

# 日中笹川医学奨学金制度 第46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：2025年9月19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

趙 群 笹川医学奨学金進修生同学会理事長

祝 辞 施 泳 中華人民共和国駐日本国大使館公使

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 10組（第46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 橋本 朋幸 笹川保健財団常務理事

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	<p>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）</p> <p>②研究費・・・・・・・・・・・・・・・・・・・・・月額10万円（受入機関に支給）</p> <p>※日本に滞在している期間に対して支給する。</p> <p>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。</p>
研究者の義務	<p>① 日本滞在期間中は日本国法令を遵守すること</p> <p>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</p> <p>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</p> <p>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</p> <p>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）</p> <p>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</p> <p>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</p> <p>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</p>
主な行事	<p>●入国前</p> <p>日本語研修 11月～翌年1月</p> <p style="text-align: right;">[於；中国医科大学語学研修センター（遼寧省瀋陽市）]</p> <p>3月 結団式 [於；北京]</p> <p>●入国後</p> <p>4月～8月の間に日本入国</p> <p>9月 〈共同研究コース〉研究者集会 [於；日本財団ビル（東京）]</p>

# 研究発表

発表 順番	中国側研究者	日本側研究者	掲載 頁
共同研究テーマ（英文・日文）			
	復旦大学附属中山医院胸外科 王 帥 主治医師、丁 建勇 主任医師	順天堂大学大学院医学研究科呼吸器外科学講座 鈴木 健司 主任教授	
①	Clinical outcomes of different surgical approaches of thymectomy for myasthenia gravis 重症筋無力に対する胸腺摘除術の異なる手術アプローチの臨床転帰		P. 1
	中国医学科学院腫瘍医院乳腺外科 孔 祥溢 副主任医師 張 文祥 主治医師、陸 葉 医師	国立がん研究センター中央病院乳腺・腫瘍内科 米盛 勸 科長	
②	Exploring the Feasibility of Preoperative Tumor-Bed Boost, Oncoplastic Surgery, and Adjuvant Radiotherapy Schedule in Early-Stage Breast Cancer:A Phase II Clinical Trial 早期乳癌に対する術前腫瘍床ブースト照射とオンコプラスティック乳房温存手術および術後放射線治療スケジュールの併用に関する第II相臨床試験		P. 2
	天津市第三中心医院肝胆疾病研究所 駱 瑩 副研究員	大阪大学大学院工学研究科 応用化学専攻有機工業化学領域 松崎 典弥 教授	
③	In Vitro Construction of NASH Model : Mimicking Pathological Collagen Remodeling NASHモデルのin vitro構築：コラーゲンの病理学的バイオミメティックリモデリング		P. 3
	中国医学科学院北京協和医院 転化医学国家重大科技基礎施設 陳 宏達 研究員 張 越倫 助理研究員、駱 晨雨 大学院生	国立がん研究センターがん対策研究所疫学研究部 岩崎 基 部長	
④	Identification of colorectal cancer risk factors using Mendelian randomization and development of risk prediction models メンデルランダム化解析による結腸直腸癌のリスク因子の同定とリスク予測モデルの開発		P. 4
	川北医学院口腔医学院 劉 英 教授 劉 為 講師、唐 婉容 准教授	東北大学大学院歯学研究科附属歯学イノベーションリエゾンセンター国際連携推進部門 洪 光 教授・副研究科長	
⑤	Asymmetric porous nZnO/P34HB composite membrane with antibacterial property for alveolar bone regeneration 抗菌性を有する非対称多孔質ナノZnO/P34HB複合膜の歯槽骨再生への応用		P. 5
	西安医学院第一附属医院檢驗科 李 卓 教授・主任技師 周 建 講師・副主任技師、張 娜 檢驗技師	秋田大学大学院医学系研究科医学専攻 病態制御医学系器官病態学講座 後藤 明輝 教授	
⑥	A Multigene Mutation Signature for Prognostic Stratification in Tongue Oral Squamous Cell Carcinoma 舌扁平上皮癌における予後層別化のための多遺伝子変異シグネチャー		P. 6
	西安交通大学第二附属医院消化内科 戴 菲 教授・主任医師 秦 斌 准教授、全 曉静 助理研究員	獨協医科大学消化器内科 入澤 篤志 主任教授、郷田 憲一 教授	
⑦	Diagnostic performance of EUS, CE and ME in predicting the invasion depth of early gastric cancer : a comparative study 早期胃癌の浸潤深達度評価におけるEUS・CE・MEの診断能の比較研究		P. 7
	天津医科大学総医院血液内科 劉 春燕 主任医師、閻 莉 主治医師	金沢大学附属病院血液内科 細川 晃平 准教授	
⑧	The role of NK-B cell interaction in the immunopathogenesis of aplastic anemia under inflammatory conditions 炎症状態における再生不良性貧血の免疫病態におけるNK-B細胞相互作用の役割		P. 8
	首都医科大学附属北京口腔医院国際医療部 周 建 教授・主任医師 郜 康 大学院生、張 天翼 大学院生	愛知医科大学医学部歯科口腔外科学講座 各務 秀明 特任教授	
⑨	The Role and Mechanisms of 3D Spheroid Culture in Enhancing the Potency of Dental Stem Cells for Pulp-Dentin Complex Regeneration 歯髄象牙質複合体再生における、三次元スフェロイド培養を用いた歯源性幹細胞の潜在能力増強の作用機序に関する研究		P. 9
	首都医科大学附属北京潞河医院腫瘍センター 全 営営 主治医師	国立がん研究センター東病院消化管内科 設楽 紘平 科長	
⑩	TROP2 Expression and Its Association with Clinicopathological Features and Biomarkers in Gastric and GEJ Adenocarcinomas 胃および胃食道接合部腺癌におけるTROP2発現と臨床病理学的特徴およびバイオマーカーとの関連性		P. 10

Clinical outcomes of different surgical approaches of thymectomy for myasthenia gravis  
重症筋無力に対する胸腺摘除術の異なる手術アプローチの臨床転帰



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【Abstract】

Myasthenia gravis (MG) is the most common neuromuscular junction disorder, characterized by fatigable muscle weakness resulting from autoimmune-mediated destruction at the postsynaptic membrane. Thymectomy has been a cornerstone of MG treatment since its initial therapeutic benefit was reported in the 1940s. While median sternotomy has traditionally been the standard approach for extended thymectomy, minimally invasive thoracoscopic techniques have gained favor due to reduced postoperative pain, shorter hospital stays, improved cosmetic outcomes, and faster recovery. However, the comparative efficacy of these surgical approaches in achieving long-term neurological remission remains a subject of ongoing debate, with no global consensus established over the past three decades.

To address this gap, we conduct a multi-center retrospective cohort study in collaboration with Mayo Clinic (USA), Juntendo University (Japan), and Zhongshan Hospital (China). In this study, multi-institutional database is retrospectively reviewed of MG patients received thymectomy with or without thymoma. We analyze patients treated between January 1, 2006, and December 31, 2022. To date, over 300 patients with MG who underwent thymectomy—with or without thymoma—have been collected. Moreover, additional 158 cases of MG from Mayo Clinic are analyzed. The primary endpoints are neurological outcomes, including remission rates and time to achieve remission. Secondary endpoints encompass perioperative outcomes (postoperative complications, hospital and ICU stay durations, and incidence of postoperative myasthenic crisis) and oncological outcomes (histopathological findings, progression-free survival, and overall survival). Multivariable logistic regression analysis is performed to identify independent risk factors for MG remission and postoperative myasthenic crisis. This ongoing international study represents the largest known population-based cohort of MG patients undergoing thymectomy with comprehensive clinicopathological data. Through robust international collaboration, we aim to clarify the long-term effectiveness of different surgical approaches to thymectomy, inform global surgical standards in the management of MG, and contribute significantly to the advancement of thoracic surgery.

## 発表②

### Exploring the Feasibility of Preoperative Tumor-Bed Boost, Oncoplastic Surgery, and Adjuvant Radiotherapy Schedule in Early-Stage Breast Cancer : A Phase II Clinical Trial

早期乳癌に対する術前腫瘍床ブースト照射とオンコプラスティック乳房温存手術および術後放射線治療スケジュールの併用に関する第II相臨床試験



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#### 【Abstract】

**Background:** Oncoplastic breast-conserving surgery (OBCS) enhances cosmetic outcomes but poses challenges for postoperative tumor-bed localization during radiotherapy, especially for delivering boost doses. Ultra-hypofractionated whole-breast irradiation (u-WBRT) shortens treatment time compared to conventional schedules. To address tumor-bed delineation inaccuracies and reduce boost-related toxicities, we investigated a novel paradigm: delivering a single-fraction preoperative tumor-bed boost followed by breast-conserving surgery (BCS/OBCS) and adjuvant u-WBRT, in early-stage breast cancer patients.

**Methods:** This single-arm, phase II trial enrolled patients <55 years with biopsy-confirmed, unicentric, node-negative early-stage breast cancer. All patients received a single 10 Gy preoperative tumor-bed boost guided by MR-Linac-based IMRT. BCS or OBCS was performed within 7–15 days, followed by u-WBRT (26 Gy in 5 fractions) within 6 weeks postoperatively. Surgical complications were recorded using the Clavien–Dindo classification. Patient-reported outcomes were assessed using the BREAST-Q (version 2.0) questionnaire. A propensity score-matched control group receiving conventional postoperative radiation was used for comparison. **Results:** Thirty-six patients completed the full protocol. Grade >2 acute radiation toxicity occurred in 11.1%, meeting the Simon’s two-stage design safety threshold. Surgical complications occurred in 19.4% of patients, mainly delayed wound healing (11.1%), but all completed local treatment as planned. Cosmetic results were favorable, with 94.5% reporting excellent or good outcomes. Compared to matched controls, patients in the novel protocol group showed comparable satisfaction in breast aesthetics, psychosocial and sexual well-being, but had lower chest physical well-being ( $P = 0.045$ ) and higher perception of arm swelling ( $P = 0.01$ ). No local recurrence or distant metastasis was observed during a median follow-up of 9.8 months (range 2.4–18.9).

**Conclusion:** This study confirms the technical feasibility and safety of preoperative single-fraction boost combined with u-WBRT in early-stage breast cancer. The approach addresses challenges of tumor-bed localization post-OBCS while reducing overall radiation duration. Although patient-reported chest physical well-being was slightly lower, oncological and cosmetic outcomes remained favorable. These findings support further evaluation in randomized controlled trials with long-term follow-up to assess efficacy and patient quality of life.

## In Vitro Construction of NASH Model : Mimicking Pathological Collagen Remodeling NASHモデルのin vitro構築 : コラーゲンの病理学的バイオミメティックリモデリング



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### 【Abstract】

Nonalcoholic steatohepatitis (NASH), representing an advanced stage of nonalcoholic fatty liver disease (NAFLD), is an increasingly prevalent global health concern with limited therapeutic options. Although in vitro models of NASH frequently employ lipopolysaccharide (LPS) and free fatty acid (FFA) stimulation to simulate steatosis and inflammation, these models inadequately capture the complexity of the hepatic microenvironment, particularly the abnormal remodeling of the extracellular matrix (ECM). This remodeling, characterized by excessive collagen deposition and abnormal crosslinking, is pivotal in the progression from NAFLD to NASH and constitutes a crucial target for the development of in vitro models. Consequently, constructing the dynamic pathological process of collagen remodeling in vitro is essential for establishing a NASH model.

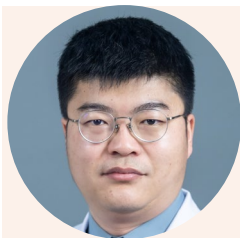
Under aging or pathological conditions, advanced glycation end-products (AGEs) facilitate non-enzymatic crosslinking of collagen, resulting in enhanced matrix stiffness and increased mechanical strength. Additionally, these mechanical alterations in the ECM influence the behavior of liver-resident cells, including hepatic stellate cells, Kupffer cells and sinusoidal endothelial cells. By incorporating AGEs into an in vitro NASH model, it is possible to dynamically modulate collagen remodeling to better mimic in vivo fibrosis progression, thus addressing limitations of conventional models that fail to simulate pathological ECM deposition and stiffening. To the best of our knowledge, none of these studies reported the use of AGEs to induce pathological collagen remodeling in vitro to construct a NASH model with disease-relevant mechanical properties, nor to mimic disease progression.

In this project, our objective is to develop an in vitro model of NASH that replicates the mechanical properties associated with pathological collagen remodeling observed in vivo. This model is based on the cell assembled viscous tissue by sedimentation (CAViTs) method, previously established by Professor Matsusaki. The CAViTs technique enables the formation of vascularized hepatic tissue (VHT) that display physiologically relevant sinusoidal structures and enhanced liver specific functions. To better mimic the pathological collagen in vivo, we introduced AGEs-induced collagen crosslinking into the VHT model. This model allows us to study the interactions between collagen remodeling and the liver microenvironment, offering insights into how these changes influence liver cell behavior and fate in NASH progression.

## 発表④

Identification of colorectal cancer risk factors using Mendelian randomization and development of risk prediction models

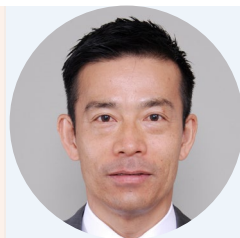
メンデルランダム化解析による結腸直腸癌のリスク因子の同定とリスク予測モデルの開発



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### 【Abstract】

This study aims to identify causal risk factors for colorectal cancer (CRC) and to develop effective risk prediction models tailored to East Asian populations. Genetic data were obtained from publicly available genome-wide association studies (GWAS) involving East Asian individuals, and epidemiological data were derived from established cohort studies. Mendelian randomization (MR) analyses were conducted to infer the causal effects of traditional lifestyle and environmental exposures, as well as novel plasma metabolites, on CRC risk. Key risk factors identified through MR were incorporated into risk prediction models developed using both statistical and machine learning approaches. Model performance was evaluated through discrimination and calibration metrics, and validated using independent datasets. The potential public health utility of these models was further assessed through population-based cohorts and microsimulation modeling to estimate their impact on CRC prevention and screening efficiency. This integrative approach provides novel insights into CRC etiology and supports the development of precision prevention strategies in East Asian populations.

Asymmetric porous nZnO/P34HB composite membrane  
with antibacterial property for alveolar bone regeneration

抗菌性を有する非対称多孔質ナノZnO/P34HB複合膜の歯槽骨再生への応用



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【Abstract】

**Objective:** Various factors such as tumor, trauma, age-related changes, and periodontitis may lead to the width or height defect of alveolar bone. Guided bone regeneration (GBR) has emerged as the primary clinical solution for bone augmentation in implant rehabilitation. This study aims to construct an asymmetric porous composite GBR membrane with antibacterial property for alveolar bone defect repair.

**Methods:** An asymmetric porous poly(3-hydroxybutyrate-co-4-hydroxybutyrate) (P34HB) membrane loading with zinc oxide nanoparticles (nZnO; 0-1.5%) was fabricated via solvent exchange. The membrane has a loose, porous bone-facing side to facilitate osteogenic cell infiltration and differentiation and a dense mucosa-facing layer loaded with nZnO for antibacterial protection and soft tissue barrier function. The membrane was characterized using SEM, EDS, XPS, and mercury porosimetry. In vitro evaluations included hemocompatibility, degradation, and osteogenic potential assessments using BMSC cultures.

**Results:** The 0.5% nZnO/P34HB membrane demonstrated optimal properties: uniform nZnO distribution, controlled pore architecture, and suitable mechanical strength. It exhibited excellent hemocompatibility, sustained degradation. BMSC cultures showed enhanced osteogenic differentiation, as evidenced by alkaline phosphatase activity and mineralization assays.

**Conclusion:** The developed 0.5% nZnO/P34HB membrane combines asymmetric porosity, controlled biodegradability, and osteogenic properties, making it a promising candidate for clinical GBR applications. Future studies will focus on antimicrobial properties and in vivo validation performance.

## A Multigene Mutation Signature for Prognostic Stratification in Tongue Oral Squamous Cell Carcinoma

舌扁平上皮癌における予後層別化のための多遺伝子変異シグネチャー



李 卓

*LI Zhuo*

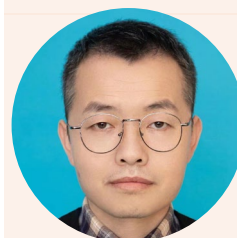
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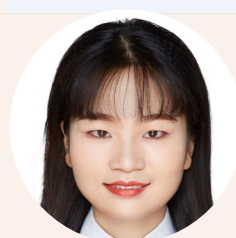
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## 【Abstract】

**Introduction:** Head and neck cancer, the sixth most common malignancy worldwide, is a major contributor to cancer-related morbidity and mortality. Tongue oral squamous cell carcinoma (TOSCC) is the most frequent subtype of head and neck cancer globally. In this study, we employed next-generation sequencing (NGS) to elucidate the genetic characteristics of TOSCC and constructed a prognostic model based on gene mutation profiles.

**Method:** TOSCC tissues were collected from 34 patients who underwent surgical resection. Targeted sequencing (MiSeq) was performed on 34 FFPE samples for which analysis was feasible. Amplicon libraries for each DNA sample were prepared using the AmpliSeq for Illumina Cancer Hotspot Panel v2 (CHPv2) and the AmpliSeq Library PLUS Kit (Illumina, San Diego, CA, USA), following the manufacturer's protocol without modifications. Meanwhile, based on the measured data, genes with mutation frequencies exceeding 5% were screened to construct a prognostic analysis model, and an external validation set of 159 samples was obtained from the The Cancer Genome Atlas (TCGA) database and combined with sequencing data to validate the model.

**Result:** A total of 42 different genes were detected in our samples. TP53 was the most frequently mutated gene (23%), followed by PTEN (8%), ATM (7%), PTPN11 (6%), CDKN2A (6%) and VHL (5%). Subsequently, multivariate Cox regression analysis of the data from the above 34 patients was performed, and a prognostic risk model based on the 6 genes with high mutation frequencies (TP53, ATM, CDKN2A, PTEN, PTPN11 and VHL) was established and evaluated. Then, the model was validated by the integrated validation set including 159 samples from the TCGA database and the 34 sequenced samples in this study, which showed significant statistical differences in the survival curves ( $P=0.025$ ) between the high-risk group ( $n=82$ ) and the low-risk group ( $n=111$ ). Finally, we used this model in combination with the clinical characteristics of the patients (pStage, TNM stage, smoking and alcohol consumption, etc.) to construct a clinical evaluation nomogram model. The results showed that the model can effectively predict the prognosis of TOSCC patients. The AUC values of 1-year, 3-year and 5-year survival rates were 0.811, 0.847 and 0.796, and the corresponding C-index were 0.696, 0.701, and 0.699, respectively. These results indicate that the model has certain stability and clinical application potential.

**Discussion:** This study showed that TP53 mutations were the most common, and a higher number of total variants was associated with better clinical outcomes, similar to what has been observed in OSCC. Moreover, the prognostic model constructed using the 6 genes with high mutation frequencies exhibited strong prognostic predictive ability for TOSCC.

# Diagnostic performance of EUS, CE and ME in predicting the invasion depth of early gastric cancer : a comparative study

## 早期胃癌の浸潤深達度評価におけるEUS・CE・MEの診断能の比較研究



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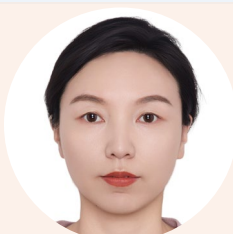
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秦 斌

*QIN Bin*

Associate Professor



全 曉静

*QUAN Xiaojing*

Attending physician

### 【Abstract】

**Background and aim:** The treatment strategy for early gastric cancer (EGC) largely depends on the depth of tumor invasion. Endoscopic ultrasonography (EUS), conventional endoscopy (CE) and magnifying endoscopy (ME) are all considered useful for predicting the invasion depth of EGC. This study aimed to compare the diagnostic performance of EUS alone versus EUS combined with CE and /or ME in evaluating invasion depth, and to establish an effective diagnostic strategy.

**Patients and methods:** Consecutive patients with submucosal invasive EGC between April 2016 and September 2024 were retrospectively enrolled, while those with mucosal cancer were prospectively recruited from May to July 2025 at Dokkyo medical university hospital. All patients underwent EUS, CE, and ME. Histological diagnosis, based on the Japanese Classification of Gastric Carcinoma, was established as the gold standard and was used to categorize lesions into two groups: (1) mucosal (M) or minute submucosal invasion  $<500\mu\text{m}$  from the muscularis mucosae (SM1), and (2) SM invasion  $\geq 500\mu\text{m}$  (SM2 or deeper). We prepared three image catalogs of EUS alone (A), EUS plus CE (B), and EUS plus CE and ME (C). Endoscopic images were independently reviewed by three gastroenterologists (Q.XJ, Q.B and G.K). Initial invasion depth predictions (M-SM1 vs. SM2 or deeper) were made using catalog A, followed by catalog B, and catalog C. The primary outcome was the diagnostic accuracy values and interobserver agreement of EUS alone and EUS combined with CE and/or ME in assessing invasion depth.

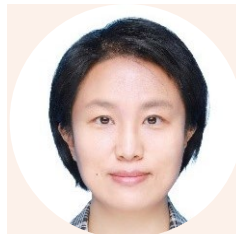
**Results:** Twelve M/SM1 and 12 SM2 tumors from a total of twenty-four patients were included. EUS alone achieved a sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy of all 86.1%. EUS plus CE resulted in a slight decrease in specificity (80.5%), PPV (81.5%) and accuracy (83.3%). However, EUS plus CE, and ME yielded the highest diagnostic accuracy (87.5%) with improved sensitivity (88.9%) and NPV (88.6%).

Interobserver agreement was almost perfect for EUS alone ( $\kappa=0.83$ ), but moderate in both EUS+CE and EUS+CE+ME ( $\kappa=0.50$ ). High-confidence rate was 64% with EUS alone, increasing to 74% with EUS+CE, and 79% with EUS+CE+ME, no significant difference was observed ( $P=0.064$ ).

**Conclusions:** EUS showed the highest reliability and suboptimal accuracy in evaluating the invasion depth of SM2 EGC. Combining EUS with CE and ME improved diagnostic performance and physicians' confidence levels. These findings suggest that combining EUS with CE and ME appears promising in predicting the invasion depth of EGC.

The role of NK-B cell interaction in the immunopathogenesis of aplastic anemia under inflammatory conditions

炎症状態における再生不良性貧血の免疫病態におけるNK-B細胞相互作用の役割



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【Abstract】

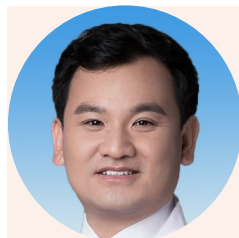
Aplastic anemia is a disease caused by immune abnormalities leading to pancytopenia, clinically characterized by severe bleeding, infection, and anemia. The current treatment is mainly based on immunosuppressive therapy. At present, research on the pathogenesis of diseases is mostly focused on aspects related to immune function. The applicant's previous research has found that in such patients, NK cells exhibit a significant inflammatory activation state, and the expression of inflammatory factors is increased. After improving NK cells, the disease status of mice can also be significantly improved. And the results of peripheral blood single-cell sequencing of the patient showed that there is ITGB2/CD18 - FCER2/CD23 interaction between NK cells and B cells. At present, research on NK-B cell interaction is limited, and its mechanism of action is not clear. Therefore, this project aims to analyze the role of corresponding immune abnormalities in the occurrence and development of AA disease from the perspective of NK-B cell interaction.

Our research content is divided into three parts:

- (1) Patient Experiments: Based on the previous single-cell sequencing results, we will further investigate the expression of inflammatory factors in the plasma and NK cells of patients with initial clinical diagnosis, improvement after treatment, and recurrence after treatment using molecular biology techniques. At the same time, we will also detect the expression of ITGB2/CD18 - FCER2/CD23 in the NK-B cell interaction pathway and conduct correlation analysis with indicators such as B cell function, proliferation and apoptosis status, disease severity, and therapeutic efficacy.
- (2) Cell Experiments: This study focuses on how NK cells under inflammatory conditions regulate B cell function through the ITGB2/CD18 - FCER2/CD23 signaling axis. We will establish an in vitro co-culture system using ITGB2-knockdown NK cells and B cells to investigate the impact of NK cells on FCER2 expression and functional alterations in B cells under stimulation by high levels of inflammatory factors (e.g., S100A, S100B) and the underlying molecular mechanisms.
- (3) Animal Experiments: Using SAA mouse models, we will evaluate the therapeutic potential of modulating the ITGB2/CD18 - FCER2/CD23 signaling pathway, aiming to identify novel therapeutic targets.

# The Role and Mechanisms of 3D Spheroid Culture in Enhancing the Potency of Dental Stem Cells for Pulp-Dentin Complex Regeneration

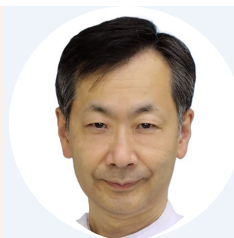
歯髄象牙質複合体再生における、三次元スフェロイド培養を用いた歯源性幹細胞の潜在能力増強の作用機序に関する研究



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## 【Abstract】

The functional regeneration of the dentin-pulp complex is pivotal for tooth preservation, yet the molecular mechanisms governing odontoblast differentiation remain poorly understood. In the current study, we revealed a distinct NKD1+ subpopulation exhibiting secretory odontoblast characteristics, which was specifically induced in dental pulp stem cells (DPSCs) by Wnt3a, but not by Wnt5a or Wnt10a through single-cell transcriptomic profiling. We then found that the NKD1+ subpopulation was evolutionary conservation, which were consistently identified in the odontoblast layers of developing tooth germs in both murine and miniature pig models, as well as within the apical open area in human molars. This conserved spatial distribution and co-localization with DSPP strongly indicates that NKD1+ cells were active dentin-secreting odontoblasts. Analysis of gene regulatory networks using SCENIC identified MSX1 as a key transcription factor regulating the specification of NKD1+ lineage. Mechanistically, Wnt3a orchestrates a tripartite cascade: upregulating NKD1/MSX1 expression, triggering NKD1 membrane detachment, and facilitating direct NKD1-MSX1 interaction to promote MSX1 nuclear translocation. CUT&Tag analysis demonstrated MSX1 occupancy at promoters of odontogenic regulators, establishing its necessity for odontogenic gene activation. Murine pulp exposure models validated that Wnt3a-activated NKD1-MSX1 signaling significantly enhances reparative dentin formation. This study delineates an evolutionarily conserved Wnt3a-NKD1-MSX1 axis that resolves stem cell heterogeneity into functional odontoblast commitment, providing both mechanistic insights into dentin-pulp regeneration and a foundation for targeted regenerative therapies.

TROP2 Expression and Its Association with Clinicopathological Features and Biomarkers in  
Gastric and GEJ Adenocarcinomas胃および胃食道接合部腺癌におけるTROP2発現と臨床病理学的特徴およびバイオマーカー  
との関連性

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## 【Abstract】

**Background:** Trophoblast cell surface antigen 2 (TROP2), a transmembrane glycoprotein, is expressed highly in solid tumors including gastric or gastroesophageal junction (GEJ) cancer (GC/GEJC). It is expected to become a novel therapeutic biomarker in the treatment of patients with metastatic GC/GEJC (mGC/GEJC). A randomized phase III trial of a TROP2-targeting ADC is currently underway in the third-line or later treatment setting for patients with mGC/GEJC. However, the characterization of TROP2 and its co-expression with currently actionable biomarkers in GC/GEJC remains unknown.

**Methods:** We conducted a single-institute retrospective cohort study of patients who enrolled in a prospective biomarker analysis at the National Cancer Center Hospital East between July 2024 and July 2025. Appropriate cutoff levels for TROP2 expression by immunohistochemistry (IHC) were explored, using different thresholds of  $\geq 25\%$ ,  $\geq 50\%$ , and  $\geq 75\%$  of tumor cells exhibiting moderate to strong staining intensity. Patient characteristics, including select biomarkers (HER2, MMR, EBV, CLDN18.2, and PD-L1), as well as clinical outcomes, were compared between TROP2-high and TROP2-low groups.

**Results:** A cohort of 272 patients with GC/GEJC was analyzed (full cohort), of whom 169 were initially diagnosed with mGC/GEJC (metastatic cohort). High TROP2 expression defined by cutoffs of  $\geq 25\%$ ,  $\geq 50\%$  and  $\geq 75\%$  was observed in 87.1%, 79.4% and 62.1% in full cohort and 86.4%, 78.7% and 60.4% in metastatic cohort, respectively. TROP2 expression showed no significant association with age, sex, tumor location (GEJ vs. gastric), Borrmann classification (Type 4 vs. others), or metastatic sites. However, higher TROP2 expression was associated with enrichment of intestinal-type histology and non-signet-ring cell carcinoma (non-SRCC). In metastatic cohort, TROP2-high ( $\geq 75\%$ ) was observed in 50.9% of non-SRCC, which was significantly higher than 26% observed in TROP2-low cases. TROP2 high expression was independent of dMMR, EBV-positive, CLDN18.2-positive, and PD-L1-positive irrespective to cutoff levels, while HER2-positive GC/GEJC was more frequently observed in the TROP2-high group ( $\geq 75\%$ ). The dual positive case was calculated as 11.0% in full cohort and 11.2% in metastatic cohort.

**Conclusions:** High TROP2 expression correlates with intestinal-type, non-SRCC and HER2-positivity in patients with GC/GEJC. Further investigation into clinical outcome will be discussed in the presentation.

**Keywords:** Gastric cancer, TROP2, clinicopathologic feature, biomarkers

## 『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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