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Enhancing host resistance to infections

β Androstenediol epimers (Δ^5 androstane, 3 β , 17 α diol; and Δ^5 androstane, 3 β , 17 β diol) consist of a metabolic endocrine node which functions to regulate host resistance to infections and malignancies. The β Androstane steroids (17 β AED/AET) up-regulate immunity and increase host resistance against lethal infection by viruses, and bacteria. The β Androstenes increase the levels of the TH1 cytokines, IL-2, IL-3, and IFN γ and counteract hydrocortisone mediated immune suppression. Treatment with a single dose of either 17 β AED or 17 β AET protected the host from whole body lethal radiation and led to the recovery of the remaining hematopoietic precursor cells. Increased host resistance protected the host from lethal infection by DNA or RNA viruses such as herpes virus, coxsackievirus B4, influenza, and arthropod borne viruses. Similar protection against lethal Gram positive and Gram-negative bacteria infections has been observed. The specificity of 17 β AED/17 β AET is illustrated by the finding that the 17 α epimer (17 α AED inhibits proliferation and mediates apoptosis in tumor cells of murine and human origin and induces autophagy in human glioblastoma. In summary, these agents provide a unique new avenue for the control, mitigation, and prevention of diseases by viral, bacterial infections. The androstenes are a new subclass of steroid hormones with specific and unique physiological properties.

Biography

Roger M Loria is an Emeritus Professor in Microbiology, Immunology with joint appointments in Pathology and Emergency Medicine. He has obtained his Doctorate from Boston University, Postdoctoral studies by the Massachusetts Heart Association, Greater Boston Chapter and a sabbatical at Harvard Medical School, Boston, MA. He is a Research Fellow in Children's Hospital in Infectious Disease Unit. He has more than 80 publications in peer reviewed journal and more than 25 patents. He is a Member of several Editorial Boards.

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