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Variations in the Human Serum Albumin Gene: Molecular and Functional Aspects

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Abstract

In healthy adult humans, human serum albumin (ALB; OMIM # 103600) is continuously released by liver hepatocytes at a rate of 14 grammes per day, with a half-life of roughly 19 days. ALB is the most abundant protein in plasma (about 35–50 gr/L), accounting for 60–65 percent of total proteins and accounting for about 80 percent of oncotic plasma pressure. having esterase-like enzymatic activity and catalytic characteristics based on heme ALB may potentially have an anticoagulant effect by binding antithrombin and preventing platelet aggregation, according to several studies. Recent research suggests that it may play a role in human innate immunity.

Keywords: Albumin; Anticoagulant; Nanomedicine

Introduction

The mature ALB molecule is a single polypeptide chain of 585 amino acids with a molecular mass of 66.5 kDa and no prosthetic groups, covalently attached lipids, or carbohydrates. The protein's three-dimensional structure has now been determined with a resolution of 2.3. ALB contains a 67 percent -helix content and no -sheet secondary structure, according to crystallographic investigations, and is folded into a heart-shaped molecule with three homologous domains (I–III). Each domain is divided into two subdomains (A and B), each with its own helical folding pattern connected by flexible loops. In neutral solution and crystal structures, the overall conformations are extremely similar. ALB is a member of the albumin superfamily, which includes -fetoprotein, vitamin D-binding protein (Gc-globulin), and afamin, among others. -Fetoprotein is essential throughout foetal development, but it is absent in maturity. The -fetoprotein-related gene, the superfamily's fifth gene, has been detected in primates as well, but it has numerous pathogenic mutations in humans that make it an inactive pseudogene. Extracellular matrix protein 1 (ECM1) is also a member of this superfamily, according to recent phylogenetic and structural research. The single-copy genes for ALB, vitamin D-binding protein, afamin, -

fetoprotein, and the -fetoprotein-related gene are all co-dominantly expressed and are located near the centromere on chromosome 4 at position 4q13.3. The five genes are strongly related in humans and all other animals studied, having evolved from a common ancestor through a sequence of duplication events. The relationship between these genes and ECM1, which is located in a different location (1q21.3), has yet to be determined.

Alloalbuminaemia

At the protein and/or gene level, seventy-four different mutations have been identified, resulting in seventy-one unique genetic variations of ALB and pro-albumin. The list of variants, as well as their primary structural and functional features, are summarised in. A more extensive presentation can be found on a dedicated website that our organisation maintains and updates on a regular basis.

Alloalbumin Frequency and Detection

Albumins from heterozygous people appeared on electrophoretic examination as a double band (bisalbuminaemia or alloalbuminaemia) or, less frequently, as a single broadened band. The uncommon homozygous cases, on the other hand, showed a net single band. This method divides the variants into two groups based on their migration speed: those that migrate quicker (more anodic) in comparison to standard ALB, and those that migrate more slowly. Standard serum protein electrophoresis, on the other hand, could only detect alloalbumins with mutations involving charged residues on the molecule's surface. More sophisticated techniques, such as electrospray time-of-flight mass spectrometry, have found novel variations with minimal or no charge changes as compared to the normal protein in recent years (silent albumins, as ALB Lyon, ALB South Pacific, ALB Ilan). Nonetheless, there are likely numerous alloalbumins that have yet to be discovered, resulting in an underestimation of the prevalence of variations in the general population.

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