Lawrence J. Ellison Institute for Transformative Medicine of USC

# SCIENCE CLUB

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## "Signaling by the Oncogenic Fusion Protein BCR-FGFR1, a Client of the Molecular Chaperone HSP90"

**Friday, June 21, 2019**
**12:00 P.M. - 1:00 P.M.**
Q & A to follow with lunch provided

## Harkness Auditorium

HSC - Clinical Sciences Building, **CSC 250**
2250 Alcazar Street, 2nd Floor, Los Angeles, CA

## Abstract:

Fibroblast Growth Factor Receptors (FGFRs) are part of the Receptor Tyrosine Kinase (RTK) family and are essential in the activation of various downstream signaling pathways and are necessary for cell differentiation and proliferation. However, mutation and translocation of FGFRs leads to aberrant activation of signaling, which often results in cancer. The t(8;22) (p11;q11) chromosomal translocation, results in the BCR-FGFR1 fusion protein. Patients who harbor this translocation are usually diagnosed with 8p11 myeloproliferative syndrome (EMS), which can progress to atypical Chronic Myeloid Leukemia (aCML), or Acute Myeloid Leukemia (AML) if left untreated. Unlike BCR-ABL, BCR-FGFR1 is rare, poorly characterized and not well understood, resulting in few therapies and clinical advancements for patients positive for this oncogenic fusion. This work seeks to analyze how the BCR-FGFR1 fusion leads to cancer, both through its biochemical and biological characterization. Additionally, although tyrosine kinase inhibitor therapies have traditionally been used to treat patients with aberrantly activated tyrosine kinases, the use of TKIs often results in drug resistance. Thus, it is crucial to establish additional therapeutic strategies in treating hematological cancers. Here we aim to investigate the regulation of BCR-FGFR1 in the cell to establish novel therapeutic targets for patients who are positive for this fusion protein.