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)　心理的ストレス反応に関わる神経機構、神経機能修飾因子とその作用分子機構の解析
2)　病態モデルにおける神経伝達物質、一酸化窒素の脳内動態とそれに対する薬物作用の解析

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)　脳血管性痴呆病態モデル系における和漢薬および和漢薬成分の抗痴呆作用と神経保護作用の評価
2)　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)　受容体遺伝子発現系を用いた薬物作用と作用機序に関する電気生理学的解析

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)　慢性脳虚血により発現する脳内遺伝子のクローニングとその機能解析
2)　うつ病態関連脳内遺伝子の発現変化と抗うつ薬・和漢薬の作用解析

◇原著論文

1) **Hussein G., Nakamura M., Zhao Q., Iguchi T., Goto H., Sankawa U., and Watanabe H.: Antihypertensive and neuroprotective effects of astaxanthin in experimental animals. *Biol. Pharm. Bull.*, 28: 47-52, 2005.**

Abstract: Astaxanthin is a natural antioxidant carotenoid that occurs in a wide variety of living organisms. We investigated, for the first time, antihypertensive effects of astaxanthin (ASX-O) in spontaneously hypertensive rats (SHR). Oral administration of ASX-O for 14 d induced a significant reduction in the arterial blood pressure (BP) in SHR but not in normotensive Wistar Kyoto (WKY) strain. The long-term administration of ASX-O (50 mg/kg) for 5 weeks in stroke prone SHR (SHR-SP) induced a significant reduction in the BP. It also delayed the incidence of stroke in the SHR-SP. To investigate the action mechanism of ASX-O, the effects on PGF(2alpha)-induced contractions of rat aorta treated with N(G)-nitro-l-arginine methyl ester (L-NAME) were studied in vitro. ASX-O (1 to 10 μM) induced vasorelaxation mediated by nitric oxide (NO). The results suggest that the antihypertensive effect of ASX-O may be due to a NO-related mechanism. ASX-O also showed significant neuroprotective effects in ischemic mice, presumably due to its antioxidant potential. Pretreatment of the mice with ASX-O significantly shortened the latency of escaping onto the platform in the Morris water maze learning performance test. In conclusion, these results indicate that astaxanthin can exert beneficial effects in protection against hypertension and stroke and in improving memory in vascular dementia.

2) **Mahakunakorn P., Tohda M., Murakami Y., Watanabe H., and Matsumoto K.: Effects of Choto-san and Its Related Constituents on Endogenous Antioxidant Systems. *Biol. Pharm. Bull.*, 28: 53-57, 2005.**

Abstract: We previously reported that Choto-san acts as an antioxidant and cytoprotective agents against H_2O_2-induced oxidative damage in NG108-15 cells, and the effect is due at least partly to the phenolic compounds. To further investigate the detail mechanisms of this cytoprotection, effects of Choto-san and related compounds on enzyme activities of antioxidant systems were examined. Choto-san (5-100 μg/ml) and Chotoko (5-100 μg/ml) stimulated the activity of superoxide dismutase (SOD), catalase and glutathione peroxidase (GPX). These also increased the level of glutathione. Although Choto-san without Chotoko (w/o CKO) did not show the effects on SOD and catalase, GPX activity and glutathion content also, but weakly, stimulated by w/o CKO. The effects of phenolic compounds, epicatechin, caffeic acid and quercetin were also investigated. Epicatechin stimulated catalase, GPX and glutathion content, but not SOD. On the other hand, caffeic acid stimulated SOD activity but had no effects on others. Quercetin stimulated all, although intensities were different among. These results suggest that simultaneous induction of cellular antioxidant defense systems by Choto-sam and its related constituents may be an important mechanisms underlying the protective effects of Choto-san on ischemia-induced neuronal cells injury, and the characteristics of the stimulative effects of phenolic compounds were depend on enzymes.

3) **Hayashi H., Tohda M., Watanabe H., Murakami Y., and Matsumoto K.: The Effects of Choto-san on the mRNA Expression of Alzheimer's Disease Related Factors in the Permanent Ischemic Rat Brain. *Biol. Pharm. Bull.*, 28: 744-746, 2005.**

Abstract: Choto-san is a Kampo medicine that has been used clinically for the treatment of dementia. We measured the mRNA expressions of some factors related to Alzheimer's disease in a dementia model rat brain. The expressions of beta-amyloid precursor protein, gamma-secretase, alpha7 nicotinic acetylcholine receptor, neprilysin, and insulin degrading enzyme (IDE) were significantly increased on day 4 after permanent occlusion of the bilateral common carotid arteries (2VO). Choto-san inhibited the

enhancement of IDE expression caused by 2VO, although it failed to show any effects on the expressions of the other molecules. These results suggest that Choto-san may produce a state in which it is not necessary to induce IDE expression to demonstrate the anti-dementia effects.

4) **Hussein G., Goto H., Oda S., Iguchi T., Sankawa U., Matsumoto K., and Watanabe H.: Antihypertensive Potential and Mechanism of Action of Astaxanthin: II. Vascular Reactivity and Hemorheology in Spontaneously Hypertensive Rats. *Biol. Pharm. Bull.*, 28: 967-971, 2005.**

Abstract: The current study was designed to determine the effects of a dietary astaxanthin (ASX-O) on vascular reactivity in spontaneously hypertensive rats (SHR), in order to verify its antihypertensive action mechanism. We evaluated contractions induced by phenylephrine (Phe), angiotensin II (Ang II) and the xanthine/xanthine oxidase (Xan/XOD) system, and relaxations induced by sodium nitroprusside (SNP) as well as endothelium-dependent relaxations mediated by acetylcholine (ACh) in thoracic aorta of the SHR, with and without ASX-O intervention. We also investigated the effects of ASX-O on blood rheology using a microchannel array system. In this study, ASX-O showed a significant modulatory effect on nitric oxide (NO)-induced vasorelaxation by the NO-donor SNP ($p<0.05$). However, it did not show significant effects in restoring the impaired endothelium-dependent relaxation to ACh in the SHR. On the other hand, the constrictive effects by Phe, Ang II and Xan/XOD were ameliorated by ASX-O ($p<0.05$). ASX-O also demonstrated significant hemorheological effect by decreasing the microchannel transit time of whole blood. In conclusion, the results suggest that ASX-O may act in modulating the blood fluidity in hypertension, and that the antihypertensive effects of ASX-O may be exerted through mechanisms including normalization of the sensitivity of the adrenoceptor sympathetic pathway, particularly [alpha]-adrenoceptors, and by restoration of the vascular tone through attenuation of the Ang II- and reactive oxygen species (ROS)-induced vasoconstriction.

5) **Murakami Y., Zhao Q., Harada K., Tohda M., Watanabe H., and Matsumoto K.: Choto-san, a Kampo formula, improves chronic cerebral hypoperfusion-induced spatial learning deficit via stimulation of muscarinic M_1 receptor. *Pharmacol, Biochem. Behav.*, 81: 616-625, 2005.**

Abstract: A recent double-blind and placebo-controlled study demonstrated a beneficial effect of Choto-san, a Kampo (traditional medicine of Japan) formula, on cognitive impairment in patients with vascular dementia. However, the neuronal mechanism underlying the therapeutic effects of this formula remains to be clarified. Using a chronic cerebral hypoperfusion model, we investigated the effect of Choto-san on cognitive dysfunction in mice to clarify its mechanism of actions. Chronic cerebral hypoperfusion was induced by permanent occlusion of both the common carotid arteries (2VO). Choto-san and *Uncaria*, a major constituent of Choto-san, caused an improvement in 2VO-induced learning deficits, whereas *Uncaria*-free Choto-san did not. The effects of Choto-san and *Uncaria* were blocked by pirenzepine, a selective muscarinic M_1 antagonist. In a tube-dominance test, 2VO induced an increased rates of assertive behavior in mice. 2VO mice administered Choto-san showed significantly reduced rates of assertive behavior compared to vehicle-treated controls, whereas *Uncaria*-free Choto-san and *Uncaria* had little effect on 2VO-induced assertive behavior. 2VO caused a significant decrease in the level of acetylcholine (ACh) contents in the brain, and the daily administration of Choto-san or *Uncaria* raised the ACh level to that in the sham-operated controls. These results suggest that Choto-san has an ameliorating effect on the spatial memory deficit caused by chronic hypoperfusion, and that the effect is mainly attributable to *Uncaria*. Moreover, it was suggested that the effects of Choto-san and *Uncaria* are at least partly mediated by stimulation of the muscarinic M_1 receptor.

6) **Nguyen T.T.H., Murakami Y., Tohda M., Watanabe H., and Matsumoto K.: Social isolation stress-induced oxidative damage in mouse brain and its modulation by majonoside-R2, a Vietnamese ginseng saponin. *Biol. Pharm. Bull.*, 28: 1389-1393, 2005.**

Abstract: Stressors with a physical factor such as immobilization, electric foot shock, cold swim, etc., have been shown to produce oxidative damage to membrane lipids in the brain. In this study, we investigated the effect of protracted social isolation stress on lipid peroxidation activity in the mouse brain and elucidated the protective effect of majonoside-R2, a major saponin component of Vietnamese ginseng, in mice exposed to social isolation stress. Thiobarbituric acid reactive substance (TBARS), one of the end products of lipid peroxidation reaction, levels were increased in the brains of mice subjected to 6-8 weeks of mice to social isolation stress. Measurements of NO metabolites (NO_x^-) also revealed a significant increase of NO production in the brains of socially isolated mice. Moreover, the depletion of brain glutathione content, an endogenous antioxidant, in socially isolated animals occurred in association with the rise in lipid peroxidation. The intraperitoneal administration of majonoside-R2 (10–50 mg/kg) had no effect on TBARS, NO, or glutathione levels in the brains of group-housed control mice but it significantly suppressed the increase in TBARS and NO levels and the decrease in glutathione levels caused by social isolation stress. These results suggest that subjecting mice to 6-8 weeks of social isolation stress produces oxidative damage in the brain partly via enhancement of NO production, and that majonoside-R2 exerts a protective effect by modulating NO and glutathione systems in the brain.

7) **Matsumoto K., Morishige R., Murakami Y., Tohda M., Takayama H., Sakakibara I., and Watanabe H.: Suppressive effects of isorhynchophylline on 5-HT_{2a} receptor function in the brain: behavioural and electrophysiological elucidation. *Eur. J. Pharmacol.*, 517: 191-199, 2005.**

Abstract: Isorhynchophylline (IRHY) is a major oxindole alkaloid found in *Uncaria* species which have long been used in traditional Chinese medicine. Here, we investigated the effects of IRHY and IRHY-related alkaloids on 5-HT receptor-mediated behavioural responses in mice and 5-HT-evoked current responses in *Xenopus* oocytes expressing 5-HT_{2A} or 5-HT_{2C} receptors. IRHY dose-dependently inhibited 5-HT_{2A} receptor-mediated head-twitch but not 5-HT_{1A} receptor-mediated head-weaving responses evoked by 5-methoxy-N,N-dimethyltryptamine. Pretreatment with reserpine, a monoamine-depleting agent, enhanced the head-twitching, but did not influence the effect of IRHY on the behavioural response. Isocorynoxeine (ICOX), an IRHY-related alkaloid in which the configuration of the oxindole moiety is the same as in IRHY, also reduced the head-twitch response in reserpinized mice over the same dose range as IRHY, while both rhynchophylline and corynoxeine, stereoisomers of IRHY and ICOX, did not. None of the alkaloids tested had an effect on meta-chlorophenylpiperazine-induced hypolocomotion, a 5-HT_{2C} receptor-mediated behavioural response. In experiments *in vitro*, IRHY and ICOX dose-dependently and competitively inhibited 5-HT-evoked currents in *Xenopus* oocytes expressing 5-HT_{2A} receptors, but had less of a suppressive effect on those in oocytes expressing 5-HT_{2C} receptors. These results indicate that IRHY and ICOX preferentially suppress 5-HT_{2A} receptor function in the brain probably via a competitive antagonism at 5-HT_{2A} receptor sites and that the configuration of the oxindole moiety of IRHY is essential for their antagonistic activity at the 5-HT_{2A} receptor.

8) **Li X., Matsumoto K., Murakami Y., Tezuka Y., Wu Y., and Kadota S.: Neuroprotective effects of *Polygonum multiflorum* on nigrostriatal dopaminergic degeneration induced by paraquat and maneb in mice. *Pharmacol. Biochem. Behav.*, 82:345-352, 2005.**

Abstract: The neuroprotective effects of *Polygonum multiflorum* extract (PME) and its two fractions, ethanol-soluble PME (PME-I) and -insoluble PME (PME-II) fractions, on the degeneration of nigrostriatal dopaminergic neurons induced by a combination of paraquat and maneb (PQMB) were investigated in male C57BL/6 mice. The mice were treated twice a week over 6 weeks with intraperitoneal injections of PQMB. This combination caused a reduction of spontaneous locomotor activity, motor incoordination, and declines of dopamine levels in the striatum and tyrosine hydroxylase-positive neurons in the substantia nigra. Administration of PME and PME-I once daily for a

total of 46 days during the 6-week period of PQMB treatment and an 8-day period following the termination of PQMB treatment significantly attenuated the impairment of behavioral performance and the decrease in striatal dopamine levels and substantia nigral tyrosine hydroxylase-positive neurons in the PQMB-treated animals, whereas the administration of PME-II had no effect on these behavioral, neurochemical and histological indices. The present findings suggest that PME has a beneficial influence on parkinsonism induced by PQMB and that the effects of PME are attributable to some substance(s) included in the ethanol-soluble fraction of PME.

9) **Zhao Q., Murakami Y., Tohda M., Watanabe H., and Matsumoto K.: Preventive effect of chotosan, a Kampo medicine, on transient ischemia is mediated by stimulation of muscarinic M_1 but not nicotinic receptor. *Biol. Pharm. Bull.*, 28: 1873-1878, 2005.**

Abstract: We have previously shown using a water maze task that transient 2 vessel occlusion (T2VO) induced learning deficit in mice and that the deficit was prevented by pre-treatment of mice with chotosan, a Kampo prescription. In this study, we investigated the mechanism underlying the preventive effect of chotosan on T2VO-induced learning deficit. Chotosan administration 1 hour before T2VO operation prevented leaning impairment. The extract of *Uncaria*, a major constituent of chotosan, also had a protective effect on learning impairment in T2VO mice, whereas *Uncaria*-free chotosan did not had no beneficial effect on maze performance of T2VO mice. The ameliorative effect of chotosan was blocked by pirenzepine, a muscarinic M_1 antagonist, but not by mecamylamine, a nicotinic receptor antagonist. Acetylcholine (ACh) content in the hippocampus of T2VO mice was significantly lower than that in the hippocampus of sham-operated control mice. Chotosan and *Uncaria* administration attenuated T2VO-induced reduction of ACh levels in the brain. These results suggest that the preventive effect of chotosan on transient ischemia-induced learning impairment is mainly attributable to the effect of *Uncaria* and that the ameliorative effect is mediated by stimulation of muscarinic M_1 receptor.

10) **Sukma M., Tohda M., Watanabe H., and Matsumoto K.: The mRNA expression differences of RNA editing enzymes in differentiated and undifferentiated NG108-15 cells. *J. Pharmcol. Sci.*, 98: 467-470, 2005.**

Abstract: We previously reported that NG108-15 cells contain intrinsic serotonin 2C receptor (5-HT2CR). The effects of imipramine, a 5-HT2CR antagonist, on cell growth, cell viability, and the 5-HT2CR mRNA level were investigated in this study. Repeated treatment with imipramine at concentrations of 1 – 10 μM for 5 days inhibited cell growth in a concentration-dependent manner without affecting cell viability. In addition, the level of 5-HT2CR mRNA was elevated. At 30 μM, imipramine significantly reduced cell viability. Our findings suggest that the effect of imipramine on neuronal growth may be related to its effects on 5-HT2CR.

11) **Nguyen M.T.T., Aware S., Tezuka Y., Shi L., Zadi F.H., Ueda J., Tran Q.L., Murakami Y., Matsumoto K., and Kadota S.: Hypouricemic effect of acacetin and 4,5-O-dicaffeoylquinic acid methyl ester on serum uric acid levels in potassium oxonate-pretreated rats. *Biol. Pharm. Bull.*, 28: 2231-2234, 2005.**

◇総　説

1) **Matsumoto K., Pinna G., Puia G., Guidotti A., and Costa E.: Social isolation stress-induced aggression in mice: a model to study the pharmacology of neurosteroidogenesis. *Stress*, 8(2): 85-93, 2005.**

2) 松本欣三, A. Guidotti, E. Costa: ストレスと睡眠・情動障害：神経ステロイド・allopregnanolone 系の関与, 日本薬理学雑誌, 126(2): 107-112, 2005.

◇学会報告 （*: 特別講演、シンポジウム、ワークショップ等）

1) Sumanont Y. Murakami Y., Tohda M., Vajragupta O., Watanabe H., Matsumoto K.: Manganese complexes of curcumin and diacetylcurcumin suppress NO production in the rat hippocampus: a microdialysis study. 第 78 回日本薬理学会年会, 2005, 3/22-24, 横浜.

2) 林寿枝, 東田道久, Saelim N., 村上孝寿, 松本欣三: アミロイド前駆蛋白および関連因子 mRNA 発現制御による初代培養細胞の状態変化. 第 78 回日本薬理学会年会, 2005, 3/22-24, 横浜.

3) Hussein G., Goto H., Oda S., Iguchi T., Sankawa U., Matsumoto K., Watanabe H: Antihypertensive action mechanisms of astaxanthin in SHR. 第 78 回日本薬理学会年会, 2005, 3/22-24, 横浜.

4) Hussein G., Goto H., Nakamura M., Zhao Q., Iguchi T., Oda S., Sankawa U., Matsumoto K., Watanabe H: Antihypertensive and neuroprotective potentials of astaxanthin. 日本薬学会第 125 年会, 2005, 3/29-31, 東京.

5) Hussein G., Goto H., Oda S., Iguchi T., Sankawa U., Matsumoto K., Watanabe H: Antihypertensive and neuroprotective potentials of astaxanthin in experimental animals. The 14th International Symposium on Carotenoids, 2005, 7/17-22, Edinburgh, UK.

6) Hussein G., Goto H., Oda S., Iguchi T., Sankawa U., Matsumoto K., Watanabe H: Mechanisms of the antihypertensive effects of astaxanthin in spontaneously hypertensive rats. The 14th International Symposium on Carotenoids, 2005, 7/17-22, Edinburgh, UK.

7) Li X., Murakami Y., Matsumoto K., Tezuka Y., Wu Y., Kadota S.: Protective effect of *Polygonum multiflorumon* nigrostriatal dopamine neuron damage caused by paraquat and manbe in mice. 第 22 回和漢医薬学会大会, 2005, 8/20-21, 東京.

8) 趙琦, 村上孝寿, 東田道久, 榊原巌, 松本欣三: 釣藤鈎の抗高血圧作用及び一過性脳虚血誘発空間認知障害に対する予防作用：華鈎藤と鈎藤の薬理作用の差異. 第 22 回和漢医薬学会大会, 2005, 8/20-21, 東京.

9) 村上孝寿, 李霞, 松本欣三, 手塚康弘, 門田重利: パーキンソン病モデル動物の黒質線条体ドパミン神経系障害に対する何首烏の保護効果. 第 56 回日本薬理学会北部会, 2005, 10/4, 新潟.

10) Hussein G., Goto H., Oda S., Iguchi T., Sankawa U., Matsumoto K., Watanabe H: Antihypertensive potential and action mechanisms of astaxanthin in spontaneously hypertensive rats. 第 1 回アスタキサンチン研究会, 2005, 11/8, 東京.

11) 村上孝寿, 趙琦, 東田道久, 渡邉裕司, 松本欣三：脳虚血誘発のマウスの空間認知障害に対する釣藤散の効果. 第 16 回天然薬物の開発と応用シンポジウム, 2005, 11/10-11, 東京.

12) Tohda M., Matsumoto K.: Up-regulation of amyloid precursor protein mRNA expression and its putative role in neuron. The 3rd Takeda science foundation symposium on pharmasciences, 2005, 12/5-7, 東京.

◇招待講演

1) 松本欣三: 釣藤散―その抗痴呆効果の薬理学的裏付け―.平成 16 年度北陸調剤情報セミナー, 2005, 2/27, 金沢.

2) Tohda M.: Identification of the Intrinsic Factors Involving the Generation and/or Regulation of Psychological Disorders by Wakan-yaku, 2005/7/14-15, Toyama.

3) 松本欣三: 漢方薬成分によるストレスの緩和. ストレス社会が求める癒し系食品の開発. 日本食品機械研究会, 2005, 7/20?, 東京.

4) 松本欣三: 脳血管性痴呆モデル系における釣藤散の薬理作用. 日本東洋医学会北陸支部石

川部会「湯本求眞記念学術講演会」, 2005, 9/24, 金沢.

5) 松本欣三: 脳血管性認知症病態モデル系における釣藤散の薬理作用とその機序. 大学勤務医のための漢方医学セミナー, 2005, 11/19, 岡山.

6) 松本欣三: 脳血管性認知症と和漢薬：病態モデル系における釣藤散の作用を中心として. 富山漢方会, 2005, 11/25, 富山.

◇共同研究

学内

1) 門田重利：富山大学和漢医薬学総合研究所,「何首烏のドパミン神経損傷に対する保護作用」, 2004, 5～

2) 後藤博三：富山大学和漢医薬学総合研究所,「Astaxanthin 含有ヘマトコッカス藻抽出物の薬理作用に関する研究」, 2001, 4/1～

国内

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2) 三川　潮： 富山県伝統医薬センター,「Astaxanthin 含有ヘマトコッカス藻抽出物の薬理作用に関する研究」, 2001, 4/1～

海外

1) グエン・チー・スー・フォン：ベトナム薬物研究所,「ベトナム人参の薬理作用の研究」, 1994, 4 -

2) Erminio Costa, Alessandro Guidotti：米国イリノイ州立大学シカゴ校精神医学研究所,「ストレス病態における神経活性ステロイドの役割」, 1997, 4-

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4) Li Song: 瀋陽薬科大学,「ストレス誘発の情動障害及び学習記憶障害に関する神経薬理学的研究」, 2005, 2/16-

◇研究費取得状況

1) 文部科学省科学研究費、萌芽的研究（代表：松本欣三）「攻撃行動制御因子の単離同定と神経生理機能に関する研究」 210 万 (1/2 年目)

2) 文部科学省科学研究費、基盤研究 B（代表：松本欣三）「漢方薬の薬効を利用した脳血管性痴呆治療標的分子の探索・同定とその生理機能解析」 850 万 (1/3 年目)

3) 文部科学省科学研究費、基盤研究 B（代表：東田道久）「和漢薬をプローブとした生体内機能分子の同定と生理機能・病態変化の解析」 1040 万 (1/3 年目)

4) 文部科学省科学研究費、２１世紀中核的研究拠点形成プログラム（分担：松本欣三）「東洋の知に立脚した個の医療の創生」 200 万

5) 富山県、和漢薬・バイオテクノロジー研究費、（代表：松本欣三）「神経精神性障害に対する薬理学的及び分子生物学的解析と応用に関する基礎研究」 70 万

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Dr. Ghazi Hussein (Assistant of Khartoum University, 2001, 4/1-)

本学学術交流事業

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井口知美：Astaxanthin 含有ヘマトコッカス藻抽出物の高血圧及び脳虚血性学習障害に対する効果

平尾顕三：一過性脳虚血誘発の空間学習認知障害に対する釣藤散の保護効果とその機序