disulfide surrogates of bioactive peptides,
including  $\mu$ -conotoxin KIIIA, a potent inhibitor of voltage-gated sodium
channels. The bioactivities and structures of the synthetic disulfide
surrogates were verified by voltage clamp assays, NMR, and X-ray
crystallography; and stability studies established that the disulfide
replacements effectively overcame the problems of disulfide reduction and
scrambling that often plague these pharmacologically important peptides. By
expanding access to structurally more diverse disulfide surrogate peptides,
the NCL-assisted DADA strategy is expected to facilitate the development of
peptide-based diagnostic and therapeutic agents.<br/>
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  <title>Cone snail analogs of the pituitary hormones oxytocin/vasopressin
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identification of conopressins and conophysins.</title>
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  <p><b>Cone snail analogs of the pituitary hormones
oxytocin/vasopressin and their carrier protein neurophysin. Proteomic and
transcriptomic identification of conopressins and conophysins.</b></p>
  <p>Biochim Biophys Acta Proteins Proteom. 2020
May;1868(5):140391</p>
  <p>Authors: Kumar S, Vijayasathathy M, Venkatesha MA, Sunita P,
Balaram P</p>
  <p>Abstract<br/>
    Transcriptomic analysis of cone snail venom duct tissue has
permitted the identification of diverse conopressin/conophysin precursor
sequences from seven distinct Conus species. Multiple precursor isoforms are
present in C.monile, C.lividus and C.loroisii. Aqueous extracts of the venom
duct tissue from C.monile yield a band, at ~ 15-20 kDa on SDS-PAGE. In-gel
trypsin digestion, followed by mass spectrometry establishes the presence of
two distinct conopressin/conophysin isoforms that differ at position 8 in
the predicted conopressin nonapeptide sequence. Mass spectrometric analysis
of aqueous extracts revealed the presence of four conopressin related
peptides, whose sequences could be deduced from MS/MS fragmentation
patterns. The four sequences determined in this study are CFIRNCPKG*,
CFIRNCPKG*, CFIRNCPKG* and CFIRNCPKG* (* indicates amide), which were further
confirmed by comparison with chemically synthesized peptides. A conophysin
with a mass of 9419.7 Da was also detected, corresponding to one of the
isoforms revealed by the transcriptome data. Complete conservation of
fourteen Cys residues and the key residues involved in peptide hormone
binding is established by comparison of conophysin sequences, with the
crystallographically characterized sequence of bovine neurophysin, in
complex with vasopressin. A survey of available sequences for
oxytocin/vasopressin peptides in both vertebrates and invertebrates
establishes the conopressins as a distinct group in this family. C-terminal
amidated, truncated conopressin analogs may arise by alternate post-
translational processing.<br/>
  </p><p>PMID: 32058072 [PubMed - in process]</p>
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  <author>Kumar S, Vijayasathathy M, Venkatesha MA, Sunita P, Balaram
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  <title>The inhibitory effect of Ph $\alpha$ 1 $\beta$  toxin on diabetic neuropathic pain

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 <p>The inhibitory effect of Ph α 1 β toxin on diabetic neuropathic pain involves the CXCR4 chemokine receptor.</p>
 <p>Pharmacol Rep. 2020 Feb;72(1):47-54</p>
 <p>Authors: da Silva Junior CA, de Castro Junior CJ, Pereira EMR, Binda NS, da Silva JF, do Nascimento Cordeiro M, Diniz DM, Cecilia FS, Ferreira J, Gomez MV</p>
 <p>Abstract

 BACKGROUND: Diabetic neuropathy is a common cause of painful diabetic neuropathy (PDN). C-X-C chemokine receptor type 4 (CXCR4) expression is increased in peripheral nerve samples from diabetes patients, suggesting a role for CXCR4 in PDN. Therefore, we evaluated the effects of Ph α 1 β , ω -conotoxin MVIIA, and AMD3100 in a model of streptozotocin (STZ)-induced PDN in rodents and naïve model of rats with the activation of the CXCR4/stromal cell-derived factor 1 (SDF-1) signal.

 METHODS: Diabetic neuropathy was induced by intraperitoneal (ip) injection of STZ in Wistar rats. Naïve rats were intrathecally injected with SDF-1 to test the CXCR4/SDF-1 signal. The effects of Ph α 1 β intrathecal (it), ω -conotoxin MVIIA intrathecal (it), and AMD3100 intraperitoneal (ip) on rat hypersensitivity, IL-6, and the intracellular calcium [Ca²⁺]_i content of diabetic synaptosomes were studied.

 RESULTS: The drugs reduced the hypersensitivity in diabetic rats. SDF-1 (1.0 μ g/it) administration in naïve rats induced hypersensitivity. Ph α 1 β (100 pmol/it) or AMD3100 (2.5 μ g/ip) reduced this hypersensitivity after 2 h treatments, while ω -conotoxin MVIIA did not have an effect. IL-6 and [Ca²⁺]_i content increased in the spinal cord synaptosomes in diabetic rats. The drug treatments reduced IL-6 and the calcium influx in diabetic synaptosomes.

 CONCLUSIONS: Ph α 1 β , ω -conotoxin MVIIA, and AMD3100, after 2 h of treatment of STZ-induced PDN, reduced hypersensitivity in diabetic rats. In naïve rats with CXCR4/SDF-1 activation, the induced hypersensitivity decreased after 2 h treatments with Ph α 1 β or AMD-3100, while ω -conotoxin MVIIA did not affect. The inhibitory effects of Ph α 1 β on PDN may involve voltage-dependent calcium channels.

 </p><p>PMID: 32016848 [PubMed - in process]</p>
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 <author> da Silva Junior CA, de Castro Junior CJ, Pereira EMR, Binda NS, da Silva JF, do Nascimento Cordeiro M, Diniz DM, Cecilia FS, Ferreira J, Gomez MV</author>
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 <title>Structure and Activity Studies of Disulfide-Deficient Analogues of α 0-Conotoxin GeXIVA.</title>

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  <p><b>Structure and Activity Studies of Disulfide-Deficient
Analogues of  $\alpha$ O-Conotoxin GeXIVA.</b></p>
  <p>J Med Chem. 2020 Feb 27;63(4):1564-1575</p>
  <p>Authors: Xu P, Kaas Q, Wu Y, Zhu X, Li X, Harvey PJ, Zhangsun D,
  Craik DJ, Luo S</p>
  <p>Abstract<br/>
   $\alpha$ O-conotoxin GeXIVA from Conus generalis is a potent antagonist of the
   $\alpha$ 9 $\alpha$ 10 nAChR and analgesic in animal models of pain. This peptide has two
  disulfide bond cross-links, and the bead and ribbon isomers have similar
  inhibitory activity against  $\alpha$ 9 $\alpha$ 10 nAChRs. We synthesized 12 disulfide-
  deficient analogues of bead GeXIVA, and all remained potent inhibitors of
   $\alpha$ 9 $\alpha$ 10 nAChR. The most potent disulfide-deficient analogue displayed IC50
  values of 6 and 33 nM at rat and human  $\alpha$ 9 $\alpha$ 10 nAChRs, respectively,
  representing less than a 2-fold increase compared with bead GeXIVA. The
  disulfide-deficient analogs and parent peptides also do not have a well-
  defined structure according to NMR spectroscopy. Molecular simulations
  suggest that the disulfide bonds and termini of GeXIVA do not establish
  stable interactions with the receptor. Overall, this study proposes that the
  structure of the analgesic peptide GeXIVA could be simplified through
  disulfide bond deletions and potentially termini truncations.<br/>
  </p><p>PMID: 31986036 [PubMed - in process]</p>
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  <author> Xu P, Kaas Q, Wu Y, Zhu X, Li X, Harvey PJ, Zhangsun D, Craik
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    <p><b>Expression of  $\alpha$ 3 $\beta$ 2 $\beta$ 4 nicotinic acetylcholine receptors by rat

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adrenal chromaffin cells determined using novel conopeptide antagonists.

J Neurochem. 2020 Jan 22;:

Authors: Hone AJ, Rueda-Ruzafa L, Gordon TJ, Gajewiak J, Christensen S, Dyhring T, Albillos A, McIntosh JM

Abstract

Adrenal chromaffin cells release neurotransmitters in response to stress and may be involved in conditions such as post-traumatic stress and anxiety disorders. Neurotransmitter release is triggered, in part, by activation of nicotinic acetylcholine receptors (nAChRs). However, despite decades of use as a model system for studying exocytosis, the nAChR subtypes involved have not been pharmacologically identified. Quantitative real-time PCR of rat adrenal medulla revealed an abundance of mRNAs for $\alpha 3$, $\alpha 7$, $\beta 2$, and $\beta 4$ subunits. Whole-cell patch-clamp electrophysiology of chromaffin cells and subtype-selective ligands were used to probe for nAChRs derived from the mRNAs found in adrenal medulla. A novel conopeptide antagonist, PeIA-5469, was created that is highly selective for $\alpha 3\beta 2$ over other nAChR subtypes heterologously expressed in *Xenopus laevis* oocytes. Experiments using PeIA-5469 and the $\alpha 3\beta 4$ -selective α -conotoxin TxID revealed that rat adrenal medulla contain two populations of chromaffin cells that express either $\alpha 3\beta 4$ nAChRs alone or $\alpha 3\beta 4$ together with the $\alpha 3\beta 2\beta 4$ subtype. Conclusions were derived from observations that acetylcholine-gated currents in some cells were sensitive to inhibition by PeIA-5469 and TxID, while in other cells, currents were sensitive only to TxID. Expression of functional $\alpha 7$ nAChRs was determined using three $\alpha 7$ -selective ligands: the agonist PNU282987, the positive allosteric modulator PNU120596, and the antagonist α -conotoxin [V11L,V16D]ArIB. The results of these studies identify for the first time the expression of $\alpha 3\beta 2\beta 4$ nAChRs as well as functional $\alpha 7$ nAChRs by rat adrenal chromaffin cells.

PMID: 31967330 [PubMed - as supplied by publisher]

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author> Hone AJ, Rueda-Ruzafa L, Gordon TJ, Gajewiak J, Christensen S, Dyhring T, Albillos A, McIntosh JM

category>J Neurochem

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<title>Differential Expression of Nicotine Acetylcholine Receptors Associates with Human Breast Cancer and Mediates Antitumor Activity of α -Conotoxin GeXIVA.</title>

<link><https://www.ncbi.nlm.nih.gov/pubmed/31963558?dopt=Abstract></link>

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<p>Differential Expression of Nicotine Acetylcholine Receptors Associates with Human Breast Cancer and Mediates Antitumor Activity of α -Conotoxin GeXIVA.</p>

<p>Mar Drugs. 2020 Jan 17;18(1):</p>

<p>Authors: Sun Z, Zhangsun M, Dong S, Liu Y, Qian J, Zhangsun D,

Luo S</p>

<p>Abstract

Nicotinic acetylcholine receptors (nAChRs) are membrane receptors and play a major role in tumorigenesis and cancer progression. Here, we have investigated the differential expression of nAChR subunits in human breast cancer cell lines and breast epithelial cell lines at mRNA and protein levels and the effects of the α 0-conotoxin GeXIVA, antagonist of α 9 α 10 nAChR, on human breast cancer cells. Reverse transcription polymerase chain reaction (PCR) demonstrated that all nAChR subunits, except α 6, were expressed in the 20 tested cell lines. Real time quantitative PCR (QRT-PCR) suggested that the mRNA of α 5, α 7, α 9 and β 4 nAChR subunits were overexpressed in all the breast cancer cell lines compared with the normal epithelial cell line HS578BST. α 9 nAChR was highly expressed in almost all the breast cancer cell lines in comparison to normal cells. The different expression is prominent ($p < 0.001$) as determined by flow cytometry and Western blotting, except for MDA-MB-453 and HCC1395 cell lines. α 0-conotoxin GeXIVA that targeted α 9 α 10 nAChR were able to significantly inhibit breast cancer cell proliferation in vitro and merits further investigation as potential agents for targeted therapy.

</p><p>PMID: 31963558 [PubMed - in process]</p>

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<author> Sun Z, Zhangsun M, Dong S, Liu Y, Qian J, Zhangsun D, Luo S</author>

<category>Mar Drugs</category>

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<title>RgIA4 Accelerates Recovery from Paclitaxel-Induced Neuropathic Pain in Rats.</title>

<link>https://www.ncbi.nlm.nih.gov/pubmed/31877728?dopt=Abstract</link>

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<p>RgIA4 Accelerates Recovery from Paclitaxel-Induced Neuropathic Pain in Rats.</p>

<p>Mar Drugs. 2019 Dec 21;18(1):</p>

<p>Authors: Huynh PN, Giuvelis D, Christensen S, Tucker KL, McIntosh JM</p>

<p>Abstract

Chemotherapeutic drugs are widely utilized in the treatment of human cancers. Painful chemotherapy-induced neuropathy is a common, debilitating, and dose-limiting side effect for which there is currently no effective treatment. Previous studies have demonstrated the potential utility of peptides from the marine snail from the genus Conus for the treatment of neuropathic pain. α -Conotoxin RgIA and a potent analog, RgIA4, have previously been shown to prevent the development of neuropathy resulting from the administration of oxaliplatin, a platinum-based antineoplastic drug. Here, we have examined its efficacy against paclitaxel, a chemotherapeutic drug that works by a mechanism of action distinct from that

of oxaliplatin. Paclitaxel was administered at 2 mg/kg (intraperitoneally (IP)) every other day for a total of 8 mg/kg. Sprague Dawley rats that were co-administered RgIA4 at 80 µg/kg (subcutaneously (SC)) once daily, five times per week, for three weeks showed significant recovery from mechanical allodynia by day 31. Notably, the therapeutic effects reached significance 12 days after the last administration of RgIA4, which is suggestive of a rescue mechanism. These findings support the effects of RgIA4 in multiple chemotherapeutic models and the investigation of $\alpha 9\alpha 10$ nicotinic acetylcholine receptors (nAChRs) as a non-opioid target in the treatment of chronic pain.

</p><p>PMID: 31877728 [PubMed - in process]</p>

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<author> Huynh PN, Giuvelis D, Christensen S, Tucker KL, McIntosh JM</author>

<category>Mar Drugs</category>

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