Some Problems Encountered in Commercial Flocks of Japanese Quail and Utility Pigeons

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INTRODUCTION

Average consumption of poultry meat in Australia is just below 25 kg *per capita* per year and more than 90% of this is derived from broiler chickens. Commercial production of ducks and turkeys is controlled by large and/or integrated poultry companies. There is a small but viable commercial game bird industry composed of small companies and entrepreneurs. Other bird meats in Australia are derived from farmed ratite birds, wild harvested mutton birds (shearwater chicks), and shot ducks and quail.

In this presentation I will discuss observations on various episodes encountered as an avian pathologist working at Department of Agriculture, Veterinary Research Institute, Parkville Vic 1977-87 with due acknowledgement to my colleagues Drs Don Barr, Mike Forsyth, Jim Gould, Michael Hindmarsh, and Peter Scott, and many associates especially the late Vic Beddome. Besides necropsy of birds at VRI, farms were visited and treatment programs recommended; therapeutic agents are referred to by generic names rather than trade names, and dose rates are not given in this article. Some other veterinarians (private practitioners, company and government) were also involved in some of these flocks. Prescribed restrictions imposed by district veterinary officers for control of zoonotic diseases usually prohibited sale of animals for slaughter and movement of birds or bird products from off the farm, and disease control programs were put in place and progress monitored.

COMMON HUSBANDRY

The Japanese quail breeders were kept on wire. On most farms birds were hatched once or twice weekly and then brooded either in a common area and later transferred to growing pens or sheds, or they were brooded and grown in the same area as the breeders. Therefore it was not uncommon to have 6-8 different age groups in adjacent pens or near-by sheds. Replacement breeders were selected from amongst the better grown progeny. A rearing mortality rate of 5% to 6 weeks was considered achievable for Japanese quail. In the case of utility pigeons, each breeding pair was expected to rear 10-12 squabs per year.

Typical sheds had earthen floors, and rice hulls were often used for litter. Some farms had more than one species, even in adjacent pens in the same shed. Many farms not only had breeders, hatching facilities and grower pens, but also small registered processing works. Some Japanese quail were grown under contract. Suitable feeds were available commercially, although some squab producers used free-choice feeding of grains and supplements.

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JAPANESE QUAIL (see Table 1 for summary)

JQ1. Clinical signs were depression, diarrhoea, tremors in 10%, and some paresis with paddling in poults. Pathology included haemorrhagic enteritis, polyserositis and meningoencephalitis. *Salmonella hessarek var 27* was isolated from affected birds, and also from trapped rats and hatchubator debris. The breeder flocks were given furazolidone via feed for 2 weeks and each batch of poults were given furaltadone via drinking water (DW) for 5 days. There was greater attention to hygiene, sick birds were culled, and better rodent control was implemented. Further cases were not reported.

JQ2. Young poults exhibited depression and diarrhoea with a high incidence of yolk sac infection, haemorrhagic enteritis and caseous typhlitis. *Salmonella typhimurium* was isolated from affected poults. They were given furaltadone via DW for the first 7-10 days, and hatchery hygiene was improved including exclusion of dirty eggs. This successfully controlled the disease.

JQ3. Although overall mortality from sudden death in breeders was only about 5% mortality, up to 50% of the young breeders recently introduced from the grower pens were dying. Necropsy revealed peritonitis, splenitis and hepatitis. *Pasteurella multocida* was isolated. Sick birds were culled, the flock was treated with oxytetracycline via DW for 7 days, and introductions of replacement breeders were segregated as batches rather than dispersed amongst older birds.

JQ4. This farm had a recurrent and serious problem of infraorbital sinusitis and conjunctivitis in young quail. *Haemophilus paragallinarum* was isolated from affected sinuses. The farmer purchased laying hens from a local market and kept them in the same shed with the quail and it was probably those hens which introduced the infection (Reece et al 1981). The hens were removed, affected quail were culled and the flock was treated with sulphonamides. There was limited depopulation but there were recurring episodes of disease. Finally, all birds were removed and after several months new breeders from an unaffected flock were purchased. There were no further outbreaks. The farmer had to purchase large numbers of young quail for rearing and processed quail to fulfil contracts until the new breeders were established.

JQ5. The onset of conjunctivitis and sinusitis in adults was of sudden onset and coincided with a decrease in egg production. Affected birds had a tracheitis and airsacculitis from which *Mycoplasma gallisepticum* was isolated. The farmer was in the habit of buying-in replacement breeders as well as selecting from his own stock and it is probable that one batch of bought-in young breeders were infected. Treatment with sulphonamides then amoxycillin via DW for 2 weeks suppressed clinical signs for short periods but there were relapses. Eventually the entire breeder flock was culled, and replaced by young birds from an unaffected farm (Reece et al 1986a).

JQ6. Many adult breeders were affected with a chronic sinusitis, depression and poor egg production although mortality appeared proportionally higher in young breeders. Necropsy revealed tracheitis, airsacculitis and sinusitis from which *Mycoplasma gallisepticum* was isolated. There was no evidence of clinical disease in large number of growers in a separate shed. Culling of severely affected birds and treatment with sulphonamides and lincomycin-spectinomycin via DW for periods of up to two weeks resulted in mild clinical improvement but relapses occurred. Eventually the entire breeder flock was culled, the shed spelled for several weeks, and replacement breeders were purchased from an apparently unaffected flock (Reece

et al 1986a).

JQ7. Many breeders had small lacerations of their foot-pads, with weight loss and poor fertility. On necropsy there was severe pododermatitis and tenosynovitis from which *Staphylococcus aureus* was isolated. The wire floors of the breeder cages had many small sharp projections of galvanising material, sufficient to rip paper pulled across it. The wire was sanded, affected birds were culled, and the remainder were treated for 10 days via DW with potentiated sulphonamides.

JQ8. Many growing quail were depressed with sneezing and catarrhal sinusitis. Growth rate was poor. Necropsy revealed mucoid sinusitis and tracheitis, and cryptosporidia were observed on mucosa of sinuses. Few affected birds died but many were culled. The first cases were associated with dusty feed which was replaced. Treatment with sulphonamides, tetracyclines, or lincomycin-spectinomycin via DW had little benefit and cryptosporidiosis continued to be an intermittent problem.

JQ9. Growers and some breeders were affected with depression and sinusitis. Significant mortality and poor growth occurred in growers. This was of recent onset and may have been associated with introductions. *Mycoplasma gallisepticum* was isolated from affected sinuses and cryptosporidia were observed on the sinus mucosa. All birds were treated with tylosin via the DW for 5 days and there was marked clinical improvement with a reduction in mortality rates and improved growth rates.

JQ10. Many grower flocks were affected for a long time before assistance was sought. There appeared to be several waves of mortality, the first at 2-7 days (10-20% mortality) in poorly grown depressed poults with yolk sac infections, polyserositis and haemorrhagic enteritis from which *Salmonella typhimurium* was isolated. Between 2-4 weeks losses were 20-30%, mainly associated with sinusitis, airsacculitis and tracheitis. These yielded *Mycoplasma gallisepticum* and additionally there were many cryptosporidia on the sinus mucosa. Deaths from polyserositis, airsacculitis and severe sinusitis continued to 6 weeks and older and growth rate was poor. Many of the birds examined at this age had a mild to moderate enteritis associated with *Eimeria spp* in the small intestines. The feed contained amprolium, sulphaquinoxaline and ethopabate as a coccidiostat. Extensive culling of affected birds and treatment with amprolium, chlortetracycline, multivitamins via the drinking water resulted in little clinical improvement. Similar problems continued .

JQ11. This farm was affected with intermittent problems of depression, poor growth and pneumonia and airsacculitis associated with *Aspergillus fumigatus*. The brooding area was dusty but clean up did not prevent further problems. Most of the poults were purchased from another breeder, and they were frequently kept in the hatchubator for 2-3 days post-hatch before despatch. Examination of poults removed from the hatchubators revealed well-developed granulomas in some lungs. The incidence of fungal problems substantially declined following improved hatchery procedures.

JQ12. This farm had been experiencing minor problems of poor growth and mortality in young quail for some months, and then the mortality rate increased dramatically. Substantial losses (80-95%) were encountered in every flock placed for several months before assistance was sought. At 2-4 weeks, 2-5% of birds exhibited retropulsion associated with fungal spondylitis of the last few cervical vertebrae, another 5-10% had tremors and paresis associated with fungal encephalitis. Most birds necropsied had severe fungal pneumonia and airsacculitis. A

variety of fungi were isolated from lesions, including multiple species from individual birds - *A. fumigatus, Penicilliium spp, Rhizopus spp, Pullularia spp* and *Mucor spp*. The farm was very dusty with thick built up dust on the incubator and hacthubators, on brooders, and hessian curtains surrounding the brooding areas. The air inlets into the incubator were cleaned, dust was removed from flat surfaces, the hessian curtains were removed, and the brooders cleaned. Clean up between batches was instituted. Within a few batches, total mortality to 6 weeks was less than 5% (Reece et al 1086b). However, 2 years later there were several flocks with more than 50% mortality rates and the same procedures were again successful in reducing the incidence in subsequent flocks.

JQ13. This farm experienced 20-30% mortality in growing birds 2-4 weeks of age with 1% showing retropulsion associated with fungal spondylitis of the last few cervical vertebrae, 5% showing tremors and paddling associated with fungal encephalitis, and the remainder with fungal pneumonia and airsacculitis. *A. fumigatus, Absidia spp* and *Rhizopus spp* were isolated from lesions. The incidence of clinical problems was reduced to negligible by reducing dust levels, removing brooding curtains, increasing ventilation and thorough disinfection and clean-up between batches (Reece et al 1986b).

JQ14. There was one batch only affected with 70% mortality at 2-4 weeks. Approximately 1% of poults were presented blind with fungal keratitis, and 5% had tremors and paresis due to fungal encephalitis. Fungal pneumonia and airsacculitis were common but retropulsion and spondylitis were not a feature. *A.fumigatus, Absidia spp, Rhizopus spp* and *Mucor spp* were isolated from lesions. This was the same farm as JQ13 but 2 years later. Brooding curtains had been reintroduced, and the ventilation reduced to reduce brooding costs.

The curtains were abandoned and attention was paid to ventilation. Large numbers of poults in adjacent pens in the same shed (both one week older and younger) were not affected.

JQ15. Four sequential batches of 2500 quail poults were affected at 2-4 weeks of age. The growth rate was poor, feathering was poor and 40-60% of poults had encrustations at the commisures and polyps on the phalanges. Histologically, the polyps were composed of retained keratin layers, with hyperplasia and hypertrophy of the stratum spongiosa typical of pantothenic acid deficiency, with some superficial bacterial infection. Affected poults were culled. Treatment with multivitamin B preparation via DW was associated with some clinical improvement and a reduced incidence in subsequent flocks. Subsequent poults reared on another delivery of feed from the same mill were not affected. The feed in question was assayed and found to contain 35ug/gm of pantothenic acid.

JQ16. The poults were given a turkey prestarter feed (28% crude protein) to 2 weeks of age, followed by a broiler starter feed (22% crude protein). The problem occurred in 8 sequential batches of 4000 poults which developed flaccid paralysis with poor growth rates and 40-60% mortality at 2-3 weeks of age following feeding of the broiler feed. The broiler feed was labelled to contain 90ppm monensin, and assays of the incriminated feed revealed normal monensin concentrations (89ppm). The affected birds did not have histological lesions in adductor and other muscles (Reece et al 1985). Subsequent flocks were given a commercial game bird starter feed which did not contain ionophorous coccidiostats, and there was better growth rate, no flaccid paralysis and normal mortality (<5% to 6 weeks).

JQ17. Sequential flocks were affected with paresis, flaccid paralysis and high mortality (75-

90%). There appeared to be some association with a particular batch of feed, yet feeding trials with that feed did not induce clinical problems and other farmers using the same batch of feed were unaffected. The affected poults were reared on wire in small groups of 20-30 poults. No adults were affected but there was a 66% (100/150) mortality from flaccid paralysis in a small flock of pheasant poults on the same farm. There were no histological lesions and assays of feed did not reveal any known toxins (Reece et al 1986c). Subsequent flocks given another batch of feed were not affected, and it did recur in subsequent years with similar negative results of tests. It was assumed to be due to botulism, although this was not able to be confirmed.

JQ18. This flock suffered a 35% mortality in 2 days at 2 weeks of age during extremely hot weather in mid summer. The brooding shed was uninsulated and in an exposed position, the gas brooder was 0.6m above the litter and on, the thermostat was broken, and there were heavy hessian curtains around the brooding area. The farmer reported that the poults were panting prior to death and carcasses examined were severely autolysed. A new thermostat was installed for the next batch and subsequent batches were brooded satisfactorily.

JQ19. During mid winter, 2 batches, 2 and 3 weeks of age respectively, in adjacent pens were depressed, huddled, inactive, and suffered a high mortality. Necropsy revealed distended ureters, litter eating, and poor bodily condition. Following a diagnosis of chilling it was revealed that a portion of the roof had blown off and several areas of the floor had become flooded. The brooders had been switched off. The surviving birds did not grow well.

JQ20. The majority of this flock 600 poults had died by 5 days of age; the rest were culled. Necropsy revealed emaciated birds with empty gastrointestinal tracts, swollen gall bladders, and resorbed yolk. It was discovered that they had been given turkey breeder pellets not starter crumble.

JQ21. This farmer encountered repeated problems with high first week mortality (20-30%) and necropsy revealed poor to emaciated poults with empty gastrointestinal tract or litter eating. Brooder management seemed satisfactory. The poults were purchased from another breeder and they were left in the hatchubator for 2-3 days until there were sufficient numbers hatched to make up orders, and then sent overnight by rail. Subsequent batches were removed after one day in the hatchubator and sent by better services. First week mortality fell to less than 5%.

JQ22. Four sequential flocks were affected at about 4 weeks of age with 20-30% showing poor growth, tremors, ataxia and incoordination. Histologically, there was extensive degeneration and necrosis of Purkinje cells of the cerebellar cortex. Both males and females were affected. The starter and grower feeds contained the coccidiostat dinitolmide at 125ppm (assayed at 88-106ppm) but no nitrofurans. Subsequent flocks given the same feed were not affected and the cause was not determined.

UTILITY PIGEONS (see Table 2 for summary)

UP1. Birds of all ages were found dead in good condition. Necropsy revealed focal hepatic necrosis and pharyngo-oesopheal diphtheresis. Amphophilic intranuclear inclusion bodies were found in hepatocytes and pharyngeal-oesophageal epithelial cells, as well as pancreatic acinocytes, tracheal epithelial cells and cells in other organs. Herpes virus was isolated from

affected livers in chick kidney cell cultures. Following this first major episode, intermittent problems occurred in subsequent years.

UP2. Mortality mainly occurred in squabs reared on the floor rather than in nest boxes. affected squabs were in poor condition with hepatomegaly, splenomegaly and airsacculitis. Chlamydia were identified in splenic impressions. The farm was placed under prescribed restrictions, and the flock was treated with chlortetracycline via DW for 45 days. Backward breeders and poor squabs were culled, and nesting on the floor was discouraged. Many moribund and dead birds were examined in the ensuing years, and psittacosis was not diagnosed.

UP3. There was no history of clinical disease in birds from this flock but freedom from *Chlamydia psittaci* was required to supply yearlings to a particular market. Using a mammalian CFT, many of the breeders were sero-positive to *C.psittaci*, and chlamydia were isolated from faecal swabs and spleens from a few sero-positive birds. The flock was treated with chlorotetracycline via DW for 45 days and sero-negative breeders were transferred to a clean isolated unit. Their squabs reared in isolation were sero-negative.

UP4. These pigeons were overcrowded and nests were frequently abandoned leading to death of squabs and partially incubated eggs so much so that production per breeding pair was only 3-5 squabs per year whereas it should have been 10-12. During winter, the shed became wet, damp and cold, and there was a sudden increase in mortality of adults and squabs. Necropsy revealed poor condition, airsacculitis, polyserositis, hepatomegaly, splenomegaly and hepatic granulomas. Chlamydia were observed in splenic smears and *Salmonella typhimurium* was isolated from lesions. The flock was placed under prescribed conditions and treated with chlortetracycline via DW for 45 days. Squabs hatched during that period were killed, breeders in poor condition were culled, the shed was weatherproofed, hygiene was improved and new sheds erected. Each pair of breeders subsequently reared 8-10 squabs/year, and no further outbreaks of clinical disease were encountered.

UP5. The breeders were in groups of 5-800 pairs in old, open-sided sheds. The litter was caked and many birds nested on the floor. All ages were affected with up to 20% of squabs dying and 10% of adults. Some showed tremors, others were found dead in good condition, whereas others were in poor condition. Necropsy revealed polyserositis, airsacculitis, splenomegaly, hepatomegaly and hepatic granulomas. Chlamydia were demonstrated in splenic smears and *S.typhimurium* was isolated form lesions. The farm was put under prescribed restrictions. Salmonellae isolated from this farm were resistant to tetracyclines so the flock was treated with furaltadone via DW for 10 days, and then chlortetracyclines via DW for 45 days. The mortality rate declined during treatment but clinical disease occurred after cessation: this cycle was repeated several times. Control was finally achieved in conjunction with culling 1500 adults and juveniles, construction of more housing, regular replacement of litter, and improved farm hygiene. In the meantime all squabs hatched were killed and no stock were allowed off the farm.

UP6. This flock had been affected for some months before assistance was sought. Squabs were dying in poor to emaciated condition with large pharyngeal ulcers associated with trichomonads; some also had peritonitis and splenomegaly from which *S.typhimurium* was isolated. Adults were also dying in poor condition with peritonitis, splenomegaly, oophoritis and orchitis associated with salmonellosis. The affected adults harboured heavy burdens of slender pigeon lice (*Columbica columborale*) although few lice were observed on other apparently healthy birds. The flock was treated with chlortetracycline and dimetridazole simultaneously via

DW for 7 days, then furaltadone via DW for 5 days. They were given proprietary feed containing dimetridazole at 125ppm. Subsequent mortality was negligible, and growth rate of the squabs improved.

UP7. The birds were housed in 3 sheds of about 200 breeding pairs each but only birds in one shed were affected. That shed leaked during wet weather. Squabs were found dead in good condition (10% across farm but 30% of those in that shed) and necropsy revealed peritonitis and splenomegaly with positive splenic impression smears for chlamydia in some, and focal hepatic necrosis with intranuclear herpes like inclusions in others. The farm was put under prescribed restrictions. Moribund squabs and breeders in poor condition were culled, and the shed was waterproofed. No further clinical cases were detected and monitoring failed to reveal any further cases of chlamydia in culled or dead birds. Therefore no treatment was undertaken.

UP8. The annual production of squabs per breeding pair was less than 3 during the previous year: the shed was overcrowded, nests were frequently abandoned and there were insufficient nest boxes available. During winter there was a sudden rise in mortality of squabs (50%). These were generally in poor condition with airsacculitis, splenomegaly and hepatomegaly associated with chlamydia and/or *S. yphimurium*. Others had pharyngeal ulceration due to trichomoniasis. The farm was placed under prescribed restrictions and the flock was treated with chlortetracyclines via DW for 45 days. Adults in poor condition were culled, an adequate number of nest boxes were installed, and proprietary feed containing dimetridazole was given. This brought the mortality problem under control and production improved. However, intermittent problems with chlamydiosis and/or paratyphoid continued despite several 45 day treatment periods with chlortetracycline. During this time sale of squabs was not permitted.

UP9. This farmer had recently commenced rearing utility pigeons for squab production and they were fed a whole grain only diet. About 5% of adults were in poor condition with swollen crops and intermittent regurgitation. On necropsy the lining of the crop was thickened and contained abundant yeast typical of *Candida albicans*. Affected birds were culled and the remainder were given proprietary pelleted feed. No further problems were noted.

UP10. Within 6-8 weeks of eating a recent delivery of feed most, if not all adults displayed a variety of neurological disturbances including ataxia, incoordination, a rolling gait, an inability to fly, and an inability to mate. Culled birds had necrosis of Purkinje cells in the cerebellar cortex. Very few adults died, but nests were abandoned and squabs died of chilling and/or starvation. The feed accidentally contained the coccidiostat dinitolmide at 185-226ppm. It was shown experimentally that feeding this feed, and experimental feed containing a similar concentration of dinitolmide, for 6-8 weeks caused clinical sings similar to the field cases and necrosis of Purkinje cells in the cerebellar cortex (Reece and Hooper 1984). Severely affected breeders were culled and the fertility of the remainder was extremely low. Many adults had marked incoordination and tremors 2 years later and were unable to mate and/or rear squabs. Suitable numbers of replacement breeders were not available for purchase and all farmers were unable to fulfil contracts to supply squabs. Financial compensation was not achieved until 5 years after the event. In the meantime it proved extremely difficult to purchase sufficient squabs to fulfill contracts. At least another two farmers purchased feed from the same batch and were similarly affected but were not able to pursue the matter through court and obtain compensation.

UP11 This flock was treated with furaltadone via DW during summer. All the medicated water was consumed within a few hours. The adults were ataxic the next day and the nests were

abandoned. None of the adults died, and necropsy of affected birds failed to reveal any neurological pathology: the squabs died on neglect and partially incubated eggs were discarded. Within a week the adults had all recovered and production was back to normal after 2 months (Reece et al 1985).

DISCUSSION

Some of the cases mentioned in this article have been reported in greater detail elsewhere as indicated. They have been included here to complete the overview of the types of problems encountered, and to show either the impact of disease and/or the effectiveness or otherwise of various control programs. The diseases and problems reported in this article were not unexpected: what was unusual was the high and repeated mortality rates which were encountered and to some extent tolerated. In many cases the impact of these diseases was rapidly diminished by appropriate control and/or treatment programmes, including attention to basic hygiene and management. It needs to be emphasised that although these were commercial flocks, many of the "farmers" had little or no previous experience of intensive livestock production, and the local veterinary practitioners often did not have the experience or inclination to get involved.

In many of these cases treatment with appropriate chemotherapeutic agents in conjunction with management changes such as culling and attention to hygiene, lead to a marked decrease in the incidence and severity of clinical signs. The infectious agents were probably not eliminated, and in some cases relapses occurred which were also treated in a similar manner. In the case of the mycoses, control was achieved without recourse to chemotherapy. Antibiotic treatment of flocks affected with mycoplasmosis suppressed clinical disease for short periods: control was only achieved by radical management procedures, namely culling of all breeders and their replacement by mycoplasma-free birds.

Until recently the legal importation of avian species into Australia was banned. Therefore, these birds were to some extent inbred, and there has been limited opportunity for selection based on quantitative production characteristics. This had significant implications when disease control programmes required depopulation and replacement by breeders free of specific diseases: in such instances birds often were not available in the numbers needed.

Chlamydiosis was diagnosed in utility pigeon flocks only and was rare in other commercial poultry flocks. It was a common diagnosis in racing pigeons and psittacine birds in Victoria during this same period (Reece et al 1992). It is highly likely that many utility pigeon flocks in Victoria had subclinical infection with *C.psittaci* and *Salmonella spp*. Problems occurred with stress conditions which precipitated clinical disease, or during attempts to obtain verification of freedom from disease for particular markets or contracts.

It is interesting to note that in the above cases with fungal pneumonia and airsacculitis, respiratory distress was not a presenting clinical sign. The main problems were poor growth and death, with a variable but low incidence of retropulsion, tremors and/or keratitis.

Respiratory cryptosporidiosis of various poultry species is a well recognised disease (Goodwin 1989). The impact of cryptosporidiosis on flocks varied greatly: some cases were of a single flock where previous and subsequent batches, and associated flocks, were not clinically affected even though there was no attempt at treatment or prevention of spread. On the other hand, some farms had recurrent problems, usually in association with other respiratory

pathogens. With the possible exception of tylosin, none of the treatment regimes instituted had any ameliorating effect. In this series, sinusitis was a clinical feature in Japanese quail infected with *H.paragallinarum, M.gallisepticum*, and respiratory cryptosporidiosis, and in several flocks more than one respiratory pathogen was involved. Many birds with cryptosporidiosis had to be culled but mortality as such was low, whereas in flocks with mycoplasmosis death was common. Appropriate disease and control programs varied depending upon the causative agent, and in the case of sinusitis, diagnosis was not possible on the basis of flock histories and clinical signs alone: laboratory testing was essential.

The cutaneous lesions observed in case JQ15 (also encountered in an unrelated pheasant flock) resembled that induced by a deficiency in the B vitamin pantothenic acid (Austic and Scott 1984), and there was also evidence of feed association and response to vitamin B treatment. A similar episode was reported by Raidal (1995). The recommended feed inclusion rate of pantothenic acid for young Japanese quail was 10mg/kg (Anonymous 1984), although there was some indications that their requirements may be greater: up to 40mg/kg (Dark 1986), which was not much greater than the concentration found in this feed (35mg/kg).

Pigeons are monogamous altricial birds with both parents involved in incubating the eggs and rearing squabs. Hence, toxicity induced neurological disorders of adult pigeons due to dinitolmide or furlatadone (and also in racing pigeons overdosed with dimetridazole) lead to the abandonment of incubating eggs and squabs, and in the former also to long-term infertility from inability to mate.

Some of the episodes reported in the article were directly attributable to poor management; they could readily have been avoided by attention to detail. Many other flock diseases were investigated but the cause of the problem was not clearly identified and/or follow-up information was inadequate. Very few of these farms generated sufficient income to provide a livelihood. In order to boost income some farmers also kept other game birds or poultry, had other on-farm enterprises (dairy and beef cattle, sheep, wheat, tourism), or had other employment. Therefore, management of the birds was sometimes sporadic and capital investment for alterations was not always readily available.

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TABLE 1. SUMMARY OF JAPANESE QUAIL FLOCK PROBLEMS.

Case	Age affected	Duration	Flock size	Sick %	Dead %	Diagnosis
JQ1	7-10 d	1 w	4 x 1200	30	25	*S. hessarek*
JQ2	3-4 d	1 w	5 x 1500	50	40	*S. typhimurium*
JQ3	8-12 w	2 w	1200	5%	5	*P. multocida*
JQ4	2-4 w	2-6 w	12 x 1000	10-20	10-15	*H. paragallinarum*
JQ5	8-12 w	3 m	1000*	12-20	10	*M. gallisepticum*
JQ6	8-16 m	3 m	3000*	30-50	30	*M. gallisepticum*
JQ7	1-2 y	months	800	10	2	bumblefoot
JQ8	3-6 w	2-4 w	1000*	10-15	5	cryptosporidiosis
JQ9	2-6 w	2-4 w	3000*	30-60	25-50	*M. gallisepticum* & cryptosporidiosis
JQ10	2 d- 6 w	4 w	2500*	50-90	40-80	*S. typhimurium, M. gallisepticum, Eimeria* spp, cryptosporidiosis
JQ11	2-4 w	2 w	4 x 1000	5	5	*A. fumigatus*
JQ12	2-6 w	2-4 w	3000*	80-95	80-95	fungi
JQ13	2-4 w	2-4 w	5 x 3000	25	25-30	fungi
JQ14	2-4 w	2 w	7000	70	60	fungi
JQ15	2-3 w	2 w	4 x 2500	40-60	40-60	pantothenic acid deficiency
JQ16	2-3 w	3-4 w	8 x 4000	40	25	monensin toxicity
JQ17	3-6 w	4 w	7 x 2500	75-90	75-90	flaccid paralysis
JQ18	2 w	2 d	1500	35	35	heat stress
JQ19	2 & 3 w	5 d	2 x 4000	40	25	chilling
JQ20	1-5 d	5 d	600	100	60	wrong feed
JQ21	1-5 d	5 d	3300*	20-30	20	non-starters
JQ22	4 w	4w	4 x 3000	20-30	20-30	cerebellar degeneration

d=days; w=weeks; m=months; * multiple batches affected

TABLE 2: SUMMARY OF UTILITY PIGEON FLOCK PROBLEMS

Case	Age affected	Duration	Flock size	Sick %	Dead %	Diagnosis
UP1	4-8 m	4 m	1500 pr 800juv	5	5	pigeon herpes
UP2	2-4 w	intermittent	500 pr 200 juv	1 sq	1	chlamydiosis
UP3	adults	?	300 pr 100 juv	neg	nil	chlamydiosis
UP4	2-4 w	1-3 m	300 pr 100 juv	25-30	10 ad 20 sq	chlamydiosis & *S. typhimurium*
UP5	all	6 m	2000 pr 1000 juv	10-20 sq	5-10 ad 5-10 juv	chlamydiosis & *S. typhimurium*
UP6	all	months	100 pr 50 juv	10 sq	10 sq 2 qd	*S. typhiurium,* trichomoniasis & lice
UP7	4-10 m	2 m	600 pr 100 juv	10 sq	10 sq	pigeon herpes & chlamydiosis
UP8	all	6 m	400 pr	50 sq 5 ad	50 sq 5 ad	chlamydiosis, *S. typhimurium* & trichomoniasis
UP9	adults	6 m	100 pr	5	2	C.albicans
UP10	adults	2 m#	800 pr 500 juv	100 ad	neg ad 100 sq	dinitolmide toxicity
UP11	adults	3 d	200 pr	100 ad	neg ad 50 sq	furaltadone toxicity

m = months; ad = adult; juv = juvenile; sq = squab; neg = negligible