INFLUENCE OF POST-MORTEM GLYCOLYSIS AND DEPHOSPHORYLATION OF HIGH ENERGY PHOSPHATES ON POULTRY TENDERNESS

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BY

A. W. Khan

Division of Biology

National Research Council

Ottawa, Canada

SUMMARY

To study the influence of post-mortem glycolysis and dephosphorylation of high energy phosphates on meat tenderness, tests were made on poultry meat from epinephrine-treated and untreated birds, and by treating pre-rigor excised pectoralis major muscles with iodoacetate and fluorodinitrobenzene. All these treatments did not affect dephosphorylation of adenosine triphosphate and adenosine diphosphate, and the onset of rigor mortis as determined by isometric tension development. Epinephrine and iodoacetate treatments inhibited lactic acid formation, while iodoacetate and fluorodinitrobenzene treatments caused a rapid depletion of adenosine triphisphate and adenosine diphosphate. Epinephrine treatment gave more tender meat while iodoacetate and fluorodinitrobenzene caused toughness. The significance of these results in post-mortem tenderization of meat is discussed.

INTRODUCTION

The importance of post-mortem glycolysis and dephosphorylation of adenosine triphosphate in determining meat quality has been shown for poultry (deFremery and Pool, 1963), beef (Marsh, 1954) and pork (Briskey, 1963). In poultry, glycolysis occurring immediately before and during slaughtering and bleeding has also been shown to affect quality by causing a low post-slaughter pH and toughness (Khan and Nakamura, 1970). More recent work in our laboratory has shown that holding poultry meat at 30-37°C during the onset of rigor mortis caused toughness, and that this toughening effect of high temperature occurred when (a) the pH level of the meat dropped from a value of about 6.3 to its ultimate value, and (b) the adenosine triphosphate content dropped below 40% of its initial concentration (Khan, 1971). The work has been extended to study in more detail the influence of post-mortem glycolysis and dephosphorylation of high energy phosphates on poultry tenderness. This paper describes the results of preliminary experiments made on pectoralis major

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muscles in which the formation of lactic acid and regeneration of adenosine triphosphate were inhibited by treatment with iodoacetate or fluorodinitrobenzene or in which glycolysis was minimized by an ante-mortem epinephrine treatment.

EXPERIMENTAL

Pectoralis major muscles were obtained from male chickens (eviscerated weight 1.5-2.0 kg), hatched and reared in the laboratory under similar environmental and nutritional conditions. To obtain muscles of high post-slaughter pH (6.7-7.0), well-rested birds were restrained in a metal funnel during slaughtering and bleeding to minimize voluntary as well as involuntary struggling. After slaughtering and bleeding, both pectoralis major muscles were excised immediately. For minimizing post-mortem glycolysis, an intramuscular dose of 4 mg of epinephrine/kg body weight was administered 16 hr. before slaughtering. For arresting regeneration of adenosine triphosphate (ATP) and glycolysis, excised breast muscle from one side was injected with neutralized iodoacetate (5 mM, 25 ml/100 g muscle), or with 2,4-dinitrofluorobenzene (5 mM, 25 ml/100 g muscle); the other half was treated with physiological saline solution (25 ml/100 g muscle) and used as the control. The muscles were aged at 2-4°C and analyses were carried out during the first 48 hr. post-mortem.

The progress of dephosphorylation of high energy phosphates was followed by monitoring the ATP and ADP contents by means of (a) differential spectroscopy (Khan and Frey, 1971), (b) phosphate analysis (Khan and Frey, 1971) and (c) chromatographic analysis using Dowex 1-X8 (formate) column (Terasaki *et al.*, 1965).

The progress of glycolysis was followed by monitoring pH changes in the test muscle. Onset of rigor mortis was followed using a transducer-amplifier system to measure isometric tension development (Khan and Frey, 1971).

Shear force value of the cooked meat was determined using a texture press system equipped with a meat shear cell as described earlier (Khan, 1971).

RESULTS AND DISCUSSION

Typical post-mortem changes in epinephrine-iodoacetate- and fluorodinitrobenzene-treated muscles are shown in Fig. 1, 2 and 3. Epinephrine and iodoacetate treatments stopped the formation of lactic acid while fluorodinitrobenzene was only partially effective in this regard. All these treatments did not prevent contraction during rigor as was shown both by measurement of isometric tension development and determination of the ATP and ADP contents. Fluorodinitrobenzene and iodoacetate treatments markedly increased the rate of dephosphorylation of ATP and ADP, while epinephrine treatment had little effect.

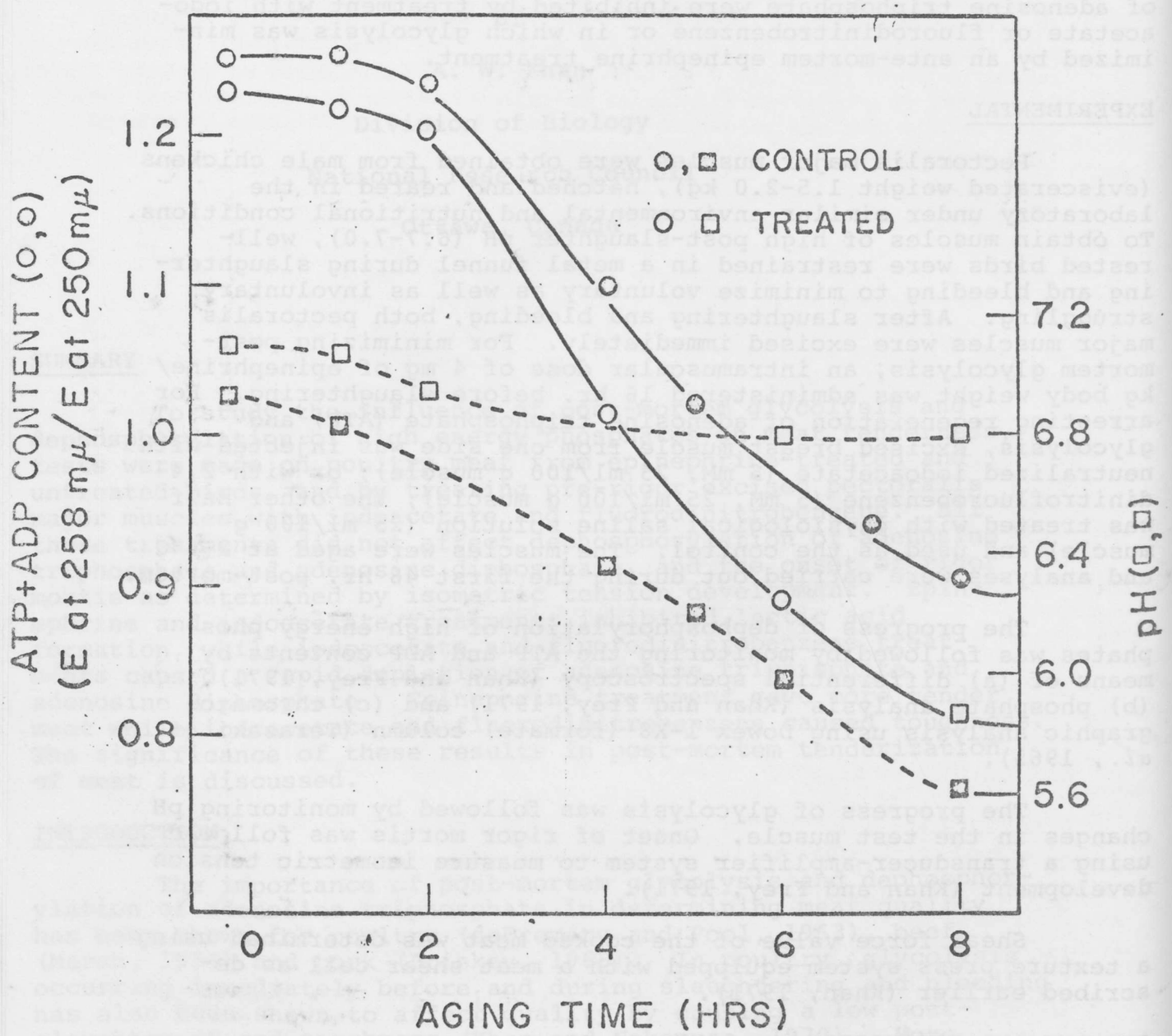

Fig. 1. Effect of ante-mortem epinephrine treatment on post-mortem dephosphorylation of adenosine triphosphate and adenosine diphosphate and pH of chicken breast muscle.

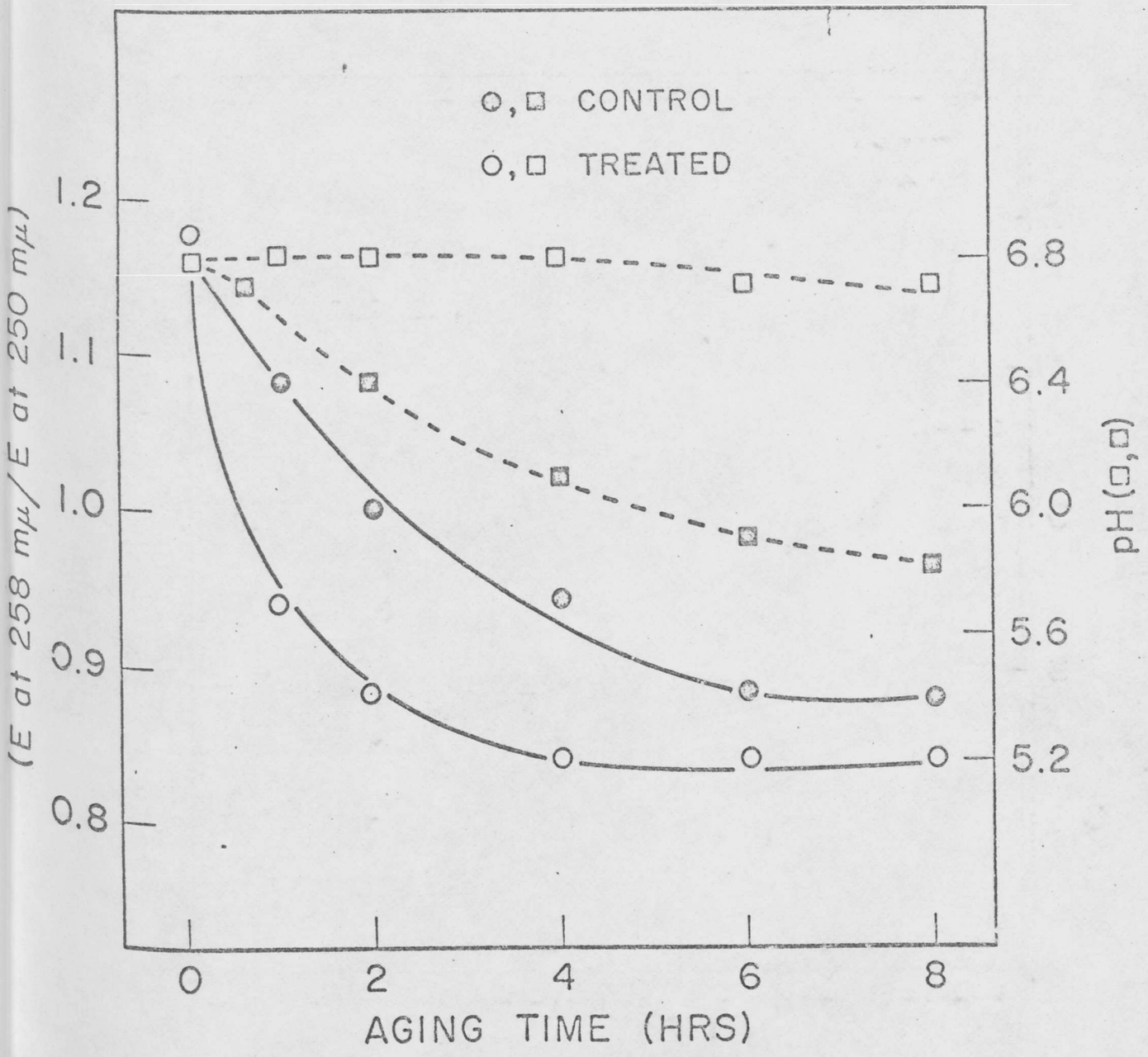

Fig. 2. Effect of iodoacetate treatment on post-mortem dephosphorylation of adenosine triphosphate and adenosine diphosphate and pH of chicken breast muscle.

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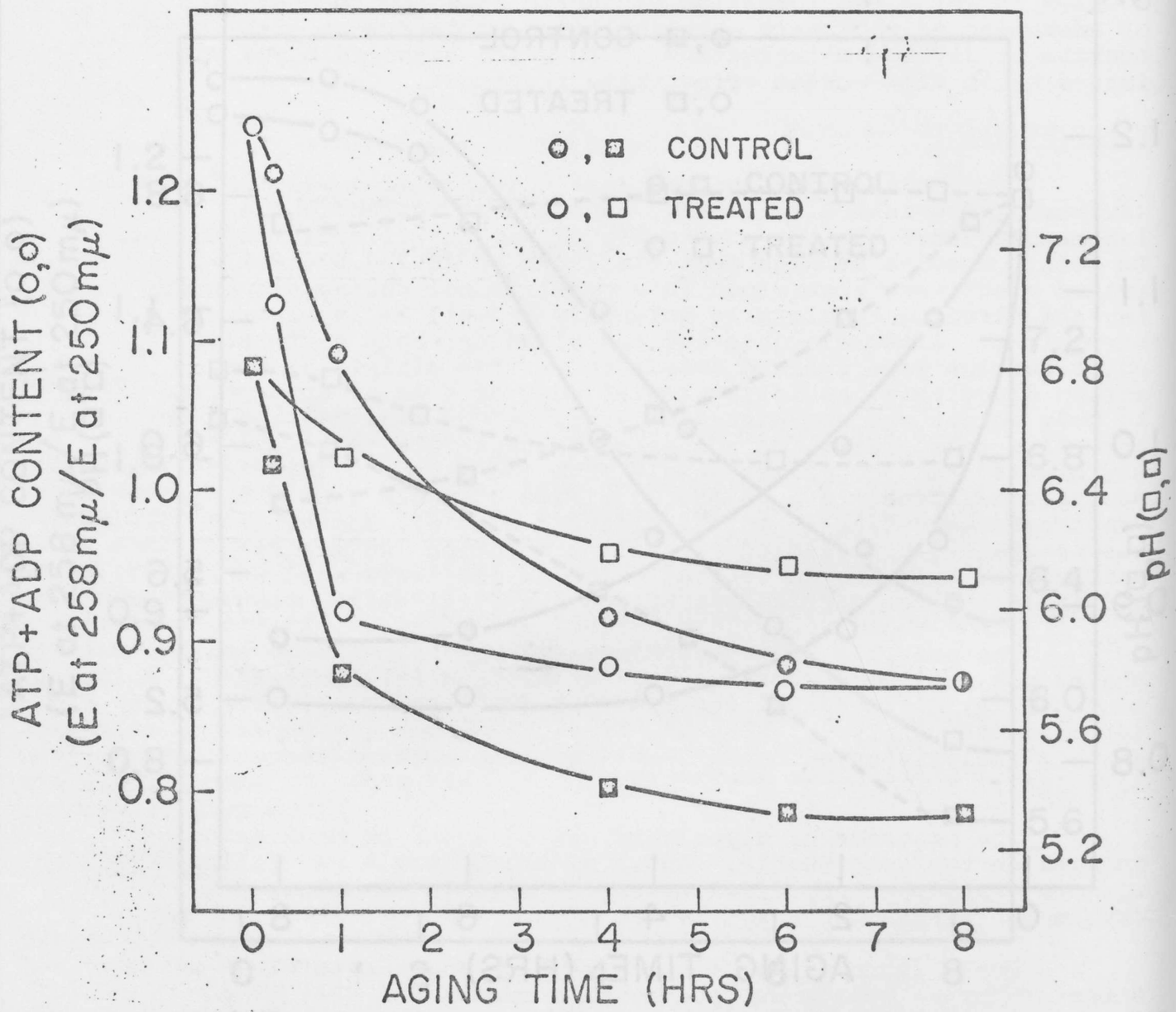

Fig. 3. Effect of fluorodinitrobenzene on post-mortem dephosphorylation of adenosine triphosphate and adenosine diphosphate and pH of chicken breast muscle.

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Table 1. Effect of epinephrine, iodoacetate and fluorodinitrobenzene treatments on tenderness (shear force value, 48 hr. postmortem) of poultry breast meat.

Treatment	Shear force Value (kg)
Control	2.6 - 2.9
Epinephrine	2.4 - 2.6
Control	2.3 - 2.8
Iodoacetate	4.3 - 4.6
Control	2.0 - 2.8
Fluorodinitrobenzene	4.2 - 6.0

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Treatment of muscle tissue with fluorodinitrobenzene and iodoacetate caused toughness, while epinephrine treatment slightly increased tenderness (Table 1). Of the three inhibitors, fluorodinitrobenzene treatment had the greatest deleterious effect on tenderness. After the onset of rigor mortis, both iodoacetate and fluorodinitrobenzene had no deleterious effect on tenderness.

The treatments used in these tests have been known to inhibit the three possible ways by which ATP can be resynthesized anaerobically from ADP in muscle. Fluorodinitrobenzene stops the phosphorylation of ADP by phosphocreatine, the main source of high energy phosphates. This phosphorylation reaction has been shown to take place very early in contraction and enables the muscle to keep up a high level of immediately available energy in the form of ATP. Epinephrine stops new high-energy phosphate generation by minimizing glycolysis; it does not interfere with the other two sources of energy supply. Iodoacetate stops the third possible source of ATP by inhibiting adenylate kinase (myokinase). This enzyme acts on two molecules of ADP, and can catalyse the transfer of phosphate from one molecule to the second, yielding a molecule of ATP.

In muscle, the phenomena of post-mortem glycolysis, dephosphorylation of ATP, stiffening and tenderness are closely related. Results show that absence of glycolysis did not prevent stiffening during rigor, and did not prevent toughness except in epinephrine-treated muscles. Post-mortem glycolysis causes a net gain of three molecules of ATP for each 6-carbon unit of the glycogen converted to lactic acid. Since minimization of post-mortem glycolysis by epinephrine treatment did not produce toughness, ATP generation as a result of glycolysis does not appear to play an important role in determining tenderness. Inhibition of ATP generation from the action of adenylate kinase by iodoacetate, and inhibition of ATP generation from phosphocreatine by fluorodinitrobenzene in particular, caused a rapid dephosphorylation of high energy phosphates as well as toughness. However, the action of these inhibitors on biochemical and enzymatic changes occurring during post-mortem aging in myofibrillar proteins is not known, and further tests are being carried out to elucidate the effect of these changes on tenderness.

REFERENCES

Briskey, E.J. 1963. Proc. Campbell Soup Co., Meat tenderness symposium, Camden, N.J. p. 195.
deFremery, D. and M.F. Pool. 1963. J. Food Sci. 28, 173.
Khan, A.W. 1971. J. Food Sci. 36, 120.
Khan, A.W. and A.R. Frey. 1971. Can. Inst. Food Technol. J. 4, in press.
Khan, A.W. and R. Nakamura. 1970. J. Food Sci. 35, 266.
Marsh, B.B. 1954 J. Sci. Food Agric. 5, 70.
Terasaki, M., M. Kajikawa, E. Fujita, and K. Ishii. 1965. Agr. Biol. Chem. 29, 208.