Abstract:
Cancer is a disease of single cells behaving badly, with mutations to certain proteins causing uncontrolled cellular proliferation. This has led to the development of therapies targeting those proteins, such as vemurafenib for melanoma. Yet while most cells will respond to drug, some do not, ultimately repopulating the tumors and causing relapse. This process is often thought to be Darwinian with genetic origins as well. Here, we show that the acquisition of resistance may in fact have non-genetic origins, and we decompose the resistance process into an early, transient priming phase before the addition of drug, following by a later reprogramming phase consisting of a stepwise epigenetic progression. We discuss some implications for cancer treatment as well as cell fate plasticity.

Bio:
Arjun Raj went to UC Berkeley, where he majored in math and physics, earned his PhD in math from the Courant Institute at NYU, and did his postdoctoral training at MIT before joining the faculty in Penn Bioengineering in 2010, where he is currently an assistant professor. His research focus is on the developed experimental techniques for making highly quantitative measurements in single cells and models for linking those measurements to cellular function. His ultimate goal is to achieve a quantitative understanding of the molecular underpinnings of cellular behavior.

Date : 10 March 2017 (Friday)
Time : 4:00 p.m.
Venue : Lecture Theatre C
The Hong Kong University of Science & Technology
Clear Water Bay, Kowloon

(Host faculty: Dr. Angela Wu)

All are Welcome!!