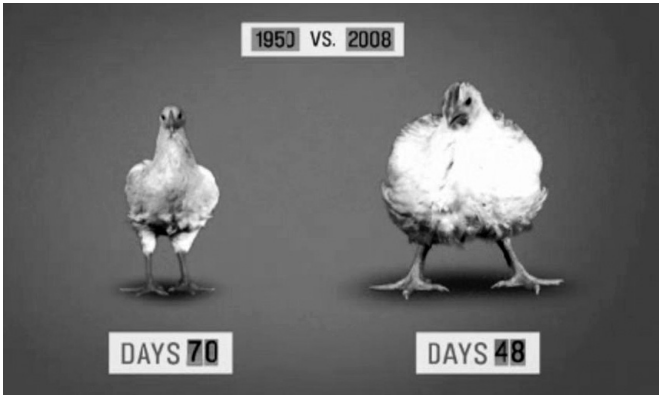


Did Myostatin Play A Key Role In Revolutionizing The Broiler Chicken Industry?

by Vicki Johnson

I had heard 'rumors' back in the late-1970s, when we were importing the first Piedmontese, that the major shift toward increased muscle mass in broiler chickens was initiated in the 1960s by heavy use of a few breeding birds that exhibited a natural mutation. It was simply called 'double muscling' at the time because the myostatin gene had not yet been discovered. We now know the effect is due to a disrupted or 'affected' myostatin gene.

The normal active version of myostatin works to restrict muscle growth.

Piedmontese muscling is due to a natural mutation in the myostatin gene where an amino acid substitution affects / disrupts the myostatin gene normal function – allowing for increasing muscle growth.

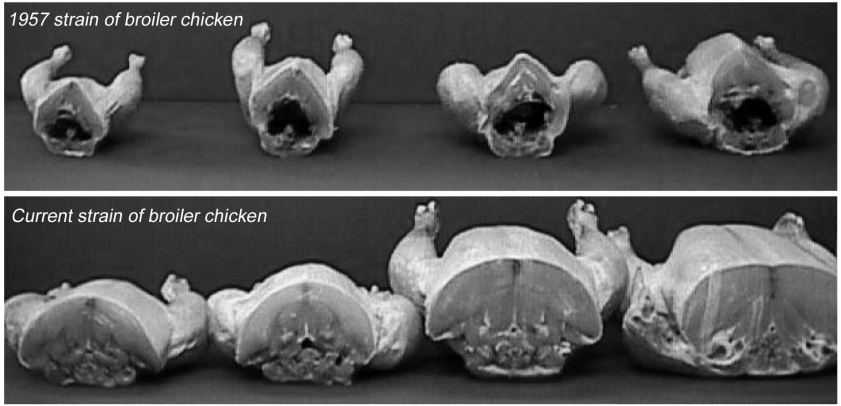
In 2013 at the NAPA Bull Sale Banquet, Lee Leachman gave a brief report on the broiler chicken industry's profound improvement in production from 1957 through 2001 – with credit being given to 'breeders genetic selection toward more muscle' in the development of new broiler bird strains. Leachman concluded that the same selection pressure to improve muscling is actively employed by most US beef breeds.

However, research now confirms the rumors that I heard in the 1970s. Modern chicken, turkey and quail lines actually ***do*** carry the genetics of 'affected' myostatin!

In chickens, there are twelve amino acid substitutions of myostatin and of these, nine substitutions were predicted to affect normal myostatin protein function - meaning, the governor on muscle growth could be turned down or off to varying degrees. [Source: Insilico Analysis of Myostatin Gene in Selected Poultry Species; Article in Journal of Advances in Biology & Biotechnology · March 2018]

Additional research into the body weight of chickens from egg-layer lines versus broiler meat-lines confirms that they carry different variants of affected myostatin. i.e. the meat lines have affected myostatin variants that ***do not exist*** in the layers. [Source: Polymorphism of the myostatin gene and its association with growth traits in chicken; Bhattacharya et al]

(cont. p.19)



From: **Performance changes in poultry and livestock following 50 years of genetic selection:** http://www.lohmann-information.com/content/l_i_41_2006-12_artikel5.pdf

Did Myostatin Play A Key Role In Revolutionizing The Broiler Chicken Industry? (cont from p.14)

While most beef breeds today continue to improve carcass composition using genetic selection toward improved muscling, most breeds simply do not naturally possess an affected myostatin gene variant that they can select towards.
Keep in mind, there are nine known variants of affected / disrupted myostatin in cattle, and they all behave a little differently.
Piedmontese carry the unique C313Y variant of affected myostatin and our registered stock must be dna verified as homozygous for it; NAPA is the first beef breed registry in the world to base animal registration on a specific testable gene. (cont. p.20)

Modern chicken lines used in agriculture demonstrate the dramatic effects of human-directed evolution; broiler chickens selected for rapid growth provide one example. The main objectives of broiler selection are complex but have generally selected for increased muscle mass and decreased time from hatch to market, combined with an increased feed efficiency. The heavy muscled productive chickens pictured here are from the Ross 308 Line, that produced between 11 to 13.6% heavier hot carcass weights than the 1957 Line.

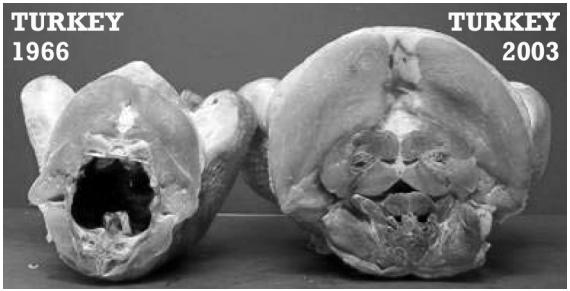
Feed Efficiency and Myostatin:
2019 research comparing High Feed Efficiency to Low Feed Efficiency broiler chickens confirms that the High Efficiency birds had “down regulation” or reduced active myostatin levels compared to Low Efficiency birds.
In other words, the High Feed Efficient bird’s normal myostatin was affected / disrupted resulting in less ‘active’ myostatin in their system.
“Thus, differences in Feed Efficiency in the PedM broiler line in this study could be due in part to different haplotypes of the MSTN (myostatin) gene. Muscle development in the high Feed Efficiency PedM phenotype would be fostered in part by a down regulation of expression of myostatin.”
[Source: Gene Expression Essential for Myostatin Signaling and Skeletal Muscle Development Is Associated With Divergent Feed Efficiency in Pedigree Male Broilers; Lassiter et al.]

Did Myostatin Play A Key Role In Revolutionizing The Broiler Chicken Industry?

The other variants of affected myostatin that do exist in some cattle may be erratic in performance and variable in muscle effect, so selection towards improved muscle in other breeds, by "accidently selecting" towards other variants of the gene, may not provide as profound or as consistent an effect as breeders might hope.

What I mean by "accidently selecting" is building a breeding program based on visual preference given to heavier muscled individuals without actually testing or knowing the genetics behind it. "Accidently selecting" toward heavier muscling may well lead to breeding animals that carry 2 different variants of 'affected' myostatin (being homozygous, the animal would most likely exhibit greatly improved muscle mass themselves) but they are only able to pass forward 1 of the variants to an offspring at a time. Which variant a calf receives can have a significant impact in individual performance, and different offspring inheriting different variants leads to inconsistent results in a calf crop.

For example, the American Angus Association has listed myostatin as a genetic "defect" and the breed is known to carry a low incidence of myostatin variant nt821. Angus that are test verified to carry 1 copy of that gene (heterozygous) will be allowed to retain registry status but will be marked as "carriers", and Angus that are test verified to carry 2 copies of nt821 (homozygous) will have their registry certificate revoked. However, testing for the nine possible gene variants is not mandatory! (If you don't look, you won't find - and you cannot direct or improve, either.)



It seems logical to me that commercial beef producers today should avail themselves of the genetics for improved muscle and carcass composition in the ONLY breed that already has generations-worth of genetic selection towards one consistent myostatin gene variant (C313Y) so they can guarantee immediate carcass performance in one crossbreeding season.
There is no need to reinvent the wheel or spend decades trying to perfect a different variant of 'affected' myostatin.
With the NAPA-registered Piedmontese being homozygous for C313Y, all the commercial cattle producer needs is to use a good NAPA-registered Pied-bull on his current cow herd.

That will move US beef production consistently in the same direction that may have helped to revolutionize the broiler chicken industry.●