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LVI SAIB Meeting – XV SAMIGE Meeting

SAIB-SAMIGE Joint Meeting 2020 – *Online*

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LVI Annual Meeting Argentine Society for Biochemistry and Molecular Biology (SAIB)

XV Annual Meeting Argentinean Society for General Microbiology (SAMIGE)

SAIB-SAMIGE – Online Joint Meeting 2020

ORAL COMMUNICATIONS

MONDAY NOVEMBER 2, 2020

14:00-16:00 CELL BIOLOGY I

Chairpersons: Cecilia Álvarez- Javier Valdez Taubas

14:00-14:13
CB-C01-017
FROM CARTOONS TO QUANTITATIVE MODELS IN GOLGI TRANSPORT
Nieto F, Quirós N, Mayorga LS

14:15-14:28
CB-C02-054
CSP DRIVES *TRAN*S SNARE ASSEMBLY DURING ACROSOMAL EXOCYTOSIS
Flores Montero K, Berberián MV, Ruete MC

14:30-14:43
CB-C03-208
KCTD15, A NOVEL PROTEIN INVOLVED IN CELL TRAFFICKING
Zarelli VEP, Lopez de Armentia MM, Colombo MI.

14:45-14:58
CB-C04-239
INTRACELLULAR TRAFFICKING OF INFLUENZA VIRUS M1 PROTEIN AT LATE STAGES OF THE INFECTIOUS CYCLE
Drake Figueredo A, Morellatto Ruggieri L, Magadán JG

15:00-15:13
CB-C05-237
THE HIV-1 ACCESSORY PROTEIN Vpu TARGETS HOST SLC1A5 (ASCT2) AMINO ACID TRANSPORTER
Morellatto Ruggieri L, Drake Figueredo A, Magadán JG

15:15-15:28
CB-C06-218
INTERACTION BETWEEN PROTEIN TYROSINE PHOSPHATASE 1B (PTP1B) AND EGFR AT ER-PM JUNCTIONS
Perez Collado ME, Arregui CO

15:30-15:43
NS-C01-202
INTERNEURONAL EXCHANGE AND FUNCTIONAL INTEGRATION OF SYNAPTOBREVIN VIA EXTRACELLULAR VESICLES
Vilcaes AA, Chanaday NL, Kavalali ET

15:45-15:58
CB-C07-004
BIOLOGICAL RELEVANCE OF 14-3-3 ACETYLATION DURING OSTEOGENIC LINEAGE DETERMINATION
Frontini-López YR, Uhart M, Bustos DM

CB-C03-208
KCTD15, A NOVEL PROTEIN INVOLVED IN CELL TRAFFICKING

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Kctd15 belongs to a family of proteins, the Potassium Channel Tetramerization Domain (KCTD) proteins because it contains a BTB domain at the N terminal that acts as a scaffold interface. Kctd15 has been demonstrated to antagonize neural crest formation by affecting Wnt signaling and AP-2 transcription factor function during embryonic development in zebrafish. However, the function of Kctd proteins is still under characterization. Preliminary results from our lab indicate that overexpression of Kctd15 induced the formation of giant vacuoles in mammalian cells. These vacuoles do not acquire Rab5, Rab7, or CD63 or dextran internalized by endocytosis, suggesting that these compartments do not belong to the early endocytic pathway. Furthermore, they are not acidic/lysosomal structures since the vacuoles were not marked by DQ-BSA or LysoTraker. We also analyzed trans-Golgi (TGN) markers such as GFP-Rab29, GFPRab32, GFP-Rab34, and GFP-Rab38. We observed that these proteins were recruited to the membrane of the vacuoles, colocalizing totally or partially with KCTD15. We also studied the production of vacuoles by overexpressing mutated versions of KCTD15 for the SUMOylation domain. We used the K278R mutant where lysine was mutated to arginine at position 278, and a truncated version of KCTD15 (K234), which lacks 49 amino acids at the C-terminal, totally losing the target domain for SUMOylation. Interestingly, we observed that the mutant K278R produced vacuoles as large, or even larger, than its WT counterpart, whereas the truncated form K234 did not generate these vacuoles. This would indicate that the domain necessary for the formation of these structures seems to reside in the C-terminal region within those 49 amino acids. In order to further characterize the nature of the generated vacuoles, we used the specific marker, TGN38, one of the few known resident integral membrane proteins of the TGN. Since TGN38 moves through both the endocytic and exocytic pathways, it is useful for the identification of post-Golgi trafficking motifs. Thus, we overexpressed KCTD15 WT or the truncated form K234, tagged with FOS, observing that TGN38 colocalized with KCTD15 WT at the vacuoles' membrane but not with K234 where the Golgi signal for TGN38 was still observed, indicating that the vacuoles of KCTD15 could be generated from the most posterior region of the trans-Golgi. By mass spectrometry, Kctd15 was able to pull down Vps26 and Vps35, components of the retromer complex which participate in recycling components from endosomes to the trans-Golgi network (TGN). Our results show that Vps26 and KCTD15 colocalize at the vacuole while Vps35 and Vps29 (another retromer component) seem to be inside. Taken together, our results suggest that Kctd15 is likely involved in the retromer-TGN trafficking and that the c-terminal domain of the protein seems to be critical for this process. Further studies are necessary to determine the role of kctd15 in this transport.

CB-C04-239
INTRACELLULAR TRAFFICKING OF INFLUENZA VIRUS M1 PROTEIN AT LATE STAGES OF THE INFECTIOUS CYCLE

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Influenza A –the main responsible for seasonal "flu"– is an RNA virus containing a single-stranded and segmented RNA of negative polarity and belongs to the *Orthomyxoviridae* family. In humans, influenza A mainly affects the upper respiratory tract causing considerable morbidity and mortality with local epidemic outbreaks and occasionally pandemic worldwide spread. The World Health Organization (WHO) estimates that seasonal circulating influenza results in about 3–5 million cases of severe illness and about 250,000 to 500,000 deaths. The replication cycle of influenza A fully depends on the host cell metabolic pathways. Thus, the translation of the viral mRNAs is divided between cytosolic (PB1, PB2, PA, NP, NS1, NS2, and M1) and endoplasmic reticulum (ER)-associated ribosomes (HA, NA and M2). It is clear that M1, the main viral capsid protein, plays a critical role during the influenza infectious cycle by controlling the entry, replication, and nuclear export of a complete set of viral genomes and proteins (vRNPs). However, little is known about the role of M1 during vRNPs trafficking *in route* to host the plasma membrane where the viral particles are being assembled. Focusing on the late stages of influenza A infectious cycle, our results indicate that M1 associates with acidic compartments at the last stages, mainly colocalizing with typical late endosomal/lysosomal markers such as Rab7a, Rab9a, CD63, LAMP1, and the LysoTracker probe. Interestingly, bafilomycin A1, an inhibitor of the vesicular proton pump, induces specific re-location of viral M1 from late membranous compartments to the cytosol, suggesting that a functional organelle is required for M1 proper cellular targeting. Therefore, we speculate that late endosomes/lysosomes might act as pre-assembling platforms where not only M1 but other structural influenza proteins such as HA, NA or M2 and vRNPs transiently converge and eventually interact one with another in order to form maturing intermediate viral particles just before to reach the host cell surface.