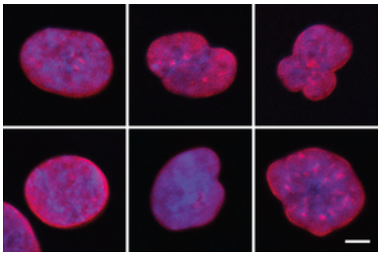
# ASRC - City College of New York

## Seminar in Biochemistry, Biophysics & Biodesign



**SEMINAR LOCATION:**
ASRC Main Auditorium
85 St. Nicholas Terrace

- *Current Cleared4 Pass or CCNY ID with gold V-22 sticker required for entrance*
- *Masks are required*
- *Maximum occupancy: 30*

**THE SEMINAR WILL ALSO BE AVAILABLE ON ZOOM:**

Click here for Zoom link

**HOST:**
Kevin Gardner
kgardner@gc.cuny.edu

**FOR MORE INFORMATION, CONTACT:**
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The Biochemistry Seminar series is supported in part by the CUNY Institute for Macromolecular Assemblies; the CCNY Science Division, and the Advanced Science Research Center at the Graduate Center of the City University of New York.

ADVANCED SCIENCE RESEARCH CENTER
THE GRADUATE CENTER
CITY UNIVERSITY OF NEW YORK

## Wednesday, November 10, 2021

**12:00 – 1:00 PM**

CUNY Advanced Science Research Center, Main Auditorium
85 St. Nicholas Terrace, New York, NY

# Eduardo Torres

Assistant Professor, Dept. of Molecular, Cell & Cancer Biology
University of Massachusetts Chan Medical School

## Aneuploidy disrupts cellular physiology and metabolism

ABSTRACT An abnormal number of chromosomes or aneuploidy accounts for most spontaneous abortions as missegregation of a single chromosome during development is often lethal. Humans with trisomies for chromosomes 13 or 18, which cause Patau and Edwards syndromes, respectively, are born with severe developmental defects and die soon after birth. Only individuals with trisomy 21, which causes Down syndrome, can live to adulthood but show cognitive disabilities, increased risk for leukemias, autoimmune disorders, and clinical symptoms associated with premature aging. Notably, the incidence of aneuploidy increases with age in both somatic and germline tissues in apparently healthy individuals. The mechanisms by which aneuploidy affects cellular function to cause Down syndrome or promote aging are not well understood. Our studies revealed that aneuploidy disrupts the integrity and morphology of the nuclear membrane. Because mutations that affect nuclear morphology cause premature aging, we hypothesize that the aneuploidy effects on the nucleus drive phenotypic anomalies associated with premature aging in Down syndrome. In addition, to characterize aneuploidy-driven phenotypes in human cells, we performed global transcriptome, proteome, and phenotypic analyses of primary fibroblasts from individuals with Patau (trisomy 13), Edwards (trisomy 18), or Down syndromes. On average, mRNA and protein levels were increased by 1.5-fold in all trisomies, with a subset of proteins enriched for subunits of macromolecular complexes showing signs of post-transcriptional regulation. Lastly, we show that several aneuploidy-associated phenotypes are present in trisomy 21 cells, including lower viability and increased dependency on serine-driven lipid biosynthesis. Our studies establish a critical role of aneuploidy, independent of triplicated gene identity, in driving cellular defects associated with Down syndrome.