UNC team describes novel inflammatory protein function

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A UNC-led team of scientists describes the function of a previously uncharacterized protein that dramatically influences inflammation.

A majority of the NLR family of proteins function as activators of inflammation. However, scientists at UNC report that a newly identified NLR protein, NLRC3, was able to inhibit a major inflammatory pathway that is controlled by a protein called NF-Kappa B. NF-Kappa B activation has been long associated with inflammation and cancer promotion. Their article appears in the August 5,2012 online publication of the journal Nature Immunology.

The UNC team previously reported that another NLR family member, NLRP12, was also able to inhibit NF-Kappa B activation. However, in their new study, the team reported that NLRC3 inhibits this major inflammatory pathway through a completely different mechanism. The researchers show that NLRC3 directly interacts with the molecule TRAF6 and forms a novel, previously uncharacterized protein complex described as a 'TRAFasome'. TRAF6 is a key regulator of NF-kappaB and is a critical step in the regulation of inflammation.

In pre-clinical models, the team was able to show that NLRC3 and the formation of the TRAFasome was important in regulating the immune response during endotoxic shock, a serious hyperinflammatory process typically associated with severe infection.

Monika, Scheneider, first author of the paper and a postdoctoral research associate at UNC Lineberger Comprehensive Cancer Center, explains, "Our research reveals greater insight into the mechanisms controlling inflammation and identifies potential therapeutic targets."

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