CDB SEMINAR

Speaker: **Hiromi Hirata**

<Depertment of Molecular, Cellular and Developmental Biology,
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Title: "Genetic and Electrophysiological Analysis of Zebrafish Early Behaviors"

Date: Monday, March 14
Time: 16:00 P.M.～17:00 P.M.
Place: 1F Auditorium of Building C, CDB

Summary:

When wild type zebrafish embryos are touched at 24 hours post-fertilization (hpf), they typically perform 2 rapid alternating coils of the tail. In contrast, accordion (acc) mutants fail to coil their tails normally but contract the bilateral trunk muscles simultaneously to shorten the trunk resulting in a pronounced dorsal bend. Electrophysiological recordings from muscles showed that the output from the central nervous system is normal in mutants suggesting a defect in muscles. In fact, relaxation in acc muscle is significantly slower than normal. In vivo imaging of muscle calcium transients revealed that cytosolic Ca2+ decay is significantly slower in acc muscle. Thus, it appears that the mutant behavior is caused by a muscle relaxation defect due to the impairment of Ca2+ reuptake. Indeed, acc mutants carry a mutation in ATP2A1 gene that encodes the sarco(endo)plasmic reticulum Ca2+-ATPase 1 (SERCA1), a Ca2+ pump found in the muscle sarcoplasmic reticulum (SR) that is responsible for pumping Ca2+ from the cytosol back to the SR. Since SERCA1 mutations in humans lead to Brody disease, an exercise-induced muscle relaxation disorder, zebrafish accordion mutants could be a useful animal model for Brody disease.

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