

# Parthenogenesis in birds: a review

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## Abstract

Parthenogenesis or 'virgin birth' is embryonic development in unfertilized eggs. It is a routine means of reproduction in many invertebrates. However, even though parthenogenesis occurs naturally in even more advanced vertebrates, like birds, it is mostly abortive in nature. In fact, multiple limiting factors, such as delayed and unorganized development as well as unfavorable conditions developing within the unfertilized egg upon incubation, are associated with termination of progressive development of parthenogenetic embryos. In birds, diploid parthenogenesis is automictic and facultative producing only males. However, the mechanisms controlling parthenogenesis in birds are not clearly elucidated. Additionally, it appears from even very recent research that these mechanisms may hinder the normal fertilization process and subsequent embryonic development. For instance, virgin quail and turkey hens exhibiting parthenogenesis have reduced reproductive performance following mating. Also, genetic selection and environmental factors, such as live virus vaccinations, are known to trigger the process of parthenogenesis in birds. Therefore, parthenogenesis has a plausible negative impact on the poultry industry. Hence, a better understanding of parthenogenesis and the mechanisms that control it could benefit commercial poultry production. In this context, the aim of this review is to provide a complete overview of the process of parthenogenesis in birds.

*Reproduction* (2018) 155 R245–R257

## Introduction

Parthenogenesis is a unique form of reproduction where embryonic development occurs in unfertilized eggs. Beatty (1967) defined parthenogenesis as 'the production of an embryo from a female gamete without any genetic contribution from a male gamete and with or without eventual development into an adult'. Therefore, any degree of development in unfertilized eggs is regarded as parthenogenesis. Moreover, it is distinct from asexual forms of reproduction, such as fission and budding, as because it involves the female gametes, and is often, regarded as an incomplete form of sexual reproduction (Mittwoch 1978).

Charles Bonnet, a naturalist and philosopher from the mid-eighteenth century, first reported the phenomenon of parthenogenesis in aphids (<https://hpsrepository.mbl.edu/handle/10776/1745>, accessed on 10/20/2017). In fact, parthenogenesis is a very common, naturally occurring phenomenon among the lower orders of the animal kingdom, especially invertebrates (Suomalainen 1950, Edwards *et al.* 2003, Lourenço 2008). Among vertebrates, natural occurrence of parthenogenesis yielding live offspring has been documented in some lower order vertebrates, such as Whiptail lizards (David & Moore 1993), Komodo dragons (Watts *et al.* 2006), hammerhead sharks (Chapman *et al.* 2007) and Boa constrictor and python snakes (Booth *et al.* 2011, 2014) in captivity as well as copperhead and cottonmouth pit

viper snakes (Booth *et al.* 2012) in the wild. Even though naturally occurring parthenogenesis is phylogenetically widespread among these vertebrate lineages, the establishment of parthenogenesis in higher orders of the animal kingdom appears to be difficult (Suomalainen 1950). In higher order vertebrates, natural parthenogenesis is usually abortive in nature (Whittingham 1980). In fact, in mammals, the functional specialization of paternal and maternal genomes, genomic imprinting, act as a barrier for natural parthenogenesis (Kono 2006). On the other hand, genomic imprinting is believed to be absent in birds; therefore, natural parthenogenesis exist but is generally abortive with a few notable exceptions in turkeys and chickens (Olsen 1975). Interestingly, birds are the highest order vertebrates where naturally occurring parthenogenesis results in adult parthenogens, and hence, avian species can serve as an ideal model to study parthenogenesis to gain more information on the evolution of sexual mode of reproduction as well as the association of ovarian cancers with spontaneous parthenogenetic development in mammalian oocytes. Moreover, among avian species, the Chinese painted quail (*Coturnix chinensis*) serve as an excellent model for developmental studies on parthenogenesis (Parker & McDaniel 2009) due to their small size, short generation interval and early age of sexual maturity when compared to chickens and turkeys (Tsudzuki 1994). Also, these

**Table 1** Avian model of choice for developmental studies vs applied studies on parthenogenesis.

	Family	Order	Age at sexual maturity	Egg production	Incubation length	Adult body weight	References*	Recent findings on parthenogenesis
Preferred species for developmental studies								
Chinese painted quail ( <i>Coturnix chinensis</i> )	Phasianidae	Galliformes	6–8 week	60–80%	16–17 day	50 g	Tsudzuki (1994)	Discovery (Parker and McDaniel 2009) A heritable trait (Parker et al. 2010) Incubational egg weight loss (Wells et al. 2012) Hindrance to normal fertilization (Parker et al. 2012) Influence of incubational temperature (Santa Rosa et al. 2015) Parthenogens alter egg environment as viable embryos (Santa Rosa et al. 2016b) Resembles early embryonic mortality and inhibits fertility (Santa Rosa et al. 2016a) Parental sex effect (Parker et al. 2017)
Preferred species for applied studies								
Commercial turkey ( <i>Meleagris gallopavo</i> )	Phasianidae	Galliformes	28 week	60–80%	28 day	10 kg	<a href="http://www.aviagenturkeys.com/uploads/2015/12/07/POBRB10_V3_BUT%2010_BreederGoals_UK.pdf">http://www.aviagenturkeys.com/uploads/2015/12/07/POBRB10_V3_BUT%2010_BreederGoals_UK.pdf</a> , accessed on 2/12/2018	Incidence is 3% (Bakst et al. 2016)
Commercial broiler breeders ( <i>Gallus domesticus</i> )	Phasianidae	Galliformes	24 week	60–80%	21 day	2.5 kg	<a href="http://en.aviagen.com/assets/Tech_Center/Ross_PS/Ross708-PS-PO-EN-2016.pdf">http://en.aviagen.com/assets/Tech_Center/Ross_PS/Ross708-PS-PO-EN-2016.pdf</a> , accessed on 2/12/2018	No recent studies available

\*References for age at sexual maturity, egg production, incubation length and adult body weight.

characteristics make parthenogenesis research using quail as a model extremely cost- and time-effective (Table 1).

In birds, as in other vertebrate lineages parthenogenesis appears to be phylogenetically widespread and was first reported in the chicken by Oellacher (1872). Thereafter, this phenomenon has been discovered in a variety of avian species, such as pigeons (Bartelmez & Riddle 1924), turkeys (Olsen & Marsden 1954a), zebra finches (Schut et al. 2008) and Chinese painted quail (Parker & McDaniel 2009). In all these species, almost all parthenogenetic development is unorganized and abortive. However, following intense genetic selection for parthenogenesis Olsen (1975) was eventually able to hatch approximately 1% of unfertilized turkey eggs.

Parthenogenesis in birds is diploid, automictic and facultative producing only males (Olsen 1975).

In fact, the majority of vertebrates, such as varanid lizards, elasmobranch fishes and boa and python snakes, exhibit facultative parthenogenesis (Watts et al. 2006, Chapman et al. 2007, Booth et al. 2011, 2014), whereas a small number (lizards and a single species of snake, the Brahminy blind snake) exhibit obligate parthenogenesis (a form different than facultative in both mechanism and outcome; Booth & Schuett 2016). In animals exhibiting facultative parthenogenesis, an egg may develop either by normal fertilization or parthenogenetically. In automictic parthenogenesis, the early state of meiosis is similar to eggs undergoing fertilization resulting in chromosome reduction. Subsequently, the diploid chromosomes are restored through either fusion of two haploid nuclei (the egg nucleus and the second polar body nucleus, known as terminal fusion automixis), the formation of a restitution nucleus or endomitosis. Parthenogens

**Table 2** A summary of parthenogenesis observed in the unfertilized eggs from different virgin avian species.

Avian species	Number of hens	Number of eggs	Incidence (%)	Most advanced stage of development observed	Hatched parthenogens (%)	Comments	References
Commercial turkey	10	266	3	Unorganized sheets of epithelial cells	0	Based on microscopic examination of 8 8-day incubated eggs	Bakst <i>et al.</i> (2016)
Beltsville small white turkey	30	934	16.9	Hatched poults	0.88	Based on macroscopic examination of 10-day incubated eggs	Olsen & Marsden (1968), Olsen (1975)
Dark Cornish chicken	375	15,629	6.38	Hatched chicks	0.03	Based on macroscopic examination of 10 10-day incubated eggs	Olsen (1966b), Sarvella (1970)
Barred Plymouth rock chicken	51	1245	0.16	Unorganized sheets of epithelial cells	0	Based on macroscopic examination of 10 10-day incubated eggs	Olsen (1966b)
White Leghorn chicken	87	984	0	No development	0	Based on macroscopic examination of 10 10-day incubated eggs	Olsen (1966b)
Chinese painted quail	298	4000	4.8	Unorganized sheets of epithelial cells	0	Based on macroscopic examination of 10 10-day incubated eggs	Parker & McDaniel (2009)
Zebra Finch	3	34	21	Irregular cell nuclei in the germinal disc area	0	Based on microscopic examination of fresh eggs	Schut <i>et al.</i> (2008)
Pigeon	Not reported	Not reported	Not reported	Fluid-filled subgerminal cavity	0	Based on microscopic examination of 4–9-day incubated eggs	Bartelmez & Riddle (1924)

developing through this type of parthenogenesis always have a diploid number of chromosomes (Suomalainen 1950, Olsen 1975, Lampert 2008). However, in the initial stages of development, turkey parthenogens have a high proportion of haploid cells, which are replaced by diploid cells as development advances (Sato & Kosin 1960). Cytological studies in turkey parthenogens have established that they are diploid males (Sato & Kosin 1960, Cassar *et al.* 1998a,b). However, Portnoy *et al.* (2014) reported the production of the first haploid vertebrate parthenogen in a whitetip reef shark.

It appears that these mechanisms regulating parthenogenesis in birds may actually work against normal fertilization. Recently, studies on quail reported that virgin hens exhibiting parthenogenesis have reduced fertility and hatchability following mating (Parker *et al.* 2012, Santa Rosa *et al.* 2016a). Moreover, as parthenogenesis is present in the modern poultry industry (Bakst *et al.* 2016), it is possible that the reproductive performance of commercial poultry species may be negatively impacted by parthenogenesis. Therefore, a better understanding of parthenogenesis and the mechanisms that control it could benefit the commercial poultry industry. In this review, we will try to summarize all available information on the process of avian parthenogenesis, including its potential impact on the poultry industry.

## Incidence of parthenogenesis

### Parthenogenesis in virgin birds

In 1945, Kosin found parthenogenetic development in 15% of fresh unfertilized eggs of Barred Plymouth Rock and White Leghorn hens, but this development ceased when the eggs were incubated. An abortive type of parthenogenesis that could be revived upon incubation was found in 1952 in the unfertilized eggs of Beltsville Small White (BSW) turkeys and then in 1953 in Dark Cornish chickens (Olsen 1975). The BSW turkeys were isolated from males for at least 224 days; and interestingly, 16.3% of the eggs laid had some degree of macroscopically detectable development upon incubation (Olsen & Marsden 1954b). In 1958, Poole and Olsen detected parthenogenesis in Dark Cornish hens by macroscopic examination of 10-day incubated eggs and found that 21.9% of the total hen population laid at least 1 egg that contained parthenogenetic development. Of the 81 hens that laid eggs containing embryonic development, 30 hens were responsible for laying at least 2 or more eggs that exhibited parthenogenesis. In fact, 2 hens were responsible for producing 5 embryos from 20 unfertilized eggs. More recently, Parker and McDaniel (2009) reported that in a virgin Chinese painted quail population 27% of the hens laid at least 1 egg that exhibited macroscopically detectable parthenogenetic development upon 10 days

of incubation. Of 4000 eggs examined, 4.8% exhibited some degree of development. In fact, this incidence of parthenogenesis in quail is close to that found in modern commercial turkeys, 3% (Bakst *et al.* 2016).

Interestingly, upon microscopic examination of fresh unfertilized turkey eggs, Kosin and Nagra (1956) found that 80% contained nucleated cells, and Haney and Olsen (1958) found that 90% contained nucleated cells in sectioned blastodiscs. However, upon incubation for 10 days macroscopically detectable parthenogenetic development was often reduced to 16–18% (Olsen 1975). In fact, this reduction in the incidence of parthenogenesis following incubation was due to disintegration of the nuclei within a few days after the onset of incubation (Kosin 1945, Schut *et al.* 2008). Hence, microscopic examination of fresh eggs was considered more accurate than macroscopic examination after incubation for determining the incidence of naturally occurring, rudimentary parthenogenesis due to the decline in detectable parthenogenesis after incubation (Poole & Olsen 1958; Table 2).

### **Parthenogenesis in mated birds**

Interestingly, Olsen (1962a) discovered that parthenogenesis occurs not only in virgin hens but also in the unfertilized eggs from mated BSW hens. In this study, genetic feather color markers were used which revealed that at least some eggs from mated turkeys develop parthenogenetically. However, it was concluded that parthenogenetic development did not hinder the normal fertilization process even though diploidy is restored in parthenogenetic development at the same time as fertilization would occur in the infundibulum, which could possibly result in polyploidy if sperm were to penetrate an egg already undergoing parthenogenetic development (Olsen 1962a, 1967a). More recently, Parker *et al.* (2012 and 2014) reported parthenogenesis in eggs from mated Chinese painted quail hens that exhibited the parthenogenesis trait as virgins. However, when these virgin hens exhibiting the parthenogenesis trait were mated, it was found that as the incidence of parthenogenesis in virgins increased, hatchability in mated birds decreased (Parker *et al.* 2012). Also, as generation of selection for the parthenogenesis trait increased, hatchability of eggs set and hatchability of fertile eggs decreased (Parker *et al.* 2014). Similarly, Schom *et al.* (1982) reported a negative correlation between the incidence of parthenogenesis in virgin hens and hatch of fertile eggs ( $r = -0.93$ ) in artificially inseminated Broad Breasted White turkeys. In addition, both male and female quail selected for the parthenogenetic trait appear to be responsible for the reduction in hatchability (Parker *et al.* 2017). In fact, parthenogenetic line females contribute to this reduction in hatchability following mating by increasing early embryonic mortality and the percentage of eggs

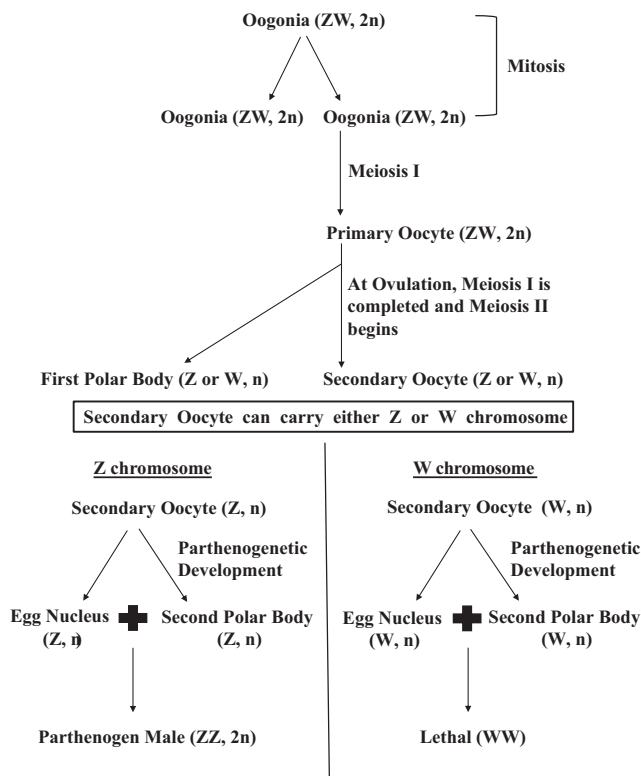
exhibiting parthenogenesis. However, the reduction in hatchability for parthenogenetic line males is due to decreased fertility as well as an increased incidence of parthenogenesis (Parker *et al.* 2017).

Similar to hatchability, genetic selection for the parthenogenetic trait negatively affected overall fertility in Chinese painted quail (Parker *et al.* 2014, Santa Rosa *et al.* 2016a). This reduction in fertility in birds selected for parthenogenesis was due to fewer sperm penetrating the egg. In fact, the average number of sperm penetration holes in the perivitelline layer of birds selected for parthenogenesis was 45 vs 171 sperm holes in eggs from the unselected control birds. Also, the percentage of eggs without any sperm holes was 21% higher in birds exhibiting parthenogenesis as opposed to the control birds (Santa Rosa *et al.* 2016a). Parker *et al.* (2017) found that males selected for the parthenogenetic trait were indeed responsible for this lower fertility due perhaps to poor semen quality, therefore allowing for more unfertilized eggs and hence more parthenogenetic eggs. These recent findings contradict Olsen's (1962a) earlier hypothesis that mechanisms of parthenogenesis do not interfere with the mechanisms of normal fertilization and embryonic development in birds. However, Olsen (1975) did report that hatched turkey parthenogens had poor semen quality.

### **Molecular mechanism of parthenogenesis**

Research has shown that avian parthenogens studied so far are males consisting mostly of diploid cells with ZZ chromosomes due to heterogamety (ZW) in the avian female sex. For example, the microscopic and histological examination of gonads from BSW turkey parthenogens revealed that all parthenogens were males (Poole & Olsen 1957). In another study, molecular sexing of BSW turkey parthenogens revealed the absence of the W chromosome indicating that all parthenogens examined were males (Cassar *et al.* 1998a). In fact, this is similar to that observed in caenophidian snakes with the ZW sex-determining system where all parthenogenetic offspring produced were males (Booth & Schuett 2016). In addition, turkey parthenogens at their initial stages of development were mosaics of haploid, diploid and polyploid cells (Sato & Kosin 1960, Harada & Buss 1981). However, as development progressed, the proportion of haploid cells declined, such that in hatched turkey parthenogens more than 87% of the cells were diploid (Sato & Kosin 1960, Cassar *et al.* 1998b). However, Sarvella (1970) reported triploidy (ZZW) in an adult chicken that was likely a parthenogen. In fact, parthenogenetic embryos begin their development from haploid ova, and eventually, the diploid chromosome number is established (Darcey *et al.* 1971, Deford *et al.* 1979, Harada & Buss 1981). However, the mechanism of restoration of diploidy is not yet completely understood.





**Figure 1** A schematic representation of the proposed molecular mechanism of avian parthenogenesis – Terminal Fusion Automixis.

The proposed molecular mechanism for avian parthenogenesis is either the retention of the second polar body with the egg nucleus or absence of meiosis II (Fig. 1; Sato & Kosin 1960, Olsen & Buss 1972, Olsen 1975). During normal fertilization, viable sperm present at the infundibulum penetrate the ovum, thus meiosis II is completed, expelling the second polar body and allowing the fusion of haploid sperm and egg pronuclei to form a diploid zygote (Olsen 1942, Olsen & Fraps 1944). However, when viable sperm are absent or when sperm fail to penetrate the ovum, the second polar body is likely not completely expelled from the ovum. Eventually, the second polar body and the egg nuclei may fuse and form a reconstituted nucleus containing a diploid number of chromosomes (Sato & Kosin 1960, Olsen 1975). Due to the ZW sex determining system in the avian species, the second polar body and the egg nuclei can carry either Z or W chromosome. The fusion of the two carrying Z chromosomes results in the formation of a homogametic (ZZ) male, whereas the fusion of the two W chromosomes is lethal (WW; Harada & Buss 1981). Therefore, the unfertilized ovum carrying the Z chromosome resumes development as a diploid cell with all genetic material of maternal origin and yields a male parthenogen (Olsen 1975).

Also, cytological studies have shown that avian parthenogens are heterozygous at certain loci. Based on

skin grafting (Poole *et al.* 1963, Poole 1965) and down color marker (Olsen 1966a) tests, turkey parthenogens were shown to be heterozygous at one or a few loci. Normal meiotic reduction and crossing over followed by the retention of the second polar body with the egg nucleus could produce diploid males containing chromosomes heterozygous within crossover regions for any loci at which the dam was also heterozygous (Olsen 1975).

## Characteristics of parthenogenesis

### Before and after oviposition

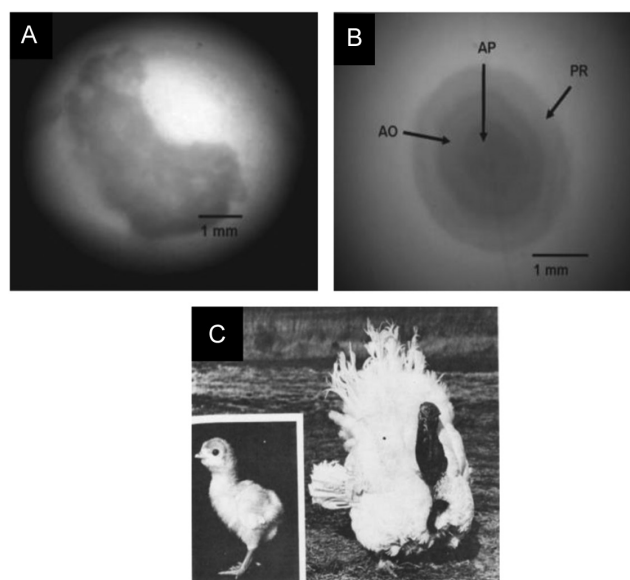
Prior to lay, parthenogenetic embryos experience a time lag in development when compared to normal embryos from fertilized eggs (Olsen 1965a). Turkey parthenogens undergo first cleavage at the uterus (Haney & Olsen 1958) as opposed to the magnum in normal fertilized embryos (Olsen & Fraps 1944). Hence, the first cleavage is delayed by about 2–3 h in unfertilized parthenogen embryos (Haney & Olsen 1958). Following cleavage, cells tend to fuse, pile up and form multiple layers rather than spread laterally as a single-layered blastoderm (Olsen 1965a). This lack of cellular organization is hypothesized to be due to the absence of sperm (Bartelmez & Riddle 1924, Olsen 1975). In addition, during early parthenogenetic development and even at lay, the cells are solely large yolk-laden blastomeres as opposed to small epithelial-type cells found in freshly laid fertilized eggs (Olsen 1965a). Also, these parthenogenetic cells are often necrotic, disorganized and arrested in development (Olsen 1942, Parker & McDaniel 2009).

At lay, parthenogens are in the early blastula stage (Haney & Olsen 1958), whereas fertilized embryos are often in the early gastrula stage of development (Hays & Nicolaidis 1934). Hence, the delayed parthenogens require 2 additional days of incubation as compared to fertile eggs to hatch (Olsen 1965a). In fact, in fresh Chinese painted quail eggs, the germinal discs exhibiting parthenogenetic development were slightly smaller than those of fertilized eggs due to delayed embryonic development (Parker & McDaniel 2009). Moreover, in the fresh eggs from virgin and non-mated Barred Rock and White Leghorn hens examined by Kosin (1945), 15% of the blastodiscs showed cells containing intact nuclei and in the process of mitosis. However, the duration of mitosis was brief, terminating at approximately 24 h after oviposition and thus, could not be revived upon incubation. Also, in zebra finches with parthenogenetic development, germinal discs contained irregular shaped and aggregated nuclei (Schut *et al.* 2008). However, Bakst *et al.* (1998) observed well-organized area pellucida, area opaca and a distinct periblastic ring in six out of the ten fresh unfertilized turkey blastoderms with parthenogenetic development.

### During incubation

During incubation, parthenogens require 2 additional days (Olsen 1965a) and this additional time required is probably utilized to reorganize the unorganized embryonic cells to form a normal blastoderm (Olsen 1975). Cytological studies using unfertilized BSW turkey eggs with parthenogenetic development during the first 4 days of incubation demonstrated varying degrees of transformation of yolk-laden blastomeres into multiple layers of epithelial-type cells. In some instances, thickened sheets of epithelial-type cells were seen to cover the entire yolk surface; however, embryos were formed in only 20% of the unfertilized eggs (Olsen 1975). In fact, this unorganized embryonic development makes it difficult to classify avian parthenogens using the standard stages of embryonic development described by Hamburger and Hamilton (1951). More recently, in BSW turkeys selected for parthenogenesis, parthenogenetic development within the first 24–48 h of incubation appeared as unorganized sheets of cells closely resembling early embryonic mortality in fertilized eggs (Cassar *et al.* 1998a). Also, Schut *et al.* (2008) suggested that the unfertilized zebra finch eggs containing parthenogenetic development were advanced enough to appear as early dead embryos. Further, parthenogenetic embryos from unselected virgin Chinese painted quail after 10 days of incubation, resembled very early dead embryos with unorganized, membranous and undifferentiated cells (Parker & McDaniel 2009). Interestingly, a few of the quail parthenogens exhibited an organized development with an area pellucida, area opaca and distinct periblastic ring similar to fresh unincubated-fertilized eggs. In fact, the average germinal disc size of 10-day incubated parthenogens was 3.7 mm as opposed to 4 mm in fresh unincubated-fertilized eggs. However, the germinal disc of 10-day incubated parthenogens was wider than both fresh and 10-day incubated unfertilized eggs with no development (Fig. 2; Parker & McDaniel 2009).

Recently, in virgin (Santa Rosa *et al.* 2016b) and mated (Santa Rosa *et al.* 2016a) Chinese painted quail genetically selected for parthenogenesis, over incubation, parthenogens were shown to modify the environment within the egg albumen. For example, as parthenogen size increased from 0 through 12 days of incubation, albumen pH was lowered as the parthenogens were actively multiplying and undergoing cellular respiration utilizing albumen O<sub>2</sub> and releasing CO<sub>2</sub>. Similarly, as parthenogen size increased, albumen Ca<sup>2+</sup> concentration also increased, possibly due to ionic release from the egg yolk at the low albumen pH. Also, albumen Na<sup>+</sup> concentration increased perhaps to generate an osmotic gradient for the movement of water from the albumen to the yolk for the formation of subembryonic fluid. Conversely, parthenogens decreased albumen Cl<sup>-</sup> as it was likely passively transported into the blastoderm



**Figure 2** Parthenogenetic development observed in Chinese painted quail and turkeys. (A) 10-day incubated germinal disc of Chinese painted quail exhibiting unorganized, irregular parthenogenetic development; (B) 10-day incubated germinal disc of Chinese painted quail with organized parthenogenetic development exhibiting an area opaca (AO), area pellucida (AP) and periblastic ring (PR); Photo credit: Parker & McDaniel 2009 Parthenogenesis in unfertilized eggs of *Coturnix chinensis*, the Chinese painted quail, and the effect of egg clutch position on embryonic development. *Poultry science* 88(4) 784–790, by permission of Oxford University Press, (C) Parthenogenetic male turkey and (inset) newly hatched turkey poul sired by him (Photo credit: Olsen 1975, USDA ARS).

(Santa Rosa *et al.* 2016b). Likewise, parthenogens lowered albumen proteins as they were mobilized for growth (Santa Rosa *et al.* 2016a,b). Interestingly, the albumen analyses revealed that eggs containing parthenogens were similar to early embryonic mortality in fertilized eggs (Santa Rosa *et al.* 2016a). Previously, based on macroscopic analyses Cassar *et al.* (1998b) and Parker and McDaniel (2009) reported the same in BSW turkeys and Chinese painted quail, respectively. Therefore, it is apparent that avian parthenogens are viable embryos capable of modifying their environment. However, avian parthenogens in most instances lack advanced growth (Olsen 1975, Santa Rosa *et al.* 2016b).

Interestingly, developing turkey parthenogenetic embryos were found to be smaller than normal fertilized embryos. In fact, parthenogenetic embryos, from 9 through 25 days of incubation, had lower body weights and lengths as well as testes, spleen and thyroid weights as compared to normal embryos of the same age, and the differences in development showed a delay of about 3 days throughout the embryonic period (Olsen 1970). This delay in development in turn resulted in delayed hatching and most hatched turkey parthenogen poults were weaker than those from fertilized eggs. Further, most mature parthenogen males have nonfunctional or

underdeveloped testes; and in fact, those with normal testes produced poor quality semen at maturity (Olsen 1973, 1975). However, a few turkey parthenogens have good sperm quality and have successfully sired normal turkey poults (Fig. 2; Cassar *et al.* 1998b, Olsen 1975). However, the majority of hatched turkey parthenogens have a shorter lifespan, average 4 years, as well as slower growth rate as opposed to normal turkeys perhaps due to their high degree of homozygosity resulting in the expression of lethal alleles (Olsen 1973). Interestingly, caenophidian snake parthenogens also exhibit developmental abnormalities often resulting in stillborn offspring or reduced lifespan (Booth & Schuett 2016).

### Limiting factors of parthenogen development

The most common macroscopic expression of embryonic parthenogenetic failures in turkey eggs consists of unorganized, delicate and irregularly shaped embryonic tissues that may be closely aggregated (1 cm diameter) or extended over the entire surface of the yolk (<http://oregonstate.edu/instruct/ans-tparth/index.html>, accessed on 10/31/2017). In Chinese painted quail, the majority of parthenogenetic failures appear as unorganized sheets of epithelial cells confined within a germinal disc of  $\leq 7$  mm in diameter (Parker & McDaniel 2009, Parker *et al.* 2010). Multiple limiting factors could be associated with the termination of the progressive development of parthenogenetic embryos; and probably, it is the cumulative effect of all the limiting factors that impair their development. Again, parthenogens exhibit a highly unorganized type of growth and are delayed in development at lay, and thus, most cannot be revived upon incubation (Olsen 1975). Haney and Olsen (1958) found that 97.4% of the blastodiscs of fresh unfertilized turkey eggs underwent cleavage and that 87.2% of the blastodiscs contained nucleated cells. However, after 9–10 days of incubation, only 37.3% of unfertilized turkey eggs demonstrated some degree of development. In fact, Poole and Olsen (1958) demonstrated that freshly laid eggs from strains of Dark Cornish chickens, which had the highest average incidence of early cleavage, showed the highest incidence of parthenogenesis after incubation. It is possible that cells in the more advanced stages of embryonic development at oviposition adapt easier to standard incubation temperatures, whereas cells in early embryonic development at lay are less likely to survive these conditions because standard incubation temperature (37.5°C) is less than the hen's body temperature (41°C) at which early embryonic development occurs. Interestingly, Parker and McDaniel (2009) reported that in virgin Chinese painted quail, the first egg in a clutch sequence is 2 times more likely to exhibit parthenogenesis compared to subsequent eggs in the clutch sequence. In fact, the egg destined to be first in a clutch stays in the hen's body 16 h longer than subsequent eggs in the clutch (Warren & Scott 1935).

The additional time that the first egg remains in the hen's body could allow delayed parthenogenetic embryos a greater opportunity for embryonic development due to increased length of exposure to the hen's body temperature. In addition, Cassar *et al.* (1998b) demonstrated that in BSW turkeys, parthenogens with small populations of haploid cells at lay are the ones that are likely to continue development and hatch, because those that survive beyond day 10 of incubation are predominantly diploid.

Upon incubation, the unfavorable conditions developing within the unfertilized egg may further prevent the progressive development of parthenogenetic embryos. Santa Rosa *et al.* (2016b) demonstrated that Chinese painted quail eggs exhibiting parthenogenetic development have a low albumen pH from the day of lay throughout 12 days of incubation, whereas fertilized eggs at lay have a low pH, then on the second day of incubation pH increases before a decline on the third day. Again, this low albumen pH appears to be due to a high CO<sub>2</sub> concentration. Also, eggs with parthenogenetic development are heavier at set and lose less weight (water) over incubation as opposed to eggs with no development (Wells *et al.* 2012, Santa Rosa *et al.* 2016b). Therefore, Santa Rosa *et al.* (2016b) hypothesized that the lack of moisture loss in these eggs may increase hydration of the eggshell membrane leading to the closure of the eggshell pores, hence blocking gas exchange and leading to the accumulation of CO<sub>2</sub> produced by the parthenogen, thus lowering albumen pH. In addition, parthenogenetic eggs in the early blastula stage of development have high albumen Ca<sup>2+</sup> and Na<sup>+</sup> concentrations. These high ion concentrations along with the acidification of the egg albumen may adversely affect mitotic division during early embryonic development leading to cessation of the cell cycle and subsequent cellular death (Santa Rosa *et al.* 2016b). Therefore, the abortive nature of the vast majority of avian parthenogens is possibly because of the unfavorable conditions developing within the egg albumen in addition to their unorganized and delayed development. However, some internal or external cues may provide an organizing effect for avian parthenogenetic embryos, resulting in hatching parthenogens.

### Factors influencing parthenogenesis

Parthenogenesis in birds is facultative, but there is no clear evidence as to what factors may have contributed to the transition from a complete sexual form to the parthenogenetic form of reproduction. However, it is believed that selective adaptation to adverse environmental conditions may be responsible for this transition in avian species. Some of the environmental contributing factors suggested include: quality and availability of food, changes in day length and temperature, viral and bacterial infections and hormonal



changes (Olsen 1975). Even in various mammalian species, the spontaneous activation of ovarian oocytes resulting in parthenogenetic development and subsequent appearance of ovarian teratomas or teratocarcinomas has been described and is postulated to be due to hormonal changes (Whittingham 1980). In fact, ovarian teratomas are the most common ovarian neoplasms in young women and are usually benign in nature (Crum 1999).

On the other hand, parthenogenesis can be artificially induced in both vertebrates and invertebrates by a variety of physical and chemical stimuli. For example, thermal activation can induce parthenogenetic development in the unfertilized eggs of the silkworm (Astaurov 1967). More recently, phospholipase C $\zeta$  was shown to stimulate parthenogenetic development in human oocytes to yield embryos as far as the blastocyst stage (Rogers *et al.* 2004). The various factors influencing naturally occurring parthenogenesis are discussed in detail below.

## Genetic factors

### *Genetic selection for parthenogenesis*

Avian parthenogenesis is heritable as the incidence of parthenogenesis can be increased by genetic selection for the trait (Olsen *et al.* 1968, Olsen 1975, Parker *et al.* 2010). In BSW turkeys, selective breeding increased the incidence of parthenogenesis almost threefold in five generations, from 16.7% to 41.5%. Interestingly, there was an increase in parthenogen size as generation of selection increased. In fact, there was also an increase in the relative number of advanced parthenogenetic embryos from 0.2% to 11.7% (Olsen 1965*b*). In Pozo Gray turkeys, genetic selection for five generations increased the levels of macroscopically detectable parthenogenesis from 1.1% to 18.6% (Olsen & Buss 1967). Similar results were observed in Dark Cornish chickens where intense genetic selection for the parthenogenetic trait increased the incidence of parthenogenesis as well as parthenogen size (Sarvella 1973). Furthermore, genetic selection also increased the number of Chinese painted quail eggs exhibiting parthenogenesis (Parker *et al.* 2010). In fact, the incidence, in terms of percentage of hens exhibiting parthenogenesis, was almost doubled in four generations, from 36.5% to 68.1%. Also, genetic selection increased the percentage of eggs containing parthenogenetic development 3-fold by the fourth generation of selection. In fact, this increase in the incidence of parthenogenesis appears to be due to a decrease in clutch size with genetic selection for parthenogenesis (Parker *et al.* 2010). As a result of a shortened clutch sequence, more first eggs in a clutch sequence with a higher tendency to exhibit parthenogenesis were produced (Parker & McDaniel 2009). Moreover, as the generation of selection increased, quail parthenogen size also increased and was almost 65% greater in the second generation as

opposed to the parent generation (Parker *et al.* 2010). However, as the generation of selection increased, the differences in hens exhibiting parthenogenesis between generations became less. For example, in quail, Parker *et al.* (2010) revealed that the increase in incidence of parthenogenesis was 10.9 percentage points between the parent generation and first generation, whereas the increase was only 3.7 percentage points between the third and fourth generations. Likewise, Olsen (1975) also revealed that in BSW turkeys, the difference in incidence of parthenogenesis between a random population and the first generation was 9 percentage points, whereas between generations three and four, the difference was only 4.2 percentage points.

Olsen *et al.* (1968) hypothesized that parthenogenesis in chickens and turkeys is controlled by a single locus, autosomal recessive gene. When birds from high and low parthenogenetic lines were crossed, the average incidence of parthenogenesis in the virgin progenies was intermediate with respect to the two parental strains. Further, F2 and back cross methods were used to test the hypothesis in chickens (Olsen *et al.* 1968, Olsen 1975). Moreover, Schom *et al.* (1982) calculated heritability of 0.24 for the parthenogenetic trait in Broad Breasted White turkeys.

### *Genetic strains and varieties*

Previous studies show that the occurrence of parthenogenesis varies with breeds, strains, lines and types of matings. For example, Olsen (1966*b*) studied the incidence of parthenogenesis in 10-day incubated unfertilized eggs from different varieties of chickens. In this study, Araucanas, New Hampshire and Game chickens showed no predisposition to parthenogenesis. White Leghorns, Barred Plymouth Rocks and Rhode Island Reds showed a very low incidence of parthenogenesis. However, Dark Cornish hens showed a high incidence, with the Beltsville strain of Dark Cornish having the highest incidence followed by the Silver Cornish. In fact, of 1143 unfertilized eggs classified as exhibiting parthenogenetic development, 1136 were from the Cornish hens. In contrast to chickens, all the nine different varieties of turkeys tested for parthenogenesis by Olsen and Marsden (1968) showed some degree of parthenogenesis, ranging from 3.6 to 22.4%. Hence, turkey eggs were more predisposed to parthenogenetic development as opposed to chicken eggs (Olsen 1975).

Interestingly, in turkeys, the type of mating, inbreeding or outbreeding, influences the virgin progenies incidence of parthenogenesis. In fact, in BSW turkeys, the incidence of parthenogenesis as well as the percentage of macroscopically recognizable embryos were highest for unfertilized eggs laid by daughters of inbred sires and outbred dams as compared to daughters of inbred sires and inbred dams (Olsen 1972). On the other hand, Savage and Harper (1986) demonstrated



that in turkeys, the selection for low semen volume increased the incidence of parthenogenesis, probably as a means of genome survival. Of the two lines of Medium White turkeys selected for low and high semen volume for ten consecutive generations, the virgin birds from the line selected for low semen volume had a significantly higher incidence of parthenogenesis for the two generations studied. On the other hand, natural parthenogenesis has been described in only one species of quail, *Coturnix chinensis* (Parker & McDaniel 2009), and finches, *Taeniopygia guttata* (Schut *et al.* 2008).

## Age

The overall incidence of parthenogenesis and parthenogenetic embryos in young virgin turkey hens is higher than that in older hens. Also, young hens have more parthenogenetic embryos during the first laying season as opposed to their second laying season (Olsen 1967b). However, laying season itself was found to have no significant effect on the incidence of parthenogenesis (Olsen 1968). In addition, Sarvella and Gehman (1975) reported that the incidence of parthenogenesis was greater in double-yolked eggs as opposed to single-yolked eggs. In fact, double-yolked eggs are common among young commercial layers and broiler breeder hens during the first three months of lay (Jaap & Muir 1968, Harms & Abdallah 1995). Also, young hens often have erratic clutches resulting in smaller clutches and more first eggs of the clutch sequence (Robinson *et al.* 1990). As the first egg of a clutch sequence has a higher tendency to exhibit parthenogenesis (Parker & McDaniel 2009), it is possible that clutch position has an effect on parthenogenesis exhibited in young hens. On the other hand, as the hen approaches peak egg production, the number of eggs in a clutch sequence increases, and, as a result, the incidence of parthenogenesis declines (Parker & McDaniel 2009).

## Environmental factors

### Temperature

In mammals, low temperature storage of unfertilized eggs has been shown to induce parthenogenetic development (Austin 1956). However, in birds, high egg storage or incubational temperatures were shown to increase parthenogenetic development (Schom *et al.* 1982, Santa Rosa *et al.* 2015). Schom *et al.* (1982) reported that storing turkey eggs at a higher temperature (17°C) prior to incubation resulted in a 12.1% incidence of parthenogenesis whereas storage at 6°C yielded only 3.2%. Likewise, Santa Rosa *et al.* (2015) found that elevated storage temperature (40°C) increased parthenogen size in Chinese painted quail eggs. Also, elevated incubation temperature (42°C) following storage at 20°C for 0–3 days increased the incidence of

parthenogenesis yet lowered albumen pH possibly due to more CO<sub>2</sub> production by the parthenogens. However, a combination of higher temperature during storage (40°C) and incubation (42°C) inhibited parthenogenetic development. On the other hand, egg storage temperature (20, 30 and 40°C) was found to have no effect on the incidence of parthenogenesis when eggs were incubated at standard temperature (Santa Rosa *et al.* 2015). Similarly, Sarvella (1974) observed no differences for the incidence of parthenogenesis due to storage temperature in chicken eggs. Eggs incubated within an hour of oviposition showed no differences in parthenogenetic development as compared to eggs stored at 12.8°C for 3 days.

## Viruses

Varieties of live poultry viruses, following either natural infection or vaccination, are known to enhance parthenogenetic development in chickens and turkeys. For example, in Dark Cornish chicken eggs following vaccination of hens with live fowl pox virus, the incidence of parthenogenesis was enhanced more than ninefold as opposed to eggs laid before vaccination. In fact, the incidence was highest in the eggs laid 30–60 days after vaccination (Olsen 1956). Likewise in turkeys, live fowl pox virus vaccination was observed to increase the incidence of parthenogenesis as well as embryo size (Olsen 1956, Olsen & Poole 1962, Olsen & Buss 1967). Additionally, twin, triplet and quadruplet parthenogenetic embryos were found in the unfertilized eggs laid by unselected BSW turkeys recently vaccinated (Olsen 1962b). Vaccination of parent stock was found to even increase the number of parthenogenetic embryos observed in the eggs of non-vaccinated progenies as compared to progenies of non-vaccinated parents, but the level of parthenogenesis decreased as the generations without fowl pox vaccination increased (Olsen 1975). In contrast, Sarvella and Gehman (1975) reported that live fowl pox virus had no stimulating effect on parthenogenetic development following *in ovo* injection into chicken eggs.

Unlike fowl pox virus, a DNA virus, RNA viruses have been found to affect only the incidence of parthenogenesis but not on the size of the embryos. For instance, leukosis virus following a natural outbreak of visceral lymphomatosis in white leghorn chickens increased the incidence of parthenogenesis. As observed with fowl pox virus, the predisposition toward parthenogenesis was transmitted from the infected hens to the unfertilized progenies, but only 4 of 17 hens acquired the potential to exhibit parthenogenesis (Olsen 1966c). Likewise, live Rous sarcoma virus (Olsen 1961) and live Newcastle disease virus (Olsen 1975) following vaccination in turkeys increased the number of unfertilized eggs exhibiting parthenogenetic development.

On the other hand, [Olsen \(1962c\)](#) demonstrated that killed virus vaccines, Newcastle disease, fowl pox and Rous sarcoma, had no effect on the incidence of macroscopically detectable parthenogenesis in the unfertilized eggs laid by vaccinated unselected BSW turkeys. Also, other components in live virus vaccines, such as tissue and blood, were found to have no effect, thus indicating that only the live viruses were causative agents in altering parthenogenesis. However, *in ovo* administration of killed fowl pox virus vaccine into chicken eggs also had no stimulating effect on parthenogenesis ([Sarvella & Gehman 1975](#)).

Even though the mechanism of action of viruses on parthenogenesis is not clearly understood, two possible modes of action can be postulated. First, the virus may have a direct effect on the embryo. In fact, the direct action of the virus can cause fusion of the cells ([Huang et al. 1981](#)), which may induce parthenogenetic development or may have an organizing effect in the eggs where parthenogenetic development has already been initiated resulting in more advanced embryonic development. Second, the virus may have an indirect effect on the virgin hen that then effects parthenogenetic development. The virus may act as a stress factor and reduce egg production ([Leslie 2000](#)), which in turn results in shorter clutch lengths and more first-of-sequence eggs, thus resulting in a higher incidence of parthenogenesis ([Parker & McDaniel 2009](#)). Previously, it was hypothesized that DNA and RNA viruses act differently; and the DNA virus, fowl pox, is probably a potent organizer as well as a stimulant to cellular proliferation resulting in more developed embryos. However, both DNA and RNA viruses were suggested to have the potential to alter the bird's genetic material after gaining entrance into the ova where viral DNA could alter meiotic cell divisions such that parthenogenetic development is initiated in the ova. Further, the altered genetic material is transferred to the future generations such that they acquire a strong predisposition to parthenogenesis ([Olsen & Buss 1967](#), [Olsen 1975](#)). However, additional research on the mechanism of action of viruses on avian parthenogenesis is needed to make accurate conclusions.

### Potential impact of parthenogenesis on the poultry industry

Recently, [Parker et al. \(2010\)](#) reported that genetic selection for parthenogenesis in Chinese painted quail decreased egg production and clutch length. In fact, clutch length appeared to affect both the incidence of parthenogenesis and egg production. As the incidence of parthenogenesis was greatest for the first eggs in the clutch sequence ([Parker & McDaniel 2009](#)), a decrease in egg production due to shorter clutch sequences increased the number of first eggs in the clutch sequence and thus increased the percentage of

eggs exhibiting parthenogenetic development ([Parker et al. 2010](#)). A similar negative correlation between parthenogenetic development and egg production was observed by [Schom et al. \(1982\)](#) in Broad Breasted White turkey hens. A reduction in egg production would result in huge economic losses to the poultry industry. As parthenogenesis exist in the modern poultry industry ([Bakst et al. 2016](#)), it may negatively affect egg production resulting in economic losses. Therefore, a better understanding of parthenogenesis could lead to economic gains in egg production.

As parthenogenesis impairs hatchability and fertility ([Schom et al. 1982](#), [Parker et al. 2012, 2014](#), [Santa Rosa et al. 2016a](#)), it could potentially directly impact these parameters in the commercial poultry industry. In fact, both males and females selected for the parthenogenetic trait were found to negatively impact reproductive performance. For instance, parthenogenetic line females lay heavier eggs and these eggs lose less weight during incubation as opposed to unselected control line females. Both parthenogenetic line dams and sires exhibited a lower hatchability and albumen pH as well as a higher incidence of parthenogenesis vs control parents. Further, parthenogenetic line males were responsible for the reduced fertility by lowering the number of sperm penetrating the egg ([Parker et al. 2017](#)). Previously among chickens, the Cornish breeds were found to exhibit the highest incidence of parthenogenesis ([Olsen 1966b](#)). Because of its breast conformation, the Cornish chicken was used extensively to develop the modern day broiler breeder chicken ([Delany 2003](#)). Therefore, it is possible that the parthenogenetic trait in the Cornish chicken has been inherited by the modern day broiler breeder. Hence, it is hypothesized that fertility and hatchability in the commercial broiler industry are currently being negatively impacted by the presence of parthenogenesis. However, additional research on parthenogenesis and its impact on the reproductive performance of commercial broilers are required to test this hypothesis.

Additionally, it has been shown that parthenogens resemble early embryonic mortality in fertilized eggs ([Olsen & Buss 1972](#), [Cassar et al. 1998a](#), [Parker & McDaniel 2009](#), [Santa Rosa et al. 2016a](#)). For example, [Santa Rosa et al. \(2016a\)](#) reported that in mated Chinese painted quail, parthenogens alter albumen characteristics similar to early dead embryos from fertilized eggs. Eggs exhibiting parthenogenetic development had a lower albumen pH, protein and O<sub>2</sub> concentrations, yet higher CO<sub>2</sub> concentration as opposed to infertile eggs but they were similar to eggs with early embryonic mortality. Interestingly, in order to distinguish parthenogens from early dead embryos that developed from fertilized eggs in mated turkeys, genotypic sexing was used by [Cassar et al. \(1998a\)](#), and it was concluded that at least some early dead embryos without W chromosomes could in fact be parthenogens. In the poultry industry,

hatch residue analysis is vital to determine fertility and hatching failures of each flock (Wilson 1994). Because most avian parthenogens resemble early embryonic mortality during hatch residue analysis, it is possible that the percentage of early dead embryos reported in the industry as well as the percentage of fertilized eggs may be inflated because these parthenogenetic eggs are actually a product of unfertilized eggs that fail to develop properly. This in turn warrants additional research to identify the genetic makeup of early dead embryos using genotyping techniques, like microsatellite DNA fingerprinting and amplified fragment-length polymorphism (AFLP), to confirm parthenogenetic development. In fact, genotyping is successfully used for accurate identification of parthenogens in other vertebrates, like sharks and snakes (Chapman *et al.* 2007, Booth *et al.* 2014).

Finally, vaccination is a routine practice in the modern poultry industry, especially live pox and Newcastle disease vaccinations. However, these live viruses are known to enhance parthenogenetic development in poultry (Olsen 1956, 1962b, Olsen & Buss 1967). Also, the effect of these viruses on parthenogenesis is transferred from vaccinated parent generations to non-vaccinated progenies (Olsen & Buss 1967). Eventhough there is no information on the effect of current virus vaccine strains on parthenogenesis in poultry, it is possible that the current virus strains affect the birds similarly to that observed in the 1960s.

## Conclusion

Parthenogenesis is a naturally occurring spontaneous phenomenon in birds. Even though most avian parthenogens are abortive in nature and the mechanism to restore diploidy is not fully understood, it is proven that all avian parthenogens are homogametic males with mostly diploid chromosomes. The reasons for the transition from a complete sexual form to a parthenogenetic form of reproduction is still debatable, but certain environmental cues, in addition to epigenetic cues, can trigger the process of parthenogenesis. Moreover, the process of parthenogenesis has been witnessed not only in virgin birds but also in mated birds and appears to interfere with the mechanisms of normal fertilization and embryonic development in fertilized eggs. Therefore, parthenogenesis may currently be adversely affecting the poultry industry. Hence, it is imperative to have a better understanding of avian parthenogenesis in order to improve the reproductive performance of commercial poultry and to prevent economic losses. With recent advances in molecular techniques, future research on avian parthenogenesis should focus on the proximate mechanisms employed by parthenogens to restore diploidy as well as on the reasons for the delayed and abortive development of parthenogens by

studying the differential expression of genes and other epigenetic abnormalities, like erroneous chromatin remodeling. Using DNA-based genotyping methods, accurate identification of parthenogens from early dead embryos should be conducted to accurately calculate the fertility and hatchability loss in the poultry industry due to parthenogenesis. Another key area of future research could be investigating the role of modern virus vaccine strains on parthenogenesis, including their mechanisms of action. Also, even though in birds parthenogenesis is phylogenetically widespread, most of the studies are restricted to galliformes (chickens, turkeys and Chinese painted quail); hence, there is a need for further studies on other avian species in order to establish the evolutionary basis of parthenogenesis in birds. In addition, these future researches on avian parthenogenesis may enhance our knowledge of parthenogenesis in higher vertebrates, like mammals, and may have a direct implication on our understanding of reproductive disorders, such as ovarian teratomas as well as the production of human stem cells.

## Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this review.

## Funding

This publication is a contribution of the Mississippi Agricultural and Forestry Experiment Station and this material is based upon work that is supported by the National Institute of Food and Agriculture, U.S. Department of Agriculture, Hatch project under accession number MIS-329200.

## Acknowledgements

The authors would like to thank Dr Holly Parker, Midian Nascimento dos Santos, and all of the undergraduate and graduate students for their assistance with this project.

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Received 29 November 2017

First decision 18 January 2018

Revised manuscript received 15 February 2018

Accepted 19 March 2018