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ALL ARE WELCOME

24 Apr 2024 (Wed), 3pm The Auditorium (Level 1)

Hosted by: Dr PEK Jun Wei

Understanding the role of translation dysregulation in reproductive and intellectual disorders



Dr. Greenblatt's established his lab at the University of British Columbia in 2021. Dr. Greenblatt received his PhD in Biophysics studying cellular protein quality control in the lab of Ron Kopito at Stanford University. His postdoctoral training in reproductive biology and genetics was performed in Allan Spradling's lab at the Carnegie Institution.

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Defects are in oocyte development are a major cause of human miscarriage, which is estimated to occur with a frequency of 30-70%. Upon the cessation of growth, oocytes shut down transcription and survive using ongoing gene expression from stored mRNAs. Neurons similarly utilize gene expression from mRNAs stored at synapses. We use the model organism Drosophila to (1) study the factors that limit natural oocyte survival and (2) determine mechanisms underlying translational control from stored mRNAs, which remain poorly understood. Our findings implicate instability in translation, and in particular the reduced activity of stored ribosomes, as a major driver of oocyte inviability. We also found that selective translation is mediated by a complex of RNA binding proteins including FMR1, the most commonly mutated gene underlying heritable intellectual disability and premature ovarian failure, mechanisms suggesting that underlying translational dysfunction in Drosophila oocytes underlie aspects of human reproductive and intellectual disorders.

Recent Publications:

- 1. K. Flanagan, A. Baradaran-Heravi, Q. Yin, K. Dao Duc, A.C. Spradling, and E.J. Greenblatt. FMRP- dependent production of large dosage-sensitive proteins is highly conserved. Genetics iyac094 (2022).
- 2. E.J. Greenblatt, R. Obniski, C. Mical, and A.C. Spradling. Prolonged ovarian storage of mature Drosophila oocytes dramatically increases meiotic instability. eLife 8:e49455 (2019).
- 3. E.J. Greenblatt and A.C. Spradling. Fragile X mental retardation 1 gene enhances the translation of large autism-related proteins. Science 361:709-712 (2018).