CHAPTER

20

Reproductive management of poultry

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OUTLINE

Introduction

Poultry production has markedly increased globally over the last 50 years. This is summarized in Table 20.1. The increase in rate of poultry production over 50 years is over twice the rate of increase in human population.[1]

Chicken production is divided into two distinct sectors: meat and eggs. There are disparate lines of birds derived from different strains with greatly different genetics. An analogous situation exists for ducks. Production of poultry meat depends on three distinct reproductive phases:

1. A system of primary breeding companies with pedigree flocks undergoing intensive genetic selection for improvement. These produce grandparent stock/great grandparent stock and, following multiplication, leads to broiler breeders (or their equivalent in other poultry species). In the USA, there were 114 million broiler breeder pullets placed for broiler egg production (and hence broiler chick) in 2017.[2]
2. Broiler breeder production of fertile eggs. In the USA, there were 1.2 billion hatching eggs produced in 2017.[2]
3. Incubation of the fertile eggs in hatcheries to produce broiler chicks, turkey poults, ducklings and goslings. In the USA, there were 185 million broiler chicks placed per week in August 2018 (*cf.* 227 million eggs set per week)[3] and 292 million turkey poults hatched in 2017.[4]

Production of eggs depends on three distinct reproductive phases:

1. Primary breeding companies with pedigree flocks undergoing intensive genetic improvement. These produce replacement pullets. In the USA, there are 115 million re-placement pullets per year.[5] The average number of layers in the USA in 2017 was 375 million.[2] In the USA, the average hen produces 281 eggs per year.[4]
2. Re-cycling hens toward the end of egg production cycle.

Physiological control of reproduction

Embryonic development of the reproductive system

In contrast to the situation in mammals, the sex chromosomes in male birds are ZZ (homozygous) compared to ZW (heterozygous) in females. In males, the two testes are internal and accessory organs such as the prostate and seminal vesicles are absent. The testes develop due to gene dosing with increased expression of the Z-linked transcription factor gene, double-sex and mab-3-related transcription factor 1 (DMRT1).[6,7] Anti-Müllerian hormone (AMH) is synthesized and secreted by the embryonic testis with greater expression in the embryonic testes than the ovaries.[8-10] AMH directs the regression of the paired Müllerian ducts.[8-10]

TABLE 20.1   Increases in poultry meat and egg production globally.

	1966	1991	2016	Fold increase
POULTRY PRODUCTION IN MILLION METRIC TONS				
Poultry meat				
Chicken	10.0	38.2	107.1	10.7
Turkey	1.07	3.47	6.06	5.7
Duck	0.42	1.38	4.53	10.8
Geese/guinea fowl	0.19	0.76	2.54	13.4
Eggs[a]				
Hen's	16.5	36.6	73.9	4.5
Other birds[b]	0.79	2.6	6.9	8.7
POPULATION IN BILLION				
World	3.41	5.42	7.30	2.14

[a] Based on weight of egg including shell.
[b] Predominantly duck eggs.
Data from FAO.[3]

In females, only the left ovary and oviduct develop in all avian species and closely related dinosaurs; the latter based on fossil evidence from the early Cretaceous period.[11] The avian oviduct is derived from the embryonic Müllerian duct; the former term encompassing the entire reproductive tract from infundibulum to the cloaca.[7] Regression of the right oviduct is induced by AMH.[10] Parenthetically, AMH also plays an important role in development of tubules in the testes.[8,9] The embryonic female gonad expresses the rate-limiting enzyme for the production of estrogens, aromatase (CYP19A1) but expression is not found in the embryonic male gonads.[12,13] In turn, the estrogens, such as estradiol, induce growth of the oviduct.[7]

Egg development

The egg is comprised of the yolk, yolk membranes, egg white, shell membranes and finally the egg shell. Each of these components are developed along specific regions of the female reproductive tract together with the ovary.

Yolk

The egg yolk is a mature ovum (oocyte) that is produced by the ovary. The maturation of the ovum involves multiple processes including deposition of yolk proteins/lipids.

Yolk protein/lipoproteins/phosphoproteins were assigned to three categories based on centrifugation of diluted yolk:

- Low-density fraction with a very high lipid composition
- Granules composed of heavy and light chain lipovitellins, phosvitin and a yolk glycoprotein.[14]
- Soluble proteins.

The soluble proteins[15] consist of the following:

- α livetins (serum albumen)

- β livetins (serum α_2-globulin containing transport proteins)
- γ livetins (serum γ -globulin predominantly immunoglobulin Y).

Egg yolk livetins (α, β, and γ-livetin) have recently been shown to exert anti-inflammatory properties.[16]

Yolk precursors: Yolk precursors are synthesized in the liver. Two major yolk precursors are very-low-density lipoprotein (VLDL) and vitellogenin. Very-low-density lipoprotein (VLDL) has the following characteristics:

- Globular micelle-like
- Non-polar core of triglycerides and cholesterol esters
- Coated with amphiphilic mix of phospholipid, free cholesterol (FC) and twp apolipoproteins.[17] Chicken vitellogenin has been purified from plasma of estrogen treated adult male chickens.[18] It is a dimer with a molecular weight 480,000.[18] It is a dimer composed of two polypeptide monomers each with a molecular weight of about 170,000.[19] There are about 220−235 phosphate moieties per monomer vitellogenin[18] and the lipid component is about 20%. Hepatic expression of vitellogenin is induced by estrogens.[19]

Yolk deposition: A specific receptor is responsible for transfer of vitellogenin and very-low-density lipoprotein (VLDL) across the oocyte plasma membrane to fill the oocyte with yolk.[20,21] Within the oocyte, vitellogenin is cleaved proteolytically to form the yolk proteins, heavy and light chain lipovitellin (20% lipid), phosvitin and a yolk glycoprotein. These are incorporated into yolk granules. Deposition of γ livetins is very high in small follicles <200 mg,[22] but decreases during development of large follicles.[23] For the last four days of development of the follicles, yolk is being deposited at 2.5 cm^3 or greater per day.[24] Once the ovum (egg yolk) has matured, ovulation is stimulated by the pituitary hormone,

luteinizing hormone (LH). An extensive explanation of hormonal control of female reproduction follows. If ovulation is successful the ovum is normally received into the infundibulum.

Egg white

The egg white or albumin of the egg is produced by the magnum of the oviduct. The magnum is the longest section of the oviduct where the ovum spends approximately 4 h accumulating egg white proteins.[25]

Among the constituents of egg white are the following proteins:

- Ovalbumen - 50% of egg white proteins
- Ovotransferrin (conalbumen) 12% (this chelates metal ions particularly iron)
- Ovomucoid -11%
- Lysozyme -3.5%
- Ovomucin 1—3%
- Avidin 0.05%.[26]

Antimicrobial peptides and proteins[27—29] are present in the egg white and include the following:

- Gallin or ovodefensin
- β-defensin 11
- Cathelicidin
- Cystatin - a cysteine protease inhibitor
- Lysozyme-a bacteriolytic enzyme
- Ovoinhibitor.

Eggshell membranes

Following albumin deposition and addition of water ("plumping fluid") to the developing egg, the eggshell membrane is added in the isthmus; this taking approximately 1 h.[25] The eggshell membranes are 93% protein[30] contains proteins including collagens, ovoalbumin, bacteriolytic enzymes such as ovotransferrin and lysozyme together with clusterin peptides and ovodefensins/defensins such as gallin.[30,31] These are also glycosaminoglycans including galactosaminoglycan.[32,33]

Egg shell

The formation of the egg shell in the uterus/shell gland is the final yet it is of longest in duration taking approximately 19 h.[25] This is due to the extensive structure of the shell. The egg shell is 97% inorganic (calcium carbonate).[26,34] Of the remaining 3% (the decalcified egg shell) is 79% protein with the matrix phosphoproteins including the following: ovocleidin-17, ovocleidin-116, ovocalyxin-32 and osteopontin.[30,35]

The fully formed egg is retained in the shell gland just distal to the vagina of the oviduct until oviposition.

Male reproduction

Unlike many mammals, the testes of poultry and other birds are in the abdominal cavity.[36,37] Following sexually maturation, adult male birds produce semen containing large numbers of spermatozoa (see Table 20.2). Production of spermatozoa is critical to fertilization of the ovulated ovum and, hence, the production of broiler chicks (see Broiler breeder reproduction section), layer pullet chicks and turkey poults (see Artificial insemination section).

Spermatogenesis is a complex process that is tightly regulated by neuroendocrine and endocrine mechanisms (discussed later in hormonal control of reproduction).[36,37] Spermatozoa are

TABLE 20.2 Semen characteristics in poultry.

Poultry type	Semen/ejaculate volume (mL)	Concentration of spermatozoa (#x 10^9 mL^{-1})	References
Layer type (white Leghorn)	0.65	3.55	108,109
Broiler breeders	0.29	6.33	125
Turkeys	0.44	6.76	126

produced in the seminiferous tubules of the testes.[36,37] Each mature spermatozoan consists of an acrosome containing the nucleus that holds the DNA information, attached to a microtubular flagella enabling it to be propelled from the cloaca of the hen to the infundibulum of the oviduct.

Once spermatozoa enter the vagina of the oviduct, they are stored in the utero-vaginal junctions or the infundibular sperm storage tubules.[36] Similar number of utero-vaginal junctions can be found in commercial broiler breeders and turkeys.[38] Spermatozoa can retain their ability to fertilize the oocyte for several weeks, however fertility begins to decline post 7 days after insemination and is markedly lower 10 days after insemination.[39]

Hormonal control of reproduction

Hormones are critically important to the optimal functioning of the gonads, the photoperiodic stimulation of reproduction, sexual and maternal behavior and induced molting. The major androgen produced by the testes is testosterone. Interestingly, it was demonstrated by Berthold in 1849, in the first endocrine study, that a testicular factor was essential to both male behaviors such as crowing, mating and aggression in chickens and to the development of the secondary sexual characteristics such as the rooster's comb and wattle.

Pituitary gland and reproduction: The gonads are controlled by the anterior pituitary hormones, LH and follicle stimulating hormone (FSH). These gonadotropins play a critical role in the development and maintenance of the gonads. LH stimulates production of progesterone by granulosa cells from large follicles[40] and testosterone by Leydig cells of the testes.[41] FSH increases proliferation of granulosa cells, expression of both steroidogenic acute regulator (StAR) and inhibin α genes in granulosa cells and release of progesterone with the effect progressively greater with tissue from larger

follicles.[42,43] In addition, prolactin can exert an inhibitory effect on the chicken ovary.[44]

Hypothalamic control of gonadotropin release: There are two gonadotropin releasing hormones (GnRHs) in the chicken (cGnRH-I and cGnRH-II) and two receptors for GnRH (cGnRHR1 and cGnRHR3). GnRH-II is much more potent than GnRH-I in hens in stimulating LH release by 36 fold.[45] However, GnRH-II is not detected in the median eminence.[46,47] There is high expression of GnRHR3 in the pituitary gland.[48] Therefore, the releasing hormone for LH is chicken GnRH-I and the receptor is cGnRHR3.

Chicken gonadotropin-inhibitory hormone (GnIH) is a peptide with 12-amino-acids.[49] While GnIH inhibits both the synthesis and the release of gonadotropins in chickens,[50] the physiological relevance of GnIH still requires clarification.

The ovary produces the following:

- Estrogens, primarily estradiol. Estrogens induce the following: development of the oviduct, production of yolk precursors (VLDL and vitellogenin) (see above) by the liver, production of egg white proteins by the oviduct and, with androgens, formation of medullary bone (a labile source of calcium). In addition, estrogens allow the expression of female behaviors and moderate the release of luteinizing hormone (LH).
- Progesterone. Among its roles are stimulating production of a specific egg white protein (avidin) and stimulating the release of LH.
- Androgens, predominantly testosterone. Androgens are essential to the development of medullary bone.[26,51]

Ovarian hormones and growth factors also play critical roles in follicular development. For instance, activin A increases expression of both FSH and LH receptors but decreases cell proliferation of granulosa cells.[52] Moreover, development of small follicles is suppressed by epidermal growth factor receptor ligands such as transforming growth factor α.[57]

In contrast, bone morphogenetic protein 6 enhanced responsiveness to FSH.[43,53]

Testicular functioning is controlled in a similar manner to the ovary. LH stimulates the Leydig cells to produce testosterone.[41] The Sertoli cells produce nutrients to the maturing spermatozoa and are under the control of FSH. Testosterone is produced from the Leydig cells.

Light and reproduction

Photoperiodic induction of reproduction

There is seasonality of egg production in chickens when held under a natural photoperiod in the temperate zone. Egg production increases markedly after the winter solstice and declines beginning prior to the autumnal equinox.[54] The physiological basis of this annual cycle is photoperiodic stimulation of reproduction by long daylengths; these inducing the development of functioning gonads.[54] Red light is detected by photo-pigments in the hypothalamus[54−56] with the most important photoreceptor influencing the hypothalamic release of GnRH-I being red opsin.[55] The photoperiodic mechanism involves light coinciding temporarily with the light sensitive (photo-sensitive) phase of a circadian rhythm. This leads to release of GnRH-I, synthesis and secretion of LH and FSH and, hence, gonadal resurgence.[54]

Chickens Pullets are reared under short daylengths (6L:18D or 8L:16D). They are transferred to longer daylengths (12L:12D) at breed specific physiological ages[45] to stimulate gonadal development. In studies where pullets were transferred to daylengths of 10L:14D or 11L:13D, plasma concentrations of LH did not increase, but marked increases in plasma concentrations of LH daylengths were observed with daylengths 13L:11D or greater.[54] Perhaps surprisingly, daylengths were interpreted differently depending on the previous photoperiod. Transfer of pullets from photoperiods of either 4L:20D or 20L:4D to 12L:12D were followed by, respectively increases and decreases in plasma concentrations of LH.[54] Thus, the same photoperiod can be interpreted as either photostimulatory or photoinhibitory.

Commercially, pullets are brought into lay by increasing both the photoperiod and the light intensity (Table 20.3). There are breed and genetic line specific differences between ages at

TABLE 20.3 Photoperiodic stimulation of egg production in poultry hens by photoperiod and light intensity.

	Age at photo-stimulation (weeks)	Photoperiod prior to photostimulation	Photoperiod at photostimulation	References
Layer pullets	17	12L:12D	13L:11D[a]	75
Broiler breeders	≥21	8L:16D	12L:12D[b]	64
Turkeys	29	6L:18D	14L:10D	127
		Light intensity		
		Prior to photostimulation (lux)	After to photostimulation (lux)	
Layer pullets	17	20−25	30	75
Broiler breeders	≥ 21	5−7	>50 to <100	64
Turkeys	29	20−100	120	127

[a] Daylength 13L:11D (week 17) and thereafter increased by $1/4$ hour per day every week until 16L:8D.
[b] Daylength 12L:12D (week 21 or later) and thereafter increased by 1 h per day every week until >14L:9D.

TABLE 20.4 Seasonal differences in photoperiodic responses of 54 week-old turkey hens after 8 weeks on 6L:18D.

	Summer	Winter
Photoperiod inducing reproduction	≥14L:10D	≥ 11.5L:12.5D
Night interruption with 1 h light relative to dawn at the times indicated	≥16 to 17	≥10 to 11

Based on Siopes.[60]

which photostimulation is performed (see Table 20.3). Egg production in broiler breeder hens is improved when photostimulation is delayed from 18 to 21 weeks old.[57] There is also evidence that it may be advantageous to delay photostimulation of broiler breeders to 24 weeks old.[58]

Turkeys: Turkeys come into lay with increasing daylengths.[59] There are seasonal differences (see Table 20.4).[60] There is a rapid increase in plasma concentrations of both LH and FSH when males and females are exposed to long day-lengths.[61]

Photorefractoriness and reproduction

Photorefractoriness is the loss of the ability to respond to the stimulatory effects of long photoperiods. Photorefractoriness can be "broken" by re-exposure to short day-lengths. This is seen in turkeys with prolonged exposure to long day-lengths with the signs of photorefractoriness being decreased egg production and molting.[59,60] The decline in egg production during the production cycle of chickens might also be attributed to photorefractoriness. Indeed, there is greater sensitivity of older hens to reduced daylength with an over 90% decrease in egg production in 105 weeks old hens compared to a 26% decline in 28 weeks old hens.[62] Moreover, plasma concentrations of LH were only decreased in the older hens.[62]

Light intensity and poultry reproduction

While, light intensities greater than 1 lux are required for photoperiodic induction of egg production,[63] considerably higher light intensities are employed in commercial poultry production (Table 20.3). For instance, in broiler breeders, light intensity is increased from before photostimulation about 6 lux in the pullet phase to >50 lux after photostimulation at 21 or 22 weeks of age.[64]

Other effects of light intensity: Light intensity has other effects. For instance, increasing light intensity in immature pullets is associated with increased plasma concentrations of FSH.[65] Moreover, the ability of a short pulse of light to photostimulate chickens is influenced by light intensity.[66] In addition, the ratio of the light intensity during the subjective day to that during the subjective night is important in entraining the rhythm of oviposition.[67]

Nutrition and reproductive management

Overview

In poultry, nutrition is integrally linked to the hypothalamo-pituitary-gonadal axis. It has been known for 50 years that egg production in hens stops quickly following fasting.[68] The administration of mammalian or avian gonadotropin restores, albeit partially, egg production in starved hens[68]; this suggesting that underlying cause is the lack of pre-ovulatory LH surges. Fasting is followed rapidly by decreases in plasma concentrations of LH,[69] body weight together with precipitous declines in ovarian and oviductal weights.[70] Similarly, production of eggs and plasma concentrations of LH decrease quickly after reducing calcium or sodium in the diet of hens.[71,72] In young chickens, protein deficiency also has been demonstrated to rapidly cause atrophy of gonads, decrease circulating concentrations of LH and depress responsiveness to GnRH.[73]

The *NRC Nutrient Requirements of Poultry* has been invaluable to researchers and important to the poultry industry recommending minimum levels of nutrients in the feed.[74] These requirements are based on the published research prior to the development of the specific edition of *NRC Nutrient Requirements of Poultry.* Primary breeders publish age specific nutrient recommendations for each of their genetic lines.[64,75−77] Examples of such recommendations for energy, protein, lysine and calcium are summarized in Table 20.5. It is clear that the recommendations for calcium content in laying hen diets are very high due to the demands of eggshell formation. Moreover, the levels of calcium in diets are higher as the production cycle proceeds presumably due to the increasing size of the egg. This is the case irrespective of whether the recommendations are for layer or broiler breeder hens (Table 20.5).

Broiler breeder reproduction

Nutrition of broiler breeder pullets and reproductive management

Feeding programs are designed to achieve target body weights throughout growth with markedly lower weights at 24 weeks old (*ad libitum* fed 5.65 kg; restricted to achieve target weights 3.06 kg).[78] These programs not only decrease the feed needs of the broiler breeder but also reduce mortality and increase egg production.[78]

Broiler breeder pullets can be fed nutrient restricted diets by programs where birds are fed daily or skip-a-day or feeding four or five or six days per week. There were greater body weights and lower liver weights in 16 week-old pullets fed daily than skip-a-day despite the birds despite their receiving the same amount of feed.[79] There were also higher hepatic concentrations of both lipid and glycogen together with the expression of lipogenic enzymes in skip-a-day fed pullets.[79,80]

Mench considered that feed restrict of broiler breeder females may be associated with physiological stresses and increased incidence of abnormal behaviors.[81] The severity of feed restriction needs to be progressively greater with generation exhibiting increased size/growth rates in broiler chickens.[82] The strategy in feed restriction is to reduce caloric intake while maintaining amount of feed consumed. This goal is achieved by increasing the percentage of crude fiber in the diet. Skip-a-day programs have been considered helpful in increasing uniformity within flocks and reducing abnormal behaviors.[83]

Induced molting or re-cycling to increase egg production

Hens can be induced (or forced) to molting at the end of their laying cycle resulting in improved egg production at a lower cost than using replacement pullets (Fig. 20.1). In the USA, 19.7 % of laying hens are molted (re-cycled) each month.[5] This process can involve severe nutritional restriction including starvation and/or withholding water and/or reduction in photoperiod.[70,84−86] Alternate methods of induced molting include an extremely high zinc diet (20,000 ppm) followed by a conventional layer feed beginning at day 12[87] and sodium/chloride-deficient diets.[88,89]

Broiler breeders are rarely molted, but under certain circumstances, molting may be performed. Most broiler breeder molt programs are achieved by restricting feed consumption and supplementing water containing essential micronutrients allowing utilization of fat stores. This reduced fat stores such that hens achieve a more pullet-like body composition before being photostimulated again. In addition to feed restriction, to induce molting in broiler breeder hens, the daylength is decreased to 8L:16D and light intensity is reduced.[85,86] Production levels with molted broiler breeders are about 10% less than their previous laying cycle.[85,86] The lower production level appears to be due to there being fewer follicles after a forced molt compared to their initial lay cycle.[25]

TABLE 20.5 Dietary levels of nutrients for chickens recommended by primary breeders.

	Energy kcal kg^{-1}	Crude protein[a]	Lysine[a]	Digestible lysine[a]	Calcium[c]
LAYER LINE[b]					
Grower	2977−3087	17.5	0.96	0.88	1.0
Developer	2977−3131	16.0	0.83	0.76	1.0
Prelay	2911−2955	16.5	0.85	0.78	2.5
Layer feed 1 (first egg to 2% below peak)	2844−2955	16.0	0.881	0.805	4.15
Layer feed 2 (2% below peak to 90% production)	2844−2944	15.5	0.821	0.750	4.30
Layer feed 3 (89−85% production)	2822−2922	15.25	0.777	0.710	4.40
Layer feed 4 (84−80% production)	2800−2844	15.0	0.761	0.695	4.60
Layer feed 5 (<80%)	2778−2822	14.75	0.745	0.680	4.65
BROILER BREEDERS[c]					
Cobb 500F					
Starter (0−4 weeks)	2796	18.54	1.00	0.90	1.00
Grower (5−18 weeks)	2581	14.45	0.59	0.49	0.99
Pre-Breeder (19−22 weeks)	2761	15.43	0.72	0.61	1.45
Breeder 1 (23−40 weeks)	2761	15.43	0.72	0.64	2.89
Breeder 2 (>41 weeks)	2749	14.50	0.58	0.62	3.08
BROILER BREEDER[d]					
Cobb 700					
Starter (0−4 weeks)	2900	19.00	1.04	0.93	1.00
Grower (5−16 weeks)	2700	15.00	0.72	0.61	1.00
Developer (17 weeks to 1st egg)	2800	15.00	0.74	0.62	1.30
Breeder 1 (1st egg to 35 weeks)	2850	15.0	0.75	0.66	3.05
Breeder 2 (36+ weeks)	2750	14.5	0.72	0.64	3.25
Ross 308 and 708[e]					
Grower (4 weeks−5% egg production)	2800	14−15	0.68	0.61	0.9
Breeder 1 (5% eggs - 35 weeks)	2800	15	0.67	0.60	3.0
Breeder 2 (35−50 weeks)	2800	14	0.62	0.56	3.2
Breeder 3 (>50 weeks)	2800	13	0.58	0.52	3.4

[a] *Expressed as % of feed for broiler breeder pullets and hens but g day^{-1} for layers (total feed consumption ~97 g after 35 weeks old).*
[b] *Based on Hy-line.[75]*
[c] *Based on Vantress.[64]*
[d] *Based on Cobb-Vantress.[128]*
[e] *Based on Ross.[76,77]*
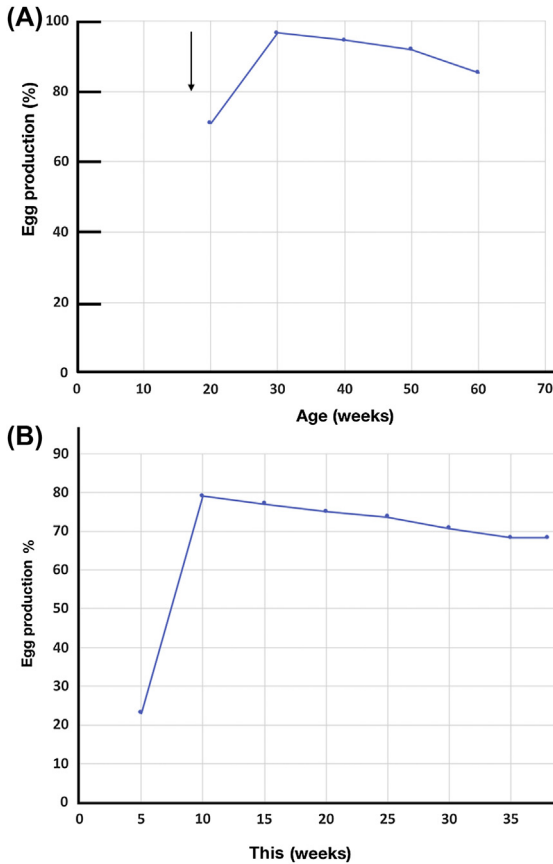
(A)

(B)

FIG. 20.1   Egg production in the first (A) and second cycle (B) of egg production. Time is age in weeks. *Arrow* indicates induction of lay at 17 weeks old. *Data from Yilmaz Dikmen et al. and Gordon et al.*[114,124].

FIG. 20.2   Changes in egg production (%) and feather loss (number of wing cast feathers lost per day for 47 hens). *Data from Hoshinon et al.*[90]

LH, estradiol (E_2) and progesterone were lower in molting hens than in laying hens or fully recycled hens.[90]

The effects of an industry molting system on organ weights together with circulating concentrations of ions and corticosterone were evaluated.[91] The approach was to combine salt and protein deprivation[91]:

• Layer diet [17.2 % protein, fiber 2.45 %, 0.4 % sodium chloride (NaCl) and metabolizable energy (ME) 1270 Mcal kg^{-1}]

• Pre-molt diet [17.2% protein, fiber 3.6 %, 0 % added NaCl & ME - 1270 Mcal kg^{-1}] for 3 days.

• Molt feed 1 [9.7% protein, fiber 3.6 %, 0 % added NaCl & ME - 1218 Mcal kg^{-1}] for 21 days

• Molt feed 2 [9.7% protein, fiber 3.6 %, 0.3 % added NaCl & ME - 1214 Mcal kg^{-1}] for 3 days

The terms, forced or induced molt, are open to question as it presumes that molting (loss of feathers) causes rejuvenation of reproduction performance. Molting occurs after resumption of normal feeding and is temporally shifted from ovarian recrudescence (see Fig. 20.2). When feed is withdrawn for 8 days and water withdraw for 2 days, egg production had completely ceased by 6 days (see Fig. 20.2).[90] Molting occurred after the resumption of feeding and there were concomitant increases in circulating concentrations of T_3 and corticosterone (see Fig. 20.2).[90] Circulating concentrations of
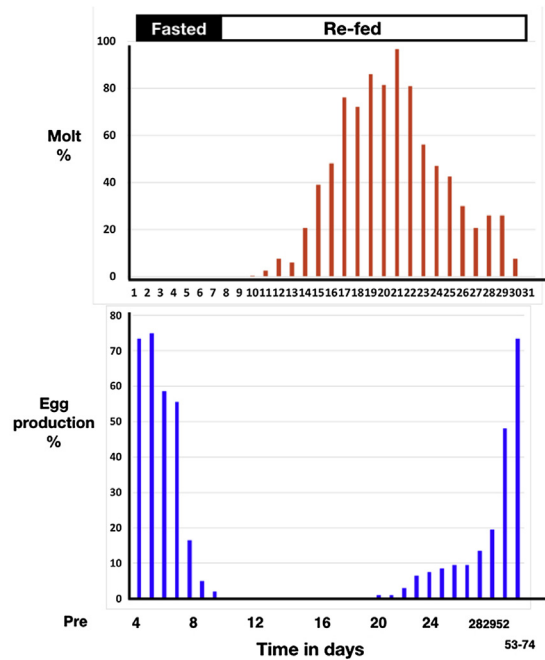
- Molt feed 3 [16.0% protein, fiber 3.2 %, 0.4 % added NaCl & ME - 1274 Mcal kg^{-1}] for 7 days
- Layer diet [17.2 % protein, fiber 2.45 %, 0.4 % added sodium chloride & ME - 1270 Mcal kg^{-1}]
- Photoperiod was maintained at ∼17 h light per day.

This approach is effective in stopping egg production and decreasing body weight and organ weights.[91] It is accompanied by reductions in circulating concentrations of sodium and chloride (see Table 20.6).[91] The egg production cycle of about 21 weeks of age to 45−60 weeks old can be extended with a molt between 108 and 120 weeks old.[91]

The physiological mechanism underlying induced molting included decreased release of GnRH from the median eminence and consequently lack of the pre-ovulatory LH surge. Ovulation completely ceased with 4 days of feed withdrawal.[92] Plasma concentrations of LH and progesterone were decreased with 2 days of feed withdrawal.[92] The GnRH content of the median eminence was similarly decreased but not until 4 days of feed withdrawal.[92] There are also

decreases in the number of gonadotropes expressing LH.[93] Oviductal regression occurs due to lack of estrogens and is accompanied by increased expression of peptidases with, for example, expression of the peptidase, cathepsin L.[94]

It is questioned whether re-cycling/forced molting of hens is consistent with one of the "The Five Freedoms," namely: "1. Freedom from hunger and thirst" and the need for "ready access to fresh water and a diet to maintain full health and vigor."[95] There is both evidence for and against the process being physiologically stressful. An indicator of stress, heterophil: lymphocyte ratio, was increased after 7 days after feed withdrawal to a forced molt.[96] Similarly, induction of molting was accompanied by increased the percentage of heterophils (day 7 and 14) and of eosinophils in one study.[97] However, the evidence of molt induction influencing plasma concentration of the stress hormone, corticosterone, is circumspect. No changes in plasma concentrations of corticosterone in force molted hens were reported (also see Table 20.6).[91,94]

In laying hens subjected to induced molting, plasma concentrations of corticosterone were higher on day 10 in hens subjected to complete

TABLE 20.6 Changes in body and organ weights together with plasma concentrations of sodium, chloride and corticosterone in hens subjected to a molt using an industry approach of a combination of salt and protein deprivation.[91]

Parameter	Physiological state					
	Control	Molt week 1	Molt week 2	Molt week 3	Molt week 4	SEM (n = 5)
Body weight loss (%)	3^a	12^{a,b}	19^b	21^b	13^{a,b}	4.9
Ovary (g)	25^a	18^{ab}	13^b	13^b	18^{a,b}	5.0
Oviduct (g)	64^a	44^b	25^c	27^c	46^b	10
Liver (g)	41.9^a	38.9^a	29.0^b	29.7^b	37.0^{a,b}	2.3
Small intestine (g)	64.1^a	52.9^{a,b}	46.3^b	51.4^{a,b}	62.3^{a,b}	3.4
Plasma Na$^+$ (Mequiv L^{-1})	169^a	147^b	147^b	146^b	150^b	3.7
Plasma Cl$^-$ (Mequiv L^{-1})	120^a	116^b	117^b	116^b	119^{a,b}	3.7
Plasma CORT (pg mL^{-1})	296	313	498	282	548	100

^{a,b,c} Different leters in a row indicate difference p<0.05.

feed withdrawal than those in which molting was induced by a high zinc diet or in a whole-grain barley diet.[98] There are also shifts in immune responses including cytokine expression. During starvation induction of molting, there is a reduced delayed type hypertensive response in the wattle.[99] Feed withdrawal for 6–8 days is associated with increased oviductal expression of the following: interleukin (IL)-6, IL 8 and interferon γ.[100] Similarly, expression of cytokines in response to an endotoxin challenge increased in the uterus of molting compared to laying hens.[101]

Other aspects of reproductive management in poultry

Artificial insemination

Artificial insemination (AI) is used very widely in turkey production but not in the production of layer pullets or broiler chicks.[102] Hens are inseminated weekly with diluted semen (for spermatozoa concentrations in turkey ejaculate see Table 20.2). While the technique is labor intensive, it takes advantage of the presence of spermatozoa storage glands in the oviduct.[102] These release spermatozoa after oviposition such that newly ovulated ovum can be fertilized prior to the addition of egg white, membranes and the shell.[102] The AI techniques that were developed for poultry have been applied to the conservation of endangered wild birds when they fail to mate in captive conditions.[103] AI can be employed alone or coupled with cryopreservation.[103]

Approaches to reduce broodiness

It is advantageous to suppress broodiness (maternal behavior) and the consequent decrease in egg production in poultry. Broodiness has been effectively genetically eliminated in commercial chickens irrespective of whether broilers or laying hens.[104] Approaches that could reduce the unfavorable effects of broodiness in turkeys include: genetics and active or passive immunization with antisera reducing available prolactin. Administration of antisera to turkey prolactin increased egg production in turkeys.[105] An alternate approach is active immunization. Similarly, egg production was markedly increased in turkey hens actively immunized against VIP (with VIP being conjugated to keyhole limpet hemocyanin) due to impairment of prolactin release and consequently reduction in incubation behavior(s).[106,107] It was hoped that antisera could also enhance reproduction in males. Young male chickens exhibited multiple changes after active immunization against both inhibin and VIP. These included increases in semen volume and spermatozoa mobility together with spermatozoa concentrations.[108] In contrast, this improvement in reproductive performance was not seen in old roosters actively immunized against both inhibin and VIP.[109] Antibody approaches do not appear to be widely adopted due to expense of registration.

Cage free (colony), conventional, enriched and free-range systems

Both egg production and bird health are influenced by the systems employed to maintain (chicken) hens. Egg production was greater in the hens in conventional caging compared to an aviary system. Much lower (~40%) mortalities were reported in hens in conventional caging compared to an aviary system.[110] Moreover, there was a greater incidence of keel deformities in hens in a cage free system compared to those in conventional cages.[111] There are also differences in leg bone characteristics with hens under the cage free conditions having increased cortical cross-sectional area and cortical density of their humerus and tibia compared to those maintained in conventional cages.[112] Moreover, there was there was greater stiffness of both humerus and tibia and increased percentage ash in the humerus in hens in a cage free system.[112,113]

However, in another study, there were little differences between tibia and humerus parameters between hens in cage-free or conventional cage systems.[111] In laying hens at the end of the laying cycle, there were differences in bone characteristics depending on the environment under which the pullets were raised.[112]

Production has been compared between Lohmann Brown layer hens in convention layer housing, enriched convention layer housing and free-range systems. Perhaps unexpectedly, egg production was greater in hens in a free-range system (average production over cycle 89.3%) compared to conventional layer housing (87.3% with enrichment and 87.1% without).[94] In contrast, there was greater feed intake and higher feed conversion ratios in hens in a free-range system.[114] In another study, egg production was greater in hens with an enriched environment compared to conventional caging.[110]

Ovulation issues in broiler breeders

Broiler breeders are prone to ovulation issues. Examples include internal ovulation and double ovulation of a (double "yolked" eggs). The latter is due to pair of large yellow follicles in the follicular hierarchy.[25] Duplicate ovulation is considered to be a result of "over-stimulated" hens coming into production or pullets not ready for light stimulation or to over-feeding.[113,115] Internal lay of the ovum is followed the constituents of ovum being absorbed, but can result in a substrate for bacterial growth like *Escherichia coli*.[116]

Uses of components of eggs

Yolk as source of antibodies

A major soluble protein type in yolk are the γ livetins or the immunoglobulins (discussed section above). Yolk IgY from chicken immunized against pathogens (rotaviruses, bovine coronavirus, enterotoxigenic *E. coli* and *Salmonella* spp.)

offers potential to replace antibiotics in livestock production. This may be particularly important with the restriction of antibiotic use and growing consumer resistance to their use. A meta-analysis of 22 studies determine that hyperimmune yolk IgY was effective against diarrhea in piglets.[117] Similarly, oral administration of IgY was efficacious in poultry and calves.[117,118]

Other products of eggs

Egg white and its constituents have important functional properties. Examples include the following. Avidin is used in multiple biochemical tests. Lysozyme (bacteriostatic and bactericidal) has been used as a preservative, for instance, in cheese making.[27]

Egg shell membranes or egg shell membrane powder have been used as natural bandage for burns and injuries in traditional Asian medicine. Egg shell membrane powder and its carbohydrate constituents have been recently been demonstrated to exert anti-inflammatory effects[119,120] supporting their effectiveness.

Transgenic chickens as bioreactors

In the chick embryo, primordial germ cells migrate from the germinal crescent to the genital ridge ultimately becoming male or female gametes. The primordial germ cells carry the genetics to the next generation and, thus, if this can be manipulated we have a multi-generational method of producing transgenic animals. Chicken primordial germ cells can be obtained from blood of chick embryos and maintained in culture.[121] This has been used to both insert and/or "knock out" genes.[121]

Transgenic hens can be used as bioreactors to produce pharmaceutical proteins in their egg white when oviduct specific promoters are used.[122] This concept has been applied to the production of human erythropoietin[123] and tumor necrosis factor receptors.[115]

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