– Special Seminar –

THIS SEMINAR WILL BE AVAILABLE VIA ZOOM ONLY:
9:00 AM PDT
12:00 PM EDT
5:00 PM BST
7:00 PM IDT

Click here to register
Or go to:
https://tinyurl.com/Varda-S-B

HOST:
Amédée des Georges
ajd9478@nyu.edu

Organized by Manfred Philipp. Sponsored by the CUNY Academy for the Humanities and Sciences, the CUNY Graduate Center's Biochemistry Doctoral Program, the CUNY Graduate Center's Interdisciplinary Concentration in Cognitive Science, the CUNY Advanced Science Research Center, and Americans for Ben-Gurion University.

THE CITY UNIVERSITY OF NEW YORK

AMERICANS FOR BEN-GURION UNIVERSITY
Partners in the Remarkable

Wednesday, April 3

Seminar by Zoom 12:00 - 1:00 PM

Varda Shosan-Barmatz

National Institute of Biotechnology in the Negev
Department of Life Sciences
Ben-Gurion University of the Negev
Beer Sheva, Israel

Mitochondrial gatekeeper VDAC1 overexpression and oligomerization lies at the intersection of programmed cell death, inflammation and disease

ABSTRACT Recently, we demonstrated that induction of apoptosis leads to VDAC1 overexpression and oligomerization regardless of the cell type, apoptosis inducer used, that all affect the mitochondria, yet acting via different mechanisms. Accordingly, we proposed a new concept for apoptosis induction by which apoptosis inducers, stress, or diseases state, induce VDAC1 overexpression, and thereby shift the equilibrium between monomeric and oligomeric states. This promotes the formation of a large channel within the VDAC1 homo-oligomer, which then acts as a conduit for pro-apoptotic protein release and subsequent apoptosis. Oligomeric VDAC1 is also at the nexus of mitochondrial DNA (mtDNA) release and is implicated in impairing the innate immune system because the mtDNA fragments released into the cytosol trigger type-I interferon signaling and inflammation. Moreover, we have demonstrated that overexpression of VDAC1 is a common threat in diabetes, and in neurodegenerative, cardiac, and autoimmune diseases. In addition, others have demonstrated associations between VDAC1 overexpression and oligomerization, and acute liver injury, rheumatoid arthritis, spinal cord injury, and COVID-19. Thus, inhibiting VDAC1 overexpression and/or oligomerization represents an effective strategy to treat these diseases. With the perception of VDAC1 as an innovative target for the control of dysregulated cell metabolism, inflammation, and programed cell death associated with various diseases, we have developed the new VDAC1-interacting molecules, VBIT-4 and VBIT-12. These molecules prevent VDAC1 oligomerization, cell death, mitochondrial dysfunction, and inflammation and abolish the pathophysiology of various diseases as demonstrated in mouse models for type-2-diabetes, lupus, colitis, Alzheimer's disease, acute liver injury, spinal cord injury, and COVID-19. Our findings implicate VDAC1 as the link between mitochondrial dysfunction and a wide range of diseases, and place it at the crossroads between metabolism, cell survival, cell death, and inflammation.