複合薬物薬理学分野　Division of Medicinal Pharmacology

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◇研究目的

中枢神経系疾患の病態と発症機構に関する薬理学的研究を行うとともに，和漢薬をはじめ，複合成分からなる薬物の薬効に関する計量薬理学的評価，作用本体の追求および分子レベルでの作用機序の解明を目的とした研究を行っている。

◇研究概要

Ⅰ）　中枢神経系疾患の病態と発症機構に関する基礎研究

1) 心理的ストレスで起こる異常行動および薬物応答性変化の神経機構の解析および神経機能修飾因子の役割に関する研究
2) 病態モデルにおける神経伝達物質等の内在性因子や天然薬物成分の脳内動態に関する研究

Ⅱ）　複合薬物及びその成分の中枢作用に関する神経薬理学的研究

1) 脳血管性認知症をはじめとする認知症の病態モデル系における和漢薬および和漢薬成分の抗認知症作用と神経保護作用の評価ならびに作用分子機構の解明
2) 神経保護薬をはじめ，新規リード化合物の開発をめざした伝統薬物・民族薬の薬理作用の探索と作用機序の解析
3) ツメガエル受容体遺伝子発現系を用いた神経伝達受容体の機能解析，薬物作用およびその作用機序に関する電気生理学的研究

Ⅲ）　遺伝子発現を指標とした薬物作用の解明と和漢薬作用に関する研究

1) 慢性脳虚血等のストレスにより発現する脳内遺伝子 vof のクローニングとその生理機能解析
2) 和漢薬および抗うつ薬を利用したうつ病態関連因子のクローニング，発現変化の作用解析，およびうつ病発症機序の分子的解明

◇原著論文

1)　Noh J.S., Park C.H., Kim H.Y., Zhao Q., Yamabe N., Matsumoto K., and Yokozawa T.: Chinese prescription Kangen-karyu prevents dyslipidaemia and oxidative stress in mouse model of type 2 diabetes. J. Pharm. Pharmacol. 63, 111-9; 2011.

Abstract: OBJECTIVES: We have investigated the effects of Kangen-karyu, a Chinese prescription, on the lipid metabolism and oxidative stress in a type 2 diabetes model. **METHODS:** Male db/db mice were divided into three groups: control (vehicle), Kangen-karyu 100 or 200 mg/kg body weight/day orally administered mice. Age-matched non-diabetic m/m mice were used as a normal group. **KEY FINDINGS:** The administration of Kangen-karyu reduced hyperglycaemia and hyperlipidaemia in db/db type 2 diabetic mice through a decline in the serum levels of glucose and lipids, and an improvement of lipoprotein profiles. The increased oxidative stress in db/db mice was attenuated by the administration of

Kangen-karyu through inhibiting the generation of reactive oxygen species and lipid peroxidation. The enhanced hepatic triglyceride and total cholesterol levels of the db/db mice were significantly reduced by Kangen-karyu administration through down-regulation of sterol regulatory element-binding protein-1 and lipogenic enzymes in liver. Furthermore, the expressions of hepatic nuclear factor-kappa B (NF-κB) and cyclooxygenase-2 and inducible nitric oxide synthase protein levels were also augmented in db/db mice. However, Kangen-karyu reduced the expressions of these inflammatory proteins by inhibiting NF-κB activation in db/db type 2 diabetes. **CONCLUSIONS:** This study suggests that Kangen-karyu may improve oxidative stress via the regulation of dyslipidaemia in type 2 diabetes.

2) **Yamada M., Hayashida M., Zhao Q., Shibahara N., Tanaka K., Miyata T., and Matsumoto K.: Ameliorative effects of yokukansan on learning and memory deficits in olfactory bulbectomized mice. J. Ethnopharmacol. 135, 737-46; 2011.**

Abstract: AIM OF THE STUDY: Yokukansan (YKS) is a Japanese traditional herbal medicine and has been used for the treatment of the behavioral and psychological symptoms of dementia (BPSD). The present study aimed to clarify the effects of YKS on learning and memory impairments, and its mechanisms of action in olfactory bulbectomized (OBX) mice, one of the animal models of Alzheimer's disease (AD). **MATERIALS AND METHODS:** OBX or sham-operated ddY mice were treated with YKS or donepezil (DPZ), a reference drug, and their cognitive performances were tested by the modified Y-maze test, novel object recognition test, and fear conditioning test to elucidate the spatial working memory, non-spatial short-term memory, and long-term memory, respectively. After completing the behavioral experiments, the expression level of cholinergic marker proteins and the activity of acetylcholinesterase (AChE) in the brain were analyzed by western blotting and Ellman's method, respectively. **RESULTS:** OBX caused spatial working memory and non-spatial working memory impairments that were reversed by YKS and also by DPZ; however, YKS failed to affect the long-term memory deficits. Amelioration of the spatial working memory by YKS was reversible by scopolamine, a muscarinic receptor antagonist. YKS treatment reversed OBX-induced down-regulation of choline acetyltransferase and muscarinic muscarinic M_1 receptor expression without affecting muscarinic M_3 receptor expression or AChE activity. **CONCLUSION:** These results demonstrate that YKS improves short-term memory deficit caused by OBX and that the effect is at least partly mediated by muscarinic receptor stimulation and the normalization of central cholinergic systems. The present findings also suggest that YKS has a therapeutic effect not only on BPSD, but also on memory impairment of AD.

3) **Zhao Q., Yokozawa T., Tsuneyama T., Tanaka K., Miyata T., Shibahara N., Matsumoto K.: Chotosan-induced improvement of cognitive deficits in senescence-accelerated mouse (SAMP8) involves normalization of angiogenic/neurotrophic factors and neuroplasticity systems in the brain. Chin. Med. 6, 33 (on line); 2011.**

Abstract: BACKGROUND: Chotosan (CTS, Diaoteng San), a Kampo medicine (ie Chinese medicine) formula, is reportedly effective in the treatment of patients with cerebral ischemic insults. This study aims to evaluate the therapeutic potential of CTS in cognitive deficits and investigates the effects and molecular mechanism(s) of CTS on learning and memory deficits and emotional abnormality in an animal aging model, namely 20-week-old senescence-accelerated prone mice (SAMP8), with and without a transient ischemic insult (T2VO). **METHODS:** Age-matched senescence-resistant inbred strain mice (SAMR1) were used as control. SAMP8 received T2VO (T2VO-SAMP8) or sham operation (sham-SAMP8) at day 0. These SAMP8 groups were administered CTS (750 mg/kg, p.o.) or water daily for three weeks from day 3. **RESULTS:** Compared with the control group, both sham-SAMP8 and T2VO-SAMP8 groups exhibited cognitive deficits in the object discrimination and water maze tests and emotional abnormality in the elevated plus maze test. T2VO significantly exacerbated spatial cognitive deficits of SAMP8 elucidated by the water maze test. CTS administration ameliorated the cognitive deficits and emotional abnormality

of sham- and T2VO-SAMP8 groups. Western blotting and immunohistochemical studies revealed a marked decrease in the levels of phosphorylated forms of neuroplasticity-related proteins, N-methyl-D-aspartate receptor 1 (NMDAR1), Ca2+/calmodulin-dependent protein kinase II (CaMKII), cyclic AMP responsive element binding protein (CREB) and brain-derived neurotrophic factor (BDNF) in the frontal cortices of sham-SAMP8 and T2VO-SAMP8. Moreover, these animal groups showed significantly reduced levels of vasculogenesis/angiogenesis factors, vascular endothelial growth factor (VEGF), VEGF receptor type 2 (VEGFR2), platelet-derived growth factor-A (PDGF-A) and PDGF receptor α (PDGFRα). CTS treatment reversed the expression levels of these factors down-regulated in the brains of sham- and T2VO-SAMP8. **CONCLUSION:** Recovery of impaired neuroplasticity system and VEGF/PDGF systems may play a role in the ameliorative effects of CTS on cognitive dysfunction caused by aging and ischemic insult.

4) Utsintong M., Rojsanga P., Ho K.Y., Talley T.T., Olson A.J., Matsumoto K., and Vajragupta O.: Virtual Screening against Acetylcholine Binding Protein. Journal of Biomolecular Screening. PMID: 21956172; 2011.

Abstract: The nicotinic acetylcholine receptors (nAChRs) are a member of the ligand-gated ion channel family and play a key role in the transfer of information across neurological networks. The X-ray crystal structure of agonist-bound α(7) acetylcholine binding protein (AChBP) has been recognized as the most appropriate template to model the ligand-binding domain of nAChR for studying the molecular mechanism of the receptor-ligand interactions.Virtual screening of the National Cancer Institute diversity set, a library of 1990 compounds with nonredundant pharmacophore profiles, using AutoDock against AChBPs revealed 51 potential candidates. In vitro radioligand competition assays using [(3)H] epibatidine against the AChBPs from the freshwater snails, Lymnaea stagnalis, and from the marine species, Aplysia californica and the mutant (AcY55W), revealed seven compounds from the list of candidates that had micromolar to nanomolar affinities for the AChBPs. Further investigation on α(7)nAChR expressing in Xenopus oocytes and on the recombinant receptors with fluorescence resonance energy transfer (FRET)-based calcium sensor expressing in HEK cells showed that seven compounds were antagonists of α(7)nAChR, only one compound (NSC34352) demonstrated partial agonistic effect at low dose (10 μM), and two compounds (NSC36369 and NSC34352) were selective antagonists on α(7)nAchR with moderate potency. These hits serve as novel templates/scaffolds for development of more potent and specific in the AChR systems.

5) Zhao Q., Matsumoto K., Tsuneyama K., Tanaka K., Li F., Shibahara N., Miyata T., and Yokozawa T.: Diabetes-induced central cholinergic neuronal loss and cognitive deficit are attenuated by tacrine and a Chinese herbal prescription, kangen-karyu: elucidation in type 2 diabetes db/db mice. J. Pharmacol. Sci. 117, 230-42; 2011.

Abstract: We investigated the effect of kangen-karyu (KK), a Chinese herbal prescription, on cognitive deficits and central cholinergic systems of type 2 diabetic db/db mice. Seven-week-old db/db (Y-db/db) mice received daily administration of test drugs during an experimental period of 12 weeks. At 18 weeks of age (O-db/db), the animals underwent the water maze test. Compared with age-matched control strain mice (O-m/m), vehicle-treated O-db/db mice showed impaired learning and memory performance. KK (100 - 200 mg/kg per day) and the reference drug tacrine (THA: 2.5 mg/kg per day) ameliorated the performance of O-db/db mice without affecting their serum glucose level. O-db/db mice had lower levels of brain-derived neurotrophic factor (BDNF) mRNA and its protein in the brain than O-m/m mice. Expression levels of central cholinergic marker proteins in the hippocampus and the number of cholinergic cells in the medial septum and basal forebrain were also significantly lower in O-db/db than in O-m/m mice, whereas no significant differences in the expression levels of these factors and the cell number were found between Y-m/m and Y-db/db mice. KK and THA treatment significantly reversed the

down-regulated levels of cholinergic markers, choline acetyltransferase-positive cell number, and BDNF expression in db/db mice. These findings suggest that KK as well as THA prevents diabetes-induced cognitive deficits by attenuating dysfunction of central cholinergic systems.

6) **Park C.H., Noh J.H., Kim J.H., Tanaka T., Zhao Q., Matsumoto K., Shibahara N., and Yokozawa T.: Evaluation of morroniside, iridoid glycoside from Corni Fructus, on diabetes-induced alterations such as oxidative stress, inflammation, and apoptosis in the liver of type 2 diabetic db/db mice. Biol. Pharm. Bull. 34, 1559-65; 2011.**

Abstract: The present study was conducted to examine whether morroniside has an ameliorative effect on diabetes-induced alterations such as oxidative stress, inflammation, and apoptosis in the liver of type 2 diabetic *db*/*db* mice. Morroniside (20 or 100 mg/kg body weight/d, *per os* (*p.o.*)) was administered every day for 8 weeks to *db*/*db* mice, and its effect was compared with vehicle-treated *db*/*db* and *m*/*m* mice. The administration of morroniside decreased the elevated serum glucose concentration in *db*/*db* mice, and reduced the increased oxidative biomarkers including the generation of reactive oxygen species and lipid peroxidation in the liver. The *db*/*db* mice exhibited the up-regulation of nicotinamide adenine dinucleotide phosphate oxidase subunits, NF-E2-related factor 2 (Nrf2), heme oxygenase-1, nuclear factor-kappa B, cyclooxygenase-2, inducible nitric oxide synthase, monocyte chemotactic protein-1, and intracellular adhesion molecule-1 levels in the liver; however, morroniside treatment significantly reduced those expressions. Moreover, the augmented expressions of apoptosis-related proteins, Bax and cytochrome *c*, were down-regulated by morroniside administration. Hematoxylin–eosin staining showed that the increased hepatocellular damage in the liver of *db*/*db* mice improved on morroniside administration. Taking these into consideration, our findings support the therapeutic evidence for morroniside ameliorating the development of diabetic hepatic complications *via* regulating oxidative stress, inflammation, and apoptosis.

7) **Sukma M., Tohda M., Suksamran S., and Tantisira B.: γ-Mangostin increases serotonin 2A/2C, muscarinic, histamine and bradykinin receptor mRNA expression. J. Ethnopharmacol. 135, 450-4; 2011.**

Abstract: AIM OF THE STUDY: γ-Mangostin is a xanthone found in the fruit hulls of Garcinia mangostana L., which have long been used in Southeast Asia as a traditional medicine for the treatment of abdominal pain, dysentery, wound infections, fever and convulsions. Recent studies have revealed that γ-mangostin exhibits a variety of pharmacological activities, including serotonin 2 (5-HT(2)) receptor antagonism, anti-inflammatory effects and analgesic effects. To explore the mechanism of γ-mangostin responsible for these pharmacological activities, especially its effects on some related receptors, we investigated the effects of γ-mangostin on 5-HT(2), histamine (H(1)) and bradykinin (BK(2)) receptor gene expression in neuroblastoma (NG 108-15) cells in vitro. Additionally, to extend the study of the pharmacological properties, we examined the effect of γ-mangostin on the muscarinic (M(4)) receptor. **MATERIALS AND METHODS:** NG 108-15 cells were cultured in vitro and treated with γ-mangostin or a 5-HT(2) receptor antagonist (either imipramine or ketanserin). Then, the levels of mRNA for 5-HT(2A/2C) receptors were evaluated by semi-quantitative RT-PCR. The preventive effect of serotonin on the enhancement effects was also revealed. Additionally, the effects of γ-mangostin on the muscarinic, histamine and bradykinin receptors were determined. **RESULTS:** Chronic application of γ-mangostin at a concentration of 0.1 μM induced a significant increase in the level of 5-HT(2A/2C) receptor mRNA. These effects were prevented by serotonin. Moreover, γ-mangostin up-regulated the M(4), H(1) and BK(2) receptors. **CONCLUSION:** The ability of γ-mangostin to enhance the expression of 5-HT(2A/2C), muscarinic, histamine and bradykinin receptor mRNA suggests that this compound has antagonistic effects. These pharmacological properties may partly account for the benefits of using mangosteen in the treatment of inflammation, pain and neuropsychiatric symptoms.

8) **Hang P.T.N., Phuong D.T., Phuong N.T., Xuyen P.T., Ha Q.T.L., Khoi N.M., and Tohda M.: Establishment of the cell culture model to study anti-depressant effect on primary culture cortical cells and NG108-15 cell line. J. Medicinal Materials-Hanoi 16, 277-81; 2011.**

Abstract: In vivo model are ideal for studying the effects of drugs on all of living subjects. However, they are limited in being able to explore cellular and molecular mechanisms of drugs. Culture models have improved these disadvantages. In this study, we establish the cell culture model to study anti-depression effects of imipramine, an antidepressant and hypericin that extracted from *Hypericum perforatum* L. acclimatized Vietnam using BNIP-3 gene as the depressive factor. The result shows that both imipramine and hypericin significantly stimulated the expression level of BNIP-3 gene in the primary culture cortical cell and NG108-15 cell line without effect on β-actin expression level. In conclusion, the cell culture was established to evaluate screening/molecular mechanism of antidepression effect. Furthermore, *Hypericum perforatum* L. acclimatized Vietnam is the potential candidate for studying the antidepressant in Vietnam.

◇総説

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◇学会報告 （*: 特別講演，シンポジウム，ワークショップ等）

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4) 稲田千香子，山田麻利名，趙琦，松本欣三：Anti-neurodegenerative effect of tacrine on N-methyl-D-aspartate(NMDA)-induced neuronal cell death in organotypic hippocampal slice culture. 第 84 回日本薬理学会年会，2011, 3/22-24，横浜.
5) 山邉典子，朴鑽欽，大和田滋，岡本拓也，松本欣三，柴原直利，横澤隆子：慢性腎臓病に対する漢方方剤の有効性，日本薬学会第 131 年会，2011, 3/28-31，静岡.
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* 8) 東田道久:和漢薬の理論を基盤にした各種うつ病発症機序の分類・解明と作用薬の開発をめざして．第 28 回和漢医薬学会，2011, 8/27-28，富山.
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* 11) 松本欣三:生活習慣病・加齢からくる認知症と和漢薬. 生体機能と創薬シンポジウム 2011,

2011, 9/1-2，東京.

12) 東田道久，ミンマライラック サリン，小林直史，松本欣三：補中益気湯の抗うつ及び消化器系活性化作用に関する検討 : 抗うつ薬との違いについて．第 62 回日本薬理学会北部会，2011, 9/29-30，仙台.

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* 14) 松本欣三，趙琦，常山幸一，田中謙，李峰，宮田健，横澤隆子：加齢・糖尿病に起因する認知行動障害と漢方薬による実験的予防・治療．第 11 回日本臨床中医薬学会学術大会シンポジウム，2011, 11/12，東京.

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16) 松本欣三，山田麻利名，林田未希，柳雅樹，趙琦，田中謙，宮田健：嗅球摘出マウスの学習記憶障害に対する抑肝散の効果 : 中枢コリン神経系を介した改善作用．第 39 回薬物活性シンポジウム，2011, 11/21-22，福岡.

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◇その他

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◇共同研究

研究所内

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◇研究費取得状況

1) 文部科学省科学研究費, 基盤研究 A（代表：東田道久）「うつ病のすべてがわかる和漢薬：発病機序の分子的解明から新規抗うつ薬開発まで」560 万 (3/5 年目)
2) 学長裁量経費教育研究費「持続発展型国際交流と人材育成をめざした「拠点大学方式による学術交流事業」活用プログラム」（代表：松本欣三）80 万
3) 学長裁量経費教育研究費「第 13 回国際伝統医薬シンポジウム「伝統医薬・天然薬物を駆使した問題疾患の予防・治療―臨床・前臨床研究の国際的最前線」（松本欣三）200 万
4) 重点配分経費教育研究費 一般横澤隆子（富山大）（分担：松本欣三）76.3 万
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