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[J Exp Med.](#) 2007 Jan 22;204(1):11-5. Epub 2007 Jan 16.

# The antiinflammatory activity of IgG: the intravenous IgG paradox.

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How high doses of intravenous IgG (IVIg) suppress autoimmune diseases remains unresolved. We have recently shown that the antiinflammatory activity of IVIg can be attributed to a minor species of IgGs that is modified with terminal sialic acids on their Fc-linked glycans. Here we propose that these Fc-sialylated IgGs engage a unique receptor on macrophages that, in turn, leads to the upregulation of an inhibitory Fcgamma receptor (FcgammaR), thereby protecting against autoantibody-mediated pathology.

PMID: 17227911 [PubMed - indexed for MEDLINE]

PMCID: PMC2118416

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