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Articles of Significant Interest in This Issue

A CCN1–Yes-Associated Protein Feedback Loop Regulates Physiological and Pathological Angiogenesis

The cellular communication network factor 1 (CCN1) is a matricellular protein with critical functions in blood vessel development, regeneration, and stability. CCN1 controls the flow of information between the cells and their immediate environment. Lee et al. (e00107-19) show that the CCN1 gene is transcriptionally activated in vascular sprouts via Yes-associated protein (YAP), a transcriptional coactivator of growth and differentiation genes. Forced expression of CCN1 leads to YAP inactivation and cytoplasmic sequestration. Loss of this CCN1 negative feedback on YAP activity results in aberrant vascular growth, similar to that seen in mice with CCN1 deficiency or in pathological conditions associated with dysregulated CCN1 gene expression.

Metabolic Regulation of DNA Damage Response Signaling by Nitric Oxide

The metabolic coupling of glycolysis and mitochondrial oxidation in pancreatic β cells is required for secretion of the appropriate amount of insulin to clear glucose from the bloodstream. Oleson et al. (e00153-19) show that it is this same cell-type-selective regulation of intermediary metabolism that allows nitric oxide to protect β cells from DNA damage-directed apoptosis. In β cells and in non-β cells forced to derive ATP solely by mitochondrial oxidation, nitric oxide and inhibitors of mitochondrial respiration attenuate DNA damage response (DDR) signaling and apoptosis. These findings identify cellular metabolism as a novel regulator of DDR signaling and DNA damage-associated apoptosis.

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