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## Oral Communication Abstract
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# Paricalcitol ameliorates hypoxia- and TGF-β1-induced injury in kidney pericytes

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**Objectives:** Kidney fibrosis contributes the progression of chronic kidney disease. The emerging role of renal pericytes on kidney fibrosis has revealed recently. Paricalcitol is reported to have anti-inflammatory, antifibrotic, and antioxidant effects. We evaluated the effect of paricalcitol in hypoxia- and transforming growth factor (TGF)-β1-induced kidney pericyte injury.

**Methods:** Pericytes were cultured and treated with TGF-β1 (5 ng/mL) or $H_2O_2$ (300 μM) or exposed to hypoxia (1% $O_2$ and 5% $CO_2$). For paricalcitol treatment, cultured pericytes were pretreated with paricalcitol (20 ng/mL) for 90 min before inducing injury. We measured cell migration and wound healing in TGF-β1 and paricalcitol-exposed pericytes.The gene expression of fibrosis, oxidative stress markers, and related pathways were evaluated by real-time PCR and western blot analysis.

**Results:** TGF-β1 induced pericyte-to-myofibroblast transition (PMT) and paricalcitol attenuated the expression of upregulated fibrosis markers, including alpha-smooth muscle actin (α-SMA), fibronectin, vimentin, and matrix metallopeptidase-9 (MMP-9) by inhibiting Smad2 phosphorylation. Paricalcitol inhibited TGF-β1-induced cell migration in pericytes. Hypoxia induced oxidative stress injury by activation of hypoxia-inducible factor (HIF)-1α pathway, such as HIF-1α, prolyl hydroxylase 3 (PHD3), and glucose transporter 1 (GLUT-1), and leaded to increased expression of fibrosis markers by upregulation of TGF-β/Smad pathway in pericytes. Paricalcitol ameliorated activation of HIF-1α and TGF-β/Smad signaling pathways in hypoxic pericytes, in particular, by inhibition of Smad2 phosphorylation.

**Conclusions:** Paricalcitol ameliorated PMT by inhibition of Smad2 signaling pathway and oxidative stress in hypoxia- and TGF-β1-induced pericyte injury.