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Avian diseases which affect egg production and quality

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Abstract: This chapter addresses diseases and syndromes which have been shown, or are reported, to have adverse effects on egg production and quality. However, any disease of poultry can adversely affect egg production and quality indirectly, by affecting the health of the bird. The main topics are the effect on egg production and quality of bacteria (*Salmonella*, *Mycoplasma*, *Escherichia coli*, infectious coryza, *Ornithobacterium*, *Gallibacterium*, spirochaetosis), viruses (infectious bronchitis virus, egg drop syndrome, swollen head syndrome, avian encephalomyelitis, influenza, Newcastle disease, laryngotracheitis), syndromes (fatty liver haemorrhagic syndrome, cage layer osteoporosis) and toxic agents. A short section on clinical perspectives reports on information obtained from practising poultry veterinarians as such observations can provide valuable starting points for future research.

Key words: egg quality, avian disease, virus, bacteria.

16.1 Introduction

Any disease of poultry can adversely affect egg production and quality either directly, by having effects on the reproductive system, or indirectly, by affecting the health of the bird. Respiratory infections which result in air sacculitis may, in turn, infect the ovary and oviduct. In addition, some diseases infect the oviduct and ovary by ascending infection. It is frequently difficult to diagnose the cause or causes of reduced egg quality because it is often a combination of factors that leads to poor egg quality. Management,

nutrition and disease may, in combination, result in a reduction in egg internal quality and/or egg shell quality. The most recent comprehensive review of diseases affecting egg quality is that of Spackman (1987) who, although focusing on egg shell quality, also discussed the effects of disease on egg internal quality. This chapter addresses diseases and syndromes which have been shown, or are reported to, have adverse effects on egg production and quality. Emphasis will be on developments since Spackman's 1987 review, except where earlier information is fundamental to the discussion. The main experimental developments that have occurred since Spackman's review are new information about the effect of Australian strains of infectious bronchitis virus on the oviduct and egg quality and the reporting of egg apex abnormalities caused by *Mycoplasma synoviae* (particularly in combination with infectious bronchitis virus).

16.2 Effects of bacteria on egg production and quality

16.2.1 *Salmonella*

Aetiology

The genus *Salmonella* (family Enterobacteriaceae) consists of more than 2500 serologically distinguishable variants and can be divided into non-motile and motile serotypes (Gast, 2008). All salmonellae that are associated with poultry are members of a single genetically defined species, *Salmonella enterica*.

Epidemiology

The non-motile serotypes, *Salmonella enterica* serovar Gallinarum (fowl typhoid) and *Salmonella enterica* serovar Pullorum (pullorum disease), can cause septicaemic disease in poultry. The motile serotypes are often referred to as paratyphoid (PT) salmonellae and include *S. enterica* serotype Enteritidis and *S. enterica* serotype Typhimurium (usually abbreviated as *S. Enteritidis* and *S. Typhimurium*). The paratyphoid salmonellae are often asymptomatic in avian species, but can cause food-borne illness in humans (Gast, 2008). Age and genetic strain influence the resistance of birds to avian enteric salmonellosis (Beal *et al.*, 2005; Berchieri *et al.*, 2001).

Clinical signs in layers: egg production and quality

In general, young birds are more susceptible to *Salmonella* infections than mature birds. *S. Gallinarum* and *S. Pullorum* cause septicaemic disease resulting in decreased egg production and hatchability, as well as morbidity and mortality (Shivaprasad, 2000). Transovarian infection can occur (Berchieri *et al.*, 2001). Experimental infection of birds with *Salmonella* Enteritidis increased the incidence of hairline cracks in eggs, leading to an increased risk of contamination (Guard-Bouldin and Buhr, 2006).

16.2.2 Mycoplasma

Aetiology

Mycoplasmas are eubacteria devoid of cell walls and are members of the class Mollicutes, Order I Mycoplasmatales. Genus I, *Mycoplasma*, has more than 100 species of which 25 infect avian species, and 10 of which infect chickens (Kleven, 2008).

Epidemiology

Mycoplasma gallisepticum causes chronic respiratory disease (CRD) in chickens as well as infectious sinusitis in turkeys and often co-occurs with a respiratory virus infection (Ley, 2008). Oviduct infection can occur owing to proximity to infected air sacs (Nunoya *et al.*, 1997). *M. synoviae* also causes respiratory disease but can become systemic, causing disease primarily in joints and tendon sheaths in chickens and turkeys (Kleven, 2008). *M. gallisepticum* and *M. synoviae* also infect a range of other avian species.

Clinical signs in layers: egg production and quality

Spackman (1987) states 'The role of Mycoplasmata in egg production and quality is somewhat controversial'. At that time, it was not clear to what extent *Mycoplasma synoviae* (MS) or *M. gallisepticum* (MG), alone, were involved in abnormal eggshells and production losses and what was caused by interactions with other infectious agents such as infectious bronchitis virus (Giambrone *et al.*, 1977). MG had been implicated in production losses and, in the case of breeder flocks, decreased hatchability. Until about 2000, the main concern in flocks of laying hens was *Mycoplasma gallisepticum* (Kleven 2008; Kleven *et al.*, 1990; Stipkovits and Kempf, 1996; Yoder 1986) although there were reports of MS in layer flocks (Branton *et al.*, 1989; Mohammed *et al.*, 1986, 1987a,b; Morrow *et al.*, 1990; Opitz, 1983; Reece *et al.*, 1986a). MS was regarded as being a problem predominantly in broiler and turkey flocks and has also been reported from other poultry species such as ducks (Bencina *et al.*, 1988a; Yamada and Matsuo, 1983), geese (Bencina *et al.*, 1988b) and pigeons (Reece *et al.*, 1986b). The production of eggs with abnormal shells has been reported for geese infected with *Mycoplasma* sp strain 1220 (Dobos-Kovacs *et al.*, 2009). The vertical transmission of MS was recognized relatively early (Carnaghan, 1961; MacOwan *et al.*, 1984; Roberts and McDaniel, 1967). Both MG and MS have the potential to cause salpingitis in laying birds (Domermuth *et al.*, 1967). Opitz (1983) reported MS infection on 48% of farms surveyed in Maine, US. Mohammed *et al.* (1986) reported prevalence of MG as 73% in southern California and 3% in central California and MS as 91% in southern California and 32% in central California. An economic analysis indicated that there was no association between MS infection and egg production but that MG resulted in an estimated 127 million eggs lost in southern California (Mohammed *et al.*, 1987b).

A recent study by Feberwee *et al.* (2009a) has shown that MS, isolated from the oviduct of birds producing abnormal eggs, can induce eggshell apex abnormalities (EAA). These abnormalities are characterized by changes in the colour and texture of the shell in the region of the air sac. When this region of the egg shell is observed under the scanning electron microscope, the mammillary layer and the lower part of the palisade layer are missing. These EAAs ceased after an injection of long-acting oxytetracycline but reoccurred 12 days later. The incidence of EAAs was higher in the presence of infectious bronchitis virus. The presence of EAAs was accompanied by shell thinning, increased translucency and reduced shell breaking strength. A later experiment with SPF white layers (Feberwee *et al.*, 2009b) investigated the ability of an MS vaccine to protect against egg shell abnormalities in the presence of an infectious bronchitis challenge. EAAs were produced only in birds that were challenged with *M. synoviae*, whether or not they had been previously vaccinated for MS. Again, this eggshell abnormality was associated with reduced shell strength. Vaccination reduced the incidence and delayed the appearance of EAAs in eggs from birds challenged with MS but did not completely prevent the occurrence of EAAs.

16.2.3 *Escherichia coli*

Aetiology

Escherichia is the type genus of the family Enterobacteriaceae and *E. coli* is the type species of the genus *Escherichia*. *E. coli* causes colibacillosis in most avian species, with younger birds being most susceptible (Barnes *et al.*, 2008).

Epidemiology

E. coli is commonly found in the intestines of poultry and is transmitted to eggs primarily by faecal contamination of the shell surface followed by entry into the egg.

Clinical signs in layers: egg production and quality

Escherichia coli affects production and, potentially egg quality, in laying hens by causing colibacillosis associated with salpingitis (Trampel *et al.*, 2007). Other avian species such as ducks may also be affected (Bisgaard, 1995). Many serotypes of *E. coli* are found in poultry but it is only the avian pathogenic *E. coli* (APEC) which possess specific virulence factors and are capable of causing salpingitis and peritonitis (Landman and Cornelissen, 2006). Salpingitis may be caused by either a systemic infection or by ascending infection of the oviduct from the cloaca. The study of Ozaki and Murase (2009) reported postmortem findings of fibrinous exudates in the vagina, caseous exudates in the upper oviduct, degenerated ovaries and a thickened and oedematous oviduct mucosa. The stress associated with the onset of lay may act as a precipitating factor in colibacillosis outbreaks (Zanella *et al.*,

2000). Virulent *Mycoplasma synoviae* can act as a complicating factor in *E. coli* peritonitis syndrome (Raviv *et al.*, 2007). However, Vandekerchove *et al.* (2004) concluded that colibacillosis outbreaks are not necessarily associated with other respiratory pathogens.

16.2.4 Ornithobacterium

Aetiology

Ornithobacterium rhinotracheale is a Gram-negative, non-motile, pleomorphic, rod-shaped, non-sporulating bacterium (Chin *et al.*, 2008).

Epidemiology

O. rhinotracheale causes respiratory disease in chickens and a wide range of other avian species (van Empel and Hafez, 1999). A high seroprevalence of *O. rhinotracheale* was found in a study in the north central region of the United States (Heeder *et al.*, 2001). The organism is transmitted vertically (van Empel and Hafez, 1999).

Clinical signs in layers: egg production and quality

Clinical signs are respiratory disease and the severity of the disease is worsened when birds have coexisting infections with other respiratory disease agents (Thachil *et al.*, 2009). It can also develop into peritonitis. In commercial laying flocks, *O. rhinotracheale* results in production drops, decreased egg size, misshapen eggs and increased mortality (Sprengrer *et al.*, 2000). Similar symptoms have been reported for flocks of broiler breeders (Chin *et al.*, 2008).

16.2.5 Gallibacterium

Aetiology

The genus *Gallibacterium* is in the family Pasteurellaceae and contains avian bacteria formerly known as *Pasteurella haemolytica*, *Actinobacillus salpingitidis* or *Pasteurella anatis*. It includes the species *G. anatis* and *G. genomospecies* 1 and 2. *G. anatis* comprises two biovars, a haemolytic biovar haemolytica and a non-haemolytic biovar anatis (Barnes *et al.*, 2008; Christensen *et al.*, 2003; Neubauer *et al.*, 2009).

Epidemiology

Gallibacterium spp can be isolated from a wide variety of birds.

Clinical signs in layers: egg production and quality

Gallibacterium anatis, biovar haemolytica, has been suggested as causing peritonitis and salpingitis in chickens (Christensen *et al.*, 2003; Barnes and Nolan, 2008) and other species (Bisgaard, 1995) and has been isolated from laying birds suffering from reproductive disorders (Neubauer *et al.*,

2009). Similar symptoms have been induced experimentally (Bojesen *et al.*, 2004).

16.2.6 Spirochaetosis

Aetiology

Spirochaetes are classified in the Order Spirochaetales which contains three families, Spirochaetaceae, Brachyspiraceae and Leptospiraceae, and a total of nine genera (Hampson and Swayne, 2008). However, spirochaetes which cause avian intestinal spirochaetosis (AIS) are all of the family Brachyspiraceae, genus *Brachyspira*.

Epidemiology

AIS occurs primarily in flocks of layers and broiler breeder hens so is a disease of birds that are producing eggs. A survey conducted in eastern Australia reported that birds in 43% of broiler breeder and 68% of layer flocks were infected with intestinal spirochaetes but no broiler flocks were infected (Stephens and Hampson, 1999). This study identified *Serpulina pilosicoli* (now *B. pilosicoli*) and *S. intermedia* (now *B. intermedia*) among the flocks but could not identify other species found. Colonization can be enhanced by a wheat-based diet (Phillips *et al.*, 2004) and this could not be consistently and significantly reduced by the use of endogenous feed enzymes. A recent paper (Ivanics *et al.*, 2009) reported problems in Hungary in layer flocks.

Clinical signs in layers: egg production and quality

Intestinal spirochaetes colonize the caecum and/or rectum and can cause diarrhoea (Hampson and Swayne, 2008; Ivanics *et al.*, 2009). The disease also results in reduced egg production and hatchability and eggshell quality deteriorates (Ivanics *et al.*, 2009).

16.3 Effects of viruses on egg production and quality

16.3.1 Infectious bronchitis virus

Aetiology

Infectious bronchitis (IB) is a poultry viral disease caused by a coronavirus, an enveloped RNA virus. The coronavirus belongs to the Coronaviridae family. The essential coronavirus characteristic is antigenic plasticity, because of the variable amino acid sequences of spicules on the surface. Many serotypes exist and the most frequent is the Massachusetts strain, and new variants like the 'Qx' strain.

Epidemiology

IB affects *Gallus* of all ages, but is more severe in young poultry. The virus spreads in aerosol and is transmitted by the respiratory route. Droppings and

nasal discharge are the virulent matter. Transmission is horizontal either directly from bird to bird or indirectly via personnel or material (Cavanagh and Gelb, 2008).

Clinical signs in layers: egg production and quality

With both respiratory and genital tropism, IB infection involves respiratory signs in broilers, drops in egg production and deterioration in egg quality in layers. Some strains are also nephropathogenic. Sevoian and Levine observed internal and external egg quality alteration in laying hens experimentally infected with Massachusetts IB strain (Sevoian and Levine, 1957).

In layers, the disease causes drops in egg production, commonly by from 5 to 10%, and by up to 50%. A commercial chicken flock from which IB virus was recovered showed a drop in egg production of 15%. The drop lasted for approximately 4 weeks and production did not return to the pre-infection level (Cook, 1984). The severity of the production drop may vary with the period of lay and with the causative virus strain. An Arkansas strain experimental infection in White Leghorn layers studied the effects of the virus on egg production (Muneer *et al.*, 1986). Infected hens laid fewer eggs and the shell quality and internal quality were inferior. Egg production was 62% in infected birds and 77% in controls 15 days post-infection. IB-infected hens laid eggs with a watery albumen consistency and yolk size was smaller.

Chousalkar and Roberts studied the effect of Australian IB strains on egg production and egg quality. The classic 'IB egg', an egg that is wrinkled and corrugated, as described by Dhinakar Raj and Jones (1997), was not observed. Although there was no decline in egg production, there was a deterioration of egg internal quality (albumen) and a loss of egg shell colour (Chousalkar and Roberts, 2009). In addition, egg shape changed, with IB causing eggs to become more elongate. Studies of the histopathology of the oviduct of White Leghorn (Chousalkar *et al.*, 2007a), Hy-Line Gray (Chousalkar *et al.*, 2007b) and Isa Brown hens (Chousalkar *et al.*, 2009a) as well as ultrastructural investigations (Chousalkar and Roberts, 2007a,b; Chousalkar *et al.*, 2009a) confirmed that IB induces pathology in various regions of the oviduct of laying hens. Virus was also isolated from the oviduct of previously challenged laying hens (Chousalkar *et al.*, 2009b).

Coronavirus infection in the shell gland cells causes declines in egg shell quality: thin, soft, misshapen or pale unpigmented shells. The thin and watery albumen occurs when the coronavirus affects the cells of the magnum. Haugh unit values are reduced. A day-old coronavirus infection can also create false layers. Layers are then unable to lay due to the oviduct damage. This situation has been described in France in breeders and layers associated with a new variant strain, Qx (Robineau and Moalic, 2009).

16.3.2 Egg drop syndrome (EDS)

Aetiology

Egg drop syndrome (EDS) virus is caused by duck adenovirus A and was first described in laying hens in 1976. The adenovirus is a member of the genus *Atadenovirus* and family *Adenoviridae*.

Epidemiology

Egg drop syndrome affects chickens and quail. Ducks and geese seem to be natural hosts of the virus. The virus is transmitted vertically by eggs. Droppings also contain the virus that can be horizontally transmitted by the oral route. Infected wild birds can be a contamination source for poultry (Adair and Smyth, 2008).

Clinical signs in layers: egg production and quality

The disease is characterized by drops in egg production and increased incidence of abnormal eggs. Birds remain generally healthy. Egg production is usually reduced by from 10% to 40%. The fall in production can be rapid, and the drop in egg production usually lasts from 4 to 10 weeks (McFerran and Smyth, 2000). The first sign is loss of shell pigments. This loss of colour is followed by thin, soft, rough and granular shell and shell-less eggs. Higashihara and coworkers observed few clinical signs except production of abnormal eggs in hens infected orally with EDS virus (Higashihara *et al.*, 1987). Egg production was about 20% lower and aberrant eggs were shell-less, soft-shelled, thin shelled and pale coloured. In this study, internal quality was not altered. In EDS 76 field cases reported by Van Eck, watery and thin albumen was reported (Van Eck *et al.*, 1976).

16.3.3 Swollen head syndrome

Aetiology

Swollen head syndrome is an infectious disease caused by an avian pneumovirus from the Paramyxoviridae family. The virus is enveloped. It is a single-stranded, RNA virus 80–200 nm.

Epidemiology

The swollen head syndrome affects chickens and guinea fowl. Respiratory signs occur in young birds and the adults are affected by drops in egg production. The transmission of the virus is lateral by aerosol through the respiratory route. It is spread by both airborne and mechanical (feed, water and equipment) routes (Gough and Jones, 2008).

Clinical signs in layers: egg production and quality

Respiratory signs occur in young birds and adults are affected by drops in egg production usually by from 5% to 30%. The disease lasts from 2 to 3 weeks. An avian pneumovirus infection study using laying hens was conducted

by Cook. Intravenous inoculation caused a drop in egg production of up to 25% and a high incidence of soft and thin-shelled eggs. Some respiratory signs were also observed (Cook *et al.*, 2000).

16.3.4 Avian encephalomyelitis

Aetiology

Avian encephalomyelitis is a poultry viral disease caused by a picornavirus from the Picornaviridae family. It is an RNA virus, not wrapped and icosahedric.

Epidemiology

Avian encephalomyelitis affects chickens, pheasants, quails and turkeys. The disease occurs in young birds, usually less than three weeks of age and also in layers. Vertically transmission in eggs is the main route of contamination. The virus is also excreted in faeces by the horizontal route from bird to bird (Calnek, 2008).

Clinical signs in layers: egg production and quality

The disease is characterized by neurological signs in young birds with uncoordination, ataxia and tremors. Affected laying hens show a temporary drop in egg production (5–10%), but not neurological signs. Meroz reported a severe drop in egg production of from 75% to 51% associated with encephalomyelitis in a commercial laying flock aged 43 weeks (Meroz *et al.*, 1990). Production returned to its previous level within two weeks. No clinical signs and no abnormal eggs were observed in the flock.

16.3.5 Influenza

Aetiology

Avian influenza is a viral infection caused by influenza A virus, which is a member of the family Orthomyxoviridae. These are RNA viruses classed in three types A, B and C. Influenza A viruses are isolated in birds, swine, horse and human. Types C and D are isolated in humans. The glycoproteins haemagglutinin (H) and neuraminidase (N) characterize the virus subtypes. 16 H antigens and 9N antigens have been described by Swayne and Halvorson (2008).

Epidemiology

Influenza virus has been recovered from domestic and wild avian species all over the world. Migratory waterfowl are a wild reservoir of influenza virus. Turkeys and chickens are highly susceptible. Avian influenza virus transmission is horizontal and includes direct and indirect contact (Swayne and Halvorson, 2008).

Clinical signs in layers: egg production and quality

Clinical signs vary depending on species infected, bird age and the virulence of the viral subtype. Mortality of up to 90%, without any clinical signs of illness, can be observed in a highly pathogenic influenza outbreak. In a low pathogenic outbreak, some birds may also develop general symptoms with cough, diarrhoea, depression, anorexia and drops in egg production. In the United States, influenza A virus was isolated from commercial laying hens 55 weeks old with up to 69% mortality and severe decrease in egg production. Egg production dropped from as high as 80% to 13% (Johnson and Maxfield, 1976). With low pathogenic influenza A virus, egg drop production is less severe. In 1997 and 1998, in Pennsylvania, H7N2 (nonpathogenic) influenza A virus was diagnosed in commercial leghorn laying hens (Ziegler *et al.*, 1999). Mortality was less than 4% and egg production declined by from 2% to 4%.

16.3.6 Newcastle disease*Aetiology*

Newcastle disease is an infectious disease affecting domestic and wild birds and is caused by a paramyxovirus, of the family Paramyxoviridae and genus *Rubulavirus*. The paramyxoviruses are RNA viruses. They are enveloped with two glycoproteins haemagglutinin-neuraminidase (HN) and glycoproteins F, for virus attachment and fusion.

Epidemiology

Newcastle disease affects all poultry species, but the pathogenicity varies with host. Chickens are highly susceptible, but ducks and geese are less susceptible. The virus spreads through horizontal transmission, in bird secretions (nasal discharge) and droppings. Birds are contaminated by inhalation or ingestion directly from bird to bird or indirectly by mechanical means (personal, material). Vertical transmission is controversial. The virus can survive for several weeks in the environment in contaminated manure or material (Alexander and Senne, 2008).

Clinical signs in layers: egg production and quality

Clinical signs are variable depending on host species and virus strain. Newcastle virus is classed into pathotypes velogenic, mesogenic and lentogenic. The viscerotropic velogenic strains cause high mortality and enteric lesions. Neurotropic velogenic strains cause also high mortality with respiratory and nervous signs. With mesogenic strains, clinical signs are respiratory and nervous with low mortality level. Lentogenic stains cause respiratory disorders particularly in young birds. In adult birds, egg production is affected by mesogenic strains. Layers are depressed and anorexic. A partial to complete drop in egg production is observed. Egg quality is affected with thin-shelled eggs.

16.3.7 Laryngotracheitis

Aetiology

Laryngotracheitis (LT), also known as infectious laryngotracheitis (ILT), is caused by a member of the family Herpesviridae, subfamily Alphaherpesviridae, genus *Iltovirus*. It is taxonomically identified as *Gallid herpesvirus* (Guy and Garcia, 2008).

Epidemiology

Laryngotracheitis is a respiratory disease of chickens that can result in production losses due to mortality, morbidity and decreased egg production (Jordan, 1966; Bagust *et al.*, 1986; Guy and Garcia, 2008). The chicken is the primary natural host and, although the disease may affect chickens of all ages, it is mostly observed in adult birds. However, the onset of lay can affect the rate of shedding of carrier birds (Hughes *et al.*, 1989).

Clinical signs in layers: egg production and quality

Clinical signs involve primarily the respiratory system with nasal discharge, respiratory depression, mucoid tracheitis, sinusitis, conjunctivitis, gasping and expectoration of blood mucus. Decreased egg production and failure to thrive appear to be secondary to the effects on other body systems. There is no direct evidence for effects of ILT on egg quality.

16.4 Effects of syndromes on egg production and quality

16.4.1 Fatty liver haemorrhagic syndrome (FLHS)

Fatty liver haemorrhagic syndrome (FLHS) is a metabolic disease affecting caged hens in high production. It is characterized by fat infiltration into the liver, haemorrhage and mortality. The disease is associated with a high energy ration and probably induced by mycotoxins, hot weather and dietary lipids. High levels of plasma oestradiol also increase the FLHS risk (Haghighi-Rad and Polin, 1981). Clinical signs are essentially mortality in full production and sudden drops in egg production. The disease can cause up to 5% mortality during the laying cycle (Julian, 2005). The liver is friable, enlarged, yellow, engorged with fat and haemorrhagic. Restricted feeding may prevent the disease. Fatty liver and hepatic steatosis is frequently confused with fatty liver syndrome. Hepatic steatosis is associated with a low protein and high energy level in the ration and causes little mortality and egg production drops (Julian, 2005).

16.4.2 Cage layer osteoporosis

Osteoporosis is a metabolic disorder affecting bone structure and reduction in bone mass. The bones are more fragile and susceptible to fractures. The disease may occur with calcium (Ca) depletion. This may occur because

of inadequate dietary Ca, vitamin D3 and phosphorus (P) (Julian, 2005). If Ca is insufficient in the diet, in order to produce eggs, hens withdraw the mineral from cortical bone. Phosphorus deficiency causes osteoporosis, often called cage layer fatigue. Phosphorus is essential to the medullary bone structure. Vitamin D3 deficiency results also in osteoporosis by affecting Ca metabolism. Adequate nutrition helps to reduce this disorder. More exercise results also in better bone quality but may not decrease the fracture incidence. Lines resistant to osteoporosis have been selected (Whitehead and Fleming, 2000).

16.5 Effects of toxic agents on production and egg quality

Various toxic agents and contaminants can produce disease in poultry. Contamination of feed with mycotoxins has the potential to reduce production and egg quality (Garaleviciene *et al.*, 2001; Chowdhury and Smith, 2004, 2005; Chowdhury *et al.*, 2005; Pandey and Chauhan, 2007). Chowdhury and coworkers showed that mycotoxins result in increased plasma uric acid concentrations, most likely due to effects on the liver, and also cause immunosuppression. Some of these effects are mediated via a reduction in feed intake of the contaminated feed (Suksupath *et al.*, 1989). Egg production in broiler breeders has been shown to be reduced by a mycotoxin but only at relatively high levels (Brake *et al.*, 2002).

Ingestion of crude oil resulted in lower Haugh units in poultry (Ekweozor *et al.*, 2002). Vanadium has also been shown to reduce albumen quality by reducing the amount of crude ovomucin per millilitre of thick egg albumen (Toussant and Latshaw, 1999). The ovomucin content of the thin albumen was not affected by vanadium supplementation of the diet.

16.6 Clinical perspectives

Opinions were sought from poultry veterinarians via organizations such as the American Association of Avian Pathologists, the World Veterinary Poultry Association and the Australian Veterinary Poultry Association. A number of poultry veterinarians offered an opinion from their own clinical experience. A veterinarian from New Zealand commented on the problems associated with rearing birds to point of lay in cages and then placing them in floor systems (barn, free range). This often results in problems with parasites such as worms and coccidia and producers are not always clearly aware of what treatments are required. This veterinarian also mentioned problems with nutrition that are blamed on disease. He is of the opinion that infectious bronchitis tends to be blamed for shell and albumen quality problems when the cause may be something else (or a combination of other factors).

A veterinarian from the UK, Dr David Burch from Octagon Services Ltd, offered the opinion that egg drops are not so much a syndrome as a production failure. Dr Burch maintains a website on which information about disease and egg drops is provided (Burch, 2010). A veterinarian from Australia offered the following list of diseases and other factors which affect egg production and quality: *Mycoplasma gallisepticum*, *Mycoplasma synoviae*, Egg Drop Syndrome-76, avian influenza (high and low pathogenicity), Newcastle disease virus, infectious bronchitis virus, turkey rhinotracheitis, avian herpesvirus and avian encephalomyelitis virus. Rarely, paramyxoviruses and other viruses can cause problems. This veterinarian also stressed the importance of management factors in achieving good egg quality: feed, water, heat, cold, electricity and light.

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