Porcine Muscle Glycogen Structure and its Association with Other Muscle Properties.*† (27999)

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(Introduced by P. H. Phillips)
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The quantity of glycogen stored in muscle at time of death is important in determining post-mortem chemical and physical properties of the muscle only if: (a) the glycogen is available or accessible for degradation(1) and (b) the enzymes are not inhibited by a decreasing pH(2). The rate at which the glycogen is broken down post-mortem has been found to have a more pronounced influence on the properties of the muscle than total amount of glycogen present at time of death (3).

Bovine muscles with differing functional requirements contained glycogen with various branching characteristics(4). Briskey and Lawrie(5) also reported that glycogen samples isolated from different bovine muscles at pre-rigor and post-rigor periods were broken down at unequal rates by phosphorylase. Larner *et al.*(6) found that muscle phosphorylase preferentially degraded the larger glycogen molecules while Stetten and Stetten(7) discovered that glycogen build up was rapid on glycogen molecules of small size. Sayre *et al.*(8) found marked differences in the amount of muscle glycogen present in the muscle of pigs of different breeds.

However, comparisons between breeds showed no tendency for elevated glycogen stores to result in a rapid rate of post-mortem glycolysis. These findings indicated that the branching characteristics of the glycogen molecule might differ under various nutritional or hereditary influences and might be a factor in regulation of the rate and amount of post-mortem glycolysis.

Experimental and results. The external and internal chain lengths of glycogen(9,10) were determined on samples isolated(11) from the *longissimus dorsi* of pigs, from 3 breeds, (Hampshire, Chester White, Poland China) which were previously found to differ in amount of glycogen, rate of post-mortem glycolysis(12) and physical properties of the post-rigor muscle(13). Fig. 1 shows the average amount of muscle glycogen in the 3 breeds.

The average external chain lengths of glycogen from muscles frozen at various times post-mortem tended to decrease by approximately one glucose residue during post-mortem glycolysis (Table I). The chain lengths of Hampshire glycogen sampled immediately after death tended to be longest and those of the Poland China glycogen shortest for the 3 breeds. Although the branching characteristics of the Chester White glycogen were similar to those for Hampshire glycogen at time of death the chain lengths of Chester White glycogen appeared to shorten more rapidly during post-mortem glycolysis. There also seemed to be a marked shortening of internal chain lengths in the Chester White glycogen samples.

Sucrose feeding (Table II) which has been

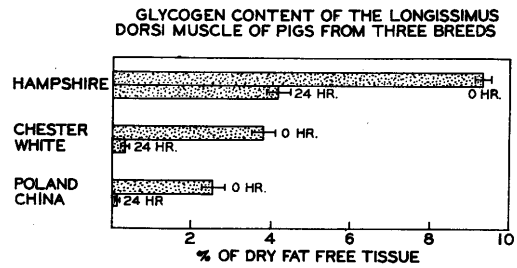

FIG. 1. 0 hr = sample frozen immediately after exsanguinations; 24 hr = sample obtained 24 hr post-mortem. ⊢⊣ indicates ± standard error of mean.

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TABLE I. Breed Comparison of Branching Characteristics of Porcine *Longissimus Dorsi* Glycogen.

	Avg external and internal chain length*					
	0 hr†		1 hr		24 hr	
Breed	External	Internal	External	Internal	External	Internal
Hampshire	10.1 (6)‡	2.3	10.0 (8)	2.7	9.5 (6)	2.5
Chester White	10.0 (5)	3.1	8.9 (5)	1.2	7.1 (2)	1.0
Poland China	9.2 (8)	1.7	9.3 (8)	2.4	8.7 (2)	2.8

* Chain length expressed as No. of glucose residues.
† Time post-mortem.
‡ No. of observations.

TABLE II. Influence of Nutritional Status on Branching Characteristics of Porcine *Longissimus Dorsi* Glycogen.

	Avg external and internal chain length*					
	0 hr†		1 hr		24 hr	
Treatment	External	Internal	External	Internal	External	Internal
50% sucrose feeding (2 wk)	10.4 (5)‡	2.5	10.0 (4)	2.5	9.3 (2)	2.4
No. sucrose fasted 70 hr	9.0 (4)	1.6	8.4 (5)	1.3	—	—

* Chain length expressed as No. of glucose residues.
† Time post-mortem.
‡ No. of observations.

shown to elevate the muscle glycogen stores (14), tended to lengthen both the external and internal chain length of muscle glycogen when compared to glycogen from the muscle of animals which were fasted 70 hr prior to slaughter. These findings suggest that glycogen structure may be altered by the nutritional status of the animal as well as by genetic influences.

The data also suggest that an alteration in the external chain length of the glycogen molecule may influence the rate at which the molecule is degraded. In all cases the external chain length was shortened during post-mortem glycolysis. The Chester White pigs, which exhibited the slowest rate of post-mortem glycolysis had the highest rate and the greatest amount of alteration in the structure of the glycogen molecule. Conversely the Poland China pigs had the most rapid rate of glycolysis, while the chain lengths of the muscle glycogen were only slightly shortened during the entire post-mortem glycolytic period. These findings tend to support the hypothesis of Lawrie, Manners and Wright(4) that a decreased rate of anaerobic glycolysis may be due, in part, to an alteration in the molecular structure of muscle glycogen.

Summary. The average chain length of glycogen from porcine muscles decreased by approximately one glucose residue during post-mortem glycolysis. There was however, a more severe decrease in both external and internal chain lengths of the glycogen from the Chester White muscles which had a slow rate of anaerobic glycolysis. Sucrose feeding tended to lengthen both the external and internal chain lengths of the muscle glycogen and accelerated the rate of anaerobic glycolysis. These findings suggest that glycogen structure may be altered by nutritional as well as genetic influences and, in both instances, may influence the rate at which the molecule is degraded.

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Effect of D-Aldosterone on Cation Content of Isolated Rabbit Atria* (28000)

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Few substances are capable of stimulating active Na-K transport across cell membranes. Perhaps the most physiologically important of these agents is the hormone aldosterone (1). This naturally occurring steroid has been shown to stimulate active Na-K transport in a variety of tissues including isolated toad bladder(2), frog skin(3), renal tubule (4) and skeletal muscle and brain(5,6). Equivocal effects have been noted with respect to RBC ion transport, however(7,8).

Since aldosterone has been implicated in production of myocardial edema and derangements in ion metabolism in certain cardiac disease states(9), we undertook experiments to determine whether or not the biologically active d-form of aldosterone(10) was capable of modifying the Na or K content of cardiac tissue. Also, since aldosterone given *in vivo* has been shown to enhance K accumulation in skeletal muscle(5,6), we were particularly interested in determining whether the d-form of the steroid could prevent the K loss from isolated left atrial tissue which occurs with prolonged electrical stimulation *in vitro*. Thus, as a working hypothesis, we are questioning whether this steroid is capable of stimulating active Na-K transport in the isolated heart as it has been claimed to do in other tissues(2-6).

Materials and methods. Left atria from hearts of White New Zealand rabbits were studied. The animals were sacrificed by a blow on the head and the heart quickly removed and placed in a dissecting dish containing oxygenated bicarbonate-Ringer solution(11). The left atrium was dissected free from the ventricles and all visible fat, connective and vascular tissue removed. Experiments were divided into 5 groups: *Group I.* Freshly dissected tissue was blotted on filter paper to remove adhering solution and then immediately placed in a drying oven at 110°C until a constant dry weight was obtained. Total tissue K and Na were determined from nitric acid digests of the atria using a flame photometer with an internal lithium standard. *Group II.* After atria were dissected from the ventricles, they were placed in a muscle bath at a constant resting tension for 260 minutes. The muscle chamber was constantly perfused with Ringer's solution at 30°C. At the end of this period, the atria were removed from the bath and prepared for K and Na analysis as in Group I. *Group III.* Tissue prepared and handled in same manner as Group II except that the atria were stimulated at a constant rate of 200 beats/min for the entire time the tissue was *in vitro*. *Group IV.* Same as Group III except that 200 minutes after placing in the muscle chamber, a d-aldosterone (1.1×10^{-6}M)-Ringer solution was substituted for the normal Ringer perfusing the atria. Drug action continued for one hour, then the tis-

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