APOBEC (“apolipoprotein B mRNA editing enzyme, catalytic polypeptide-like”) is a family of evolutionarily conserved cytidine deaminases.

A mechanism of generating protein diversity is mRNA editing. Members of this family are C-to-U editing enzymes. The N-terminal domain of APOBEC like proteins is the catalytic domain, while the C-terminal domain is a pseudocatalytic domain. More specifically, the catalytic domain is a zinc dependent cytidine deaminase domain and is essential for cytidine deamination. RNA editing by APOBEC-1 requires homodimerisation and this complex interacts with RNA binding proteins to form the editosome.

The apoB RNA-editing enzyme, catalytic polypeptide-like (APOBEC) family of proteins includes APOBEC1, APOBEC3, and activation-induced deaminase, all of which are zinc-dependent cytidine deaminases active on polynucleotides and involved in RNA editing or DNA mutation. In contrast, the biochemical and physiological functions of APOBEC2, a muscle-specific member of the family, are unknown, although it has been speculated, like APOBEC1, to be an RNA-editing enzyme. Here, we show that, although expressed widely in striated muscle (with levels peaking late during myoblast differentiation), APOBEC2 is preferentially associated with slow-twitch muscle, with its abundance being considerably greater in soleus compared with gastrocnemius muscle and, within soleus muscle, in slow as opposed to fast muscle fibers. Its abundance also decreases following muscle denervation. We further show that APOBEC2-deficient mice harbor a markedly increased ratio of slow to fast fibers in soleus muscle and exhibit an approximately 15-20% reduction in body mass from birth onwards, with elderly mutant animals revealing clear histological evidence of a mild myopathy. Thus, APOBEC2 is essential for normal muscle development and maintenance of fiber-type ratios; although its molecular function remains to be identified, biochemical analyses do not especially argue for any role in RNA editing [1)].

Sato et al. found two proteins, APOBEC-2 (RNA-editing enzyme) and Gamma-synuclein (breast cancer related protein), which have not been recognized as denervation-induced proteins to date. The results might prove to be beneficial in elucidating the molecular mechanisms of denervation-induced muscle atrophy [2)].

1)
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