Division of Internal Medicine
Department of Human Biology and Pathophysiology

Outline

The Takahashi Laboratory is primarily interested in elucidating the pathophysiology of metabolic diseases, such as diabetes and dyslipidemia, and the mechanisms of therapeutic agents to generate original insights in the laboratory. We use state-of-the-art techniques including molecular biology, genetics, physiology and biochemistry.

If you would like to conduct research in our laboratory, we are flexible in designing a topic to suit your individual interests, such as the fusion of dentistry with medicine and molecular biology (e.g. basic research on bone and periodontal bacteria and glucose metabolism). We currently perform cell culture and molecular biological analyses (e.g. DNA, RNA: especially non-coding RNA, proteins, and extracellular secretory vesicles: especially exosomes) on daily basis, so you can leave the learning of these research techniques to us.

Of course, you may not know where to start at first, so we will support you in all aspects of your research activities, including choosing a specific research topic that will lead to novel knowledge, searching and reading literature, experimental procedures, troubleshooting, discussing results, writing and submitting papers, and writing research proposals. We will provide you with the best support for your overall research activities. Please feel free to contact us!

Faculty members

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Current research interests

1. Elucidation of molecular patho-mechanisms in metabolic diseases such as diabetes and obesity
2. Elucidation of molecular crosstalk between skeletal muscle, bone (osteo-sarcopenia) and metabolic organs
3. Involvement of non-coding RNAs (especially long non-coding RNAs) in various pathological conditions
4. Involvement of extracellular secretory vesicles (especially exosomes) in various pathological conditions

Fig. 1 :

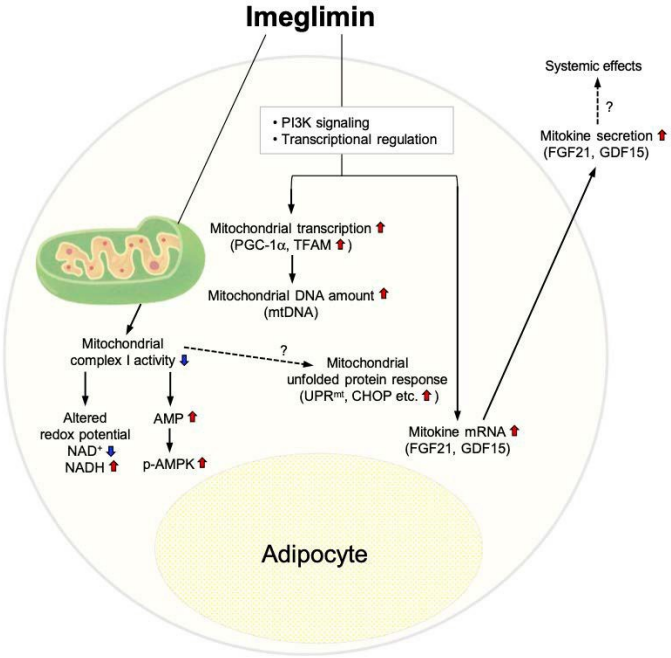
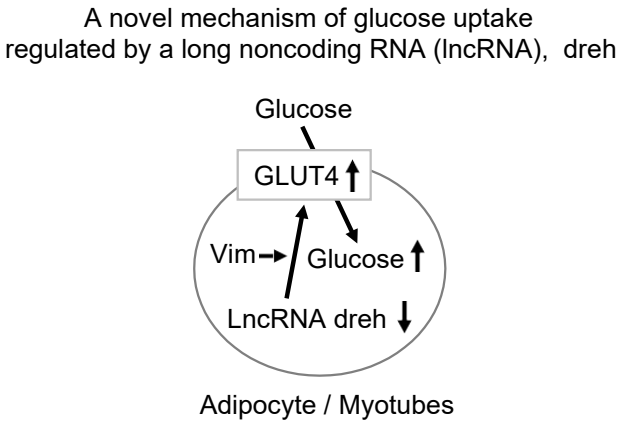


Fig. 2 :



Published papers (selected)

1. Takahashi N, Kimura AP, Yoshizaki T, Ohmura K. Imeglimin modulates mitochondria biology and facilitates mitokine secretion in 3T3-L1 adipocytes. ***Life Sciences*** 349:122735, 2024. (IF=6.1) (**Fig. 1**: Discovered and reported that Imeglimin, a novel diabetes drug, acts on adipocytes)

2. Takahashi N, Kimura AP, Ohmura K, Naito S, Yoshida M, Ieko M. Knockdown of long noncoding RNA dreh facilitates cell surface GLUT4 expression and glucose transport through the involvement of vimentin in 3T3-L1 adipocytes. ***Gene*** 735:144404, 2020. (IF=3.5) (**Fig. 2**: The discovery of long non-coding RNA Dreh involved in glucose uptake in adipocytes)

3. Takahashi N, Kimura AP, Otsuka K, Ohmura K, Naito S, Yoshida M, Ieko M. Dreh, a long noncoding RNA repressed by metformin, regulates glucose transport in C2C12 skeletal muscle cells. ***Life Sciences*** 236:116909, 2019. (IF=6.1) (**Fig. 2**: The discovery of long non-coding RNA Dreh involved in metformin-induced glucose uptake in skeletal muscle cells)

4. Takahashi N, Kimura AP, Naito S, Yoshida M, Kumano O, Suzuki T, Itaya S, Moriya M, Tsuji M, Ieko M. Sarcolipin expression is repressed by endoplasmic reticulum stress in C2C12 myotubes. ***Journal of Physiology and Biochemistry*** 73:531-538, 2017. (IF=3.4)

5. Takahashi N, Yoshizaki T, Hiranaka N, Kumano O, Suzuki T, Akanuma M, Yui T, Kanazawa K, Yoshida M, Naito S, Fujiya M, Kohgo Y, Ieko M. The production of coagulation factor VII by adipocytes is enhanced by tumor necrosis factor-α or isoproterenol. ***International Journal of Obesity*** 39:747-754, 2015. (IF=4.9)

6. Takahashi N, Yoshizaki T, Hiranaka N, Suzuki T, Yui T, Akanuma M, Kanazawa K, Yoshida M, Naito S, Fujiya M, Kohgo Y, Ieko M. Endoplasmic reticulum stress suppresses lipin-1 expression in 3T3-L1 adipocytes. ***Biochemical and Biophysical Research Communications*** 431:25-30, 2013. (IF=3.1)

7. Takahashi N, Yoshizaki T, Hiranaka N, Suzuki T, Yui T, Akanuma M, Oka K, Kanazawa K, Yoshida M, Naito S, Fujiya M, Kohgo Y, Ieko M. Suppression of lipin-1 expression increases monocyte chemoattractant protein-1 expression in 3T3-L1 adipocytes. ***Biochemical and Biophysical Research Communications*** 415:200-205, 2011. (IF=3.1)