Complement plays a vital role in host defense for both the innate and adaptive immune systems. But overactive complement can be self damaging. Diseases where this occurs include, but are not limited to, paroxysmal nocturnal hemoglobinemia, macular degeneration, Alzheimer disease, multiple sclerosis, rheumatoid arthritis, atherosclerosis, myasthenia gravis, ALS, Parkinson disease, and malaria infection. We have identified aurin tricarboxylic acid as an orally effective compound that blocks bystander lysis caused by membrane attack complex formation as well as C3 convertase formation in the alternative pathway. It is effective in the nanomolar range. When given orally to Alzheimer disease type B6SJL-Tg mice, it inhibits MAC formation in serum and improves memory retention. Mice given the enormous dose of 500 mg/kg/day show no evidence of harm to any organ upon autopsy. Using ELISA type assays, we established that ATA binds directly to C9 preventing its binding to C5b-8. We also established that it binds to Factor D preventing its cleavage of PC3bB to the C3 convertase PC3bBb. We conclude that ATA, by inhibiting two stages of the alternative pathway, and the final stage of the classical pathway, will become a very broad spectrum therapeutic agent.