INDUCTION OF DPM CHANGES IN BROILER CHICKENS
AND CHARACTERISTICS OF MYOPATHY SYMPTOMS

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Abstract

The aim of the study was to induce deep pectoral myopathy (DPM) lesions in 42 to 45-day-old broiler chickens of the Ross 308 and Flex genetic lines, kept under standard intensive breeding management conditions applied in Poland, and to assess the degree of myopathy. A total of 110 and 120 carcasses, respectively, were examined. The study method consisted of the exposure of the birds to a stress factor (forced wing flapping) at specific time intervals prior to slaughter and at different durations of the stress factor. As a result of the conducted experiment, DPM symptoms appeared in the examined chickens. The symptoms of the progressing anomaly were divided into four stages. The first stage was characterised by the occurrence of bloody extravasations, stage II was characterised by a pale pink colour of muscles, stage III – greening of the muscle tissue, while stage IV was connected with necrosis and white-grey-green colour of muscles. The application of the forced wing flapping several days before slaughter (1, 3 d) resulted in the incidence of earlier symptoms of myopathy – stages I and II. Stimulation of wing flapping 5, 7, and 14 d before slaughter caused subsequent DPM stages (i.e. stages III and IV). In the group of the youngest birds subjected to the stress factor at 21 d before slaughter, DPM lesions were not found. Moreover, the longer the duration (15-60 s) of the stress factor, the greater the intensity of this phenomenon was observed. Bilateral DPM symptoms occurred more frequently than unilateral symptoms. Recorded results show that increased wing flapping is a significant factor inducing DPM in 42 to 45-day-old broiler chickens. These investigations indicated a possibility to determine the degree of DPM lesions depending on the passage of time from the induction of the anomaly to the slaughter of birds.

Key words: broiler chickens, deep pectoral myopathy, stress, wing flapping.

Deep pectoral myopathy (DPM) is a degenerative myopathy of pectoralis minor muscles, also referred to as tender muscles. The myopathy is characterised by changes in colour and texture of the muscle tissue.

The anomaly was observed for the first time on turkey breeding farms, in turkey hens from breeding flocks in Canada, the USA, and U.K in 1967-1968. In 1968, it was described by Dickinson (4) and named as degenerative myopathy. In Poland, cases of DPM in turkeys have been observed starting from 1991. The anomaly was recorded in breeding turkey hens, i.e. heavy and medium-heavy layer hens (51-54 weeks old) to be slaughtered after the completion of the reproduction period. Among 26,169 examined carcasses, DPM symptoms were detected in 15.6% of all analysed birds (8). Data show that the scale of this phenomenon is a considerable entity. In broiler chickens myopathy of pectoralis minor muscles has been observed in recent years (1, 2, 6, 7).

Aetiology of DPM has not been thoroughly clarified. Intensive genetic selection towards rapid weight gain of birds, a high proportion of pectoral muscles in the carcass, as well as intensive management systems applied for these birds are indicated as primary causes of muscle susceptibility (1, 2, 3, 6, 7, 9, 11). DPM was not found in extensive organic, ecological, or “label rouge” management systems, in which other genetic lines of birds, with slower growth rates, are used. In turn, a factor stimulating and inducing DPM symptoms is probably related with the birds being bothering, scared, or otherwise made to be involved in intensive wing flapping several days before slaughter (1, 3, 5, 11). It was proven experimentally that a relatively short period of wing flapping is sufficient to induce DPM symptoms (3). In the course of effort caused by wing flapping or birds being bothered during e.g. preventive vaccination, weighing, etc., the pectoralis minor, which is responsible for raising wings, contracts, and swells considerably. According to Siller (11) the pectoralis minor muscle increases in volume by 20% or by as much as 25%, as it was reported by Bilgili and Hess (3). Excessive pressure presses on blood vessels, causing their occlusion. Suppression of the blood flow and thus also oxygen to myoglobin as a consequence leads to anoxia and tissue necrosis (10). The anomaly affects valuable carcass elements, causing serious economic losses in poultry processing plants. For this reason it is essential to gain insight into the
mechanism of DPM development and to determine methods preventing the myopathy.

The aim of the study was to verify or confirm the hypothesis that DPM of pectoralis minor muscles in broiler chickens kept under standard management conditions applied in Poland may have been induced by forced wing flapping in experimental birds, as well as the evolution of characteristic traits of muscles affected by this degeneration in time.

**Material and Methods**

The experimental material comprised pectoral muscles of 42-day-old broiler chickens of the Ross 308 genetic line and 45-day-old chickens of the Flex genetic line. The chickens were kept in a typical animal housing facility of a selected commercial farm. The birds were kept on litter (in pens) under standard environmental conditions. Mechanical exhaust ventilation, watering and feeding systems were automated. A standard feeding regime was applied. Temperature in the brooder house was reduced on average from 33°C in the 1st week of life of chickens to 18°C in the 6th week. The total number of examined chickens was 110 in experiment 1 (Ross 308 line) with 10 birds in each group and 120 in experiment 2 (Flex line) with 12 birds in each. The birds were divided into 11 (experiment 1) or 10 (experiment 2) equal groups. The performed experiments were approved by the Local Ethics Commission for Experimentation on Animals. The experiments were conducted according to the procedure originally developed in cooperation with Professor S. Bilgili (USA) within the framework of our cooperation with Auburn University, Alabama, USA.

The experiments were conducted as follows. In the first part (a) of experiment 1, forced wing flapping lasting exactly 45 s was induced in chicken of different age, that is, at different time before slaughter, ranging from 21 to 1 d (Table 1a). In turn, in the second part (b) of this experiment birds at the same age were exposed to the same stress factor 3 d before slaughter, acting in individual groups for 15, 30, 45, and 60 s (Table 1b).

The last group comprised the control, in which physical activity was not forced. In the experiment 2, the first group of birds was excluded, since it resulted from the experiment 1 that chickens were too young for DPM symptoms to be initiated in them. Moreover, in the experiment 2, the number of wing flaps (the complete wing movement upwards and downwards) was controlled, corresponding to the four durations of stimulated wing flapping. Table 1 presents a design of the performed experiment.

**Results**

On the basis of thorough observations, the development of DPM was divided into four stages depending on changes in colour, colour intensity, and texture of the muscle tissue. The 1st stage of DPM was characterised by the occurrence of haemorrhages and clotted blood in the vessels (Fig. 1). Stage II of myopathy was manifested by the pale pink colour of the pectoralis minor muscle and necrotic and fibrotic lesions in muscle tissues (Fig. 2). Stage III was characterised by the greening of the muscle tissue, first of all in the central part of the muscle (Fig. 3). In stage IV, the muscle was completely affected by necrosis and took white-grey-green colour (Fig. 4).

Frequencies of individual DPM stages are presented in Figs 5, 6, and 7. The colours in these figures for individual DPM stages are approximate representations of natural colour of muscles.

As a result of the experiments conducted on the farm, DPM lesions were initiated in the pectoral muscles. In the experiment 1, the lesions occurred in 39% of the analysed muscles (Fig. 7a). However, in comparison of the results from the experiments 1 and 2, with the first group of birds being excluded, as it was the case in the experiment 2, chickens with DPM symptoms accounted for 43% of population. In the experiment 2, the myopathy occurred in 64% of the analysed pectoralis minor muscles (Fig. 7b).

**Table 1**

**Experimental design**

<table>
<thead>
<tr>
<th>Part 1 (a)</th>
<th>Part 2 (b)</th>
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<tr>
<td><strong>Group</strong></td>
<td>Duration of stress factor (s)</td>
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n.a. – not applicable
Fig. 1. Deep pectoral myopathy – stage I.

Fig. 2. Deep pectoral myopathy – stage II.

Fig. 3. Deep pectoral myopathy – stage III.

Fig. 4. Deep pectoral myopathy – stage IV.

Fig. 5a, b. Occurrence of DPM symptoms in broiler chickens exposed to a stress factor - wing flapping – experiment 1 (Fig. a - part 1, Fig. b - part 2).
Fig. 6a, b. Occurrence of DPM symptoms in broiler chickens exposed to the stress factor - wing flapping – experiment 2 (Fig. a - part 1, Fig. b - part 2).

Fig. 7a, b. Frequency of DPM symptoms in broiler chickens exposed to the stress factor - wing flapping (Fig. a - experiment 1, Fig. b - experiment 2; part 1 together with part 2).

Fig. 8a, b. DPM lesions in broiler chickens subjected to the stress factor - wing flapping at different time before slaughter – part 1 (Fig. a - experiment 1, Fig. b - experiment 2).
Fig. 9. Frequency of DPM in the pectoral minor muscle of tested chickens influenced by duration of stress factor. R=0.73; R²=0.54; the standard error of estimation: 17.79; significance level P<0.038; confidence interval: 0.95.

Fig. 10. Unilateral lesions in pectoral muscles.

Fig. 11. Bilateral lesions in pectoral muscles.

Fig. 12. Pectoralis minor and major muscles with DPM lesions.
Early stages of myopathy, *i.e.* stages I and II, were observed much more frequently than the stages III and IV in both experiments. Moreover, in part I of both experiments, all the distinguished development stages of the anomaly were found. In turn, in part 2 in which 3 d before slaughter an increasing duration (15–60 s) of the stress factor was applied, later stages of myopathy, *i.e.* III and IV, were not detected even at the longest duration of encouraged wing flapping. Thus, the time of 3 d before slaughter is too short for the greening of pectoralis minor muscles to develop.

Results recorded in the first part (a) of the experiments 1 and 2 are presented in Fig. 8. In both experiments symptoms of the anomaly did not develop in the controls. In the group of birds subjected to the stress factor 21 d before slaughter, *i.e.* at the age of 21 d, DPM lesions were not observed. Probably this was connected with the too young age of those chickens, and the resulting smaller weight of pectoral muscles, for myopathy symptoms to develop. Induced wing flapping in birds on days 14 and 7 (in experiment 2 also 5 d) before slaughter caused the occurrence of DPM stage III. The application of the stress factor on 7 d (in experiment 2 also 5 d) before slaughter resulted in the appearance of stage IV of the anomaly. Early symptoms of myopathy stages I and II were observed in chickens subjected to the stress factor 3 and 1 d before slaughter.

The effect of the duration of the stress factor on the intensity of myopathy incidence was analysed in part 2 of the experiments 1 and 2. The longer was the duration of the stress factor within the range of 15 to 60 s, the higher was the increase in the frequency of the anomaly incidence in chickens, which on average amounted from 29.6% to 75.8% of the analysed population. Pearson's correlation analysis (*r* = 0.73) showed a statistically significant positive dependence between the duration of the stress factor and the frequency of the anomaly. This was also confirmed by the analysis of linear regression and the scatter diagram (Fig. 9). Linear regression coefficient was 0.99, which means that if the duration of intensive wing flapping increases by 1 s, the frequency of DPM incidence in broiler chickens may be expected to increase by 0.99%.

Moreover, the longer the duration of the stress factor (15, 30, 45, 60 s), the higher the number of wing flapping movements was observed (12, 24, 33, 40, respectively). This dependence was statistically significant (*r* = 0.94), but it was not directly proportional to the passage of time. It was observed that the increasing duration of the stress factor probably caused greater fatigue of pectoral muscles in birds and in the final period of stress action a progressively lower wing motion activity was noted.

On the basis of macroscopic examination of pectoral muscles it was stated that myopathy of pectoralis minor muscles may occur bi- and unilaterally (Figs 10, 11). The symptoms appeared bilaterally with a greater frequency in the experiment 1, amounting to 85%, while in the experiment 2 it was 81%.

DPM lesions affect not only minor, but also major muscles (Fig. 12), which definitely constitutes a novel finding. Lesions in superficial muscles in the experiment 1 occurred in 76%, and in the experiment 2 in 84% of major muscles, corresponding to minor muscles with DPM symptoms. Extravasations were observed on these muscles, accompanied by grey-green colour of certain areas of tissues.

**Discussion**

Deep pectoral myopathy found in gallinaceous poultry, first of all in adult turkeys/turkey hens from breeding flocks, in recent years has appeared in many countries including Poland, affecting very young, 6 to 7-week-old broiler chickens (6, 7). The primary cause of this phenomenon is connected with the intensive genetic selection and breeding towards rapid weight gains, primarily concerning pectoral muscles (1, 2, 3, 6, 7, 9, 11). Such human interference with nature and animal physiology, being the result of economic consideration, may not be freely conducted with any consequences whatsoever. Such an activity results in the occurrence of numerous muscle pathologies, including myopathy of pectoralis minor muscles. On the basis of the experiments conducted on the farm, it was shown that the key role in the induction of DPM lesions in 42-, 45-day-old broiler chickens is played by the response of the chickens to a stress factor such as sudden wing flapping. In case of the experiment 1, the frequency of myopathy (43%) was lower than in the experiment 2 (64%). This might have resulted from the use of different genetic lines of chickens (Ross 308 vs. Flex) and different ages of chickens (42 and 45 d, respectively), and thus different body weights of chickens and weight of pectoral muscles. Moreover, the effect of time from the moment of anomaly initiation to the time of slaughter on the degree of advancement of DPM lesions in pectoral muscles was also determined. It was found that exposure to the stress factor at 5, 7, 14 d before slaughter results in greening of minor muscles. Forced wing flapping at 5 and 7 d before slaughter (weight of chickens of approx. 2.36 kg and 2.38 kg, respectively) initiates the last DPM stage (IV) in breast muscles of the chickens. It is possible that in this period before slaughter advanced changes in muscle colour and texture occur very rapidly, leading to necrotic lesions, which become white-grey-green in colour. Wight *et al.* (12) found that the diseased muscle becomes superficially green after 9 d and at that time greyish areas may be found. These authors conducted experiments on 20-week-old chickens and it should be remember that in the times of these studies (the early 1980’s) the analysed genetic material differed from the contemporary. It also needs to be stressed that induction of the anomaly at 14 d before slaughter does not cause the occurrence of DPM stage IV, but stage III in both experiments. In that period, birds have a lower body weight (approx. 1.84 kg), and thus also lower weight of breast muscles. The frequency of DPM increases with an increase in body weight of chickens (6, 7). Probably in that period lesions connected with ischemia in pectoral muscles are weaker and occur with lower dynamics. Induction of enhanced wing motion
activity at 5, 7, and 14 d before slaughter caused the appearance of DPM stage III. It needs to be remembered that most authors do no distinguish III and IV stages at the advanced stage of DPM development. In turn, hyperaemia and pale pink muscle colour occur as a result of the action of the stress factor at least 1 to 3 d before slaughter. It was concluded by Bilgili and Hess (3) that the early stage (stages I, II in our study) of the anomaly continued for a few days after the initial event or incident stimulated DPM lesions (3). In the group of chickens subjected to the stress factor at 21 d before slaughter (with slaughter occurring at day 42 of life), no DPM lesions were observed. Probably at this age chickens are too young (have lower weight of breast muscles) for DPM symptoms to be initiated.

On the basis of conducted investigations and recorded results, four stages of anomaly development were distinguished depending on changes in hue, intensity of colour, and texture of the muscle tissue. It was stated that myopathy affects pectoralis minor muscles, but also major muscles, which is a novel finding, and that bilateral symptoms occur more frequently in breast muscles. During sanitary and veterinary inspection at the slaughter line lesions indicating the occurrence of DPM are imperceptible. Detection of myopathy symptoms is possible only during carcass dissection, particularly after trimming of pectoral muscles. Thus it is advisable to provide efficient communication between processors (monitoring of the anomaly at the poultry processing plant) and suppliers (poultry breeders), which will facilitate actions aiming at the control/prevention of the anomaly.

In conclusion, enhanced, intensive wing flapping is a significant factor inducing DPM in 42- to 45-day-old broiler chickens. In order to reduce the incidence rate of the anomaly it is essential to identify and eliminate factors causing intensive wing flapping in birds on commercial farms. Development of individual DPM stages depends on the age of chickens and the time, which has passed from the induction of the anomaly by forced wing flapping to the slaughter. The longer the duration of the stress factor from 15 to 60 s, the significantly greater increase is observed in the frequency of the anomaly in broiler chickens.

References

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