令和８年度　第８回　大学院セミナー

令和８年　５月　13 日

分　野　名 Area of Research (責任者名)(内線)	幹細胞生物学　分野 責任者名(　李　桃生)　　内線(　7099　)
演　　題 Title	核小体 ATR シグナル伝達軸は、ストレス感知とオートファジー誘導を連動させる（Nucleolar ATR signaling axis couples stress sensing to autophagy induction）
講　師　等 Presenter	原爆後障害医療研究所　原研幹細胞　准教授 川端　剛　先生（Tsuyoshi Kawabata）
概要 Abstract	Autophagy maintains cellular homeostasis through lysosomal degradation of cytoplasmic components, yet the signaling mechanisms coupling nuclear stress to autophagosome biogenesis remain unclear. Here, we demonstrate that ATR kinase, canonically known as a replication stress sensor, regulates starvation-induced canonical autophagy. ATR inhibition and genetic reduction suppressed autophagic flux and autophagosome formation, and patient-derived Seckel syndrome cells showed impaired autophagy under both basal and starvation conditions. Phosphoproteomic screening of membrane-enriched fractions combined with an siRNA functional screen identified a nucleolar scaffold protein as an ATR substrate required for autophagosome biogenesis. Its knockdown impaired LC3 puncta formation, ATG9 vesicle recruitment, and WIPI2 induction upon starvation. Mechanistically, loss of this nucleolar factor caused hyperphosphorylation of the autophagy transcription factor TFEB and reduced its nuclear translocation in an ATR-dependent manner, and a fraction of TFEB co-localizes with this protein at nucleoli. These findings define an ATR–nucleolus–TFEB signaling axis that connects nucleolar stress sensing to activation of autophagy, revealing a non-canonical role for ATR as an integrator of metabolic stress and cellular quality control.
開 催 日 時 Date and Time	令和８年　５月　27 日（　水　） 17：30　～　19：00
開 催 方 法 Online/Face to face	オンライン：TEAMS
備　　考 Notes	受講を希望者は、以下のメールに連絡ください。 （E-mail: litaoshe@nagasaki-u.ac.jp） If you would like to participate in this seminar, please contact: litaoshe@nagasaki-u.ac.jp.

☐先端医療科学特論（基礎編）
☐先端新興感染症病態制御学特論
☐日本語（Japanese）
☐対面（Face to face）
☐先端医療科学特論（臨床編）
☑先端放射線医療科学特論
☑英語（English）
☑オンライン(Online)