21. Rose TM, Bruce AG. Oncostatin M is a member of a cytokine family that includes leukemia-inhibitory factor, granulocyte colony-stimulating factor, and interleukin 6. Proc Natl Acad Sci U S A 88 (1991): 8641-8645.

22. West NR, Owens BMJ, Hegazy AN. The oncostatin M-stromal cell axis in health and disease. Scand J Immunol 88 (2018): e12694.

23. Richards CD. The enigmatic cytokine oncostatin M and roles in disease. ISRN Inflamm 2013 (2013): 512103.

24. Hermanns HM. Oncostatin M and interleukin-31: Cytokines, receptors, signal transduction and physiology. Cytokine Growth Factor Rev 26 (2015): 545-558.

25. Liu J, Modrell B, Aruffo A, et al. Interactions between oncostatin M and the IL-6 signal transducer, gp130. Cytokine 6 (1994): 272-278.

26. Gearing DP, Bruce AG. Oncostatin M binds the high-affinity leukemia inhibitory factor receptor. New Biol 4 (1992): 61-65.

27. Mosley B, De Imus C, Friend D, et al. Dual oncostatin M (OSM) receptors. Cloning and characterization of an alternative signaling subunit conferring OSM-specific receptor activation. J Biol Chem 271 (1996): 32635-32643.

28. Fossey SL, Bear MD, Kisseberth WC, et al. Oncostatin M promotes STAT3 activation, VEGF production, and invasion in osteosarcoma cell lines. BMC Cancer 11 (2011): 125.

29. Rawlings JS, Rosler KM, Harrison DA. The JAK/STAT signaling pathway. J Cell Sci 117 (2004): 1281-1283.

30. Zarling JM, Shoyab M, Marquardt H, et al. Oncostatin M: a growth regulator produced by differentiated histiocytic lymphoma cells. Proc Natl Acad Sci U S A 83 (1986): 9739-9743.

31. Mittal M, Siddiqui MR, Tran K, et al. Reactive oxygen species in inflammation and tissue injury. Antioxid Redox Signal 20 (2014): 1126-1167.

32. Eming SA, Martin P, Tomic-Canic M. Wound repair and regeneration: mechanisms, signaling, and translation. Sci Transl Med 6 (2014): 265sr6.

33. Grenier A, Dehoux M, Boutten A, et al. Oncostatin M production and regulation by human polymorphonuclear neutrophils. Blood 93 (1999): 1413-1421.

34. Hurst SM, McLoughlin RM, Monslow J, et al. Secretion of oncostatin M by infiltrating neutrophils: regulation of IL-6 and chemokine expression in human mesothelial cells. J Immunol 169 (2002): 5244-5251.

35. Albasanz-Puig A, Murray J, Namekata M, et al. Opposing roles of STAT-1 and STAT-3 in regulating vascular endothelial growth factor expression in vascular smooth muscle cells. Biochem Biophys Res Commun 428 (2012): 179-184.

36. Weiss TW, Kvakan H, Kaun C, et al. The gp130 ligand oncostatin M regulates tissue inhibitor of metalloproteinases-1 through ERK1/2 and p38 in human adult cardiac myocytes and in human adult cardiac fibroblasts: a possible role for the gp130/gp130 ligand system in the modulation of extracellular matrix degradation in the human heart. J Mol Cell Cardiol 39 (2005): 545-551.

37. Zhang X, Li J, Qin JJ, et al. Oncostatin M receptor beta deficiency attenuates atherogenesis by inhibiting JAK2/STAT3 signaling in macrophages. J Lipid Res 58 (2017): 895-906.

38. Rozario T, DeSimone DW. The extracellular matrix in development and morphogenesis: a dynamic view. Dev Biol 341 (2010): 126-140.

39. Li L, Zhao Q, Kong W. Extracellular matrix remodeling and cardiac fibrosis. Matrix Biol 68-69 (2018): 490-506.

40. Cheresh P, Kim SJ, Tulasiram S, et al. Oxidative stress and pulmonary fibrosis. Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease 1832 (2013): 1028-1040.

41. Siwik DA, Pagano PJ, Colucci WS. Oxidative stress regulates collagen synthesis and matrix metalloproteinase activity in cardiac fibroblasts. Am J Physiol Cell Physiol 280 (2001): C53-60.

42. Zhao W, Zhao T, Chen Y, et al. Oxidative stress mediates cardiac fibrosis by enhancing transforming growth factor-beta1 in hypertensive rats. Mol Cell Biochem 317 (2008): 43-50.

43. Liu C, Yang Q, Fang G, et al. Collagen metabolic disorder induced by oxidative stress in human uterosacral ligament-derived fibroblasts: A possible pathophysiological mechanism in pelvic organ prolapse. Mol Med Rep 13 (2016): 2999-3008.

44. Lee HB, Yu MR, Song JS, et al. Reactive oxygen species amplify protein kinase C signaling in high glucose-induced fibronectin expression by human peritoneal mesothelial cells. Kidney Int 65 (2004): 1170-1179.

45. Nybo T, Cai H, Chuang CY, et al. Chlorination and oxidation of human plasma fibronectin by myeloperoxidase-derived oxidants, and its consequences for smooth muscle cell function. Redox Biol 19 (2018): 388-400.

46. Allon M, Litovsky S, Young CJ, et al. Medial fibrosis, vascular calcification, intimal hyperplasia, and arteriovenous fistula maturation. Am J Kidney Dis 58 (2011): 437-443.

47. Marden G, Wan Q, Wilks J, et al. The role of the oncostatin M/OSM receptor beta axis in activating dermal microvascular endothelial cells in systemic sclerosis. Arthritis Res Ther 22 (2020): 179.

48. Richards CD, Botelho F. Oncostatin M in the Regulation of Connective Tissue Cells and Macrophages in Pulmonary Disease. Biomedicines 7 (2019).

49. Luft R, Landau B. Mitochondrial medicine. Journal of internal medicine 238 (1995): 405-421.

50. Sorescu D, Griendling KK. Reactive oxygen species, mitochondria, and NAD(P)H oxidases in the development and progression of heart failure. Congest Heart Fail 8 (2002): 132-140.

51. Madamanchi NR, Runge MS. Mitochondrial dysfunction in atherosclerosis. Circ Res 100 (2007): 460-473.

52. Turrens JF. Mitochondrial formation of reactive oxygen species. The Journal of physiology 552 (2003): 335-344.

53. Chen Q, Vazquez EJ, Moghaddas S, et al. Production of reactive oxygen species by mitochondria: central role of complex III. J Biol Chem 278 (2003): 36027-36031.

54. Nicholls DG, Budd SL. Mitochondria and neuronal survival. Physiological reviews 80 (2000): 315-360.

55. Madamanchi NR, Moon SK, Hakim ZS, et al. Differential activation of mitogenic signaling pathways in aortic smooth muscle cells deficient in superoxide dismutase isoforms. Arterioscler Thromb Vasc Biol 25 (2005): 950-956.

56. Crompton M. The mitochondrial permeability transition pore and its role in cell death. Biochem J 341 (1999): 233-249.

57. Halestrap AP, Clarke SJ, Javadov SA. Mitochondrial permeability transition pore opening during myocardial reperfusion--a target for cardioprotection. Cardiovasc Res 61 (2004): 372-385.

58. Di Lisa F, Canton M, Menabo R, et al. Mitochondria and reperfusion injury. The role of permeability transition. Basic Res Cardiol 98 (2003): 235-241.

59. Honda HM, Korge P, Weiss JN. Mitochondria and ischemia/reperfusion injury. Annals of the New York Academy of Sciences 1047 (2005): 248-258.

60. Sakakura K, Nakano M, Otsuka F, et al. Pathophysiology of atherosclerosis plaque progression. Heart Lung Circ 22 (2013): 399-411.

61. Sluimer JC, Gasc JM, van Wanroij JL, et al. Hypoxia, hypoxia-inducible transcription factor, and macrophages in human atherosclerotic plaques are correlated with intraplaque angiogenesis. J Am Coll Cardiol 51 (2008): 1258-1265.

62. Semenza GL. Hypoxia-inducible factor 1: oxygen homeostasis and disease pathophysiology. Trends Mol Med 7 (2001): 345-350.

63. Semenza GL. Targeting HIF-1 for cancer therapy. Nat Rev Cancer 3 (2003): 721-732.

64. Weidemann A, Johnson R. Biology of HIF-1α. Cell Death & Differentiation 15 (2008): 621-627.

65. Gao L, Chen Q, Zhou X, et al. The role of hypoxia-inducible factor 1 in atherosclerosis. J Clin Pathol 65 (2012): 872-876.

66. Vink A, Schoneveld AH, Lamers D, et al. HIF-1 alpha expression is associated with an atheromatous inflammatory plaque phenotype and upregulated in activated macrophages. Atherosclerosis 195 (2007): e69-75.

67. Houben E, Hellings N, Broux B. Oncostatin M, an Underestimated Player in the Central Nervous System. Front Immunol 10 (2019): 1165.

68. Battello N, Zimmer AD, Goebel C, et al. The role of HIF-1 in oncostatin M-dependent metabolic reprogramming of hepatic cells. Cancer Metab 4 (2016): 3.

69. Chang SH, Hwang CS, Yin JH, et al. Oncostatin M-dependent Mcl-1 induction mediated by JAK1/2-STAT1/3 and CREB contributes to bioenergetic improvements and protective effects against mitochondrial dysfunction in cortical neurons. Biochim Biophys Acta 1853 (2015): 2306-2325.

70. Demyanets S, Kaun C, Rychli K, et al. The inflammatory cytokine oncostatin M induces PAI-1 in human vascular smooth muscle cells in vitro via PI 3-kinase and ERK1/2-dependent pathways. Am J Physiol Heart Circ Physiol 293 (2007): H1962-1968.

71. Lijnen HR. Pleiotropic functions of plasminogen activator inhibitor-1. J Thromb Haemost 3 (2005): 35-45.

72. Ghosh AK, Vaughan DE. PAI-1 in tissue fibrosis. Journal of cellular physiology 227 (2012): 493-507.

73. Liu RM. Oxidative stress, plasminogen activator inhibitor 1, and lung fibrosis. Antioxid Redox Signal 10 (2008): 303-319.

74. Liu RM, Choi J, Wu JH, et al. Oxidative modification of nuclear mitogen-activated protein kinase phosphatase 1 is involved in transforming growth factor beta1-induced expression of plasminogen activator inhibitor 1 in fibroblasts. J Biol Chem 285 (2010): 16239-16247.

75. Liu RM, Gaston Pravia KA. Oxidative stress and glutathione in TGF-beta-mediated fibrogenesis. Free Radic Biol Med 48 (2010): 1-15.

76. Ueno M, Maeno T, Nomura M, et al. Hypoxia-inducible

factor-1alpha mediates TGF-beta-induced PAI-1 production in alveolar macrophages in pulmonary fibrosis. Am J Physiol Lung Cell Mol Physiol 300 (2011): L740-752.

77. Louis K, Hertig A. How tubular epithelial cells dictate the rate of renal fibrogenesis? World J Nephrol 4 (2015): 367-373.

78. Zhang Q, Wu Y, Ann DK, et al. Mechanisms of hypoxic regulation of plasminogen activator inhibitor-1 gene expression in keloid fibroblasts. J Invest Dermatol 121 (2003): 1005-1012.

79. Zhang Q, Wu Y, Chau CH, et al. Crosstalk of hypoxia-mediated signaling pathways in upregulating plasminogen activator inhibitor-1 expression in keloid fibroblasts. J Cell Physiol 199 (2004): 89-97.

80. Misra S, Shergill U, Yang B, et al. Increased expression of HIF-1alpha, VEGF-A and its receptors, MMP-2, TIMP-1, and ADAMTS-1 at the venous stenosis of arteriovenous fistula in a mouse model with renal insufficiency. J Vasc Interv Radiol 21 (2010): 1255-1261.

81. Wu J, Peng L, McMahon GA, et al. Recombinant plasminogen activator inhibitor-1 inhibits intimal hyperplasia. Arterioscler Thromb Vasc Biol 29 (2009): 1565-1570.

82. Furumoto T, Fujii S, Nishihara K, et al. Maladaptive arterial remodeling with systemic hypertension associated with increased concentrations in blood of plasminogen activator inhibitor type-1 (PAI-1). Am J Cardiol 93 (2004): 997-1001.

83. Tominaga Y, Miyagawa S, Kawamura T, et al. Mechanism of Cardiac Repair in Rat Myocardial Infarction Model Treated with Extracellular Vesicles from Differentiating Induced Pluripotent Stem Cell-Derived Cardiomyocytes. Circulation 144 (2021): A10495-A.

84. Baldwin JF, Sood V, Elfline MA, et al. The role of urokinase plasminogen activator and plasmin activator inhibitor-1 on vein wall remodeling in experimental deep vein thrombosis. J Vasc Surg 56 (2012): 1089-1097.

85. Kaikita K, Schoenhard JA, Painter CA, et al. Potential roles of plasminogen activator system in coronary vascular remodeling induced by long-term nitric oxide synthase inhibition. J Mol Cell Cardiol 34 (2002): 617-627.

86. Ji Y, Strawn TL, Grunz EA, et al. Multifaceted role of plasminogen activator inhibitor-1 in regulating early remodeling of vein bypass grafts. Arterioscler Thromb Vasc Biol 31 (2011): 1781-1787.

87. Hisada Y, Garratt KB, Maqsood A, et al. Plasminogen activator inhibitor 1 and venous thrombosis in pancreatic cancer. Blood Adv 5 (2021): 487-495.

88. Meltzer ME, Lisman T, de Groot PG, et al. Venous thrombosis risk associated with plasma hypofibrinolysis is explained by elevated plasma levels of TAFI and PAI-1. Blood 116 (2010): 113-121.

89. Diebold I, Kraicun D, Bonello S, et al. The 'PAI-1 paradox' in vascular remodeling. Thromb Haemost 100 (2008): 984-991.

90. Cesari M, Pahor M, Incalzi RA. Plasminogen activator inhibitor-1 (PAI-1): a key factor linking fibrinolysis and age-related subclinical and clinical conditions. Cardiovasc Ther 28 (2010): e72-91.

91. Meade ES, Ma YY, Guller S. Role of hypoxia-inducible transcription factors 1alpha and 2alpha in the regulation of plasminogen activator inhibitor-1 expression in a human trophoblast cell line. Placenta 28 (2007): 1012-1019.

92. Reilly CF, Fujita T, Mayer EJ, et al. Both circulating and clot-bound plasminogen activator inhibitor-1 inhibit endogenous fibrinolysis in the rat. Arterioscler Thromb 11 (1991): 1276-1286.

93. Gorlatova NV, Cale JM, Elokdah H, et al. Mechanism of inactivation of plasminogen activator inhibitor-1 by a small molecule inhibitor. J Biol Chem 282 (2007): 9288-9296.

94. Hertig A, Berrou J, Allory Y, et al. Type 1 plasminogen activator inhibitor deficiency aggravates the course of experimental glomerulonephritis through overactivation of transforming growth factor beta. FASEB J 17 (2003): 1904-1906.

95. Lackie PM. Molecular portfolios: cells interacting with matrix in repairing airway epithelium. Clin Exp Allergy 38 (2008): 1840-1843.

96. Vayalil PK, Iles KE, Choi J, et al. Glutathione suppresses TGF-beta-induced PAI-1 expression by inhibiting p38 and JNK MAPK and the binding of AP-1, SP-1, and Smad to the PAI-1 promoter. Am J Physiol Lung Cell Mol Physiol 293 (2007): L1281-1292.

97. Vayalil PK, Olman M, Murphy-Ullrich JE, et al. Glutathione restores collagen degradation in TGF-beta-treated fibroblasts by blocking plasminogen activator inhibitor-1 expression and activating plasminogen. Am J Physiol Lung Cell Mol Physiol 289 (2005): L937-945.

98. Higgins PJ, Slack JK, Diegelmann RF, et al. Differential regulation of PAI-1 gene expression in human fibroblasts predisposed to a fibrotic phenotype. Exp Cell Res 248 (1999): 634-642.

99. Tuan TL, Zhu JY, Sun B, et al. Elevated levels of plasminogen activator inhibitor-1 may account for the altered fibrinolysis by keloid fibroblasts. J Invest Dermatol 106 (1996): 1007-1011.

100. Chan JC, Duszczyszyn DA, Castellino FJ, et al. Accelerated skin wound healing in plasminogen activator inhibitor-1-deficient mice. Am J Pathol 159 (2001): 1681-1688.

101. Schvartz I, Seger D, Shaltiel S. Vitronectin. The international journal of biochemistry & cell biology 31 (1999): 539-544.

102. Bauman KA, Wettlaufer SH, Okunishi K, et al. The antifibrotic effects of plasminogen activation occur via prostaglandin E2 synthesis in humans and mice. J Clin Invest 120 (2010): 1950-1960.

103. Eitzman DT, McCoy RD, Zheng X, et al. Bleomycin-induced pulmonary fibrosis in transgenic mice that either lack or overexpress the murine plasminogen activator inhibitor-1 gene. J Clin Invest 97 (1996): 232-237.

104. Hattori N, Degen JL, Sisson TH, et al. Bleomycin-induced pulmonary fibrosis in fibrinogen-null mice. J Clin Invest 106 (2000): 1341-1350.

105. Senoo T, Hattori N, Tanimoto T, et al. Suppression of plasminogen activator inhibitor-1 by RNA interference attenuates pulmonary fibrosis. Thorax 65 (2010): 334-340.

106. Wilberding JA, Ploplis VA, McLennan L, et al. Development of pulmonary fibrosis in fibrinogen-deficient mice. Ann N Y Acad Sci 936 (2001): 542-548.

107. Sisson TH, Hattori N, Xu Y, et al. Treatment of bleomycin-induced pulmonary fibrosis by transfer of urokinase-type plasminogen activator genes. Hum Gene Ther 10 (1999): 2315-2323.

108. Swaisgood CM, French EL, Noga C, et al. The development of bleomycin-induced pulmonary fibrosis in mice deficient for components of the fibrinolytic system. Am J Pathol 157 (2000): 177-187.

109. Simone S, Loverre A, Cariello M, et al. Arteriovenous fistula stenosis in hemodialysis patients is characterized by an increased adventitial fibrosis. J Nephrol 27 (2014): 555-562.

110. Iglesias-De La Cruz MC, Ruiz-Torres P, Alcami J, et al. Hydrogen peroxide increases extracellular matrix mRNA through TGF-beta in human mesangial cells. Kidney Int 59 (2001): 87-95.

111. Kucia-Tran JA, Tulkki V, Scarpini CG, et al. Anti-oncostatin M antibody inhibits the pro-malignant effects of oncostatin M receptor overexpression in squamous cell carcinoma. J Pathol 244 (2018): 283-295.

112. Elokdah H, Abou-Gharbia M, Hennan JK, et al. Tiplaxtinin, a novel, orally efficacious inhibitor of plasminogen activator inhibitor-1: design, synthesis, and preclinical characterization. J Med Chem 47 (2004): 3491-3494.

113. Hennan JK, Elokdah H, Leal M, et al. Evaluation of PAI-039 [1-benzyl-5-[4-(trifluoromethoxy)phenyl]-1H-indol-3-yl(oxo)acetic acid], a novel plasminogen activator inhibitor-1 inhibitor, in a canine model of coronary artery thrombosis. J Pharmacol Exp Ther 314 (2005): 710-716.

114. Sillen M, Declerck PJ. Targeting PAI-1 in Cardiovascular Disease: Structural Insights Into PAI-1 Functionality and Inhibition. Front Cardiovasc Med 7 (2020): 622473.

115. Simon DI, Simon NM. Plasminogen activator inhibitor-1: a novel therapeutic target for hypertension? Circulation 128 (2013): 2286-2288.

116. Kairuz EM, Barber MN, Anderson CR, et al. C-type natriuretic peptide (CNP) suppresses plasminogen activator inhibitor-1 (PAI-1) in vivo. Cardiovasc Res 66 (2005): 574-582.

117. Waters DW, Blokland KEC, Pathinayake PS, et al. STAT3 Regulates the Onset of Oxidant-induced Senescence in Lung Fibroblasts. Am J Respir Cell Mol Biol 61 (2019): 61-73.

118. Jung RG, Motazedian P, Ramirez FD, et al. Association between plasminogen activator inhibitor-1 and cardiovascular events: a systematic review and meta-analysis. Thromb J 16 (2018): 12.

119. Morrow GB, Whyte CS, Mutch NJ. A Serpin With a Finger in Many PAIs: PAI-1's Central Function in Thromboinflammation and Cardiovascular Disease. Front Cardiovasc Med 8 (2021): 653655.