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Matrix biology highlights

Edited by Tom Neill and Jason Zoeller

1). Context dependent protective or promotive laminin function
Reference|Spenlé, C., Lefebvre, O., Lacroute, J., Méchine-Neuville, A., Barreau, F., Blottière, H.M., Duclos, B., Arnold, C., Hussenet. T., Hemmerlé, J., Gullberg, D., Kedinger, M., Sorokin, L., Orend, G., Simon-Assmann, P. "The laminin response in inflammatory bowel disease: protection or malignancy?" 2014. *PLoS One* 9(10):e111336.
http://www.ncbi.nlm.nih.gov/pubmed/25347196
A recent report from Spenlé and colleagues published in PLoS One describes abnormal overexpression of laminin α1 and α5 in human and mouse colitis samples that was associated with p53 expression[1]. To further explore the relevance of these observations, the authors utilized transgenic mice that overexpress intestine-specific *Lama1* or *Lama5*. These mice were less vulnerable to inflammation but more prone to cancer progression in vivo (Fig. 1). These results define unique laminin function related to gastrointestinal inflammation and cancer.

2). Jun and the diffuse large B-cell lymphoma (DLBCL) microenvironment
Reference|Blonska, M., Zhu, Y., Chuang, H.H., You, M.J., Kunkalla, K., Vega, F., Lin, X. "Jun-regulated genes promote interaction of diffuse large B-cell lymphoma with the microenvironment." 2014. *Blood* http://www.ncbi.nlm.nih.gov/pubmed/25533033.

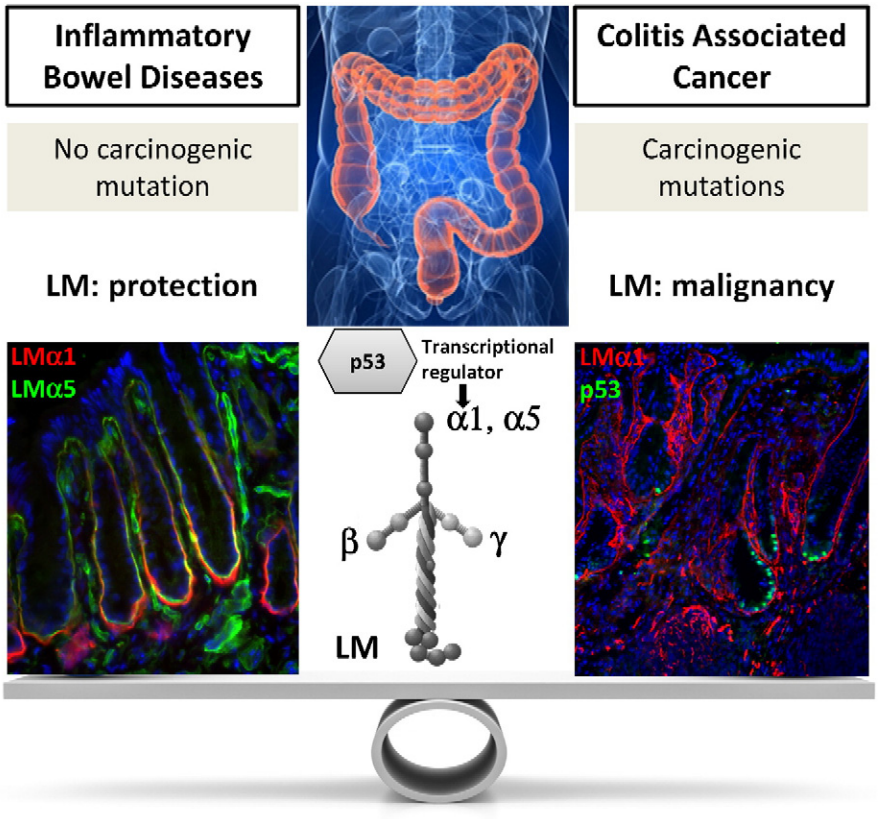

Fig. 1. Laminins reduce inflammation response and promote cancer progression. Laminins (LM) can act in either a protective or a pro-tumorigenic manner depending on the carcinogenic status of the epithelial cells. The p53 transcription factor acting directly on the LMα1 chain here undeniably plays a major regulatory role. Figure kindly provided by Simon-Assmann P.

http://dx.doi.org/10.1016/j.matbio.2015.02.009

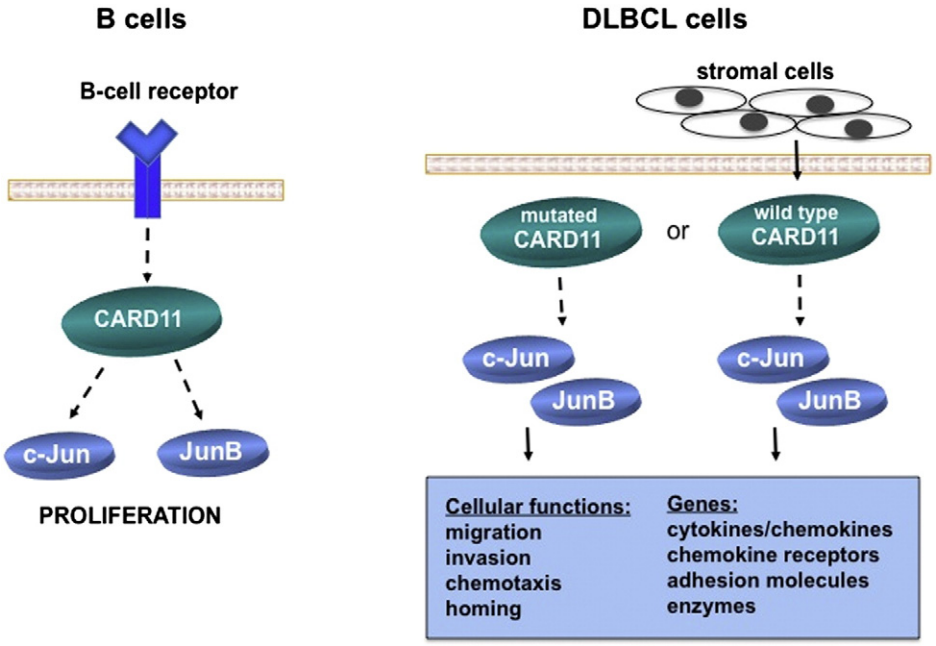

Fig. 2. (Left) Normal B cells, the stimulation of B cell receptor leads to activation of adaptor protein CARD11 and accumulation of c-Jun and JunB transcription factors. (Right) Diffuse large B-cell lymphoma (DLBCL) cells; elevated Jun proteins induce genes that mediate cell migration, invasion, chemotaxis and homing. Figure kindly provided by Blonska M.

A recent report from Blonska and colleagues published in Blood defines crosstalk between DLBCL and the tumor microenvironment mediated via Jun-controlled genes[2]. These results were based upon investigation of the adhesion defect observed in response to c-Jun and JunB knockdown. DLBCL cell lines with reduced Jun lost extracellular matrix attachment abilities in vitro. Furthermore, loss of Jun blocked tumor growth and reduced the dissemination of tumor cells to the bone marrow in vivo. The clinical relevance of these observations was evaluated in patient biopsies alongside PET/CT scans to assess disseminated cells. Supportive of the mouse data, patient samples with high Jun were associated with extranodal disease. These results support Jun-related function at the secondary sites. Coordinated via Jun signals, the production of various cytokines and growth factors was defined as one possible explanation for these observations. Moreover, the identified Jun-target genes (*ITGAV*, *FoxC1* and *CX3CR1*) underscore additional microenvironment-related interactions (Fig. 2).