

Abstract

Although inflammation is a primary means for fighting infection, improper regulation of inflammation can result in life threatening conditions. Statins, prescribed to lower cholesterol, also have anti-inflammatory properties. These effects are mediated in part due to decreased synthesis of isoprenoid intermediates that are required by multiple GTP-binding proteins to attach at cell membranes. This study establishes a method for investigating if inhibition of CDC42, a potential master regulator, is sufficient to mimic the anti-inflammatory properties of statins and thus revealing if CDC42 plays a central role in mediating the anti-inflammatory action of statins on macrophages. Macrophages are one of the first immune cells activated in the inflammatory pathway. Expression of macrophage inflammatory surface proteins is used as a measurement of inflammatory response. Here, we compare the expression of macrophage inflammatory surface proteins between untreated macrophages and macrophages treated with lovastatin or ML 141, a small molecule inhibitor that acts selectively on CDC42.

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