

日中笹川医学奨学金制度 第47期＜共同研究コース＞ 研究者集会



主 催：公益財団法人 日中医学協会
笹川医学奨学金進修生同学会

開催日：2026年9月16日

会 場：日本財団ビル大会議室

Program

研究者集会（14：30～18：00 日本財団ビル2階 大会議室）

開会宣言 小川 秀興 日中医学協会会長

挨拶 跡見 裕 日中医学協会理事長

胡 秀英 笹川医学奨学金進修生同学会常務理事

祝 辞 苗 允 中華人民共和国駐日本国大使館科学技術部参事官

尾形 武寿 日本財団会長

研究発表

座 長 林崎 良英 日中医学協会理事、

日中医学（日中医学協会-日本財団）協力委員会委員長

発表者 9組（第47期研究者・日本側共同研究者）

総 評

懇 親 会（18：00～19：00 同ビル2階 第1～4会議室）

挨拶 喜多 悦子 笹川保健財団会長

乾杯挨拶 新井 一 日中医学協会業務執行理事

〈共同研究コース〉概要



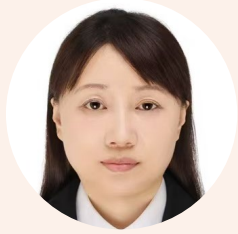
中国の医療関係者が、日本の医療関係者を行う共同研究活動を支援する。

助成期間	最長6カ月間（4月1日以降に日本に入国）
招請者数	年間10チームまたは個人
研究機関	日本国内の大学、病院、研究所等
奨学金	①日本滞在中の生活費（宿舎費を含む）・・・月額25万円（研究者に支給） ②研究費・・・・・・・・・・・・・・・・・・・・・月額10万円（受入機関に支給） ※日本に滞在している期間に対して支給する。 一時出国等により日本不在の期間が1か月を超える場合、当該月は支給しない。
研究者の義務	① 日本滞在期間中は日本国法令を遵守すること ② 本制度応募時に提出した「誓約書・保証書」の内容を遵守すること ③ 世界の著名な専門学術誌に研究成果を英文論文で発表すること ④ 発表論文に日中笹川医学奨学金（Japan China Sasakawa Medical Fellowship）助成を受けたことを記載すること ⑤ 発表論文を日中医学協会と笹川医学奨学金進修生同学会に提出すること（発表論文は本制度成果資料として保存） ⑥ 9月に日本で開催する〈共同研究コース〉研究者集会において日本側共同研究者と共に共同研究の内容を発表すること ⑦ 日本入国後、日本側共同研究者と「共同研究日本滞在計画書」を提出すること ⑧ 「共同研究日本滞在計画書」の内容に変更が生じた場合は、即時、日中医学協会に電子メール等文書で通知すること
主な行事	●入国前 日本語研修 11月～翌年1月 [於；中国医科大学語学研修センター（遼寧省瀋陽市）] 3月 結団式 [於；北京] ●入国後 4月～8月の間に日本入国 9月 〈共同研究コース〉研究者集会 [於；日本財団ビル（東京）]

研究発表

発表 順番	中国側研究者	日本側研究者	掲載 頁
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①	四川省医学科学院/四川省人民医院腎臓内科 劉 馳 副研究員	国立成育医療研究センター研究所移植免疫研究室 李 小康 室長	P. 1
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②	山西医科大学第一医院護理部 李 莉 副主任護理師 崔 紅宙 皮膚科副主任 畢 紅梅 看護師長	香川県立保健医療大学保健医療学部看護学科 吳 小玉 教授	P. 2
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④	山東大学齐鲁医院口腔颌面外科 韓 亦冰 助理研究員	信州大学医学部分子医化学教室 平塚 佐千枝 教授	P. 4
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⑥	中国医科大学附属第一医院胸外科 孫 艷彬 教授 劉 思源 大学院生	東京大学大学院医学系研究科呼吸器外科学 佐藤 雅昭 教授	P. 6
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⑨	浙江省人民医院放療科 石 磊 主任醫師 李 文鑫 主治醫師	浜松医科大学医学部細胞分子解剖学講座 瀬藤 光利 教授	P. 9
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FSTL1 Exacerbates Lupus Nephritis by Promoting Th17 Cells Activation
FSTL1によるTh17細胞活性化促進を介したループス腎炎病態増悪の解析



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【Abstract】

Immune dysregulation is one of the main causes of lupus nephritis (LN), and regulatory T (Treg) cells play an important role in the pathogenesis of LN. Recent studies have shown that Follistatin-like 1 (FSTL1) significantly promotes inflammation and fibrosis in kidney diseases and is elevated in various autoimmune disorders, highlighting its important role in immune regulation. However, whether FSTL1 is associated with the immune imbalance in LN has not been reported. In this study, MRL/lpr spontaneous lupus mice were used as a model. Disease progression in the LN group was assessed by monitoring urinary creatinine and urinary protein weekly and calculating the urinary creatinine-to-protein ratio. Single-cell sequencing was further used to analyze the relationship between FSTL1 and proteins involved in Treg cells differentiation pathways. The proportion of Treg cells subsets and the expression levels of related cytokines, such as TGF- β and IFN- γ , were measured. Our findings indicated immunofluorescence findings indicated that renal FSTL1 expression in LN patients was higher than that in the control group. Subsequently, in an LN mouse model, renal RNA-seq analysis showed that FSTL1 expression in the LN group was higher than that in the control group. Bioinformatics analysis and protein-protein interaction networks suggested that upregulated FSTL1 promotes an increase in IL-6, and that the interactions of FSTL1 with IL-6 and TGF- β 1 are closely associated with the Th17 cell differentiation pathway. Meanwhile, Western blot analysis confirmed that renal FSTL1 expression was higher in the LN group compared with the control group. Comprehensive assessment of lupus disease severity indicated that the LN group exhibited significantly greater severity than the control group. Flow cytometry revealed that the proportion of Th17 cells in the kidneys and spleens of the LN group was significantly higher than that in the control group. Finally, both disease severity and Th17 cell proportion were higher in the FSTL1 overexpression group than in the PBS control group.

The Application of Traditional Chinese Medicine Appropriate Techniques
in Elderly Dermatitis and Eczema: A Sino-Japanese Joint Study
高齢者の皮膚炎・湿疹に対する中国伝統医療適正技術の応用：日中共同研究



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【Abstract】

Background: Dermatitis and eczema are prevalent chronic skin diseases in older adults, bringing physical-psychological-economic burdens to patients and families. Though TCM appropriate techniques benefit chronic inflammatory skin disorders, objective standardized assessment frameworks covering disease severity, TCM patterns and psychological status are insufficient, hindering the international promotion of integrated Chinese-Western medicine. This Sino-Japanese joint study intends to construct an intelligent assessment-intervention system for elderly dermatitis and eczema combining Chinese and Western medicine.

Methods: A multi-dimensional“disease-TCM pattern-emotion”scale will be developed incorporating EASI scoring, TCM pattern features and psychological indicators, followed by cross-cultural adaptation and preliminary validation in China and Japan. A multicenter study will recruit 100 participants in China and 100 in Japan to collect clinical data for verifying scale applicability and clinical effects of TCM appropriate techniques, and build an integrated dataset for statistical analysis.

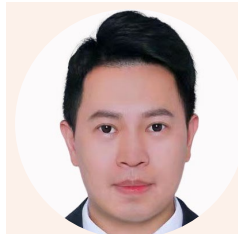
Results: This study will generate a cross-culturally feasible standardized evaluation system for elderly dermatitis and eczema, enhance objectivity and reproducibility for efficacy evaluation of TCM appropriate techniques, and realize the integration of TCM experience, geriatric nursing, psychological care and AI-aided skin monitoring. Further outputs include personalized TCM-AI intervention schemes and a Sino-Japanese academic exchange platform.

Conclusion: This work can provide clinical and methodological evidence for precise management of geriatric chronic skin diseases, standardization of TCM appropriate techniques, and global dissemination of integrated Chinese-Western medicine nursing.

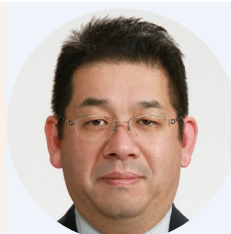
Surgery versus Radiofrequency Ablation for Small Hepatocellular Carcinoma:

A Validation Study Based on the SURF Trial

小型肝細胞癌に対する外科切除とラジオ波焼灼療法の比較：SURF試験に基づく検証研究



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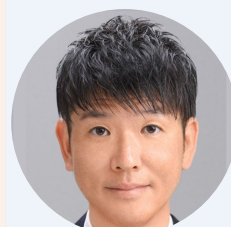
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【Abstract】

The SURF trial (a multicenter RCT and parallel observational cohort from Japan) demonstrated equivalent overall survival (OS) and recurrence-free survival (RFS) between surgery and radiofrequency ablation (RFA) for small hepatocellular carcinoma (HCC) (≤ 3 cm, ≤ 3 nodules), with a lower local recurrence rate after surgery. However, the etiology of HCC in China is predominantly hepatitis B virus (HBV)-related, whereas SURF mainly enrolled HCV patients. Whether these findings are applicable to Chinese patients remains unknown. Objective: To externally validate the SURF trial conclusions in a Chinese HBV-predominant cohort, comparing surgery versus RFA for small HCC in terms of OS, RFS, and local recurrence, and to identify treatment selection factors. Methods: This is a single-center retrospective cohort study at the Affiliated Hospital of North Sichuan Medical College. Consecutive patients with initial HCC (≤ 3 cm, ≤ 3 nodules, Child-Pugh A/B) treated with curative-intent surgery or RFA between 2015 and 2020 will be enrolled. Baseline characteristics, treatment details, and long-term follow-up data will be collected. To minimize selection bias, inverse probability of treatment weighting (IPTW) will be applied to balance confounders. Kaplan-Meier curves and Cox proportional hazards models will be used to compare OS and RFS. Subgroup analyses will be performed by tumor size and etiology. The local recurrence rate and odds ratios for selecting surgery over RFA will be evaluated. Expected Results: We hypothesize that OS and RFS will show no significant difference between the two groups, consistent with SURF, while surgery will yield a significantly lower local recurrence rate. Tumor diameter >2 cm is expected to be a strong predictor for choosing surgery. These results will provide Chinese-specific evidence to guide individualized treatment decisions and bridge the gap between Japanese trial data and Chinese clinical practice. Conclusion: This validation study will test the generalizability of the SURF trial in a different etiological setting and contribute to the global body of evidence for small HCC management.

PADI2-MYH10 Citrullination Drives OSCC Vascular Invasion
口腔扁平上皮癌の血管侵襲におけるPADI2-MYH10シトルリン化



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【Abstract】

Introduction: Oral squamous cell carcinoma (OSCC) frequently spreads through early interactions between tumor cells and blood vessels. However, the molecular mechanism that promotes intravascular tumor cell cluster (IVCT) formation remains unclear. This joint study aims to clarify whether the ER stress-Ca²⁺-PADI2-MYH10 citrullination axis contributes to OSCC vascular invasion and to identify potential biomarkers or therapeutic targets for preventing vascular dissemination.

Method: OSCC specimens were classified according to vascular invasion status using HE staining, CD31/D2-40, Pan-CK and fibrin(ogen)-related pathological assessment. Clinical variables, coagulation markers and survival-related parameters were analyzed. Fibrin-associated and citrullinated proteins were examined by immunoprecipitation, immunoblotting and proteomic analysis. Tumor-endothelial interactions were further evaluated using tumor-conditioned medium, co-culture systems, tumor-cell aggregation assays, migration/invasion assays, calcium imaging, MYH10 knockdown and PADI inhibition. Mouse tumor models were used for preliminary in vivo validation.

Result: Preliminary clinical analysis identified IV-negative and IV-positive OSCC cases, with IV positivity associated with advanced tumor features, increased fibrinogen levels and poorer prognosis. Histological analysis showed perivascular fibrinogen and citrullinated fibrinogen deposition around invasive tumor nests and IVCT regions. Molecular studies indicated activation of endothelial PADI2/HYOU1 signaling and enrichment of MYH10 in fibrin- or citrulline-associated protein complexes. Functionally, fibrin(ogen) and citrullination promoted tumor-cell aggregation and tumor-endothelial remodeling. MYH10 knockdown reduced tumor-cell migration and invasion, while PADI inhibition attenuated tumor growth and related circulating biomarkers in experimental models.

Discussion: These findings suggest that OSCC vascular invasion may be driven by a fibrin-rich and citrullination-dependent tumor-vascular niche. The ER stress-Ca²⁺-PADI2-MYH10 axis may represent a key mechanism linking endothelial activation, tumor-cell aggregation and IVCT formation. Further clinicopathological validation, MYH10 citrullination site mapping and rescue experiments will be performed to support a joint English manuscript for international publication.

Nrf2 transduced abnormal biomechanics and regulates chondrocyte ferroptosis in osteoarthritic cartilage of temporomandibular joint
 Nrf2シグナル伝達による変形性顎関節症における軟骨細胞の細胞死メカニズムに関する研究



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【Abstract】

Osteoarthritis (OA) is a chronic and highly prevalent joint disease, causing pain and disability in the aging population. Ferroptosis, a non-apoptotic modality of cell death, is defined as an iron-dependent regulated necrosis that is caused by extensive lipid peroxidation-mediated membrane damage. Ferroptosis plays an important role in OA cartilage. Temporomandibular joint osteoarthritis (TMJ OA) is the fourth major subtype disease of the temporomandibular disorders (TMD), and has become the leading cause of orofacial pain in the oral and maxillofacial region. The cause of cartilage degeneration and chondrocyte ferroptosis in TMJ OA caused by abnormal dental occlusion remains unclear. Recently, our previous study have shown that the experimental unilateral anterior crossbite (UAC) induced OA-like lesions such as ferroptosis, cartilage thinning and reduced cartilage matrix in the mouse TMJs. Glutathione peroxidase 4 (GPX4), which can be inhibited by iron-chelating agents and lipophilic antioxidants, as the core molecule that regulates ferroptosis and plays a key role in cartilage degeneration and matrix loss. NF-E2-related factor 2 (Nrf2), a biomechanics-stimulation transduced signal, is downregulated expression in cell under cyclic mechanical stress, which is involved in the regulation of cell differentiation and death. We speculate that Nrf2-GPX4 mediated biomechanics-stimulation transduction may exist in OA chondrocytes, which promotes ferroptosis of osteoarthritic chondrocytes and thus plays an important role in causing chondrocyte loss and matrix loss. In this joint project, a lot of advanced technologies will be adopted such as Nrf2/GPX4 conditional knockout mice, proteomics, DNA-protein chip-seq and other advanced molecular biological assays. We demonstrated the biomechanic-stimulation signal transduction effect of Nrf2-GPX4 negatively regulating ferroptosis in osteoarthritic chondrocytes. This study not only improves the basic theory of OA cartilage degeneration, but also provides a new strategy for the prevention and treatment of OA by targeting the regulation of ferroptosis.

Integrating Single-Cell Analysis and ctDNA for Biomarker Discovery in NSCLC
NSCLCにおけるバイオマーカー探索を目的としたシングルセル解析とctDNA解析の統合



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【Abstract】

Lung cancer is a leading cause of cancer-related mortality, and postoperative recurrence remains a major challenge in non-small cell lung cancer (NSCLC). Responses to systemic therapies, including chemoimmunotherapy and targeted therapy, vary considerably among patients, creating a need for reliable biomarkers of treatment response, recurrence risk, and disease status. Circulating tumor DNA (ctDNA) provides a minimally invasive means of detecting molecular residual disease and tracking tumor dynamics. Longitudinal ctDNA studies, including analyses of the TRACERx cohort, have shown that ctDNA can identify patients at high risk of recurrence and monitor disease after surgery.

Professor SATO Masaaki leads the Japanese team at the Department of Thoracic Surgery, The University of Tokyo. His team studies thoracic surgery, precision lung resection, neoadjuvant treatment for lung cancer, and related translational research. Professor SUN Yanbin's team at the Department of Thoracic Surgery, The First Hospital of China Medical University, focuses on locally advanced NSCLC and has used single-cell RNA sequencing and spatial transcriptomics to characterize tumor microenvironment remodeling after neoadjuvant chemoimmunotherapy.

This project will further analyze and validate single-cell findings associated with treatment response and integrate them with longitudinal ctDNA and clinical follow-up data from patients with NSCLC. Public single-cell RNA-seq and spatial transcriptomic datasets will be used to expand the analytical cohort and identify the cell types and spatial contexts associated with candidate biomarkers. The project aims to integrate single-cell-derived biological features with ctDNA-based monitoring to identify clinically relevant biomarkers for predicting treatment response and postoperative recurrence in NSCLC.

Clinical Significance of Rim Sign in Carotid Artery Stenosis 頰動脈狭窄症におけるRim signの意義



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Postgraduate student

【Abstract】

Introduction: Carotid artery stenosis is an important cause of acute ischemic stroke. Association between the presence of lipid rich necrotic core with intraplaque hemorrhage in the carotid plaque and cerebral ischemic events or ischemic complication after neuro-endovascular surgery were reported. While carotid calcified plaques are generally considered stable lesions, certain lesions—such as the “Rim sign” which is diagnosed using CT angiography—have been reported to be associated with clinical symptoms and unstable plaque components, and recently have attracted increasing attention. The purpose of this study is to elucidate the relationship between Rim sign and unstable plaques quantitatively and to clarify its clinical significance.

Materials and Methods: Between January 2014 and December 2022, 66 cases of carotid artery stenosis which underwent quantitative plaque assessment based on preoperative CT angiography and MRI plaque imaging were included. We analyzed the association between the presence of Rim sign, degree of it (distribution ratio) and the volume of unstable components (intraplaque hemorrhage and lipid rich necrotic core) on MRI plaque imaging, as well as patient characteristics.

Results: There were 53 male patients (80.3%) with a mean age of 72 ± 3 years. The Rim sign was observed in 28 patients (42%). Symptomatic lesions were present in 17 of 28 patients (61%) in the Rim sign-positive group and in 15 of 38 patients (39%) in the Rim sign-negative group. The volume of unstable components on MRI plaque imaging was significantly larger in the Rim sign-positive group than in the Rim sign-negative group (Median [interquartile range]: Rim sign-positive group vs. Rim sign-negative group: $294.1 [184.9-527]$ mm³ vs. $65.8 [2.36-141.4]$ mm³, $p < 0.0001$). Furthermore, the volume of unstable components was inversely correlated with the distribution ratio of the Rim sign ($r = -0.389$, $p = 0.0404$).

Conclusions: Presence of the Rim sign is associated with larger volume of lipid rich necrotic core and intraplaque hemorrhage in carotid plaque. Moreover, in the Rim sign positive case, inverse correlation between degree of Rim sign and unstable component volume was found. These results suggest that the Rim sign may become useful imaging marker for risk stratification of acute ischemic stroke using CT angiography.

Mechanisms Underlying Soluble Uric Acid-Induced TRPV1 Sensitization in DRG Neurons 可溶性尿酸により誘導されるDRGニューロンのTRPV1感作機序



程 継東

CHENG Jidong

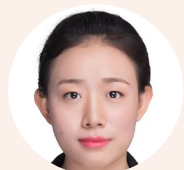
Professor; Dean of Instruction
Department of Internal
Medicine, Xiang'an Hospital of
Xiamen University, School of
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付 善潘

FU Shanpan

Postgraduate Student



郝 玥美

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【Abstract】

Hyperuricemia is a major risk factor for gout and is increasingly recognized as a potential modulator of peripheral sensory nerve function. Although monosodium urate crystals are known to trigger acute gouty inflammation and pain, whether soluble uric acid alters sensitivity of primary sensory neurons remains unclear. This study aimed to investigate whether soluble uric acid sensitizes dorsal root ganglion (DRG) neurons through transient receptor potential vanilloid 1 (TRPV1) channels and to explore the underlying mechanism.

DRG neurons were acutely isolated from rats and cultured in MEME-COMP medium containing soluble uric acid at concentrations 3, 6, or 10 mg/dL for 24 h. TRPV1-mediated neuronal activity was evaluated by calcium imaging using capsaicin stimulation, while TRPV1 expression in DRG neurons was assessed by immunofluorescence. Intracellular reactive oxygen species (ROS) levels were measured using DCFH-DA staining. N-acetylcysteine (NAC) was used to examine the involvement of oxidative stress.

Soluble uric acid treatment showed no obvious cytotoxicity in primary DRG neurons. Calcium imaging revealed that 6 and 10 mg/dL soluble uric acid enhanced capsaicin-induced calcium responses in TRPV1-positive neurons, with prolonged response duration and delayed calcium peak observed at 10 mg/dL. In contrast, 3 mg/dL soluble uric acid showed no significant effect compared with the control group. Immunofluorescence analysis further showed that 6 and 10 mg/dL soluble uric acid increased both the proportion of TRPV1-positive neurons and TRPV1 fluorescence intensity, with a greater effect at 10 mg/dL. These findings indicate that soluble uric acid enhances DRG neuronal sensitivity by upregulating TRPV1 expression. Further experiments demonstrated that ROS levels increased in a concentration-dependent manner after treatment with 3, 6, and 10 mg/dL soluble uric acid. Co-treatment with 10 or 20 mM NAC reduced intracellular ROS accumulation and reversed the 10 mg/dL uric acid-induced increase in TRPV1 expression, suggesting that oxidative stress contributes to soluble uric acid-mediated TRPV1 upregulation.

Together, these results suggest that soluble uric acid sensitizes DRG neurons by enhancing TRPV1 expression and function, at least partly through oxidative stress. This mechanism may help explain how hyperuricemia contributes to peripheral neuronal hypersensitivity and may provide a potential therapeutic target for preventing uric acid-related pain sensitization.

X-Ray FLASH Whole Body Irradiation Study- Exploring Acute Lipidomic and Spatial Alterations
Following Ultra-High Dose Rate Exposure
X線FLASH全身照射研究—超高線量率被ばく後の急性脂質プロファイル
および空間的変化の解明



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【Abstract】

FLASH radiotherapy (FLASH-RT) exhibits remarkable normal tissue protection, yet the multi-omic mechanisms-particularly systemic lipid metabolism remodeling-remain elusive. To address this, a high-fidelity mouse model was constructed to support integrated multi-omics investigations following whole-body FLASH irradiation (WB-FLASH). On November 17, 2025, C57BL/6J mice were subjected to whole-body irradiation using a ZJSG X-FLASH accelerator. Three cohorts were established: Conventional radiotherapy (CONV-RT, 10 Gy/min), FLASH (200 Gy/s), and a non-irradiated Blank control (n=7) serving as a biological baseline. EBT4 film dosimetry verified an average field dose of 7.14 Gy with exceptional uniformity (Inline/Crossline Symmetry: 100.5%/102%). Systematic harvesting occurred across three critical windows: 4 h (CONV-RT 14, median sampling 6.2 h; FLASH 10, median sampling 7.7 h), 24 h (CONV-RT 15, FLASH 15), and 48 h (CONV-RT 15, FLASH 15). A total of nineteen distinct tissues-including eye, blood, skin, salivary gland, brain, spinal cord, lung, thymus, heart, liver, spleen, kidney, intestine, colorectum, and leg muscle-were snap-frozen in liquid nitrogen and archived at -80° C. Physical quality assurance confirmed stable 200 Gy/s output and homogeneous dose distribution. A comprehensive, temporally stratified biobank spanning 48 hours post-irradiation has been successfully established, with balanced subject distribution ensuring robust statistical power. Future investigations will integrate untargeted LC/MS lipidomics with spatial mapping to profile acute lipid remodeling at 4h, 24h, and 48h following FLASH versus conventional whole-body irradiation. Multivariate analyses and pathway enrichment will identify key peroxidation signatures-particularly PE-OOH dynamics-that distinguish FLASH-sparing from CONV-induced toxicity. This spatiotemporal lipidomic framework is expected to elucidate the metabolic basis of the FLASH effect, providing a robust foundation for clinical translation.

第47期〈学位取得コース〉研究者名簿（2026年度招請）

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	低温劣化に対する高い耐性を有する新しい表面改質ジルコニアを用いた骨 形成の促進	課程博士 歯学		
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	大腸における腫瘍内マイクロバイオームと分子・臨床サブタイプに基づく 予測モデルの構築	課程博士 医学		
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	顕微鏡下手術針のビジョンベーストラッキングとスキルに基づくロボット 針操作に関する研究	課程博士 工学		

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	静水圧の上昇が肝細胞のリポファジーに与える影響と関連機構	医学		

『日中笹川医学奨学金制度』沿革

1985年	財団法人日中医学協会設立
1986年	中国衛生部、日中医学協会、笹川記念保健協力財団の間で『笹川医学奨学金制度』協定書に調印—10年間に1,000名の研究者を招請
1987年	笹川医学奨学金制度開始—第1期生来日
1991年	笹川医学奨学金制度5周年記念式典を北京・人民大会堂で開催 帰国した研究者が同窓会組織「笹川医学奨学金進修生同学会」（笹川同学会）を結成し、中国全 域の医療水準向上及び日中間の医学・医療交流の促進・深化を目的に、辺境地域の医療従事者の 育成や被災地等におけるボランティア診療、日本人専門家を招き学術交流会・学術セミナーの開 催等の活動を行う
1992年	帰国した研究者の中から特に優秀な研究者を再招請する特別研究者招請事業開始
1996年	『日中笹川医学研究者制度（第二次制度）』協定書に調印—1998年から10年間に1,000名の研究者 を招請
1997年	笹川医学奨学金制度10周年記念行事を北京・人民大会堂で開催
1998年	第20期生帰国、受け入れ者数1,000名を達成 第二次制度開始—第21期生来日
2007年	日中笹川医学研究者制度20周年記念式典を北京・人民大会堂で開催 日本財団、中国衛生部の間で『日中笹川医学奨学金制度（第三次制度）』協定書に調印 —2008年9月から5年間に150名の研究者を招請
2008年	第三次制度開始—第31期生来日、特別研究者招請事業終了
2013年	日本財団、中国国家衛生・計画生育委員会の間で『日中笹川医学奨学金制度（第四次制度）』 協定書に調印—2014年から5年間に150名の研究者を招請
2014年	第四次制度開始—第36期生来日
2016年	日中笹川医学奨学金制度30周年記念式典を東京で開催
2017年	日本財団、中国国家衛生・計画生育委員会、日中医学協会の間で『日中笹川医学奨学金制度（第五 次制度）』協定書に調印—日中医学交流の新たな形を目指し、2018年から〈学位取得コース〉と 〈共同研究コース〉で構成
2018年	第五次制度開始—第40期生来日
2023年	日中笹川医学奨学金制度35周年記念式典を北京・人民大会堂で開催 日本財団・中国国家衛生健康委員会・日中医学協会の間で『日中笹川医学奨学金制度（第六次制 度）』協定書に調印—〈学位取得コース〉〈共同研究コース〉を進化発展させると共に〈ポスト ドクターコース〉を新設
2024年	第六次制度開始—第45期生来日

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