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Isoforms of vitamin E differentially regulate inflammation

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Vitamin E regulation of disease has been extensively studied but most studies focus on the α-tocopherol isoform of vitamin E. These reports indicate contradictory outcomes for anti-inflammatory functions of the α-tocopherol isoform of vitamin E with regards to animal and clinical studies. These seemingly disparate results are consistent with our recent studies demonstrating that purified natural forms of vitamin E have opposing regulatory functions during inflammation. We have determined that α-tocopherol inhibits whereas γ-tocopherol elevates allergic inflammation, airway hyperresponsiveness, leukocyte transendothelial migration, and endothelial cell adhesion molecule signaling through protein kinase Cα. Moreover, we determined that α-tocopherol is an antagonist and γ-tocopherol is an agonist of PKCα through direct binding to a regulatory domain of PKCα. Furthermore, in a clinical study, we determined that increasing serum concentrations of γ-tocopherol associated with worse lung function, while increasing serum concentrations of α-tocopherol associated with better lung function. In summary, we have determined mechanisms for opposing regulatory functions of α-tocopherol and γ-tocopherol on inflammation. Information from our studies will have significant impact on the design of clinical studies and on vitamin E consumption.

Biography

Joan M. Cook-Mills completed her Ph.D from Michigan State University and postdoctoral studies from the University of Illinois in Chicago. She was an Assistant Professor at the University of Cincinnati and is now an Associate Professor at Northwestern University. She has published reviews, book chapters and more than 35 papers in reputed journals. She has served as a member of several study sections for the American Heart Association and NIH.

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