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THE EXQUISITE DESIGN OF SOMATIC HYPERMUTATION TO ENHANCE ANTIBODY DIVERSITY, BINDING AFFINITY AND SELF-TOLERANCE

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ABSTRACT

Somatic hypermutation (SHM) of antibody genes is commonly cited as preeminent evidence of the Darwinian “survival of the fittest” in nature. Just as SHM generates antibodies with the strongest binding affinity through random mutation and selection in somatic cells of an individual, the same process occurring in germ cells on a population level can result in macroevolution according to proponents of the latter theory. Although SHM superficially appears to produce point mutations randomly, such mutations are actually generated by a well-designed intricate mechanism contributing to species preservation. Non-random characteristics of SHM are summarized as well as recent findings illuminating its amazing role in rehabilitating self-reactive antibodies, a process called clonal redemption (CR). Unlike haphazard mutations in germ cells purported to provide the raw material for Darwinian evolution, SHM occurs at specific sites in specific genes in specific cells and tissues at a specific developmental stage at a specific rate (increased 10^6 x normal) and duration within specific organisms for a specific purpose. SHM plays an integral role in improving antibody binding affinity in many vertebrates, diversifying the primary antibody repertoire in some mammals, and redeeming auto-antibodies as examined in mice and humans. Activation-induced cytidine deaminase (AID) mediates SHM by deaminating cytidine to uracil while five different, complex DNA damage repair enzyme pathways ultimately replace the uracil-guanine pair with other base pairs. Mutations in AID, which also mediates immunoglobulin class switch recombination, may result in a hyper IgM syndrome, other immune disorders and carcinogenesis. Normally, up to 70% of developing B cells are self-reactive and are deleted or rendered anergic (nonreactive). CR salvages anergic B cells, as they bind foreign antigen and receive T cell co-stimulation. Consequently, they undergo SHM and selection resulting in decreased affinity for self-antigen and increased affinity for foreign antigen. Up to 1000-fold discriminatory affinity between two structurally similar antigens can occur before and after CR. It defies logic to assert that the sophisticated system of SHM, which induces, directs, repairs and abrogates mutations to enable optimal humoral immunity, could have arisen by a non-directed mutational mechanism. Rather, SMH reveals evidence of exquisite design by an omnipotent Creator.

KEYWORDS

somatic hypermutation, antibody, Darwinian evolution, self tolerance

THE AUTHOR

Upon obtaining a PhD in biology education and a MD degree, Frank Maas spent several years teaching biological sciences from the elementary to graduate school levels. In addition, he performed basic science and clinical research in immunology for several years at multiple academic centers. He also obtained a master’s degree in theology as he has a keen interest in issues concerning the intersection of science and religion.