The 716th Kitasato Medical Society Invitational Academic Lecture Series Abstract

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Regulation of tissue morphogenesis by endothelial cell-derived signals

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Endothelial cells (ECs) form an extensive network of blood vessels that has numerous essential functions in the vertebrate body. In addition to their well-established role as a versatile transport network, blood vessels can induce organ formation or direct growth and differentiation processes by providing signals in a paracrine (angiocrine) fashion. Tissue repair also requires the local restoration of vasculature. ECs are emerging as important signaling centers that coordinate regeneration and help to prevent deregulated, disease-promoting processes. Vascular cells are also part of stem cell niches and have key roles in hematopoiesis, bone formation, and neurogenesis. Here, we review these newly identified roles of ECs in the regulation of organ morphogenesis, maintenance, and regeneration.

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Endothelial epsins as regulators and potential therapeutic targets of tumor angiogenesis

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VEGF-driven tumor angiogenesis has been validated as a central target in several tumor types deserving of continuous and further considerations to improve the efficacy and selectivity of the current therapeutic paradigms. Epsins, a family of endocytic clathrin adaptors, have been implicated in regulating endothelial cell VEGFR2 signaling, where its inactivation leads to nonproductive leaky neo-angiogenesis and, therefore, impedes tumor development and progression. Targeting endothelial epsins is of special significance due to its lack of affecting other angiogenic-signaling pathways or disrupting normal quiescent vessels, suggesting a selective modulation of tumor angiogenesis. This review highlights seminal findings on the critical role of endothelial epsins in tumor angiogenesis and their underlying molecular events, as well as strategies to prohibit the normal function of endogenous endothelial epsins that capitalize on these newly understood mechanisms.