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Thrombospondin 1, 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 chemoattractant and proinflammatory activator for macrophages in inflamed AT, and promotes obesity-induced inflammation and IR.

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Efficacy of dietary therapy in the prevention of headache symptoms in migraine (without aura)

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In the last few years, it has become apparent that a relationship between migraine and insulin resistance may exist. The aim was to report on the efficacy of dietary therapy in the management and treatment of headache symptoms. The dietary regimen employed required a chronic migraine (without aura) patient to: (1) completely eliminate alcohol, monosodium glutamate, artificial sweeteners, sugar and food containing sucrose natural or otherwise from his diet; (2) restrict the consumption of fruit, dairy and fat; (3) consume at least six small meals per day making sure the interval between meals did not exceed 3 hours; (4) increase his intake of complex carbohydrate and water ; (5) upon waking in the middle of the night consume a light snack containing complex carbohydrate and (6) avoid all medication deemed to impact glucoregulation. The results suggested that the implementation of the prescribed dietary regimen was success in reducing headache frequency by at least 70% when compared to pre-treatment levels. Further support for dietary therapy was provided by evidence that any deviation from the prescribed dietary regimen immediately resulted in a 2.5 fold increase in need for headache related pain medication. The data highlights that in some cases the individual's diet may be contributing to the development of headache symptoms. Thus, dietary therapy should also be explored when deciding on the best treatment and management strategy for migraine patients.

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