
Myostatin b knockout tilapia exhibits increase in fillet yield and feed conversion efficiency but decrease in Streptococcus resistance and reproductive capacity

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Abstract

Precision breeding of mighty tilapia (MiT-1) with 7 bp and 13 bp deletion in exon 1 of *myostatin b* (*mstnb*) gene leading to loss of function were established from NT1 Taiwan tilapia by CRISPR/Cas9 genome editing to F5 generation and F3 generation, respectively, which both show high fillet yield phenotype (50~60%). The MiT-1 strain exhibits significant double-muscle phenotype with increase in muscle mass of the dorsal, abdominal, and lateral regions, resulting in notable increases in body height, body width, and body weight but shorter body length. The condition factor (CF) of *mstnb* bb (6.05 ± 0.72) is significantly better than that of *mstnb* BB (3.63 ± 0.23) at 9-month adult tilapia. Both male and female MiT-1 adult tilapia exhibit significantly double muscle phenotype with higher fillet yield although male tilapia is still larger than female. However, enhanced muscle growth in *mstnb* KO tilapia may have a negative effect on pathogen resistance. Survival rate was reduced to 13.3 % of homozygotes bb (n=30) and to 23.3 % of heterozygotes Bb (n=30) compared with 46.7 % of wild-type BB (n=30) sibling after being challenged by high virulent *Streptococcus iniae* 89353 (3.6×10 CFU/g body weight) by Intraperitoneal Injection. However, the reduced disease resistance, and low reproductive capacity is beneficial for biosafety and breed control in aquaculture due to obstacle to inbreeding of *mstnb* homozygotes. Furthermore, *Bidens pilosa* extract power as a multi-functional feed additive to enhance the growth, metabolism, and immunity of tilapia, can be further applied in *mstnb* heterozygous and homozygous tilapia.

Keywords: tilapia, myostatin, CRISPR/Cas9, genome editing, fillet yield, disease, Streptococcus iniae, reproduction

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