EFFECTS OF IN OVO EXPOSURE TO ACETYLSALICYLIC ACID AND HYPERTHERMIA ON THE HATCHABILITY AND THYROID HORMONE CONCENTRATIONS IN NEWLY-HATCHED CHICKS

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Abstract

The effect of acetylsalicylic acid (ASA) injected in ovo on the 18th d of incubation on the hatchability, course of hatching, and thyroid hormone concentrations (thyroxine (T4) and triiodothyronine (T3)) in the blood of chicks exposed to control (CON) or hyperthermic (HT) conditions during hatching were investigated. Eggs (n=540) from a broiler breeder flock were incubated under standard conditions. On the 18th d of incubation they were injected with 0, 5, or 10 mg of ASA/egg and subsequently incubated at 37.2°C (CON) and 38.5°C (HT). The processes of external pipping and hatching were monitored from the 460th h of incubation, and the embryopathological analysis of the dead embryos was performed. Blood samples from 12 chicks from each group were collected immediately after hatching, and at the 12th, 24th, and 36th h following. The T4 and T3 concentrations in the plasma samples were measured radioimmunologically. There were no significant alterations in the embryonic mortality and chick hatchability between the CON and HT incubation conditions. ASA treatment did not affect the hatching parameters under CON conditions; however, under HT conditions the lower dose of ASA reduced embryonic mortality and increased the hatchability. The hatching of the chicks was accelerated by about 8 h under HT conditions, too. A gradual decrease in T4 and T3 levels in CON and HT chicks was found, following the hatching. In embryos incubated under CON conditions, ASA treatment significantly increased the T4 concentration at the moment of hatching. The opposite ASA effect was found at the 12th h after the hatching, as well as in chicks exposed to HT conditions (both at the moment of hatching and at the 12th h after it). Plasma T3 concentrations in HT conditions were significantly lower at the moment of hatching and at 12th h after hatching. ASA treatment decreased the T3 level at the time of hatching in chicks kept under CON conditions. The results obtained indicate that ASA injected in ovo at the 18th d of incubation does not affect the hatchability under hyperthermic conditions. The results of the thyroid hormone determinations suggest that treatment of the chicken embryo with ASA a few days before hatching reduces the unfavourable effects of heat stress conditions, which may appear in the hatch.

Key words: chick embryo, acetylsalicylic acid, hatch, hyperthermia, thyroid hormones.

Acetylsalicylic acid (aspirin, ASA) is used as a non-steroidal anti-inflammatory drug (NSAID) in human beings and many animal species. ASA is a specific irreversible inhibitor of cyclooxygenase (COX) activity during the synthesis of prostaglandins from arachidonic acid, and therefore it is used as an antipyretic, analgesic, and anti-inflammatory agent. ASA is absorbed completely through the digestive tract. It reaches the maximum concentration in blood after 2 h following treatment, while its half-time in blood is estimated at about 40 h (30, 33, 36).

In poultry production, several indications for the use of ASA have been proposed, including respiratory diseases, digestive coccidial and bacterial infections, inadequate intestinal equilibrium to sustain good weight gain, broiler ascites, locomotor disturbances, stimulation of egg production, improving egg shell quality, and as a compound, which improves the reproductive results (2, 13, 22). Moreover, ASA effectively reduces the body temperature of the chicken (29) and may mitigate the effect of heat stress (16, 19).

Heat stress is of major concern in poultry production. The chicken broilers and adult birds are always exposed to heat stress during very hot summer weather. It leads to increased mortality, and reduces the growth rate in broilers (16, 29). However, the first time when the organism of a chick can be exposed to hyperthermia is the hatching period. This results from the worsening of the micro-environmental condition in the course of hatching in the incubator (23). This always results in many stunted chicks with poorly closed navels and red hock, and later to increased mortality and slower
post-hatch growth. Moreover, disturbances in the function of the cardio-vascular system (myocarditis, damage to the navel and chorioallantoic vessels) and the neglect of immunity were observed (10, 19, 23).

ASA injected in ovo during the last phase of the incubation could protect the chick embryo and/or one-day chick against the unfavourable effects of heat stress in the hatchery, and improve their quality. This assumption is based on the metabolic properties of ASA in the organism, leading to a decrease in the body temperature. In the literature there is no data concerning the effect of ASA on chick embryo development. Therefore, the aim of the present study was to examine the consequence of ASA in ovo injections on the hatchability and hatch course of chicks under hyperthermic conditions. Since thyroid hormones are involved in the reaction to heat stress conditions and play a crucial role during the last phase of chicken embryogenesis (8, 10, 18, 28), their concentrations in the blood plasma of chicks injected in ovo with ASA and exposed to hyperthermia were also investigated.

Material and Methods

Eggs (weighing on average 62.3 ±5.2 g) from the same broiler breeder flock of the Ross 308 line were incubated (Masalles 65 DIGIT) during the first 18 d under standard conditions: T-37.8°C, RH-55%. The eggs were candled on the 5th and 18th d of the incubation, and unfertilised eggs with mortalised embryos were discarded. On the 18th d of the incubation, the eggs (n=540) were randomly divided into three equal groups, and in ovo injected with 0, 5.0, and 10.0 mg of ASA (Polpharma, Poland) dissolved in 50 µl of 0.9% Natrium chloratum (Polpharma, Poland). The administered doses of ASA were calculated on the basis of the daily doses of drinking water for adult hens (0.5 and 1.0 g ASA/L) (3, 13). ASA was injected into the albumin that lets the embryo swallow it before the onset of pipping. Before the injection, the surface of every egg was disinfected with 70% ethanol. A small hole in the shell (φ - c.a. 2 mm) was made with an 18G needle at the big end of the egg, and the injections were performed across the chorioallantoic membrane into the albumin with a 26G needle. After manipulation, the holes were sealed by Parafilm (Sigma, USA). The eggs of each injected group were divided into two equal subgroups (n=90 in each group), and sent to the control and experimental as means ±S.E.M., and considered significant at P<0.05.

Coaparin® (Polfa, Poland) following chick decapitation. The blood samples were centrifuged for 10 min (2,000×g), and the individual plasma samples were kept at −20°C the hormones could be determined. The plasma thyroxine (T4) and triiodothyronine (T3) concentrations were measured radioimmunologically, according to the method earlier described (1, 28). Standard solutions of T4 and T3 were prepared in chicken plasma free from endogenous thyroid hormones. Antibodies against T4 and T3 (Sigma USA) have no, or else very weak, cross reactivities with other iodothyronines. The radioactive iodothyronines were obtained from NEN (Belgium). The lowest limit of sensitivity for T4 and T3 was 1.25 and 0.08 ng/mL, respectively. The intra-assay coefficients of variation for T4 and T3 were 5.8% and 6.2%, respectively.

The results of the pipping and hatching courses were presented as medians and means ±SD and, analysed by the Kruskal-Wallis Three Way Analysis of Variance on Ranks for Failed Normality Test. The received data of each group were demonstrated with a linear regression y = a + bx, where y is the per cent of the pipped /hatched chicks; x stands for the incubation hour; a the intercept, i.e. the estimated start of the pipping or hatching process; b the slope, i.e. the degree of the synchronisation of the pipping or hatching processes, the time (h) necessary to pip or hatch 1% of chicks (17).

The hatchability and embryopathological results were statistically analysed by a z test, while the results of thyroid hormones were analysed by three-way analysis of variance, followed by Tukey’s multiple range test. The statistical analyses were performed using Sigma-Stat 2.03 (SPSS Science Software, USA), while the figures was prepared using Grapher 4.0 (Golden Software Inc., USA). Because the radiomimmoasay revealed that there were no significant differences in the plasma thyroid hormone levels between the male and female embryos during the incubation process, which is in agreement with previous findings (27), the data from both sexes were combined. The results were presented as means ±S.E.M., and considered significant at P<0.05 and highly significant at P<0.01.

Results

The results of the hatching and embryopathological analyses are shown in Tables 1 and 2, while the results of the external pipping and hatching courses are presented in Tables 3 and 4, respectively.
Table 1
Effect of the in ovo injection of acetylsalicylic acid (ASA) and hyperthermia in the hatcher on the hatchability of chicken embryos

<table>
<thead>
<tr>
<th>Item</th>
<th>Standard (control) (37.2°C)</th>
<th>Hyperthermia (38.5°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA dose (mg per egg)</td>
<td>0  5 10</td>
<td>0  5 10</td>
</tr>
<tr>
<td>N</td>
<td>90 90 90</td>
<td>90 90 90</td>
</tr>
<tr>
<td>Embryos dead before external pipping (%)</td>
<td>5.6&lt;sup&gt;a&lt;/sup&gt; 7.8&lt;sup&gt;b&lt;/sup&gt; 4.4&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>5.6&lt;sup&gt;a&lt;/sup&gt; 2.2&lt;sup&gt;ac&lt;/sup&gt; 5.6&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Embryos dead after external pipping (%)</td>
<td>1.1&lt;sup&gt;ab&lt;/sup&gt; 4.4&lt;sup&gt;b&lt;/sup&gt; 1.1&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>2.2&lt;sup&gt;ab&lt;/sup&gt; 0.0&lt;sup&gt;a&lt;/sup&gt; 0.0&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hatched chicks (%)</td>
<td>93.3&lt;sup&gt;ab&lt;/sup&gt; 87.8&lt;sup&gt;b&lt;/sup&gt; 94.4&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>92.2&lt;sup&gt;ab&lt;/sup&gt; 97.8&lt;sup&gt;a&lt;/sup&gt; 94.4&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

a,b,c - the values in the rows marked with different letters differ significantly (P<0.05).

Table 2
Results of the embryopathological analysis of chicken embryos exposed to acetylsalicylic acid (ASA) and hyperthermia in the hatcher

<table>
<thead>
<tr>
<th>Item</th>
<th>Standard (control) (37.2°C)</th>
<th>Hyperthermia (38.5°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA dose (mg per egg)</td>
<td>0  5 10</td>
<td>0  5 10</td>
</tr>
<tr>
<td>N</td>
<td>90 90 90</td>
<td>90 90 90</td>
</tr>
<tr>
<td>Malposition</td>
<td>1  3 4</td>
<td>1  2</td>
</tr>
<tr>
<td>Incomplete retraction of the yolk sack</td>
<td>5  6 4</td>
<td>5  2</td>
</tr>
<tr>
<td>Pain during injection</td>
<td>1  1 -</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 3
Course of the pipping of chicks injected in ovo with acetylsalicylic acid (ASA) and exposed to hyperthermic conditions in the hatcher

<table>
<thead>
<tr>
<th>Thermal conditions in the hatcher</th>
<th>ASA dose (mg per egg)</th>
<th>n</th>
<th>Pipping</th>
<th>Synchronisation of pipping</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>First</td>
<td>Last</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time of incubation (h)</td>
<td></td>
</tr>
<tr>
<td>37.2°C</td>
<td>0</td>
<td>85</td>
<td>482</td>
<td>510</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>83</td>
<td>480</td>
<td>510</td>
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<tr>
<td></td>
<td>10</td>
<td>86</td>
<td>480</td>
<td>510</td>
</tr>
<tr>
<td>38.5°C</td>
<td>0</td>
<td>85</td>
<td>478</td>
<td>510</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>88</td>
<td>476</td>
<td>510</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>85</td>
<td>476</td>
<td>510</td>
</tr>
</tbody>
</table>

a – estimated start of the pipping process; b – the degree of the synchronisation of the pipping processes, i.e. the time (h) necessary to pip 1% of chicks; S<sub>b</sub> – regression error; a,b,c - the values in the columns marked with different letters differ significantly (P<0.05).

Table 4
Course of the hatching of chicks injected in ovo with acetylsalicylic acid (ASA) and exposed to hyperthermic conditions in the hatcher

<table>
<thead>
<tr>
<th>Thermal conditions</th>
<th>ASA dose (mg per egg)</th>
<th>n</th>
<th>Hatching</th>
<th>Synchronisation of hatching</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time of incubation (h)</td>
<td></td>
</tr>
<tr>
<td>37.2°C</td>
<td>0</td>
<td>84</td>
<td>496</td>
<td>522</td>
</tr>
<tr>
<td></td>
<td>5.0</td>
<td>79</td>
<td>490</td>
<td>522</td>
</tr>
<tr>
<td></td>
<td>10.0</td>
<td>85</td>
<td>494</td>
<td>522</td>
</tr>
<tr>
<td>38.5°C</td>
<td>0</td>
<td>83</td>
<td>474</td>
<td>520</td>
</tr>
<tr>
<td></td>
<td>5.0</td>
<td>88</td>
<td>484</td>
<td>522</td>
</tr>
<tr>
<td></td>
<td>10.0</td>
<td>85</td>
<td>484</td>
<td>522</td>
</tr>
</tbody>
</table>

a – estimated start of the hatching process; b – the degree of the synchronisation of the hatching processes, i.e. the time (h) necessary to hatch 1% of chicks; a,b,c - the values in the columns marked with different letters differ significantly (P<0.05).
Fig. 1. The effect of acetylsalicylic acid (ASA) injected in ovo on the 18th d of incubation on plasma thyroxine (T₄) (a, b) and triiodothyronine (T₃) (b, c) levels in newly-hatched chicks incubated from 19 d of embryogenesis under control (a, c) or hyperthermic (b, d) conditions. Each value represents the mean ±SE from 12 determinations; a-e – the values marked with different letters in each panel differ significantly (P<0.05); * P<0.05 in comparison with the appropriate values under control conditions.
There were no significant alterations in the embryonic mortality and chicken hatchability between the control and hyperthermic chickens. ASA treatment did not affect these parameters under control conditions. However, the lower dose of ASA (i.e., 5 mg/egg) significantly (P ≤ 0.05) reduced embryonic mortality and increased the hatchability under the hyperthermic conditions (Table 1).

The cases of the incomplete retraction of the yolk sack and the malpositions (2.2%-6.7%) were similarly frequent in each group (P ≥ 0.05), and the pain of embryos caused by injection occurred in only two single cases (P ≥ 0.05, Table 2).

In comparison to the control embryos, the earlier pipping (Table 3) and hatching (Table 4) of chicks exposed to hyperthermic conditions by 6 and 8 h (P ≤ 0.05), respectively, were noted. However, the synchronisation degrees of both processes were significantly lower under the hyperthermic conditions (P ≤ 0.05, Tables 3 and 4). ASA did not affect the degrees of external pipping and hatching under the standard conditions only. The analysis of the degree of the synchronisation process (b parameter) revealed that, under the control conditions, 1% of chicks from the control group and the groups treated with 5 and 10 mg of ASA/egg pipped every 12.0, 12.6, and 12.6 min (P ≥ 0.05, Table 3) and hatched every 11.4, 12.0, and 13.2 min, respectively (P ≥ 0.05, Table 4). Under the hyperthermic conditions, 1% of chicks from the group injected with 10 mg of ASA/egg needed to pip on average every 16.8 min, while the chicks from the groups treated with 0 and 5 mg of ASA needed to pip by 1.8 and 1.2 min less, respectively (P ≤ 0.05, Table 3). However, under these conditions the lowest degrees of hatching synchronisation occurred in the non-treated group (b = 0.28, i.e., every 16.8 min for 1% of chicks), while this indicator for both the ASA treated groups was shorter than 2.4 min (P ≤ 0.05, Table 4).

As shown in Figs 1a and 1b, significant interactions (P ≤ 0.001) between the thermal conditions, the time after hatching, and the dose of ASA on the thyroxine concentrations were observed. The highest concentrations of T₄ in the control (21.5 ± 2.17 ng/mL) and heat stress (27.9 ± 2.84 ng/mL) chicks were found at the moment of hatching. They were significantly different (P ≤ 0.01; Figs 1a and 1b). At 12th h following the hatching, T₄ concentrations sharply decreased in both groups by 39% and 65%, respectively (P ≤ 0.01).

During the next few hours of the experiment, a gradual increase in T₄ concentrations was observed. Under the control conditions at the 36th h, and under the hyperthermic conditions at the 24th h, after the hatching T₄ concentrations were 1.37- and 1.29-fold higher in comparison with the appropriate values at the 12th h (P ≤ 0.05; Figs 1a and 1b). At the moment of the hatching, in the embryos incubated under control conditions and injected with 10 mg of ASA/egg, a significant increase (by 44%) in the T₄ concentration was noted (P ≤ 0.01). On the other hand, at the 12th h after the hatching, both doses of ASA significantly decreased the T₄ concentrations by 31% and 44%, respectively (P ≤ 0.01; Fig. 1a). A similar effect of ASA was found under the hyperthermic conditions at the moment of hatching where the T₄ concentrations in the blood plasma of chicks treated with 5 or 10 mg of ASA/egg were 36% and 45%, respectively, lower in comparison with the control value (P ≤ 0.01; Fig. 1b). There were no significant alterations in the T₃ levels in the ASA-treated chicks at the other time intervals, both hatched under the control and hyperthermic conditions. However, the T₃ concentrations under the hyperthermic conditions were significantly lower in comparison with the appropriate control values at the 24th and 36th h after hatching (P ≤ 0.05, Figs 1a and 1b).

The plasma T₃ concentration, shown in Figs 1c and 1d, decreased gradually from 3.21 ± 0.42 ng/mL and 2.42 ± 0.23 ng/mL under the control and heat stress conditions at the moment of hatching to 1.07 ± 0.06 and 0.88 ± 0.03 ng/mL at 36th h after hatching, respectively (P ≤ 0.01). However, significant interactions (P ≤ 0.05) between the hyperthermic conditions, the period of staying in the hatcher, and the dose of ASA on the plasma T₃ concentrations were found. Under the hyperthermic conditions, significantly lower levels of T₃ were observed, not only at the moment of hatching, but also at the 12th h following it, where the T₃ concentrations were 25% and 18% lower (P ≤ 0.05) than under the control conditions (Figs 1c and 1d). The effect of the ASA injection occurred only in those chicks kept under the control conditions at the time of hatching. In the chicks treated with 10 mg of ASA/egg, the concentration of T₃ was by 33% lower in comparison with the control group (P ≤ 0.01; Fig. 1c). In the other examined time intervals after the hatching, both under the control and hyperthermic conditions, the level of this hormone did not differ significantly between the control and ASA-treated groups (P ≥ 0.05; Figs 1c and 1d).

**Discussion**

The present study has shown that ASA injected in ovo three days before the expected time of the hatching did not affect the chick’s hatchability, and did not delay the time of the hatching, both under the control and hyperthermic conditions in the hatcher. However, there were significant alterations observed in iodothyronine concentration in the blood plasma of the newly hatched chicks.

In the reported study, no embryotoxic effect of the applied doses of ASA was observed. This statement is confirmed by the observation that the embryo mortality mostly resulted from evident post-manipulation injuries (only two embryos were injured, each of them from a different group), or the incomplete retraction of the yolk sack. It seems that the last malformation was not a consequence of the ASA application because: 1) it was found in various groups and 2) the retraction of the yolk sack should be completed by the 19d of incubation (26), i.e., before the applied manipulation. These observations are consistent with the data reported by Bruggeman et al. (7), who
revealed that the embryo sensibility to manipulations seems to be associated with the stage of its development. Moreover, the hatchability of chicks from eggs hatched under standard (T-37.2°C; RH-70%) and hyperthermic conditions (T-38.5°C; RH-65%) was very similar. These results are in agreement with the opinion that the avian embryo at hatching period is much more heat-stress resistant than during the first two weeks of incubation (12). However, too high a temperature in the hatcher does have an effect on the organism of the newly hatched chick.

In the experiment performed, the permanent elevation of the temperature in the hatcher from the recommended 37.2°C to 38.5°C accelerated the hatching of the chicks by about 8 h. The dependency of the chick embryogenesis rate on the incubation temperature, described by several other authors (6, 11, 12, 19), is related to the changes in the metabolic rate in the developing embryo. The hatching acceleration can be one of the reasons for the no-closing navel and post-haemorrhagic anaemia in chicks. Moreover, the prolonged keeping of chicks under hyperthermic conditions usually leads to disturbances in the neuro-hormonal and immunological processes (23). Accordingly, it seems that the possible effect of ASA should appear during the hatching and/or post-hatching (neonatal) period.

A gradual decrease in the thyroid hormone concentration in the blood plasma of the newly hatched chicks, as observed in our experiment, is in agreement with the data of earlier studies (9, 14, 27, 32). It has been reported that during the last phase of chicken embryogenesis, a gradual increase in T4 and T3 towards the maximal values at the stage of internal pipping (when the beak of the chicken embryo perforates the internal membrane and the transition to lung respiration occurs) and external pipping (when the hatching chicken makes a hole in the egg shell) takes place. Thereafter, a quick decrease in both the iodothyronines in the blood circulation occurs. The increase in the thyroid hormone levels in the blood circulation at the last stage of embryogenesis is indispensable for the hatched chick (10), and is correlated with the rate of the hatching processes (27). It has already been established that the alterations in the T4 and T3 concentrations during the last phase of embryogenesis, and after the hatching process, resulted in the function of the hypothalamo-thyroid axis, which is responsible for thyroidal T3 secretion, and the activity of deiodinase type I (D1) and type III (D3) in the liver and kidneys. The D1 enzyme, which mainly catalyses the outer ring deiodination (ORD), is responsible for the conversion of T4 to T3. This metabolically active iodothyronine can be degraded into an inactive hormone, 3,3′-dideoxythyronine, by means of inner ring deiodination (IRD) by deiodinase D3. It has been found that during the last phase of embryogenesis, and after the hatching, hepatic D1 activity is relatively high and remains stable during the process of hatching, while hepatic D3 activity decreases substantially, most profoundly between the internal pipping and the external pipping stages (4, 9, 31). Under the hyperthermic conditions, a significant increase in the T4 concentration with a concomitant decrease in T3 in the blood plasma was observed. This result is consistent with the assumption that heat stress is accompanied by a decrease in the basal metabolic rate and plasma triiodothyronine, the metabolically active hormone (5). The changes observed in T4 and T3 in the blood of hyperthermic chickens can not only be explained by higher a T4 secretion rate from the thyroid gland, but also by an increase in hepatic D3 deiodinase activity, which metabolises T3 into 3,3′-T2. In the present study, the degree of the synchronisation under the hyperthermic conditions was generally lower in comparison with the standard ones. It cannot be excluded that this effect is associated with the decrease in the T3 concentration in the blood plasma of the embryos kept at the higher temperature at the moment of hatching. This assumption is in agreement with observations of the reaction of newly hatched chicks to heat stress as described by Yahav et al. (34).

The most outstanding result of this study was the effect of ASA on the thyroid hormone concentration in the blood plasma, and the observed interaction between the ASA treatment and the thermal conditions in the hatcher. In previous studies, ASA has been used as an effective reducer of body temperature and as a heat stress suppressor in broiler chickens and Japanese quails (13, 16, 29). Moreover, since it effectively decreases thyroid hormone levels, therefore, it has often been used in hyperthyroid therapy (35). May and McNaughton (21) have reported that feeding with the ASA supplementation reduces the level of iodothyronines (particularly T3) in the blood of the laying hen. The present study has confirmed that ASA treatment at the 18th d of incubation resulted in a significant decrease in the T3 concentration at the stage of hatching. Taking into consideration that, at the same time, the aspirin treatment increased the T4 concentration, it cannot be excluded that ASA may directly affect the D1 and/or D3 activity in peripheral tissues. The effect of ASA under hyperthermic conditions was different, since both applied doses of ASA significantly decreased the T4 concentrations, both at the moment of hatching and at the 12th h after it. At the same time, no changes in the T3 concentration in hyperthermic chickens were observed. It seems likely that the increase in the thyroxine concentration in the blood plasma is related to the direct inhibitory effect of ASA on the hypothalamo-pituitary-thyroid axis (20, 24). Moreover, it cannot be excluded that this mechanism is associated with the suppression of prostaglandin synthesis (mainly PGE and PGF2α), which are potent regulators of thyreotropin (TSH) and iodothyronines secretion from the pituitary and thyroid gland, respectively (3, 20, 30, 36). Further investigations are necessary to explain the mechanism by which ASA affects the function of the thyroid gland and the peripheral metabolism of thyroid hormones in chickens during the last stages of the embryogenesis and neonatal period.

In conclusion, the results obtained indicate that ASA injected in ovo at the 18th d of incubation does not affect the hatching parameters; however, it diminishes
embryonic mortality and elevates their hatchability under hyperthermic conditions. The results of the thyroid hormone determinations suggest that the treatment of the chicken embryo with ASA a few days before hatching reduces the unfavourable effects of heat stress conditions, which may appear in the hatcher.

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