Chapter 2

Post - Mortem Quality Changes in Fish

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Information about the post mortem changes of fish is imperative to appreciate the processors involved in the spoilage and quality changes. The knowledge is beneficial for better control of the quality of raw material. Understanding the factors that cause changes in quality helps to find ways to prevent the changes and maintain the quality and freshness of the raw material.

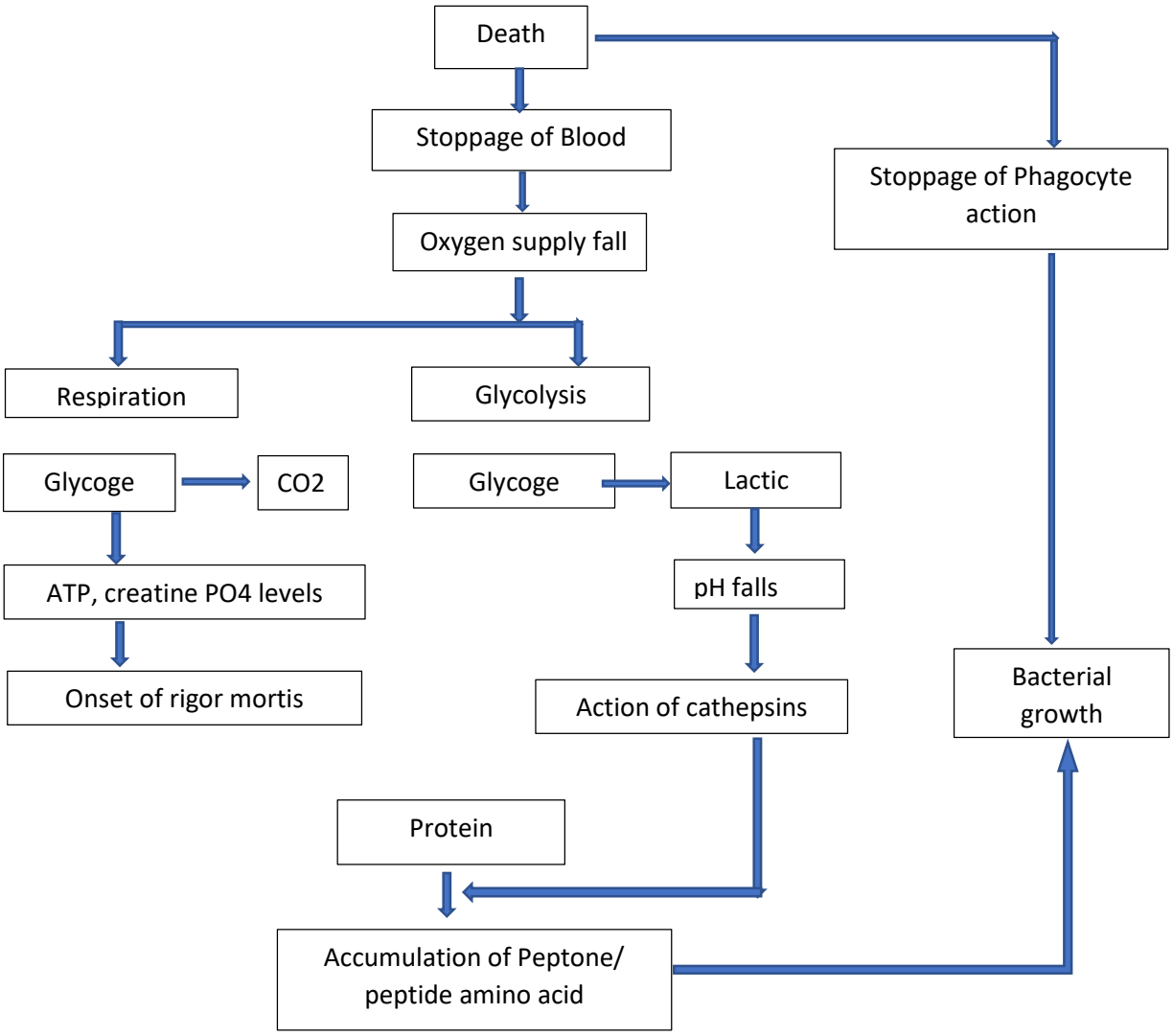
Fish is a food item of good acceptability and nutritional value. But it is a highly perishable item and quality deterioration very fast if not preserved properly. The changes leading to spoilage of fish are highly complex. Both biochemical and microbiological processes contribute the quality deterioration. The enzymes naturally present in the system are primarily responsible for the post-mortem biochemical changes.

During the period after death till its consumption a large number of physicochemical changes take place, which can be classified into three stages.

1. Pre-rigor state –in which the meat is soft and pliable and is characterised biochemically by a fall in ATP and creatine phosphate and glycolysis.
2. Rigor mortis-stiff and rigid condition, which extends from 1 to 7 h and is affected by a number of factors.
3. Post rigor- a stage during which meat tenderisation takes place making the meat organoleptically acceptable.

Pre rigor state

It is the stage of the fish immediately after death and before the onset of rigor mortis. The first sign of dead fish is the unusually high mucus on the body. In live fish mucus, a glycoprotein formed of mucin, is secreted at a controlled rate and plays a role in preventing the entry of microbes by its anti-bacterial and lysosomal action. However, after death the properties of the mucus are altered or lost and it no longer acts in controlling microbial invasion.



Following death, the circulation of bold to body ceases leading to depletion of oxygen supply to the tissues is cut off. This results in the inability of body tissues to synthesise adenonsine-5' triphosphate (ATP) as electron transport chain (ETC) and oxidative phosphorylation mechanisms are no longer operative. This results in the depletion of ATP and creatine phosphate. Alongside this the anaerobic conversion of glucose to lactic acid takes place, leading to drop in pH. This accelerates rigor mortis and protein denaturation. The drop in pH accelerates action of cathepsins and other proteolytic enzymes. This leads to accumulation of various metabolites, flavour, bacterial growth and ultimately spoilage.

Rigor mortis

Normal aerobic oxidation of glucose through oxidative chain produces 39 molecules of ATP per molecule of glucose. After death as anaerobic condition is created and glycogen can no longer be converted in to CO_2 and water. The major supply of ARP is, thus, cut off. Anaerobic

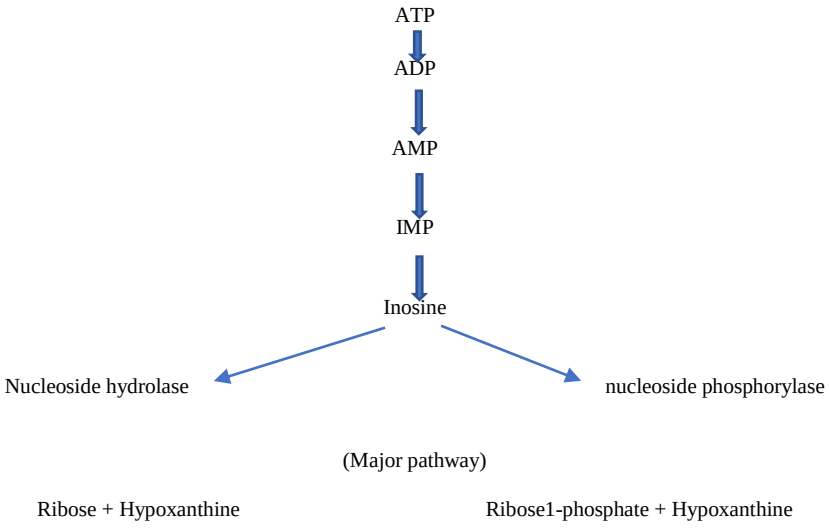
conversion of glycogen to lactic acid takes place, leading to the production of just 3 molecule of ATP.

Besides, the ATP available is also depleted by sarcoplasmic ATPase for the phosphorylation of glycogen to glucose-1-phosphate. For some time after death the ATP concentration is maintained at the expense of creatine phosphate by the action of the enzyme creatine kinase. The ADP released as a result of ATPase activity is rephosphorylated to ATP and free creatine is formed. Thus, in the early post mortem stages the concentration of ATP is maintained and the concentration of creatine phosphate decreases rapidly. Eventually due to the continued activity of ATPase, the concentration of ATP as well as creatine phosphate falls.

Drop in ATP level initiates combination of actin and myosin leading to the formation of actomyosin. This results in a rigid condition of the muscle, called rigor mortis. In cod it was reported that a 5% drop in ATP level leads to onset of rigor mortis.

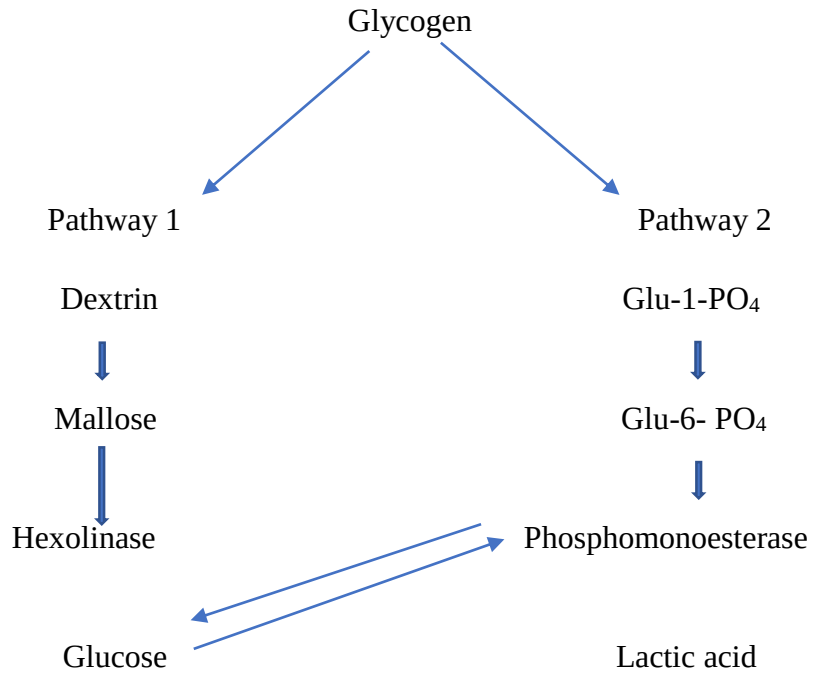
Post mortem metabolism of ATP

ATP is converted in to adenosine-5' –diphosphate (ADP) by sarcoplasmic ATPase, which is by myokinase. Conversion of adenosisne-5' monophosphate (AMP) to IMP is effected out by a deaminase action. IMP, which plays a role in the flavour of good quality fish, is dephosphorylated to inosine. Decomposition of inosine produces hypoxanthine, which is considered to be a quality index for freshness and quality of fish.



Post mortem glycolysis

On cessation of oxygen supply to the muscle tissues, glycogen the main carbohydrate source of muscle, is no longer oxidized to Co2 and water but broken down to lactic acid by anaerobic glycolysis, which is reported to take place by two pathways- hydrolytic or amylolytic pathway, and phosphorolytic pathway.



Post mortem pH

As lactic acid accumulates in the system, the pH of the system falls form the initial physiological pH of 7.2-7.4 to the ultimate post mortem pH of 5.3 - 5.5 in well fed and rested animals which have high levels of tissue glycogen. In fish relatively high pH is required to prevent toughness of meat and the final pH is attained in 24 hrs. Fish which was made to struggle just before death have a lower glycogen level and hence the ultimate pH would be around 6 – 6.6.

Time course of rigor mortis

The development of rigor is closely related to temperature. The length of time between death and onset of rigor is determined by the relative activities of enzyme systems responsible for ATP degradation. This in turn is controlled by the relative concentrations of ATP, creatine phosphate and glycogen in the muscle tissue at the time of death. In well-fed and well –rested animals the levels of these chemicals are high so that a longer delay period is observed prior to development of rigor, producing meat of low pH and high quality. Subjecting the fish to starvation or struggling would inevitably result in a much shorter delay period producing meat of inferior quality.

A prominent post-mortem change is the joss of water bound to protein molecules due to the loss of water holding capacity falls. This is related to the drop in pH to 5.3-5.5 which is almost close to the isoelectric pH of fish meat. During post rigor aging of meat, the water holding

capacity of meat was found to increase. This is attributed to an increased osmotic pressure within the fibres or alterations of the electrical charges on protein molecules involved and is related to the movement of ions to and from the muscles.

Changes in muscle proteins

The muscle myofibrillar protein, particularly actin and myosin changes in relation to rigor mortis, the actin and myosin are dissociated in the pre rigor stage. Depletion of ATP gradually associates the two leading to the formation of actomyosin. The fish sarcoplasmic proteins are far more stable than myofibrillar protein. They possess better thermo stability and solubility than their counterparts in other meats and not appear to play roles in muscle texture.

Following the resolution of rigor, a gradual tenderization of meat occurs and the post rigor meat is organoleptically well accepted compared to that in rigor.

Mechanism

Meat tenderization following rigor mortis is an important process because it imparts to the meat its final texture and flavour immediately before consumption. The important changes takin place during the process includes the following

1. The water holding capacity is increased
2. The level of water insoluble non-protein nitrogen namely peptides and amino acid level increase duo to the action of proteolytic enzymes.
3. It is also shown that the resolution of rigor is not related to separation of actin and myosin form actomyosin but is the result of the weakening and disintegration of z-line of A-band of muscle fibre.

Changes produced by naturally occurring bacteria

Microorganisms are found on all the outer surfaces (skin and gills) and in the intestines of live and newly caught fish. The total number of organisms vary enormously. A normal range of 10^2-10^7 cfu (colony forming units)/cm2 on the skin surface. The gills and the intestines both contain between 10^3 and 10^9 cfu/g.When the fish gets into the processing area the bacterial count on the skin is often high. If the fish is not washed well with clean water a lot of bacteria can get in the processing area and contaminate the fish flesh during filleting. The flesh can also get contaminated with mesophilic bacteria from the people in the working area. So personal hygiene is also very important. Highest numbers of bacteria of fresh fish are present in digestive tract, but numbers in outer surface can be increased upto 10^8 during spoilage. Temperature plays very important role in controlling microbial growth. Higher temperatures (around 37°C) can increase the microbial growth.

Main Spoilage bacteria found in fish are *Pseudomonas, Shewanella putrefaciens, Photobacterium phosphoreum, Enterobacteriacea.* Usually Pathogens found in natural environment of fish are *Clostridium botulinum, Listeria monocytogenes, Vibrio parahaemolyticus, V. vulnificus, Aeromonas hydrophila* and *Plesiomonas shigelloides*. Other pathogens that can contaminate from the environment are *Staplylococcus aureus*, *Salmonella* species, *Escherichia coli etc.* In post-rigor stage, bacterial spoilage becomes very fast. Growth of bacteria contributes to the development of spoilage odours because of production microbial metabolites.

The rise of muscle pH from acidic to alkaline range for accumulation of volatile bases like ammonia and trimethylamine produced by spoilt fish enhances bacterial growth. Trimethylamine is produced by the reduction of trimethylamine oxide by bacterial enzyme as well as by tissue enzymes.