

Fig. 1. Map of chromosome 4 (Archibald et al., 1994). The location starting with S are microsatellite markers. Others are genes. The left map is for females, right for males and combined in the middle. The figures at marker and gene locations are cM.

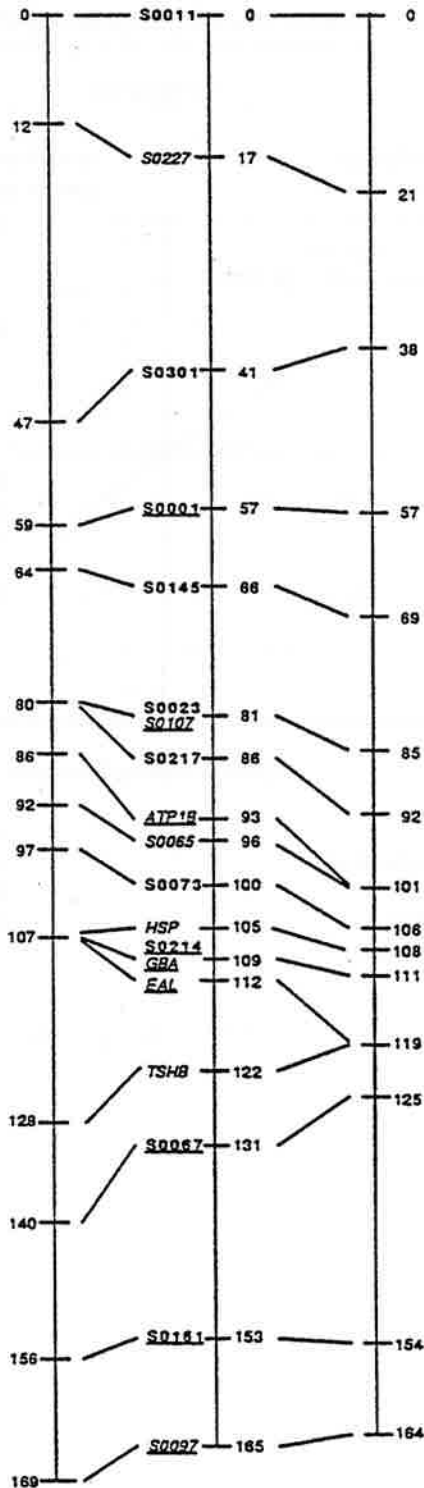


TABLE 1. CARCASS YIELDS AND COMPOSITION BY TREATMENT^a AND WEIGHT^b GROUP

Components	<u>Transgenic</u>		<u>Control</u>		SEM	<u>Significance, P <</u>		
	H	L	H	L		T	W	T x W
<u>Separable carcass</u>								
Lean	65.9	67.0	56.1	65.0	1.4	.01	.01	.02
Fat	16.5	14.3	29.6	20.3	1.8	.01	.01	.05
Bone	17.6	18.7	14.3	14.7	.9	.01	NS	NS
<u>Percentage of carcass</u>								
Shoulder	28.3	26.6	26.8	26.3	.7	NS	NS	NS
Loin	24.8	26.1	28.7	29.0	.9	.01	NS	NS
Ham	28.5	29.1	25.4	26.7	.7	.01	NS	NS
Belly	18.7	18.2	19.0	17.9	1.3	NS	NS	NS
<u>Total carcass fat^c</u>	2.9	10.0	19.1	15.1	2.4	.01	NS	.04

^aTreatment = Transgenic vs. Control

^bWeight group = Heavy (H = 93 kg) vs. Light (L = 54 kg)

^cTotal carcass fat evaluated chemically

TABLE 2. SEPARABLE CARCASS CUT COMPONENTS AND INTRAMUSCULAR FAT BY TREATMENT AND WEIGHT GROUP

Components	Transgenic		Control		SEM	Significance, P <		
	H	L	H	L		T	W	T x W
<u>Separable lean</u>								
Shoulder	65.9	67.0	56.1	65.0	1.4	.01	.01	.02
Loin	60.1	62.4	49.6	54.6	2.4	.01	NS	NS
Ham	74.5	71.4	62.0	70.0	1.5	.01	NS	.01
Belly	61.7	56.4	48.3	52.5	3.0	.02	NS	NS
<u>Separable fat</u>								
Shoulder	16.5	14.3	29.6	20.3	1.8	.01	.01	.08
Loin	17.2	15.3	31.1	26.2	2.8	.01	NS	NS
Ham	13.7	15.9	27.1	20.1	1.9	.01	NS	.04
Belly	23.7	26.0	38.1	29.8	3.8	.04	NS	NS
<u>Separable bone</u>								
Shoulder	17.6	18.7	14.3	14.7	.9	.01	NS	NS
Loin	22.7	22.3	19.3	19.3	1.4	.05	NS	NS
Ham	13.1	12.7	10.9	10.0	.6	.01	NS	NS
Belly	14.6	17.7	13.6	17.7	1.8	NS	.08	NS
<u>Intramuscular fat</u>								
Shoulder	2.6	4.1	11.7	10.8	1.8	.01	NS	NS
Loin	1.6	3.7	10.1	7.5	.8	.01	NS	.02
Ham	1.6	3.1	5.7	4.6	1.3	.08	NS	NS
Belly	3.1	4.2	16.7	13.1	2.6	.01	NS	.05

Figure 1:

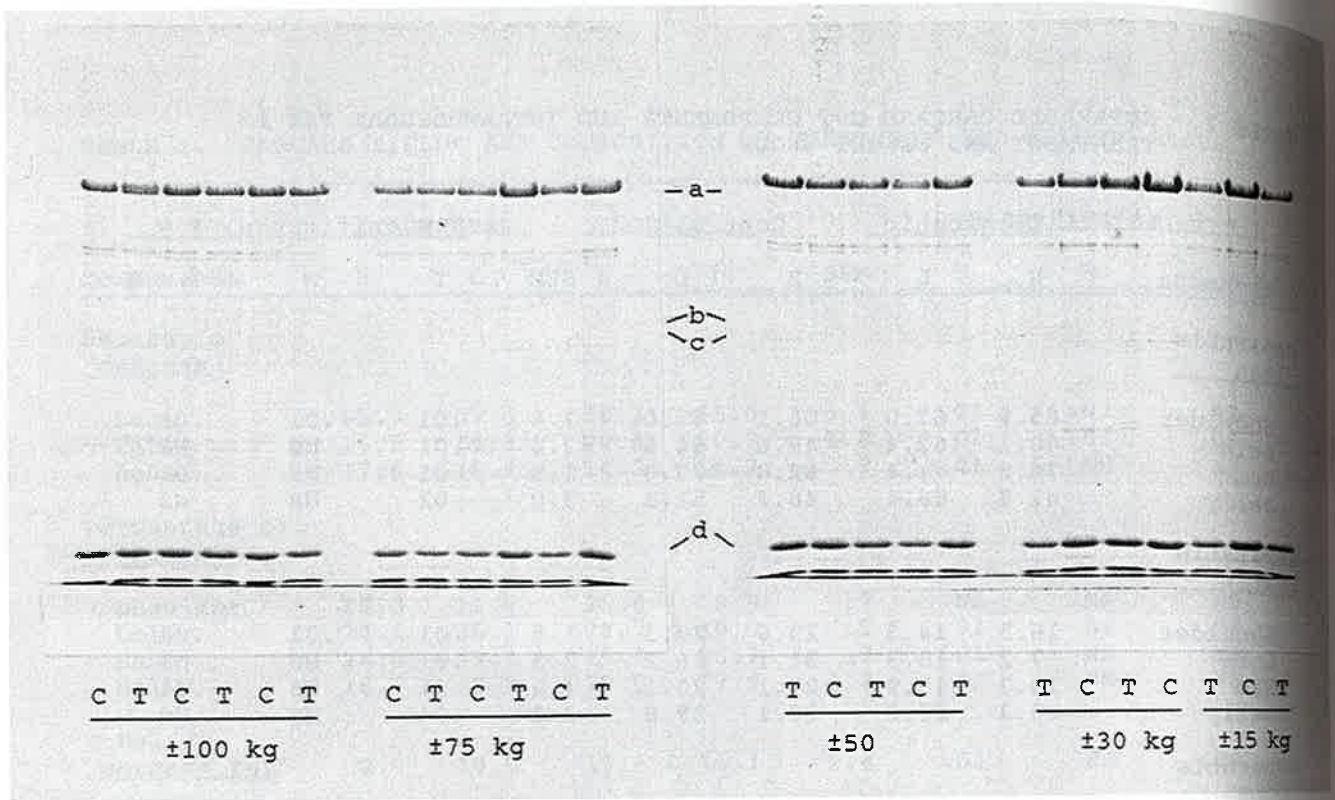


Figure 2:

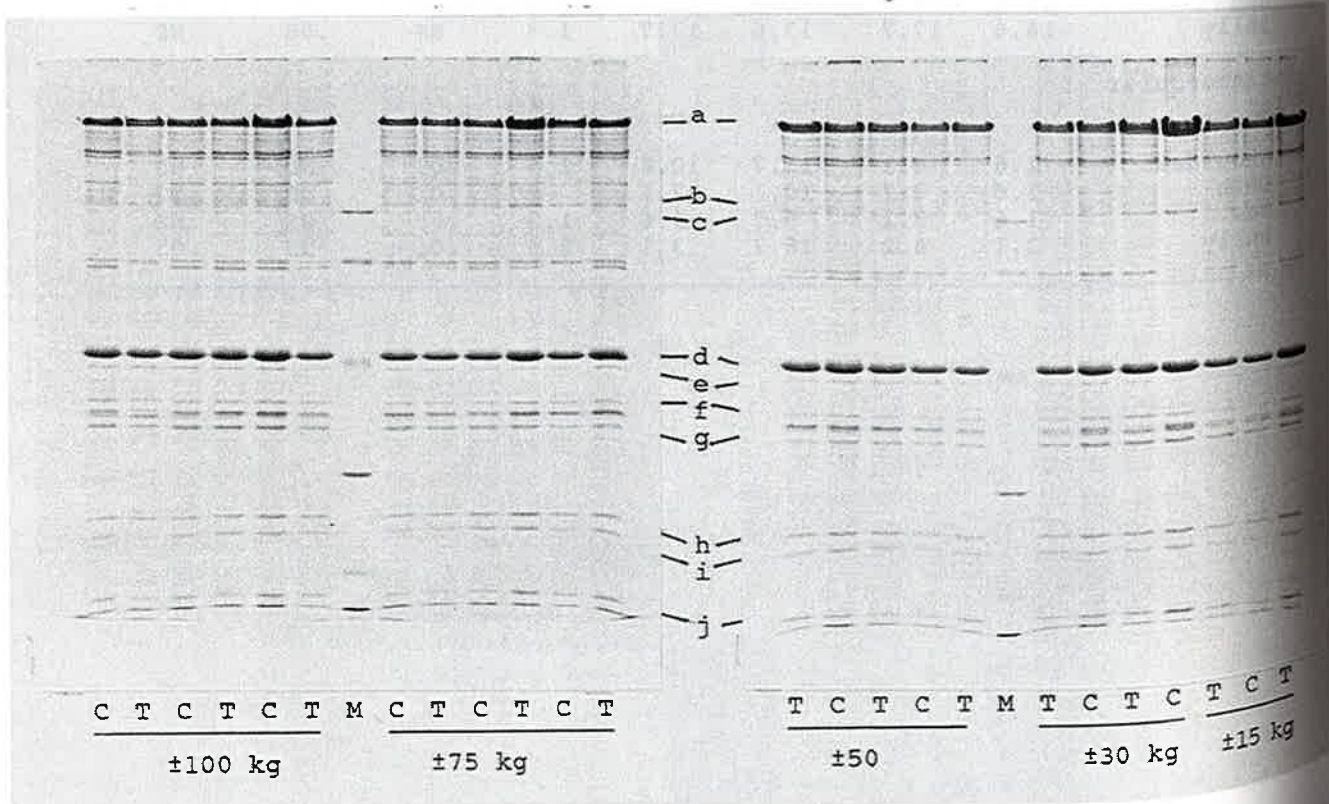


Figure 3:

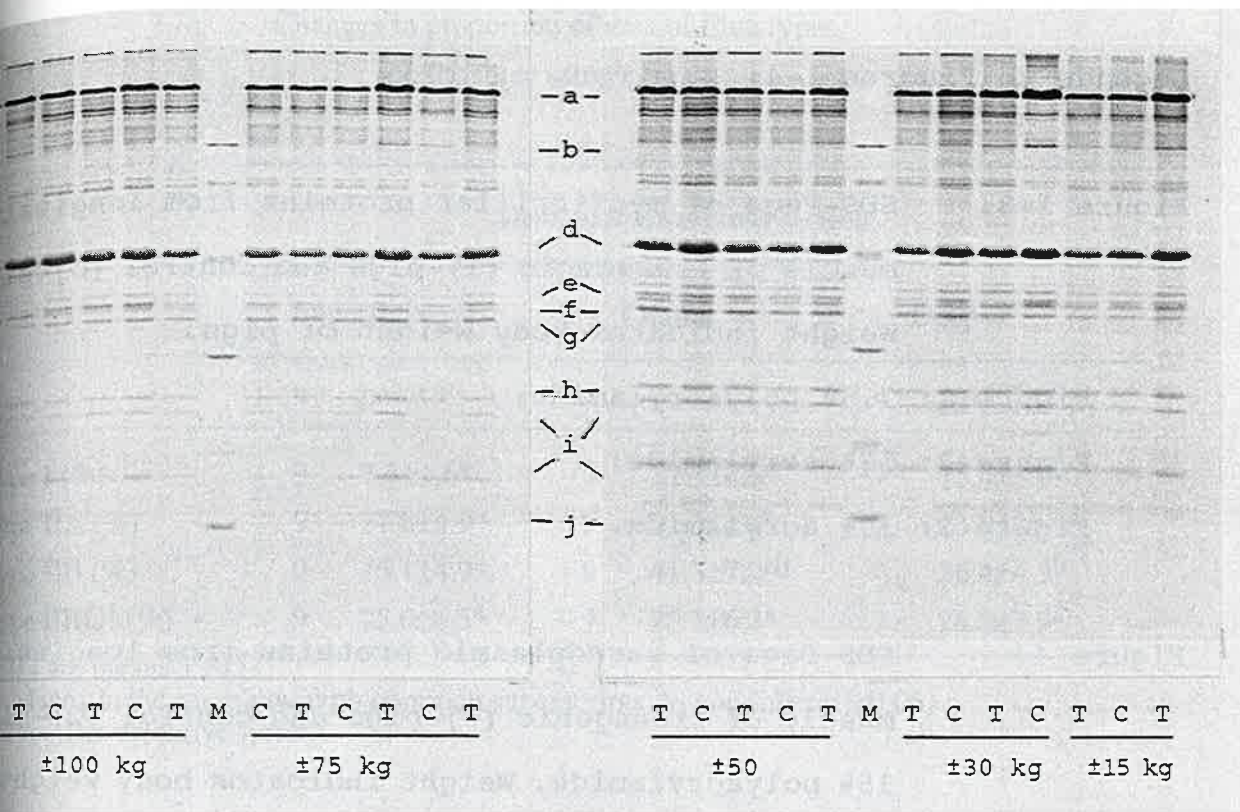
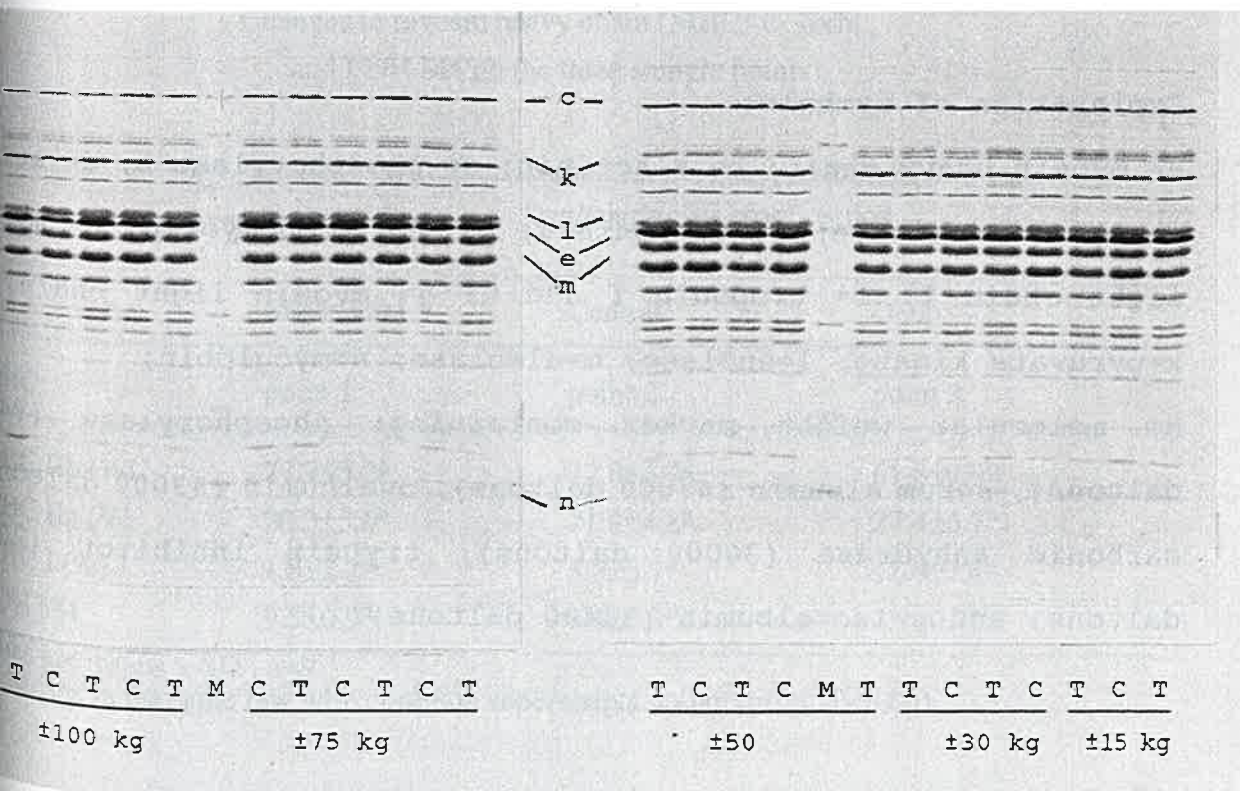


Figure 4:



Legends to Figures 1-4:

Figure 1-3: SDS-Page of myofibrillar proteins from longissimus muscle of transgenic (T)-pigs and control (C)-pigs. Weight indicates body weight of pigs.

Figure 1: 7.5% polyacrylamide;

Figure 2: 12% acrylamide;

Figure 3: 15% acrylamide.

Figure 4: SDS-Page of sarcoplasmic proteins from longissimus muscle of transgenic (T)-pigs and control (C)-pigs; 15% polyacrylamide. Weight indicates body weight of pigs.

Explanation of symbols:

a= myosin heavy chain; b= α -actinin; c= phosphorylase B; d= actin;
e= creatine kinase; f= troponin T; g= α - β tropomyosin; h= myosin
light chain I; i= troponin I and C; j= myosin light chain II;
k=pyruvate kinase; l=enolase; m=aldolase; n=myoglobin;
M= molecular weight marker containing: phosphorylase (94000
daltons), serum albumin (67000 daltons), ovalbumin (43000 daltons),
carbonic anhydrase (30000 daltons), trypsin inhibitor (20100
daltons) and α -lactalbumin (14400 daltons).

Table 1
Changes in proportion of area of fibre types
on the three sample points

<i>Semitendinosus</i> cross section					
Superficial		Central		Deep	
n	point 1	n	point 2	n	point3
Type I (%)	9 7.3±1.6 ^a	9 12.3±4.6 ^a	7 15.4±3.0 ^b		
Type IIA (%)	9 21.8±4.9 ^a	9 21.3±6.4 ^a	7 23.9±23.9 ^a		
Type IIB (%)	9 46.1±4.9 ^a	9 41.3±7.2 ^{ab}	7 36.4±4.3 ^b		
Type IIBO ¹ (%)	9 25.0±6.5 ^a	9 25.1±6.3 ^a	7 24.3±7.9 ^a		

Values are means ± SD :

ab - Means in the same row with common superscripts do not differ (P<0.05)

1 - Type IIB oxidative fibres

Table 2
Changes in myosin heavy chain (MHC) isoform
and LDH M4 on the three sample points

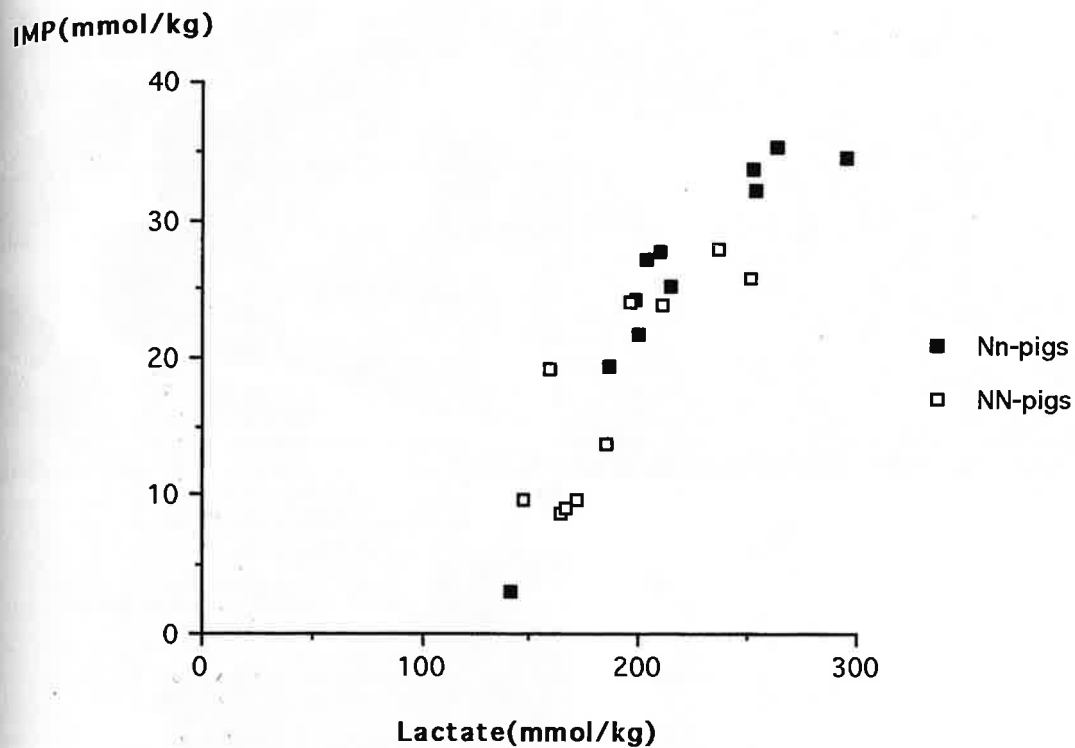
<i>Semitendinosus</i> cross section			
	Superficial	Central	Deep
MHC	point 1	point 2	point 3
MHC IIB (%)	77.7±5.2 ^a	72.8±6.4 ^a	61.8±9.9 ^b
MHC IIA (%)	20.3±5.6 ^a	21.8±4.8 ^a	27.4±6.7 ^b
MHC I (%)	1.8±2.2 ^a	5.3±3.7 ^a	11.8±7.4 ^b
LDH M4	9.5±0.9 ^a	8.4±0.9 ^b	7.9±0.7 ^b

Values are means ± SD ; n=9

ab - Means in the same row with common superscripts do not differ (P<0.05)

	STUDY A at exsanguination			STUDY B 30 min after exsanguination		
	Hal ^N Hal ^N (n=10)	Hal ⁿ Hal ⁿ (n=10)		Hal ^N Hal ^N (n=10)	Hal ^N Hal ⁿ (n=11)	
Pigs with giant fibres	0	3		9	10	
ATP	17,5±3,0	9,3±4,8	***	4,8±4,0	3,0±4,4	n.s.
ADP	5,9±1,3	5,4±1,3	n.s.	4,7±3,1	2,2±2,4	n.s.
AMP	1,8±0,9	3,7±2,6	*	0,7±0,7	0,4±0,6	n.s.
IMP	2,1±1,4	9,3±3,5	***	17,2±7,8	26,2±9,6	*
Lactate	90±47	165±29	***	189±35	220±43	n.s.
Glycogen	232±78	136±57	**	94±49	103±48	n.s.
pH	6,62±0,19	6,32±0,07	***	6,11±0,22	5,98±0,22	n.s.
EEL	21,7±1,8	27,0±3,7	**	16,4±2,8	23,5±5,2	**

Table 1. Metabolic response and pH immediately at exsanguination (study A) and 30 min after exsanguination (study B) in *M. longissimus dorsi* in halothane gene free pigs (Hal^NHal^N) and in carriers of the halothane gene (HalⁿHalⁿ, Hal^NHalⁿ). ATP, ADP, AMP, IMP, lactate and glycogen concentrations are expressed in mmol/kg. EEL shows the surface reflectance values. Significance level (Student's unpaired t-test) between carriers and non-carriers of the halothane gene in study A and B is indicated (n.s.=not significant, *** =p<0.001, **=p<0.01 and *=p<0.05).



Giant fibres

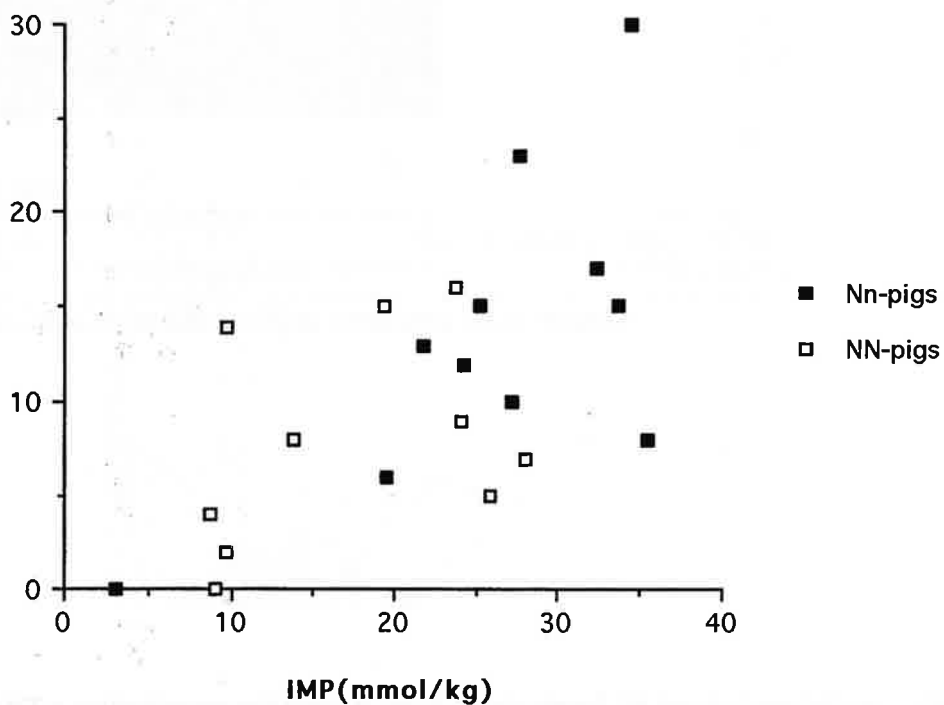


Figure 1. The number of giant fibres revealed in the NADH-stain on a circular screen (19 cm diameter) of a Visopan-microscope (55x) in relation to IMP concentration. IMP concentration in relation to lactate concentration. Samples were taken from 21 pigs (*M. longissimus dorsi*) 30 min after exsanguination.

Nn-pigs = carriers of the halothane gene, NN-pigs = non-carriers of the halothane gene

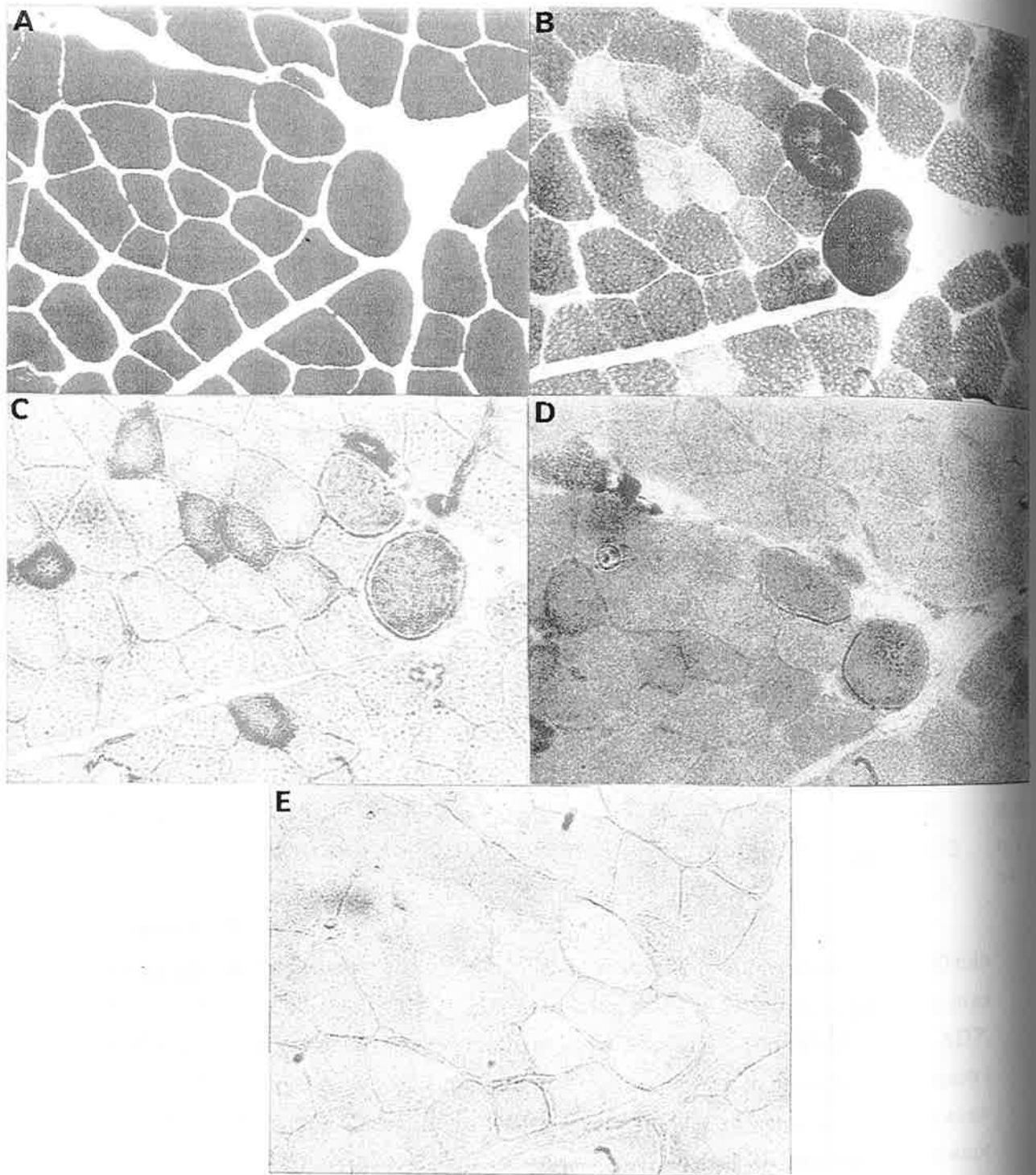


Figure 2. Serial sections of *M. longissimus dorsi* stained for myofibrillar ATPase after , alkaline preincubation (pH=10.3) = (A), acid (pH=4.6) = B, NADH-tetrazolium reductase = C, Sudan black B =D and PAS.=E The muscle sample was taken 30 min after exsanguination. Note the two giant fibres in all sections.

Figure 1. General outline of the QUA-SI-PORK model.

Figure 2. General outline of relevant processes taking place before slaughter.

Figure 3. A simple example of regulation.

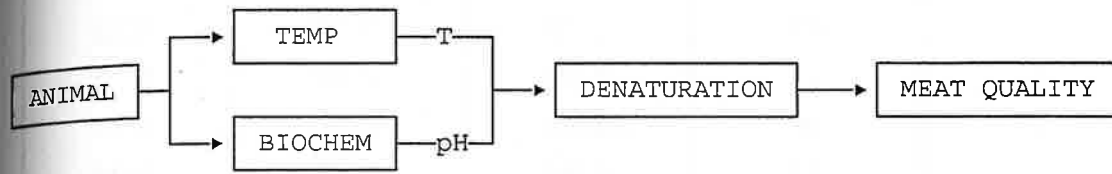


Figure 1. General outline of the QUA-SI-PORK model.

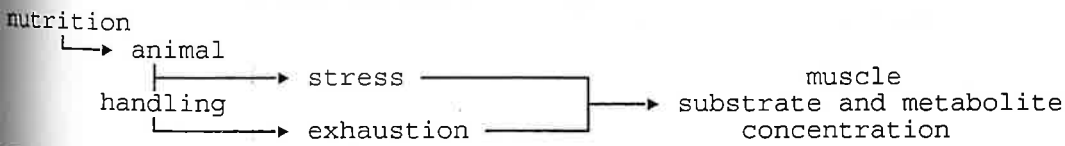


Figure 2. General outline of relevant processes taking place before slaughter.

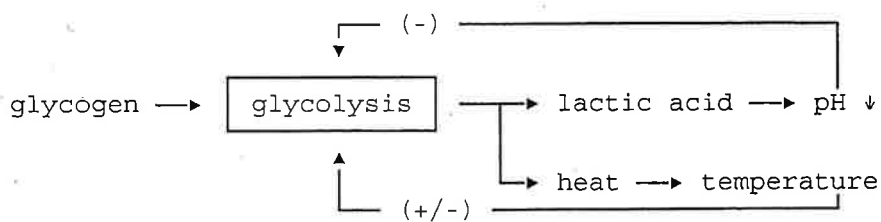


Figure 3. A simple example of regulation.

Temperature	A	Log B	c	s ²
1	44	1.71	-10.48	0.93
4	43	1.22	-1.50	0.25
5	19	1.66	-7.82	0.16
7	16	2.83	-2.26	0.15
10	13	4.64	-3.53	0.04
15	16	4.99	-4.86	0.05
22	20	7.26	-6.82	0.03
29	27	6.89	-7.95	0.12
37	37	2.50	-5.77	0.21

Table 1. The mean values of A, B and c and the variance s² for each temperature.

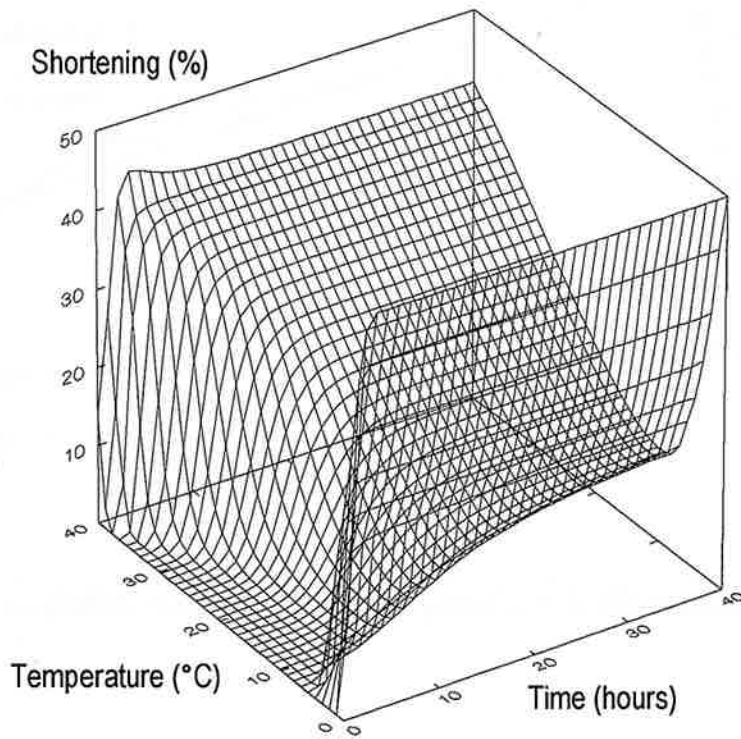
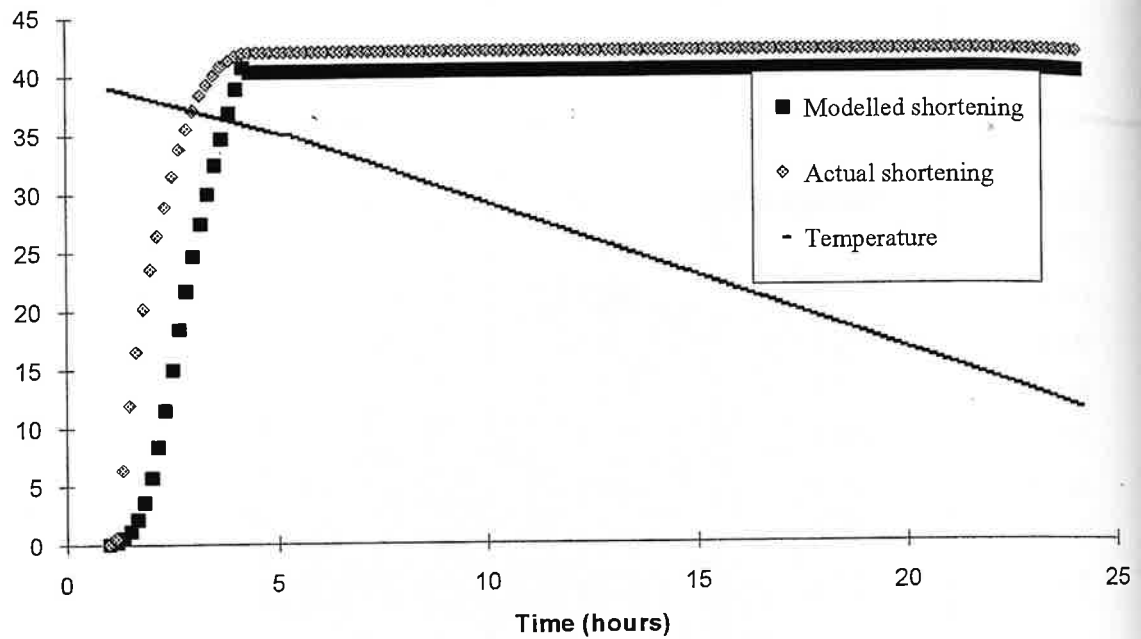


Figure 1. Modelled shortening versus time and temperature for *M. semimembranosus*.

Temperature (°C), Shortening (%)

Figure 2. Modelled and actual shortening versus time for slow chilling of *M. semimembranosus*.

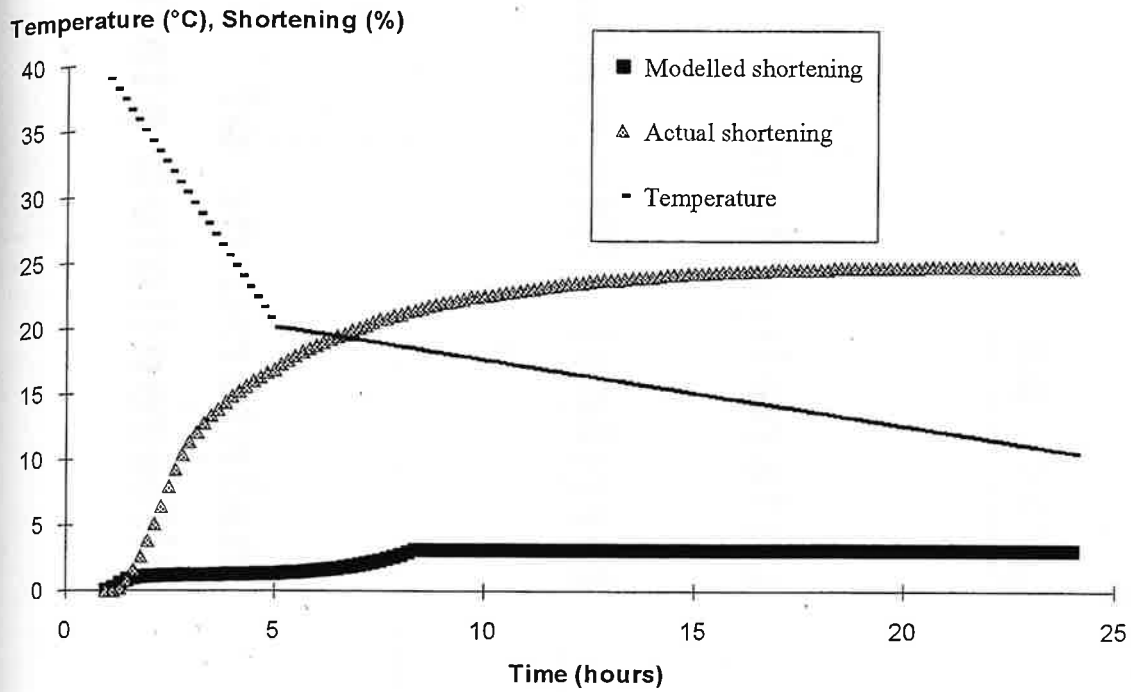


Figure 3. Modelled and actual shortening versus time for fast chilling of *M. semimembranosus*.

MYOGENESIS

tissues genes

MESODERM

myf-5



Myf-4



Myf-3



Myf-6



MYOFIBERS

MYOFIBERTYPES

process

determination

terminal differentiation

**maintenance of
differentiated state**

Legend tot the figure

Figure 1

The myf-5 gene is activated in mesoderm cells in the early embryo resulting in determination of the cells to the myogenic cell lineage. The transiently activated myf-6 gene may participate in this process. The action of myf-3 and myf-4 induces terminal myogenic differentiation in the cells. Myf-6 is activated again in terminally differentiated cells, suggesting its involvement in maintenance of the differentiated state.