METABOLIC PROFILE IN DIFFERENT TISSUES AND POSTMORTEM GLYCOGENOLYSIS IN SKELETAL MUSCLE OF PIGS WITH DIFFERENT *PRKAG3* GENOTYPES

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Introduction

Many pigs of Hampshire origin carry the *PRKAG3* mutation (previously known as the RN^- mutation) which results in an almost twofold increase in glycogen content in skeletal muscle (Monin et al., 1992, Estrade et al., 1993, Enfält et al., 1997). This has a large impact on meat characteristics such as pH, water content, technological yield and lean meat content (Monin et al., 1987, Monin et al., 1992, Fernandez et al., 1991, Lundström et al., 1996, Enfält et al., 1997). The *PRKAG3* mutation has beneficial effects for lean meat content but detrimental effects for water-holding capacity and processing yield. The aim of this study was to investigate if the *PRKAG3* mutation in pigs has any influence on the metabolic profile in muscle, liver and heart and if the mutation has any influence on rate of glycogenolysis and lactateproduction in skeletal muscles postmortem.

Material and methods

Eight clinically healthy pigs (Yorkshire/ Swedish Landrace x Hampshire) were studied. Four pigs were carriers and four were non-carriers of the *PRKAG3* mutation as shown by DNA analyses on the blood (Milan et al., 2000). The pigs were housed in pens (4 pigs/pen) with straw as bedding. They were fed a commercial finisher diet *ad lib* and had free access to water. The pigs were killed with a captive bolt and exsanguinated when they weighed 67 ± 13 kg . Samples of the heart, liver, *m. biceps femoris*, and *m. longissimus dorsi* were collected, frozen in liquid nitrogen and stored at minus 80° C until analyzed. Glycogen, enzyme activities, ATP, G-6-P and lactate concentrations were analysed as previously described by Lowry and Passonneau 1973, Essen et al.1980, and Essen-Gustavsson et al., 1984. Differences between carriers and non-carriers of the *PRKAG3* mutation were tested for statistical significance using the Students unpaired T-test.

Results and discussion

Enzyme activities and glycogen concentrations in heart, liver and muscle are shown in table 1. Glycogen levels were higher in skeletal muscles of carriers compared with non-carriers of the *PRKAG3* mutation. This was expected due to the mutation and is in agreement with several earlier studies (Monin et al., 1992, Estrade et al., 1993, Enfält et al., 1997, Essen-Gustavsson et al., 2005, Ylä-Ajos et al., 2007). In *m. biceps femoris* oxidative capacity was higher in carriers of the *PRKAG3* mutation than in non-carriers as shown by higher CS and HAD activities. In *m. longissimus dorsi* , HAD activities were higher in the carriers indicating a higher capacity for fat oxidation and LDH activities were lower indicating a lower glycolytic capacity. No differences were seen in glycogen concentration or enzyme activities between genotypes in heart and liver. These findings are consistent with the observation that the *PRKAG3* gene shows a very restricted tissue distribution (Lebret et al., 1999, Mahlapuu et al., 2004). It is primarily expressed in white skeletal muscle and no significant gene expression has been documented in the heart or liver. That the *PRKAG3* mutation has an influence also on the metabolic profile in different skeletal muscles is in agreement with one earlier study (Lebret et al., 1999).

Table 1. Mean values ± S.D. for citrate synthase (CS), 3-OH-acyl CoA dehydrogenase (HAD) and lactate dehydrogenase (LDH) activities and glycogen content in *m longissimus*, *m. biceps femoris* , heart and liver. * denotes a significant difference ($p < 0.05$) between carriers and non-carriers of the *PRKAG3* mutation.

	CS mmol/kg/min	**HAD** mmol/kg/min	**LDH** mmol/kg/min	**Glycogen** mmol/kg
M.longissimus				
Carrier	9.7 ± 0.4	26.6 ± 3.4*	1771 ± 130*	468 ± 37*
Non-carrier	8.2 ± 2.9	17.5 ± 0.8	2094 ± 54	287 ± 26
M.biceps				
Carrier	32.5 ± 1.2*	32.5 ± 2.5*	1527 ± 177	481 ± 20*
Non-carrier	23.2 ± 5.6	20.7 ± 3.3	1574 ± 369	303 ± 32
Heart				
Carrier	140 ± 12	132 ± 6	353 ± 19	27 ± 8
Non-carrier	152 ± 9	133 ± 2	353 ± 26	24 ± 9
Liver				
Carrier	17 ± 1	88 ± 21	176 ± 18	888 ± 114
Non-carrier	15 ± 2	89 ± 11	182 ± 11	816 ± 249

The amount of glycogen degraded in *m. longissimus dorsi* and *m. biceps femoris* from 10min to 24h postmortem did not differ between carriers and non-carriers (table 2). This is in agreement with results from a recent study (Ylä-Ajos et al., 2007). The amount of ATP degraded in *m. longissimus dorsi* and the amount of lactate and G-6-P produced from 10min to 24h postmortem did not differ between carriers and non-carriers (table 2). Low ATP levels in muscle were associated with high G-6-P and lactate levels. Even if the carriers of the *PRKAG3* mutation have higher glycogen content and a different oxidative and glycolytic capacity in the muscles compared to the non-carriers this does not seem to influence the rate of glycogenolysis and lactate production postmortem.

Table 2. Mean values ± S.D. for ATP, G-6-P, lactate and glycogen concentrations (mmol/kg) in *m. longissimus dorsi* and glycogen concentration in *m. biceps femoris* at different times post-mortem
* denotes a significant difference ($p < 0.05$) between carriers and non-carriers of the *PRKAG3* mutation.

	ATP *m. longissimus dorsi*	**G-6-P** *m. longissimus dorsi*	**Lactate** *m. longissimus dorsi*	**Glycogen** *m. longissimus dorsi*	**Glycogen** *m. biceps femoris*
10 min					
Carrier	19.7 ± 5.6	14 ± 13	134 ± 31	475 ± 36*	481 ± 20*
Non-carrier	17.0 ± 3.1	8 ± 3	127 ± 28	294 ± 16	303 ± 26
60 min					
Carrier	16.7 ± 7.5	12 ± 9	149 ± 60	463 ± 40*	489 ± 46*
Non-carrier	16.8 ± 5.6	7 ± 11	133 ± 46	304 ± 15	310 ± 57
120 min					
Carrier	13.9 ± 8.3	16 ± 8	176 ± 31	432 ± 40*	489 ± 32*
Non-carrier	10.0 ± 7.5	13 ± 16	191 ± 65	250 ± 64	245 ± 42
24 hour					
Carrier	0.5 ± 0.6	48 ± 18	304 ± 82	310 ± 51*	364 ± 32*
Non-carrier	0.9 ± 0.9	46 ± 16	295 ± 22	152 ± 21	122 ± 18

Conclusion

The *PRKAG3* mutation influences glycogen content and the metabolic profile in skeletal muscles but not in heart and liver. The rate of glycogenolysis during 24 h post mortem is not influenced by the *PRKAG3* mutation.

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