第 18 回 寄生虫感染免疫研究会

（高知・寄生虫・研究のキーワードで作成した生成 AI イメージです・・・）

令和 8 年 1 月 29 - 30 日

開催場所：高知城ホール

世話人代表　徳島大学　安友康二

概要

・**日時：**令和 8 年 1 月 29 - 30 日

・**場所：**高知城ホール

・**参加費：2000 円**　学生無料

・**意見交換会**（p.10 参照）**5500 円**（実費）

・**発表時間：**20 分

・**質疑：**10 分

時間は座長の裁量によって調節可能です

・**COI の開示：**

必要な場合は、スライドでお示し下さい。

(口頭でも可)

会場

一般財団法人 高知県教育会館
高知城ホール　4 階多目的ホール

所在地：〒780-0850 高知県高知市丸ノ内二丁目 1 番 10 号
TEL: 088-822-2035
FAX: 088-822-2037

アクセス

🚗高知インターチェンジから車で 20 分

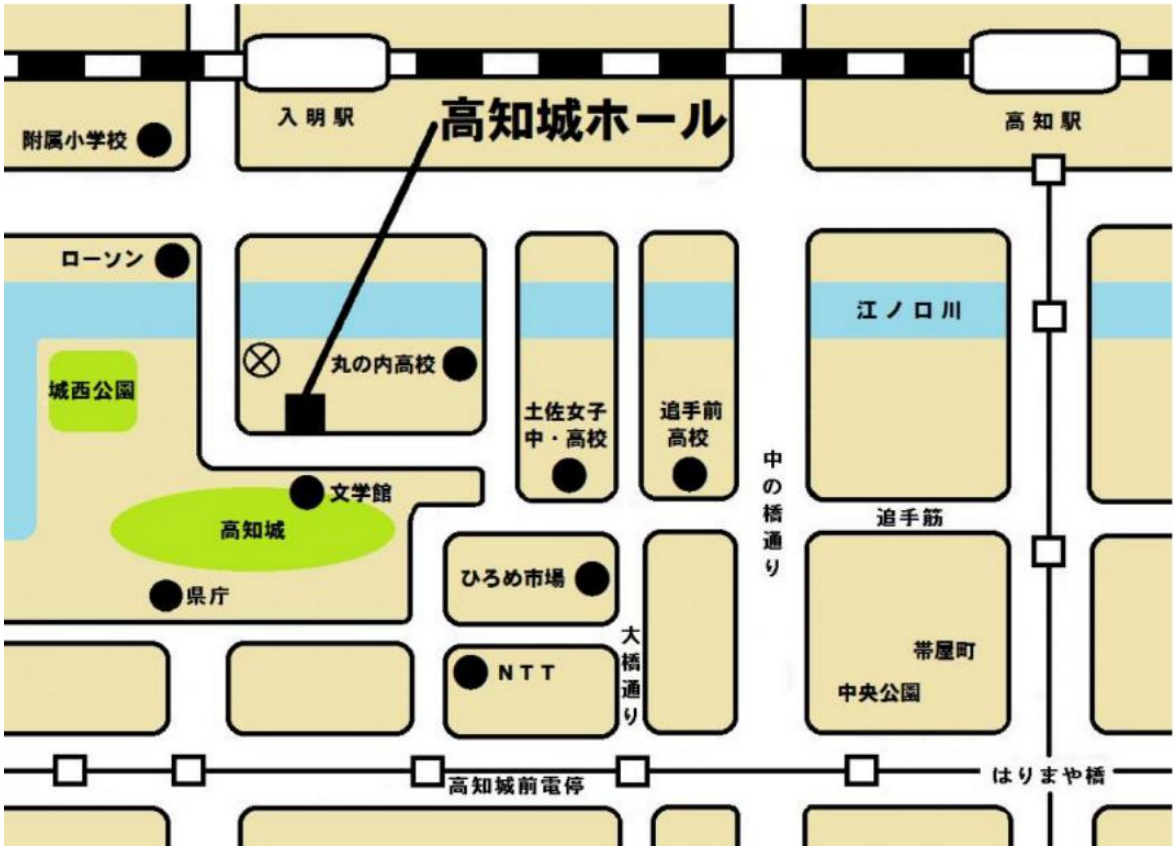
🚗JR 高知駅から車で 5 分

🚗高知龍馬空港から車で 30 分

🚗高知港から車で 15 分

🚗高知龍馬空港からバスで約 40 分

🚋ときでん交通電停「高知城前」から徒歩約 10 分



タイムテーブル

【1 月 29 日（木)】

12:00	受付
12:50	開会の挨拶
13:00	セッション A　座長：吉田裕樹（佐賀大学）
14:30-15:00	休憩
15:00	セッション B　座長：久枝一（国立感染症研究所）
17:00	写真撮影
17:05-17:20	世話人会

【1 月 30 日（金)】

8:30	開場
9:00	セッション C　座長：前川洋一（岐阜大学）
10:30-11:00	休憩
11:00	セッション D　座長：小林隆志（大分大学）
12:00	閉会の挨拶

プログラム

【1 日目】

1 月 29 日 13:00-14:30

セッション A

座長：吉田裕樹（佐賀大学）

A-1
13:00-13:30

One Health–Based Assessment of Toxoplasma Infection and Identification of Novel Virulence Factors

○Naganori Kamiyama[1,2], Eito Tanabe[1], Yoshiaki Hayashi[1], Mai Ueno[1], Nozomi Sachi[1], Sotaro Ozaka[1], Yomei Kagoshima[1], Supanuch Ekronarongchai[1], Tipanan Khunsri[1], Masaaki Okamoto[1], Masahiro Yamamoto[3], Takashi Kobayashi[1,2]

[1]Department of Infectious Disease Control, Faculty of Medicine, Oita University, Yufu, Oita, Japan., [2]Research Center for GLOBAL and LOCAL Infectious Diseases, Oita University, Yufu, Oita, Japan., [3]Department of Immunoparasitology, Research Institute for Microbial Diseases, Osaka University, Suita, Osaka, Japan.

A-2
13:30-14:00

Role of Th1-type Treg in immune regulation at mucosal site following *Toxoplasma* infection

○Miwa Sasai, Masahiro Yamamoto.

Dept. of Immunoparasitology, RIMD, IFReC, CiDER, CAMaD, The University of Osaka

A-3
14:00-14:30

The alteration of gene expression in *Trichinella* under different host environments

○Yoichi Maekawa, Sukhonthip Khuangchiangkhwang, Zhiliang Wu

Department of Parasitology and Infectious Diseases, Gifu University Graduate School of Medicine

s1 月 29 日 15:00-17:00

セッション B

座長：久枝一（国立感染症研究所）

B-1
15:00-15:30

Visual Detection of Malaria Parasite-Parasitized Erythroblasts in Peripheral Blood via Immunization-Based Model

Kumpei Ito[1,†] , Yuki S. Tateishi[1,†], ○Takashi Imai[1,2,3,4,5], Shinya Miyazaki[6] , Yukiko Miyazaki[6],Wataru Kagaya[7] , Mai Nakashima[8,9], Miho Sase[1], Misato Yoshioka-Takeda[1], Chikako Shimokawa[1], Kyoko Hayashi[1], Kentaro Itokawa[5,10], Osamu Komagata[5,10], Ha Ngo-Thanh[2,11], Aoi Shimo[1,3],Tamasa Araki[1,12], Takeshi Annoura[1], Takashi Murakami[3] and Hajime Hisaeda[1]

[1]Department of Parasitology, National Institute of Infectious Diseases (NIID), Japan Institute for Health Security (JIHS), [2]Department of Infectious Diseases and Host Defense, Gunma University, [3]Department of Microbiology, Saitama Medical University, [4]Leprosy Research Center, NIID, JIHS, [5]Antimicrobial Resistance Research Center, NIID, JIHS, [6]Department of Protozoology, Institute of Tropical Medicine, Nagasaki University, [7]Department of Ecoepidemiology, Institute of Tropical Medicine, Nagasaki University, [8]Department of Molecular Protozoology, Research Institute for Microbial Diseases (RIMD), Osaka University, [9]Department of Tropical Medicine and Malaria, Research Institute, National Center for Global Health and Medicine (NCGM), JIHS, [10]Department of Medical Entomology, NIID, JIHS, [11]National Hospital for Tropical Disease, Hanoi 10000, Vietnam, [12]Research Center for Biosafety, Laboratory Animal and Pathogen Bank, NIID, JIHS, [†]These authors contributed equally to this work.

B-2
15:30-16:00

Mechanisms underlying the IL-27-mediated regulation of *Plasmodium*-specific memory CD4$^+$ T cells

○Katsuyuki Yui[1, 2, 7], Sanjaadorj Tsogtsaikhan[2], Shin-Ichi Inoue[2], Hirotaka Matsumoto[3], Odsuren Skbaatar[2], Ganchimeg Bayarsaikhan[2], Masahiro Yamaoto[4], Hiroki Yoshida[5], Daisuke Kimura[2], Julius Hafalla[6], Maria Lourdes Macalinao[7]

[1]Shionogi Global Infectious Dis Div, Inst Trop Med, Nagasaki Univ, [2]Div Immunol, Dept Mol Microbiol Immunol, Grad Sch Biomed Sci, Nagasaki Univ; [3]Sch Informat Data Sci, Nagasaki Univ; [4]Dept Immunoparasitol, Res Inst Microbial Dis, Osaka Univ, [5]Div Mol Cell Immunosci, Dept Biomol Sci, Facul Med, Saga Univ; [6]Dept Infect Biol, Fac Infect Trop Dis, LSHTM, UK; [7]TMGH, Nagasaki Univ

B-3
16:00-16:30

Access and utilization of host-derived iron by *Leishmania* parasites

○Yasuyuki Goto

Laboratory of Molecular Immunology, Department of Animal Resource Sciences, Graduate School of Agricultural and Life Sciences, The University of Tokyo

B-4

16:30-17:00

Recent advances in malaria and leishmaniasis research in Malaria Immunology Lab, IMSUT

○Cevayir Coban[1,2,3]

[1]Division of Malaria Immunology, Department of Microbiology and Immunology, Institute of Medical Science (IMSUT), University of Tokyo, JAPAN, [2]International Vaccine Design Center (VDesC), Institute of Medical Science (IMSUT), University of Tokyo, JAPAN, [3]The University of Tokyo Pandemic Preparedness, Infection and Advanced Research Center (UTOPIA), The University of Tokyo, JAPAN

【2 日目】

1 月 30 日 9:00-10:30

セッション C

座長：前川洋一（岐阜大学）

C-1

9:00-9:30

T-Cell Immunogenicity of AI-Predicted Malaria Vaccine Elements Assessed by IFN-γ Enzyme-Linked Immunospot Assay

Shara Bakytbek[1,2,3], Agnieszka Gromadka[4], Wataru Kagaya[3,5], Huai Chuang[3,6], ○Yarob Ibraheem[1,2,3], Farzaneh Valanezhad[1,2,3], Ayako Hyuga[1,2,3], Marius Gheorghe[4], Daiki Miura[7], Alexandru Odainic[4], Shin-Ichi Inoue[2,8], Shusaku Mizukami[3,9], Satoshi Kaneko[3,5], Kazuhide Onoguchi[7], Sebastian Kapell[4,10], Kaidre Bendjama[4], Osamu Kaneko[3,6], Shinjiro Hamano[1,2,3]

[1]Department of Parasitology, Institute of Tropical Medicine (NEKKEN), The Joint Usage/Research Center on Tropical Disease, Nagasaki University, Nagasaki, Japan, [2]Graduate School of Biomedical Sciences, Nagasaki University, Nagasaki, Japan, [3]The Vaccine Research and Development Center, DEJIMA Infectious Disease Research Alliance (DIDA), Nagasaki University, Nagasaki, Japan, [4]NEC Oncoimmunity AS, Oslo, Norway, [5]Department of Eco-epidemiology, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan, [6]Department of Protozoology, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan, [7]AI Drug Development Division, NEC Corporation, Tokyo, Japan, [8]Department of Molecular Microbiology and Immunology, Division of Immunology, Nagasaki University, Nagasaki, Japan, [9]Department of Immune Regulations, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan, [10]Department of Vaccine Informatics, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan

C-2
9:30-10:00

Modulating Intracellular Protein Production from mRNA by Attenuating Innate Immune Responses

Satoshi Miyagawa[1], Mayumi Taniguchi[1,2], Sayuri Nakamae[1,3], Jiun-Yu Jian[1], Katsuyuki Yui[4], Kenji Hirayama[2], Shigeru Kawakami[5] and ○Shusaku Mizukami[1,2]

[1]Department of Immune Regulation, Shionogi Global Infectious Diseases Division (SHINE), Institute of Tropical Medicine (NEKKEN), Nagasaki University, [2]School of Tropical Medicine and Global Health (TMGH), Nagasaki University, [3]Department of Clinical Product Development, Division of Clinical Medicine and Research, NEKKEN, Nagasaki University, [4]SHINE, NEKKEN, Nagasaki University, [5]Department of Pharmaceutical Informatics, Graduate School of Biomedical Sciences, Nagasaki University

C-3
10:00-10:30

Sequential Class Switching Drives Long-Lived IgE-Producing Plasma Cells in a Percutaneous Food Allergy Model

○Michio Tomura, Mayuko Hashimoto, Yutaka Kusumoto

Laboratory of Immunology, Faculty of Pharmacy, Osaka Ohtani University

---

1 月 30 日 11:00-12:00

セッション D

座長：小林隆志（大分大学）

D-1
11:00-11:30

Identification of the immunoreactive component in *Giardia intestinalis*

○Shigenari Ishizuka[1], Yasunobu Miyake[1], Makoto Kazama[2], Fumika Michi[2,3], and Hiroki Yoshida[1]

[1]Molecular and Cellular Immunoscience, Department of Biomolecular Sciences, Faculty of Medicine, Saga Univ., [2] NEKKEN Bio-Resource Center (BRC), Institute of Tropical Medicine (NEKKEN), Nagasaki Univ., [3]Central Laboratory, Institute of Tropical Medicine (NEKKEN), Nagasaki Univ.

D-2
11:30-12:00

An Integrated Strategy Toward Control and Elimination of Schistosomiasis in Kenya: The SATREPS Kenya Project

○Shinjiro Hamano, M.D., Ph.D.
Department of Parasitology; Institute of Tropical Medicine (NEKKEN), Nagasaki University

意見交換会

日時：令和 8 年 1 月 29 日(木)　19 時より

場所：個室四季料理　くらり

〒780-0822 高知県高知市はりまや町３丁目１－１２

TEL：088-875-7770

URL: https://kurari.owst.jp/

←お店の HP の QR コード

アクセス：

とさでん『蓮池町通』『高知橋』電停から徒歩 2 分

JR 高知駅から徒歩 5 分／グリーンホテルからすぐ

Map は WEB ページにも掲載されています

予約名：寄生虫感染免疫研究会

会費：5500 円（4000 円のコース＋2 時間飲み放題 1500 円）

抄録集

A-1

One Health–Based Assessment of Toxoplasma Infection and Identification of Novel Virulence Factors

○Naganori Kamiyama[1,2], Eito Tanabe[1], Yoshiaki Hayashi[1], Mai Ueno[1], Nozomi Sachi[1], Sotaro Ozaka[1], Yomei Kagoshima[1], Supanuch Ekronarongchai[1], Tipanan Khunsri[1], Masaaki Okamoto[1], Masahiro Yamamoto[3], Takashi Kobayashi[1,2]

[1]Department of Infectious Disease Control, Faculty of Medicine, Oita University, Yufu, Oita, Japan., [2]Research Center for GLOBAL and LOCAL Infectious Diseases, Oita University, Yufu, Oita, Japan., [3]Department of Immunoparasitology, Research Institute for Microbial Diseases, Osaka University, Suita, Osaka, Japan.

Toxoplasma gondii is a zoonotic parasite that infects all warm-blooded animals. Primary infection in a non-immune pregnant woman can cause congenital toxoplasmosis, leading to severe fetal complications such as hydrocephalus and miscarriage. In Japan, the seroprevalence of anti-Toxoplasma antibodies is estimated to be less than 10%, which is low even by global standards. Given Japan's declining birthrate and aging population, clarifying the prevalence of Toxoplasma infection in humans and animals is an urgent public health issue. Using a government–academia collaborative One Health approach, this study aimed to comprehensively assess the prevalence of Toxoplasma infection in humans and animals, including pigs and wild boars, in Oita Prefecture. Our goal is to establish a system in which pregnant women can give birth without excessive concern about Toxoplasma infection and to promote effective prevention of infection in livestock.

Furthermore, because no effective vaccine against Toxoplasma has yet been developed, vaccine research is actively underway worldwide. The efficacy of live-attenuated vaccines using genetically modified Toxoplasma strains has been demonstrated in mouse models. Therefore, identifying novel Toxoplasma virulence factors may provide critical insights for the future development of preventive vaccines. To identify new virulence factors, we performed RNA sequencing to comprehensively compare gene expression profiles between virulent and attenuated Toxoplasma strains and generated knockout strains of candidate genes that were highly expressed in virulent strains. We then conducted infection experiments using these gene-deficient strains to identify novel virulence factors associated with Toxoplasma pathogenicity.

A-2

Role of Th1-type Treg in immune regulation at mucosal site following *Toxoplasma* infection

○Miwa Sasai, Masahiro Yamamoto.
Dept. of Immunoparasitology, RIMD, IFReC, CiDER, CAMaD, The University of Osaka

Toxoplasma gondii is an intracellular parasitic protozoan capable of infecting all warm-blooded animals. Cases of congenital toxoplasmosis due to vertical transmission from infected mothers and acquired toxoplasmosis associated with immunosuppression have been reported within Japan. The primary route of *T. gondii* infection in humans is through oral ingestion of contaminated soil, water, or meat. However, it remains unclear which immune mechanisms control the infection within the host after oral inoculation and subsequently maintain homeostasis. Here, we investigated changes in the immune responses following oral infection with the *T. gondii*. Our results revealed that oral infection with *T. gondii* cysts induces a highly inflammatory response in the intestine. Inflammation is crucial for eliminating pathogens, but if it isn't resolved and returned to a steady state after elimination, it can lead to inflammatory diseases. Using the VeDTR mouse system, that newly developed in our laboratory, we investigated how inflammation is regulated following oral inoculation. We specifically labeled only Th1-type cells among regulatory T cells and eliminated Th1-type regulatory T cells (Th1-Treg) using diphtheria toxin, revealing that the mice exhibited high susceptibility following T. gondii infection. We will present the latest findings on the Th1-Treg-mediated immunosuppression mechanism in *T. gondii* cyst infection. If this inflammation is not controlled, it can lead to inflammatory diseases.

A-3

The alteration of gene expression in *Trichinella* under different host environments

○Yoichi Maekawa, Sukhonthip Khuangchiangkhwang, Zhiliang Wu
Department of Parasitology and Infectious Diseases, Gifu University Graduate School of Medicine

Trichinella spp. parasitize a wide range of hosts, including mammals, birds, and reptiles. However, the mechanisms underlying this broad host range remain unclear. To address this, we infected *Trichinella* parasites into murine hosts with distinct immune backgrounds.
When *Trichinella spiralis* was infected into antibody-deficient mice (Ab-KO), adult female worms recovered from these hosts were larger than those obtained from wild-type (WT) mice. Based on this observation, we hypothesized that the worms alter their gene expression programs to adapt to different host environments.

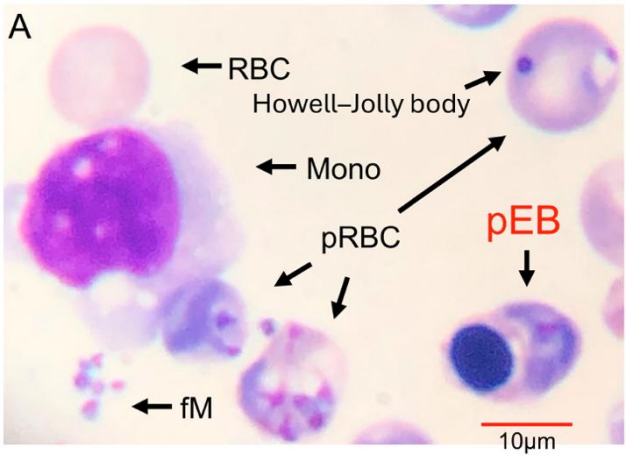
Using whole-transcriptome analysis, we found that gene expression profiles differed between adult worms derived from WT and Ab-KO mice. We initially speculated that genes related to body structure or growth would be upregulated in worms from Ab-KO mice; however, we did not observe clear changes in these categories. Instead, we found that several elastase-1 genes were downregulated in worms derived from Ab-KO hosts. We are currently investigating how host immune environments influence parasite gene expression and how these transcriptional changes relate to parasite development and host adaptation.

B-1

Visual Detection of Malaria Parasite-Parasitized Erythroblasts in Peripheral Blood via Immunization-Based Model

Kumpei Ito[1,†] , Yuki S. Tateishi[1,†], ○Takashi Imai[1,2,3,4,5], Shinya Miyazaki[6] , Yukiko Miyazaki[6],Wataru Kagaya[7] , Mai Nakashima[8,9], Miho Sase[1], Misato Yoshioka-Takeda[1], Chikako Shimokawa[1], Kyoko Hayashi[1], Kentaro Itokawa[5,10], Osamu Komagata[5,10], Ha Ngo-Thanh[2,11], Aoi Shimo[1,3],Tamasa Araki[1,12], Takeshi Annoura[1], Takashi Murakami[3] and Hajime Hisaeda[1]

[1]Department of Parasitology, National Institute of Infectious Diseases (NIID), Japan Institute for Health Security (JIHS), [2]Department of Infectious Diseases and Host Defense, Gunma University, [3]Department of Microbiology, Saitama Medical University, [4]Leprosy Research Center, NIID, JIHS, [5]Antimicrobial Resistance Research Center, NIID, JIHS, 6 Department of Protozoology, Institute of Tropical Medicine, Nagasaki University, 7 Department of Ecoepidemiology, Institute of Tropical Medicine, Nagasaki University, 8 Department of Molecular Protozoology, Research Institute for Microbial Diseases (RIMD), Osaka University, 9 Department of Tropical Medicine and Malaria, Research Institute, National Center for Global Health and Medicine (NCGM), JIHS, 10 Department of Medical Entomology, NIID, JIHS, 11 National Hospital for Tropical Disease, Hanoi 10000, Vietnam, 12 Research Center for Biosafety, Laboratory Animal and Pathogen Bank, NIID, JIHS
[†] These authors contributed equally to this work.



This study reports a murine model that enables the direct visualization of malaria parasite–parasitized erythroblasts (pEBs, see fig on right) in peripheral blood, overcoming a long-standing technical barrier in malaria research. Although erythroblasts have recently been recognized as host cells for *Plasmodium* parasites, their extreme rarity in circulation has previously restricted analyses to invasive bone marrow or spleen sampling.

We established an immunization–challenge model in which C57BL/6 mice were first immunized with non-lethal *Plasmodium yoelii* 17XNL and subsequently challenged with *Plasmodium berghei* ANKA. Immunized mice were protected from experimental cerebral malaria, survived longer, and developed sustained high parasitemia. Under these conditions, pEBs became consistently detectable in peripheral blood using standard Giemsa-stained smears.

All blood-stage parasite forms—rings, trophozoites, schizonts, merozoites, and gametocytes—were observed within erythroblasts, including rare images of parasite development during erythroblast enucleation. The appearance of pEBs correlated strongly with high parasitemia (>60%) and robust stress erythropoiesis, rather than anemia alone, as lethal infection with *P. yoelii* 17XL failed to produce circulating pEBs.

The study provides the first systematic morphological catalog of parasitized erythroblasts in peripheral blood and demonstrates that Plasmodium development within erythroid precursors can be studied *in vivo* without transgenic parasites or invasive tissue sampling. The authors propose that circulating pEBs may influence parasite propagation, anemia, and immune recognition, particularly through MHC class I–dependent $CD8^+$ T cell responses.

Overall, this work establishes a practical experimental platform for studying erythroblast–parasite interactions and highlights pEBs as a previously overlooked but potentially important component of malaria pathogenesis, immunity, and vaccine research.

B-2

Mechanisms underlying the IL-27-mediated regulation of *Plasmodium*-specific memory CD4$^+$ T cells

○Katsuyuki Yui[1, 2, 8], Sanjaadorj Tsogtsaikhan[2], Shin-Ichi Inoue[2], Hirotaka Matsumoto[3], Odsuren Skbaatar[2], Ganchimeg Bayarsaikhan[2], Masahiro Yamaoto[4], Hiromitsu Hara[5], Hiroki Yoshida[6], Daisuke Kimura[2], Julius Hafalla[7], Maria Lourdes Macalinao[8]

[1]Shionogi Global Infectious Dis Div, Inst Trop Med, Nagasaki Univ; [2]Div Immunol, Dept Mol Microbiol Immunol, Grad Sch Biomed Sci, Nagasaki Univ; [3]Sch Informat Data Sci, Nagasaki Univ; [4]Dept Immunoparasitol, Res Inst Microbial Dis, Osaka Univ; [5]Dept Immunol, Grad Sch Med Den Sci, Kagoshima Univ; [6]Div Mol Cell Immunosci, Dept Biomol Sci, Facul Med, Saga Univ; [7]Dept Infect Biol, Fac Infect Trop Dis, LSHTM, UK; [8] TMGH, Nagasaki Univ

Malaria infection elicits both protective and pathogenic immune responses, and cytokines play critical roles in the regulation of the immune responses. We investigated the role of IL-27 in the development of immunological memory to malaria during chronic malaria infection using MHC-II-restricted malaria antigen-specific T-cell receptor transgenic mouse, PbT-II, and *Plasmodium chabaudi* infection model.

C57BL/6 mice were transferred with PbT-II cells and were infected with *P. chabaudi*. Neutralization of IL-27 during the first week of infection promoted the expansion of PbT-II cells 2-3 weeks of the infection, which were maintained at high levels during chronic infection. PbT-II cells in IL-27-neutralized mice generated unique subpopulations of memory cells that displayed distinct gene expression patterns, cytokine production, and proliferative capacity from those in untreated mice. These CD4$^+$ T cells were maintained independent of active infection and appears to play protective roles in the secondary infection. Further studies demonstrated that IL-27 receptor signaling in both PbT-II cells and other T cells was critical for this regulation, suggesting the complexity of cytokine-mediated network regulation on the generation and maintenance of memory CD4$^+$ T cells during chronic malaria.

These findings demonstrate that IL-27, which is produced during the acute phase of malaria infection, acts at the time of T cell priming inhibiting the development of unique Th1 memory precursor CD4$^+$ T cells via cytokine network, suggesting implications for the development of vaccines and other strategic interventions.

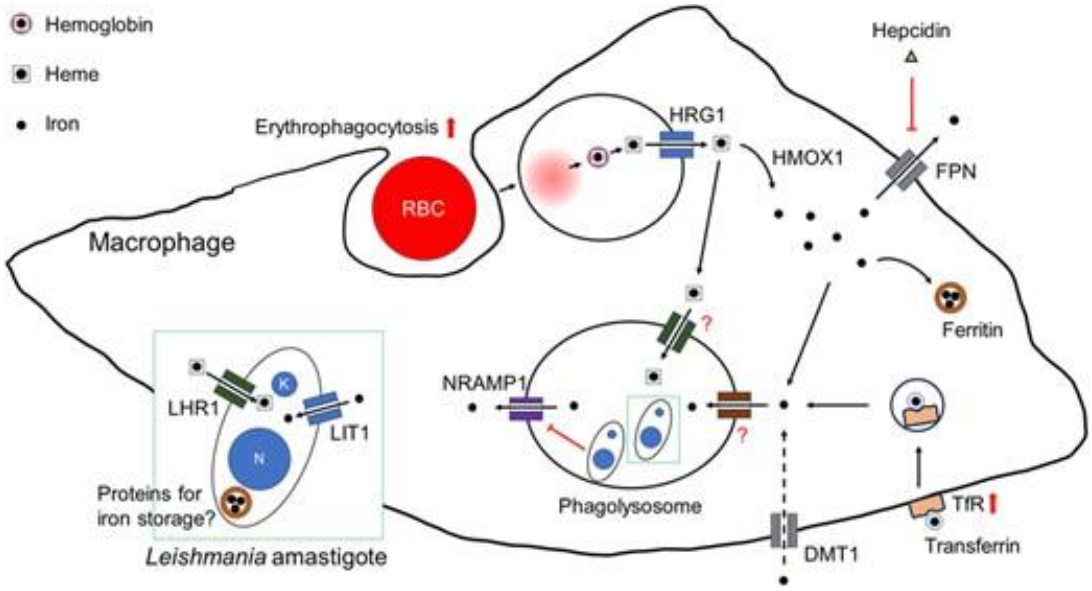
B-3

Access and utilization of host-derived iron by *Leishmania* parasites

○Yasuyuki Goto

Laboratory of Molecular Immunology, Department of Animal Resource Sciences, Graduate School of Agricultural and Life Sciences, The University of Tokyo

Iron is involved in many biochemical processes including oxygen transport, ATP production, DNA synthesis and antioxidant defense. The importance of iron also applies to *Leishmania* parasites, an intracellular protozoan pathogen causing leishmaniasis. Leishmania are heme-auxotrophs, devoid of iron storage proteins and the heme synthesis pathway. Acquisition of iron and heme from the surrounding niche is thus critical for the intracellular survival of *Leishmania* inside the host macrophages. Moreover, *Leishmania* parasites are also exposed to oxidative stress within phagolysosomes of macrophages in mammalian hosts, and they need iron superoxide dismutase for overcoming this stress. Therefore, untangling the strategy adopted by these parasites for iron acquisition and utilization can be good targets for the development of antileishmanial drugs. Here, I would like to introduce our research addressing acquisition and utilization of iron/heme by *Leishmania* parasites during infection to macrophages.



B-4

Recent advances in malaria and leishmaniasis research in Malaria Immunology Lab, IMSUT

○Cevayir Coban[1,2,3]

[1]Division of Malaria Immunology, Department of Microbiology and Immunology, Institute of Medical Science (IMSUT), University of Tokyo, JAPAN, [2]International Vaccine Design Center (VDesC), Institute of Medical Science (IMSUT), University of Tokyo, JAPAN, [3]The University of Tokyo Pandemic Preparedness, Infection and Advanced Research Center (UTOPIA), The University of Tokyo, JAPAN

Antimicrobial resistance (AMR) in pathogens including protozoan parasites (particularly for malaria and leishmaniasis) is a major challenge to global disease control. In this talk, I will focus on recent advances in our understanding of host–parasite interactions and discuss emerging strategies to address AMR in parasites, with an emphasis on host-directed interventions and immune-based approaches for the prevention and treatment of malaria and leishmaniasis.

C-1

T-Cell Immunogenicity of AI-Predicted Malaria Vaccine Elements Assessed by IFN-γ Enzyme-Linked Immunospot Assay.

Shara Bakytbek[1,2,3], Agnieszka Gromadka[4], Wataru Kagaya[3,5], Huai Chuang[3,6], ○Yarob Ibraheem[1,2,3], Farzaneh Valanezhad[1,2,3], Ayako Hyuga[1,2,3], Marius Gheorghe[4], Daiki Miura[7], Alexandru Odainic[4], Shin-Ichi Inoue[2,8], Shusaku Mizukami[3,9], Satoshi Kaneko[3,5], Kazuhide Onoguchi[7], Sebastian Kapell[4,10], Kaidre Bendjama[4], Osamu Kaneko[3,6], Shinjiro Hamano[1,2,3]

[1]Department of Parasitology, Institute of Tropical Medicine (NEKKEN), The Joint Usage/Research Center on Tropical Disease, Nagasaki University, Nagasaki, Japan, [2]Graduate School of Biomedical Sciences, Nagasaki University, Nagasaki, Japan, [3]The Vaccine Research and Development Center, DEJIMA Infectious Disease Research Alliance (DIDA), Nagasaki University, Nagasaki, Japan, [4]NEC Oncoimmunity AS, Oslo, Norway, [5]Department of Eco-epidemiology, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan, [6]Department of Protozoology, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan, [7]AI Drug Development Division, NEC Corporation, Tokyo, Japan, [8]Department of Molecular Microbiology and Immunology, Division of Immunology, Nagasaki University, Nagasaki, Japan, [9]Department of Immune Regulations, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan, [10]Department of Vaccine Informatics, Institute of Tropical Medicine (NEKKEN), Nagasaki University, Nagasaki, Japan

Malaria continues to be a substantial global health burden, with an estimated 282 million cases and 610,000 deaths worldwide. Although partially effective vaccines have been deployed, their limited protective efficacy underscores the need for improved vaccine strategies capable of inducing robust cellular immune responses. Protective immunity against malaria is closely associated with the availability and function of $CD4^+$ T cells and $CD8^+$ T cells, which target both the asymptomatic liver stage and the symptomatic blood stage of *Plasmodium falciparum* infection. In this study, malaria vaccine candidate antigens were selected using an AI-based prediction approach, designed to provide broad population coverage across multiple HLA class I and II alleles. Peripheral blood mononuclear cells (PBMCs) were obtained from malaria-exposed individuals (n = 41) and stimulated *ex vivo* with overlapping peptide pools representing the selected vaccine elements. Antigen-specific T-cell responses were assessed using a high-sensitivity interferon-gamma (IFN-γ) Enzyme-Linked ImmunoSpot (ELISPOT) assay. The frequency of epitope-specific IFN-γ responses was quantified to evaluate circulating memory T-cell populations and to functionally validate the *in silico* epitope predictions. Collectively, these findings enhance our understanding of malaria-specific cellular immune responses in naturally exposed individuals and provide experimental support for incorporating AI-driven antigen selection into next-generation malaria vaccine development.

C-2

Modulating Intracellular Protein Production from mRNA by Attenuating Innate Immune Responses

Satoshi Miyagawa[1], Mayumi Taniguchi[1,2], Sayuri Nakamae[1,3], Jiun-Yu Jian[1], Katsuyuki Yui[4], Kenji Hirayama[2], Shigeru Kawakami[5] and ○Shusaku Mizukami[1,2]

[1]Department of Immune Regulation, Shionogi Global Infectious Diseases Division (SHINE), Institute of Tropical Medicine (NEKKEN), Nagasaki University, [2]School of Tropical Medicine and Global Health (TMGH), Nagasaki University, [3]Department of Clinical Product Development, Division of Clinical Medicine and Research, NEKKEN, Nagasaki University, [4]SHINE, NEKKEN, Nagasaki University, [5]Department of Pharmaceutical Informatics, Graduate School of Biomedical Sciences, Nagasaki University.

Early innate immune responses, including type I interferon signaling, are rapidly induced during pathogen infection and play critical roles in host defense. Previous studies have shown that pathogen-derived immune evasion factors, such as viral NS1 proteins, can enhance protein expression from exogenously delivered mRNA by suppressing innate immune activation. However, how modulation of distinct innate immune regulatory pathways differentially impacts intracellular protein production from mRNA under infection-associated conditions remains incompletely understood.

In this study, we examined whether attenuation of innate immune signaling through a mechanistically distinct immune evasion factor could enhance intracellular protein production from mRNA. Co-expression of the SARS-CoV-2 viral protein ORF6 significantly increased reporter protein expression from mRNA, particularly under conditions of high-dose mRNA transfection and poly(I:C) stimulation that mimic infection-associated innate immune activation. This enhancement was accompanied by suppression of IFN-β and interferon-stimulated gene expression, suggesting that ORF6 primarily modulates upstream innate immune signaling pathways that indirectly constrain intracellular protein production from mRNA.

Together, these findings extend previous observations by demonstrating that immune evasion factors operating through distinct regulatory pathways can differentially modulate intracellular protein production from mRNA. In addition, these findings suggest that pathway-specific innate immune regulation under infection-associated immune conditions may influence protein expression from mRNA-based platforms, including in contexts relevant to parasite infection immunity.

C-3

Sequential Class Switching Drives Long-Lived IgE-Producing Plasma Cells in a Percutaneous Food Allergy Model

○Michio Tomura, Mayuko Hashimoto, Yutaka Kusumoto
Laboratory of Immunology, Faculty of Pharmacy, Osaka Ohtani University

Recent studies have shown that allergen-specific, high-affinity IgE–producing long-lived plasma cells (IgE-LLPCs), which are mainly maintained in the bone marrow, play a critical role in sustaining allergen-specific IgE during periods without antigen exposure and in driving type I allergic responses. During sensitization, two functionally distinct IgE-producing plasma cell populations are generated in the draining lymph nodes: short-lived plasma cells producing low-affinity IgE and long-lived plasma cells producing high-affinity IgE capable of inducing anaphylaxis. While IgE responses induced during parasitic infections are generally thought to arise via direct class switching from IgM^+ B cells, short-lived IgE-producing plasma cells similarly arise via direct class switching and preferentially localize to the spleen. In contrast, IgE-LLPCs in allergic settings are generated through a sequential IgM–IgG1–IgE class-switch pathway accompanied by affinity maturation, and predominantly reside in the bone marrow, where they persist long after sensitization.

To examine whether percutaneous sensitization induces IgE-LLPCs, we established a chronic skin-sensitization food allergy model using IgE-Venus reporter mice, in which IgE-producing plasma cells can be identified as $Venus^+$ cells. Repeated epicutaneous application of ovalbumin following tape stripping induced $CD138^+$ IgE-producing plasma cells in the bone marrow and spleen only after prolonged sensitization, whereas short-term sensitization was insufficient. These findings support the notion that sequential class switching and prolonged antigen exposure, rather than sensitization frequency, are critical for the generation of IgE-LLPCs. These cells persisted for at least 25 weeks after the final sensitization without further antigen exposure, indicating their maintenance as IgE-LLPCs. Consistent with this persistence, allergen-specific IgE remained detectable and oral antigen challenge elicited allergic symptoms, supporting a functional role for IgE-LLPCs in sustaining food allergy responses.

In addition, we will present single-cell transcriptomic analyses of long-lived plasma cells of other immunoglobulin classes.

D-1

Identification of the immunoreactive component in *Giardia intestinalis*

○Shigenari Ishizuka[1], Yasunobu Miyake[1], Makoto Kazama[2], Fumika Mi-ichi[2,3], and Hiroki Yoshida[1]

[1]Molecular and Cellular Immunoscience, Department of Biomolecular Sciences, Faculty of Medicine, Saga Univ., [2] NEKKEN Bio-Resource Center (BRC), Institute of Tropical Medicine (NEKKEN), Nagasaki Univ., [3]Central Laboratory, Institute of Tropical Medicine (NEKKEN), Nagasaki Univ.

Host immune system employs innate immune receptors to immediately detect and efficiently eliminate pathogenic microorganisms by inducing immune cell activation. While the receptors for bacteria, fungi, and viruses have been well characterized, receptors involved in protozoa recognition have not fully investigated. To address this point, we examined the reactivity of 50 human immune receptors against 10 protozoan species utilizing a cell-based high-throughput reporter system. Among those, the paired receptors MIRA and MIRI (myeloid immunoglobulin-like receptor for activation and inhibition) exhibited specific reactivity to Giardia intestinalis lysate. Experiments using MIRA or MIRI-expressing reporter cells, as well as Ig fusion protein, suggests that the ligand is an intracellular protein. As a next step, we plan to fractionate Giardia proteins by liquid chromatography in combination with our reporter system to track the ligand activity, followed by mass spectrometry to identify the molecular structure.

D-2

An Integrated Strategy Toward Control and Elimination of Schistosomiasis in Kenya: The SATREPS Kenya Project

○Shinjiro Hamano, M.D., Ph.D.
Department of Parasitology; Institute of Tropical Medicine (NEKKEN), Nagasaki University

Schistosomiasis remains a major public health challenge in sub-Saharan Africa, with over 90% of global cases occurring in this region. The SATREPS Project, led by Nagasaki University and in partnership with Kenyan institutions, aims to establish an evidence-based, integrated control program to accelerate the transition from disease control to elimination. The project targets two major endemic areas: Homa Bay County (*Schistosoma mansoni*) and Kwale County along the Indian Ocean coast (*Schistosoma haematobium*).

The project is built upon three interlinked research pillars:
(1) Transmission monitoring,
(2) Drug discovery and development, and
(3) Behavior change communication (BCC).

For transmission monitoring, the project has developed a high-sensitivity human monitoring tool based on novel antigen combinations (Serpin and RP26) that outperform conventional microscopy and show strong potential for elimination-phase surveillance in both *S. mansoni* and *S. haematobium* endemic settings.

In parallel, innovative environmental DNA (eDNA) surveillance technologies were developed, including the QuickConc method, which significantly improves *Schistosoma* DNA detection from water bodies and enables rapid, electricity-free field processing.

In the drug discovery component, a unique *in vitro–in vivo* screening platform was established for Kenyan isolates of *S. mansoni* and *S. haematobium*. Screening of approximately 70,000 chemical compounds will generate a sustainable pipeline for identifying lead candidates with both prophylactic and therapeutic activity, strengthening Kenya's local research capacity for anti-schistosomal drug development.

The BCC pillar integrates epidemiological and social science approaches to identify behavioral and environmental drivers of transmission. Community-based interventions in Mbita and Kwale revealed persistent knowledge gaps, unsafe water-contact practices, and infrastructure limitations, which are now being translated into locally adapted intervention packages aligned with Kenya's national NTD Breaking Transmission Strategy.

Through strong collaboration between Nagasaki University, KEMRI, Maseno University, county governments, and international partners, the SATREPS Kenya Project is building a scalable, data-driven model for schistosomiasis elimination. The tools, protocols, and policy linkages developed under this program will provide a robust foundation for expanding elimination efforts across endemic regions in Kenya and beyond.