Coping with the PSE syndrome in poultry meat

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Introduction:

PSE syndrome regards to meat defects. It is generally described as a major defect affecting the colour (paler than normal), the texture (softer than normal) and the water holding capacity (lower than normal) of the meat. All these quality issues are becoming a concern to the industry.

PSE syndrome has first been described in pork mainly since the beginning of the 60's and more recently (in the 90's) in poultry, essentially in turkeys and chickens. In may 2007, a request (over the last 20 years) as "PSE meat" on the PubMed web site (http://www.ncbi.nlm.nih.gov/entrez/) gave one hundred responses: 2/3 of them concerned pig and the remaining 1/3 poultry with 15 papers concerning turkeys and 15 concerning chickens.

If the syndrome seems to be well described and understood in pork, it is not the same case in poultry because no definitive paper has yet been published to give a clear ethology of what is sometimes still called PSE-like syndrome.

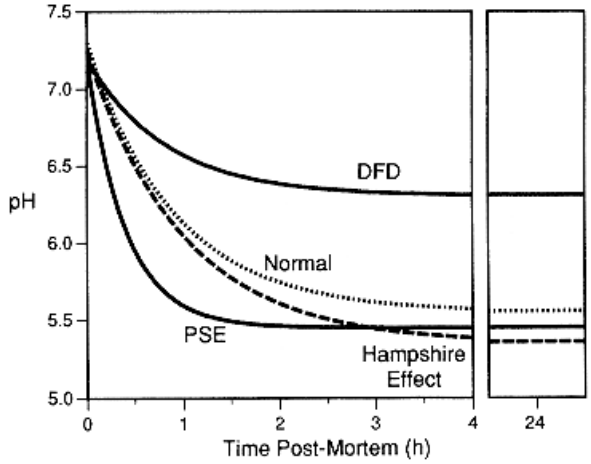
In this paper, we will try to draw what seems to be more or less clear with the PSE syndrome in poultry and first try to give an appropriate definition of it. Then, we will examine all the known consequences of its presence on meat quality and the related consequence for poultry industry. Finally, we will examine some possible hypotheses which have been investigated to explain its presence within certain flocks of birds.

1 - What is a PSE meat ?

Muscle to meat transformation.

At the moment of death, muscle tissue undergoes anoxia due to the blood deprivation consequent to bleeding operations. To try to maintain its homeostasis, muscle continues to produce ATP with its only available metabolic pathway to produce energy: the anaerobic glycolysis. This metabolic process, that breaks down carbohydrates and sugars through a series of reactions, produces ATP and lactate which accumulates and produces the acidification of the muscle tissue. In normal cases, the final pH of the meat (pHu) is around 5.6-5.7 but it can also be lower (Hampshire effect) or higher (DFD meats). All these abnormal values concern defects of the amplitude of pH fall and have several consequences on meat qualities. On the other hand, the pH fall can exist with an appropriate amplitude but with an increased speed leading to reach ultimate pH values within a few minutes after death. In slaughter-houses, these minutes correspond to slaughter operations including animal hanging, anaesthesia, bleeding, feather picking, evisceration, etc… that is to say all the operations done previously carcass cooling. In consequences, a low muscle pH value will be encountered while muscle temperature is still high, leading to a final combination of low pH and high temperature values. These physico-chemical conditions are largely unfavourable to maintain muscle proteins integrity and will therefore cause protein alterations leading to water excessive release, loss of protein functionality and meat texture alterations.

Figure 1. Effect of time *post-mortem* on pH for several meat quality types (Murray, 1995)



In consequence, PSE meats are necessary associated with an increase of the speed of pH fall while muscle temperature remains normal or higher than normal. The maintenance of this relatively high body temperature is due to scalding operations, post-mortem persistence of metabolisms and to limited cooling rates of carcasses to prevent cold shortening of muscle fibres. It is commonly admitted that, 20 min after bleeding, a muscle pH value around 6.0, or below, and a muscle temperature around 35° C, or higher, are sufficient to finally produce PSE meats. From the Figure 1, it must also be retained that PSE meats do not present an abnormal pHu value. This make them difficult to be identify in processing plants where they are often mistaken with meats presenting an abnormally low pH value.

Acceleration of post-mortem glycolysis.

Anaerobic glycolysis is normally present in muscles to provide ATP when dioxygene is lacking. In that case, when one molecule of glucose is degraded, 2 molecules of ATP and 2 molecules of lactate are produced. Glucose is present in few quantities within the muscle cell but is largely available from the degradation of glycogen. Thus, glycogen represents the stock of local energy of muscle fibres. It is present in large quantities in white muscle fibres and to a restricted extend in red muscle fibres. The speed of glycogen breakdown will direct the speed of lactate accumulation and consequently the speed of pH fall. Different levels of regulation of anaerobic glycolysis are present in the muscle cell and their modulations will affect the speed of pH fall. The first key level is AMPc content because its increase will activates the phosphorylase kinase A which is requested to begin glycogenolysis. In muscle cell, AMPc is essentially produced from ATP by the adenylate cyclase, an enzyme which can be activated by several factors such as hormones or calcium. Regulations by hormones can be evoked in living organism but are of less interest in our case because they are no more available during *rigor mortis* onset. On the contrary, calcium is largely present in sarcoplasmic reticulum of muscle cells and could therefore plays a central role regulation of post mortem muscle glycogenolysis. It is this hypothesis which is generally used to explain the appearance of PSE meat in porks.

Is regulation of muscular calcium level the central point in all PSE meats ?

In pigs, numerous studies have reported that acceleration of pH fall leading to PSE meats have a genetic determinism (Fujii et al., 1991). This is due to a mutation on a gene (called Hal gene or halothane gene sensibility) coding for the ryanodine receptor which is a calcium-release channel present within the sarcoplasmic reticulum membrane. When this gene is non functional, high quantities of free calcium can be release in muscular sarcoplasm. This increase in Ca2+ of the sarcoplasm enhances the conversion of ATP in AMPc by adenylate cyclase but also activates glycogen phosphorylase A. It is one the first enzyme involved in glycogen conversion into glucose which will fuel glycolysis. This mechanism explains how pH fall can be accelerate only by deregulation of calcium metabolism as in the case of the ryanodine receptor mutation described in pigs sensible to halothane. In turkeys, Sams et al.

(1999) found very small differences in meat quality parameters between halothane positive and negative turkeys. Even if Chiang et al. (2004 and 2003) reported the identification of two αRYR alleles and the characterisation of αRYR transcript variants in turkey skeletal muscle, they concluded that there was no significant relationship between the presence of the transcripts variants and meat quality. An other, excessive release of Ca2+ associated to PSE meats has also been reported by Soares et al. (2003) in chickens and by Cheah et al. (1995) in pigs, due to enhances activity of phospholipase A2. On the contrary, Molette et al. (2005) reported that high post mortem muscular glycolysis in turkeys could be due to direct modifications of enzymes of the glycolysis and not necessary to modifications in calcium metabolism. Nevertheless, even if the post-mortem process leading to PSE syndrome in poultry is similar to pork, the fundamental biochemical events that lead to its development are not well established. We must hypothesis that a new biochemical mechanism of PSE meat development in poultry remains to be proposed to well understand the fundamental origins of its development.

2 - What are the characters of PSE meats?

As suggested above, PSE meats can easily by mistaken with meats presenting abnormally low pHu values. This is due to the fact that, in the two cases, observed meat defects can be very similar: the colour of the meat is supposed to be paler than normal, the water released during meat storage or processing is higher than normal and the texture of the meat is more or less altered. It must also be considered that meat can present the combined characters of an increased speed of pH fall associated to a pHu value lower than normal.

The colour of poultry PSE meats.

The colour of the meat is very important for a consumer because it is the only sensorial quality of the product that it can easily appreciate when it makes its choice in the meat store. The colour of the poultry meat must not be too pale or too dark but also very homogenous. This colour can be appreciated by visual judgement (not easy to perform and relatively long and expensive) but also by physical measurements done with a chromameter. This last mean has the favour of processing plant managers because it is easy to perform, rapid, non invasive and relatively cheap. The pale colour of the PSE meat is associated to an increase of the L* (lightness) value in pig (Fisher et al., 2000). In poultry, results are less consensual when PSE meats are identified on the basis of low early pH values. Pietrzak et al. (1997) reported higher L* values in PSE turkey meats while Rathgeber et al (1999), Hahn et al (2002) or Molette et al (2002) did not find differences in L* values between fast and normal glycolyzing muscles. On the contrary, Zhang and Barbut (2005) in chickens and Barbut (1998, 1996, 1993) in turkeys proposed to use high L* value (>51-53) to detect PSE-like meats characterised by paler colour and altered water holding capacity and texture. However, it is noticeable that all these studies were done with meat samples collected 24h post mortem when only ultimate pH could be recorded. Therefore, a negative correlation between L* and ultimate pH values was reported indicating an other origin of the colour defect rather due to an abnormal amplitude of pH fall than to an increase of the speed pH fall. More recently, Fraqueza et al. (2006) failed to report a relationship between pH_{15} and L* values samples but confirm the relationships between excessive L* (≥50) and low pH_{24} (<5.8) values in PSE-like turkey breast meats.

The texture of the meat.

In pork, as in poultry, modifications of the texture is associated with PSE meat (Mc Kee and Sams, 1997). These modifications give a meat which appears to be softer prior cooking and tougher after cooking. The texture of the meat is mainly defined by three components (Maltin et al., 2003) which are the length of sarcomeres, the quantity and quality of the muscular collagen and the level of muscular proteolysis. According to several authors, the two first components of meat texture seem not to be modified in PSE meats. On the contrary, delayed or lack of maturation due to alterations of muscular proteolytic enzymes are reported in PSE meats from pork (Monin et al., 1999) and from turkeys (Popiesch et al., 1992). In that case,

authors observed, in PSE samples, limited alterations of muscular proteins post-mortem which are hypothesised to be due to a lower activity of modified proteolytic enzymes because of alterations of their structure and/or limited access to their substrates. This hypothesis is in good accordance with all the observed alterations of muscular proteins extractabilities due to the combination of a high temperature and a low pH in PSE muscles. Unfortunately, until today, no detailed investigations have been conducted on mechanisms of maturation in PSE poultry meats.

The water holding capacity of PSE meat.

The water holding capacity of meat is essentially due to proteins. In consequence, it will be altered by modifications of the muscular proteome due to PSE development. In PSE meats, the exudative condition is due to the fact that proteins are incapable of holding water because of the decrease of the space between actin and myosin (Offer, 1991). The loss of water begins naturally during the onset of rigor mortis and continues during meat storage. In poultry, loss of water within the first 24 h post slaughter is multiplied by 1.3 to 7 fold in PSE meats (Mc Kee and Sams, 1997, Owens et al., 2000). If the losses of water are important during storage they will be consequently reduced during cooking but PSE meats have still a lower cooking yield than normal ones. If PSE meats are further processed (by marinating or dry-cured transformations), their processing yields will also be lowered when compared to non PSE ones. Because it affects economic loss for the industry, but also the safety of the product, this huge reduction in water holding capacity of PSE meats makes them undesirable for meat processors.

3 - What are the PSE meat origins in poultry?

From a practical point of view, this question remains the most important for the poultry industry because its answers will necessary describe conditions of production and/or slaughtering that must be followed to avoid PSE meat appearance. Unfortunately, we must admit that, for the moment, no one is able to describe such conditions and only few recommendations can be drawn. Nevertheless, in pigs, even if the ethology of the PSE syndrome is clearly established, the problem has not yet been completely solved because it has also been demonstrated that best growing performances are generally found in animals carrying Hal modified genes.

Frequency of PSE meat in poultry.

Despite its recognised negative effects on meat quality, few detailed surveys of poultry PSE meats occurrence in commercial conditions have been reported. The major difficulty of this type of study remains in collecting data because it implies to measure early pH values in slaughter houses when it is not easy to do . Some studies have been conducted in North America (Barbut, 1996, Sams et al. 1999, Owens et al., 2000, Woelfel et al. 2002) or more recently in Europe (Fraqueza et al., 2006). Most of these studies are based on L* value measurements at different times post-mortem even if this character is not necessary sufficient to detect PSE meat in poultry (Molette et al., 2002). According to these studies, PSE meat occurrence can vary from 5 to 40 % within a flock. If the first number is retained, this low value might not represent a true problem for the processors because solutions can be found in sorting defective meats or by mixing them with normal ones (dilution of the problem). On the contrary, a higher occurrence represents a huge problem because no economically acceptable solution is available.

Possible origins of PSE meats in poultry.

Genetics ?

Based on the results obtained in pigs, the genetic origin of the PSE meat defect in poultry has probably been the most followed lead. We have seen above that the presence of αRYR transcript variants in turkey skeletal muscle failed to explain the appearance of PSE meat defects. Because PSE meat defects in poultry have been described recently, it has been

supposed that this syndrome could be associated with modern lines of birds selected for increased growth rate and/or beast meat yield. Fernandez et al. (2001 and 2002) in turkeys and Berri et al (2001) in broilers have compared the characteristics of the meat coming from lines differing in their growing rates. Both of these studies showed that increased growth rates are associated with a decrease of the speed of pH fall which is the opposite of what is observed in PSE meats syndrome. For Le Bihan-Duval et al. (2001) genetic correlations between pH_{15min} and body weight or breast meat yield in fast growing broilers are very low (0.07 and 0.13 respectively) which does not strengthen the idea of a negative effect of an increased growth rate on the quality of the meat.

Feeding programs ?

Very few studies have investigated this field in poultry while in pig magnesium, sucrose and vitamins have been reported to have an effect on the development of the PSE syndrome. Nevertheless, Olivo et al. (2001) reported that increasing vitamin E in the feed of chickens is useful to prevent proteins denaturation and therefore susceptible to decrease PSE syndrome effects on meat quality.

Ante-mortem factors ?

Most of these factors are close to the moment of slaughter and their influences on the process of muscle to meat conversion have been largely studied.

The influence of the season, and more generally of an increase of the outside temperature before slaughter, on the occurrence of the PSE syndrome in poultry have been documented (Soares et al., 2003, Debut et al., 2003, Olivo et al., 2001, Owens et al., 2000, McKee and Sams, 1997, Northcutt et al., 1994). Despite these numerous reports, it remains difficult to identify this environmental factor as one of the determining factor of PSE meat in poultry because some studies report a negative influence on some meat quality parameters (mainly colour and water holding capacity) while others report no influences at all. One more time, if a chronic or an acute thermic stress necessary gives PSE meats in pork, it seems not to be the case in poultry.

The duration of transport from poultry house to the abattoir is an other source of ante mortem stress susceptible to favour PSE syndrome development. Nevertheless, Owens and Sams (2000) and Debut et al. (2003) failed to demonstrate such an influence in poultry.

The duration of rest before slaughter is generally short in poultry slaughterhouses. This is probably the reason why its influence on the development of the PSE syndrome has not been widely studied in chickens or turkeys. In broilers, it seems that a time of rest of 4 hours do not modify meat colour and texture (Kannan et al, 1997).

Slaughtering and post mortem conditions?

To avoid pain all the animals killed in the EC must be anaesthetised before bleeding. In poultry, this can be done with a water-bath (electronarcosis) or by gaseous systems. Both of those systems have been suspected to increase the frequency of PSE meats in chickens or in turkeys but the results published by Froning et al., (1978) or Northcutt et al. (1998) failed to demonstrate it. Because the persistence of a high temperature in the muscle during the onset or *rigor mortis* is one of the two conditions necessary to PSE syndrome development, it is clear that the cooling rate of the carcasses can influence the occurrence of PSE meats. However, this cooling must not be done too early because it could cause meat defects such as cold shortening. Nevertheless, Mc Kee and Sams (1997), Molette et al. (2003), Alvarado and Sams (2004) and Sams and Alvarado (2004) failed to demonstrate that early chilling significantly modify texture, colour and WHC of poultry meats.

Conclusions:

PSE meat have concerned, for a long time, pigs but the development of similar defects in colour, water holding capacity and texture of poultry meats have conducted scientists to design experiments more or less close to those already realised in mammals. However, and even if there is some similarities, it seems that the ethology of these

syndromes is not based on the same metabolic processes. In the two cases, it is the combination of a low initial pH and a high muscle temperature values which lead to muscular proteins denaturations implied in the PSE syndrome. The nature of these denaturations and the biochemical events leading to acceleration of post-mortem muscular glycolysis in poultry are still unclear. A lot of work must be done in those fields and the use of combined biochemical tools such has functional genomics, proteomics and metabolomics will be helpful. The determination of rearing factors facilitating the PSE syndrome development in poultry will then be more evident and significant recommendations to farmers and meat processors will therefore be edited. To well achieve these goals, it seems important that scientists and professionals collect a maximum of data concerning specifically this syndrome and well distinguish it from other poultry meat defects such as simple variation in colour or ultimate pH values.

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