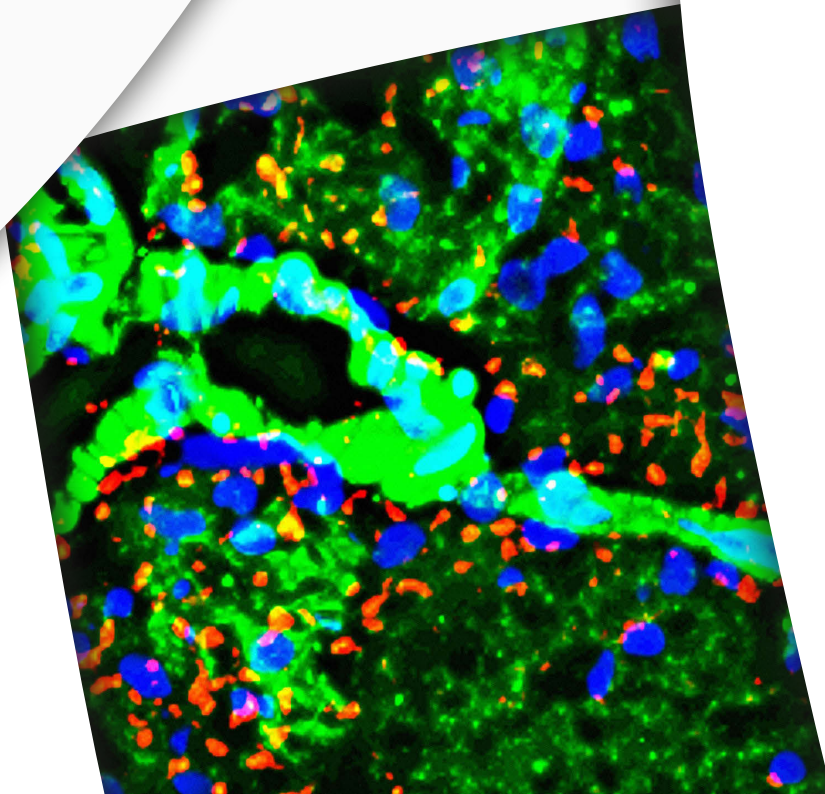# TAU PEPTIDES
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TAU PEPTIDES OFFERED BY BACHEM

Alzheimer's disease is a neurodegenerative disease characterized by extracellular amyloid-β (Aβ) plaques and intracellular neurofibrillary tangles containing pathological tau protein aggregates. Even though both, Aβ and tau, are related to Alzheimer's disease, studies have shown that the degree of tau-related pathology correlates better with the degree of dementia than the amyloid plaque burden. Human tau, which exists in six isoforms of different length, is used in cerebrospinal fluid as a sensitive and specific biomarker for Alzheimer's disease, usually determined by ELISA.
In addition to our catalog portfolio of over 250 Alzheimer's disease related research products, Bachem now offers a comprehensive choice of tau peptides including phosphorylated sequences as new catalog products.

Tau Protein

Microtubules are part of the cytoskeleton, tubulin polymers with various cellular tasks. For instance, they play a role in stabilizing cell shape and during mitosis, and act as tracks for intracellular transport by motor proteins. Microtubules are stabilized by various types of microtubule-associated proteins which bind to their surface and promote their self-assembly from tubulin subunits. Tau (Microtubule-Associated Protein Tau, MAPT) [1] stabilizes the microtubules of the neuronal cytoskeleton und thus is important for their regular function. Tau protein is primarily expressed in neuronal cell bodies and axons.

The binding affinity of tau for microtubules is regulated by the phosphorylation state of the protein. In healthy neurons, the protein is phosphorylated by a number of kinases and dephosphorylated by phosphatases [2,3]. An equilibrium of phosphorylated and non-phosphorylated forms serving as tau pool exists in the cytosol. Phosphorylation is reverted when tau binds to tubulin. Glycogen-synthase kinase-3β (GSK-3β), cyclin-dependent protein kinase 5 (cdk5), and cAMP-dependent protein kinase (PKA) are amongst the most important tau kinases [4]. Protein phosphatase PP2A was found to be the major tau phosphatase ([3,5]. In Alzheimer's disease (AD) or other pathological conditions known as tauopathies, phosphorylation of the protein is dysregu-

TAU PROTEIN

Tau protein is primarily expressed in neuronal cell bodies and axons and, depending on its six isoforms, has a length of 352 to 441 amino acids.

lated leading to a decrease of the tubulin-binding capacity. Whereas the microtubules are destabilized, hyperphosphorylated tau protein aggregates yielding the so-called neurofibrillary tangles (NFTs), one of the hallmarks of AD [6-9]. In NFTs, the extent of tau phosphorylation was found to be 3 to 4 times higher than in cytosolic tau of healthy and AD brains [10].

Tau is a rather disordered highly flexible protein. Due to its random coil conformation

Table 1: Isoforms of human Tau protein (N insert, R repeat).

Clone*	Inserts/ Repeats	Number of AA	MW (kDa)	Sequence
htau23 (fetal tau)	0N/3R	352	36.7	MAEPRQEFEVMEDHAGTYGLGDRKDQGGYTMHQDQEGDTDAGLKAEEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDDKKAKGADGKTKIATPRGAAPPGQKGQANATRIPAKTPPAPKTPPSSGEPPKSGDRSGYSSPGSPGTPGSRSRTPSLPTPPTREPKKVAVVRTPPKSPSSAKSRLQTAPVPMPDLKNVKSKIGSTENLKHQPGGGKVQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQVEVKSEKLDFKDRVQSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEIVYKSPVVSGDTSPRHLSNVSSTGSIDMVDSPQLATLADEVSASLAKQGL
htau37	1N/3R	381	39.7	MAEPRQEFEVMEDHAGTYGLGDRKDQGGYTMHQDQEGDTDAGLKESPLQTPTEDGSEEPGSETSDAKSTPTAEAEEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDDKKAKGADGKTKIATPRGAAPPGQKGQANATRIPAKTPPAPKTPPSSGEPPKSGDRSGYSSPGSPGTPGSRSRTPSLPTPPTREPKKVAVVRTPPKSPSSAKSRLQTAPVPMPDLKNVKSKIGSTENLKHQPGGGKVQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQVEVKSEKLDFKDRVQSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEIVYKSPVVSGDTSPRHLSNVSSTGSIDMVDSPQLATLADEVSASLAKQGL
htau39	2N/3R	410	42.6	MAEPRQEFEVMEDHAGTYGLGDRKDQGGYTMHQDQEGDTDAGLKESPLQTPTEDGSEEPGSETSDAKSTPTAEDVTAPLVDEGAPGKQAAAQPHTEIPEGTTAEEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDDKKAKGADGKTKIATPRGAAPPGQKGQANATRIPAKTPPAPKTPPSSGEPPKSGDRSGYSSPGSPGTPGSRSRTPSLPTPPTREPKKVAVVRTPPKSPSSAKSRLQTAPVPMPDLKNVKSKIGSTENLKHQPGGGKVQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQVEVKSEKLDFKDRVQSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEIVYKSPVVSGDTSPRHLSNVSSTGSIDMVDSPQLATLADEVSASLAKQGL
htau24	0N/4R	383	40.0	MAEPRQEFEVMEDHAGTYGLGDRKDQGGYTMHQDQEGDTDAGLKAEEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDDKKAKGADGKTKIATPRGAAPPGQKGQANATRIPAKTPPAPKTPPSSGEPPKSGDRSGYSSPGSPGTPGSRSRTPSLPTPPTREPKKVAVVRTPPKSPSSAKSRLQTAPVPMPDLKNVKSKIGSTENLKHQPGGGKVQIINKKLDLSNVQSKCGSKDNIKHVPGGGSVQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQVEVKSEKLDFKDRVQSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEIVYKSPVVSGDTSPRHLSNVSSTGSIDMVDSPQLATLADEVSASLAKQGL
htau34	1N/4R	412	43.0	MAEPRQEFEVMEDHAGTYGLGDRKDQGGYTMHQDQEGDTDAGLKESPLQTPTEDGSEEPGSETSDAKSTPTAEAEEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDDKKAKGADGKTKIATPRGAAPPGQKGQANATRIPAKTPPAPKTPPSSGEPPKSGDRSGYSSPGSPGTPGSRSRTPSLPTPPTREPKKVAVVRTPPKSPSSAKSRLQTAPVPMPDLKNVKSKIGSTENLKHQPGGGKVQIINKKLDLSNVQSKCGSKDNIKHVPGGGSVQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQVEVKSEKLDFKDRVQSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEIVYKSPVVSGDTSPRHLSNVSSTGSIDMVDSPQLATLADEVSASLAKQGL
htau40	2N/4R	441	45.9	MAEPRQEFEVMEDHAGTYGLGDRKDQGGYTMHQDQEGDTDAGLKESPLQTPTEDGSEEPGSETSDAKSTPTAEDVTAPLVDEGAPGKQAAAQPHTEIPEGTTAEEAGIGDTPSLEDEAAGHVTQARMVSKSKDGTGSDDKKAKGADGKTKIATPRGAAPPGQKGQANATRIPAKTPPAPKTPPSSGEPPKSGDRSGYSSPGSPGTPGSRSRTPSLPTPPTREPKKVAVVRTPPKSPSSAKSRLQTAPVPMPDLKNVKSKIGSTENLKHQPGGGKVQIINKKLDLSNVQSKCGSKDNIKHVPGGGSVQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQVEVKSEKLDFKDRVQSKIGSLDNITHVPGGGNKKIETHKLTFRENAKAKTDHGAEIVYKSPVVSGDTSPRHLSNVSSTGSIDMVDSPQLATLADEVSASLAKQGL

* designations taken from [9]

Insert 1: ESPLQTPTEDGSEEPGSETSDAKSTPTAE
Insert 2: DVTAPLVDEGAPGKQAAAQPHTEIPEGTT

Repeat 1: QTAPVPMPDLKNVKSKIGSTENLKHQPGGGK
Repeat 2: VQIINKKLDLSNVQSKCGSKDNIKHVPGGGS
Repeat 3: VQIVYKPVDLSKVTSKCGSLGNIHHKPGGGQ
Repeat 4: VEVKSEKLDFKDRVQSKIGSLDNITHVPGGGN

AD ALZHEIMER'S DISEASE – CHANGES IN TAU

crystals of the protein could not be obtained [11]. The loose, unfolded structure prevented the determination of the exact binding site of tau to microtubules. Tau is rather stable to treatment with acids or heating [12]. Tau consists of four domains characterized by their amino acid composition and function: the N-terminal "projection domain", a proline-rich region, the microtubule-binding part, and the C-terminal sequence [9]. The protein is a dipolar molecule, its N-terminal region contains a large proportion of acidic amino acid residues (pI 3.8), whereas the proline-rich middle part and the C-terminus are dominated by basic amino acids (pI 11.4 and 10.8, respectively) [8]. The distribution of charges can be changed by post-translational modifications associated with pathological conditions [13]. Tau exists in six major isoforms differing in number of tubulin-binding domains and length, they vary in size from 352 to 441 amino acid residues (see Table 1). They also differ in biological activity. The short isoforms allow plasticity of the cytoskeleton whereas the longer ones may preferentially play a role in its stabilization [14]. In adult human brains, the six isoforms can be found whereas in fetal brains, only the shortest isoform is expressed [15]. A central domain of tau, residues 198 to 369 for the longest isoform, aggregates with tubulin and promotes formation of microtubules. It is characterized by 3 to 4 repeat domains, all involved in binding [16] (Fig. 1). The N-terminal part is called projection domain as it projects from the surface of the microtubule. It varies in length, as 1 or 2 short sequences can be inserted. The projection domain interacts with other cytoskeletal elements and the neuronal plasma membrane, and is involved in signal transduction [17].

Tau Pathology

The mechanism of tau aggregation is not completely understood. Association with microtubules preserves the disordered structure of the protein. Aberrant post-translational modifications and conformational changes of tau go together with loss of affinity for tubulin and risk of aggregate formation. Phosphorylation of two or three Ser/Thr residues is required for the optimal function of tau, for pertaining its unfolded structure in free form, whereas hyperphosphorylation (> 6 phosphoserines/threonines [7]) inactivates the protein. A part of the hyperphosphorylated protein aggregates yielding NFTs or tau fibers (paired helical filaments, PHFs), the rest (about 40%) remains dissolved in the cytosol disrupting microtubules [10,18]. The longest tau isoform consisting of 441 amino acids contains about 80 Ser and Thr residues, all potential phosphorylation sites. Up to now, more than 40 sites have been identified in tau isolated from AD brains. The equilibrium between phosphorylation and dephosphorylation is regulated by various enzymes, so hyperphosphorylation may also result from the inability to revert this modification of tau by action of a phosphatase. Quantitative in vitro studies demonstrated that phosphorylation of tau at Ser^{262}, Thr^{231}, and Ser^{235} inhibited microtubule binding by ~35%, ~25%, and ~10%, respectively [19]. Evaluation of the binding kinetics between hyperphosphorylated and normal tau suggested that Ser^{199}/Ser^{202}/Thr^{205}, Thr^{212}, Thr^{231}/Ser^{235}, Ser^{262}/Ser^{356}, and Ser^{422} could be critical phosphorylation sites that turn tau into an inhibitory molecule that destabilizes microtubules by removing tubulin-associated protein [20.21]. Further phosphorylation at Thr^{231}, Ser^{396}, and Ser^{422} promoted self-aggregation of tau into filaments.
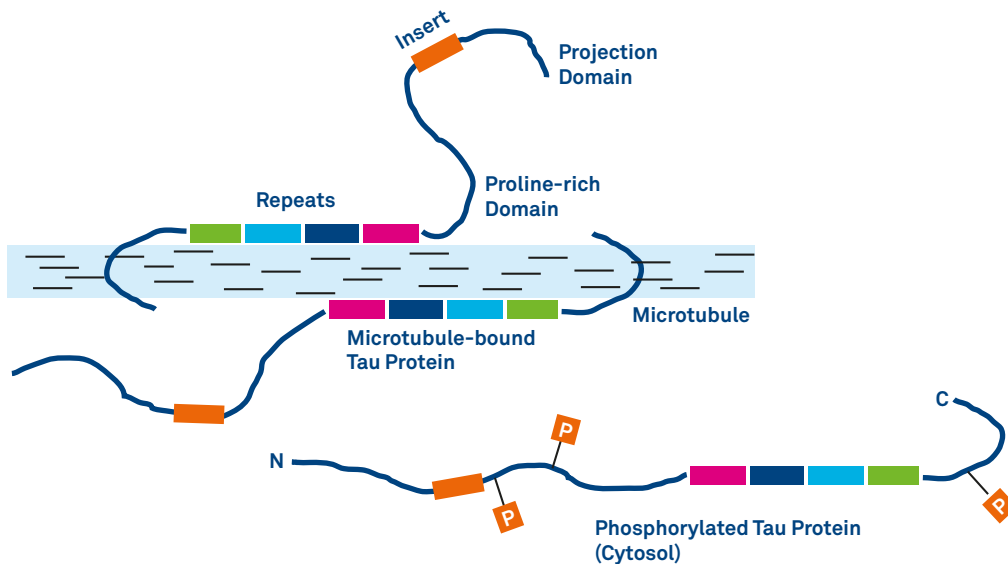
Fig. 1: Structure and normal function of tau. The protein contains 2-3 phosphoserine/threonine residues.

N^ϵ Acetylation of lysines may also be involved in the regulation of tau as well as in pathological processes [22]. This modification mediated by histone acetyltransferases (HAT) eliminates positive charges. Aberrant acetylation could interfere with the binding of tau to microtubules [23]. Acetylation of three lysine residues essential for tau binding has been detected. Acetylation of one of them, Lys[280], could play a role in tangle formation [23,24]. Hyperphosphorylation promotes N^ϵ acetylation. The side chain modification can be reverted by the NAD-dependent histone deacylase SIRT1 [25], so an equilibrium of acetylation/deacetylation could be maintained. Activity of SIRT1 is reduced in AD brains [26].

Enzymatic degradation is a major modification of tau protein in AD. Caspase-3-mediated cleavage at Asp[421] [27] and further C-terminal truncation promotes maturation of NFTs [28]. The truncated proteins could serve as biomarkers for AD. N-terminal truncation at Asp[13] is catalyzed by caspase-6, its role being less clear. Regular tau clearance via ubiquitinylation and degradation effected by the 26S proteasome or by autophagy is impeded in AD brains [29,30]. Glycation [31], deamidation [32], oxidation, and nitration [33,34] of tau are further modifications of the protein associated with tauopathies, whereas O-glycosylation of serine/threonine with N-acetylglucosamine

reduces aberrant phosphorylation [35,36]. This beneficial modification of tau relies on an intact glucose metabolism in the brain, a condition not always met in case of AD. Accordingly, diabetes has been established as a risk factor for the disease [37].

Conformation strongly affects the enzymatic (de)phosphorylation of serines and threonines in the vicinity of a proline residue. The peptidyl-prolyl cis/trans isomerase Pin 1 isomerizes phosphoserine/threonine-proline motifs. A trans-cis conversion of the $pThr^{231}$-Pro^{232} bond has been observed during early stages of AD. Pin1 prevents the accumulation of the resulting pathogenic cis form of tau by reverting the isomerization allowing dephosphorylation by PP2A [38]. Trans-tau promotes microtubule assembly. In AD brains, activities of both Pin 1 and PP2A are decreased [39]. An antibody developed against cis ($pThr^{231}$)-tau was shown to block brain injury and tauopathy [40,41].

VQIINK (tau 275-280) and VQIVYK (tau 306-311, PHF6), two hexapeptide motifs from the N-termini of repeats R2 and R3 with a marked tendency for β-sheet formation are involved in the aggregation of hyperphosphorylated tau yielding neurotoxic polymers [24,42]. PHF formation can be abolished by incorporating proline in one of the segments [43]. The chaperone Hsp90 binds to

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the VQIVYK motif [44] and, together with cochaperones, assists in tau clearance via autophagy or proteasome-mediated degradation [45]. Do et al. could demonstrate that the fragments Aβ (25-35), GSNKGAIIGLM, and tau (273-284), GK**VQIINK**KLDL, could interact affecting the self-assembly process of both Aβ and tau [46].

Tau and Alzheimer's Disease

Aggregation of β-amyloid peptides (which result from processing of amyloid precursor protein) into senile plaques as well as formation of neurofibrillary tangles (NFT) from tau protein are associated with Alzheimer's disease. Neither plaques nor NFT are a cause of neurodegeneration, but markers of damage and progression of AD. Nevertheless, both proteins play a central role in the development of AD and hence serve as biomarkers measured in cerebrospinal fluid [47]. It is not absolutely clear yet which of them is the real culprit responsible for eventual neuronal death. A number of observations point at tau, for example, neurofibrillary degeneration, and not β-amyloidosis, correlates with the presence of dementia in humans [48-50]

Neurofibrillary degeneration due to aberrant phosphorylation of tau could be prevented by inhibiting the involved enzymes, which makes GSK-3β, cdk5, PKA and other kinases attractive targets in developing AD cures [1,4,46]. Dephosphorylation could be promoted by upregulating PP2A, a phosphatase with broad substrate specificity.

Lithium inhibits GSK-3β, as it competes with magnesium [51,52]. In clinical studies, lithium was administered alone or in combination with valproate [53]. Further GSK-3β-inhibiting compounds as aloisines, flavopiridol, hymenialdisine, paullones, and staurosporine are under evaluation. The small molecule GSK-3β inhibitor tideglusib has reached clinical phase II for mild-to-moderate AD [54]. Aloisines and flavopiridol also inhibit cdk5, as does the purine olomucin. A reduction of tau hyperphosphorylation by administration of another purine, the selective cdk5 inhibitor roscovitine, could be achieved in mice and rats [55,56]. PP2A activity can be increased by down-

regulating the two endogenous inhibitor proteins I_1^{PP2A} and I_2^{PP2A} [6,57]. The NMDA antagonist memantine, a well-established drug for the management of moderate to severe AD, reverses tau hyperphosphorylation by regenerating PP2A [58]. The antioxidant melatonin has also been shown to restore the activity of the inhibited enzyme [59,60].

Davunetide (AL-108), an octapeptide corresponding to murine activity-dependent neuroprotective protein (74-81), binds to tubulin and promotes microtubule assembly [61]. Formulations for intranasal application and injection have been evaluated in clinical studies. Clinical tests were also performed with methylene blue (methylthioninium chloride) [62]. The dye prevents tau assembly and disrupts existing aggregates, though the mode of action is not known yet [63].

Results obtained from immunotherapeutical studies using AD models are encouraging [64]. Immunizations with Aβ or tau fragments showed positive effects such as improvement of locomotor activity and ability to perform cognitive tasks. Active immunization with phosphorylated tau fragments as $(pSer^{396,404})$-Tau (379-408) [65], $(pSer^{202}, pThr^{205})$- Tau (195-231), $(pThr^{212}, pSer^{214})$-Tau (207-220), or $(pThr^{231})$-Tau (224-238) [66] decreased tau pathology.

Conclusion

Numerous promising therapeutic approaches tackling the neuropathological effects of either aberrant tau modification or β-amyloid aggregate formation are being followed currently. This gives rise to hope that an effective cure for AD will be developed in the foreseeable future.

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Immunotherapy targeting pathological tau conformers in a tangle mouse model reduces brain pathology with associated functional improvements.
J. Neurosci. 27, 9115-9129 (2007)

[66] **M. Boimel et al.**
Efficacy and safety of immunization with phosphorylated tau against neurofibrillary tangles in mice.
Exp. Neurol. 224, 472-485 (2010)

TAU PEPTIDES FOR ALZHEIMER'S RESEARCH

Bachem's offer for Alzheimer's research comprises a broad choice of tau protein fragments and amyloid peptides. Please see also our Product Monograph "Amyloid Peptides".

For more details on our Alzheimer's disease peptides, please go to:
shop.bachem.com

TAU PEPTIDES

Microtubule-Associated Protein (142-161) (human)
(Tau Peptide (422-441))
H-8062 NEW
SPQLATLADEVSASLAKQGL

Tau Peptide (1-16)
H-8004 NEW
MAEPRQEFEVMEDMAG

Tau Peptide (45-73)
(Exon 2/Insert 1 Domain)
H-7804 NEW
ESPLQTPTEDGSEEPGSETSDAKSTPTAE

Tau Peptide (74-102)
(Exon 3/Insert 2 Domain)
H-7806 NEW
DVTAPLVDEGAPGKQAAAQPHTEIPEGTT

(Ser(PO$_3$H$_2$)^{202},Thr(PO$_3$H$_2$)^{205})- Tau Peptide (194-213)
H-8166 NEW
RSGYSSPG(p)SPG(p)TPGSRSRTP

(Thr(PO$_3$H$_2$)^{231})-Tau Peptide (225-237)
H-8048 NEW
KVAVVR(p)TPPKSPS

Tau Peptide (244-274)
(Repeat 1 Domain)
H-7982 NEW
QTAPVPMPDLKNVKSKIGSTENLKHQP-GGGK

Acetyl-Tau Peptide (244-274)
(Repeat 1 Domain)
H-7808 NEW
Ac-QTAPVPMPDLKNVKSKIG-STENLKHQPGGGK

Tau Peptide (245-274)
(Repeat 1 Domain)
H-7978 NEW
TAPVPMPDLKNVKSKIGSTENLKHQP-GGGK

Tau Peptide (255-314)
(Repeat 2 Domain)
H-8038 NEW
NVKSKIGSTENLKHQPGGGKVQIINK-KLDLSNVQSKCGSKDNIKHVPGGGSVQI-VYKPVD

(Ser(PO$_3$H$_2$)^{262})-Tau Peptide (260-264)
PAb Blocking
H-7966 NEW
IG(p)STE

Tau Peptide (268-282)
H-8068 NEW
HQPGGGKVQIINKKL

Tau Peptide (273-284)
H-8102 NEW
GKVQIINKKLDL

Acetyl-Tau Peptide (273-284) amide
H-8168 NEW
Ac-GKVQIINKKLDL-NH$_2$

Tau Peptide (274-288)
H-8072 NEW
KVQIINKKLDLSNVQ

Tau Peptide (275-305)
(Repeat 2 Domain)
H-7812 NEW
VQIINKKLDLSNVQSKCGSKDNIKHVP-GGGS

Tau Peptide (277-291)
H-8074 NEW
IINKKLDLSNVQSKC

Tau Peptide (295-309)
H-6076 NEW
DNIKHVPGGGSVQIV

Tau Peptide (298-312)
H-8078 NEW
KHVPGGGSVQIVYKP

Tau Peptide (301-315)
H-8082 NEW
PGGGSVQIVYKPVDL

Tau Peptide (304-318)
H-8084 NEW
GSVQIVYKPVDLSKV

Tau Peptide (306-317)
H-8096 NEW
VQIVYKPVDLSK

TAU PEPTIDES (CONTINUED)

Tau Peptide (306-336) (Repeat 3 Domain)
H-7814 NEW
VQIVYKPVDLSKVTSKCGSLGNIHHKP-GGGQ

Biotinyl-Tau Peptide (306-336) (Repeat 3 Domain)
H-8028 NEW
Biotinyl-VQIVYKPVDLSKVTSKCGSLGNI-HHKPGGGQ

Tau Peptide (307-321)
H-8086 NEW
QIVYKPVDLSKVTSK

Tau Peptide (323-335)
H-7818 NEW
GDTSPRHLSNVKK

Tau Peptide (337-368) (Repeat 4 Domain)
H-7816 NEW
VEVKSEKLDFKDRVQSKIGSLDNITHVP-GGGN

Tau Peptide (379-408)
H-8172 NEW
RENAKAKTDHGAEIVYKSPVVS-GDTSPRHL

$(Ser(PO_3H_2))^{396.404}$ Tau Peptide (379-408)
H-8164 NEW
RENAKAKTDHGAEIVYK(p)SPVVSGDT(p)SPRH

Lys-Trp-Lys-(Ser(GlcNAc-β-D)^{400})-Tau Peptide (388 - 411)-Lys-Lys(biotinyl) amide
H-7822 NEW
KWKHGAEIVYKSSPVVS(GlcNAc-β-D)GDTSPRHLSNVKK(biotinyl)-NH$_2$

Tau Peptide (472-487)-PEG6-(507-526) (human)
H-7988 NEW
RGAAPPGQKGQANATR-amino-PEG6-propionyl-KSGDRSGYSSPGSPGTPGSR

Tau Peptide (512-525) amide
H-7984 NEW
SGYSSPGSPGTPGS-NH$_2$

PHF6 AND RELATED PEPTIDES

PHF6
H-8098 NEW
VQIVYK

Ac-Tyr-Lys-NH$_2$
H-8124 NEW
Ac-YK-NH$_2$

Ac-Val-Tyr-Lys-NH$_2$
H-8108 NEW
Ac-VYK-NH$_2$

Acetyl-PHF4 amide
H-8106 NEW
Ac-IVYK-NH$_2$

Acetyl-PHF5 amide
H-8104 NEW
Ac-QIVYK-NH$_2$

Acetyl-PHF6 amide
H-8112 NEW
Ac-VQIVYK-NH$_2$

Acetyl-PHF6KE amide
H-8122 NEW
Ac-VQIVYE-NH$_2$

Acetyl-PHF6IV amide
H-8114 NEW
Ac-VQVVYK-NH$_2$

Acetyl-PHF6QV amide
H-6118 NEW
Ac-VVIVYK-NH$_2$

Acetyl-PHF6YA amide
H-8116 NEW
Ac-VQIVAK-NH$_2$

T-Peptide (all-D-PHF6-R9)
H-8126 NEW
Ac-vqivykRRRRRRRRR-NH$_2$

TAU-RELATED PRODUCTS

Enzyme inhibitors and substrates and further peptides and biochemicals for Alzheimer's research are available on our online shop
shop.bachem.com

↳ Areas of Interest
    ↳ Alzheimer's Disease
    ↳ Tau

PIN-1 SUBSTRATES

Ac-Ala-Ala-Ser(PO₃H₂)-Pro-Arg-pNA
L-2155
Ac-AA(p)SP-pNA

Suc-Ala-Glu-Pro-Phe-AMC
I-1750
Suc-AEPF-AMC

Suc-Ala-Glu-Pro-Phe-pNA
L-1635
Suc-AEPF-pNA

H-Trp-Phe-Tyr-Ser(PO₃H₂)-Pro-Arg-AMC
I-1930
WFY(p)SPR-AMC

H-Trp-Phe-Tyr-Ser(PO₃H₂)-Pro-Arg-pNA
L-2075
WFY(p)SPR-pNA

PKA INHIBITORS AND SUBSTRATES

cAMP-Dependent Protein Kinase Inhibitor-α (5-22) amide (human, mouse, rabbit, rat)
H-3222
TTYADFIASGRTGRRNAI-NH₂

H-Arg-Gly-Tyr-Ala-Leu-Gly-OH
M-1105
RGYALG

PKA Inhibitor (6-22) amide
N-2040
TYADFIASGRTGRRNA-NH₂

PKItide
H-3234
IAAGRTGRRQAIHDILVAA

H-Arg-Lys-Ile-Ser-Ala-Ser-Glu-Phe-Asp-Arg-Pro-Leu-Arg-OH (BPDEtide)
H-3216
RKISASEFDRPLR

Calcineurin Substrate
H-2084
DLDVPIPGRFDRRVSVAAE

H-Gly-Arg-Gly-Leu-Ser-Leu-Ser-Arg-OH
H-7405
GRGLSLSR

H1-7
H-1805
RRKASGP

Kemptide trifluoroacetate salt
M-1510
LRRASLG trifluoroacetate salt

Kemptide acetate salt
M-2725
LRRASLG acetate salt

(Trp⁴)-Kemptide (Chocktide)
M-1525
LRRWSLG

(Val⁶,Ala⁷)-Kemptide
M-1515
LRRASVA

MELATONIN

Melatonin
Q-1300

N-Acetyl-2-benzyl-tryptamine (Luzindole)
Q-1885

PERI-VASCULAR CELL

Perivascular cell containing tau protein.
Fluorescence deconvolution light micrograph of a section through a perivascular cell, showing tau protein (red). Tau protein is an abundant neural protein, but aggregations of this protein are thought to play a role in brain disorders such as Alzheimer's disease. Magnification: x200, when printed 10 cm wide.

BACHEM

PIONEERING PARTNER FOR PEPTIDES

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