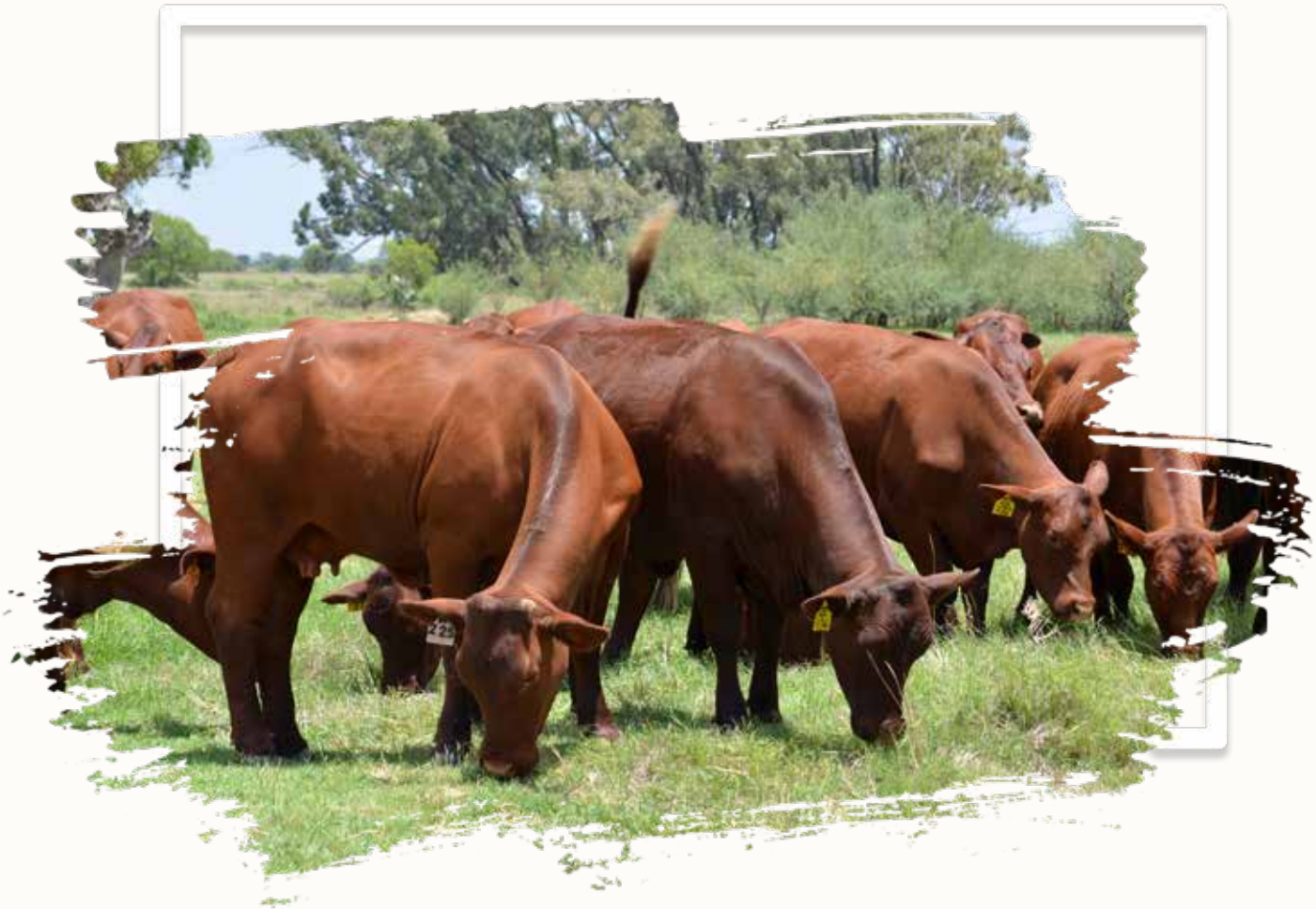

The Inheritance OF THE *Myostatin genes*

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The myostatin protein regulates normal growth and development of muscle in an animal's body. A few mutations in the gene responsible for the synthesis of this protein can disrupt myostatin's ability to function normally, causing muscles to grow uncontrollably, resulting in an animal being double-muscled. The Q204X and nt821 mutations cause a break in the myostatin protein, and affected animals will be double-muscled. The F94L mutation doesn't break the myostatin protein, but rather affects its ability to bind to muscles and ensure normal muscling. Mutations such as the, so called "green genes" appear to have very small or neutral effects when on its own but an

(compounding) additive effect is seen when more than one myostatin mutation is inherited together. This combination may cause undesired effects on traits such as fertility. These genes are currently being tested for their true effect once sufficient numbers of animals have been genotyped for accurate results. These mutations behave recessively, meaning it is almost impossible to identify carriers of double-muscling mutations, as they only appear in an affected calf, which is a result of mating two carrier animals (Figure 4).

All the myostatin mutations are single gene mutations which follow simple mendelian inheritance. In this inheritance pattern the animal can either be a homozygote (two copies of the same variant) or heterozygote (two different variants). One copy of each variant is inherited from the sire and the dam. When an animals is homozygous for the "normal"/ "wild-type"/"free" it is reported as a AA or 0, when an animal is heterozygous for a mutation meaning it has one "normal" and one mutation variant then it is given a Aa or a 1 and if it is homozygous for a mutation meaning it carries two copies of the mutation then it is given a aa or 2 in the report. Figure 1 is a punnett square which shows how the inheritance will occur when an animal homozygous for the normal "wild-type" variant (AA) is mated with another homozygous "wild-type" (AA) animal.

Figure 1 Punnett square of mating between two homozygous "wild-type" animals

		Sire	
		A	A
Dam	A	AA	AA
	A	AA	AA

As seen in Figure 1 all the progeny will have an AA homozygous "wild-type" genotype and will never transfer a myostatin mutation to their progeny. Figure 2 shows the genotype of progeny from a mating between a "wild-type" (AA) (the dam in this case) and a heterozygote carrier (Aa) (the sire).

Figure 2 Punnett square of mating between a homozygous "wild-type" and a heterozygote carrier animal

		Sire	
		A	a
Dam	A	AA	Aa
	A	AA	Aa

As seen in Figure 2 when you mate a carrier of a myostatin mutation (Aa) to a "mutation free" (AA) animal it results in 50% of the progeny being carriers of the mutation (Aa).

Figure 3 shows the mating between a homozygous "wild-type" (AA) (dam) and a homozygous carrier (aa) (sire).

Figure 3 Punnett square of mating between a homozygote "free" and homozygous carrier

		Sire	
		a	a
Dam	A	Aa	Aa
	A	Aa	Aa

As seen in Figure 3 when you mate an AA with an aa animal all resulting progeny will be carriers of the mutations.

Figure 4 shows a mating between two heterozygote carriers. In practice where a carrier bull is used in a herd of carrier cows.

Figure 4 Punnett square of mating between two heterozygote carriers

		Sire	
		A	a
Dam	A	AA	Aa
	a	Aa	aa

Therefore, as can be seen in Figure 4, when you mate two heterozygote carriers for the mutation it results in 25% of the progeny being "mutation free", 50% will be carriers of the mutation and 25% of the progeny will be homozygous affected and present as double muscled.

There is a well-known direct effect on birth weights resulting in dams with double-muscled calves experiencing calving difficulties. Affected animals will also have less fat deposition, which will lead to lower lifetime production, decreased fertility and longevity. It is recommended to ensure that sire bulls are "free" from these mutations, in order to minimize and potentially eradicate incidences of double muscling.