



PHYSIOLOGICAL STUDIES ON THE EFFECT OF *IN OVO* LEPTIN ADMINISTRATION IN JAPANESE QUAIL

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ABSTRACT

This study was carried out to investigate the effect of *in-ovo* injection of leptin hormone on some growth and reproductive parameters in Japanese quail. A total number of 1200 Japanese quail eggs were incubated at 37.5°C and 50-60 % relative humidity, with automatic turning every two hours till the 15th day of incubation. On the 5th day of incubation (day of injection), the eggs were randomly divided into four equal groups: group 1, eggs were kept without treatment as a control group (C); group 2, eggs were injected with 50.0µl normal saline as a control for leptin solvent (N.S); group 3, eggs were injected with 50.0µl normal saline containing 0.1µg leptin (0.1 L) and group 4, eggs were injected with 50.0µl normal saline containing 1.0µg leptin (1L). After hatching, hatchability percentages and hatching weights were recorded. Twenty birds (ten males and ten females) from each group were identified and weighed weekly for successive 8 weeks to evaluate the live body weights. From the 4th week of age 10 birds (5 males and 5 females) from each group were killed and plasma samples were collected to determine plasma concentrations of testosterone and estradiol E₂. The results of this study revealed that *in-ovo* administration of leptin in Japanese quail resulted in non-significant increases in hatchability percentages but it significantly increased ($P \leq 0.05$) the hatching weights. Also, this study showed that the live body weights of both male and female Japanese quail increased after *in-ovo* leptin administration. There were significant increases ($P \leq 0.05$) in the concentrations of testosterone and estradiol E₂ in leptin treated groups, compared with control and normal saline treated groups.

Key Words: *In-ovo* injection, Japanese quail, Leptin.

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1. INTRODUCTION

Leptin; 16 kDa globular protein secreted mainly from adipocytes; is a key regulator in the control of food intake, growth and reproduction as well as fat and glucose metabolism, energy expenditure, and puberty in mammals [3, 21]. Leptin also plays a role in fetal growth, immune and pro-inflammatory responses [18] as well as regulating the energy balance in birds and mammals [5, 7, 8, 27]. Avian leptin cDNA has been cloned in chicken [1, 30] and duck [6]. Cloning of the chicken leptin receptors [13, 23] and the detection of its presence in the hypothalamus [13],

pituitary gland [25] and the ovary [23] indicated the possibility of the involvement of leptin in the regulation of reproductive functions in birds at the central and peripheral levels [24]. The presence of leptin mRNA was demonstrated in the brain, bursa of Fabricius, heart, liver, muscle, and spleen of 5-day-old Leghorn embryo [1, 16]. The presence of leptin mRNA in the developing chick embryo as early as 72 hours [19] and rodent embryos [12] suggests its action in an autocrine or paracrine manner at early stages of

embryogenesis [17]. Leptin expression in the embryonic yolk sac implicates that this hormone may control energy expenditure by mediating nutrient transfer to the developing embryo [1]. It had been also reported that recombinant leptin injected *in-ovo* acted in Japanese quail as a growth factor accelerating embryonic and postembryonic development resulting in higher body weight of hatched quail [17] with subsequent higher growth rate. Its stimulatory effect was more pronounced in younger embryos than the older ones in which the yolk sac was almost resorbed. This effect may be due to the more important role of leptin in earlier developmental stages. This study was performed to determine the effects of *in-ovo* injection of leptin on hatchability percentage, hatching weight, live body weight and plasma concentrations of testosterone and estradiol E₂ in Japanese quail.

2. MATERIALS AND METHODS

2.1. Eggs incubation and injection:

A total number of 1200 Japanese quail eggs, obtained from quail project (Agricultural Production Technology Center, Faculty of Agriculture, Cairo University, Giza), were incubated at 37.5°C and 50-60 % relative humidity, with automatic turning every two hours till the 15th day of incubation. On the 5th day of incubation (day of injection), the eggs were randomly divided into four equal groups: group 1, eggs were kept without treatment as a control group (C); group 2, eggs were injected with 50.0 µl normal saline as a control for leptin solvent (N.S); group 3, eggs were injected with 50.0 µl normal saline containing 0.1 µg leptin (0.1 L) and group 4, eggs were injected with 50.0 µl normal saline containing 1.0 µg leptin (1L). Prior to injection, the eggs were disinfected by ethanol 70 % according to Kocamis *et al* [15]. The solutions were injected into the yolk through the narrow end of the egg

according to Robel and Christensen [9], then the site of injection was closed by wax and the eggs were returned into the incubator. On the 15th day of incubation, the eggs were placed in hatching boxes at 37.5°C and 70 % relative humidity within the same incubator till hatching occur between the 17th and 19th days [2].

2.2. Preparation of leptin solution:

Recombinant human Leptin expressed in *Escherichia coli* in the form of lyophilized powder (L4146) : > 97% (SDS- PAGE) was purchased from Sigma, St. Louis, MO, USA. One mg of leptin powder was dissolved in 25 ml normal saline according to Meek *et al.* [31] to prepare a stock solution containing 40 µg leptin / 1ml normal saline (2 µg leptin /50µl normal saline). To prepare leptin solution containing 1 µg leptin/50µl normal saline, each 1 ml of the stock solution was diluted by addition of 1 ml normal saline. To prepare leptin solution containing 0.1µg leptin/50 µl normal saline, each 1 ml of the stock solution was diluted by addition of 1ml normal saline to reach a concentration of 1µg leptin/50µl normal saline then diluted by addition of 18 ml normal saline to reach the desired concentration (0.1µg leptin/50µl normal saline).

2.3. Bird housing and management:

Birds of each group were housed in brooding boxes for the first three weeks of age then they were transferred to a clean well ventilated room bedded with fresh clean wood shaving forming a layer of 5 cm depth. The room floor was divided into four equal compartments provided by suitable feeders and drinkers [20]. Artificial lighting was provided for 24 hours over the experimental period according to Lamošová *et al.* [17]. The ambient temperature was 37°C for the first 2 days after hatching and then it was decreased stepwise by 3°C at 4 days intervals till reach 21°C [17]. Birds were identified using leg bands, till reaching the age of two weeks then these bands were

replaced by permanent wing bands according to Satterlee *et al.* [28]. A commercial balanced broiler starter ration containing 24.8 % crude protein and metabolizable energy of about 2950 Kcal/Kg was used for feeding of the young birds. While adult quails (from 6 weeks of age) fed diet containing 20.2% crude protein and 2809 kcal/Kg metabolizable energy [11]. Birds were allowed free access to fresh water.

2.4. Measured parameters:

After hatching, hatchability percentages and hatching weights were recorded. Twenty birds (ten males and ten females) from each group were identified and weighed weekly for successive 8 weeks to evaluate the live body weights. From the 4th week of age 10 birds (5 males and 5 females) from each group were killed and blood samples were collected in clean heparinized tubes, centrifuged at 3000 rpm/15min. Plasma samples were separated and stored at -20°C to determine the concentrations of testosterone and estradiol E₂.

2.5. Hormonal Analysis:

ELISA kits for testosterone and estradiol (E₂) (Monobind Inc., Lake Forest, CA 92630, USA) were used to determine the concentrations of testosterone and estradiol E₂ in plasma samples of male and female Japanese quail respectively.

2.6. Statistical analysis:

Data were represented as mean (±S.E). One way analysis of variance (ANOVA) was used for determining the significant difference between groups using Graph Pad Prism software (San Diego, CA, USA) *ver.* 5.04. Significant difference between mean values was determined at $P \leq 0.05$.

3. RESULTS

3.1. Hatchability percentage

In-ovo administration of leptin solution at concentrations of 0.1µg/50µl and 1.0µg/50

µl resulted in non-significant increases in the hatchability percentages of Japanese quail eggs if compared with control and normal saline treated groups (Table.1).

Table 1 Effect of *in-ovo* administration of leptin on hatchability and embryonic mortality percentages of Japanese quail

| Groups | Hatchability (%) | Embryonic mortality (%) |
|--------|-------------------------|-------------------------|
| C | 80.13±0.38 ^a | 19.87±0.38 ^a |
| NS | 80.23±0.38 ^a | 19.77±0.38 ^a |
| 0.1L | 81.00±0.17 ^a | 19.00±0.17 ^a |
| 1L | 80.23±0.41 ^a | 19.77±0.41 ^a |

Means (± S.E.) with different letters in the same column are significantly different ($P \leq 0.05$). C= control, NS= normal saline, 0.1L=0.1 µg leptin, 1L= 1µg leptin

3.2. Hatching weight

The hatching weights of Japanese quail hatched from leptin treated eggs were significantly higher ($P \leq 0.05$) than those from control and normal saline treated groups (Table. 2).

Table 2 Effect of *in-ovo* administration of leptin on hatching weights of Japanese quail

| Groups | Hatching weight (g) |
|--------|------------------------|
| C | 7.75±0.12 ^b |
| N.S | 7.61±0.09 ^b |
| 0.1 L | 9.04±0.14 ^a |
| 1 L | 9.09±0.15 ^a |

Means (±S.E.) with different letters in the same column are significantly different ($P \leq 0.05$). C= control, NS= normal saline, 0.1L=0.1 µg leptin, 1L= 1µg leptin

3.2. Live body weight of male Japanese quail

Mean body weights of male Japanese quail were higher in both treated groups across the whole observed period. The highest weights resulted after injection of the lower dose of leptin. The significance of these increases differed among weeks of age (Table. 3).

3.3. Live body weight of female Japanese quail

Mean body weights of female Japanese quail were higher in both treated groups across the whole observed period. The highest weights resulted after injection of

the lower dose of leptin. The significance of these increases differed among weeks of age (Table. 4).

3.4. Plasma concentration of total testosterone:

Plasma concentrations of total testosterone (ng/ml) in male Japanese quail were significantly ($P \leq 0.05$) higher in both leptin treated groups compared with control and normal saline treated groups. There was

non-significant difference between leptin concentrations (Table 5).

3.6. Plasma concentration of estradiol E_2 :
Plasma concentrations of estradiol E_2 (pg/ml) in female Japanese quail were higher in both leptin treated groups compared with control and normal saline treated groups. The lower concentration of leptin resulted in the highest values (Tab. 6)

Table 3 Effect of *in-ovo* administration of leptin on live body weight (g) of male Japanese quail:

| Age (weeks) | Animal groups | | | |
|-------------|---------------------------|--------------------------|--------------------------|---------------------------|
| | C | NS | 0.1L | 1L |
| 1 | 23.38±0.64 ^b | 23.50±0.47 ^b | 36.03±0.85 ^a | 35.72±0.91 ^a |
| 2 | 58.03±0.47 ^{bc} | 55.82±0.91 ^c | 66.55±1.08 ^a | 60.84±1.37 ^b |
| 3 | 122.40±0.94 ^{ab} | 117.60±2.55 ^b | 126.30±2.32 ^a | 124.00±1.97 ^a |
| 4 | 141.00±2.56 ^b | 139.00±3.02 ^b | 159.10±2.26 ^a | 155.00±1.82 ^a |
| 5 | 174.1±4.71 ^b | 170.00±4.05 ^b | 187.90±4.32 ^a | 186.60±4.13 ^a |
| 6 | 196.±4.95 ^{ab} | 189.50±4.74 ^b | 206.10±4.23 ^a | 204.90±2.92 ^a |
| 7 | 219.30±8.75 ^{ab} | 206.20±6.38 ^b | 236.50±7.66 ^a | 221.70±3.49 ^{ab} |
| 8 | 234.5±8.77 ^{ab} | 224.9±6.52 ^b | 249.1±7.65 ^a | 240.4±4.59 ^{ab} |

Means (±S.E.) with different letters in the same column are significantly different ($P \leq 0.05$). C= control, NS= normal saline, 0.1L=0.1 µg leptin, 1L= 1µg leptin.

Table 4 Effect of *in-ovo* administration of leptin on live body weight (g) of female Japanese quail

| Age (weeks) | Animal groups | | | |
|-------------|---------------|--------------|-------------|--------------|
| | C | NS | 0.1L | 1L |
| 1 | 28.94±0.82b | 28.56±0.72b | 43.64±1.41a | 43.58±1.22a |
| 2 | 66.68±1.29b | 65.25±1.86b | 76.56±1.01a | 68.59±1.27b |
| 3 | 132.8±2.06b | 133.8±2.47b | 142.8±2.98a | 141.3±2.58a |
| 4 | 157.7±1.56b | 160.5±2.78b | 186.2±3.34a | 182.6±3.70a |
| 5 | 202.6±4.19c | 206.1±1.87bc | 220.3±3.10a | 215.8±4.77ab |
| 6 | 243.6±6.10a | 224.1±6.19b | 254.6±6.84a | 246.0±4.80a |
| 7 | 261±7.37ab | 249.7±9.22b | 276.7±8.38a | 266.3±5.52ab |
| 8 | 273.6±7.47ab | 265.2±8.39b | 291.5±8.16a | 282.1±5.58ab |

Means (±S.E.) with different letters in the same column are significantly different ($P \leq 0.05$). C= control, NS= normal saline, 0.1L=0.1 µg leptin, 1L= 1µg leptin.

Table 5 Effect of *in-ovo* administration of leptin on total testosterone (ng/ml) in male Japanese quail plasma

| Groups | W4 | W5 | W6 | W7 | W8 |
|--------|------------------------|------------------------|--------------------------|--------------------------|------------------------|
| C | 0.80±0.11 ^b | 1.70±0.15 ^b | 2.63 ± 0.08 ^b | 2.66 ± 0.14 ^b | 4.46±0.26 ^b |
| NS | 0.90±0.05 ^b | 1.63±0.08 ^b | 2.46 ± 0.14 ^b | 2.80 ± 0.36 ^b | 4.26±0.26 ^b |
| 0.1L | 1.30±0.05 ^a | 2.26±0.14 ^a | 3.63 ± 0.32 ^a | 4.46 ± 0.06 ^a | 6.10±0.15 ^a |
| 1L | 1.20±0.11 ^a | 2.23±0.12 ^a | 3.40 ± 0.20 ^a | 3.36 ± 0.08 ^a | 5.66±0.12 ^a |

Means (±S.E.) with different letters in the same row are significantly different ($P \leq 0.05$). C= control, NS= normal saline, 0.1L=0.1 µg leptin, 1L= 1µg leptin.

Table 6 Effect of *in-ovo* leptin administration on plasma estradiol E₂ level (pg/ml) in female Japanese quail

| Groups | Age (weeks) | | | | |
|--------|--------------------------|---------------------------|-------------------------|--------------------------|-------------------------|
| | 4 | 5 | 6 | 7 | 8 |
| C | 66.53±0.72 ^b | 46.23 ± 2.81 ^b | 74.67±7.85 ^c | 102.3±7.20 ^b | 128.6±4.89 ^c |
| NS | 64.57±1.86 ^c | 57.73 ± 2.87 ^b | 85.03±8.25 ^c | 96.13±4.33 ^b | 158.6±9.17 ^b |
| 0.1L | 82.57±3.77 ^a | 124.6 ± 6.96 ^a | 153.7±8.00 ^a | 148.0±8.04 ^a | 265.9±9.37 ^a |
| 1L | 73.85±4.33 ^{ab} | 125.6 ± 7.82 ^a | 120.3±5.77 ^b | 140.5±10.09 ^a | 163.1±7.12 ^b |

Means (±S.E.) with different letters in the same column are significantly different ($P \leq 0.05$). C= control, NS= normal saline, 0.1L=0.1 µg leptin, 1L= 1µg leptin

4. DISCUSSION

The development of avian embryo in the isolated environment of the egg provides a unique opportunity to manipulate post hatching growth and performance by means of altering the nutritional or endocrinological status of the embryo through *in-ovo* injection [26]. Leptin *in-ovo* injection in Japanese quail acted as a growth factor accelerating embryonic and postembryonic development and resulted in higher body weight of hatched quail with subsequent higher growth rate [17]. The present study revealed that leptin *in-ovo* injection in Japanese quail during the 5th day of incubation period resulted in non-significant increases in the hatchability percentages of control and normal saline treated groups. The same results were observed by Lamošová *et al.* [17].

The obtained results also showed that leptin *in-ovo* injection in Japanese quail during the 5th day of incubation period caused significant increases in the hatching weights and the subsequent live body weight of male and female Japanese quail. Presence of leptin mRNA in the developing chick embryo, similar to rodent embryos [12] suggests that leptin has paracrine and endocrine effects on embryogenesis. Leptin expression was detectable as early as 72 hours in the developing chick embryo [19]. Leptin expression in the embryonic yolk sac implicates that this hormone may control energy expenditure by mediating nutrient transfer to the developing embryo. It is hypothesized that leptin in birds acts as a

general signal of low energy status to neuroendocrine systems that improve the utilization of nutrients [17]. Its stimulatory effect was more pronounced in younger embryos than the older ones in which the yolk sac was almost resorbed. This effect may be due to the more important role of leptin in earlier developmental stages.

Our results illustrated that leptin *in-ovo* injection in Japanese quail during the 5th day of incubation period accelerated the onset of puberty and maturity of male and female Japanese quail. These findings were confirmed by the increased concentrations of total testosterone and estradiol E₂ in male and female Japanese quail respectively. It had been revealed that leptin acts not only as a satiety, appetite-regulating hormone which controls weight gain and fat deposition [10], but that leptin is also extensively implicated in puberty and fertility regulation [4], reproductive processes and serves as a main hormonal factor that links adiposity with reproduction [3, 22]. The first indication of this association came from the observation that genetically obese mice lacking functional leptin (*ob/ob*) or leptin receptor (*db/db*) fail to undergo normal sexual maturation and remain infertile throughout life [14, 29].

Cloning of the chicken leptin receptors [13 and 23] and detection of its presence in the hypothalamus [13 and 25], in the pituitary [25] and in the ovary [23, 25] indicate the possibility that leptin might be involved in the regulation of reproductive functions in birds by acting both at the central and peripheral levels.

It could be concluded that leptin *in ovo* injection at a low concentration (0.1µg leptin/50µl normal saline) can improve growth and reproduction of Japanese quail. The role of leptin hormone in reproduction is promising and needs further investigations.

5. REFERENCES

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دراسات فسيولوجية على تأثير حقن الليبتين في بيض السمان الياباني

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قسم الفسيولوجيا - كلية الطب البيطرى - جامعة بنها

الملخص العربى

تم اجراء هذه الدراسة لتقييم تأثير حقن هرمون الليبتين فى بيض السمان اليابانى على بعض مؤشرات النمو والتكاثر. تم تحصين عدد 1200 بيضة سمان يابانى عند درجة حرارة 37.5 درجة مئوية و 50-60% رطوبة نسبية مع التقليب المستمر للبيض كل ساعتين. تم تقسيم البيض عشوائيا خلال اليوم الخامس من التحضين (يوم الحقن) الى اربع مجموعات متساوية: المجموعة الاولى لم يتم حقنها نهائيا كمجموعة ضابطة، المجموعة الثانية تم حقنها باستخدام 50 ميكروليتر محلول ملحي كمجموعة ضابطة للمذيب، المجموعة الثالثة تم حقنها باستخدام 50 ميكروليتر محلول ملحي تحتوى على 0.1 ميكروجرام ليبتين، المجموعة الرابعة تم حقنها باستخدام 50 ميكروليتر محلول ملحي تحتوى على 1 ميكروجرام ليبتين. بعد الفقس تم حساب نسبة الفقس والاوزان الحية عند الفقس. تم ترقيم عدد 20 طائر (10 ذكور و 10 اناث) من كل مجموعة و وزنهم اسبوعيا لمدة 8 أسابيع متتالية لحساب التغير فى الوزن الحى للطائر. تم ذبح عدد 10 طيور (5 ذكور و 5 اناث) بداية من الاسبوع الرابع من العمر من كل مجموعة للحصول على عينات البلازما وذلك لقياس مستوى هرمونى التيستوستيرون والاستروجين. أظهرت نتائج هذه الدراسة ان حقن هرمون الليبتين فى بيض السمان اليابانى لا يسبب زيادة معنوية فى نسبة الفقس ولكنه يؤدى الى حدوث زيادة معنوية فى وزن الطائر عند الفقس اذا ما قورن بالمجموعتين الضابطين الاولى والثانية. تشير النتائج ايضا الى ان الاوزان الحية لذكور واناث السمان اليابانى زادت زيادة معنوية بعد حقن هرمون الليبتين. أوضح التحليل الهرمونى لعينات البلازما ان حقن بيض السمان اليابانى بهرمون الليبتين أدى الى زيادة مستوى هرمون التيستوستيرون فى بلازما الذكور وزيادة هرمون الاستروجين فى بلازما الاناث

(مجلة بنها للعلوم الطبية البيطرية: عدد 23(2)، ديسمبر 2012: 71-78)