



東北大学 加齢医学研究所 概要

Institute of Development, Aging and Cancer
Tohoku University



目次 Content

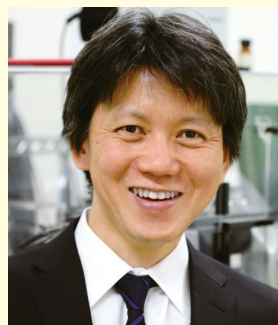
所長あいさつ Greeting & Mission	01	脳科学研究部門 Division of Brain Science	28
加齢医学研究所のミッション Mission of the IDAC	02	応用脳科学研究分野 Dept. Functional Brain Imaging	28
沿革 History	02	人間脳科学研究分野 Dept. Human Brain Science	30
歴代所長 Past Directors	03	臨床加齢医学研究分野 Dept. Nuclear Medicine and Radiology	32
組織図 Organization	04	統合生理学研究分野 Dept. Integrative Physiology	34
共同利用・共同研究拠点「加齢医学研究拠点」 Joint Usage/Research Center on Aging	05	スマート・エイジングセンター Smart-Aging Center	36
加齢制御研究部門 Division of Aging Science	06	附属医用細胞資源センター Cell Resource Center for Biomedical Research	38
遺伝子発現制御分野 Dept. Gene Expression Regulation	06	非臨床試験推進センター Pre-Clinical Research Center	38
遺伝子導入研究分野 Dept. Experimental Immunology	08	環境ストレス老化研究センター Center for Environmental Response and Aging	39
生体防御学分野 Dept. Immunobiology	10	脳 MRI センター Brain MRI Center	40
モドミクス医学分野 Dept. Modomics Biology and Medicine	12	先端研究小動物 MRI センター Small Animal MRI Center for Research Use with Technical Advantage	40
生体情報解析分野 Dept. Integrative Bioanalyticse	14	マルチオミクスコアファシリティセンター Multi-omics Core Facility	40
加齢生物学分野 Dept. Aging Biology	16	共通施設	41
老化制御学分野 Dept. of Aging Systems Science	18	実験動物管理室 Laboratory Animal Resources	41
イン・シリコ解析研究分野 Dept. In Silico	20	共通機器管理室 Center of Research Instruments	41
腫瘍制御研究部門 Division of Cancer Science	22	広報情報室 Public Relations and Information Office	41
分子腫瘍学研究分野 Dept. Molecular Oncology	22	国際戦略室 International Strategy Office	41
腫瘍生物学分野 Dept. Cancer Biology	24	各種データ Various data	42
呼吸器外科学分野 Dept. Thoracic Surgery	26	アクセスマップ Acces Map	44

所長あいさつ

Message from the Director

所長
田中 耕三

Director
Kozo Tanaka



日本人の平均寿命は戦後70年間で30年以上延長し、私たちは100年前と比べて倍近い人生を生きられるようになりました。一方65歳以上の高齢者率が30%に迫ろうとする超高齢社会においては、高齢化にともなう様々な医学的・社会的問題が顕在化しています。私たちは、これらの問題に対処するための目標として、スマート・エイジング社会の実現を掲げています。「スマート・エイジング」とは、加齢による変化に賢く対処し、個人・社会が知的に成熟することを意味し、超高齢社会において私たちが活力を持続するための鍵となる考え方です。

加齢医学研究所では、生命の誕生から発達、成熟、老化、死に至る加齢の基本的メカニズムを解明するために、分子生物学的手法を用いた遺伝子や細胞の研究から、動物実験による個体レベルの研究、さらにヒトを対象とした研究まで、多階層的な医学研究を行なっています。そしてこれらの研究によって、難治性がんの克服や認知症の予防などを目指しています。当研究所は、加齢制御研究部門、腫瘍制御研究部門、脳科学研究部門の3つの部門に加えて、環境ストレス老化研究センター、非臨床試験推進センター、脳MRIセンター、先端研究小動物MRIセンター、医用細胞資源センターを擁し、多彩な研究活動を行なっています。また認知症ゼロ社会実現を目指すスマート・エイジング学際重点研究センター、認知神経科学の社会実装を目指す応用認知神経科学センターと密接に連携しています。さらに当研究所は、超高齢社会における健康長寿の実現に向けて、文部科学省から加齢医学研究拠点に認定されており、加齢医学の中核的センターとして先導的な国内外の共同研究を展開しています。

高度高齢化社会への対応は、先進国を中心として各国共通の課題として認識されており、私たちはスマート・エイジング、すなわち全ての人が高齢期を積極的に受容し、社会で活動し続けることを可能とする方策を示すことによって、世界を先導する研究拠点となることを目指します。

The average life expectancy in Japan has increased by over 30 years since the end of World War II, and people now live almost twice as long as they did 100 years ago. However, in a super-aging society where 30% of the population is aged 65 and older, various medical and social challenges associated with aging are becoming apparent. To address these challenges, we aim to create a smart aging society, where “smart aging” refers to the intellectual growth of individuals and society by effectively adapting to the changes brought about by aging.

To understand the fundamental mechanisms of aging from birth through development, maturation, aging, and death, the Institute of Development, Aging and Cancer (IDAC) conducts research ranging from genetic and cellular studies using molecular biological techniques to organismal-level research through animal experiments and comprehensive medical studies involving human subjects. These studies aim to conquer intractable cancers and prevent dementia. In addition to its three main research areas—Aging Science, Cancer Science, and Brain Science—IDAC also includes the Center for Environmental Response and Aging, the Pre-Clinical Research Center, the Brain MRI Center, the Small Animal MRI Center for Research Use with Technical Advantage, and the Cell Resource Center for Biological Research. Furthermore, we are closely collaborating with the Smart Aging Research Center (S.A.R.C.), which aims to create a dementia-free society, and the Cognitive Neuroscience Application Center, which seeks to apply cognitive neuroscience to societal challenges. Lastly, IDAC has been designated as a research center for aging medicine by Japan’s Ministry of Education, Culture, Sports, Science and Technology (MEXT), to promote healthy longevity in a super-aging society.

Addressing the challenges of a rapidly aging society is recognized as a common issue for all countries, particularly in developed nations. Our ultimate goal is to create a smart aging society, and we aspire to become a world-leading research center in managing the complexities of a super-aging society.



加齢医学研究所のミッション

Mission of the IDAC

加齢医学研究所は、生命の誕生から発達、成熟、老化、死に至る加齢の基本的メカニズムを解明しています。得られた研究成果を応用して、加齢に伴う認知症などの脳・神経疾患や難治がんなどの克服を目指し、先端予防・診断・治療法や革新的医療機器の開発を行っています。

さらに、加齢医学の中核的研究センターとして先導的な国際共同研究を展開し、世界をリードする拠点であることを目指します。

The Institute of Development, Aging, and Cancer (IDAC) is dedicated to elucidating the basic mechanisms of aging, from birth through development, maturity, aging, and death. By applying research findings, the institute aims to overcome age-related brain and neurological diseases such as dementia and refractory cancers, and to develop advanced methods for prevention, diagnosis, treatment, and innovative medical devices. Additionally, as a core research center for aging medicine, the institute aims to lead pioneering international collaborative research and establish itself as a world-leading hub.

沿革 History

1941.12.15	抗酸菌病研究所設置 Research Institute of Tuberculosis and Leprosy founded.
1966.7.1	抗酸菌病研究所附属病院開院 Research Institute of Tuberculosis and Leprosy Hospital opened.
1977.3.23	現在地に研究所棟新築・移転 The institute was relocated to a new building at the present location.
1980.3.31	現在地に附属病院棟新築・移転 The Hospital was relocated to a new building at the present location.
1993.4.1	抗酸菌病研究所を加齢医学研究所に改組 Reorganised as Institute of Development, Aging and Cancer (IDAC)
	抗酸菌病研究所附属病院を加齢医学研究所附属病院に名称変更 Name changed to IDAC Hospital
1997.4.1	附属医用細胞資源センター設置 Cell Resource Center for Biomedical Research founded.
2000.4.1	加齢医学研究所附属病院は医学部附属病院と統合 IDAC Hospitals have merged with the Hospital of the Faculty of Medicine Hospital.
2004.4.1	国立大学法人東北大学に移行 Transitioned to a national university corporation.

2009.6.25	共同利用・共同研究拠点「加齢医学研究拠点」に認定 The Institute was accredited as a Joint Usage/Research Center.
2009.10.1 (~2017.3)	附属スマート・エイジング国際共同研究センター設置 Smart Ageing International Research Centre founded (Attached to the Institute) .
2015.4.1	附属非臨床試験推進センター設置 Pre-Clinical Research Center founded.
2016.10.1 (~2017.3)	スマート・エイジング学際研究重点拠点の開所 Smart Ageing International Research hub founded.
2017.4.1	スマート・エイジング学際研究重点拠点から、スマート・エイジング学際重点研究センターへ改組 Smart Ageing International Research Centre founded.
2019.10.10	附属環境ストレス老化研究センター設置 Center for Environmental Response and Aging founded.
2020.8.1	附属脳MRIセンター設置 Brain MRI Center founded.
2024.6.1	附属先端研究小動物MRIセンター設置 Small Animal MRI Center for Research Use with Technical Advantage founded.
2025.10.16	附属マルチオミクスコアファシリティセンター設置 Multi-Omics Core Facility Center founded.
2026.4.1	附属スマート・エイジングセンター設置 Smart-Aging Center founded.

歴代所長 Past Directors

抗酸菌病研究所長

Directors of Research Institute of Tuberculosis and Leprosy

1941.12.15 - 1948.03.30	熊谷 岱蔵	Taizo Kumagai
1948.03.31 - 1963.03.31	海老名敏明	Toshiaki Ebina
1963.04.01 - 1968.03.31	佐藤 三郎	Saburo Sato
1968.04.01 - 1974.03.31	鈴木千賀志	Chikashi Suzuki
1974.04.01 - 1978.04.01	斎藤 達雄	Tatsuo Saito
1978.04.02 - 1984.04.01	佐藤 春郎	Haruo Sato
1984.04.02 - 1987.04.01	今野 淳	Kiyoshi Konno
1987.04.02 - 1990.03.31	立木 蔚	Shigeru Tsuiki
1990.04.01 - 1991.03.31	涌井 昭	Akira Wakui
1991.04.01 - 1992.03.31	本宮 雅吉	Masakichi Motomiya
1992.04.01 - 1993.03.31	渡辺 民朗	Minro Watanabe



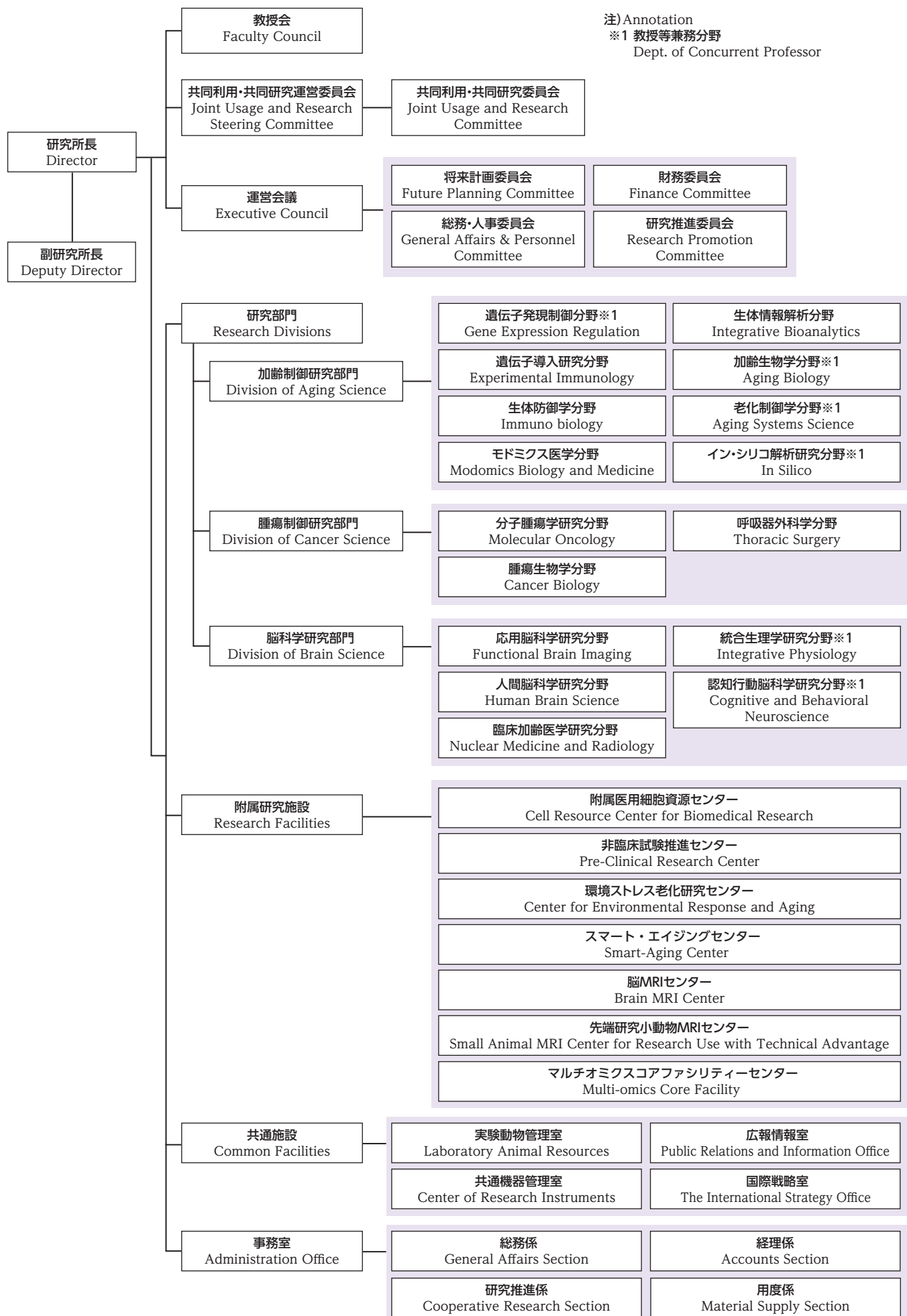
加齢医学研究所長

Directors of Institute of Development, Aging and Cancer

1993.04.01 - 1996.03.31	渡辺 民朗	Minro Watanabe
1996.04.01 - 1998.03.31	今野 多助	Tasuke Konno
1998.04.01 - 2000.03.31	藤村 重文	Shigefumi Fujimura
2000.04.01 - 2006.03.31	帯刀 益夫	Masuo Obinata
2006.04.01 - 2012.03.31	福田 寛	Hiroshi Fukuda
2012.04.01 - 2014.03.31	佐竹 正延	Masanobu Satake
2014.04.01 - 2023.03.31	川島 隆太	Ryuta Kawashima
2023.04.01 -	田中 耕三	Kozo Tanaka



組織図 Organization



共同利用・共同研究拠点 加齢医学研究拠点

当拠点は、超高齢社会における健康長寿の実現に向けて、加齢医学の中核的研究センターとして先導的な国内外の共同研究を展開します。

1. 共同利用・共同研究拠点名 「加齢医学研究拠点」

2. 認定有効期間 令和4年4月1日～令和10年3月31日（令和3年10月29日認定）

3. 拠点の概要

加齢医学研究拠点では、生命の誕生から発達、成熟、老化、死に至る加齢の基本的メカニズムを解明し、得られた研究成果を応用して加齢に伴う認知症などの脳・神経疾患や難治がんなどの加齢疾患の克服を目指します。さらに、先端予防・診断・治療法や革新的医療機器の開発を行い、超高齢社会における健康長寿の実現に向けて、加齢医学の中核的研究センターとして先導的な国内外の共同研究を展開します。

モデル動物を用いた加齢研究領域、加齢・発生分化・生体防御の基礎的研究領域、腫瘍制御研究領域、脳の発達・加齢研究領域、革新的医療機器開発の、五本柱の共同利用・共同研究を推進します。また、グローバル化、人材養成機能の強化、新分野創成、異分野融合研究の観点から、一層の機能強化を行っていきます。これにより、スマートエイジング社会の実現に貢献し、世界に新しい高齢社会のあり方を提示します。

Joint Usage / Research Center Center for Research on Aging

The Center for Research on Aging will develop leading national and international collaborative research as a core center for aging medicine, aiming to realize healthy longevity in a super-aged society.

1. Name of Joint Usage/Research Center : Center for Research on Aging

2. Period of Authorization : 2022.4.1 ~ 2028.3.31 (authorized on 2021.10.29)

3. Overview The Center for Research on Aging aims to elucidate the basic mechanisms of aging, from the birth of life through development, maturation, aging, and death. It seeks to apply the research findings to overcome age-related diseases such as dementia, brain and neurological disorders, and intractable cancers. Additionally, the Center will develop advanced preventive, diagnostic, and therapeutic methods, as well as innovative medical devices. It will conduct leading national and international collaborative research as a core center for aging medicine, with the goal of achieving healthy longevity in a super-aged society.

The Center will promote joint use and research in the following five areas:

- Aging research using animal models
- Basic research on aging, developmental differentiation, and biological defense
- Tumor control research
- Brain development and aging research
- Development of innovative medical devices

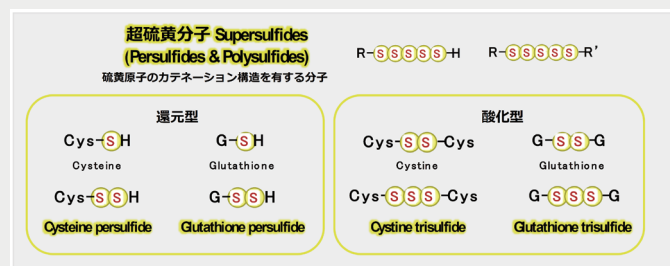
Furthermore, the Center will enhance its functions from the perspectives of globalization, human resource development, creation of new fields, and interdisciplinary research. In this way, we aim to contribute to the realization of a smart aging society and present a new model of an aging society to the world.

レドックス代謝の生化学から 遺伝子発現の理解へ

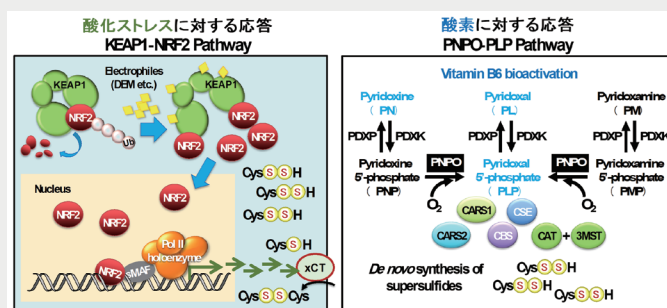
Redox metabolism underlying gene expression regulation



電子の動き、すなわち、酸化還元反応（レドックス反応）は生命の最も根源的な営みで、生命の営みを支えるエネルギー代謝を担うとともに、酸素に富んだ現在の地球環境における恒常性維持機構を担っています。レドックス恒常性の攪乱は、生体にストレスをもたらし、それに対する応答を誘導します。私たちの研究室では、急速に社会の高齢化がすすみつつある現在、健康寿命の延伸が医学にとっての極めて重要な課題であるとの認識のもと、環境にいかに対応して生命を維持するかという生体のストレス応答機構の理解を目指しています。



近年明らかにされた新規生体分子である超硫黄分子



超硫黄分子の生体内合成に関わる分子機構

主な研究テーマ

- 超硫黄分子の役割とその産生制御機構
- カルコゲン代謝が担う酸素応答機構
- 難治性がんにおける還元ストレスの意義と治療への応用
- 代謝制御から理解する炎症性腸疾患病態

研究キーワード

硫黄代謝 / KEAP1-NRF2 経路 / PNPO-PLP 経路 /
酸化ストレス / 還元ストレス

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2. Sekine H, Akaike T, Motohashi H. Oxygen needs sulfur, sulfur needs oxygen: a relationship of interdependence. *EMBO J.* 2025 Jun;44(12):3307-3326. doi: 10.1038/s44318-025-00464-7.
3. Sekine H, Takeda H, Takeda N, Kishino A, Anzawa H, Isagawa T, Ohta N, Murakami S, Iwaki H, Kato N, Kimura S, Liu Z, Kato K, Katsuoka F, Yamamoto M, Miura F, Ito T, Takahashi M, Izumi Y, Fujita H, Yamagata H, Bamba T, Akaike T, Suzuki N, Kinoshita K, Motohashi H. PNPO-PLP axis senses prolonged hypoxia in macrophages by regulating lysosomal activity. *Nat Metab.* 2024 Jun;6(6):1108-1127. doi: 10.1038/s42255-024-01053-4.
4. Takeda H, Murakami S, Liu Z, Sawa T, Takahashi M, Izumi Y, Bamba T, Sato H, Akaike T, Sekine H, Motohashi H. Sulfur metabolic response in macrophage limits excessive inflammatory response by creating a negative feedback loop. *Redox Biol.* 2023 Jul 29;65:102834. doi: 10.1016/j.redox.2023.102834.
5. Alam MM, Kishino A, Sung E, Sekine H, Abe T, Murakami S, Akaike T, Motohashi H. Contribution of NRF2 to Sulfur Metabolism and Mitochondrial Activity. *Redox Biol.* 60, 102624, 2023. doi: 10.1016/j.redox.2023.102624.



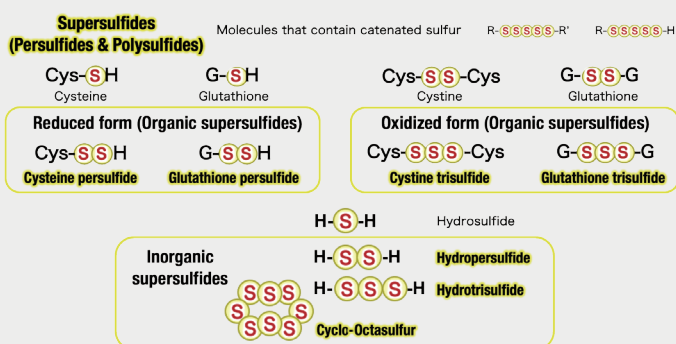
教授 (兼務)
本橋 ほづみ

Concurrent Professor
Hozumi Motohashi

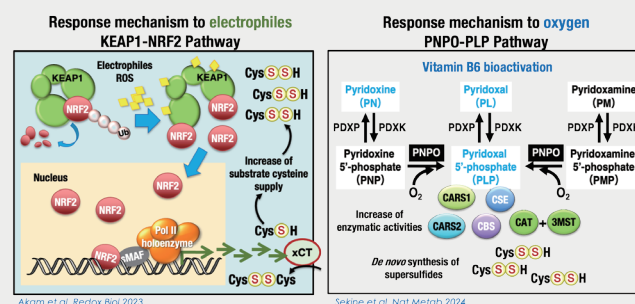


The movement of electrons—that is, redox (oxidation-reduction) reactions—constitutes one of the most fundamental processes of life. Redox reactions not only drive energy metabolism, which sustains all biological activities, but also underpin the mechanisms that maintain homeostasis in the oxygen-rich environment of present-day Earth. Disruption of redox homeostasis imposes stress on living systems and triggers adaptive responses.

In our laboratory, recognizing that the extension of healthy life expectancy is one of the most pressing challenges in medicine in this rapidly aging society, we seek to understand the biological stress response mechanisms that enable organisms to adapt to environmental changes and sustain life.



Supersulfides as a New Entity of Biomolecules



Two regulatory Pathways for Synthesis of Cysteine Persulfide as a Primary Supersulfide

Research Topics

- Physiological and pathological roles of supersulfides and regulatory mechanisms of their synthesis
- Chalcogen metabolism for stress response to oxygen
- Biological and therapeutic significance of reductive stress in intractable cancers
- Pathological basis of inflammatory bowel diseases emerging from metabolic rewiring

Research Interests

Sulfur metabolism / KEAP1-NRF2 pathway
PNPO-PLP pathway / Oxidative stress
Reductive stress

Selected Publications

1. Sung E, Murakami S, Morita M, Ida T, Akaike T, Motohashi H. Mitochondria regulate the cell fate decisions of megakaryocyte-erythroid progenitors. *Stem Cell Rep.* 2025 Nov 20;102720. doi: 10.1016/j.stemcr.2025.102720.
2. Sekine H, Akaike T, Motohashi H. Oxygen needs sulfur, sulfur needs oxygen: a relationship of interdependence. *EMBO J.* 2025 Jun;44(12):3307-3326. doi: 10.1038/s44318-025-00464-7.
3. Sekine H, Takeda H, Takeda N, Kishino A, Anzawa H, Isagawa T, Ohta N, Murakami S, Iwaki H, Kato N, Kimura S, Liu Z, Kato K, Katsuoka F, Yamamoto M, Miura F, Ito T, Takahashi M, Izumi Y, Fujita H, Yamagata H, Bamba T, Akaike T, Suzuki N, Kinoshita K, Motohashi H. PNPO-PLP axis senses prolonged hypoxia in macrophages by regulating lysosomal activity. *Nat Metab.* 2024 Jun;6(6):1108-1127. doi: 10.1038/s42255-024-01053-4.
4. Takeda H, Murakami S, Liu Z, Sawa T, Takahashi M, Izumi Y, Bamba T, Sato H, Akaike T, Sekine H, Motohashi H. Sulfur metabolic response in macrophage limits excessive inflammatory response by creating a negative feedback loop. *Redox Biol.* 2023 Jul 29;65:102834. doi: 10.1016/j.redox.2023.102834.
5. Alam MM, Kishino A, Sung E, Sekine H, Abe T, Murakami S, Akaike T, Motohashi H. Contribution of NRF2 to Sulfur Metabolism and Mitochondrial Activity. *Redox Biol.* 60, 102624, 2023. doi: 10.1016/j.redox.2023.102624.

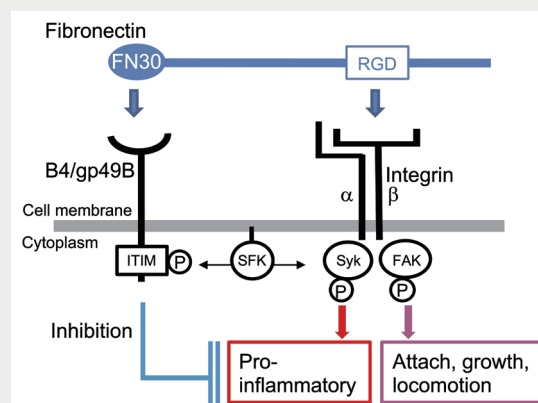
免疫抑制レセプターによる 免疫関連疾患の制御機構の解明と創薬

Drug discovery by elucidation of inhibitory receptor-mediated immune regulation



過去25年間の研究内容としては一貫して免疫抑制レセプターのはたらきをアレルギーや自己免疫疾患、遺伝性疾患との関係において解析して参りました。この間、抑制性 Fc レセプターや独自に発見した新しい抑制性レセプター PIR-B の機能が不十分になることが、全身性エリテマトーデス(SLE)に代表されるような自己免疫疾患の素因となることを報告しました。またミエロイド系細胞に発現する LILRB4 という抑制性レセプターが SLE 患者の自己抗体を産生する病原性プラズマセル表面に異所性に高発現するという知見から、LILRB4発現が病原性を持つ意外な一面も大学病院の先生方との共同で報告しました。このような免疫機構の変調が骨形成や認知症にも関与する可能性を示すこともできました。

現在は、がん・SLE・神経変性疾患の克服に向け、私たちの研究の総仕上げとして抑制レセプターを標的とした創薬研究に注力します。たとえば負の方向への免疫制御は、生体が本来持っているがんに対してはたらく免疫を弱めるという「免疫チェックポイント」のコンセプトが指摘されていましたが、これを阻害すると実際にがんに明確な治療効果が見られることが実証され臨床応用されています。私たちの研究していたLILRB4もがん免疫に関与するのではないかという単純な発想で調べてみますと、明らかにこれを阻害するとがん免疫が増強しました。また私たちは独自に、20年ほど謎のまま残されていたLILRB4の生理的リガンドタンパク質が、フィブロネクチンN末端30キロダルトン(FN30)であることを突き止めました。私たちはFN30とLILRB4の結合阻害ががんやSLEの抗体医薬として社会実装される可能性に期待して特許を申請し、LILRB4抗体医薬を実際にごん患者さまにお届けできる日が来ることを切に願っています。



図：免疫チェックポイント LILRB4 は細胞外マトリクスであるフィブロネクチンと結合し、インテグリンの活性化と炎症を抑制する

主な研究テーマ

- LILRB の生理的リガンドの探索
- 免疫チェックポイントとしての LILRB の機能解析
- 免疫チェックポイント阻害抗体医薬の開発

研究キーワード

免疫抑制 / 免疫チェックポイント / がん免疫療法
自己免疫疾患 / アルツハイマー病

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2. Itoi S et al. Myeloid immune checkpoint ILT3/LILRB4/gp49B can co-tether fibronectin with integrin on macrophages. Int Immu (2022) 34, 435.
3. Takahashi N et al. Co-localization of fibronectin receptors LILRB4/gp49B and integrin on dendritic cell surface. Tohoku J Exp Med (2022) 257, 171.
4. Su MT et al. LILRB4 promotes tumor metastasis by regulating MDSC-mediated immunosuppression and inhibiting anti-tumor exosomal miRNA secretion. Oncoimmunol (2022) 11, 2060907.
5. Su M-T et al. Blockade of checkpoint ILT3/LILRB4/gp49B binding to fibronectin ameliorates autoimmune disease in BXSB/Yaa mice. Int Immu (2021) 33, 447.



特任教授 (研究)

高井 俊行

Professor

Toshiyuki Takai



助教

与謝 玲

Assistant Professor

Rei Yosa

For the past 25 years, we have consistently analyzed the function of immune regulatory receptors in relation to allergy, autoimmune diseases, and genetic disorders. During this period, we have reported that inadequate function of the inhibitory Fc receptor and the novel inhibitory receptor PIR-B, which we originally discovered, predispose to autoimmune diseases such as systemic lupus erythematosus (SLE). In collaboration with doctors at a university hospital, we also reported that LILRB4, an inhibitory receptor expressed on myeloid cells, is ectopically highly expressed on the surface of pathogenic plasma cells that produce autoantibodies in SLE patients, an unexpected aspect of LILRB4 expression that has pathogenic properties. We were also able to show that such modulation of immune mechanisms may also be involved in bone formation and dementia. Now, we will focus on drug discovery research targeting regulatory receptors as the culmination of our research to overcome cancer, SLE, and neurodegenerative diseases. For example, the concept of “immune checkpoints” has been pointed out as a way to negatively regulate the immune system, weakening the body’s natural immunity against cancer. Based on the simple idea that LILRB4 may also be involved in cancer immunity, we investigated LILRB4 and found that inhibition of LILRB4 clearly enhanced cancer immunity. We also independently discovered that the physiological ligand protein for LILRB4, which had remained a mystery for about 20 years, is the N-terminal 30 kilodalton of fibronectin (FN30). We have applied for a patent in anticipation of the possibility of social implementation of the binding inhibition of FN30 to LILRB4 as an antibody drug for cancer and SLE, and we sincerely hope that we will be able to deliver LILRB4 antibody drugs to cancer patients in the near future.

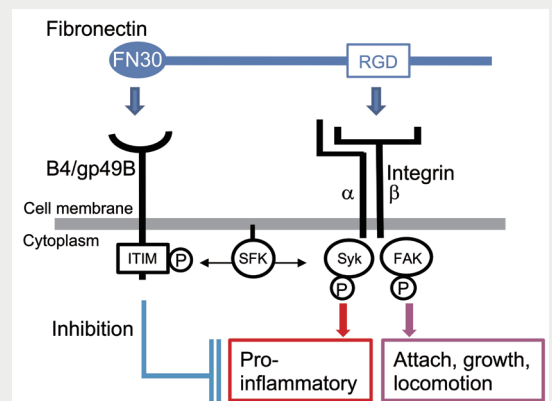


Figure: Immune checkpoint LILRB4 binds fibronectin, an extracellular component, thereby inhibiting integrin activation and proinflammatory signal.

Research Topics

- Identification of novel physiological ligand for LILRB
- Tumor immunity regulated by LILRB on monocytes
- Drug discovery for cancer by blocking immune checkpoint LILRB

Research Interests

Inhibitory receptor, immune checkpoint, cancer immunotherapy, autoimmune disease, Alzheimer's disease

Selected Publications

1. Itagaki F et al. Fibronectin on target cells attenuates natural cytotoxicity of NK cells via myeloid immune checkpoint ILT3/LILRB4/gp49B. *Int Immu* (2023) 35, 339.
2. Itoi S et al. Myeloid immune checkpoint ILT3/LILRB4/gp49B can co-tether fibronectin with integrin on macrophages. *Int Immu* (2022) 34, 435.
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4. Su MT et al. LILRB4 promotes tumor metastasis by regulating MDSC-mediated immunosuppression and inhibiting anti-tumor exosomal miRNA secretion. *Oncoimmunol* (2022) 11, 2060907.
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【基礎研究から医療へ】 最先端の免疫学で、未解明の病態に挑む

[From Bench to Bedside] Tackling unresolved disease mechanisms with cutting-edge immunology.

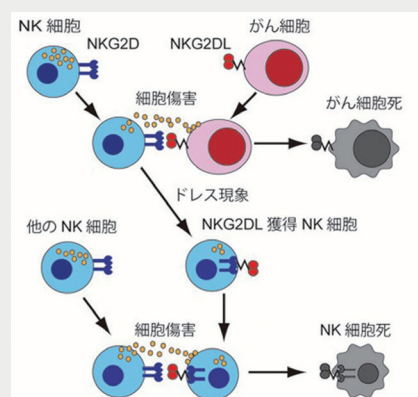


当分野は、今年で18年目を迎えます。私たちは、がん、自己免疫疾患、感染症などの疾患の克服を目指し、NK細胞やT細胞の認識機構を中心とした生体防御機構(免疫学)の研究を行っています。当分野の強みは、ゲノム編集や遺伝子組み換え技術を駆使し、モデル動物や細胞、人工抗体などを自ら作製して、新規診断・治療法の開発にまで踏み込める点にあります。

分野長の小笠原康悦はNK細胞研究における世界の第一人者であり、当分野の基幹研究として『Nature』や『Immunity』等のトップジャーナルに成果を発表しています。近年も、T細胞受容体の網羅的解析システムの開発(国際特許取得)や、トロゴサイトーシスによるNK細胞死の発見、新型コロナウイルスの変異酵素の発見、金属アレルギー発症機構の解明など、世界をリードする成果を次々と挙げています。所属する大学院生が日本学術振興会育志賞を受賞するなど、若手が世界レベルの研究に触れ、高く評価される環境が整っています。

さらに、渡辺数基助教を中心に「口腔と全身の相互作用」という新たな視点から、脳機能解析やシングルセル解析など最新の解析技術を用いた最先端の研究も展開しています。

これまで当研究室では、医学、歯学をはじめ、薬学、理学、工学、農学部など、実に多様なバックグラウンドを持つ学生が活躍してきました。研究に対する興味と熱意を持つ方を広く受け入れています。私たちと共に、世界を驚かせる研究の圧倒的な面白さと醍醐味を味わってみませんか？



図：がんを攻撃したNK細胞が細胞死に向かう仕組みの発見

主な研究テーマ

- NK細胞の機能と役割
- がんの免疫回避機構の解明
- トロゴサイトーシスの解明
- アレルギー発症機構の解明
- 口腔と全身の相互作用

研究キーワード

NK cell / NKG2D / cancer /
autoimmune / infection

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1. Watanabe, K., et al.: Highly purified hypochlorous acid water facilitates glucose metabolism and memory formation in type 2 diabetic mice associated with altered-gut microbiota. : Scientific Reports. 14, 1, 16107., 2024
2. Yasaka, K., et al.: Phospholipase D4 as a signature of toll-like receptor 7 or 9 signaling is expressed on blastic T-bet + B cells in systemic lupus erythematosus : Arthritis Research and Therapy. 25, 1, 200., 2023
3. Inamori, K., et al.: Importance of lymph node immune responses in MSI-H/dMMR colorectal cancer : JCI insight. 6, 9, e137365. 2021
4. Kosuge, M., et al.: Point mutation bias in SARS-CoV-2 variants results in increased ability to stimulate inflammatory responses: Scientific Reports. 10, 1, 17766., 2020
5. Takeda, K., et al.: IFN- γ is required for cytotoxic T cell-dependent cancer genome immunoediting: Nature Communications. 8, 14607., 2017



教授
小笠原 康悦

Professor
Koetsu Ogasawara



助教
渡辺 数基

Assistant Professor
Kazuki Watanabe

This year marks the 18th anniversary of our department. We conduct research on host defense mechanisms (immunology), focusing on the recognition mechanisms of NK cells and T cells, with the ultimate goal of overcoming diseases such as cancer, autoimmune disorders, and infectious diseases. A major strength of our laboratory is our ability to fully utilize genome editing and genetic recombination technologies to independently create original animal models, cells, and artificial antibodies. This capability allows us to advance our research all the way to the development of novel diagnostic methods and therapeutics.

Our department head, Professor Kouetsu Ogasawara, is a world-renowned pioneer in NK cell research, and our core findings are consistently published in top-tier journals such as Nature and Immunity. In recent years, we have continued to produce world-leading achievements, including the development of a comprehensive T cell receptor analysis system (for which we hold an international patent), the discovery of NK cell death caused by trogocytosis, the identification of the mutating enzyme of the novel coronavirus, and the elucidation of the pathogenic mechanisms behind metal allergies. We provide an exceptional environment where young researchers can engage in world-class science and earn high recognition, as demonstrated by one of our graduate students receiving the prestigious JSPS Ikushi Prize.

Furthermore, Assistant Professor Kazuki Watanabe is leading cutting-edge research from the novel perspective of "oral-systemic interactions". This innovative work actively incorporates the latest analytical techniques, including brain function analysis and single-cell analysis.

Throughout our history, students from highly diverse academic backgrounds—including medicine, dentistry, pharmacy, science, engineering, and agriculture—have thrived in our laboratory. We broadly welcome anyone who possesses a strong interest and deep enthusiasm for research. Why not join us and experience the profound excitement and true thrill of conducting research that astounds the world?

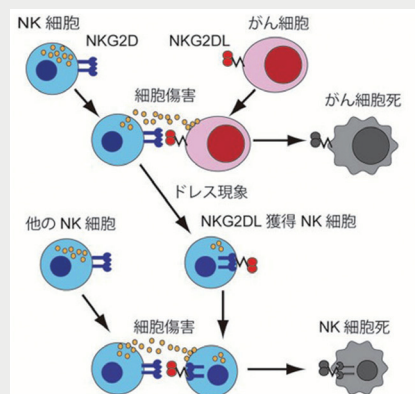


Figure: Discovery of NK cell death via trogocytosis triggered by cancer cell attack

Research Topics

- Functions and Roles of NK Cells
- Elucidation of Immune Evasion Mechanisms in Cancer
- Elucidation of Trogocytosis
- Elucidation of Pathogenic Mechanisms of Allergies
- Oral-Systemic Interactions

Research Interests

NK cell / NKG2D / cancer /
autoimmune / infection

Selected Publications

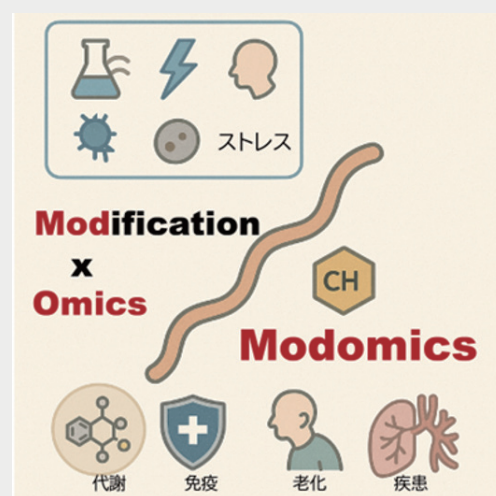
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RNA バイオロジーを通して 生命の理解と疾患治療をめざす

Understanding the complexity of life and developing treatments for diseases via RNA modification



我々の体を構成するたんぱく質、DNAやRNAといった生体分子は様々な化学修飾を受けることで新たな機能を獲得し、細胞や臓器の高次機能を制御します。また、外界からの化学的・物理的・生物学的なストレス、あるいは疾患や加齢などの内的要因が生体分子の修飾状態を変化させ、生体機能を障害し疾患発症の原因となります。これらの修飾の網羅的な解析を「モドミクス (Modification + Omics)」と名付け、私たちは中でも特にRNA修飾に注目し、先進的な質量分析装置やRNA解析手法を用いて、細胞・モデル動物・ヒトを研究材料として、RNAモドミクスと代謝・免疫・老化などの生理機能やヒト疾患病態との関わりについて研究を行っています。特に近年、RNAウイルスである新型コロナウイルスの世界的な蔓延に対して、化学修飾を人為的に導入したmRNAワクチンがパンデミックの制圧の原動力になっていることが示すように、RNAとRNAに付与される化学修飾は生命の営みを理解する上で不可欠な研究分野であり、次世代医療にとっても不可欠な技術となっています。私たちはRNAとその修飾を通して、生命の理解と疾患治療に貢献したいと考えて、日々研究を行っています。



主な研究テーマ

- RNA 修飾に基づく加齢性疾患の理解と制御
- ミトコンドリア核酸の分子基盤の理解と疾患応用

研究キーワード

RNA 修飾／核酸代謝／ミトコンドリア／質量分析

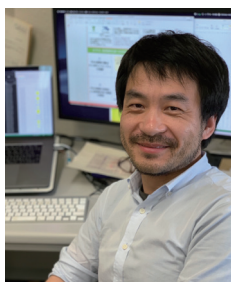
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2. Structural insights into the agonist selectivity of the adenosine A3 receptor. *Nat Commun*. 2024 Nov 7;15(1):9294.
3. N6-methyladenosine (m6A) is an endogenous A3 adenosine receptor ligand. *Mol Cell*. 2021;81(4):659-674.e7.
4. Loss of Ftsj1 perturbs codon-specific translation efficiency in the brain and is associated with X-linked intellectual disability. *Sci Adv*. 2021 Mar 26;7(13):e-abf3072.
5. Cdk5rap1-mediated 2-methylthio modification of mitochondrial tRNAs governs protein translation and contributes to myopathy in mice and humans. *Cell Metab*. 2015;21(3):428-442.



教授
魏 范研

Professor
Fan-Yan Wei



助教 松田 盛

Assistant Professor
Shigeru Matsuda

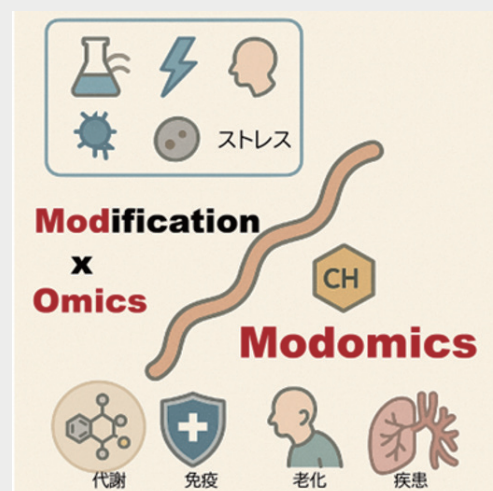
助教 谷 春菜

Assistant Professor
Haruna Tani

助教 Raja Norazireen Raja Ahmad

Assistant Professor
Raja Norazireen Raja Ahmad

Living organisms are made up of a complex array of biomolecules, including proteins, DNA, and RNA, which undergo a variety of chemical modifications in order to acquire new functions and exert control over the higher functions of cells and organs. External chemical, physical, and biological stresses, as well as internal factors, such as genetic variations and aging, can disrupt the state of these modifications, causing the onset of disease and disrupting biological functions. Our research focuses on RNA modifications in particular. By employing state-of-the-art mass spectrometry technology and RNA analysis methodologies, we aim to investigate how RNA modifications regulate physiological functions, such as metabolism and immunity. Additionally, we explore how the dysregulation of RNA modifications contributes to aging-related diseases, such as diabetes and inflammation using cells, animal models, and human subjects as research materials. Notably, the new generation of vaccines that contain chemically modified mRNA has been a driving force in the control of the COVID 19 pandemic. This underscores the essential nature of RNA and its modifications, which are key to understanding the complexity of life, as well as in the development of next-generation medicine. Our team conducts research every day with the hope of contributing to a better understanding of life and the development of new treatments for diseases through RNA and its modifications.



Research Topics

- RNA modification in human physiology and disease,
- Mitochondrial biology and disease

Research Interests

RNA modification / Mass spectrometry /
mitochondria / nucleic acid metabolism

Selected Publications

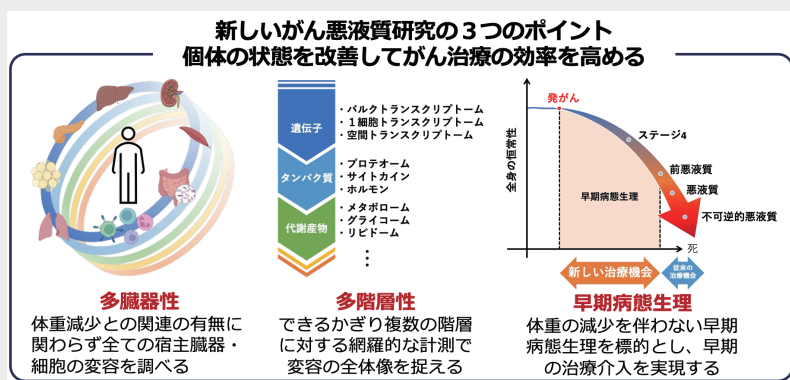
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5. Cdk5rap1-mediated 2-methylthio modification of mitochondrial tRNAs governs protein translation and contributes to myopathy in mice and humans. *Cell Metab*. 2015;21(3):428-442.

大規模データ解析によって 複雑な生命現象を理解する

Understanding Complex Biological Phenomena through Large-Scale Data Analysis



生体情報解析分野は、大規模データ解析を駆使して、がんによって個体が不調に陥るのはなぜか、日々のストレスと老化の関係はどのようなものか、デバイスの利用は生体にどのような影響をあたえるのか、といった問いに答えようとしています。近年の技術革新により、生命科学・医学分野で得ることのできる情報の量は飛躍的に増えました。いまやゲノム全体の配列を知ることは難しくありません。ある細胞における 2 万種類以上の遺伝子の発現量や数百の代謝物の量を網羅的に測定することもできます。個々人が持つ背景情報（年齢や性別、既往歴など）の蓄積にも目をみはるものがあります。技術革新の一方で、新たな課題も生まれました。大規模なデータが得られても、得られた情報が何を意味するのかを解釈することが難しい場合があるのです。異なる階層のデータ同士（たとえば遺伝子発現の情報と疾患の既往歴）を結びつけることが困難である場合も多々あります。解釈しやすいデータを得るためにはどうすれば良いか、得られた大規模データから有用な情報を抽出するにはどうすべきか、これらの基本的な問題を考えることの重要性が増しています。生体情報解析分野では、実験デザイン、大規模計測、そして情報解析までを一貫して行うことで大規模データを最大限に活用する枠組みを構築することを目指しています。



図：生体情報解析分野におけるがん悪液質研究の三つのポイント

主な研究テーマ

- がんによって個体に不調が生じるのはなぜか
- エンハンサーによる代謝・免疫・老化制御のメカニズム
- ヒトは日常生活でどのようなマルチオミクス変動を経験しているのか

研究キーワード

がん悪液質／マルチオミクス解析／エンハンサー
ヒト生物学／大規模データ解析

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1. Hojo et al. Nat Commun. 2019 Jun 13;10(1):2603. doi: 10.1038/s41467-019-10525-1.
2. Mizuno et al. Nat Commun. 2022 Jun 15;13(1):3346. doi: 10.1038/s41467-022-30926-z..
3. Vandenbon et al. Commun Biol. 2023 Jan 24;6(1):97. doi: 10.1038/s42003-023-04479-w.
4. Maeshima et al. eBioMedicine 2024 Sep;107:105271. doi: 10.1016/j.ebiom.2024.105271.
5. Nakamura et al. Cancer Sci. 2024 Mar;115(3):715-722. doi: 10.1111/cas.16078.



教授
河岡 慎平

Professor
Shinpei Kawaoka



助教
依田 真由子

Assistant Professor
Mayuko Yoda

Our laboratory seeks to answer fundamental questions by leveraging large-scale data analysis: Why does cancer lead to systemic dysfunction? How are daily stress and aging interconnected? What biological effects does device usage have on the human body? Recent technological advances have dramatically expanded the amount of information that can be obtained in life sciences and medicine. Sequencing an entire genome is no longer difficult. It is now possible to comprehensively measure the expression levels of more than 20,000 genes within a single cell, as well as hundreds of metabolites. In addition, extensive background information on individuals—such as age, sex, and medical history—can now be accumulated and analyzed. While technological innovation has greatly increased data availability, it has also introduced new challenges. Even when large-scale data are obtained, interpreting what the information truly means is often difficult. Integrating data across different layers—for example, linking gene expression profiles with clinical histories—remains particularly challenging. Addressing these fundamental questions has become increasingly important: How can we generate data that are inherently interpretable? How can we extract meaningful insights from complex, large-scale datasets? Our laboratory aims to build an integrated framework that maximizes the value of large-scale data by conducting experimental design, high-throughput measurement, and data analysis in a seamless and unified manner.

Research Topics

- Cancer cachexia
- Enhancer-dependent regulation of metabolism, immunity, and aging
- Multi-omics changes during daily human activities

Research Interests

Cancer Cachexia / Multi-Omics Analysis
Enhancer / Human Biology
Large-scale data analysis

Selected Publications

1. Hojo et al. Nat Commun. 2019 Jun 13;10(1):2603. doi: 10.1038/s41467-019-10525-1.
2. Mizuno et al. Nat Commun. 2022 Jun 15;13(1):3346. doi: 10.1038/s41467-022-30926-z..
3. Vandenbon et al. Commun Biol. 2023 Jan 24;6(1):97. doi: 10.1038/s42003-023-04479-w.
4. Maeshima et al. eBioMedicine 2024 Sep;107:105271. doi: 10.1016/j.ebiom.2024.105271.
5. Nakamura et al. Cancer Sci. 2024 Mar;115(3):715-722. doi: 10.1111/cas.16078.

マイクロバイオーーム、免疫系、細胞老化の クロストークから老化の本質を紐解く

Unveiling the Essence of Aging through the Crosstalk between the Microbiome, Immune System, and Cellular Senescence

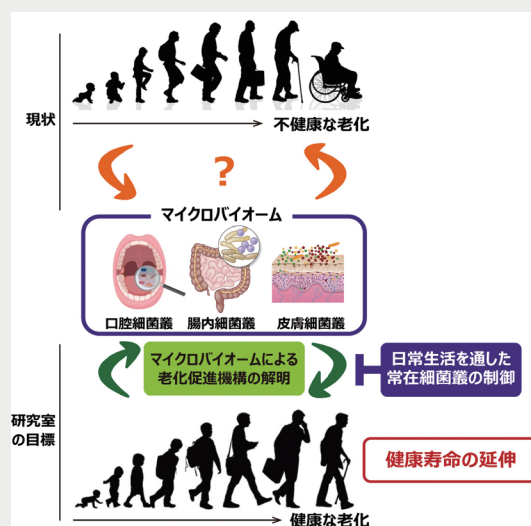


当研究室では、ヒトの体に共生する微生物群(マイクロバイオーーム)が、免疫系を介して老化プロセスにどのような影響を及ぼすのか、その詳細なメカニズムの解明に挑んでいます。

私たちは、老化を「マイクロバイオーーム・免疫系・細胞老化」の三者が織りなす相互作用の結果として捉えています。加齢に伴う細菌叢のバランスの乱れ(ディスバイオシス)は、免疫系の恒常性を揺るがすだけでなく、組織内での老化細胞の蓄積を促し、炎症性物質を分泌するSASP(老化関連分泌表現型)による慢性炎症の惹起を介して全身の老化を加速させることが示唆されています。本研究室では、腸管、口腔、皮膚という主要な共生部位を対象に、細菌叢の変化がいかにして免疫老化や細胞老化を誘発し、組織機能の低下を招くのかを多角的に解析しています。

1. 腸内細菌: 全身の免疫バランスを制御し、老化細胞の除去や蓄積に関する司令塔としての役割を解明します。
2. 口腔細菌: 慢性的な炎症波及が、いかにして遠隔組織の細胞老化や加齢性疾患を引き起こすのか、その病態連関を追究します。
3. 皮膚細菌: 外部環境との接点である皮膚をモデルとし、共生細菌が直接的に細胞老化へ介入する分子機序を特定します。

これらのクロストークを制御する機序を解き明かすことで、「健康寿命の延伸に向けた新たな介入法」の創出に貢献していきます。



図：研究室の最終到達目標：マイクロビオーームおよび免疫系の制御による健康寿命の延伸

主な研究テーマ

- 腸内細菌叢によるB細胞老化(B cell senescence)誘導メカニズムの解明
- 腸内細菌叢によるT細胞変容を介した慢性炎症(Inflammaging)の惹起と組織老化への影響の解明
- 皮膚常在細菌・免疫・細胞老化のクロストークによる皮膚老化メカニズムの解明

研究キーワード

マイクロバイオーーム／免疫系／細胞老化／老化

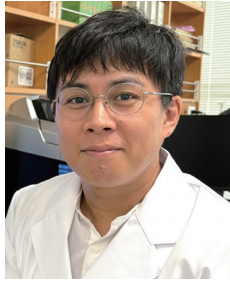
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2. Mizuno H, †Kawamoto S, Uemura K, Park JH, Hori N, Okumura Y, Konishi Y, †Hara E. B cell senescence promotes age-related changes in oral microbiota. Aging Cell, 2024;23(12): e14304. (†corresponding author)
3. †Kawamoto S, Hara E. Crosstalk between gut microbiota and cellular senescence: a vicious cycle leading to aging gut. Trends Cell Biol. 2024;34(8):626-635. (†corresponding author)
4. Matsudaira T, Nakano S, Konishi Y, Kawamoto S, Uemura K, Kondo T, Sakurai K, Ozawa T, Hikida T, Komine O, Yamanaka K, Fujita Y, Yamashita T, Matsumoto T, Hara E. Cellular senescence in white matter microglia is induced during ageing in mice and exacerbates the neuroinflammatory phenotype. Commun. Biol. 2023;6:665.
5. †Kawamoto S, Uemura K, Hori N, Takayasu L, Konishi Y, Katoh K, Matsumoto T, Suzuki M, Sakai Y, Matsudaira T, Adachi T, Ohtani N, Standley DM, Suda W, Fukuda S, †Hara E. Bacterial induction of B cell senescence promotes age-related changes in the gut microbiota. Nat. Cell Biol. 2023;25(6):865-876. (†corresponding author)



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Our laboratory is dedicated to elucidating the detailed mechanisms by which the human microbiome influences the aging process via the immune system. We perceive aging as a complex outcome of the tripartite interaction between the microbiome, the immune system, and cellular senescence.

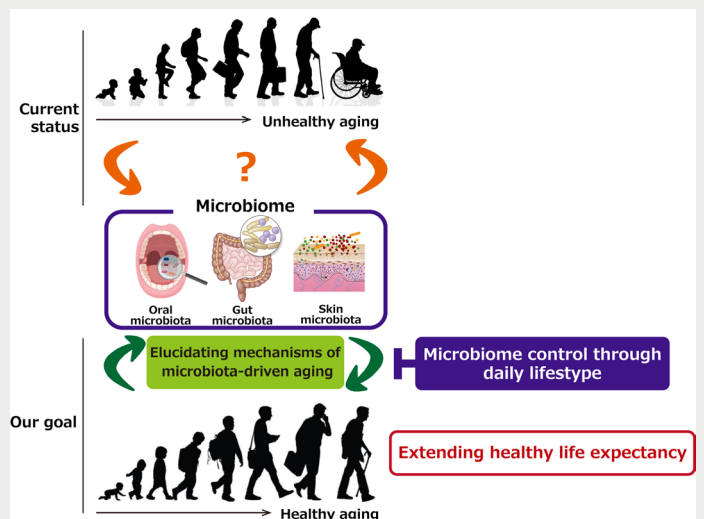
Emerging evidence suggests that age-related dysbiosis—the disruption of microbial balance—not only destabilizes immune homeostasis but also promotes the accumulation of senescent cells. These cells, in turn, accelerate systemic aging by inducing chronic inflammation through the Senescence-Associated Secretory Phenotype (SASP). Focusing on major symbiotic sites including the intestinal tract, oral cavity, and skin, we employ multifaceted approaches to analyze how microbial shifts trigger immunosenescence and cellular senescence, ultimately leading to the decline of tissue function.

1. Gut Microbiota: We investigate its role as a "master regulator" that governs systemic immune balance and modulates the clearance or accumulation of senescent cells.

2. Oral Microbiota: We explore the pathological links through which the systemic dissemination of chronic inflammation triggers cellular senescence in remote tissues and contributes to age-related diseases.

3. Skin Microbiota: Utilizing the skin—the primary interface with the external environment—as a model, we identify the molecular mechanisms by which symbiotic bacteria directly intervene in cellular senescence.

By deciphering the mechanisms that control these complex crosstalks, we aim to contribute to the development of novel interventions for extending healthy life expectancy.



Ultimate Goal: Extending healthy life expectancy by modulating the microbiome and the immune system.

Research Topics

- Elucidating the mechanisms of B-cell senescence induction by the gut microbiota
- Elucidating the induction of chronic inflammation (inflammaging) via T-cell alteration driven by the gut microbiota and its impact on tissue aging
- Deciphering the mechanisms of skin aging driven by the crosstalk between skin bacteria, the immune system, and cellular senescence

Research Interests

Microbiome / Immune system /
Cellular senescence / Aging

Selected Publications

1. †Kawamoto S, Horiguchi H, Torigoe D, Wakita M, Ito K, Sugawara S, Zhou X, Mikawa T, Park JH, Jung BK, Okumura Y, Miyagawa H, Maruya M, Hori N, Uemura K, Sugimoto M, Matsuda M, Mochizuki N, Kondoh H, Takahashi A, Oike Y, †Hara E. Reevaluating the senolytic activity of a GLS1 inhibitor and an anti-PD-1 antibody: toward greater reproducibility and methodological rigor. *EMBO Rep.*, 2026; Online ahead of print. († corresponding author)
2. Mizuno H, †Kawamoto S, Uemura K, Park JH, Hori N, Okumura Y, Konishi Y, †Hara E. B cell senescence promotes age-related changes in oral microbiota. *Aging Cell*, 2024;23(12): e14304. († corresponding author)
3. †Kawamoto S, Hara E. Crosstalk between gut microbiota and cellular senescence: a vicious cycle leading to aging gut. *Trends Cell Biol.* 2024;34(8):626-635. († corresponding author)
4. Matsudaira T, Nakano S, Konishi Y, Kawamoto S, Uemura K, Kondo T, Sakurai K, Ozawa T, Hikida T, Komine O, Yamanaka K, Fujita Y, Yamashita T, Matsumoto T, Hara E. Cellular senescence in white matter microglia is induced during ageing in mice and exacerbates the neuroinflammatory phenotype. *Commun. Biol.* 2023;6:665.
5. †Kawamoto S, Uemura K, Hori N, Takayasu L, Konishi Y, Katoh K, Matsumoto T, Suzuki M, Sakai Y, Matsudaira T, Adachi T, Ohtani N, Standley DM, Suda W, Fukuda S, †Hara E. Bacterial induction of B cell senescence promotes age-related changes in the gut microbiota. *Nat. Cell Biol.* 2023;25(6):865-876. († corresponding author)

オミクスデータ駆動型研究から老化の本質を捉え 介入による老化抑制を以てこれを証明する

Definition of the fundamental nature of aging through omics-data-driven research and its validation through interventions that suppress aging.



老化制御学分野では加齢に伴う遺伝子発現変化の包括的な評価に基づき、まだ見ぬ老化の本質に迫る omics-data driven research を展開していきます。独自のアプローチとして現在、遺伝子発現について単一種内での年齢との相関情報と、種間における寿命との相関情報の複合解析から、加齢変化の有害な側面と有益な側面を推測・実証していく研究を行っています。また別に最も注力しているアプローチとして、老化プロテオーム、ならびに加齢性転写後調節異常の評価と、それに基づく新規老化、寿命制御機構の解明に取り組んでいます。老化プロテオームの主要な特徴として APOE やイムノグロブリンの蓄積を認めており、それらを切り口とした老化メカニズムの理解と老化抑制法の開発研究を先行させています。

主な研究テーマ

- 種間比較アプローチに基づく加齢変化の有害な側面と適応的な側面の区別
- プロテオーム解析を主軸とした加齢性転写後調節異常のメカニズムと意義の解明
- 老化プロテオームの特徴である APOE と抗体の増加を司るメカニズムとその意義の解明
- 大規模オミクスデータ解析に基づく新規老化・寿命制御機構の解明
- 哺乳動物モデルの健康寿命ならびに最大寿命の延長法の評価及び開発

研究キーワード

老化 / オミクスデータ解析 / 寿命制御

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3. Takasugi, M., Yoshida, Y., Nonaka, Y., Ohtani, N. (2023) Gene expressions associated with longer lifespan and aging exhibit similarity in mammals. *Nucleic Acids Res.* 51(14):7205-7219.
4. Takasugi, M., Firsanov, D., Tomblin, G., Ning, H., Abulaeva, J., Seluanov, A., Gorbunova, V. (2020). Naked mole-rat very-high-molecular-mass hyaluronan exhibits superior cytoprotective properties. *Nat Commun* 11(1):2376
5. Takasugi, M., Okada, R., Takahashi, A., Virya, C.D., Watanabe, S., Hara, E. (2017). Small extracellular vesicles secreted from senescent cells promote cancer cell proliferation through EphA2. *Nat Commun* 8:15729



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We aim to advance omics-data-driven research to uncover previously unrecognized fundamental aspects of aging, based on the comprehensive evaluation of age-associated changes in gene expression. As a unique approach, we integrate within-species age-associated gene expression changes with cross-species lifespan-associated expression patterns to infer and experimentally validate the detrimental and beneficial components of age-associated changes. In parallel, as a central focus of our research, we investigate the aging proteome and age-related dysregulation of post-transcriptional control to identify novel mechanisms underlying aging and lifespan regulation. We have identified the accumulation of APOE and immunoglobulins as key features of the aging proteome and are advancing studies to elucidate their underlying mechanisms and to develop strategies for suppressing aging.

Research Topics

- Distinction between detrimental and adaptive aspects of aging-associated changes based on cross-species comparative approaches.
- Mechanistic and functional analysis of age-related dysregulation of post-transcriptional control, with a focus on proteome-level alterations.
- Mechanistic and functional analysis of age-associated accumulation of APOE and immunoglobulins as key features of the aging proteome.
- Identification of novel mechanisms of aging and lifespan regulation through large-scale omics data analyses.
- Evaluation and development of interventions to extend healthspan and maximum lifespan in mammalian models.

Research Interests

Aging / multi-omics analysis /
and lifespan regulation

Selected Publications

1. Takasugi, M., Nonaka, Y., Takemura, K., Yoshida, Y., Stein, F., Schwarz, J.J., Adachi, J., Satoh, J., Ito, S., Tomblin, G., Biashad, S.A., Seluanov, A., Gorbunova, V., Ohtani, N. (2024) An atlas of the aging mouse proteome reveals the features of age-related post-transcriptional dysregulation. *Nat Commun* 15 (1):8520
2. Takasugi, M., Ohtani, N., Takemura, K., Emmrich, S., Zakusilo, F.T., Yoshida, Y., Kutsukake, N., Mariani, J.M., Windrem, M.S., Chandler-Militello, D., Goldman, S.A., Satoh, J., Ito, S., Seluanov, A., Gorbunova, V. (2023) CD44 correlates with longevity and enhances basal ATF6 activity and ER stress resistance. *Cell Rep* 42(9):113130.
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ゲノムの配列情報に代表される「生命情報」を 情報科学的に扱い、生命システムの理解を目指す

The Department of In Silico Analysis focuses on understanding biological systems through the computational analysis of biological information, represented by genomic sequences.



従来の生物学は個別の議論を蓄積し、どんどん教科書を分厚くする枚挙の学問でした。もちろん個別論を見る楽しさは生物学の醍醐味ですが、生命情報ビッグバンとも言える急激な生命情報の増加(図1)により、生物学は大きく様変わりつつあります。例えば、ヒトゲノムに代表される生物の設計図情報や各ゲノム上にある遺伝子がどれぐらい利用されているかという情報(=遺伝子発現量)など、一昔前には考えられなかった程、詳細かつ膨大な生命情報が手に入る時代がこうとしています。その結果、膨大な生命情報を解析する事で、生命システムの一般原理に迫ることが出来る可能性が出てきています。

データが増えることはもちろん良い事です、データが増えれば増えるほどその処理は大変になってきます。特に、せっかく情報が増えてもちゃんと処理しないと貴重な情報を失いかねません。例えば、それぞれの遺伝子の発現量データから計算できる共発現指標という指標があります。従来は単純に遺伝子ペアの発現量の相関係数を用いていましたが、相関係数には、少数のハズレ値の影響が大きいなど様々な問題があります。そこで我々は、最初に主成分解析を行って発現量を主成分空間に射影し、主な主成分を順に抜くことで、少数のデータの影響を除いた多次元共発現度指標を考案し、この指標が従来指標に比べて遺伝子の機能的関連を上手く表現していることを示しました(図2)。

このように、我々の研究室では生命情報ビッグバンの時代における新しい情報生物学を目指して、生命情報を上手に解析するアルゴリズムの研究開発を行っています。

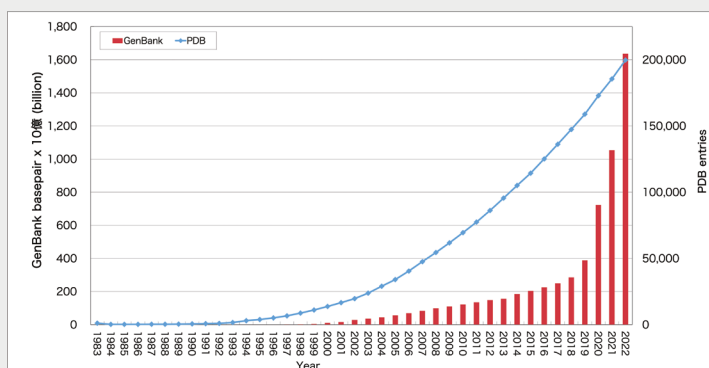


図 1

主な研究テーマ

- 計算生物学
- マルチオミクス解析
- ゲノムコホート研究

研究キーワード

バイオインフォマティクス/ゲノム情報学/
メタボローム解析/タンパク質構造解析

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1. PNPO-PLP axis senses prolonged hypoxia in macrophages by regulating lysosomal activity Nature Metabolism, 2024
Sekine H*, Takeda H*, Takeda N, ... Kinoshita K, Motohashi H
2. Genetic architecture of alcohol consumption identified by a genotype-stratified GWAS and impact on esophageal cancer risk in Japanese people Science Advances, 2024
Koyanagi YN, Nakatochi M, ... Kinoshita K, Matsuo K
3. Simulating metabolic pathways to enhance interpretations of metabolome genome-wide association studies Scientific Reports, 2025 / Kodate S, Sato M, ... Kinoshita K
4. Association of circulating metabolites and polygenic risk score with incident type 2 diabetes: a prospective community-based cohort study Cardiovascular Diabetology, 2025
Takase M, Nakaya N, ... Kinoshita K, Yamamoto M
5. Deep learning-based histopathological assessment of tubulo-interstitial injury in chronic kidney diseases Communications Medicine, 2025
Suzuki N, Kojima K, ... Kinoshita K, Shido K



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木下 賢吾

Concurrent Professor
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It is a well-known fact that traditional biology has tried to enumerate as many biological phenomena as possible, resulting to thicker and thicker textbooks. Although the increase of biological information is quite useful to improve our understanding of biological systems, the increase of information makes it quite difficult to deduce useful information from raw experimental data. In other words, better data analyses are needed to lead us to a better understanding of biological systems. These considerations help us to develop new algorithms and to apply them to analyze large-scale data in this “big bang” era of biological information.

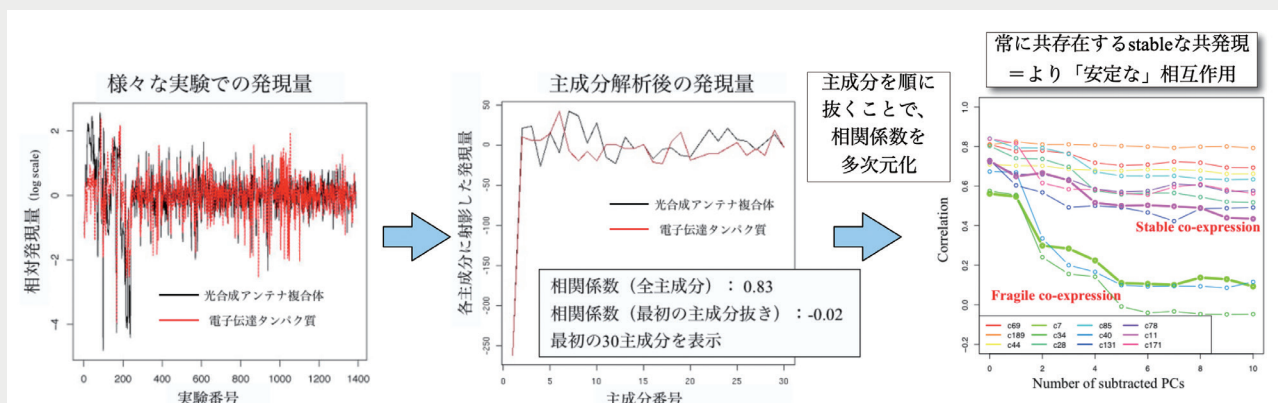


図 2

Research Topics

- computational biology
- multi-omics analyses
- genome cohort study

Research Interests

Inhibitory receptor, immune checkpoint,
cancer immunotherapy, autoimmune disease,
Alzheimer's disease

Selected Publications

1. PNPO-PLP axis senses prolonged hypoxia in macrophages by regulating lysosomal activity *Nature Metabolism*, 2024
Sekine H*, Takeda H*, Takeda N, ... Kinoshita K, Motohashi H
2. Genetic architecture of alcohol consumption identified by a genotype-stratified GWAS and impact on esophageal cancer risk in Japanese people *Science Advances*, 2024
Koyanagi YN, Nakatochi M, ... Kinoshita K, Matsuo K
3. Simulating metabolic pathways to enhance interpretations of metabolome genome-wide association studies *Scientific Reports*, 2025 / Kodate S, Sato M, ... Kinoshita K
4. Association of circulating metabolites and polygenic risk score with incident type 2 diabetes: a prospective community-based cohort study *Cardiovascular Diabetology*, 2025
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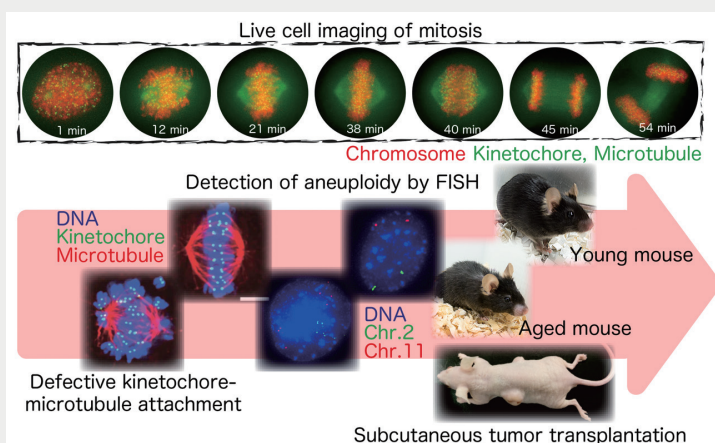
ゲノム不安定性が起こるしくみと、 そのがんや老化との関連を解明する

The Origin of Genomic Instability and its Relevance to Cancer and Aging



私たちの体には約37兆個の細胞がありますが、これは1個の受精卵が分裂を繰り返してできたものです。この1つ1つの細胞には、精巧なしくみによって遺伝情報が正確に伝えられています。一方多くのがんでは染色体の異常や遺伝子の変異が見られ、これは遺伝情報を正確に伝えるしくみの異常(ゲノム不安定性)によって起こります。ゲノム不安定性はアルツハイマー病などの疾患や老化の過程でも見られることが報告されていますが、そのはっきりしたしくみはわかっていません。私たちはこのゲノム不安定性がどのようにして起こるのか、そしてそれがどのようにがんや老化と関連しているのかについて研究しています。そしてゲノムを安定に維持することによる疾患の予防や、ゲノム不安定性をターゲットとしたがん治療の開発につなげようとしています。

私たちは、ヒト細胞における染色体分配の分子機構について明らかにしてきました。またこの機構の異常が、どのようにして多くのがんで見られる染色体不安定性をひき起こすのかについて調べています。さらに、染色体不安定性と老化との関連についても明らかにしようとしています。一方私たちが染色体分配に関連する分子として発見したCHAMP1が、最近知的障害の原因遺伝子であることがわかったため、CHAMP1の神経系の発達への役割についても研究しています。これらの研究は、培養細胞およびマウスを用いて、ライブセルイメージング、生化学的解析、ゲノム・エピゲノム解析などの手法を駆使して行っており、分子レベルから個体レベルまでの一貫した理解を目指しています。



図：分子腫瘍学研究分野の研究テーマ／細胞分裂における染色体分配の過程を観察し、がんや老化で見られるゲノム不安定性がどのようにして生じるのかを研究しています。

主な研究テーマ

- がん細胞の染色体不安定性の原因の解明
- 老化にともなう染色体不安定性の解明
- 知的障害原因分子 CHAMP1 の機能の解析
- 染色体不安定性とがん・老化との関連の解明
- 染色体不安定性を標的としたがん治療の開発

研究キーワード

がん／老化／染色体不安定性／ゲノム不安定性
染色体分配

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助教
國安 絹枝

Assistant Professor
Kinue Kuniyasu

助教
Ren Jiayi

Assistant Professor
Ren Jiayi

Our body comprises around thirty-seven trillion cells, all of which originate from a single fertilized egg. Each cell faithfully inherits genetic information through the cooperation of a number of elaborate mechanisms. By contrast, most cancer cells exhibit chromosomal abnormalities and genetic mutations caused by genomic instability, whereby the mechanisms ensuring the faithful transmission of genetic information are impaired. Genomic instability has also been observed in Alzheimer's disease and aging, although the underlying mechanisms are not fully understood. We are investigating how genomic instability occurs and its relationship with cancer and aging. Our aim is to develop a strategy to prevent disease by increasing genome stability and to develop cancer therapies by targeting genomic instability.

We are investigating the molecular mechanisms of chromosome segregation in human cells. We are also studying how disruption to these mechanisms causes the chromosomal instability observed in many cancers. Additionally, we intend to address the relationship between chromosomal instability and aging. We discovered CHAMP1, a molecule involved in chromosome segregation, and it has recently been found to be related to intellectual disability. Consequently, we are studying the role of CHAMP1 in neuronal development. We address these questions at all levels, from the molecular to the organismal, using cultured cells and mice, as well as a variety of techniques, including live cell imaging, biochemical analysis, and genomic and epigenomic analyses.

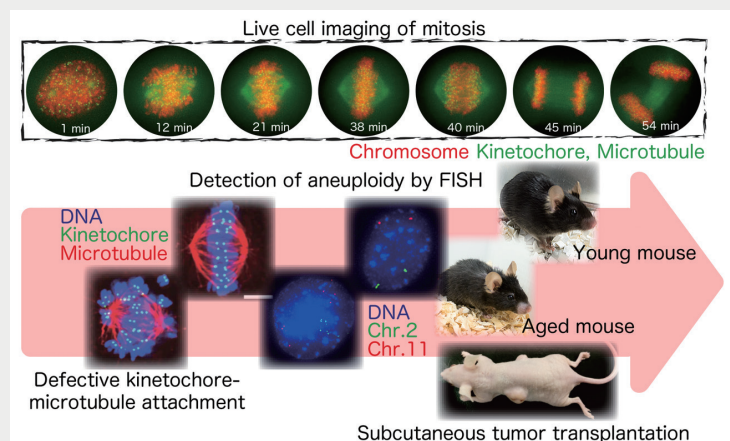


Figure: Research topics in Dept. Molecular Oncology
We study how the genomic instability seen in cancer and aging arises by observing the process of chromosome segregation in cell division.

Research Topics

- Causes of chromosomal instability in cancer cells
- Chromosomal instability associated with aging
- Functional analysis of CHAMP1, a molecule related to intellectual disability
- Relationship of chromosomal instability with cancer and aging
- Development of cancer therapies targeting chromosomal instability

Research Interests

cancer / aging / chromosomal instability /
genomic instability / chromosome segregation

Selected Publications

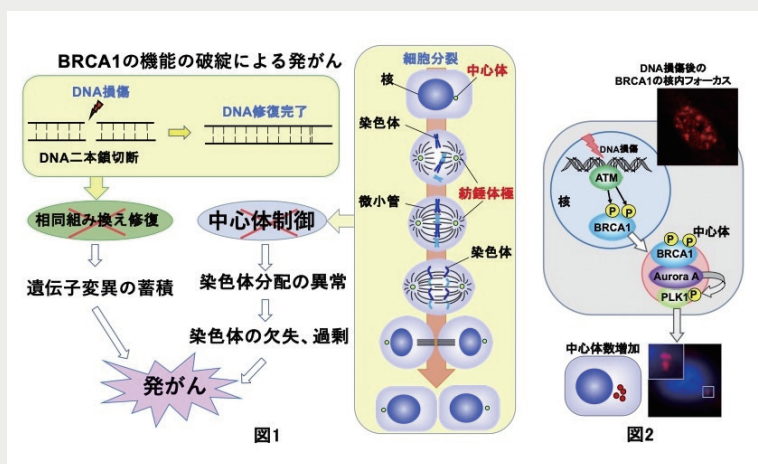
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2. Fujita H et al. CHAMP1-POGZ counteracts the inhibitory effect of 53BP1 on homologous recombination and affects PARP inhibitor resistance. *Oncogene* (2022) 41, 2706-2718.
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遺伝性乳がん卵巣がん症候群(HBOC)の原因遺伝子産物BRCA1の機能解析を中心とした研究

We conduct research primarily focused on the functional analysis of BRCA1, a gene product responsible for hereditary breast and ovarian cancer (HBOC) syndrome.



遺伝性乳がん卵巣がん症候群(HBOC)は、BRCA1やBRCA2の生殖細胞系列変異により乳がん、卵巣がん、膵がん、前立腺がんの発症リスクが上昇する遺伝性腫瘍です。BRCA1は、細胞内の多様な機構に関与しますが、私達は特に中心体制御や相同組換え修復における機能について研究を行っています(図1)。これまでBRCA1結合分子としてOlg-like ATPase 1 (OLA1)と Receptor for activated C kinase 1 (RACK1)を同定し(Mol Cell 2014, Oncogene 2019)、これらのがん由来の変異が中心体制御能に異常をもたらすことを明らかにしました。また、DNA損傷後、BRCA1が核内でのDNA損傷シグナルを中心体に運んで、中心体数を増加させる新たながん抑制能を明らかにし(Cancer Sci 2022)(図2)、さらに分裂期キナーゼとして知られるAurora AがOLA1をユビキチン化して中心体成熟を促進することも明らかにし(Cell Rep 2023)、現在は中心体を標的としたがん治療法の開発を目指しています。また、私達は相同組換え活性の新たな測定法としてAssay for Site-specific HR Activity (ASHRA)を開発し (Sci Rep 2019)、BRCA1の転写制御能を介したPARP阻害剤の耐性機構を明らかにし、さらに今後はHBOCの診断に応用することをめざしています。



主な研究テーマ

- 遺伝性乳がん卵巣がん症候群 (HBOC) の原因遺伝子産物 BRCA1 の機能解明
- Aurora A の E3 ユビキチンリガーゼ活性による中心体制御能の解明
- 中心体の DNA 損傷応答機構の解明
- 相同組換え修復能の測定による HBOC の診断法の開発
- BRCA1 の転写制御能による PARP 阻害剤の耐性機構の解明

研究キーワード

HBOC / BRCA1 / 中心体 / 相同組換え修復
PARP 阻害剤

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3. Yoshino Y, Ogoh H, Iichi Y, Sasaki T, Yoshida T, Ichimura S, Nakayama M, Wu X, Fujita H, Kikuchi M, Fang Z, Li X, Abe T, Futakuchi M, Nakamura Y, Watanabe T, and Chiba N. Knockout of Brca1-interacting factor Ola1 in female mice induces tumors with estrogen suppressible centrosome amplification. BBA - Molecular Basis of Disease, 1870 (5):167138, 2024
4. Fang Z, Li X, Yoshino Y, Suzuki M, Qi H, Murooka H, Katakai R, Shirota M, Pham T A M, Matsuzawa A, Otsuka K, Ishioka C, Mori T, and Chiba N. Aurora A polyubiquitinates the BRCA1-interacting protein OLA1 to promote centrosome maturation. Cell Reports, 42: 112850, 2023
5. Qi H, Kikuchi M, Yoshino Y, Fang Z, Ohashi K, Gotoh T, Ideta R, Ui A, Endo S, Otsuka K, Shindo N, Gonda K, Ishioka C, Miki Y, Iwabuchi T, and Chiba N. BRCA1 transports the DNA damage signal for CDDP-induced centrosome amplification through the centrosomal Aurora A. Cancer Science, 113(12):4230-4243, 2022



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Hereditary breast and ovarian cancer (HBOC) is a hereditary cancer syndrome in which germline mutations in BRCA1 and BRCA2 increase the risk of developing breast, ovarian, pancreatic, and prostate cancers. BRCA1 is involved in a wide range of cellular processes; however, our research has particularly focused on its roles in centrosome regulation and homologous recombination repair (Figure 1).

We have identified Obg-like ATPase 1 (OLA1) and Receptor for Activated C Kinase 1 (RACK1) as BRCA1-interacting proteins (Mol Cell 2014; Oncogene 2019), and demonstrated that cancer-associated mutations in these proteins lead to abnormalities in centrosome regulation. Furthermore, we revealed a novel tumor-suppressive function of BRCA1, whereby, following DNA damage, it transmits nuclear DNA damage signals to the centrosome, resulting in an increase in centrosome number (Cancer Sci 2022). We also showed that Aurora A, a mitotic kinase, ubiquitinates OLA1 to promote centrosome maturation (Cell Rep 2023). Based on these findings, we are currently aiming to develop cancer therapies targeting the centrosome.

In addition, we developed a novel method to assess homologous recombination activity, termed the Assay for Site-specific HR Activity (ASHRA) (Sci Rep 2019) (Figure 2). Using this approach, we have elucidated mechanisms of resistance to PARP inhibitors mediated by the transcriptional regulatory function of BRCA1, and we are now working toward applying this method to the diagnosis of HBOC.

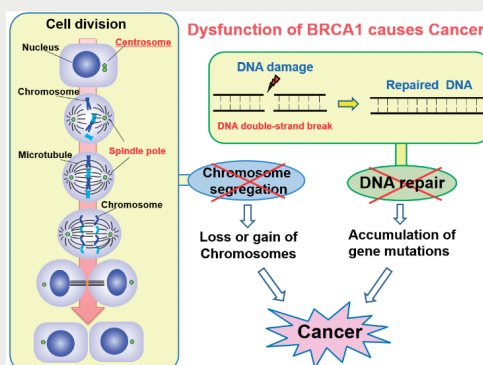


Figure 1 : Dysfunction of BRCA1 causes cancer

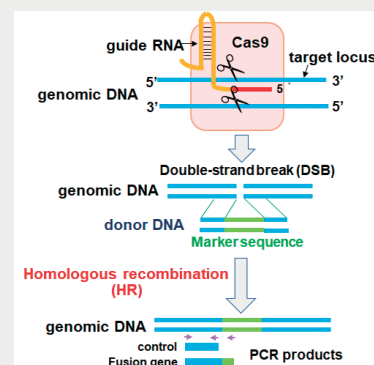


Figure 2 : Schematic of ASHRA

Research Topics

- Elucidation of the functions of BRCA1, a causative gene product of hereditary breast and ovarian cancer (HBOC) syndrome.
- Investigation of centrosome regulation mediated by the E3 ubiquitin ligase activity of Aurora A.
- Elucidation of the DNA damage response mechanisms at the centrosome.
- Development of diagnostic methods for HBOC based on the assessment of homologous recombination repair capacity.
- Elucidation of resistance mechanisms to PARP inhibitors mediated by the transcriptional regulatory functions of BRCA1.

Research Interests

HBOC / BRCA1 / Centrosome /
Homologous recombination, / PARP inhibitor

Selected Publications

1. Yoshino Y, Fang Z, Chiba N. The centrosome: a critical hub for cell cycle regulation. Trends in Cell Biology, 34(6):437-439, 2024
2. Motonari T, Yoshino Y, Haruta M, Endo S, Sasaki S, Miyashita M, Tada H, Watanabe G, Kaneko T, Ishida T, Chiba N. Evaluating homologous recombination activity in tissues to predict the risk of hereditary breast and ovarian cancer and olaparib sensitivity. Scientific Reports, 14: 7519, 2024
3. Yoshino Y, Ogoh H, Iichi Y, Sasaki T, Yoshida T, Ichimura S, Nakayama M, Wu X, Fujita H, Kikuchi M, Fang Z, Li X, Abe T, Futakuchi M, Nakamura Y, Watanabe T, and Chiba N. Knockout of Brca1-interacting factor Ola1 in female mice induces tumors with estrogen suppressible centrosome amplification. BBA - Molecular Basis of Disease, 1870 (5):167138, 2024
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肺癌と肺移植を診療・研究の両輪として 基礎研究にも注力

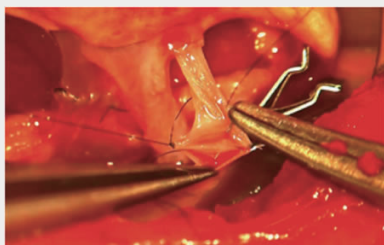
Focusing on basic research on lung cancer and lung transplantation as both medical treatment and research



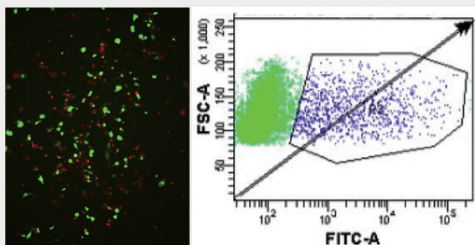
当分野は、加齢医学研究所の分野として、また、東北大学病院の診療科、呼吸器外科として活動しています。

研究の主なテーマは、肺がんと肺移植に関わるものです。手術成績向上を目指した臨床研究はもとより、多くの基礎・臨床研究分野のご協力のもと、肺癌や難治性呼吸器疾患の分子生物学的・病理学的研究、肺保存に関わる研究、虚血・再灌流性肺障害に関わる研究、肺移植後急性・慢性拒絶反応に関わる研究などを展開しています。特に免疫学は、がん研究と臓器移植研究にまたがる分野であり、当教室の研究のメインテーマとなっています。様々な肺疾患に対する細胞治療も早期の臨床応用を念頭に置いて基礎研究を展開しています。さらに、肺組織の再生に関わる研究、幹細胞から肺を作り出す研究、当研究所の使命である加齢医学研究すなわち加齢に伴う肺組織の変化が様々な疾患・病態にどのような影響をもたらすかについての研究にも力を入れています。

診療科としては、肺、縦隔、胸壁などの胸部疾患のうち、外科的治療を要するものを診療の対象としています。主な対象疾患は肺癌、転移性肺腫瘍、縦隔腫瘍、胸壁腫瘍、気胸、胸膜悪性中皮腫、終末期慢性進行性肺疾患（肺移植）などです。東北大学病院は全国に12カ所ある肺移植実施施設の一つに認定されており、東北においては唯一の肺移植施設です。2000年の本邦初となる脳死肺移植以来、2025年12月までに228例の肺移植（脳死肺移植:208例、生体肺移植:20例）を施行いたしました。肺移植後の5年生存率は約75%であり、国際登録の成績（5年生存率約55%）を凌駕する良好な成績が得られています。



ラットの左片肺移植実験。ドナーとレシピエントの肺動脈をmicrosurgical techniqueを用いて吻合している様子。用いている糸は9-0モノフィラメント糸



ヒト肺癌細胞株に対するプラスミドによる遺伝子導入。細胞核（赤）と細胞内に取り込まれたプラスミドベクター（緑）を識別し、FACSによるcell sortingを行い遺伝子導入された細胞を分離した



ブタ摘出肺を用いた体外循環灌流法による長時間肺保存の研究

主な研究テーマ

- 肺癌や難治性呼吸器疾患の分子生物学的・病理学的研究
- 肺保存に関わる研究
- 虚血・再灌流性肺障害に関わる研究
- 肺移植後急性・慢性拒絶反応に関わる研究
- 移植可能なバイオ人工肺の開発

研究キーワード

移植免疫／移植肺の保存／
肺癌のオーダーメイド治療／
バイオ人工肺

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2. Onodera K, Notsuda H, Kumata S, Watanabe T, Watanabe Y, Suzuki T, Hirama T, Oishi H, Okada Y. PD-L1 expression correlates with the oncological severity and prognosis of early-stage lung cancer. *Surg Today*. 2025 Nov;55(11):1635-1643. doi: 10.1007/s00595-025-03070-6. Epub 2025 Jun 4. PMID: 40464925; PMCID: PMC12534232
3. Suzuki T, Watanabe T, Tomiyama F, Ito T, Okada Y. Transplantation of Bioengineered Lung Using Decellularized Mouse Lungs and Primary Human Endothelial Cells. *J Vis Exp*. 2025 Mar 28;(217). doi: 10.3791/67565. PMID: 40228017.
4. Oishi H, Onodera K, Notsuda H, Watanabe T, Watanabe Y, Suzuki T, Niikawa H, Togo T, Kumata S, Okada Y. Postoperative Lung Function after Robotic- versus Video-Assisted Thoracoscopic Lobectomy for Lung Cancer: A Propensity Score-Matched Study. *Ann Thorac Cardiovasc Surg*. 2025;31(1):25-00123. doi: 10.5761/atcs.0a.25-00123. PMID: 41407370; PMCID: PMC12714413.
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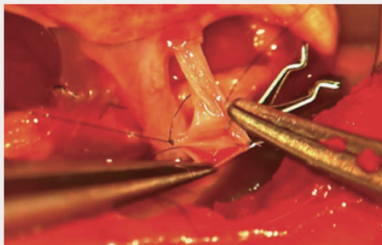
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Assistant Professor
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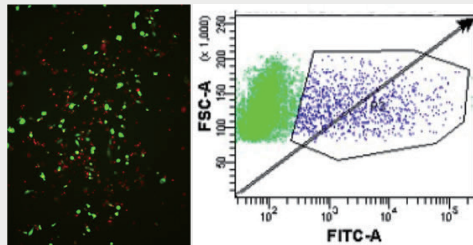
The Department of Thoracic Surgery operates as a field of the IDAC and as a department of Tohoku University Hospital, Department of Pulmonary Surgery.

The main themes of our research are related to lung cancer and lung transplantation. In addition to clinical research aimed at improving surgical outcomes, with the cooperation of many basic and clinical research fields, we are developing molecular biological and pathological research on lung cancer and intractable respiratory diseases, research related to lung preservation, research related to ischemia and reperfusion lung injury, and research related to acute and chronic rejection after lung transplantation. Immunology, in particular, is a field that spans cancer research and organ transplantation research, and is the main research theme of our department. We are also conducting basic research on cell therapy for various lung diseases with a view to early clinical application. In addition, we are also focusing on research related to the regeneration of lung tissue, the creation of lungs from stem cells, and research on aging medicine, which is the mission of our institute, i.e., how changes in lung tissue associated with aging affect various diseases and pathological conditions.

As a department, we focus on chest diseases such as lung, mediastinum, and chest wall that require surgical treatment. Major target diseases include lung cancer, metastatic lung tumors, mediastinal tumors, chest wall tumors, pneumothorax, malignant mesothelioma of the pleura, and end-stage chronic progressive lung diseases (lung transplantation). Tohoku University Hospital is one of 11 lung transplant centers in Japan and the only lung transplant center in Tohoku and Hokkaido. 160 lung transplants (144 brain-dead lung transplants and 16 living lung transplants) have been performed by December 2022 since the first brain-dead lung transplant in Japan in 2000. The 5-year survival rate after lung transplantation is approximately 75%, which is better than the rate in the international registry (5-year survival rate of approximately 55%).



An anastomosis of a rodent's left single lung transplantation. PA anastomosis was performed with a 9-0 monofilament needle and thread.



A gene transfection with plasmid. The red signals are nuclear and the green ones are plasmid vector in cells. After gene transfection, the cells are sorted out using a FACS cell sorter.



An examination of a pig's prolonged lung preservation using an ex-vivo lung perfusion.

Research Topics

- Molecular biological and pathological research on lung cancer and intractable respiratory diseases.
- Research related to lung preservation.
- Research related to ischemia and reperfusion lung injury.
- Research related to acute and chronic rejection after lung transplantation.
- Development of implantable bioartificial lungs.

Research Interests

Transplant immunity / transplant lung preservation
customized treatment of lung cancer /
bioartificial lungs

Selected Publications

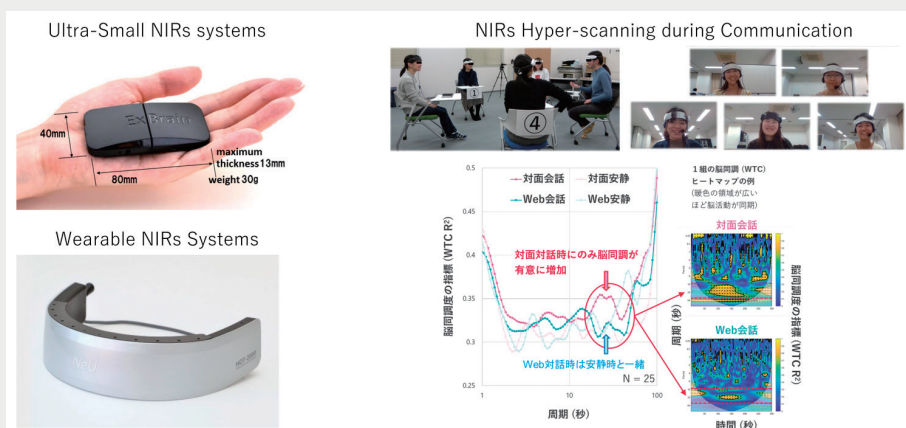
1. Hirama T, Nagao M, Okada Y. Pre-Transplant Infection Screening and Vaccination During Prolonged Waiting Periods for Lung Transplantation in Donor-Limited Asia-Pacific Settings. *Respirology*. 2026 Mar;31(3):232-5. doi:10.1002/resp.70226 PubMed PMID: 41693109; PubMed Central PMCID: PMC12963693.
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脳機能イメージングを核とした 心を豊かに穏やかに加齢するための方法論的研究

We work on a wide range of brain science studies for the purpose of social technology studies, where we can apply these findings to the field of education and welfare.



生活介入研究では、最新の脳機能イメージング技術や、認知神経科学、心理学、疫学などの知識を架橋融合した応用脳科学研究を展開し、健康な小児や成人、健全な社会生活を送っている高齢者の認知機能を維持・向上するための手法を開発します。これまでに、高齢者の運動トレーニングや音読・計算を用いた認知トレーニングやゲームを用いた認知トレーニングの開発と社会実装を行ってきました。認知神経科学の観点から、大脳の前頭前野 (PFC) の作動記憶 (ワーキングメモリ) や実行機能と呼ばれている機能 (将来の計画・企画や意思決定、行動の選択や統制などの基幹となる機能) を向上させる方法を提案します。児童・生徒を対象とした心の発達研究では、学習意欲に影響する要因を特定し、適切な介入プログラムやエビデンスに基づいた教育政策を提案するために、仙台市教育局と連携して心理学・脳科学・社会学・教育学等の手法を活用した多角的な研究を展開しています。心のデコーディング研究では、最新の脳機能イメージング技術を駆使して、個人の注意・気分・意欲・理解などの状態を脳活動から読みとるための基礎研究と、その技術を生活の中で応用する社会技術研究を展開しています。また、集団の脳活動同時計測で社会的インタラクション時の‘複数脳の相互作用・共鳴’を明らかにし、コミュニケーションや共感を促進する技術や環境のデザインに繋げる研究を行っています。



近赤外分光法ハイパースキャンニングによる脳同調定量化

主な研究テーマ

- 高齢者の認知機能維持・向上
- 小児・若年者の認知機能向上
- ICT が認知発達に与える影響
- 円滑なコミュニケーションを支える環境
- ニューロ・マーケティング

研究キーワード

超小型近赤外計測装置／ハイパースキャンニング
認知介入／コホート

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5. Preventive effect of one-session brief focused attention meditation on state fatigue: Resting state functional magnetic resonance imaging study. Yamaya N, Hashimoto T, Ikeda S, Brilliant TD, Tsujimoto M, Nakagawa S, Kawashima R. Neuroimage, 297, 120709, 2024



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川島 隆太

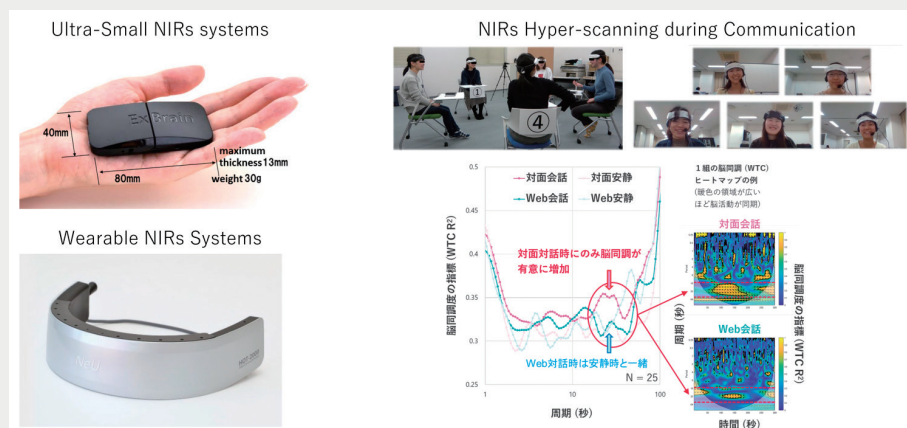
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助教
大方 翔貴

Assistant Professor
Shoki Ogata

The Department of Functional Brain Imaging is made-up of researchers from a variety of backgrounds including medicine, science, life sciences, engineering, pharmacy, linguistics, pedagogy, and the arts. We work on a wide range of brain science studies, from basic brain research for the purpose of social technology studies, where we can apply these findings to the field of education and welfare. Mainly, we focus on functional brain imaging and social technology. In functional brain imaging research, our interest is the “human mind” where we elucidate the neural underpinnings of cognitive functions, such as perception, memory, motor, linguistic and emotional processes, to reveal the mechanism of the mind that survives the complicated.



NIRs Hyper-scanning during Communication

Research Topics

- Functional mapping of higher cognitive functions of humans.
- Neuroimaging study for brain plasticity in performing cognitive intervention in humans.
- Decoding of higher cognitive activities of humans.
- Neuroimaging study for analyzing the correlation between brain and genome in humans.
- Analysis of temporospatial pattern of human brain activity by non-invasive brain measurements.

Research Interests

Cognitive intervention for elders /
Cognitive stimulation for children and adolescents /
Hyper-scanning / Neuro marketing

Selected Publications

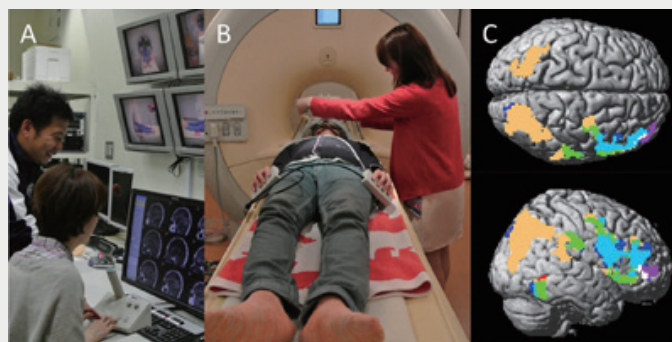
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3. Interventions for expectant and new parents designed to prevent child abuse and neglect in at-risk families: Systematic review and meta-analysis. Tamon H, Sudo M, Yoshitsugu Hm Maruyama N, Ogawa K, Takehara K, Tachibana Y, Kawashima R. *Child Abuse and Neglect*, 160, 107154, 2025
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5. Preventive effect of one-session brief focused attention meditation on state fatigue: Resting state functional magnetic resonance imaging study. Yamaya N, Hashimoto T, Ikeda S, Brilliant TD, Tsujimoto M, Nakagawa S, Kawashima R. *Neuroimage*, 297, 120709, 2024

脳を知れば人間がわかる。 世界はもっとよくなる。

Know your brain. Make the world better.



当分野は人間らしい精神と行動を実現する脳の仕組みを、脳機能計測と生理・行動計測を駆使して明らかにします。基礎から応用まで、人間性に関わるあらゆる学問領域をつなぐ「ハブhub」となる脳科学を目指します。これを通じて、人間らしい生き方、老い方、社会のあり方を脳科学的に提言し、超高齢社会におけるスマート・エイジングの技術開発を目指します。



図：MRI 実験中の操作室 (A) と撮像室 (B) の様子。
研究成果の一例 (C)：鏡の中の自己像を認知する際に働く様々な脳領域 (Sugiura et al., 2014)

主な研究テーマ

- 自己を創る脳
- 価値はどこから生まれるか
- 言葉とコミュニケーション
- 災害と生きる力
- 未来を拓く人間脳科学

研究キーワード

認知神経科学 / fMRI / 自己 / 社会 /
コミュニケーション

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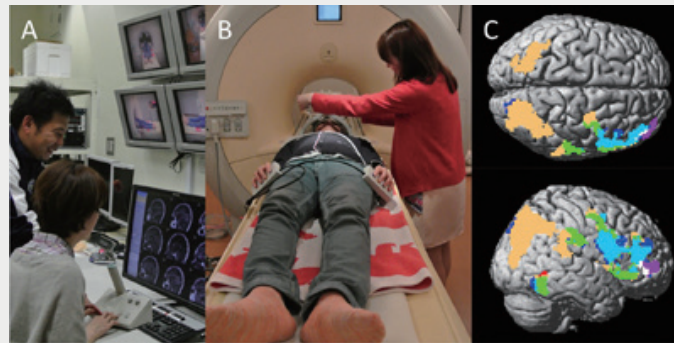
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The Department of Human Brain Science was established to reveal the neural mechanisms underlying the 'human' mind and behavior. To accomplish this, we utilize functional neuroimaging as well as physical and behavioral measurements. We strive for a brain science that is considered a 'hub', connecting different fields of basic and application sciences that address human nature. Ultimately, we want to propose new ways where our lives as an aging society can be more 'human' from a neuroscientific perspective.



A) Students examining images and results from an MRI experiment. B) A volunteer subject is thoroughly guided in the scanner room. C) Regions of the brain that are activated during self-recognition in the mirror.

Research Topics

- The Self in the Brain
- Where value comes from?
- Language and communication
- Disaster and the power to live
- Human brain science for the future

Research Interests

Cognitive Neuroscience / fMRI : fMRI
 (Functional Magnetic Resonance Imaging)
 Self / Society / Communication

Selected Publications

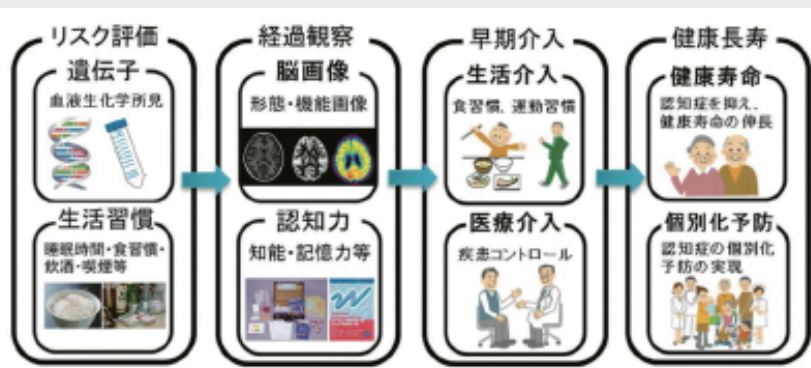
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5. Hamamoto Y, Oba K, Ishibashi R, Ding Y, Nouchi R, Sugiura M. Reduced body-image disturbance by body-image interventions is associated with neural-response changes in visual and social processing regions: A preliminary study. *Front Psychiatry*. 2024;15:1337776. doi:10.3389/fpsy.2024.1337776.

脳科学と高齢者医療の最前線に立ち、 認知症や高齢者特有の疾患に焦点を当てた 包括的な研究と診療を展開

Standing at the forefront of neuroscience and geriatric medicine,
we conduct comprehensive research and clinical practice focused on dementia
and diseases specific to the elderly.



東北大学病院における認知機能外来では認知機能低下疑い症例の診察、検査などを通しての精密な診断や治療の提供、周辺症状を呈する高度認知症の治療方針決定や対処法の相談を行なっています。また、昨今、アルツハイマー病に対する抗アミロイドβ抗体治療も積極的に導入する他、多くの認知症の治験・新薬開発に参画しています。臨床研究では、認知症の病態解明、全年代を通じた認知機能障害症例の病態解明などを最先端の画像検査や生理学的検査などを駆使して展開し、その成果を世界に向けて発信しています。さらに、高齢者医療におけるポリファーマシー問題やせん妄対策などの問題解決に向けて、臨床研究を通じてより良い医療の提供を目指しています。また、超高齢者社会の抱える課題解決に向けて、基礎的研究にも精力的に取り組んでいます。そのために、多くの企業と協力しながら、大学の多様な研究シーズを活用した産学共同研究を推進しています。当分野では脳の発達や加齢、生活習慣、遺伝要因など、高齢者の健康に関わる幅広い領域をカバーしています。具体的には、食生活や運動習慣、芸術活動など、日常生活に関わる様々な要因が高齢者の身体機能や認知機能、心理状態にどのような影響を与えるのかを明らかにするため、介入研究を実施しています。最終的には産学共同研究を通じて得られた結果を社会実装に繋げていくことを目指しており、超高齢社会への対応は大学と産業界が協力して取り組むべき重要な課題であると認識しています。結果として既に複数の大学発スタートアップ企業も創出しています。



主な研究テーマ

- 軽度認知障害患者における臨床的特徴の国際比較
- 血液バイオマーカーによる認知機能障害早期発見の試み
- MRIによるアミロイドβ可視化技術の開発
- 認知機能障害に関わる脳内神経ネットワーク異常の解明
- 健常高齢者における食生活や運動習慣と認知機能との関連の解明

研究キーワード

Alzheimer' s disease / Mild cognitive impairment
Frailty / Polypharmacy / Brain sciences.

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3. Soga K, Takahashi M, Uno A, Sinada T, Oba K, Kawashima K, Tatewaki Y, Nakase T, Taki Y. Circuit training intervention for cognitive function, gut microbiota, and aging control: study protocol for a longitudinal, open-label randomized controlled trial. Trials. 2025 Mar 18;26(1):94. doi: 10.1186/s13063-025-08807-9.
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Assistant Professor
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At the Memory Clinic of Tohoku University Hospital, we provide comprehensive care for patients with cognitive decline, including detailed diagnostic evaluations and treatment through clinical assessment and advanced testing. We also offer consultation on treatment strategies and management of behavioral and psychological symptoms in patients with advanced dementia.

In recent years, we have actively introduced anti-amyloid beta antibody therapies for Alzheimer's disease, also participated in numerous clinical trials and new drug development programs for dementia. Our clinical research focuses on elucidating the pathophysiology of dementia, as well as cognitive impairment across all age groups, utilizing cutting-edge neuroimaging and physiological assessment techniques. The outcomes of these efforts are disseminated globally.

Furthermore, we aim to improve geriatric care by addressing critical issues such as polypharmacy and delirium through clinical research. In parallel, we are actively engaged in basic research to tackle the broader challenges of a super-aging society. To this end, we promote industry-academia collaborative research by leveraging diverse research seeds within the university in partnership with various companies.

Our field encompasses a wide range of factors influencing elderly health, including brain development and aging, lifestyle habits, and genetic factors. Specifically, we conduct interventional studies to clarify how daily life factors—such as diet, exercise, and artistic activities—affect physical function, cognitive function, and psychological well-being in older adults.

Ultimately, we aim to translate findings from industry-academia collaborations into real-world applications. We recognize that addressing the challenges of a super-aging society is a critical issue that requires close cooperation between academia and industry. As a result, multiple university-originated startup companies have already been established.

Research Topics

- International comparison of clinical characteristics in patients with mild cognitive impairment
- Attempts at early detection of cognitive impairment using blood biomarkers
- Impact of diabetes on Alzheimer's disease pathology
- Elucidation of abnormal brain neural networks associated with cognitive impairment
- Investigation of the relationship between diet, exercise habits, and cognitive function in healthy older adults

Research Interests

Alzheimer's disease / Mild cognitive impairment
Frailty / Polypharmacy / Brain sciences.

Selected Publications

1. Nakase T, Takano Y, Nomura S, Baek HW, Takayama S, Ono R, Abe M, Ishii T, Tatewaki Y, Taki Y. Association of symptoms of neuropsychological long COVID with imaging and plasma biomarkers. *J Neurol Sci.* 2026 Apr 15;483:125808. doi: 10.1016/j.jns.2026.125808.
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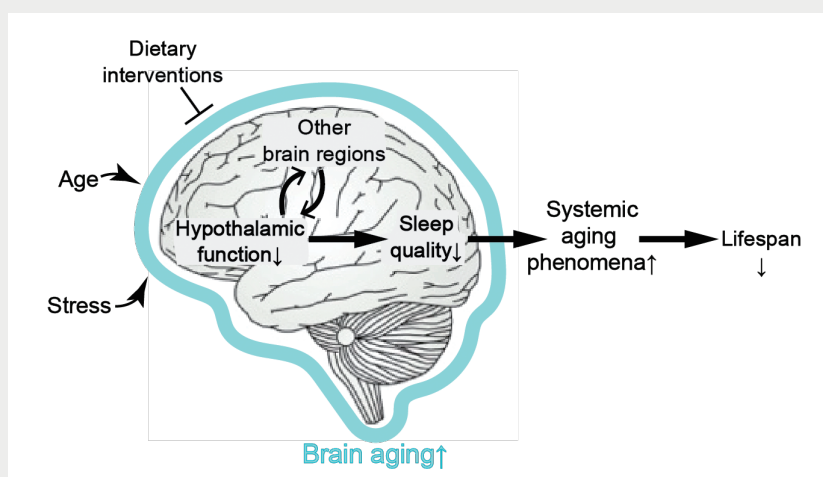
視床下部の加齢変化が駆動する 老化寿命制御機構の統合的理解

An Integrative Understanding of Mechanisms of Aging/ lifespan Control Driven by Hypothalamic Aging



歳をとると脳の働きが低下する「脳老化」が起こります。近年の研究から、脳老化は単なる加齢現象にとどまらず、寿命そのものを規定する可能性があることが示唆されています。そこで本研究分野では、脳老化を分子レベルから組織レベルで理解し、哺乳類の老化や寿命の制御における脳、特に視床下部の役割を明らかにすることを目指しています。さらに、疫学、臨床、数理モデルなど多様な分野の国内外の専門家と連携し、ヒトの健康寿命を延ばす新たな方法の開発にも積極的に取り組んでいます。これらの研究を通じて、高齢化が急速に進む社会において、人々がより長く健やかな生活を送れるようになることを目指しています。

加齢に伴い、脳、特に視床下部機能が低下すると睡眠の質が低下する。また、ストレス負荷も同様に睡眠の質の低下を誘導する。また、視床下部の機能低下は他の脳領域を含む脳の機能的ネットワークの異常を引き起こす。その結果、全身の老化現象が誘導され、最終的に哺乳類の寿命に影響が及ぼされる。食餌制限などの栄養学的介入は老化に伴う睡眠の質低下を抑制することが老化現象の遅延や寿命延伸作用につながるのかもしれない。つまり加齢に伴う睡眠変化は個体寿命を決定する脳老化の現象の一つであることが考えられる。



図：脳が全身の老化現象を調節する機構

Figure. Mechanisms by which the brain controls systemic aging phenomena and longevity.

The function of the brain, particularly the hypothalamus, declines with age, leading to poor quality of sleep. Certain stress also causes a reduced quality of sleep. Additionally, hypothalamic dysfunction leads to abnormalities in functional brain networks that involve other brain regions. Such poor sleep quality leads to several systemic aging phenomena, and ultimately affects mammalian longevity. A nutritional intervention, such as dietary restriction, delays the aging process and extends lifespan presumably through the suppression of age-associated sleep alterations. Such age-associated sleep alteration may cause aging and affect mammalian longevity. Thus, age-associated sleep alterations can be one of determinant of mammalian longevity.

主な研究テーマ

- 睡眠調節能を介した哺乳類の寿命を制御する新規視床下部神経の同定とそのメカニズムの解明
- 脳老化の可逆性及び非可逆性分子機構の解明
- 栄養学的介入による寿命延伸作用の責任神経と回路の解明
- 加齢に伴う脳機能ネットワーク変化とその生理学的機能の解明

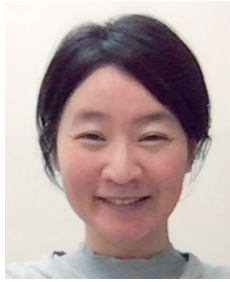
研究キーワード

視床下部／老化・寿命／睡眠／脳機能ネットワーク



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辻 将吾

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Shogo Tsuji

As we age, the brain undergoes functional decline, a process known as “brain aging.” Recent studies suggest that brain aging is not only a consequence of aging itself, but may also play a determining role in lifespan. We aim to deepen our understanding of brain aging at the molecular to tissue levels and to elucidate the role of the brain, particularly the hypothalamus, in regulating aging and lifespan in mammals. In addition, we actively collaborate with experts from diverse fields—such as epidemiology, clinical research, and mathematical modeling—both in Japan and internationally, to develop new approaches for extending healthy human lifespan. Through these efforts, we hope to contribute to a rapidly aging society by enabling people to live longer, healthier lives.

The function of the brain, particularly the hypothalamus, declines with age, leading to poor quality of sleep. Certain stress also causes a reduced quality of sleep. Additionally, hypothalamic dysfunction leads to abnormalities in functional brain networks that involve other brain regions. Such poor sleep quality leads to several systemic aging phenomena, and ultimately affects mammalian longevity. A nutritional intervention, such as dietary restriction, delays the aging process and extends lifespan presumably through the suppression of age-associated sleep alterations. Such age-associated sleep alteration may cause aging and affect mammalian longevity. Thus, age-associated sleep alterations can be one of determinant of mammalian longevity.

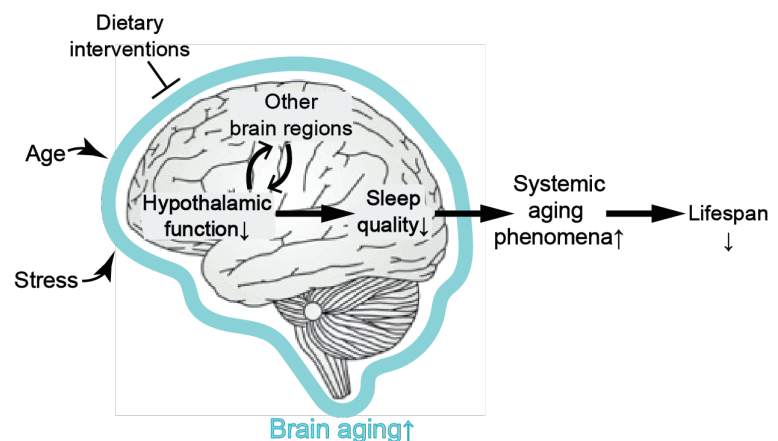


Figure. Mechanisms by which the brain controls systemic aging phenomena and longevity.

Research Topics

- Identification of a novel neuronal population in the hypothalamus that may control mammalian longevity through sleep regulation and elucidation of its mechanisms.
- Elucidation of reversible and irreversible molecular mechanisms of brain aging.
- Exploration of neurons and neuronal circuits, which are responsible for the effects of dietary/nutritional interventions in mammalian lifespan extension.
- Investigation of age-associated changes in functional brain networks and their physiological significance.

Research Interests

Hypothalamus / aging/longevity / sleep / functional brain network

Figure. Mechanisms by which the brain controls systemic aging phenomena and longevity.

The function of the brain, particularly the hypothalamus, declines with age, leading to poor quality of sleep. Certain stress also causes a reduced quality of sleep. Additionally, hypothalamic dysfunction leads to abnormalities in functional brain networks that involve other brain regions. Such poor sleep quality leads to several systemic aging phenomena, and ultimately affects mammalian longevity. A nutritional intervention, such as dietary restriction, delays the aging process and extends lifespan presumably through the suppression of age-associated sleep alterations. Such age-associated sleep alteration may cause aging and affect mammalian longevity. Thus, age-associated sleep alterations can be one of determinant of mammalian longevity.

脳の健康を科学し、社会へ届ける — 小児から高齢期まで、生涯を通じた「健康脳」の実現

"From Childhood Brain Development to Healthy Aging: Science-to-Society Translation of the 'Lifelong Healthy Brain' Through Global Collaboration"



当センターは、数万件規模の脳MRI画像データベースを基盤に、小児の健やかな脳発達から高齢期の認知症予防まで、一生涯の脳の健康を定量的に解明する包括的な研究拠点です(図1)。

研究の最大の特徴は、画像解析による基礎医学にとどまらず、「産官学民連携による社会実装」を活動の中心に据えている点です。超高齢社会の課題解決に向け、人々の「日常」に根ざした多角的な生活介入研究を、食品・モビリティ・ヘルスケア分野など異業種企業との強固な共同研究体制で推進しています。運動習慣・安全運転・食品の脳への効果実証に加え、IoTデバイスを活用した長期介入研究や、グループ音楽セッション・デジタルアート学習・回想が高齢者の認知・心理機能に与える効果の実証など、エビデンスに基づく研究成果を積み上げています。特に、ノスタルジー×生成AIによる認知症リスク低減プロジェクトでは、AIによる画像処理を駆使して、過去の航空写真や報道画像等から過去三次元空間の再現を行い、全国の過去三次元空間デジタルプラットフォームを開発しています(図2)。また、生まれてからの年数である暦年齢よりも、加齢性疾患や死亡を予測することに秀でた生物学的年齢を標的として、その進行を遅らせるための生活習慣介入の有効性評価も進めています。我々は、企業や自治体との産学官連携に加え、多くの大学・研究機関との国際共同研究(図3)など、多くの連携のもとで研究を展開しています。

加齢をネガティブに捉えず、誰もが心豊かに年齢を重ねる「スマート・エイジング」を究極の理念とし、個別最適化に基づく「生涯健康脳」を社会基盤へ還元することで、誰もが幸福を実感できる未来に貢献します。

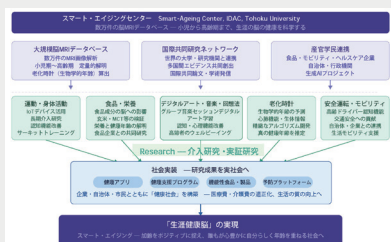


図1 スマート・エイジングセンターの活動の概要

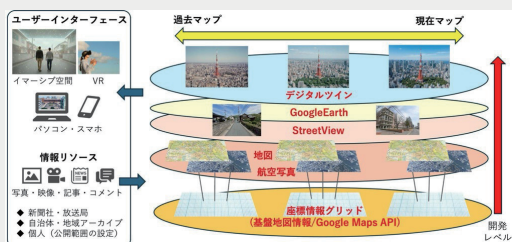


図2 ノスタルジー×生成AIによる認知症リスク低減プロジェクト



図3 国際連携部門の活動概要

誰もが気軽に楽しく脳の健康維持・認知症リスク低下を享受できる、思い出の場所の三次元空間の再現とデジタルプラットホームの開発。AIと人的支援を融合したハイブリッドアプローチのもと、医学・工学・人文学にわたるHCI研究を推進し、国際プロジェクト・共同研究を通じて高齢者が社会的価値を生み出す担い手となることを目指す。

主な研究テーマ

- 大規模脳画像解析を用いた基礎・応用研究 — 数万件のMRIデータベースから小児期の脳発達～認知症予防まで定量的に解明
- 日常をベースとした多角的な生活介入研究 — 運動・食品・デジタルアート・音楽・ノスタルジー体験の効果を実証
- 精緻な「老化時計(生物学的年齢)」の構築 — 脳画像・生体情報から真の健康年齢を予測するアルゴリズム開発
- 産官学民連携による社会実装 — 食品・モビリティ・ヘルスケア企業、自治体との共同研究を通じた実社会への還元
- 国際共同研究ネットワークでグローバルにエビデンスを創出 — 世界の研究機関と連携し、誰もが心豊かに年齢を重ねていく「スマート・エイジング」を究極の目標(理念)に、個別最適化された方法で「生涯健康脳」を実現

研究キーワード

生涯健康脳／生活介入研究／産官学民連携・社会実装
スマート・エイジング

参考文献

1. Shinada, T., Takahashi, M., Uno, A., Soga, K., & Taki, Y. (2025). Effects of group music sessions on cognitive and psychological functions in healthy older adults. *Frontiers in Aging*, 6, 1513359. <https://doi.org/10.3389/fragi.2025.1513359>
2. Uno, A., Browne, R., Shinada, T., Takahashi, M., Soga, K., Du, Y., Abiko, F. & Taki, Y. (2025). Effects of a digital visual art learning intervention in healthy older adults: a pilot randomized controlled trial. *Frontiers in Aging*, 6, 1635789. <https://doi.org/10.3389/fragi.2025.1635789>
3. Soga, K., Takahashi, M., Uno, A., Shinada, T., Oba, K., Kawashima, K., Tatewaki, Y., Nakase, T., Taki, Y. (2025). Circuit training intervention for cognitive function, gut microbiota, and aging control: study protocol for a longitudinal, open-label randomized controlled trial. *Trials* 26, 94. <https://doi.org/10.1186/s13063-025-08807-9>
4. Soga, K., Higuchi, A., Tomita, N., Kobayashi, K., Kataoka, H., Imankulova A., Salazar, C., Thyreau, B., Nakamura, S., Tsushita, Y., Uchiyama, A. & Taki, Y. (2025). Beneficial Effects of a 26-Week Exercise Intervention Using IoT Devices on Cognitive Function and Health Indicators. *Journal of Science in Sport and Exercise*, 7, 238-249. <https://doi.org/10.1007/s42978-024-00321-9>



センター長・教授
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助教 中島 淑恵

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Assistant Professor
Shingo Hayashi

助教 宮坂 真紀子

Assistant Professor
Makiko Miyasaka

助教 山谷 礼輝

Assistant Professor
Noriki Yamaya

The Smart-Aging Center is a comprehensive research hub dedicated to quantitatively elucidating lifelong brain health — from healthy pediatric brain development to dementia prevention in later life — built upon a database of tens of thousands of brain MRI scans (Figure 1).

What sets our center apart is a commitment that goes far beyond image-based basic medical research: we place "social implementation through industry-government-academia-civil collaboration" at the very core of our activities. To address the challenges of Japan's super-aging society, we advance multifaceted lifestyle intervention studies rooted in people's everyday lives, supported by strong joint research frameworks with cross-sector industries spanning food, mobility, and healthcare. Our evidence base continues to grow, encompassing studies on the effects of exercise habits, safe driving practices, and dietary factors on the brain, as well as long-term intervention research utilizing IoT devices, and randomized trials demonstrating the cognitive and psychological benefits of group music sessions and digital art learning in older adults. In particular, the Nostalgia x Generative AI Dementia Risk Reduction Project utilizes AI-powered image processing to recreate past three-dimensional spaces from historical aerial photographs and news images, developing a nationwide digital platform for past three-dimensional spaces (Figure 2).

We are also advancing the development of highly precise "Aging Clock" algorithms — using cutting-edge analysis of biometric data to elucidate the relationship between cardiorespiratory fitness and biological aging, and to predict each individual's true biological age. Our research is conducted through broad and diverse partnerships, including industry-academia-government collaboration with companies and municipalities, as well as international joint research with universities and institutions worldwide (Figure 3).

Furthermore, backed by funding from Google.org, we have launched an innovative project that uses generative AI to reconstruct historical three-dimensional environments, with the aim of scientifically validating the cognitive and psychological effects of "nostalgia experiences." Guided by our ultimate philosophy of "Smart Aging" — a vision in which aging is embraced not as decline, but as a time of rich and meaningful growth — we are committed to returning personalized "Lifelong Healthy Brain" strategies to the foundations of society, and to contributing to a future in which everyone can experience genuine well-being.

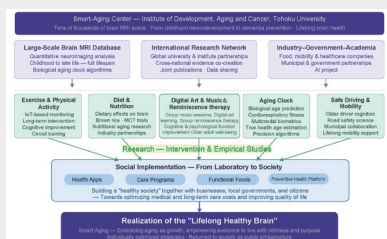


Figure 1 Caption: Overview of Research Activities at the Smart-Aging Center

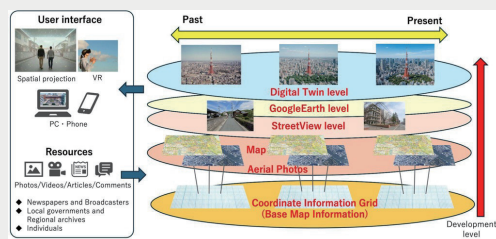


Figure 2 Caption: Nostalgia-Driven Dementia Care with Generative AI



Figure 3 Caption: International Collaboration Division

Built on a large-scale brain MRI database and broad interdisciplinary collaboration, the center advances lifestyle intervention research across five domains and translates findings into society, with the ultimate goal of realizing the "Lifelong Healthy Brain."

Development of a digital platform and three-dimensional spatial recreation of memorable places, allowing everyone to easily and enjoyably maintain brain health and reduce the risk of dementia.

Research Topics

- Foundational and Applied Research Using Large-Scale Neuroimaging — Leveraging a database of tens of thousands of MRI scans to quantitatively elucidate brain health across the full lifespan, from pediatric neurodevelopment to dementia prevention in later life.
- Multifaceted Lifestyle Intervention Research Grounded in Daily Life — Conducting randomized controlled trials to examine the effects of exercise, dietary factors, digital art learning, music sessions, and nostalgia experiences on brain health and cognitive function.
- Development of Highly Precise "Aging Clocks" (Biological Age Metrics) — Building advanced algorithms that leverage neuroimaging and multimodal biometric data to accurately predict each individual's true biological age, independent of chronological age.
- Social Implementation Through Industry-Government-Academia-Civil Collaboration — Translating research findings into real-world services and products through joint research partnerships with companies in the food, mobility, and healthcare sectors, as well as with local governments and municipalities.
- Global Evidence Generation Through an International Research Network — Collaborating with universities and research institutions worldwide, with the ultimate goal of realizing "Smart Aging" — a society in which everyone can age with richness and fulfillment — and achieving the "Lifelong Healthy Brain" through individually optimized strategies.

Research Interests

Lifelong Healthy Brain/Lifestyle Intervention Research

Industry-Academia-Government Collaboration and Social Implementation/Smart Aging

Selected Publications

1. Shinada, T., Takahashi, M., Uno, A., Soga, K., & Taki, Y. (2025). Effects of group music sessions on cognitive and psychological functions in healthy older adults. *Frontiers in Aging*, 6, 1513359. <https://doi.org/10.3389/fragi.2025.1513359>
2. Uno, A., Browne, R., Shinada, T., Takahashi, M., Soga, K., Du, Y., Abiko, F. & Taki, Y. (2025). Effects of a digital visual art learning intervention in healthy older adults: a pilot randomized controlled trial. *Frontiers in Aging*, 6, 1635789. <https://doi.org/10.3389/fragi.2025.1635789>
3. Soga, K., Takahashi, M., Uno, A., Shinada, T., Oba, K., Kawashima, K., Tatewaki, Y., Nakase, T., Taki, Y. (2025). Circuit training intervention for cognitive function, gut microbiota, and aging control: study protocol for a longitudinal, open-label randomized controlled trial. *Trials* 26, 94. <https://doi.org/10.1186/s13063-025-08807-9>
4. Soga, K., Higuchi, A., Tomita, N., Kobayashi, K., Kataoka, H., Imankulova, A., Salazar, C., Thyreau, B., Nakamura, S., Tsushita, Y., Uchiyama, A. & Taki, Y. (2025). Beneficial Effects of a 26-Week Exercise Intervention Using IoT Devices on Cognitive Function and Health Indicators. *Journal of Science in Sport and Exercise*, 7, 238–249. <https://doi.org/10.1007/s42978-024-00321-9>

附属医用細胞資源センター

Cell Resource Center for Biomedical Research

センター長
小笠原 康悦

Head of the Center
Koetsu Ogasawara

医用細胞資源センターは、癌細胞、培養細胞等の医用細胞に関する研究並びにこれらの細胞の維持保存及び各研究機関への供給を行うことを目的としています。

腹水癌細胞株及びヒト癌細胞、白血病細胞を含む培養細胞株、ハイブリドーマ等を対象とした細胞バンク事業を行っており、本ホームページ上に細胞株のカタログを公開し、希望者へ有償で供給しています。細胞株の品質管理として、マイコプラズマ検査、DNAフィンガープリンティング、動物種同定検査等を行っています。

The Cell Resource Center for Biomedical Research is dedicated to conducting research on medical cells, including cancer cells and cultured cells, as well as maintaining, preserving, and supplying these cells to various research institutions.

The cell lines include transplantable animal cell lines, such as Yoshida sarcoma and rat ascites hepatoma (AH series) cell lines as well as human, murine cell lines and hybridoma cells. The Cell Line Catalog is available on the web site.

非臨床試験推進センター

Pre-Clinical Research Center

センター長
岡田 克典

Head of the Center
Yoshinori Okada

非臨床試験推進センターは、国際標準の非臨床試験環境を整備し、基礎研究から治験その他の臨床研究まで切れ目なく研究開発支援を行うことにより、世界を先導する革新的な医療機器やヘルスケアシステム等の開発及び実用化の迅速化に貢献することを目的としています。

The Pre-Clinical Research Center aims to accelerate the development and practical application of world-leading innovative medical devices and healthcare systems. To achieve this, the Center establishes international-standard environments for pre-clinical research and provides seamless research and development support from basic research through to clinical trials and other clinical studies.

非臨床試験推進センター Pre-Clinical Research Center

心臓病電子医学分野 Department of Medical Engineering and Cardiology

分野長
准教授
白石 泰之

Head of Department
Associate Professor
Yasuyuki Shiraishi

准教授
深谷 碧

Associate Professor
Fukaya Aoi

助教
フランシス・C・チクウェート

Assistant Professor
Chikweto Francis

当分野は、本格的な臨床試験への橋渡し役を担う非臨床試験研究の環境を整え、革新的な医療機器やヘルスケアシステムの研究開発に貢献します。国際的な安全と福祉の品質基準を満たす非臨床試験推進センターで、国内外の大学や企業とのネットワークを構築し、非臨床試験研究の場をもつことで医工連携のネットワークの充実につなげます。

This Department aims to establish a robust environment for pre-clinical research that serves as a bridge to full-scale clinical trials, thereby contributing to the research and development of innovative medical devices and healthcare systems.

Operating within the Pre-Clinical Research



直接穿刺型血液ポンプ
Direct puncture blood pump

Center, which meets international quality standards for safety and welfare, the Department builds collaborative networks with universities and companies in Japan and abroad. By providing dedicated spaces for pre-clinical studies, the Department contributes to strengthening networks for biomedical engineering collaboration.

非臨床試験推進センター Pre-Clinical Research Center

呼吸不全電子医学分野 Department of Respiratory Engineering

分野長

岡田 克典

Head of Department
Yoshinori Okada

当分野は、呼吸不全患者の予後改善、生活の質の向上を目的として、肺移植医療の推進と肺移植関連の医療機器の開発、在宅酸素療法に留まらない呼吸不全に対する新しい治療法・新しい医療機器の開発に取り込んでいます。

- ・長時間肺保存を目的としたdeviceの開発
- ・抗凝固薬が不要な心肺補助装置の開発
- ・呼吸困難の生理学的機序の解明

With the goal of improving patient prognosis and quality of life (QOL) for individuals with respiratory failure, the Department promotes lung transplantation medicine, develops transplant-related medical devices, and advances new treatment approaches and medical technologies that extend beyond conventional home oxygen therapy.

- ・Development of devices to promote long term lung preservation.
- ・Development of anti-clotting drugs and heart-lung assist devices.
- ・The use of physiological mechanisms to elucidate breathing difficulties.

非臨床試験推進センター Pre-Clinical Research Center

生体計測研究分野 Department of Biomedical Measurements

分野長

西條 芳文（兼務教授）

Head of Department
Professor(Concurrent)
Yoshifumi Saijo

当分野は、加齢に関係した生理学的・形態学的・生化学的変化の計測方法や機器の開発・評価により、人の加齢の課程やメカニズムを明らかにすることを目的としています。

The Department aims to elucidate the processes and mechanisms of human aging by developing and evaluating imaging and sensor technologies designed to measure aging-related physiological, morphological, and biochemical changes.

環境ストレス老化研究センター Center for Environmental Response and Aging

センター長

魏 范研

Head of the Center
Fan-Yan Wei

環境ストレス老化研究センターは、老化プロセスと、環境化学物質、病原体、機械的刺激などの環境因子に対するストレス応答との機能的相互作用を明らかにすることを目的としています。最終目標は、防御機構を制御し、細胞保護能力を高めることによって、老化プロセスを遅らせ、老化関連疾患を緩和することです。

The Center for Environmental Response and Aging aims to clarify functional interactions between the aging process and stress response to various environmental factors, such as chemical substances, pathogens, and mechanical stimuli.

The Center's ultimate goal is to delay the aging process and alleviate age-related diseases by modulating defense mechanisms and enhancing cytoprotective capacities.

脳 MRI センター

Brain MRI Center

センター長
杉浦 元亮

Head of the Center
Motoaki Sugiura

脳MRIセンターは、高磁場磁気共鳴画像撮像装置及びこれに付帯する実験設備を人間の脳機能・形態に関する研究に供し、その環境を維持・管理することを目的としています。MRI装置の利用を希望する大学、研究機関、企業の研究者が脳計測実験に参加できます。

The Brain MRI Center was established for the joint use of high-field magnetic resonance imaging (MRI) equipment and its associated experimental facilities. These resources are made available for research on human brain function and structure. Researchers from universities, research institutions, and companies who wish to use MRI equipment may participate in brain measurement experiments.

先端研究小動物 MRI センター

Small Animal MRI Center for Research Use with Technical Advantage

センター長
佐藤 亜希子（兼務教授）

Head of the Center
Akiko Satoh

先端研究小動物MRIセンターは、センターは、小動物用デジタルMRI装置及びこれに付帯する実験設備を動物の生体機能および形態に関する研究に供し、その環境を維持・管理することを目的としています。

The Small Animal MRI Center for Research Use with Technical Advantage (RUTA) provides digital MRI systems for small animals and associated experimental equipment for research on physiological functions and morphology in vivo, while maintaining and managing an optimal research environment.

マルチオミクスコアファシリティセンター

Multi-Omics Core Facility Center

センター長
魏 范研

Head of the Center
Fan-Yan Wei

マルチオミクスコアファシリティセンターは、遺伝子、転写産物、タンパク質、代謝物などの生体分子、ならびに生体機能に影響を及ぼしうる環境因子や外因性分子を対象とする多階層オミックス解析を実施するための実験設備を整備し、これを研究に供するとともに、その利用環境を維持・管理しています。

The Multi-Omics Core Facility Center establishes and maintains experimental facilities for conducting multi-tiered omics analyses. These analyses target biomolecules such as genes, transcripts, proteins, and metabolites, as well as environmental factors and exogenous molecules that may influence biological functions. The Center makes these facilities available for research while managing the user environment.

共通施設 Common Facilities

実験動物管理室 Laboratory Animal Resources

室長
河本 新平（兼務教授）

Head of the Laboratory
Shimpei Kawamoto

実験動物管理室では、日常的な実験動物の飼育管理・品質管理に加えて、マウス生殖工学技術を用いた研究支援業務にも取り組むことで、実験動物福祉の理念である3R (Replacement, Reduction, Refinement) を推進し、適正な動物実験が実施されるようにするための環境整備に取り組んでいます。

In addition to the daily care and quality control of laboratory animals, the Laboratory Animal Resources supports research activities utilizing mouse reproductive techniques. Through these efforts, the Laboratory promotes the 3Rs (Replacement, Reduction, Refinement)—the guiding principles of laboratory animal welfare—and work to maintain an appropriate environment for animal experimentation.

共通機器管理室 Center of Research Instruments

室長
魏 范研

Head of the Center
Fan-Yan Wei

共通機器管理室は、加齢医学の基礎研究を円滑化するため、遺伝子・細胞・画像解析を中心とした約50台の機器を集中管理しています。共焦点顕微鏡やセルソーター、質量分析装置等の高度な特殊装置を揃えるだけでなく、専任スタッフによる技術サポート体制を完備し、初心者から熟練者まで誰もが精度高いデータを得られる環境を提供します。

The Center centrally manages approximately 50 instruments, primarily for genetic, cellular, and image analysis, to support basic research in aging research. In addition to providing access to advanced specialized equipment, such as confocal microscopes, cell sorters, and mass spectrometers, the Center offers comprehensive technical support by dedicated staff. This ensures a reliable environment for obtaining high-quality data, from beginners to experienced researchers.

広報情報室 Public Relations and Information Office

室長
田中 耕三

Head of the Office
Kozo Tanaka

広報情報室は、広報・情報ネットワーク・安全衛生管理の三業務を担っています。公式WebサイトやSNSでの情報発信に加え、所内ネットワーク環境や安全衛生管理体制の整備を推進し、研究環境の維持向上に努めています。

The Public Relations and Information Office manages three core functions: public relations, network administration, and safety and health management. In addition to disseminating information through the official website and social media, the Office maintains and enhances the institute's network infrastructure and safety management systems to support a stable research environment.

国際戦略室 International Strategy Office

室長
魏 范研

Head of the Office
Fan-Yan Wei

国際戦略室は、URA等の専門スタッフが、国際研究力強化の推進、研究動向の分析、戦略的な国際協働の立案、研究費獲得支援等を担い、研究所の国際的プレゼンスを向上させます。また、翻訳や英語サイト拡充を通じ、グローバルな研究環境の構築を支援します。

The International Strategy Office advances IDAC's international research capacity by analyzing global research trends, supporting the development of research strategies, promoting strategic international collaboration, and assisting in the acquisition of competitive research funding. The Office also contributes to building a global research environment through English translation of administrative documents and enhancement of IDAC's English-language website.

各種データ Various Data

職員数 Faculty & Staff 2026.5.1 現在 (as of May 1, 2026)

教 授 Professors	9(1)	事 務 職 員 Administrative Staff	15(8)
准教授 Associate Professors	6(2)	技 術 職 員 Technical Staff	7(4)
講 師 Senior Assistant Professors	2(1)	限定正職員 Limited Regular Staff	9(9)
助 教 Assistant Professors	27(9)	非常勤職員 Part Time Staff	50(44)
計 Total	125 (78)	() 内は女性で内数 () indicates the number of female staff included in counts.	

学生数 Students 2026.5.1 現在 (as of May 1, 2026)

事 項 Categories	学部生 Undergraduate	博士課程前期 Master Course	博士課程後期 Doctor Course	研究所等 研究生 Research Students	合計 Total
医学系研究科 Grad.Sch.Medicine	2 (1)	17 (11)	54 (20)		73 (32)
医学部医学科 Dept.Medicine	27 (6)		7 (1)		34 (7)
医学部保健学科 Dept. Medicine,Health Sciences	4 (3)	2 (1)			6 (4)
工学研究科 Grad.Sch.Engineering		3 (1)	2 (1)		5 (2)
国際文化研究科 Grad.Sch.International Cultural Studies			2 (2)		2 (2)
生命科学研究科 Grad.Sch.Life Sciences		14 (7)	6 (3)		20 (10)
医工学研究科 Grad.Sch.Biomedical		5 (1)	2 (0)		7 (1)
加齢医学研究所 IDAC				1 (0)	1 (0)
計 Total	33 (10)	41 (21)	73 (27)	1 (0)	148 (58)

() 内は女性で内数
() indicates the number of female staff included in counts.

運営費交付金予算 Operation Grants (単位：千円) (in units of 1,000 yen)

事 項 Categories / 年 度 Fiscal Year		2021	2022	2023	2024	2025
運営費交付金 Operation Grants	人件費 Personnel Expenses	389,873	393,001	385,594	376,740	385,945
	物件費 Non-Personnel Expenses	135,273	132,563	130,207	127,873	125,599
運営費交付金 Operation Grants Total		525,146	525,564	515,801	504,613	511,544

建物 Buildings

建物総延面積 Total Floor Area of Buildings	20,957㎡
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主な外部資金受入状況 External Funds (単位：千円) (in units of 1,000 yen)

事 項 Categories / 年 度 Fiscal Year	2021	2022	2023	2024	2025
科学研究費補助金 Grants-in-Aid for Scientific Research	199,160	206,570	254,670	171,080	180,440
受託研究費 Funds for Commissioned Research	214,751	203,670	197,317	575,291	407,396
共同研究 Joint Research	146,219	174,033	152,202	67,951	150,162
寄附金 Donations	150,372	114,330	84,669	113,833	71,354
計 Total	710,502	698,603	688,858	953,125	809,352

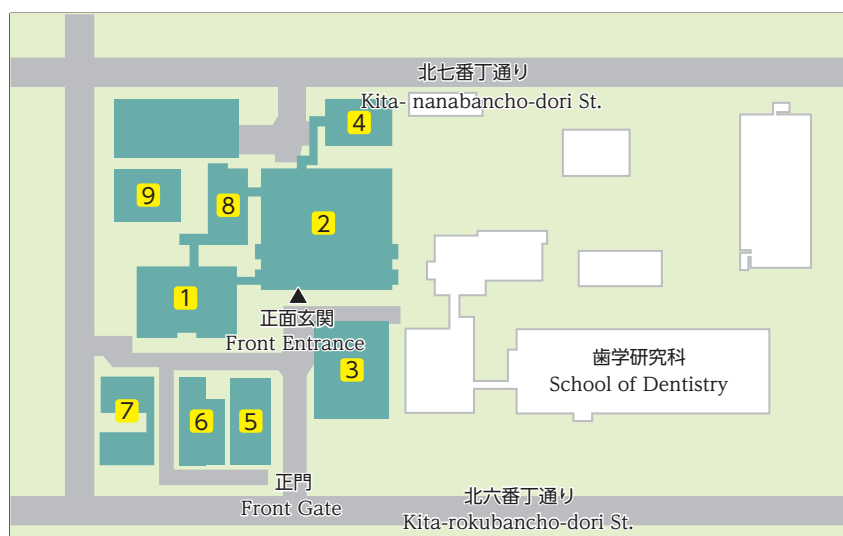
学術交流協定締結校

Agreements on Academic Exchange with Foreign Research Institutes and Universities

大学名 Name of University	国名 country	締結年月 entering date
イリノイ大学工学部 バイオアクスティクス研究所 University of Illinois	アメリカ USA	1988.8
遼寧省腫瘍病院・遼寧省腫瘍研究所 Liaoning Cancer Hospital And Institute	中国 China	1998.12
スモレンスクステートメディカルアカデミー Smolensk State Medical Academy	ロシア Russia	2003.7
キューバ・ニューロサイエンス・センター Cuba Neuroscience Center	キューバ Cuba	2008.3
マサリク大学医学部 Masaryk University	チェコ Czech Republic	2008.5
マギル大学モントリオール神経研究所 Mcgill University	カナダ Canada	2010.6
トリノ大学 University of Turin	イタリア Italy	2011.7
ユヴァスキュラ大学 University of Jyväskylä	フィンランド Finland	2012.4
コペンハーゲン大学ヘルシーエイジング研究センター University of Copenhagen	デンマーク Denmark	2012.9
西南大学心理学部 Southwest University	中国 China	2012.11
テマセック・ポリテクニク Temasek Polytechnic	シンガポール Singapore	2013.7
マッコーリー大学・オーストラリアン・スクール・オブ・アドバンスト・メディシン Macquarie University	オーストラリア Australia	2014.4
アラブ首長国連邦大学医学健康科学部 United Arab Emirates University	アラブ首長国連邦 United Arab Emirates	2016.7
レディング大学心理学・臨床言語科学部 University of Reading	イギリス United Kingdom	2016.9
シドニー大学 University of Sydney	オーストラリア Australia	2017.2
ベルゲン大学医学部 University of Bergen	ノルウェー Norway	2017.5
カスティーリャ・ラ・マンチャ大学 University of Castilla-La Mancha	スペイン Spain	2017.12
チャングン大学 Chang Gung University	台湾 Taiwan	2018.12
国立中正大学 National Chung Cheng University	台湾 Taiwan	2019.7
ジーゲン大学 University of Siegen	ドイツ Germany	2024.4
カロリンスカ研究所 Karolinska Institutet	スウェーデン Sweden	2025.8

アクセスマップ Access Map

所在配置図 External Funds



東北大学加齢医学研究所

〒980-8575 宮城県仙台市青葉区星陵町4-1

Institute of Development, Aging and Cancer,
Tohoku University 4-1 Seiryochō, Aobaku, Sendai
980-8575, Japan

- 1 実験研究棟
Center for Basic Aging Research
- 2 プロジェクト総合研究棟
Center for Clinical Aging Research
- 3 スマート・エイジング研究棟
Center for Smart Aging Research
- 4 先進フロンティア研究棟
Laboratory for Advanced Frontiers Research
- 5 ブレインダイナミクス研究棟
Laboratory for Brain Dynamics Research
- 6 ブレインイメージング研究棟
Laboratory for Brain Imaging Research
- 7 腫瘍動物実験棟
Facility for Tumor and Animal Experiment
- 8 先端医療実験棟
Aging and Cancer, Pre-clinical Research Center
- 9 動物資源実験棟
Center for Laboratory Animal Resources

星陵地区配置図 Seiryō Campus Map



仙台駅からのアクセス Access from Sendai Station



仙台市営地下鉄 Sendai City Subway

地下鉄南北線仙台駅から泉中央行きに乗車し、北四番丁駅下車後、北2出口より八幡町方面へ徒歩約15分

※詳細は仙台市交通局ホームページをご覧ください。

Take the North-South Subway Line bound for "Izumi Chuo" at Sendai Sta. and get off at "Kita-yobancho Sta." From North 2 Exit, about 15 minutes walk from that exit.

※Visit Transportation Bureau City of Sendai website for further information.

仙台市営バス Sendai City Bus

仙台駅バスのりば案内 Sendai Station Bus Stop

のりば番号 Bus Stop No.	経由地・行先 Route	下車停留所 Destination	所要時間 Time
9	北山→子平町循環 広瀬通駅経由 Kitayama-Shiheicho Loop Line Via Hirose-dori Station	東北大学病院前 Tohoku Daigaku Byouin Mae	約20分 About 20 min

※詳細は仙台市交通局ホームページをご覧ください。 Visit Transportation Bureau City of Sendai website for further information.

タクシー Taxi

仙台駅から所要時間約15分

About 15 min. from Sendai Sta.

仙台空港からのアクセス Access from Sendai Airport to Sendai Station

仙台空港から仙台駅まで仙台空港アクセス線で約25分。

Take the Sendai Airport Access Line from Sendai Airport and get off at Sendai Station. About 25 min.

東北大学 加齢医学研究所 概要

Institute of Development,
Aging and Cancer
Tohoku University

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