

## CURRICULUM VITAE

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### Education:

1991-1993: Department of Applied Chemistry, Okayama University Graduate  
School of Engineering  
1995-2000: First Department of Pathology, Hiroshima University School of  
Medicine

### Qualification:

2000: Ph.D. from Hiroshima University, Japan

### Employment and Experience:

1993-1995: Staff Scientist, DAICEL Chemical Industries, Ltd., Japan  
2000-2003: Postdoctoral fellow, Department of Geriatric Research, National  
Institute for Longevity Sciences, Japan  
2003-2005: Assistant Professor, Department of Molecular Biology, Okayama  
University Graduate School of Medicine and Dentistry, Japan  
2005-2015: Assistant Professor and Associate Professor, Cancer Research  
Institute, Kanazawa University, Japan  
2015-Present: Associate Professor, Department of Stem Cell Biology, Research  
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**Research Interest:**

Although the discovery of the tyrosine kinase inhibitor (TKI) has significantly improved the prognosis of chronic myeloid leukemia (CML) patients, a complete cure is not possible due to the existence of a rare population of CML stem cells known to be resistant to TKI therapy. We previously reported that TGF- $\beta$ -FOXO axis is essential for the TKI-resistance of CML stem cells *in vivo* (Naka *et al.* **Nature** 2010). In collaboration with Prof. Seong-Jin Kim, Seoul National University, we also found that CML stem cells take up dipeptide species as a nutrient to maintain CML stem cell survival *in vivo* (Naka *et al.* **Nat Commn** 2015). Our results will hopefully be contribute to develop novel therapeutics that can specifically suppress in CML stem cells, and thereby provide a novel avenue for curative patient therapy.

**Publications:**

1. Naka K. (co-corresponding author), Ishihara K., Jomen Y., Jin C.H., Kim D.-H., Gu Y.-K., Jeong E.-S., Li S., Krause D.S., Kim D.-W, Bae E.J., Takihara Y., Hirao A., Oshima H., Oshima M., Ooshima A., Sheen Y.Y., Kim S.J. and Kim D.K. (2016) Novel oral transforming growth factor- $\beta$  signaling inhibitor EW-7197 eradicates CML-initiating cells. **Cancer Sci.** 107(2): 140-148.
2. Naka K. (co-corresponding author), Jomen Y., Ishihara K., Kim J., Ishimoto T., Bae E., Mohny R.P., Stirdivant S.M., Oshima H., Oshima M., Kim D.-W., Nakauchi H., Takihara Y., Kato Y., Ooshima A., and Kim S.J. (2015) Dipeptide species regulate p38-MAPK-Smad3 signalling to maintain chronic myelogenous leukaemia stem cells. **Nat. Commun.** 6:8039 doi: 10.1038/ncomms9039.
3. Naka K. (co-corresponding author), Hoshii T., Muraguchi T., Tadokoro Y., Ooshio T., Kondo Y., Nakao S., Motoyama N. and Hirao A. (2010) TGF- $\beta$ -FOXO signalling maintains leukaemia-initiating cells in chronic myeloid leukaemia. **Nature** 463(7281): 676-680.