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Lethal Factors in Pekin Ducks

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PEKIN ducks were used in studying the lethal factors affecting embryonic mortality.

The experiment was carried under the usual system of mating and nutrition in the Poultry Research Station, Faculty of Agriculture, Cairo University.

The study included 3748 eggs, 2151 eggs in the first year (from January to May), and 1597 eggs in the second year (from January to April).

The lethal factors occurred in this study were :

1. *The crest syndrome*

Crested embryos showed great reduction of the upper beak, variable deficiencies in cranium, sometimes there were great hernia through which the whole brain was outside the cranium. They died at the end of the incubation. The crested embryos represented 10.67% of the total dead embryos allover the experimental period or about 42.04% of the abnormal ones.

2. *Micromelia*

These embryos showed reduction in growth. Feather growth is retarded and had "wormy" appearance. The extremities were shortened. Death occurred in late stage of incubation. This group represented 11.99% of the total dead embryos or about 47.24% of the abnormal ones.

3. *Short both mandibles*

A lethal caused abnormalities in both mandibles, especially the lower one. The upper was reduced and pointed upward. The facial parts were reduced and there was some degree of cereberal hernia.

The eyes were abnormal. This case represented 81% of the total dead embryos or 3.18 of the abnormal ones.

4. *Crooked neck* :

The characteristic features was the crooked neck and spinal column, reduction of the upper beak and the total size of the embryo, spindly legs, curled toes and oedema in the head region and the neck. The embryos having crooked neck represented 0.72% of the total dead embryos or 2.85% of the abnormal ones.

5. *Embryos that died after hatching* :

Embryos of this group were hatched but were very weak that they died within few hours after hatching and before getting them out othe hatchary. Some of them were normal but others were having bleeding originated from the yolk sac enterance position, and some of them showed failure of yolk sac absorption. The embryos of this group represented 1.19% of the total dead embryos or 4.69% of the abnormal ones.

Several lethal factors are of wide spread occurrence among various breeds of fowl, but a few of them were found in ducks.

Crested syndrome

The crested syndrome offers a good example for a very undesirable gene being adopted as the distinguishing feature of a breed. It has been found that in the underlying partio-occipital region ofthe skull there is a hole through which protrudes an encephalocele or hering of the brain. The protruding parts may include posterior parts of the cerebral hemispheres, the cerebellum, one or both optic lobes, or part of medula (Krautwald, 1910).

Studies showed that crested ducks are heterozygous for an incompletely dominant gene. The homozygotes do not hatch, and only about half the heterozygotes show a crest. This means that the penetrance of the gene is about 50%. In the homozygotes, death occurs at the end of the incubation period, or before hatching (Rust, 1932).

Requate (1960) suggested that this case in ducks is due to a single semi-dominant gene which seems to be responsible for crest formation. The great vasiability in the expression of the character suggests that it is more effective than in fowl. The development of the crest is also influenced by the total genetic millieu. Lethal effects are occasionally found in both homozygotes and heterozygotes,presumably due to genetically conditioned developmental imbalance.

Micromelia

Micromelia is an outosomal recessive gene that has been identificd in White Pekin ducks. In the homozygous condition,it is lethal in late embryonic stages. The gene has a pleiotrophic effect as found from the appearance of the homozygotes : body size is reduced, a mucoid transudate is present in the outer cervical region, feather growth is greatly retarded and "wormy" in appearance and the extremities are shortened.

Histological examination of the long bones of the extremities reveals abnormal epiphseal architecture and bone ash is reduced. This case was named Micromelia (Ash, 1968).

Mansibles missing

A lethal mutation affecting both mandibles was found in white leghorn (Marble *et al.*, 1944). The embryos showed complete absence of the lower mandible to a mere vestige, and much deformed and smaller upper mandible. It is frequently pointed upward or even curved over the front part of the head. The eyes were generally abnormal and other facial parts were reduced in size. There are always some degree of cerebral hernia. This condition has been shown to be due to a single recessive autosomal gene.

Crooked-neck dwarf

In new hampshires Pun (1954) found that embryos homozygous for the lethal gene are characterized by their crooked neck, crooked spinal column, slightly shorter upper beak, spindly legs, curly toes, and reduced sternum without keel and lateral processes. All parts of the skeleton, musculature and internal organs were reduced in size. Lethal embryos showed little growth after the 13th -16th days and usually died in the last two days of incubation, and they never hatched. It is possible to distinguish lethal from normal embryos as early as the 10th day of incubation by their culry toes.

Asmundson (1945) reorted that in New Hampshires, this syndrome is brought about by a recessive gene.

Failure of the yolk sac absorption

A recessive lethal gene was found in the offspring of a blue-black Indian hen, White Leghorn and Black Minorca males (George, 1963). That gene was responsible for the failure of yolk sac absorption mechanism, the characteristic symptom in the dead chicks was their inability to absorp the last position of yolk sac.

Material and Methods

This work has been done on the White Pekin duck (Anas Platyrhynchos) through two successive hatching seasons. The first season was during the period from January to May and the second was from January to April the next year.

Eggs were collected and kept for a period not less than 24 hr, and not more than 7 - 10 days. The eggs were given a serial number. The eggs were candled to exclude the unsuitable ones for iucubation.

Throughout the incubation period, candling was performed twice : at the 8th and the 22nd day of incubation. Dead and unhatched embryos were examined to determine their age and to detect their abnormalities and describe the existence of lethal and semi-lethal types. The abnormal embryos were classified into five groups acccording to their appearance. The first group showed the crest syndrome phenotype, the second showed the abnormal phenotype of micomelia lethal factor, the third had short both mandible, the foruth represented the embryos that died with-in few hours after hatching.

Results and Discussion

Among dead and unhatche embryos there were some that showed abnormal features as an epxression of lethal factors.

It can be seen in Table 16 that there were 317 abnormal embryos in the first year which represent 22.12% of the total dead and unhatched embryos in this year. In the second year 280 embryos or 30.47% of the total dead and unhatched were abnormal. The total number of the abnormalities were 597 or 25.38% in the two years.

These abnormal embryos can be classified into five groups according to their appearance. The first group that showed the crest syndrome phenotype represented 42.04% of the total abnormal embryos obtained in the two years.

The second group that showed the abnormal phenotype of micromelia lethal factor represented 47.24% of the total abnormalities in the two years.

The third group of abnormal embryos having short both mandible represented 3.18% of all abnormalities obtained.

The fourth group of abnormal embryos with crooked neck represented 2.85% of the total abnormal embryos.

The fifth group (4.69% of the total abnormalities) represented the embryos that died after hatching. Embryos of this group were hatched but were very weak that they died within few hours after hatching and before getting them out of the hatchary. Some of them were normal but others were having bleeding originated from the yolk sac enterance position and some of them showed failure of yolk sac absorption.

1. *The crest syndrome*

In the first season, 127 embryos showed the crest syndrome. These represented 8.86% of the total dead embryos.

In the second season, 124 embryos had the same phenotype (13.49% of the total dead embryos). The affected embryos died at the end of the incubation period. Only one of them died at the 12th day of the incubation and three died at the 22nd. The remaining embryos died in a later period.

The crested embryos showed great reduction of the upper beak, and variable deficiencies in cranium, sometimes they form a great tuberosity in which lie the cerebral hemispheres of the brain. In other cases there were great hernia in the cranium through which the whole brain was outside the cranium.

These features agreed with those found by Krautwald (1910). He found that the protruding parts may include posterior parts of the cerebral hemispher, the cerebellum, one or both optic lobes, or part of the medulla.

Rust (1932) found that crested ducks are heterozygous for an incompletely dominant gene that has only about 50% penetrence. The homozygotes do not hatch and the death occurs at a late stage of the incubation period or before hatching

Requate (1960) suggested that this case is due to a single semi-dominant gene which is responsible for crest formation. It is more effective in ducks than in fowl. The total genetic millieu is also effective in the development of the crest, and the lethal effects occurred in both homozyotes and heterozygotes are presumably due to genetically imbalanced developmental conditions.

Out of 3173 individual egg set, there were 251 embryos which showed this abnormality and 2922 did not show this abnormality : This segregation ratio was found to be in good agreement with mendelian 1:15 ratio ($X^2 = 1.28$ and $P = 0.25$). These results may suggest that the character is under the control of two recessive genes and the genotype $cr_1\ cr_1\ cr_2\ cr_2$ is suggested for the effected embryos.

2. *Micromelia*

Embryos that showed micromelia represented the highest frequency in this study. There were 145 and 137 embryos in the two seasons, respectively. There were 145 and 137 embryos in the two seasons, respectively. These represent 11.99% of the total dead embryos or about 47.24% of the abnormal ones.

These embryos were characterized by a reduction in growth. Feather growth was retarded and had wormy appearance and the extremities were shortened. Death occurred in late stage of incubation, *i.e.* after the 22nd day.

This lethal factor was first found by Ash (1968) in Pekin ducks. He suggested that this condition is due to an autosomal recessive gene, which is lethal in the homozygous condition. It has a pleiotropic effect as found from the phenotype of the homozygotes. There were a mucoid transudate present in. the cervical region. Histological examination of the long bones of the extremities revealed abnormal epiphyseal architecture and bone ash is reduced

Similar case was found in White leghorn (Asmundson, 1942)and in Indonesian land race and Kedu fowl (Fischer, 1956). These cases were marked also by shortness of the extremities, the upper beak was short and curved, and the lower beak was defected. They suggested that these a bnormalities were due to the action of two autosomal recessive genes.

There were another micromelia found in White Leghorn by Bernier (1951) which was named micromelia VII. The affected embryos have normal beak but microphthalamic eyes. The extremities were shortened and the legs were bent. This case was due to a single recessive gene.

There is another case which closely resemble micromelia and Cornish lethal, found in Japanese Quail (Hill *et al.*, 1963). The affected embryos have broad short head with bulging eyes, and a parrot-shaped beak. The neck and

trunk were broad. The extremities were shortened and were set for apart. This case was suggested to be due to an autosomal recessive gene.

3. *Short both mandible*

The affected embryos had abnormalities in both mandibles. The lower beak was reduced to a mere vestige. The upper beak was also reduced but remained from about one-quarter to two thirds of the normal size.

It was frequently pointed upward. The eyes were abnormal and other facial parts were reduced in size. There were some degree of cereberal hernia. The abnormality is recognisable in young embryos as soon as the beak begins to form. It was lethal to all homozygotes, but two of them were still alive at the day of hatch.

These features were representative to that found by Marble *et al.* (1944) in White leghorn. The numbers of embryos that showed these features were 13 and 6 for the two seasons respectively. Death occured at different stages of incubation period, two of them were still alive at hatch.

4. *Crooked neck*

The embryos carrying this lethal factor were charcterized by their crooked neck, crooked spicnal column, slightly shorter upper beak, spindly legs, curly toes, and reduced sternum without keel and lateral processes, The embryos in total were reduced in size. There were some oedema in the head region in the embryos that died in the last period of incubation. Embryos that died in small age were having a tall crooked neck, a small head and a body reduced in size, they resembled in part the embryos that carry the lethal factor micromelia.

During the two years of the study, 17 embryos having this lethal factor were recorded. This number of embryos represents a small position of the total number of chicks produced during the experimental period. This little number of embryos having crooked neck-or abnormal both mandibles-may result from a little number of heterozygous mother ducks carrying this lethal factor. On the other hand, this factor may be carried on combination with one of the other lethals that makes a great defect in the appearance of the embryos such as Micronelia lethal factor or Crest lethal factor, which when appeared in the embryos make the detection of the crooked neck-or the abnormal both mandibles factor-very difficult.

The total number of crooked neck embryos in the first year of the experiment was 12. This number is reduced in the second year to 5, and therefore the number of abnormalities caused by the abnormal both mandibles in the first year was 13 versus 6 in the second year. This reduced number of abnormalitities in the second year than in the first year may be due to several reasons :

a· The heterozygous individuals having this lethal factor were weak, they died before reaching the sexual maturity, or part of them culled during the successive culling processes that occurs throughout their living period to expell weak and low growth rate duckling chicks.

b· The heterozygotes may reach sexual maturity but they have low productivity. Due to that they gave unsufficient number of eggs. This insufficient number when studied genetically cannot give true impregnation.

c· The eggs produced by heterozygous mother ducks may have abnormalities such as,small size easily shell (Shrinked shell), high percentage of pores in the shell (high porosity), abnormal air sac or completely absent air sac. All of these abnormalities were excluded before setting the eggs in the incubation

5· *Embryos died after hatching*

Embryos of this group were hatched but were very weak that they died within few hours after hatching and before getting them out of the hatchary. Some of them were normal but others were having bleeding originated from the yolk sac enterance position, and some of them showed failure of yolk sac absorption. In the first season 20 embryos showed this case that represented (1.40%), while 8 (0.87%) embryos showed the same case in the second season.

TABLE 1. Frequency of abnormal embryos

Season	No. of eggs set	No. of dead embryos	Crest		Micromelia		Short both mandibles		Crooked neck		Died afer hatch		Total of abnormal	
			No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1st season	2151	1433	127	8.86	145	10 12	13	0.91	12	0.84	20	1.40	317	22.12
2nd season	1598	919	124	13.49	137	14.91	6	0 65	5	0.54	8	0.87	280	30.47
Total	3749	2352	251	10.67	282	11 99	19	0.81	17	0.72	28	1.19	597	25.39
Percentage			42.04		47.24		3.18		2.85		4.69		100	

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العوامل المميته فى بيض البط

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استخدم فى هذه الدراسة بيض البيض البيكى لدراسة العوامل المميتة فى الاجنه ٠ وقد استخدم النظام المعتاد بالنسبه للتزاوج والتغذيه فى محطه ابحاث الدواجن بكليه الزراعه جامعة القاهرة ٠ وقد شملت الدراسه ٣٧٤٨ بيضه منها ٢١٥١ فى السنه الاولى (من يناير حتى مايو) ، ١٥٩٧ بيضه فى السنه الثانيه (من يناير حتى ابريل) ٠

وكانت العوامل المميته التى وجدت فى هذه الدراسة :

١ ـ العامل المرتبط بوجود المعرفه : والافراد الحاملة لهذا العامل اظهرت نقصا ملحوظا فى الفك العلوى وفى عظام الراس ٠ وفى بعض الحالات ظهر فتق فى الجمجمه يكفى لخروج المخ خارجها ٠ وكانت الأجنه تنفق فى اخر فترة التفريخ ٠

٢ ـ الميكروميليا : وهذه الاجنه ابدت نقصـا فى النمـو • وتاخر نمو الريش الذى ياخذ مظهر دودى • وكانت الاطراف قصيره بدرجة كبيره وتنفق الاجنه فى مرحله متأخره من التفريخ •

٣ ـ قصر كلا الفكين : هذا العامل يسبب تشوهات فى كلا الفكين وخاصه السفلى • وكان الفك العلوى قصيرا ومتجها لاعلى واجزاء الوجه ناقصة مع وجود درجات متفاوته من الفنق المخى وتشوهات فى العينين •

٤ ـ التواء الرقبه : ومن مظاهرها المميزه قصور والتواء الرقبه والعمود الفقرى ونقص فى المنقار العلوى وفى الحجم الكلى للجنـين • وقصر الرجلـين وضعفهما والتواء الاصابع وارتشاحات مائيه فى منطقة الرأس واعلى الرقبه •

٥ ـ الاجنه التى تموت بعد الفقس : وهذه الاجنه تفقس ولكنها تكون ضعيفه جدا وتموت بعد الفقس بساعات قليله وقبل نقلها من المفقس • وبعضها يكون شكله عادى والبعض الاخر يظهر به نزف من موضع دخول كيس الصفاره والبعض يفشل فى امتصاص كيس الصغار •