

Improved Feed Efficiency in Quail with Targeted Genome Editing in Myostatin Gene

Analysis of Gene Editing on Feed Intake and Fat Growth

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INTRODUCTION

Poultry is an important meat source across the globe and increasing muscle growth that has a lower proportion of fat can greatly benefit the industry. Feed efficiency is also an aspect in need of consideration for economic viability. Myostatin (MSTN) is a well-known gene that is expressed in skeletal muscle and negatively regulates muscle growth. The MSTN gene has been found to increase muscle growth in various species but this mutation is more recent in the avian species. Interconnected with muscle growth, changes in feed efficiency and fat accretion are other factors that have not been extensively studied in MSTN mutation species. Having poultry that is more efficient at converting feed into leaner muscle can be of high value to the producers and consumers involved in the market. The MSTN mutation in avian species needs to be investigated further for potential future application in the poultry industry.

AIM

The objective of this study was to investigate the effect of MSTN mutation in avian feed efficiency and fat growth.

METHODS

1. Production and Injection of the Recombinant Adenovirus into the Quail Blastoderm
2. Generation of Germline Chimera and Identification of MSTN Mutation from Offspring
3. Generation of a MSTN Homozygous Mutant Quail Group from Heterozygous Parents
4. Measurement of Feed Consumed and Weight Gained Throughout Maturation
5. Analysis of Feed Conversion and Fat Growth Between Groups

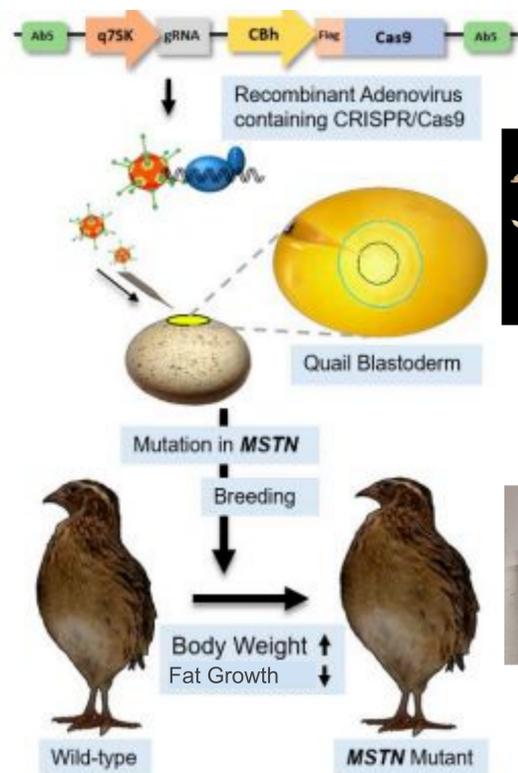


Figure 1. Overview of the study

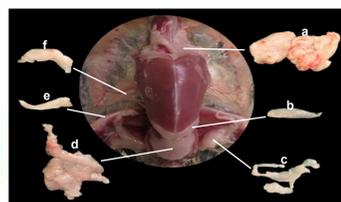


Figure 2. When analyzing fat accretion leg fat (e) and caudal breast fat (b) were measured.

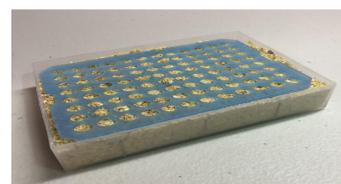


Figure 3. In order to analyze feed conversion with greater accuracy, feed was covered with filter to prevent excess waste.

RESULTS

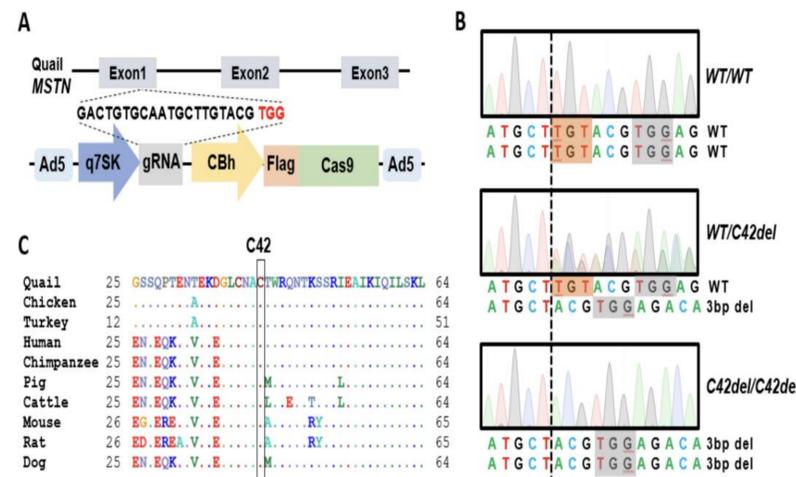


Figure 4. (A) To target quail MSTN, guide RNA was designated on exon 1. (B) Sanger sequencing chromatograms of a target region in the MSTN gene of wild-type (WT/WT), MSTN heterozygous mutant (WT/C42del), and MSTN homozygous mutant (C42del/C42del) quail were compared. (C) Amino acid sequences of MSTN protein after signal peptide were compared across species.

Positive Correlation Between Myostatin Knockout Mutant and Body Weight, Feed Intake, and Muscle to Fat Ratio

- Mutant quail experience significantly higher weight gain and body weight than WT, especially between D30 and D40
- Though feed intake was higher for mutant quail FCR was significantly lower in mutant individuals compared to WT
- MSTN mutant quail showed significantly lower percent of leg and caudal breast fat compared to WT, which contributed to better feed efficiencies

Table 1. Comparison of WG, FI, and FCR among wild type and mutant quail between age D10-40.

	Ages	Male	
		WT	Mutant
WG (g)	D10-D40	72.189 ± 1.371	81.476 ± 1.175***
FI (g)	D10-D40	328.594 ± 6.710	339.027 ± 6.217
FCR	D10-D40	4.563 ± 0.113	4.166 ± 0.076**

Table 2. Comparison leg fat, caudal breast fat, and BW among wild type and mutant quail D10-40 and D40.

	Ages	Male	
		WT	Mutant
Leg fat (%)	D10-D40	0.45009 ± 0.04244	0.29772 ± 0.01814**
CB Fat (%)	D10-D40	0.263 ± 0.02848	0.18264 ± 0.01564*
BW	D40	94.770 ± 1.3542	108.388 ± 1.4346***

All data were presented as means ± SEM. Significant differences were determined using t-test between WT and Mutant. WG, Weight gain; FI, Feed intake; FCR, Feed conversion ratio; CB, Caudal breast; BW, Body weight; n = 10 WT males, 12 mutant males, **, p < 0.01, ***, p < 0.001

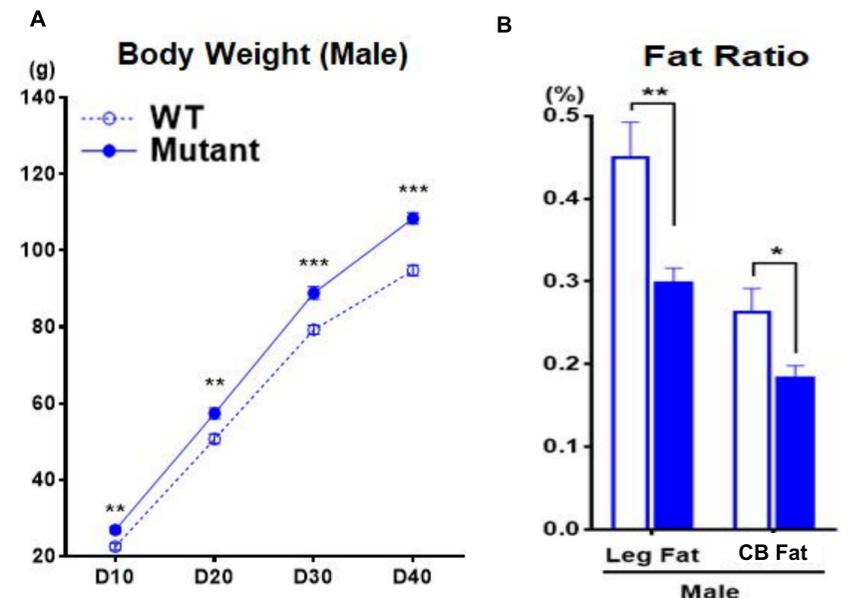


Figure 5. (A) Comparison of body weight between wild type and mutant Japanese quail between D10 and D40. (B) Relative body fat ratio of leg and caudal breast fat between WT and myostatin mutant quail.

CONCLUSION

Myostatin mutation at the C42 region of exon 1 resulted in mutant Japanese quail with higher average body weight for all time frames measures between D10 and D40, with the greatest difference between D30-D40. It was also found that feed intake of mutant quail from D10-20 and from D20-30 were significantly higher compared to those of WT, though from D30-40, no significant difference in feed intake between mutant and wild type lines. Coinciding with these results, analysis of weight gain versus feed intake showed a significantly lower FCR in mutant quail only from D30-40, which then lead to significantly lower FCR in the D10-40 period. However, due to statistical significance in lower leg and caudal breast fat in myostatin mutant quail, the case for higher feed efficiency in mutant lines can be made. This study provides important groundwork for using genetics to help increase protein yield as a food source and appeal to consumers who prefer lean protein.

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