



#### PRESS RELEASE

# The Japanese Cancer Association and Debiopharm Group Announce Winners of the 2018 JCA-Mauvernay Award

The 'JCA-Mauvernay Award 2018' is awarded to Doctors Tatsushi Igaki and Takahiro Maeda

Lausanne, Switzerland – September 28, 2018 –Debiopharm Group<sup>™</sup> (Debiopharm – www.debiopharm.com), a Swiss-based global biopharmaceutical company, will be presenting the 'JCA-Mauvernay Award' on September 29 to Doctors Tatsushi Igaki from the Laboratory of Genetics, Graduate School of Biostudies of Kyoto University for his basic research on the *Mechanisms of cell-cell competition and cooperation in tumorigenesis* and Takahiro Maeda from the Center for Cellular and Molecular Medicine of Kyushu University Hospital for his translational work on the *Identification of novel targets for leukemia therapy using the CRISPR/Cas9 gene-editing tool.* 

Doctors Igaki and Maeda will receive their Awards during the General Assembly of the 77<sup>th</sup> Annual Meeting of the Japanese Cancer Association (JCA) in Osaka which will focus this year on the theme: '*Diving Deeper into Cancer Research*'. Dr. Hitoshi Nakagama, President of the JCA and Thierry Mauvernay, President & Delegate of the Board of Debiopharm Group, will present the trophies to both scientists.

**Dr. Igaki** has been studying cell-cell communication that regulate tumor growth and progression for more than ten years. Using model organisms such as the fruit fly, he has developed the concept of "tumor-suppressive cell competition" which describes a mechanism enabling oncogenic mutant cells to be actively eliminated by surrounding normal cells. Dr. Igaki has also discovered that cell-cell cooperation can drive the progression of a tumor within its microenvironment and has dissected the molecular pathways underlying this process. Given the high degree of conservation of the identified cell-cell communication processes between flies and humans, some of the molecular players could become pharmacological targets of new therapeutic approaches.

Acute myeloid leukemia (AML) has a long-term rate survival less than 30%. **Dr. Maeda** has been working on identifying novel targets for AML therapy using the CRISPR/Cas9 gene-editing tool. He performed genome-wide CRISPR-Cas9 dropout screens utilizing AML lines exhibiting a normal karyotype and harboring functionally-normal *Trp53*. He identified DCPS as a drug target for AML therapy and demonstrated that RG3039 (a DCPS inhibitor) exhibited anti-leukemia activity and identified pre-mRNA metabolic pathway required for AML survival.

"Since 2005, our objective has been to reward groundbreaking achievement among Japanese scientists in oncology. This year's winners, Doctors Igaki and Maeda, have proved themselves to be at the forefront of oncology research. We congratulate them for their commitment and hope their work will lead to effective treatments in the years to come", said Thierry Mauvernay, President & Delegate of the Board of Debiopharm Group.

### About the JCA-Mauvernay Award

Since 2005, the Japanese Cancer Association (JCA) and Debiopharm Group have co-organized the 'JCA-Mauvernay Award'. This prize illustrates the curiosity that drives researchers as well as the scientific cooperation between Japan and Switzerland. It aims at recognizing outstanding achievements in the field of oncology amongst Japanese researchers, in both the fundamental and the clinical aspects. The award has a total value of CHF 25'000.

## About Debiopharm Group

Debiopharm Group<sup>™</sup> is a Swiss-headquartered global biopharmaceutical group including five companies active in the life science areas of drug development, GMP manufacturing of proprietary drugs, diagnostic tools and investment management. Debiopharm focuses on developing prescription drugs that target unmet medical needs. The group in-licenses and develops promising drug candidates. The products are commercialized by pharmaceutical out-licensing partners to give access to the largest number of patients worldwide.

For more information, please see www.debiopharm.com

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