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Learn How a Mutation in a 3′ UTR Results in Bulging Biceps in Beef

Although the microRNA is usually 22 bases long, it recognizes its target primarily through a “seed” region of about 5 bases in the 5′ end of the microRNA (usually at positions 2–7). This seed region recognizes targets in the 3′ UTR of the message. What happens, then, if an mRNA has a mutated 3′ UTR? Such a mutation appears to have given rise to the Texel sheep, a breed with a large and well-defined musculature that is the dominant meat-producing sheep in Europe. Genetic techniques mapped the basis of the sheep’s meaty phenotype to the *myostatin* gene. Mutations in the *myostatin* gene can prevent the proper splicing of the pre-mRNA that produce a large-muscled phenotype (Figure 1). Another way of reducing the levels of myostatin involves a mutation in the gene’s 3′ UTR sequence. In the Texel breed, there has been a G-to-A transition in the 3′ UTR of the gene for myostatin, creating a target for the *miR1* and *miR206* microRNAs that are abundant in skeletal muscle (Clop et al. 2006). This mutation causes the depletion of *myostatin* messages and the increase in muscle mass characteristic of these sheep.

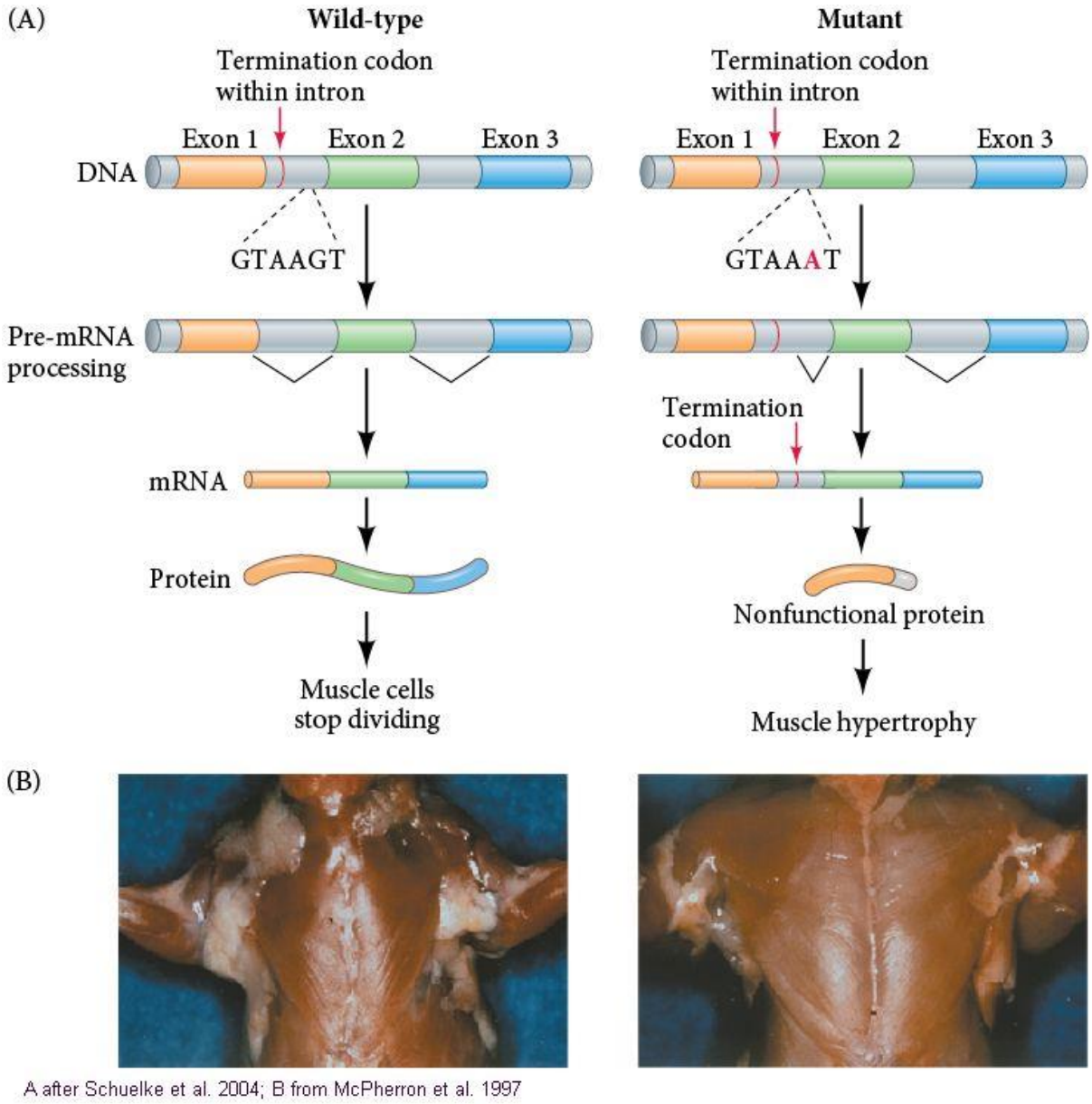

Figure 1 Muscle hypertrophy through mispliced RNA. This mutation results in a deficiency of the negative growth regulator myostatin in the muscle cells. (A) Molecular analysis of the mutation. There is no mutation in the coding sequence of the gene, but in the first intron, a mutation from a G to an A creates a new (and widely used) splicing site, which causes aberrant pre-mRNA splicing and the inclusion of an early protein synthesis termination codon into the mRNA. Thus, proteins made from that message are short and nonfunctional. (B) Pectoral musculature of a "mighty mouse" with the mutation (right) compared with the muscles of a wild-type mouse (left). (A after Schuelke et al. 2004; B from McPherron et al. 1997.)

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