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Rosiglitazone reduces the inflammatory response in a model of vascular injury in rats.

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Thiazolidinediones are ligands that bind to and activate the nuclear peroxisome proliferator-activated receptor gamma. They are widely used as insulin sensitizers for the treatment of type 2 diabetes. Several studies have implicated the peroxisome proliferator-activated receptor gamma agonists rosiglitazone and pioglitazone in inflammatory events. To assess the anti-inflammatory properties of rosiglitazone, we investigated its effects on the molecular and cellular inflammatory response induced by a carotid injury in the rat. Male Wistar rats were randomized into a rosiglitazone-treated group (10 mg kg(-1) day(-1)) and a control group (0.9% w/v NaCl). The drug or vehicle was administered by gavage for 7 days before carotid injury and for up to 21 days after injury. The inflammatory markers p38 mitogen-activated protein kinase, cyclooxygenase 2, nuclear factor-kappaB, and heat shock protein 47 and the influx and activity of cells in response to injury were measured. Rosiglitazone treatment significantly reduced the expression of the inflammatory markers compared with control group. p38 mitogen-activated protein kinase and nuclear factor-kappaB started to decrease a few hours after injury, whereas cyclooxygenase 2 and heat shock protein 47 expression decreased 7 and 14 days, respectively, after injury. Rosiglitazone also reduced neointima formation and inflammatory cell infiltration. In conclusion, rosiglitazone negatively regulated the inflammatory events involved in tissue repair at molecular and cellular levels. These results suggest that rosiglitazone plays a protective role in inflammatory vascular diseases.

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