S100A7 (psoriasin) influences immune response genes in human breast cancer. By Soma Mandal, Linda Curtis, Molly Pind, Leigh C. Murphy, and Peter H. Watson. 

This article uses unbiased global expression analysis techniques to show that S100A7 modulates expression of genes that are associated with the immune response in breast tumors. S100A7 has been associated with inflammation, T-cell chemotaxis, and host bacterial defense responses in skin. But S100A7 is also highly expressed in pre-invasive ductal carcinoma in-situ where it can promote cell survival under stress and breast tumor progression through modulation of Jab1, a master regulator of the “wound response” cell signaling pathways. The current findings are consistent with a role for S100A7 in modulating the immune response, raise the possibility that S100A7 may be a marker of this response, and support the view that immune response may be an important factor in early breast tumor progression.


The interactions between cells and their local microenvironment have profound effects on their fate. Extracellular matrix (ECM) regulates cell morphology and gene expression mediated by reorganization of actin cytoskeleton and dictated by chromatin organization. This study demonstrated that culture of breast epithelial cells within 3D-ECM induced a global histone deacetylation associated with chromatin condensation and changes in gene expression. The authors determined that cell “rounding” is sufficient to affect histone deacetylation and chromatin condensation in the absence of integrin-mediated signals from the ECM pointing to other mechanisms. Cell shape-induced changes in chromatin structure could be a common mechanism in regulation of tissue- and context-specific gene expression in vivo.


Regulation of self-renewal in testicular spermatogonial stem cells (SSCs) is not well understood. Two proteins produced by Sertoli cells, somatic cells that form the spermatogonial stem cell niche, are involved in this process. Knockout mice lacking either Ets variant gene 5 (ETV5) or glial cell-derived neurotrophic factor (GDNF) lose all SSCs, but little is known about ETV5 or GDNF regulation. This question was addressed in a new study using cultured TM4 Sertoli cells. FGFs stimulated both ETV5 and GDNF. However, other factors such as EGF stimulated production of Etv5, but not Gdnf, mRNA. Conversely, FSH and cytokines such as IL-1β and TNFα stimulate Gdnf mRNA without effects on Etv5 mRNA. Thus, regulation of each protein is unique, although there appears to be some overlap in the factors that control their production.

Role of the α1 integrin cytoplasmic tail in the formation of focal complexes, actin organization, and in the control of cell migration. By Christiane Smerling, Kerstin Tang, Werner Hofmann, and Kerstin Danker.

Cell migration is a fundamental process essential for embryonic development, tissue morphogenesis, wound healing and immune responses. Collagen-binding integrins are known to play a key role in this vital cell function. In this study, the α1 cytoplasmic tail of integrin α1β1 proved to mediate migration on collagen IV via regulation of the small GTPase Rac1 and PI3K. The conserved GFFKR motif is sufficient to convey Rac1 activation, but downregulates the amount of GTP-Rac1 in the absence of the α1-specific sequence PLKKKMEK. This polybasic sequence and active Rac1 are required for the recruitment of p110 PI3K into focal complexes and the establishment of cell polarity, which is the first function to be assigned to the C-terminus of the α1 tail.