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Extracellular matrix as an adhesion promoter

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Carbohydrates are the main component of the extracellular matrix (ECM) where they associate with proteins to form glycoproteins or proteoglycans or exist as long-chain disaccharides. All ECM proteins except elastin have associated sugar and in some cases, ECM proteins require proper glycosylation to achieve the full biological activity. It is also now clear that many ECM proteins have carbohydrate-binding domains that specifically recognize and interact with glycoconjugates with other matrix components and on the cell surface. Carbohydrates have been implicated in a wide variety of processes ranging from cell adhesion and migration to matrix assembly, growth factor sequestration and regulation, involvement in many aspects of immune function, binding of plasma proteins and control of thrombogenesis. This contribution is a method of immobilizing and processing functional multi-component structures of the ECM comprising the following successive process steps including covalent binding of an adhesion promoter layer to cell culture carriers culturing cells of a desired type on the adhesion promoter layer and thus immobilizing the ECM secreted by the cells by secretion and binding to the adhesion promoter layer and application of a decellularization protocol so as to detach matrix secreting cells from the surface while simultaneously retaining the structure and functionality of the immobilized ECM which is connected to the adhesion promoter.

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Thrombospondin1, an important mediator of obesity-associated inflammation and insulin resistance

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Thrombospondin 1 (TSP1) is a multifunctional matricellular protein. It is highly expressed in visceral fat tissue (AT) from obese and insulin resistant humans or obese rodents. Recently, both human and rodent data from our lab and others suggest that TSP1 plays an important role in obesity-associated chronic inflammation and insulin resistance (IR). The positive association of adipose tissue TSP1 with AT inflammation and IR has been observed in obese human subjects. By using global TSP1 deficient mice, we revealed a novel role for TSP1 in stimulating macrophage accumulation and activation in AT that promotes inflammation and IR resulting from high fat diet-induced obesity (DIO). Specifically, we found that feeding a high fat diet to wild type and TSP1 deficient mice for 16 weeks caused similar obesity but only mice with TSP1 deficiency remained insulin-sensitive. The protection of TSP1 deficient mice against IR was associated with reduced ATMs, decreased adipose and systemic inflammation and reduced AT fibrosis. Moreover, TSP1 deficiency protected mice from obesity-induced hypertension and kidney damage. In vitro data demonstrated that TSP1 deficient monocyte/macrophages had decreased chemotactic activity and a reduced pro-inflammatory phenotype. TSP1 treatment stimulated macrophage migration. In addition, TSP1 stimulated macrophages to produce pro-inflammatory cytokines which required TLR4 activation and was mediated by interaction between the type 1 repeats of TSP1 (TSR) and its receptor-CD36. Collectively, these data suggest that TSP1 acts as both a chemo attractant and pro inflammatory activator for macrophages in inflamed AT and promotes obesity-induced inflammation and IR.

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