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Active TGFβ recruits mesenchymal stem cells for tissue repair/remodeling

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Transforming growth factor (TGF) β is maintained in a sequestered state in extracellular matrix as a latent form, which is considered as a molecular sensor that releases active TGFβ in response to the perturbations of the extracellular matrix. The biological implication of the temporal discontinuity of TGFβ storage in the matrix and its activation is obscure. We show that active TGFβ controls the mobilization and recruitment of (messenchymal stem cells) MSCs to participate in tissue repair and remodeling. MSCs were mobilized into the peripheral blood in response to vascular injury and recruited to the injured sites where they gave rise to both endothelial cells for reendothelialization and myofibroblastic cells to form thick neointima. Intravenously injection of recombinant active TGFβ1 in uninjured mice rapidly mobilized MSCs into circulation. Further, inhibitor of TGFβ type I receptor blocked the mobilization and recruitment of MSCs to the injured arteries. Thus, TGFβ is an injury-activated messenger essential for the mobilization and recruitment of MSCs to participate in tissue repair/remodeling.

Biography

Mei Wan, MD., Ph.D. is an Associate Professor of the Center for Musculoskeletal Research, Department of Orthopaedic Surgery at Johns Hopkins University School of Medicine. For 15 years, her research focuses on understanding how TGFβ/Smads signaling regulates the behavior of mesenchymal stem cells (B.Sc.s) in tissue homeostasis, repair and remodeling. In particular, we found that active TGFβ can be released from tissue in response to perturbations to the local environment such as bone remodeling and arterial injury. The released active TGFβ stimulates the migration of B.Sc.s to participate in tissue repair or remodeling. Currently, she is an Editorial Board Member for *Journal of Bone and Mineral Research and Bone Research*.

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