

## IMPACT OF PSE AND DFD MEAT ON POULTRY PROCESSING – A REVIEW

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In the meat processing the quality of final products greatly depends on the quality of used meat. Poultry processors are faced with problem of defective meat, *i.e.* PSE (pale, soft, exudative) and DFD (dark, firm, dry). The reasons contributing to PSE of poultry meat are still not fully recognised. The PSE located in breast muscles can originate from genetics reasons, result from preslaughter stress, post-mortem slow or inadequate chilling and accelerated rigor mortis processes. The reduction of preslaughter stress, very rapid chilling and sorting of PSE meat from normal may have some use in improving quality of products and substantial elimination of economic losses.

### INTRODUCTION

Two conditions known as pale, soft and exudative (PSE), and dark, firm and dry (DFD) meat can develop in meat as a result of short- and long-term stress, respectively [Lawrie, 1998]. Especially disadvantageous is processing of defective PSE meat, which is characterised by discoloration, unfirm texture and high leakage of muscle juice. The processing value of such meat is limited and moreover cannot be considered as fully valuable culinary meat and directed to retail distribution [Alvarado, 2002]. Similarly, DFD meat with high ultimate pH > 6.3 has restricted application because is prone to microbial contamination even when initially being relatively low microbially-contaminated [Allen *et al.*, 1997]. Until now researches focussed on the estimation of influence of antemortem stressors and postslaughter conditions on quality changes in turkey and chicken breast muscles. Breast meat of glycolytic metabolism has more economical value than thigh meat.

The poultry industry suffers losses from problems referred to as “soft muscle tissue” or “summer yield problem” [Barbut, 1998]. Reliable and unquestionable detection of meat with low technological quality, leading to increased product exudate and inferior texture, is considered to be a significant economical problem in meat processing. The aim of this paper is to present reasons of poultry meat defects, classification system for identifying PSE and DFD meat and methods for reducing its occurrence.

### PHYSIOLOGICAL, PRESLAUGHTER AND POST-MORTEM FACTORS AFFECTING POULTRY MEAT QUALITY

The incidence of PSE in poultry meat depends on the genetics, antemortem and postmortem stressors including: environmental temperatures, transportation, preslaughter handling practices, chilling regimes and rapid onset of rigor mortis [Fletcher, 1992; McKee & Sams, 1998; Solomon *et al.*, 1998].

Poultry, like pigs have been subjected to intensive genetic selection for rapid muscle growth. Genetic selection in poultry resulted in more often observed stress syndrome (PSS – porcine stress syndrome) or myopathic symptoms (disappearance or weakening of muscles). The stress syndrome in poultry breast muscles is caused by mutation in the calcium channel gatekeeper proteins ( $\alpha$ - and  $\beta$ -ryanodine receptors, RYR) that controls the  $\text{Ca}^{+2}$  release from sarcoplasmic reticulum [Percival *et al.*, 1994; Sams, 1999; Solomon *et al.*, 1998]. The fluctuation in the calcium ion amounts accelerates metabolism and rises body temperature. Elevated body temperature is the source of another name for stress syndrome, *i.e.* malignant hyperthermia (MH). The genetic mutation in the RYR gatekeeper proteins causes the bird to be sensitive to the anesthetics such as halothane or depolarising agents such as succinylcholine that are homozygous for the recessive PSS/MH gene (also known as halothane gene). Muscle rigidity, increased body temperature, and increased lactic acid production can characterise

\* This review paper is dedicated to the memory of **Professor Adam Niewiarowicz** – a pioneer of PSE and DFD poultry meat research, who passed away 10 years ago.

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the response to halothane. In turkey muscles sensitive to the halothane test more often occurs incidence of PSE than in halothane tolerant birds [Owens *et al.*, 2000; Sams, 1999].

The rapid muscle growth, especially in turkey, is connected with atrophy of fibers and loss of connective integrity [Sosnicki & Wilson, 1991]. The connective tissue (endomysium) associated with individual muscle fibers cannot keep up with rapid muscle fiber growth and in result is less developed and immature [Swatland, 1990]. The fiber necrosis and focal myopathy (FM) incidence in poultry proves that muscle overgrows its own supporting system. It is not clear, in spite of similarities in the histopathological alterations if FM is the cause of PSE in turkey. FM is the abnormality that exists antemortem whereas PSE develops as a result of both preslaughter conditions and postmortem processes. Therefore, poultry meat can exhibit defaults, which can be rated as PSE or focal myopathy [Solomon *et al.*, 1998].

Among many potential stressors only a few have been evaluated in relation to poultry for their contribution to the development of PSE meat (pale and exudative). The transportation process can be stressful to the birds, and it may, consequently, affect meat quality [Owens & Sams, 2000]. During transportation (especially during heat and humid weather conditions) turkey muscles yield acidification what may be related to the pathological alterations in the muscle, *i.e.* myopathy, which increases in stressful preslaughter handling conditions [Sante *et al.*, 1995; Sosnicki *et al.*, 1998]. Some researches have reported that 3-h transportation immediately preslaughter or 4-h relief after transportation had no effect on turkey or chicken breast muscle quality related to PSE [Kannan *et al.*, 1997; Owens & Sams, 2000; Warriss *et al.*, 1999]. The breast muscles from turkeys transported for 3 h had higher pH and lower L\* values (darker colour) after 0, 2 and 24 h p.m. compared with fillets of the nontransported turkeys. It is suggested that transportation for 3 h may cause stress to the animal that accelerates metabolism to a point of depletion of muscle glycogen, resulting in higher muscle pH [Owens & Sams, 2000]. Preslaughter 10-h feed withdrawal did not have influence on glycogen content and the ultimate pH of chicken breast muscles [Savenije *et al.*, 2002; Warriss *et al.*, 1993, 1999], while turkey fasting for 15 h resulted in increased muscle pH and water holding capacity (WHC) and reduced shear force (SF) compared to the fed group [Ngoka *et al.*, 1982].

Prolonged, seasonal-type heat stress (day/night 38/32°C) subjected to turkeys was the direct reason of breast muscles higher acidification, lighter colour, higher drip and cook loss when compared to the muscles of unstressed birds [McKee & Sams, 1997]. The breast muscles of chicken exposed preslaughter to heat at 40 to 41°C for 1 h had paler colour and higher drip loss than muscles of control birds [Northcutt *et al.*, 1994]. Boulianne and King [1995] noticed higher L\* values (lighter colour) for chicken breast muscles in relation to control at simultaneously higher their alkalization. Paradoxically, this study found a relationship opposite to that observed in DFD meat, namely that redder chicken meat had a correspondingly lower pH.

The postmortem muscle temperature influences the dynamics of rigor mortis and meat quality. The elevated temperature (37 to 41°C) during processing (15–20 min

after slaughter) accelerates ATP depletion and degradation of glycogen to lactic acid (increased rate of metabolism) and produces pale, exudative meat characteristics in turkey and chicken breast muscles [McKee & Sams, 1998]. However, rigor development at 40°C increased turkey meat toughness.

The breast and leg muscles from emaciated birds or submitted to various preslaughter stressors had: darker colour, elevated pH, almost completely depleted glycogen and their SF was similar or higher than for meat from normal-coloured chicken breast fillets or unstressed birds [Boulianne & King, 1998; Fletcher, 1999; Niewiarowicz *et al.*, 1977, 1978; Walker & Fletcher, 1993]. Stress is the factor which accelerates metabolism and quick exhaustion of muscle glycogen supplies. In result glycolysis in postmortem muscle is not occurring or is slowed down because the level of substrate (glycogen) of this process undergoes substantial exhaustion [Lawrie 1998; Owens & Sams, 2000]. The Canadian researches also qualify the chicken and turkey grey-blue breast muscles from carcasses condemned for cyanosis to DFD meat [Mallia *et al.*, 2000, 2000a]. The results of Boulianne and King [1995], who examined the relationship between turkey meat colour and pH (higher pH of paler turkey breast), did not support the presence of a DFD like condition in inspected poultry meat.

Slow or inadequate chilling temperatures (30°C lub 40°C) can contribute to development of PSE in normal or fast glycolysing poultry breast muscles. This is more of the problem for turkeys due to their larger size and slower chilling rate. Therefore, rapid chilling of poultry carcasses in which occurs a rapid postmortem pH decline may, in some extent, reduce the risk for PSE meat characteristics [Alvarado & Sams, 2002; McKee & Sams, 1998; Offer & Trinick, 1991].

Incidence of PSE in poultry breast muscles may be a consequence of an accelerated rate of muscle glycolytic metabolism before slaughtering due to stress-related genetic factors. In pale turkey muscles, the rate of glycolysis is nearly two times higher than in normal muscles [Sosnicki *et al.* 1998]. It appears that the combination of antemortem stress sensitivity and glycolytic metabolism in turkey and chicken breast muscles results in accelerated rigor mortis process. The onset of rigor mortis in turkey breast muscles (ATP <1.0  $\mu\text{mol/g}$ ) may occur at pH of about 5.7 already after 20–30 min p.m. [Pietrzak *et al.*, 1997; Sosnicki *et al.*, 1995]. Accelerated rigor mortis development with poor WHC and light colour is indicative of PSE meat.

Pale, soft, exudative broiler breast meat appears different from PSE in turkey, wherein protein denaturation does not seem to be the main cause of paleness and low WHC [Van Laack *et al.*, 2000].

#### CLASSIFICATION SYSTEM FOR IDENTIFYING PSE AND DFD MEAT

Establishing a cut-off point at which it is observed lower or higher pH, L\* value or WHC than for normal meat may be used to categorise poultry breast fillets in terms of quality into PSE, normal and DFD. The pH and lightness (L\*) values in the pectoral muscle used to estimate the prevalence of PSE and DFD are presented in Table 1.

For turkey and chicken breast muscles condemned for cyanosis pH 6.1–6.2 and L\* = 45 (at 30 min p.m.) and pH 6.3

TABLE 1. Boundary of pH and lightness (L\*) values for PSE and DFD incidence in poultry breast muscle.

Breast muscle	Time p.m.	pH	L*	Reference
<b>A:PSE</b>				
young turkey <sup>a</sup>	24 h		>50/51	McCurdy <i>et al.</i> [1996]
young turkey	24 h		>53	McKee and Sams [1997]
young turkey	20 min	≤5.8	49	Pietrzak <i>et al.</i> [1994, 1997]
mature turkey hen <sup>b</sup>	24 h		≥52	Barbut [1997]
turkey	24 h		54.7	Owens <i>et al.</i> [2000a]
chicken	24 h		>49	Barbut [1997a]
chicken		5.9	>51	Bauermeister <i>et al.</i> [2000]
chicken	15 min	<5.7		Niewiarowcz <i>et al.</i> [1977, 1978]
chicken	24 h		>57	Wilkins <i>et al.</i> [2000]
chicken	24 h		>53/54	Woelfel <i>et al.</i> [1998, 2002]
<b>B:DFD</b>				
chicken			<45	Allen <i>et al.</i> [1998]
chicken			<48	Bauermeister <i>et al.</i> [2000]
chicken	15 min	>6.4		Niewiarowcz <i>et al.</i> [1977, 1978]
chicken	24 h	≥6.3		Wilkins <i>et al.</i> [2000]

<sup>a</sup> young turkey tom 17–18 weeks, <sup>b</sup> mature turkey hens 8–14 months

and L\* = 39–40, respectively, could be recommended as the cut-off points for pH- or L\*-based test for DFD [Mallia *et al.*, 2000, 2000 a]. The choice of lower value of lightness (L\*) can result in accepting more PSE meat in processing. If a higher value of lightness for young turkeys is used (such as L\* > 51), a lower by 6–17% proportion of the meat will be classified as PSE [Barbut, 1996].

#### INCIDENCE OF OCCURRING PSE AND DFD IN POULTRY BREAST MUSCLES

In Canadian poultry industry, the incidence of PSE in young turkey breast muscles ranges from 18 to 34%, in mature turkey hens from 5 to 41% and in chicken broilers from 0 to 28% [Barbut, 1996, 1997, 1997a], while about 10% of slaughtered chicken population exhibits cyanosis and DFD problem [Mallia *et al.*, 2000b]. In the USA, the occurrence of defective (PSE) turkey breast muscles is 30–41% [Owens *et al.*, 1998, 2000a] and it is 37–47% in chicken broilers [Woelfel *et al.*, 1998, 2002]. In England, occurrence of PSE is about 20% of examined chicken broilers [Wilkins *et al.*, 2000]. In Poland, the average PSE and DFD chicken meat was estimated at 6–20% [Niewiarowcz *et al.*, 1977]. Fletcher [1999] reported that approximately 7% of packages with four broiler breast fillets per package had one or more fillets that was noticeably different in color from the other fillets in the same package.

#### MICROBIOLOGY OF PSE AND DFD MEAT

The microbial resistance of chicken PSE and DFD muscles differs depending on pH, being lower for DFD muscles. The low pH of muscles is undesirable for microflora, mainly proteolytic, and extends the shelf-life of product [Allen *et al.*, 1997]. The elevated pH does not accelerate the growth of spoilage organisms but reduces the lag phase or the time that the spoilage organisms are preparing for growth [Allen *et al.*, 1997]. It is indicated by similar psychrotrophs population (specifically *P. fluorescens*) in

chicken breast muscles of PSE and DFD group stored for one and seven days and by a faster rate of objectionable odours produced in DFD than PSE chicken fillets. There is the lack of similar information for turkey breast muscles. It has been only shown that microbiological profile (salmonella, coliform, *E. coli* and aerobic plate counts) and histopathological profiles of meat from turkeys condemned for cyanosis were not different from carcasses that passed inspection. Therefore, carcasses with cyanosis should be suitable for human consumption and favourable for further processing [Mallia *et al.*, 2000, 2000b].

#### METHODS FOR REDUCING THE OCCURRENCE OF PSE- AND DFD-LIKE MEAT

Similarities in PSE reasons and results were detected between turkey breast muscles and pork muscles more than 20 years ago. However, no attempt was made to assess the magnitude of the problem, or to look for ways to separate PSE from normal meat. The problem received new interest in relation to guarantee possibly the highest quality of meat and processed poultry meat [Alvarado & Sams, 2002; Rachwał, 2000; Sams, 1999; Woelfel *et al.*, 2002]. The economic losses in Poland in 1999 year from processing of defective PSE chicken and turkey breast muscles into two types of products revealed 0.76% and 4.76% loss, respectively, in relation to the livestock value [Lesiów, 2001].

The future has both long- and short-term strategies for the problem of stress susceptibility and PSE poultry meat. Decreasing the genetic reasons causing PSE meat requires long lasting research and is remote in time. By limiting environmental stressors it is possible to decrease the incidence of defective meat but it will not fully eliminate this problem. Therefore, limiting of stressful factors during breeding, preslaughter and rapid chilling postmortem methods should be the main goal of breeders and food processors [Sams, 1999].

If PSE meat could be sorted before further processing, the PSE meat could be used with special formulations that

contain ingredients or conditions to restore meat quality (colour) and protein functionality to reduce yield losses and to improve texture [Bauermeister *et al.*, 2000; Duda, 1998, 1998a; Owens *et al.*, 2000, 2000a; Sams, 1999; Słowiński & Mroczek, 1997]. Up to now, the efforts to rectify the protein functionality losses imparted by the PSE condition, pre-rigor injection or curing chicken breast muscles with brines containing special formulations have been unsuccessful [Allen *et al.*, 1998; Alvarado & Sams, 2000; Woelfel & Sams, 1999]. However, transglutaminase added in amount of 0.6% to the curing brine may be effective in reducing the economic losses associated with poor protein binding characteristics of PSE meat in deli loaves [Bauermeister *et al.*, 2000].

Mixing PSE meat (about 10%) with normal meat may allow managing of protein and other nutrients contained in defective meat [Barbut, 1998; Duda, 1999 (personal announcement); Pospiech, 1997]. Because of high pH, the DFD chicken muscles, are microbiologically unstable and should be rapidly manufactured into heat-processed products.

## CONCLUSION

Reducing stress, rapid chilling and different processing methods to regenerate protein functional properties, especially WHC of sorted out PSE meat may need to be implemented to decrease economic losses and improve effectiveness of poultry industry. Estimation of pH value 15–20 min post mortem as well as lightness ( $L^*$ ) may be used for identification of chicken and turkey PSE meat.

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**PROBLEM WAD MIĘSA TYPU PSE I DFD W PRZEMYSŁE DROBIARSKIM – ARTYKUŁ PRZEGLĄDOWY**

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Celem pracy było przedstawienie aktualnego stanu wiedzy na temat przyczyn wad mięsa typu PSE i DFD w przemyśle drobiarskim, metod obiektywnej oceny jakości mięśni (pomiar pH i jasności barwy), potencjalnych strat wynikających z przetwarzania mięsa PSE oraz sposobu zagospodarowania wadliwego mięsa. Czynniki genetyczne, stres przedubojowy, nieprawidłowe poubojowe wychładzanie, gwałtownie następujące stężenie pośmiertne skutkują tym, że mięso może wykazywać wady typu PSE lub DFD. Ograniczenie czynników stresogennych przed ubojem, stosowanie szybkiego poubojowego wychładzania oraz oddzielenie mięśni z wadą mięsa PSE i DFD od mięśni normalnych, może zagwarantować lepszą jakość wyrobów, racjonalne zagospodarowanie wadliwego mięsa, a w konsekwencji podnieść efektywność przedsiębiorstwa przez właściwe wykorzystanie surowca.