Seminar at Graduate School of Life Sciences

Light and Dark sides of aPKC

Speaker

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Summary

Cell proliferation is controlled not only by growth factors, but also by polarization. Disrupted polarity is a hallmark of excessive cell expansion, however a molecular mechanism remains elusive. Polarity protein atypical protein kinase C lambda/iota (aPKC λ), is associated with proliferation and is an oncogene. In endothelia, suppression of aPKC λ impairs proliferation despite hyper-activated vascular endothelial growth factor (VEGF) signaling. In this seminar, I will show the molecular mechanisms to explain this discrepancy. aPKC λ phosphorylates forkhead box O1 (FoxO1) transcription factor, a gatekeeper of endothelial growth. As a result, c-Myc transcription is affected. Importantly, we confirm phosphorylation of FoxO1 by aPKC in more than 70% of the angiosarcoma patients. Our findings may provide a new therapeutic strategy for treatment of malignant cancers, such as angiosarcoma.

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