CURRICULUM VITAE

NAME: Tadashi Yamamoto

POSITION: Professor, Division of Oncology, Department of Cancer Biology, The Institute of Medical Science, The University of Tokyo, 4-6-1 Shirokanedai, Minato-ku, Tokyo 108-8639, JAPAN

DATE AND PLACE OF BIRTH: August 26, 1947, Fukui, Japan

HOME ADDRESS: 203, 3-11-17 Ebisu-minami, Shibuya-ku
Tokyo 150-0022, JAPAN

EDUCATION AND EXPERIENCE:

March, 1966	Graduated from Katsuyama Senior High School
April, 1966	Undergraduate, Osaka University
March, 1972	Bachelor of Science, Osaka University
March, 1974	Master of Science, Osaka University
March, 1977	Ph. D., Osaka University
April, 1977-August, 1977	Postdoctoral Fellow, Research Institute for Microbial Diseases, Osaka University
September, 1977-February, 1980	Visiting Fellow, Laboratory of Molecular Biology National Cancer Institute, NIH
February, 1980-January, 1981	Visiting Associate, Laboratory of Molecular Biology National Cancer Institute, NIH
January, 1981-April, 1986	Assistant Professor, Department of Oncology The Institute of Medical Science, The University of Tokyo
April, 1986-November, 1991	Associate Professor, Department of Oncology The Institute of Medical Science, The University of Tokyo
November, 1991-Present	Professor, Department of Cancer Biology (Formerly, Oncology) The Institute of Medical Science, The University of Tokyo
April, 2003-pressent	Dean, Institute of Medical Science, The University of Tokyo

AWARDS:
1984, Incentive Award from the Japanese Cancer Association
1987, Prize from Princess Takamatsu Cancer Research Fund
1990, Asahi Prize
2004-, NIH Fogarty Scholar

MEMBERSHIP:
Japanese Cancer Association (council member, Since 1986)
Japanese Society for Molecular Biology (council member, Since 1999)
American Society of Cell Biology
Award Assembly Member, the General Motors Cancer Research Fund (2000-2003)

EDITORIAL ACTIVITIES;
Editorial Board, Molecular Biology of the Cell
Editorial Board, Cell Growth and Differentiation
Editorial Board, Genes to Cells
Editorial Board, Journal of Experimental & Clinical Cancer Research
Editorial Board, Japanese Journal of Clinical Oncology

The tumor suppressor protein Tob functions as a transcriptional coregulator

Tadashi Yamamoto, Takahisa Nakamura, Toru Suzuki, Yutaka Yoshida

Division of Oncology, The Institute of Medical Science, University of Tokyo, Tokyo, 108-8639, Japan

Accumulating evidence shows that genes involved in negative control of cell growth could function as tumor suppressors. *tob*, together with *tob2*, *ana*, *pc3b*, *btg1*, and *btg2*, belongs to a family of antiproliferative genes. An exogenous expression of Tob suppresses growth of NIH3T3 cells by inhibiting G1 progression of the cell cycle. In addition, mice lacking the *tob* gene are highly prone to spontaneous formation of tumors in various tissues, supporting an idea that *tob* functions as a tumor suppressor. However, underlying mechanisms of the growth inhibition by *tob* have remained to be established. To help unravel how Tob exerts its antiproliferative function, we examined transcripts that were affected by exogenous Tob expression (gain of function) and by depletion of Tob expression (loss of function). DNA microarray analyses of about 500 cancer-related genes demonstrated that Tob affected expression of several genes, including the *p21waf1/cip1* gene and cyclin D1 gene. We further showed that overexpressed Tob activated the *p21waf1/cip1* promoter and suppressed the *cyclin D1* promoter via its interaction with histone acetylase (p300/CBP) and histone deacetylase, respectively. Furthermore, proteomic analysis revealed that Tob is a component of the CCR4-NOT transcription complex, again suggesting that Tob functions as a transcriptional regulator. Recently, we founds that one of the CCR4-NOT complex, Caf1, binds the AF-1 domain of RXRb and, together with Tob, facilitates RXRb-mediated transcription. Biological importance of Tob-Caf11 interaction will be discussed.

References

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2. Suzuki T, Tsuzuku JK, Ajima R, Nakamura T, Yoshida Y, and Yamamoto T, Phosphorylation of three regulatory serines of Tob by Erk1 and Erk2 is required for Ras-mediated cell proliferation and transformation. *Genes & Dev* 16: 1357-1370, 2002
3. Yoshida Y, Nakamura T, Komoda M, Sato H, Tsuzuku JK, Miyasaka T, Yoshida EH, Umemori H, Kunisaki RK, Tani K, Ishii S, Mori S, Suganuma M, Noda T, and Yamamoto T, Mice lacking a transcriptional corepressor Tob are predisposed to cancer, *Genes & Dev* 17: 1201-1206, 2003