40 Years of the Undergraduate Program
DEPARTMENT OF BIOLOGY · UNIVERSITY OF CRETE

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
ΠΑΝΕΠΙΣΤΗΜΙΟ ΚΡΗΤΗΣ

ΣΧΟΛΗ ΘΕΤΙΚΩΝ & ΤΕΧΝΟΛΟΓΙΚΩΝ ΕΠΙΣΤΗΜΩΝ
ΤΜΗΜΑ ΒΙΟΛΟΓΙΑΣ

Σας προσκαλούμε την **Τετάρτη 9 Δεκεμβρίου 2026 και ώρα 12.00 μ.μ. έως τις 13.00 μ.μ. στο Αμφιθέατρο Α,** στην ομιλία του Αναπληρωτή Καθηγητή, Τμήμα Φυσιολογίας της Ιατρικής Σχολής του Εθνικού και Καποδιστριακού Πανεπιστημίου Αθηνών,

κ. Κωνσταντίνου Παληκαρά.

Το θέμα της ομιλίας του είναι :
"Neuronal mitophagy in ageing and age-related neurodegeneration"

Neuronal survival during aging critically depends on the dynamic interplay between mitochondrial function and selective autophagy pathways. Disruption of this mitochondria–autophagy crosstalk is increasingly recognized as a central event in Alzheimer's disease (AD) and related tauopathies.
Using genetically tractable Caenorhabditis elegans models expressing human Aβ or tau, together with mouse models, human iPSC-derived neurons, and patient datasets, we investigate how mitochondrial dysfunction and mitophagy failure mediate neuronal vulnerability. We find that mitochondrial fragmentation and deregulation of calcium homeostasis precede overt tau aggregation and functional decline, identifying early mitochondrial impairment as a pathogenic trigger rather than a secondary consequence. Across AD systems, defective mitophagy emerges as a conserved hallmark, leading to accumulation of dysfunctional mitochondria, impaired synaptic activity, and enhanced proteotoxic stress.
Importantly, pharmacological and genetic upregulation of mitophagy mitigates amyloid and tau pathology, preserves neuronal integrity, and restores cognitive function in disease models. These findings establish a causal link between selective mitochondrial clearance and neuronal resilience.
Our findings position mitochondrial–autophagy crosstalk as a mechanistic hub integrating metabolic stress, calcium signaling, and proteostasis in neuronal aging, highlighting selective mitophagy pathways as promising therapeutic targets for age-associated neurodegeneration.

Ο Πρόεδρος του Τμήματος

Καθηγητής, Κρίτων Καλαντίδης