CYTOKINES AND EXTRACELLULAR MATRIX REMODELING

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1. Wound repair is the best model for studying extracellular matrix remodeling. The following book is presently the reference work on this topic:

Clark R.A.F. The molecular and cellular biology of wound repair, 2nd edition. Plenum Press (New-York, London), 1998.

For specific information concerning extracellular matrix remodeling in wounds, see particularly the following contributions:

1. 1. Clark R.A.F. Wound repair overview and general considerations, ibid, pp 3-50.

1.2. Riches, D.W.H. Macrophage involvement in wound repair, remodeling and fibrosis, ibid, pp 95-112

1.3. Desmoulières, A., Gabbiani, G. The role of the myofibroblast in wound healing and fibrocontractive diseases, ibid, pp 390-426.

1.4. Mignatis, P., Rifkin, D.B., Welgus, H.G., Parks, C.W. Proteinases and tissue remodeling, ibid, pp 426-474.

2. Interleukin-1 is one of the main cytokine involved in matrix remodeling:

Three complete and up-to date reviews on interleukin- 1 and its biological effects:

2.1. Roux-Lombard, P. The interleukin-1 family. Eur. Cytokine Network, 1998, 9: 565 -576.

2.2. Dinarello, C.A. Interleukin-1. Cytokine and Growth Factor Review, 1997, 8: 253-265.

2.3. Patarca, R., Fletcher, M.A. Interleukin-1: basic science and clinical applications. Critical Reviews in Oncogenesis. 1997, 8: 143-188

Three recent papers showing that interleukin-1 is also involved in fibrosis:

2.4. Jimenez, S.A., Hitraya, F., Varga, J. Pathogenesis of scleroderma collagen. Rheum. Dis. Clin. N. Am. 1996, 22: 647-674.

2.5. Nikolic-Paterson, D.J., Main, I.W., Tesh, G.H., Lan, H.Y., Atkins, R.C. Interleukin-1 in renal fibrosis. Kidney Int. 1996, 54: 588-590.

2.6. Zhang, K., Phan, S.H. Cytokines and pulmonary fibroses. Biol. Signal., 1996, 5: 232-239.

3. Tumor Necrosis Factor-α is also strongly involved in matrix remodeling:

Three recent reviews summarizing what is known about TNFα:

3. 1. Beutler, B.A. The role of Tumor Necrosis Factor in health and disease. J Rheumatol.

1999, 26 (Suppl 57): 16-21

3.2. Rink, L., Kirchner, H. Recent progress in the Tumor Necrosis Factor-α field. Int. Arch. Allergy Immunol. 1996, 111: 199-209.

3.3. Bebelmans, M.H., Van Tits, L.J., Buurman, W.A. Tumor Necrosis Factor: function, release and clearance. Crit. Rev. Immunol. 1996, 16: 1 -11.

And, to learn everything about the enzyme which liberates TNF-α from the plasma membrane (TNF-α convertase, or TACE)

3.4. Blobel, C.P. Metalloproteinase-disintegrins: links to cell adhesion and cleavage of TNF-α and Notch. Cell, 1997, 90: 589-592

4. On the expression of proteinases in wound repair:

4.1. Arumugam, S., Jang, Y.C., Chen-Jensen, C., Gibran, N.S., Isik, F.F. Temporal activity of plasminogen activators and matrix metalloproteinases during cutaneous wound repair. Surgery, 1999, 125: 587-593.

This paper is a study of the activity of the main MMPs and of the plasminogen activities in murine excisional wound.

4.2. Siméon, A., Monier, F., Emonard, H., Gillery, P., Birembaut, P., Hornebeck, W., Maquart, F.X. Expression and activation of matrix metalloproteinases in wounds: modulation by the tripeptide-copper complex glycyl-L-histidyl-L-ysine-Cu^{++}. J Invest. Dermatol., 1999, 112: 95 7-964.

This paper reports the kinetic recording of gelatinase expression and activation in dermal wounds.

4.3. Bullen, E.C., Longakey, M.T., Updike, D.L., Beuton, R., Ladin, D., Hou, Z., Howard, E.W. Tissue inhibitor of metalloproteinases-1 is decreased and activated gelatinases are increased in chronic wounds. J. Invest. Dermatol., 1995, 104: 236-240.

This paper shows an imbalance between MMPs and their inhibitors in chronic wounds, indicating that excess proteolysis may delay successful healing.

5. "Matrikines", small peptides derived from the degradation of extracellular matrix macromolecules, are able to modulate connective tissue cell activity and matrix metralloproteinase expression and/or activation.

For some examples, see these 4 papers:

5.1. Siméon, A., Monier, F., Emonard, H., Wegrowski, Y., Bellon, G., Monboisse, J.C., Gillery, P., Hornebeck, W., Maquart, F.X. Fibroblast-cytokine-extracellular matrix interactions in wound repair. Curr. Top. Pathol., 1999, 93: 95-10 1.

5.2. Maquart, F.X., Siméon, A., Pasco, S., Monboisse, J.C. Régulation de l'activité cellulaire par la matrice extracellulaire : le concept de matrikines. J Soc. Biol., sous presse.

5.3. Brassart, B., Randoux, A., Hornebeck, W., Emonard, H. Regulation of matrix metalloproteinase-2, membrane-type matrix metalloproteinase-1, and tissue inhibitor of metalloproteinases-2 by elastin-derived peptides. Clin. Exp. Metast., 1998, 16: 489-500.

5.4. : See also ref 4.2 for the role of the tripeptide glycyl-histidyl-lysine-Cu^{++} in matrix remodeling.

6. Extracellular matrix remodeling during morphogenesis is also regulated by growth factors and cytokines

See :

6. 1. Werb, Z., Chen, J.R. Extracellular matrix remodeling during morphogenesis. In: Fleischmajer, R., Timpl, R., Zerb, W., eds. Morphogenesis: Cellular interactions. Ann. N. Y Acad. Sci USA, 1998, 857: 110-118.