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On the Post-mortem Breakdown of Glycogen and ATP in Skeletal Muscle*)

SUMMARY

Bovine longissimus and sternomandibularis and rabbit leg muscles were sampled for the determination of all glycolytic metabolites from glycogen to lactate including glucose and glycerol-3-phosphate at various times from shortly after death to 24 hours post mortem. Previous work had shown a nonstoichiometric conversion of glycogen to lactate and in this work we studied the disappearance of glycogen into both glycolytic intermediates and lactate. The results support the nonstoichiometric disappearance of glycogen with a mean value of 83.55 + 13.36 % for the conversion of glycogen to intermediates and to the lactate and glucose endproducts. The total concentration of glycolytic pathway metabolites decreased during post mortem glycolysis indicating that the loss took place through a disappearance out of the normal glycolytic pathway. From the flux of the glycolytic metabolites during post mortem glycolysis it was very evident that the enzyme phosphofructokinase plays a major role in controlling the rate and extent of the post mortem glycolytic process.

Thin slices of rabbit skeletal muscle were incubated in different media immediately after death. The amounts of ATP and its metabolites in the tissue after different times of incubation were determined in order to find out which type of ATPase is responsible for the breakdown of ATP in muscle post mortem. K^+ and Na^+ in concentrations of 0.1 to 0.15 M do not activate the ATPases. No inhibiting effect of ouabain and quinidine sulfate was observed. 0.05 M $MgCl_2$ showed a strong stimulating effect on the ATP hydrolysis. The results indicate that neither membrane nor sarcoplasmatic reticulum ATPases but that most likely myofibrillar ATPases predominate in hydrolyzing ATP in skeletal muscle post mortem.

*) Abbreviations: G-1-P = glucose-1-phosphate; G-6-P = glucose-6-phosphate; F-6-P = fructose-6-phosphate; FDP = fructose-1,6-diphosphate; DAP = dihydroxyacetone phosphate; GAP = glyceraldehyde-3-phosphate; PEP = phosphoenolpyruvate; 2-PGA = 2-phosphoglyceric acid; 3-PGA = 3-phosphoglyceric acid; G-3-P = glycerol-3-phosphate; PFK = phosphofructokinase; ATP = adenosine triphosphate; ADP = adenosine diphosphate; AMP = adenosine monophosphate; IMP = inosine monophosphate; ATPase = adenosine triphosphatase; long. = longissimus muscle; stern. = sternomandibularis muscle; p.m. = post mortem

ZUSAMMENFASSUNG

Im Longissimus dorsi- und Sternomandibularis-Muskel des Rindes sowie in der Beinmuskulatur des Kaninchens wurden zu verschiedenen Zeitpunkten nach dem Schlachten (bis 24 Stdn post mortem) alle Metaboliten der Glykolyse einschließlich Glykogen, Lactat, Glucose und Glycerin-3-phosphat bestimmt. Frühere Arbeiten hatten eine nichtstöchiometrische Umwandlung von Glykogen in Lactat ergeben; in der vorliegenden Arbeit wurde der Abbau von Glykogen zu Lactat und den Intermediärprodukten der Glykolyse studiert. 83,5 ± 13,4 % des Glykogens waren nach 24 Stdn in glykolytische Intermediärprodukte sowie in Lactat und Glucose-Endprodukte umgewandelt. Die Gesamtkonzentration an Metaboliten des glykolytischen Abbaues nahm während der Glykolyse post mortem ab, ein Zeichen dafür, daß neben der Glykolyse auch andere Abbauwege in Betracht kommen. Aus der Veränderung der glykolytischen Metaboliten post mortem ergab sich, daß das Enzym Phosphofructokinase Geschwindigkeit und Ausmaß der postmortalen Glykolyse im Muskel entscheidend beeinflußt.

Dünne Schnitte der Skeletmuskulatur des Kaninchens wurden unmittelbar post mortem in verschiedenen Medien inkubiert; der Einfluß der Inkubationszeit auf den Gehalt des Gewebes an ATP und dessen Abbauprodukten post mortem wurde studiert, um festzustellen, welche Art von ATPase für den ATP-Abbau im Muskel post mortem verantwortlich ist. K^+ und Na^+ aktivierten in Konzentrationen zwischen 0,10 und 0,15 M die ATPasen nicht. Eine hemmende Wirkung von Ouabain und Chinidinsulfat wurde nicht beobachtet. 0,05 M $MgCl_2$ und 0,03 M $CaCl_2$ übten einen stark aktivierenden Effekt auf die ATP-Hydrolyse aus. Diese Resultate sprechen dafür, daß weder Membran-ATPasen noch die ATPase des sarkoplasmatischen Reticulums, sondern myofibrilläre ATPasen für den ATP-Abbau post mortem von entscheidender Bedeutung sind.

I. On the conversion of glycogen to lactate in muscle post mortem

The extent of post mortem glycolysis in muscle in combination with the rate of conversion of glycogen to lactate are important factors influencing the quality of meat.

Several groups of workers found no stoichiometric degradation of glycogen to glucose and lactic acid in bovine, porcine and ovine muscles(5,9,11,14,23). Although it had not been specifically calculated by the authors, several other papers also indicated, in the data presented, a nonstoichiometric conversion of glycogen to lactate (2,12, 21,22). The above authors have found that the breakdown of glycogen results in a conversion to lactate at rates from 50 to 100 percent. Inclusion of glycolytic intermediates in the results has not given a 100 % conversion of the glycogen to the sum of intermediates and lactate in stress resistant porcine muscle (14) and in ovine muscle (9).

Previous observations in our laboratory have shown that only about 55 to 70 % of the glycogen, which disappeared, was transformed to lactate (11,23). This paper reports on an investigation into this problem in bovine and rabbit muscle in an attempt to determine if this non-stoichiometric conversion of glycogen to glycolytic intermediates is similar to that in porcine muscle and ovine muscle.

MATERIALS AND METHODS

Bovine muscle samples from the longissimus and sternomandibularis were obtained as soon as possible after death from the city abattoir; the rabbit leg muscles (all visibly white muscles) were obtained within 1 hour after death from rabbits killed in our laboratory without anesthetic. After trimming of external fat and connective tissue, mincing and mixing, the samples were put into commercial freezer bags, tightly closed and stored at 2-4° C. Samples were taken at "0 time" (mostly about 1 hour post mortem) and then 24 hours after the "0 time" sample, with intermediate time samples being taken at 1, 2, 3, 6 or 9 hours depending on the rate of glycolysis.

Muscle extracts for the analysis of glycogen and all glycolytic intermediates including lactate were prepared by homogenizing 3 g of the muscle mince in 15 ml of ice cold 0.6 N perchloric acid. 0.20 ml of the homogenate were used for the glycogen determination. The remaining homogenate was centrifuged at 40,000 x g for 20 min at 0° C. The filtered supernatant was neutralized to a methylorange endpoint with 5.4 N KOH. After filtrating off the potassium perchlorate precipitate, the extract was stored at 0° - 4° C.

Analyses for free glucose, G-1-P, G-6-P and F-6-P were carried out in the same cuvette by combining the procedures of BERGMEYER and MICHAL (3), BERGMEYER et al. (4) and HOHORST (13). FDP, DAP and GAP were determined according to the procedure of BÜCHER and HOHORST (13). The procedure of CZOK and LAMPRECHT (7) was used for the determination of pyruvate, PEP and 2-PGA. 3-PGA was also determined with the previous analysis by the addition of phosphoglycerate mutase as described by CZOK and ECKERT (6). G-3-P was determined according to HOHORST (13).

The determination of lactate followed the method described by HOHORST (13). Glycogen was analyzed using the method described by DALRYMPLE and HAMM (8), whereby the perchloric acid homogenate is incubated in an amyloglucosidase enzyme solution. The glucose released by the enzyme was determined according to KEPPLER and DECKER (15).

RESULTS AND DISCUSSION

Table 1 summarizes the results obtained on the 8 muscle samples concerning the conversion of glycogen to lactate p.m. The muscles,

Table 1 Glycogen breakdown and formation of lactate and glycolytic intermediates post mortem in different bovine and rabbit muscles. Concentrations are expressed as µmoles per g wet tissue

	Bovine								Rabbit			
Muscle	Long.		Long.		Long.		Long.		Stern.		Stern.		Leg		Leg	
hours post mortem	2	26	4	28	1	25	1	25	1	25	1	25	1	25	1	25
Glycogen concn.	33.12	8,32	22.13	1.11	52.63	5.08	54.94	23.06	63.70	15.83	42.87	5.39	42.99	3.57	43.53	4.93
Lactate concn.	43.08	85.47	46.69	104.31	22.22	91.71	42.84	94.86	16.16	78.38	15.76	77.77	27.88	93.73	25.96	73.33
% Conversion of glycogen to lactate	85.49		129.93		73.08		81.58		64.98		82.71		83.51		61.42	
Glycolytic intermediates and lactate concn.[a]	34.24[b]	56.30[b]	42.50[c]	65.78[c]	23.44	64.06	38.49	61.73	13.43	47.17	14.43	47.21	24.54[c]	59.00[c]	23.17	48.26
% Conversion of glycogen to glycolytic intermed. and lactate	88.95		110.75		85.42		72.89		70.48		87.45		87.41		65.08	
Total glycolytic concn.[a]	67.36[b]	64.62[b]	64.63[c]	66.90[c]	76.08	69.14	94.43	84.79	77.14	62.99	57.29	52.65	67.53[c]	62.57[c]	66.70	53.2-
% Recovery of metabolites	95.93		103.51		90.88		89.79		81.66		91.90		92.66		78.3-	

[a] Expressed as glucose equivalents;

[b] For this sample only glucose, hexose phosphates and lactate were analyzed;

[c] This samale does not include 3-PGA, 2-PGA and PEP.

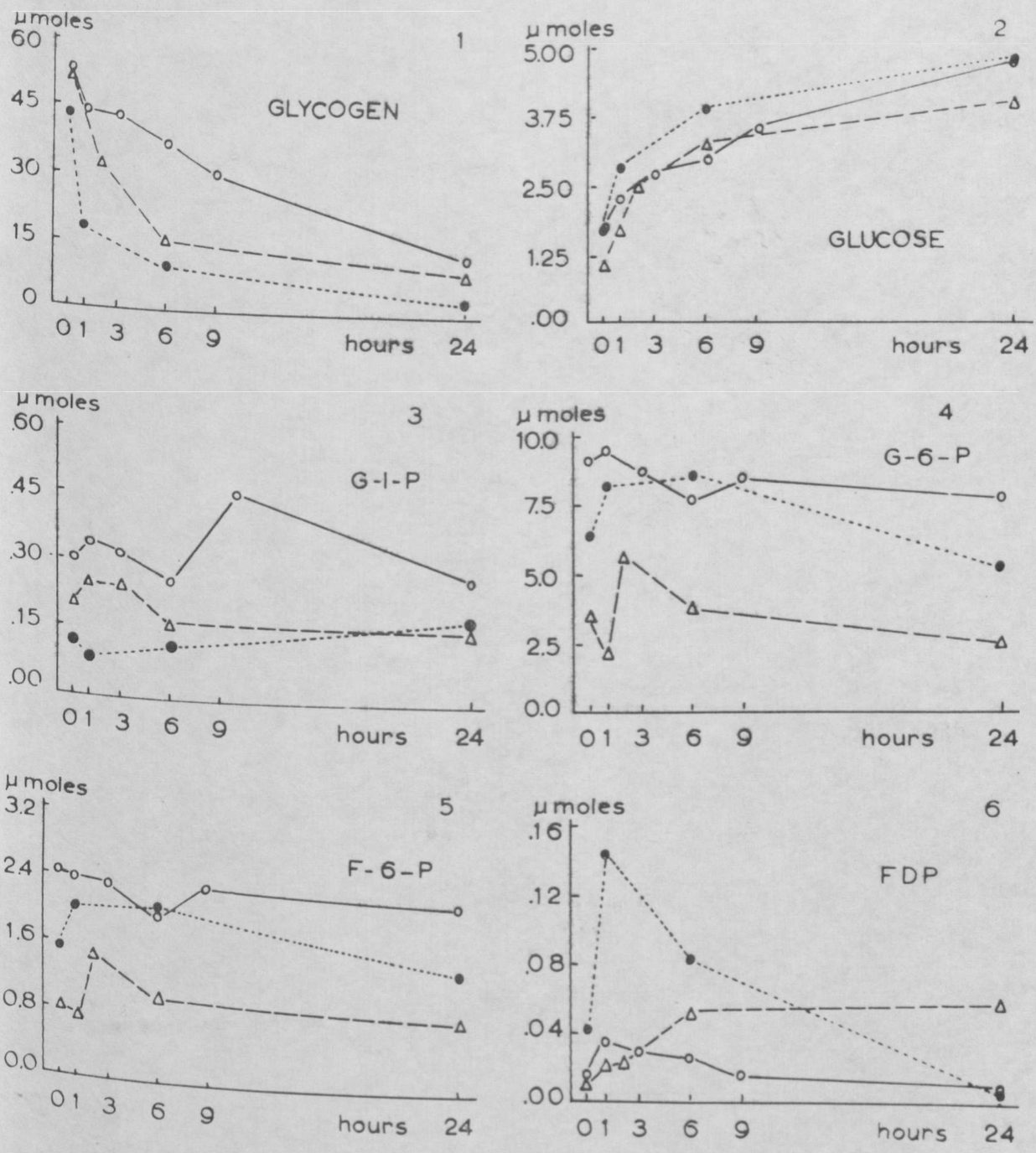

Fig. 1-6 Changes in glycogen and hexoses post mortem in bovine and rabbit muscles.
o—o: bovine longissimus; Δ---Δ: bovine sternomandibularis; ●...●: rabbit leg muscle.

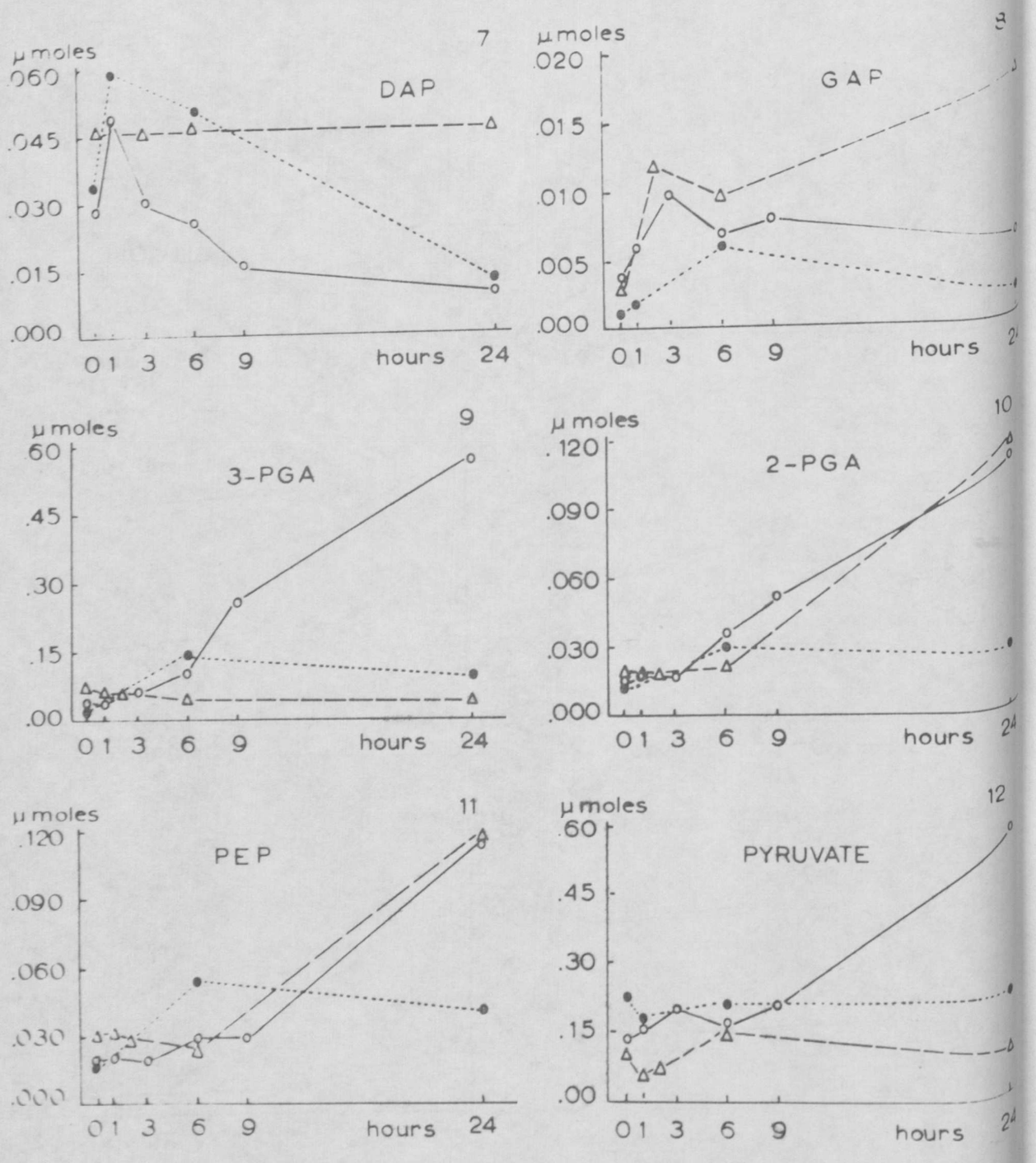

Fig. 7-12 Changes in glycolytic intermediates post mortem in bovine and rabbit muscles.
o—o: bovine longissimus; Δ---Δ: bovine sterno-mandibularis; ●....●: rabbit leg muscle

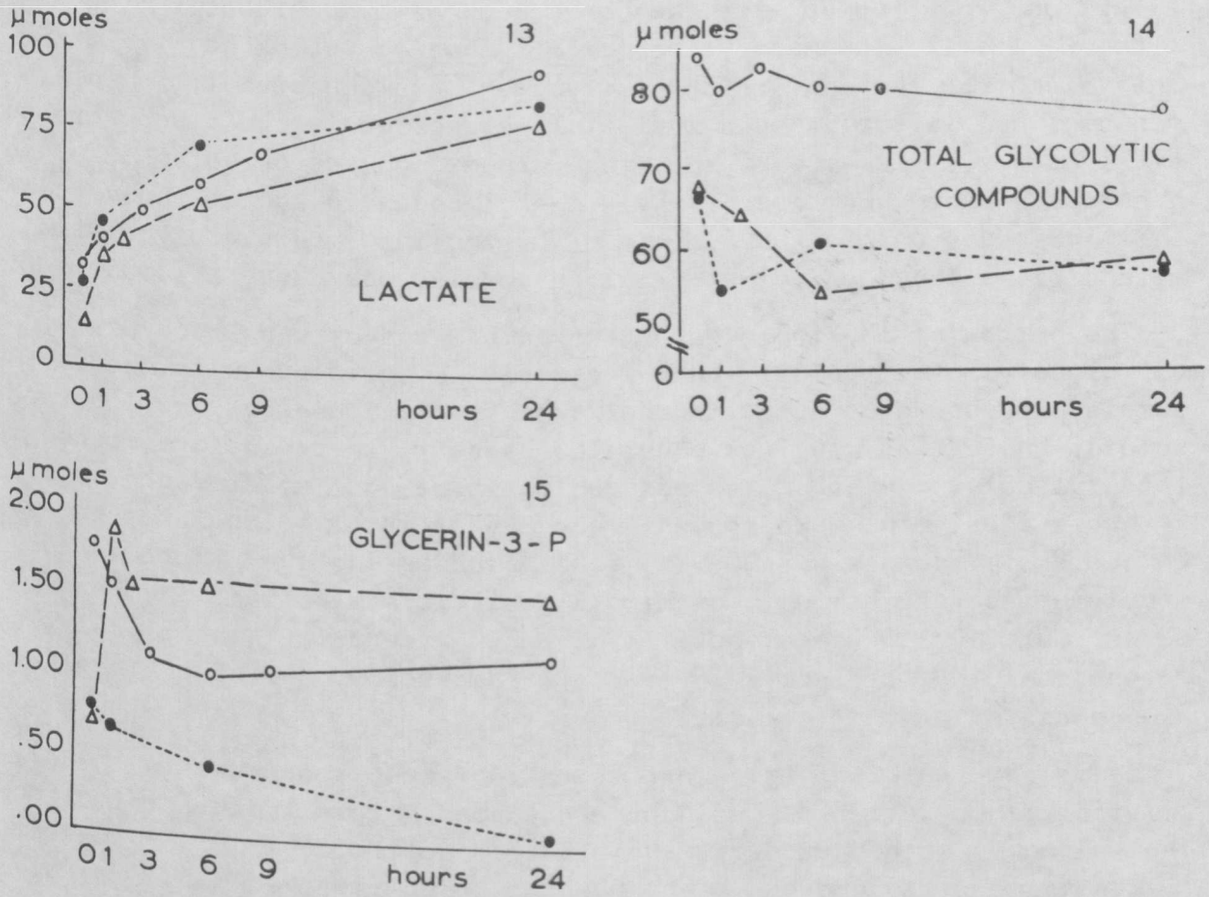

Fig. 13-15 Changes in lactate, total glycolytic metabolites and glycerol-3-phosphat post mortem in bovine and rabbit muscles.
o——o: bovine longissimus; Δ ---- Δ : bovine sternomandibularis; ●....●: rabbit leg muscles

except one, gave values for the conversion on a molar basis ranging from 60 to 85 %. The mean value for all samples was 82.83 ± 19.68 %. The values for the conversion of glycogen into the glycolytic intermediates and lactate should reflect more accurately the disappearance of glycogen. It is evident that the percent conversion values are, in general, higher than those seen in the glycogen to lactate conversion. This indicates a slight buildup p.m. of the intermediate glycolytic metabolites. The mean value for all 8 samples was 83.55 ± 13.36 %.

The bottom of the table gives the values and percent recovery of all glycolytic metabolites analyzed including glycogen with the first sample p.m. being compared to the 24 hours p.m. sample. In all but one sample, where the value was about 100 percent, there was a decrease in total glycolytic metabolites between the time soon after death until 24 hours later. The mean percent recovery of total metabolites in the 24 hours p.m. sample was 90.92 ± 7.05 % indicating that there is no complete recovery of the beginning glycolytic metabolites in most cases. Our results (see figure 14) show the trend that the glycolytic metabolites that are not recovered are lost mainly during the beginning phase of p.m. glycolysis.

It is apparent that this loss is taking place somewhere in the glycolytic pathway after glycogen breakdown and before lactate formation. The enzymatic method used for the glycogen determination is an extremely sensitive assay and has been shown to be an improved method of glycogen determination (8). The lactate analysis has been carefully standardized in an attempt to minimize the error in this determination. Therefore, glycolytic metabolites seem to disappear somewhere in the glycolytic pathway. There are many possibilities for this loss which should not be discussed here but in a publication later on. It should be only mentioned that the disappearance of pyruvate through transamination seems to be somewhat more probable than other possible ways.

Figures 1 to 15 show the changes in the glycolytic metabolites from 1 to 25 hours p.m. in the three muscles investigated. The figures for glycogen show an apparent difference in the rate of breakdown. The rabbit leg muscle had a fast rate of glycogen disappearance, the bovine long. a slow rate with the stern. having an intermediate rate. All three muscles showed an increase in the free glucose content with time p.m. The free glucose is produced by the debranching enzyme (20). The presence of endogeneous α-amylase and α-glucosidase also contributes to the free glucose level (1).

The levels of G-1-P, G-6P and F-6P are higher in the bovine long. than in the stern., which might be due to the slower breakdown of glycogen in the long. The theory that the level of the hexose phosphate intermediates is controlled mainly by phosphofructokinase (PFK) (17), is supported by our data since the buildup of hexose phosphates

by a slowdown of passage through the PFK step also slows down the breakdown of the glycogen molecule as can be seen in the case of the long. In the stern. a more active PFK results in lower levels of the hexose phosphates, slightly higher levels of FDP and the triose phosphates (DAP, GAP) and a faster rate of glycogen breakdown. This is also evident in the lactate levels where the stern. had a much lower "0 time" level than the long. but rapidly approached a similar level after 1 - 2 hours. A similar trend appears in rabbit muscle where an even faster rate of glycogen breakdown occurs possibly to compensate for the maintenance of slightly higher hexose phosphate levels than seen in the bovine stern.

The post mortem change in G-3-P also shows species and important muscle differences. In the rabbit leg muscles and in the bovine stern. there was a general maintenance of the "0 time" levels while in the bovine long. a post mortem decrease was seen. The G-3-P cycle, which is operating between the mitochondria and cytoplasm involving DAP and the production of NADH may be of minor importance after death because of the anaerobic conditions p.m.

With 3-PGA, 2-PGA, PEP and pyruvate slight species and muscle differences are evident, the reasons of which are not apparent but may be related to the different rates and extent of post mortem glycolysis.

On a molar basis it was found that the metabolites glycogen, glucose, G-6-P, F-6-P and lactate make up 95 % or more of the total glycolytic metabolites present, in the muscle at all times p.m. This is indicating the control particularly by the enzyme PFK. Glucose and lactate are not generally metabolized p.m. but they are end products of the process. It appears from our data that phosphorylase and PFK are the most important control sites since little regulation was found at pyruvate kinase and GAP dehydrogenase steps. The theory that most of the post mortem control takes place at the PFK step is supported by comparing the two bovine muscles. Lower PFK maintains high levels of F-6-P and G-6-P in the long. by slowing down the flux of these metabolites. This in turn may cause a lowering of the phosphorylase activity through high levels of G-6-P and G-1-P, therefore,giving a slower rate of glycogen breakdown. A lower activity of phosphorylase in turn should be indicated by decreased levels of the hexose phosphates but this was not the case showing clearly that the major control site is at PFK.

In the cessation of glycolysis both phosphorylase and PFK appear to play a role. This is probably due mainly to the drop in pH since the levels of metabolites including glycogen and hexose phosphates are present in amounts that would allow further glycolysis. By 24 hours p.m. the levels of glycogen remaining in the muscle were at least 10 % or more of the "0 time" level and the hexose phosphates levels were similar to the "0 time" levels. This last fact indicates that the inhibition at these control sites takes place at a similar pH, since

any inhibition of one enzyme without inhibition of the other should have resulted in significant changes in the concentration of the hexose phosphates.

II. On the type of adenosine triphosphatases operating in muscle post mortem

INTRODUCTION

The adenosine triphosphate (ATP) present in muscle tissue is broken down during the first hours after death of the animal. ATPase catalyzes the first step of this reaction, the hydrolytic splitting off of one mole of inorganic phosphate. Several ATPases exist in the muscle cell, for instance in the membranes, in the sarcoplasmatic reticulum and in the myofibrils. The question arises, which of these ATPases are responsible for the ATP breakdown p.m. This problem has been discussed extensively in the literature (for references see (10,23)). We have tried a new approach to this problem.

It is well known that most of the ATPases require ions like Na^{+}, K^{+}, or Mg^{++} and Ca^{++} for their activity (18) but the requirements vary for the different ATPases. Also some inhibitors are known which block specifically the activity of the ATPases in certain subcellular fractions (18). We investigated the influence of these factors on thin slices of rabbit muscle from shortly after death up to 24 hours p.m., following the breakdown of ATP and the formation of its metabolites in order to see which of these ATPases are operating p.m.

MATERIALS and METHODS

Rabbits were killed by a blow on the neck and the muscles (longissimus, psoas major and psoas minor) were removed as fast as possible, cut parallel to the muscle fibres into slices 1 to 1.5 mm thick and submerged into different solutions of salts and inhibitors at nearly physiological ionic strength and pH not later than 30 min after death. The incubations were carried out at 22°C. The salt concentration in all incubation media was 0.17 M. The differences between the concentrations mentioned (table 2 and figure 16) and 0.17 M were made up with Tris-HCl buffer. The pH of all incubarion media was 7.2 in the beginning and dropped to 6.6-6.9 during the 24 hours of incubation. Samples were taken at 1, 2, 4, 6, 12 and 24 hours p.m., homogenized in 1.5 M trichloracetic acid and centrifuged at 10,000 x g for 15 min. The supernatant contained the ATP and its metabolites, which were separated and determined with the TLC method described by POTTHAST and HAMM (19). The amount of each nucleotide has been expressed as percent of the sum of ATP, ADP, AMP, IMP and inosine found on the chromatogram. Hypoxanthine was not present in these samples. The

values at different times p.m. could not be related to the nucleotide content of the first sample (30 min p.m.) since in the samples at 12 and 24 hours p.m. the total amount of ATP and its metabolites was less than in the first 6 hours, probably due to the leaking of metabolites into the incubation medium.

RESULTS

With the methods described the following results for the enzymatic breakdown of ATP were obtained. With incubation in 0.17 M Tris-HCl buffer, even after 24 hours, 5 to 10 % of the ATP was not yet hydrolyzed (table 2). KCl and NaCl exhibited about 5 and 2 % resp. unhydrolyzed ATP after 10 hours of incubation (table 2), while in muscle slices incubated with 0.05 M $MgCl_2$ all ATP was hydrolyzed already by 5 hours p.m. (table 2, Fig. 16). In 0.03 M $CaCl_2$ all ATP was broken down within 3 hours p.m. (Fig. 16). Ouabain, an inhibitor for the sodium-potassium sensitive membrane ATPases, did not influence the rate of ATP breakdown (Fig. 16). Oligomycin which influences the mitochondrial ATP metabolism had no significant effect either (Fig. 16).

With the Mg^{++} and Ca^{++} chelating agents EDTA and EGTA resp. similar results were obtained as with tris buffer (Table 2). The incubation in $3 \cdot 10^{-3}$ M quinidine sulfate had no influence on ATP hydrolysis in our experiments with muscle slices (Table 2). This substance inhibits the sarcoplasmic reticulum ATPase as we have demonstrated with isolated sarcoplasmic reticulum preparations.

The changes in the amount of ADP p.m. were much less pronounced than those of ATP. In the first 6 hours p.m. ADP decreased from about 25 % to 10 % of the total metabolites in all of our experiments. After that time it remained constant till 12 hours. From the 12^{th} to 24^{th} hour p.m. it seemed in all cases to increase slightly. AMP showed no significant change during 24 hours p.m. IMP, however, exhibited remarkable changes during the time of incubation. At 1 hour after death the amount of IMP differed from 10 to 25 % of the sum of all metabolites. After 6 to 12 hours, the amount of IMP increased up to 40-65 %, depending on the incubation medium and the resulting rate of ATP hydrolysis. From 12 to 24 hours IMP decreased again to 25 - 30 % with the exception of the incubation with EDTA and EGTA where the amount of IMP remained relatively constant from 12 to 24 hours p.m.

Table 2 Influence of different incubation media on the breakdown of ATP post mortem in rabbit muscle

compound added	(M)	% ATP[a] at hours after death				
		1	2	5	10	24
Tris-buffer	0.17	52	46	26	12	7
Quinidine-sulfate	0.003	55	45	20	10	7
EDTA	0.03	45	38	15	11	10
EGTA	0.05	35	30	15	12	10
KCl	0.10	52	49	33	5	0
NaCl	0.15	53	45	22	2	0
$MgCl_2$	0.05	50	37	0	0	0
$CaCl_2$	0.03	35	10	0	0	0

[a]Percent ATP of the sum of ATP and its metabolites

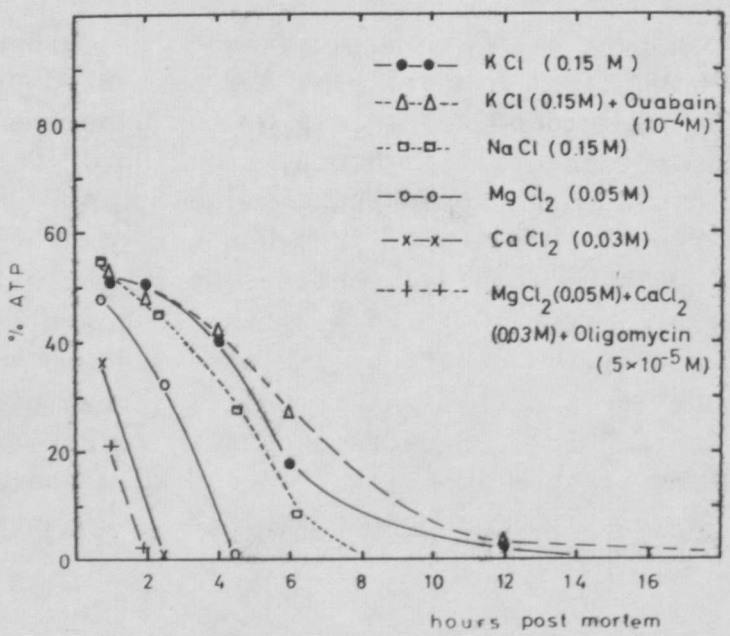

Fig. 16 Influence of different incubation media on the breakdown of ATP in rabbit muscle post mortem

DISCUSSION

The results obtained indicate that the ATP hydrolysis p.m. is due mainly to ATPases which need Mg^{++} and Ca^{++} for their activity. K^{+} and Na^{+} at concentrations of 0.1 to 0.15 M are not activators. Ouabain, which inhibits membrane ATPases, does not significantly block the ATP hydrolysis. This indicates that the effect of membrane ATPases cannot be predominant in the enzymatic breakdown of ATP p.m. MARTONOSI (16) reports that the ATPases of the sarcoplasmatic reticulum are maximally activated by about 0.1 M KCl. However, in our experiments 0.1 and 0.15 M KCl had no stimulating effect, neither has quinidine sulfate, which we found to be an inhibitor of the sarcoplasmatic reticulum ATPase. Mg^{++} and Ca^{++} ions, however, have a strong stimulating effect on the breakdown of ATP. These findings speak in favour of the predominant effect of the myofibrillar ATPases, which in intact cells are stimulated by Ca^{++} and which need Mg^{++} for their activity. At present further studies are being carried out in our laboratory on isolated subcellular fractions to confirm the reported results.

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DEGRADATION ENZYMATIQUE du TRIPOLYPHOSPHATE et du PYROPHOSPHATE (DIPHOSPHATE) AJOUTES dans la VIANDE BROYEE.

R. NERAAL et R. HAMM

L'influence de divers facteurs de grande importance technologique sur la dégradation enzymatique du tripolyphosphate (TP) et du pyrophosphate (DP) ajoutés est étudiée sur le muscle de boeuf broyé et salé à 2% de NaCl à l'aide d'une méthode par chromatographie sur couche mince. On a observé d'un animal à l'autre des différences considérables d'activité enzymatique tripolyphosphatase (TPAse) et pyrophosphatase (DPAse) sur le Longissimus dorsi. L'activité TPAse est notablement plus intense que celle de la DPAse. Pour une hydrolyse complète de 0,5% de polyphosphate dans la viande à 20°C il faut de 8 à 20 minutes pour le TP et de 2 à 15 heures pour le DP. L'activité TPAse augmente, l'activité DPAse baisse pendant les 2 premiers jours post-mortem. Ces deux effets doivent principalement reposer sur la chute du pH post-mortem. L'optimum d'activité se situe à pH 5,7 pour la TPAse et à pH 7 pour la DPAse. Le maximum d'activité polyphosphatase est observé pour une concentration en sel NaCl de 4% , alors que l'activité DPAse décroît de façon continue à mesure que la concentration en NaCl croît.

La TPAse et la DPAse sont totalement inactives à 0°C et aux températures de congélation. Les activités augmentent avec la température jusqu'à la température d'inhibition thermique des enzymes. La DPAse est plus sensible à la chaleur que la TPAse. La congélation des tissus a une influence certaine sur les activités enzymatiques.

L'augmentation de pouvoir de rétention en eau (PRE) dûe à l'addition de DP ne s'abaisse pas même après hydrolyse complète du DP. L'augmentation de PRE dûe à l'addition de TP augmente pendant la dégradation rapide de TP en DP. Seul DP semble avoir une influence sur les protéines myofibrillaires. TP semble n'être actif qu'après dégradation enzymatique en DP.

ПОСЛЕСМЕРТНОЕ РАСЩЕПЛЕНИЕ ГЛИКОГЕНА И АТФ В СКЕЛЕТНОЙ МЫШЦЕ

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В m. long. dorsi и m. sternomandibularis крупного рогатого скота и в бедренних мышцах кролика определяли в разное время после убоя (24 часа) продукты гликолиза, включая гликоген, молочную кислоту, глюкозу и глицерин-3-фосфат.

Раньшие работы не показали стехиометрического перехода гликогена в молочную кислоту; в этой работе изучали расщепление гликогена до молочной кислоты и межупроточные продукты. После 24 часа 83,5 $\pm$ 13,4 % гликогена перешло в молочную кислоту и в крайние глюкоза-продукты. Сумарная концентрция продуктов распада гликогена уменшалась после смерти; это показало что возле гликолиза можные тоже иные способы распада. Из изменения продуктов гликолиза после смерти произходит что фермент фосфофруктокиназа имеет решительное влияние на скорость и обём послесмертного гликолиза.

Тонкие слои скелетных мышц кролика подвергались непосредственно после убоя инкубации в различных средах; исследованно влияние продол жительности инкубации на содержание АТФ и продуктов его распада в ткани после убоя , что бы установили который вид АТФ-азы отвечательный для распада АТФ в мышцах после смерти. K^+ и Na^+ в концентрациях от 0,1 - 0,15 М не активируют АТФ-азы.

Ингибирующее влияние уабаина и хининдисулфата не замечено. 0,05 М $MgCl_2$ и 0,03 М $CaCl_2$ визывали очень активирующий эффект на гидролиз АТФ. Результаты показивают что ни АТФ-азы мембран ни АТФ-азы саркоплазматического ретикулума, но миофибриллярная АТФ-аза имеет решительное значение для послесмертного распада АТФ.