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**ABSTRACT:** Arterial injury induces vascular smooth muscle cells (VSMC) to modulate from a quiescent to a proliferative state characterized by cell division, migration, and secretion of matrix. These changes have been implicated in the development of intimal hyperplasia after balloon angioplasty. The transition of VSMC to a proliferative state is preceded by the accumulation of platelets and leukocytes, which may release growth factors and cytokines at the site of injury. Platelet-derived growth factor (PDGF) is secreted by platelets and a variety of cellular elements associated with the vessel wall and, as a VSMC mitogen and chemoattractant, has been implicated in the pathogenesis of intimal hyperplasia. We have found that, in addition to its effects on VSMC growth and migration, **PDGF induces the expression in VSMC of the JE and KC genes, which encode monocyte and neutrophil chemoattractants, respectively.** The induction of JE and KC by PDGF in VSMC culture involves several distinct transmembrane signaling pathways. In addition to their induction in VSMC culture, JE and KC messenger RNA accumulates rapidly and transiently in adult rabbit aorta after balloon dilatation, suggesting a role for these chemoattractants in the early vascular response to injury in vivo. **The VSMC may therefore play a dual role in vessel injury, both as a mediator of the inflammatory response through chemoattractant release and as an effector of the hyperplastic response through proliferation.**

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