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Biologically Active Peptides

Amyloid β-Proteins

Code	Compound			Price:Yen
4481-v	**Amyloid β-Protein (Human, 1-28)**	Vial	0.5 mg	15,000

New

-20℃

SP-28 (Human)

(Trifluoroacetate Form)

Asp-Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr-Glu-Val-His-His-Gln-Lys-Leu-Val-Phe-Phe-Ala-Glu-Asp-Val-Gly-Ser-Asn-Lys

(M.W. 3262.5) $C_{145}H_{209}N_{41}O_{46}$ [109770-29-8]

Purity ≧99.0% (HPLC)

Amyloidogenic Segment of Amyloid β-Protein

Amyloid β-protein (Human, 1-28) is a peptide that was synthesized chemically during the early stages of amyloid β-proteins research[1]. Over the years, many research publications using **amyloid β-protein (Human, 1-28)** have been reported, frequently including: **i)** formation of amyloid fibrils[1-5] and **ii)** expression of neurotrophic or neurotoxic activity[5-7], some of which are specific to this particular peptide. **Amyloid β-protein (Human, 1-28)** possesses only one of the fibril forming domains (central domain) and lacks the corresponding C-terminal one[5], so that this peptide reinforces a structural as well as functional interest in the research of Alzheimer's disease.

1) D.A. Kirschner, H. Inouye, L.K. Duffy, A. Sinclair, M. Lind, and D.J. Selkoe, *Proc. Natl. Acad. Sci. U.S.A.*, **84**, 6953 (1987). (*Original; Amyloid-Like Fibril Formation*)
2) B. Klajnert, M. Cortijo-Arellano, J. Cladera, and M. Bryszewska, *Biochem. Biophys. Res. Commun.*, **345**, 21 (2006). (*Amyloid Fibril Formation*)
3) A. Perálvarez-Marin, A. Barth, and A. Gräslund, *J. Mol. Biol.*, **379**, 589 (2008). (*Amyloid Fibril Formation*)
4) N.G.N. Milton and J.R. Harris, *Micron*, **40**, 800 (2009). (*Amyloid Fibril Formation*)
5) T. Wasiak, M. Ionov, K. Nieznanski, H. Nieznanska, O. Klementieva, M. Granell, J. Cladera, J.-P. Majoral, A.M. Caminade, and B. Klajnert, *Mol. Pharm.*, **9**, 458 (2012). (*Amyloid Fibril Formation & Neurotoxicity*)
6) J.S. Whitson, D.J. Selkoe, and C.W. Cotman, *Science*, **243**, 1488 (1989). (*Pharmacol.*)
7) B.A. Yankner, L.K. Duffy, and D.A. Kirschner, *Science*, **250**, 279 (1990). (*Pharmacol.*)

Code	Compound			Price:Yen
4484-v	**Amyloid β-Protein (Human, 1-38)**	Vial	0.5 mg	17,000

New

-20℃

(Trifluoroacetate Form)

Asp-Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr-Glu-Val-His-His-Gln-Lys-Leu-Val-Phe-Phe-Ala-Glu-Asp-Val-Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met-Val-Gly-Gly

(M.W. 4131.5) $C_{184}H_{277}N_{51}O_{56}S$ [131438-74-9]

Purity ≧95.0% (HPLC)

Endogenous Form of Amyloid β-Protein in Cerebrospinal Fluid

Human amyloid β-proteins are produced by enzymatic cleavages of amyloid precursor protein (APP); enzymes involved are β- and γ-secretases. Successive scissions of APP by these enzymes yield primarily the longer forms of amyloid β-protein, such as amyloid β-protein (1-49). Recently, it has been clarified that these longer forms are not the final products, but the substrates of γ-secretase, providing **amyloid β-protein (Human, 1-38)** through numerous intermediates such as amyloid β-protein (Human, 1-42) (Code 4349-v), and amyloid β-protein (Human, 1-43) (Code 4370-v)[1,2].

Amyloid β-protein (Human, 1-38) has been discovered to exist in human plasma and cerebrospinal fluid[3,4], among other locations. Functionally, **amyloid β-protein (Human, 1-38)** is reported to enhance glutamate neurotoxicity in cortical cultures at doses between 20 and 80 μM[5]. **Amyloid β-protein (Human, 1-38)** is now available from Peptide Institute, Inc.

1) M. Okochi, S. Tagami, K. Yanagida, M. Takami, T.S. Kodama, K. Mori, T. Nakayama, Y. Ihara, and M. Takeda, *Cell Rep.*, **3**, 42 (2013). (*γ-Secretase-Mediated Generation*)
2) N. Matsumura, M. Takami, M. Okochi, S. Wada-Kakuda, H. Fujiwara, S. Tagami, S. Funamoto, Y. Ihara, and M. Morishima-Kawashima, *J. Biol. Chem.*, **289**, 5109 (2014). (*γ-Secretase-Mediated Generation*)
3) J.M. Maler, H.-W. Klafki, S. Paul, P. Spitzer, T.W. Groemer, A.W. Henkel, H. Esselmann, P. Lewczuk, J. Kornhuber, and J. Wiltfang, *Proteomics*, **7**, 3815 (2007). (*Quantitation in Human Plasma*)
4) M.E. Lame, E.E. Chambers, and M. Blatnik, *Anal. Biochem.*, **419**, 133 (2011). (*Quantitation in Human Cerebrospinal Fluid*)
5) M.P. Mattson, B. Cheng, D. Davis, K. Bryant, I. Lieberburg, and R.E. Rydel, *J. Neurosci.*, **12**, 376 (1992). (*Pharmacol.; Enhancement of Glutamate Neurotoxicity*)

Orexins

Code	Compound			Price:Yen
4482-s New -20℃	**Orexin-A (Human, 17-33)** **OXA (17-33)** Tyr-Glu-Leu-Leu-His-Gly-Ala-Gly-Asn-His-Ala-Ala-Gly-Ile-Leu-Thr-Leu-NH_2 (M.W.1749.0) $C_{79}H_{125}N_{23}O_{22}$ [343268-91-7] Purity ≧99.0% (HPLC) *Orexin-1 Receptor Selective Agonist*	Vial	0.1 mg	8,000
4483-s New -20℃	**[Ala^{11}, D-Leu^{15}]-Orexin B (Human)** Arg-Ser-Gly-Pro-Pro-Gly-Leu-Gln-Gly-Arg-Ala-Gln-Arg-Leu-D-Leu-Gln-Ala-Ser-Gly-Asn-His-Ala-Ala-Gly-Ile-Leu-Thr-Met-NH_2 (M.W. 2857.3) $C_{120}H_{206}N_{44}O_{35}S$ [532932-99-3] *Orexin-2 Receptor Selective Agonist*	Vial	0.1 mg	10,000

Orexin A (Code 4346-s) and orexin B (Code 4348-s: Human, Code 4347-s: Rat & Mouse) elicit numerous biological functions including feeding regulation and wake/sleep regulation. These peptides bind to two G protein-coupled receptors, orexin 1 receptor (OX_1) and orexin 2 receptor (OX_2) to express their biological activities; orexin A interacts with both OX_1 and OX_2 non-selectively, whereas orexin B shows preferential OX_2 binding ability. To learn the contributions of these peptides to exerting an individual function of orexins, a selective ligand to each receptor subtype is required.

After an extensive structure-activity relationship study of orexin A, **orexin A (Human, 17-33)** was discovered to be 23-fold more selective to OX_1 than OX_2[1]. In the case of OX_2, **[Ala^{11}, D-Leu^{15}]-orexin B (Human)** is reported to show a 400-fold selectivity over OX_1[2], although one report described that such potency in receptor selectivity could not be reproduced[3]. These two orexin analogs may be useful tools for orexin A and orexin B distinguishing research.

1) N.A. German, A.M. Decker, B.P. Gilmour, B.F. Thomas, and Y. Zhang, *ACS Med. Chem. Lett.*, **4**, 1224 (2013). (*Original; Structure-Activity Relationship & Pharmacol.*)
2) S. Asahi, S.-I. Egashira, M. Matsuda, H. Iwaasa, A. Kanatani, M. Ohkubo, M. Ihara, and H. Morishima, *Bioorg. Med. Chem. Lett.*, **13**, 111 (2003). (*Original; Structure-Activity Relationship & Pharmacol.*)
3) J. Putura, P.M. Turunen, M.H. Jäntti, M.E. Ekholm, and J.P. Kukkonen, *Neurosci. Lett.*, **494**, 57 (2011). (*Pharmacol.*)

CEP1

Code	Compound			Price:Yen
4487-s New -20℃	**CEP1** **C-Terminally Encoded Peptide 1** **(Plant, *Arabidopsis*)** Asp-Phe-Arg-Hyp-Thr-Asn-Pro-Gly-Asn-Ser-Hyp-Gly-Val-Gly-His (M.W. 1583.6) $C_{66}H_{98}N_{22}O_{24}$	Vial	0.1 mg	7,000

Mediator of Systemic N-Demand Signaling in Plant

In plants the peptide ligand-receptor systems are involved in developmental processes with some diversities[1]. Peptide ligands in these systems are categorized into groups; the major constituents are the secreted small peptides with posttranslational modification(s) which function extracellularly[2]. This particular group of peptides is encoded by paralogous genes, in which the mature peptides are located at the C-terminal conserved domains. One such peptide is phytosulfokine (Code 4477-s), which contains two sulfated-Tyr residues.

Another member of this family is **CEP1** (**C-terminally encoded peptide 1**), isolated from *Arabidopsis* by *in silico* gene screening[3]. Very recently, a signaling systems triggered by **CEP1** has been clarified[4]: **1)** secretion of **CEP1** from root upon starvation of N-neutrient, **2)** translocation of **CEP1** to shoot and perception by Leu-rich repeat receptor kinases (LRR-RKs) which are receptors of **CEP1**, and **3)** stimulation of nitrate uptake in roots in N–rich region. This "root-to-shoot-back-to-root" signaling is considered to be effective in escaping from the retardation of root growth under the nutrient-depleted conditions. **CEP1** may help in understanding the adaptation mechanism of plants in environmentally disadvantageous situations.

1) S. Endo, S. Betsuyaku, and H. Fukuda, *Curr. Opin. Plant Biol.*, **21**, 140 (2014). (*Review*)
2) Y. Matsubayashi, *Annu. Rev. Plant Biol.*, **65**, 385 (2014). (*Review*)
3) K. Ohyama, M. Ogawa, and Y. Matsubayashi, *Plant J.*, **55**, 152 (2008). (*Original*)
4) R. Tabata, K. Sumida, T.Yoshii, K.Ohyama, H. Shinohara, and Y. Matsubayashi, *Science*, **346**, 343 (2014). (*Pharmacol.*)

Enzyme Substrates

Code	Compound			Price:Yen
3234-s	**LISA-101**	Vial	0.1 mg	10,000

New

-20℃

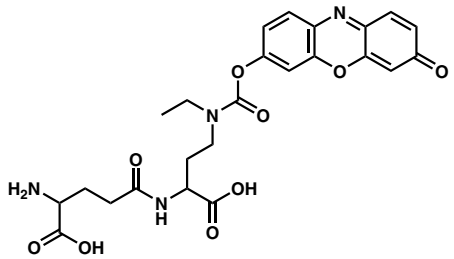
(Trifluoroacetate Form)

γ-L-Glutamyl-(2*S*)-*N*$^{\gamma}$-ethyl-*N*$^{\gamma}$-[(3-oxo-3*H*-phenoxazin-7-yl)oxy] carbonyl-2,4-diaminobutyric acid

(M.W. 514.48) $C_{24}H_{26}N_4O_9$ [1638785-74-6]

Purity ≧98.0% (HPLC)

Fluorogenic Substrate for γ-Glutamyl Cyclotransferase (GGCT)



γ-Glutamyl cyclotransferase[1] (GGCT, γ-GCT, C7orf24, EC 2.3.2.4) attracts interest as a member of glutathione cycle-related enzymes. GGCT has been found to be over-expressed in a range of cancers[2]. In particular, presence of GGCT is thought to be relevant to the malignancy of the cancer cells[3]. Additionally, it is interesting that systemic administration of siRNA of GGCT could retard the tumor growth and induce necrosis of tumor tissue while showing no obvious toxicity to normal tissues[4]. Specifically, GGCT converts γ-glutamyl amino acids (γ-Glu-Xaa, Xaa: any amino acid) into 5-oxoproline (pyroglutamate) and the corresponding amino acids (Xaa). This singular substrate preference hampered its chemical probe development for some time.

Against this background, a novel fluorogenic GGCT probe "**LISA*-101**" was successfully developed at Peptide Institute, Inc. in collaboration with Professor Yoshiki of Kyoto Pharmaceutical University and Shiga University of Medical Science[5]. GGCT treatment liberates an intact fluorophore "resorufin" from **LISA-101** which is not a fluorescence compound. This makes it possible to quantify GGCT activity with the guidance of fluorescence (e.g. Ex 571 nm/ Em 585 nm). This probe, **LISA-101**, can expand the repertoire of strategies used in cancer studies related to GGCT.

*LISA: Ligand Inspired by Substrate Acidity

1) a) M. Orlowsli, P.G. Richman, and A. Meister, *Biochemistry*, **8**, 1048 (1969); b) A.J. Oakley, T. Yamada, D. Liu, M. Coggan, A.G. Clark, and P.G. Board, *J. Biol. Chem.*, **283**, 22031 (2008). (*Isolation and Identification of GGCT*)
2) P. Gromov, I. Gromova, E. Friis, V. Timmermans-Wielenga, F. Rank, R. Simon, G. Sauter, and J.M.A. Moreira, *J. Proteome Res.*, **9**, 3941 (2010). (*Proteomic Profiling of Mammary Carcinomas*)
3) K. Takemura, H. Kawachi, Y. Eishi, K. Kitagaki, M. Negi, M. Kobayashi, K. Uchida, J. Inoue, J. Inazawa, T. Kawano, and P.G. Board, *Human Pathol.*, **45**, 331 (2014). (*GGCT as a Diagnostic Marker in Esophageal Tumors*)
4) R. Ran, Y. Liu, H. Gao, Q. Kuang, Q. Zhang, J. Tang, H. Fu, Z. Zhang, and Q. He, *J. Pharm. Sci.*, **104**, 476 (2015). (*siRNA for Treatment of Drug-Resistant Breast Cancer*)
5) a) H. Ii, Y. Nishiuchi, T. Yoshiki, and T. Yoshiya, Japan Patent JP 2014-218494 A
b) T.Yoshiya, H. Ii, S.Tsuda, S.Kageyama, T.Yoshiki and Y.Nishiuchi, *Org. Biomol. Chem.*, **13**, 3182 (2015). (*Development of LISA-101*)

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Code	Compound			Price:Yen
3235-v	**Leu-Asp-MCA**	Vial	5 mg	5,000
New	L-Leucyl-L-aspartic acid α-(4-methylcoumaryl-7-amide)			
-20℃	(M.W. 403.43) $C_{20}H_{25}N_3O_6$ [929621-97-6]			
	Purity ≧99.0% (HPLC), AMC ≦0.1%, TLC single spot			

Substrate for Porphyromonas Gingivalis Dipeptidyl-Peptidase 11

1) Y. Ohara-Nemoto, Y. Shimoyama, S. Kimura, A. Kon, H. Haraga, T. Ono, and T.K. Nemoto, *J. Biol. Chem.*, **286**, 38115 (2011).
2) S.M. Rouf, Y. Ohara-Nemoto, T. Hoshino, T. Fujiwara, T. Ono, and T.K. Nemoto, *Biochimie*, **95**, 824 (2013).
3) S.M. Rouf, Y. Ohara-Nemoto, T. Ono, Y. Shimoyama, S. Kimura, and T.K. Nemoto, *FEBS Open Bio*, **3**, 177 (2013).

Code	Compound			Price:Yen
3236-v	**Met-Leu-MCA**	Vial	5 mg	5,000
New	(Trifluoroacetate Form)			
-20℃	L-Methionyl-L-Leucine 4-methylcoumaryl-7-amide			
	(M.W. 419.54) $C_{21}H_{29}N_3O_4S$ [1009549-31-8]			
	Purity ≧99.0% (HPLC), AMC ≦0.1%, TLC single spot			

Substrate for Porphyromonas Gingivalis Dipeptidyl-Peptidase 7

1) A. Banbula, J. Yen, A. Oleksy, P. Mak, M. Bugno, J. Travis, and J. Potempa, *J. Biol. Chem.*, **276**, 6299 (2001).
2) S.M. Rouf, Y. Ohara-Nemoto, T. Hoshino, T. Fujiwara, T. Ono, and T.K. Nemoto, *Biochimie*, **95**, 824 (2013).
3) S.M. Rouf, Y. Ohara-Nemoto, T. Ono, Y. Shimoyama, S. Kimura, and T.K. Nemoto, *FEBS Open Bio*, **3**, 177 (2013).

Glycopeptides

Big Angiotensin-25

Code	Compound			Price:Yen
24009-s	**Big Angiotensin-25 (Human)**	Vial	0.1 mg	20,000

New

Bang-25

-20℃

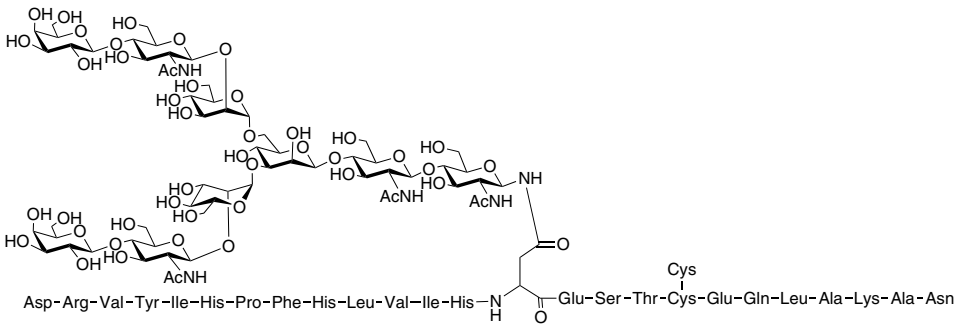
(Trifluoroacetate Form)

Asp-Arg-Val-Tyr-Ile-His-Pro-Phe-His-Leu-Val-Ile-His-[*O*-β-D-Galactopyranosyl-(1→4)-*O*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→2)-*O*-α-D-mannopyranosyl-(1→3)-[*O*-β-D-galactopyranosyl-(1→4)-*O*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→2)-*O*-α-D-mannopyranosyl-(1→6)]-*O*-β-D-mannopyranosyl-(1→4)-*O*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→4)-*N*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→4)]-Asn-Glu-Ser-Thr-Cys(Cys)-Glu-Gln-Leu-Ala-Lys-Ala-Asn

(M.W. 4677.9) $C_{195}H_{307}N_{43}O_{85}S_2$

Synthetic Product

Glycosylated Novel Endogenous Angiotensin Family Peptide



Since the discovery of the longer form of angiotensin I (Human) (Code 4007) in rat, named proangiotensin-12 (Rat) (Code 4439-v), Professor Kitamura of University of Miyazaki has endeavored to find the longer form of angiotensin family peptide(s) in humans. As a result, he clarified that such peptides in humans are not a 12 amino acid residue peptide corresponding to proangiotensin-12 (Rat), but rather a 25 amino acid residue peptide called **big angiotensin-25 (Human)**[1].

Big angiotensin-25 (Human) was isolated from urine as a major peptide when surveyed using an antibody raised against the N-terminal portion of angiotensin II. Structure analysis revealed that the primary structure of **big angiotensin-25 (Human)** is comprised of the first 25 amino acid residues of angiotensinogen and there exists two modifications in the sequence: **i)** N-glycosylation by 9-mer sugar at Asn^{14} in the typical N-glycosylation motif, Asn-Glu-Ser and **ii)** attachment of Cys on Cys^{18} through disulfide linkage. In collaboration with Professor Kitamura, we have succeeded in synthesizing **big angiotensin-25 (Human)** and confirmed that the native and synthetic **big angiotensin-25 (Human)** elute at the identical retention time on reversed phase-HPLC[1,2].

Interestingly, **big angiotensin-25 (Human)** is cleaved rapidly by chymase, directly providing angiotensin II, whereas it is slowly cleaved by renin (an enzyme generating angiotensin I). Other experimental results show that **big angiotensin-25 (Human)** immunoreactivity is located in various tissues in humans such as kidney and heart, among others. **Big angiotensin-25 (Human)** is suggested to be a precursor of tissue angiotensin II.

Chemically synthesized **big angiotensin-25 (Human)**, available now, will help to unravel the *in vivo* functional roles of this particular form of the angiotensin family peptide.

1) S. Nagata, K. Hatakeyama, M. Asami, M. Tokashiki, H. Hibino, Y. Nishiuchi, K. Kuwasako, J. Kato, Y. Asada, and K. Kitamura, *Biochem. Biophys. Res. Commun.*, **441**, 757 (2013). (*Original; Structure & Enzymatic Generation of Angiotensin II*).

2) H. Hibino, S. Nagata, K. Hatakeyama, M. Asami, M. Tokashiki, K. Kuwasako, J. Kato, Y. Asada, K. Kitamura, and Y. Nishiuchi, *Peptide Science 2013*, **50**, 151 (2014). (*Chem. Synthesis*)

SGP

Code	Compound			Price:Yen
24010	**Sialylglycopeptide**	Bulk	5 mg	30,000

New

-20℃

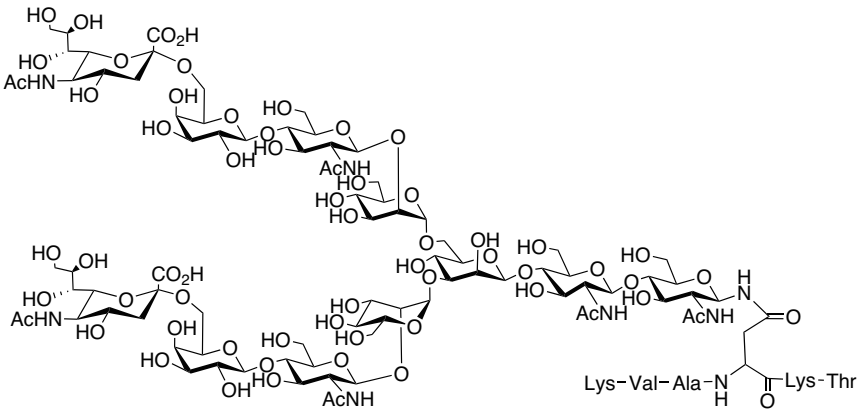
SGP (α2,6)

Lys-Val-Ala-[*O*-(5-Acetamido-3,5-dideoxy-D-glycero-α-D-galacto-2-nonulopyranosylonic acid)-(2→6)-*O*-β-D-galactopyranosyl-(1→4)-*O*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→2)-*O*-α-D-mannopyranosyl-(1→3)-[*O*-(5-acetamido-3,5-dideoxy-D-glycero-α-D-galacto-2-nonulopyranosylonic acid)-(2→6)-*O*-β-D-galactopyranosyl-(1→4)-*O*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→2)-*O*-α-D-mannopyranosyl-(1→6)]-*O*-β-D-mannopyranosyl-(1→4)-*O*-2-acetamido-2-deoxy-β-D-glucopyranosyl-(1→4)-*N*-2-acetamido-2-deoxy-β-D-glucopyranosyl]-(1→4)-Asn-Lys-Thr

(M.W. 2865.8) $C_{112}H_{189}N_{15}O_{70}$ [189035-43-6]

Purity ≧95.0% (HPLC)

Natural product isolated from egg yolk



1) A. Seko, M. Koketsu, M. Nishizono, Y. Enoki, H.R. Ibrahim, L.R. Juneja, M. Kim, and T. Yamamoto, *Biochim. Biophys. Acta*, **1335**, 23 (1997).

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3. 消光性蛍光基質(Nma-Dnp，MOCAc-Dnp，Dabcyl-EDANS の組合せなど)
4. 酵素阻害剤(アルデヒド，フルオロメチルケトン，クロロメチルケトン，ボロン酸誘導体など)
5. ジスルフィド結合含有ペプチド(1 組，2 組，3 組以上，分子内，分子間)
6. 環状ペプチドおよび枝分かれペプチド
7. 鎖状および環状デプシペプチド
8. 糖ペプチド［Asn(GlcNAc), Ser/Thr(GalNAc), Ser/Thr(GlcNAc), Ser/Thr(Gal-GalNAc), Ser/Thr(Neu-Gal-GalNAc), 1-Deoxyfructosyl 含有ペプチドなど］
9. リン酸化ペプチド［Ser(PO_3H_2)，Thr(PO_3H_2)，Tyr(PO_3H_2)誘導体］
10. フォスファターゼ抵抗性リン酸化ペプチドミミック（Ser, Thr, Tyr, His, Asp 対応）
11. 硫酸化ペプチド［Tyr(SO_3H)］
12. 蛍光標識ペプチド(FITC, Rhodamine, BODIPY, DY- シリーズなど 380 nm-730 nm に対応)
13. 安定同位体ラベルアミノ酸含有ペプチド(^{13}C，^{15}N，^{2}H など)
14. PEG 化(分子量 150 ～ 4 万まで , N 末端 , C 末端 , 側鎖)
15. フォトアフィニティ用ラベル化(N_3, Benzophenone, Diazirine など)
16. 細胞膜透過性修飾（Tat, オリゴアルギニン , Penetratin など)
17. 非天然アミノ酸含有ペプチド(ハイプシン , システインスルフィン酸など他多数)
18. アミノ基修飾(Biotinyl 化 , Myristoyl 化 , Palmitoyl 化 , Methyl 化 , Malonyl 化など種々の修飾)
19. チオール基修飾(Farnesyl 化，Geranyl 化，Palmitoyl 化など)
20. 水酸基修飾(Octanoyl 化 , Palmitoyl 化 , Palmitoleoyl 化など)
21. ペプチド結合の修飾(還元型 , スタチン型など)
22. ペプチドアルコール
23. アミノ酸誘導体 , 保護ペプチド
24. その他

受託合成量： 標準 10mg-25mg から g、kg オーダーまで承ります。（お見積り依頼の際に、ご相談下さい）

納　　期： 配列、修飾、難度、合成量により異なります（最短 1 週間～）
個別お見積いたしますのでご用命下さい。

保証純度： 通常、トリフルオロ酢酸塩でご提供いたします。
規格　（1）HPLC で検定：90% 以上（精製品）
（2）HPLC で検定：95% 以上（精製品）
（3）HPLC で検定：99% 以上（精製品）など
通常、HPLC チャート、アミノ酸分析結果、質量分析結果を添付いたします。
その他の分析項目につきましてはご相談させていただきます。

価　　格： 個別にお見積りいたしますので、E-mail、あるいは FAX にてご用命下さい。

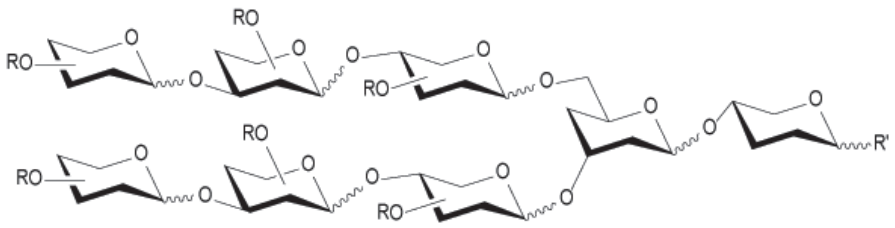
ペプチド医薬品合成（GMP）

弊社は、医薬品製造業許可を取得しており、医薬品原薬および原薬中間体の製造が可能です。
GMP 対応、治験薬 GMP 対応など個別にご相談させていただきます。
現在、ペプチド医薬品の基礎研究から上市後の受託製造まで、トータルなサービスのご提供に取り組んでいます。高品質なペプチド原薬をあらゆる場面でご提供できますので、お気軽にお問い合わせ下さい。

基礎研究 → 非臨床試験 → 臨床研究 → 治験 → 薬事申請 → 上市

糖鎖関連合成

糖を母体とした有機化合物から九糖まで実績多数



単糖・オリゴ糖	修 飾 糖	蛍光標識糖	安定同位体 ラベル糖	糖ヌクレオチド
糖 脂 質	グリコサミノグリカン フラグメント	各種糖アナログ	脂　質	糖化最終産物

抗体（ポリクローナル）作製

エピトープ選択	抗原ペプチドの合成	コンジュゲート作製
抗体作製	抗体の精製	抗体の修飾

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