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**COMPARATIVE EVALUATION OF THE DEVELOPMENT OF RECURRING  
NATURAL FOWL POX INFECTIONS IN BROILERS AND COCKERELS UNDER THE  
SAME EXPERIMENTAL CONDITIONS**

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**ABSTRACT**

Fowl pox has recently been considered in some quarters as a re-emerging infection. This is because the disease is assuming a renewed importance in some parts of the world and strains capable of causing more severe infections are being identified couple with cases of outbreaks of the disease in vaccinated chickens. Recurring outbreaks are reported in chickens especially where there is no vaccination against the disease. This work followed recurring infections of the disease involving broilers and cockerels in an experimental farm. Results showed 83.3% recurring outbreak of the disease in cockerels and no outbreak (0%) in broilers. Outbreaks occurred at 7 – 9 weeks of age. This report therefore identifies the period of 7 – 9 weeks of age as the window period of age when disease can occur if the virus is present in the environment. This is likely the period that the maternally derived antibody has waned away and is no longer protective. It also points out the marked differences in occurrence of the disease in broilers and cockerels which is likely of epidemiological importance especially in our locality where the disease is endemic. The development of cutaneous wounds due to hostilities among the cocks leading to the entry of the virus in the environment and possible antigenic variation and host specificity were suggested as possible reasons for the wide variation in the result. The need for molecular characterisation of the isolate in Nigerian was pointed out.

**Keywords: Fowl pox, chickens, Broilers and Cockerels, Nigeria**

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## INTRODUCTION

Fowl pox (FP) is a disease of global distribution [1, 2, 3, 4]. Pox disease has been reported to affect over 232 species in 23 orders of birds, including chickens, turkeys and pigeons [5]. The incidence is higher in tropical and subtropical countries [4, 6]. FP is endemic in Nigeria and is very common among the village and exotic commercial poultry with frequent outbreaks among this population of birds. It is one of the greatest challenges facing the poultry industry, especially in developing countries. Avian pox infections are associated with significant levels of morbidity and mortality in domestic and wild bird populations [7]. In commercial poultry the disease is economically important as it causes a transient drop in egg production, a reduced growth rate in young birds, poor carcass quality due to the lesions it causes on dressed chicken and mortality [1, 8]. Moreover, fowl pox lesions may compromise vision and the ability to feed, or lead to secondary bacterial or fungal infection [9, 10]. Both large and small scale enterprises are affected [4]. Susceptibility to avipoxvirus infection varies among host species, and in relation to host age (juveniles are most susceptible), immunocompetence, season and local environment [10].

The etiologic agent, Fowl pox virus (FPV) is among the largest and most complex viruses known and belongs to the Avipoxvirus genus within the Chordopoxvirinae subfamily and of the *Poxviridae* family [11, 12]. They are large, oval-shaped enveloped viruses whose genome consists of double stranded DNA ranging in size from 260 to 365 kilobase pairs and unlike most other DNA viruses, they replicate easily in the cytoplasm of infected avian cells which results in a characteristic cytopathic effect (CPE) 4 to 6 days post infection depending on the virus isolate [13, 16]. The virus produces a slow spreading disease characterized by the formation of proliferative lesions and scabs (dry form) on skin and featherless areas, and diphtheritic lesions (wet form) in the upper part of digestive and respiratory tracts [17]. The chances of mortality increases when the dry form occurs together with the wet form [18, 19].

Detection and presumptive diagnosis of the diseases is straightforward and can be made on the basis of clinical signs and gross lesions [20, 22]. Impetus for research has also been reduced because the disease can easily and effectively be controlled through vaccination [20]. Currently available vaccines are very effective and have undoubtedly contributed

immensely to the prevention of the disease in commercial poultry farming [16, 23]. However, during recent years there has been an increase in reported cases of this disease, and cases in new bird species have occurred, suggesting that avian pox may be a re-emerging avian disease [9, 24]. Davidson et al. [25] and Hess et al. [26] also reported that fowl pox outbreaks in poultry flocks in recent years have been gradually increasing because of an emerging novel type of FPV. Zhao et al. [27] reported an acute disease caused by a highly pathogenic virus causing cutaneous outbreaks in chickens in northeast China. Mortality rates of up to 100% occurred in this commercial poultry flock and this results show that the novel FPV they isolated was much more pathogenic than common FPV strains obtained from other chickens infected with the cutaneous form of fowlpox. This highly pathogenic FPV variant is a potential threat to chickens and could lead to severe ecologic effects and economic losses. Fowl pox is responsible for significant economic losses in commercial poultry, and particularly for small farmers who cannot afford vaccination and other biosecurity. It is also very important in village chickens that are predominantly not vaccinated against the disease.

The ubiquitous nature of fowl pox virus has been reported [7, 28]. Moreover, the recurrent nature of the disease in poultry farms has also been reported [29]. It has become common to find the disease recurring in many commercial and household farms, sometimes as severe outbreaks resulting in significant losses especially in unvaccinated flocks. It has been noted that the virus tends to persist in the poultry environment for extended periods of time where other viruses may not survive and the presence of photolyase gene and A-type inclusion body gene in the virus genome appear to protect the virus from environmental insults [19, 30]. For this reason the virus tends to persist in the environment that is contaminated with the virus causing infection in susceptible and unprotected chickens. This paper reports outbreak of the disease in many batches of white cockerels and the absence of outbreaks in batches of broilers reared in a particular pen and under the same rearing conditions. It also identified a particular period of disease outbreak in the birds. The possible reasons for this observation were discussed.

## **MATERIALS AND METHODS**

### **Experimental Design**

The experiment and observations were carried out in the animal house section of the Department of Veterinary Pathology and

Microbiology, University of Nigeria, Nsukka. The animal house was constructed with blocks and had concrete floor with zinc roofing. It was fly-proofed. A particular pen was used and there was an outbreak of FP in a batch of white cockerels kept in the pen. The infection occurred at the age of seven week and lasted for three weeks when the birds were disposed. Subsequent batches of broilers and cockerels, comprising of 5 batches of broilers and 6 batches of white cockerels which were reared on this pen at an interval of 3 – 4 months between the rearing of each batch were closely monitored and observed for the development of clinical signs and lesions of fowl pox. None of the batches of birds were vaccinated against fowl pox. All the batches of chickens were reared under deep litter system with feed and water given *ad libitum*. Diagnosis of the disease both in the initial outbreak and subsequent outbreaks were based on clinical signs and lesion [22, 31]. Agar Gel Immunodiffusion Test (AGID) [22] and virus isolation in embryonating chicken eggs [22, 36].

#### Batches of Chickens

**I.** A batch of 100 white cockerels brought in at day old and were reared up to 10 weeks.

- II.** A batch of 80 cockerel brought in at day old and were reared up to 10 weeks.
- III.** A batch of 60 broilers brought in at day old and were reared up to 10 weeks
- IV.** A batch of 100 cockerels brought in at day old and were reared up to 11 weeks
- V.** A batch of 100 broilers brought in at day old and reared up to 10 weeks
- VI.** A batch of 80 broilers brought in at day old and reared up to 9 weeks
- VII.** A batch of 120 white cockerels that were brought in at day old and reared up to 11 weeks
- VIII.** A batch of 80 broilers brought in at day old and reared up to 10 weeks
- IX.** A batch of 100 white cockerels brought in at day old and reared up to 10 weeks
- X.** A batch of 100 white cockerel brought in at day old and reared up to 10 weeks and
- XI.** A batch of 60 broilers that were brought in at day old and reared up to 10 weeks.

#### RESULT AND DISCUSSION

The batches of white cockerel except batch X had fowl pox outbreak between week 7 and week 9 of age (**Table 1**). This gave 83.3% disease outbreak in this breed of bird. There was no outbreak of the disease in any of the five batches of broilers during the periods of rearing in the experimental pen (**Table 1**). This gave a 0% disease outbreak in this breed of bird. The clinical disease in all the outbreaks in cockerels were similar and composed chiefly of multiple cutaneous lesions on the skin and very mild diphtheritic lesions in the buccal cavity observed only in batch VII. Mortality due to the disease was seen only in batch VII and this involved 3 chickens (3% mortality).

As stated earlier, fowl pox virus is ubiquitous and recurrent outbreaks of the disease in infected farms are quite common [29]. The results of this study showed recurrent outbreak of the disease in 83.3% the batches of white cockerels reared in the experimental pen. Surprisingly none of the batches of broilers reared in the same experimental pen showed outbreak of the disease. A striking revelation or observation in this study is therefore the marked difference in the recurrent outbreak of the disease in broilers and cockerels. Many factors may be responsible for this sharp difference in outbreaks in cockerels and broilers.

The modes of transmission of the disease may be a factor. Many different modes or routes of transmission of the disease have been identified. Transmission of virus can occur through a break in the skin or, more commonly, when vectored by biting insect such as mosquitoes and poultry ticks, mites, lice and other biting insects which may serve as mechanical vectors [32, 34]. Mockett [20] reported that the virus on its own will not break intact skin but requires some break in the skin for it to enter epithelial cells, replicate and cause disease. Cocks as they mature tend to show hostilities and fighting is common resulting in injuries and breaks in the skin which may allow the virus in the environment to penetrate resulting in infection. The fighting among the cocks is usually more pronounced as the birds get older and this may be responsible for the manifestation of the disease after 7 weeks of age. This may possibly be the time that aggressiveness and fighting habits leading to wound infliction become pronounced. Moreover, presence of maternally derived antibodies against the disease may have played some vital roles in the protection seen during the early periods of rearing. It is also possible that at above 7 weeks of age, the maternal antibody may have dropped to a non protective level and injuries due to any cause

can result in infection. As most of the cases were seen between 7 and 9 weeks of age, this may be the period that the birds may not have protective maternally derived antibodies to protect from the virus in the environment. This explains the need to vaccinate chicks before this period so as to build protective antibodies against the disease before maternally derived antibodies drop below the protective levels. Cocks on the average will also show prominent combs and wattles earlier than broilers and these are exposed areas of the skin where wounds and therefore lesions are more likely to occur. Another factor could be the inherent habit seen in pullets, layers and cocks on deep litter where the bird huddle together and sleep in close proximity to each other increasing contact between pen mates and this can encourage infection and spread of the disease within the flock. Broilers usually do not huddle together when sleeping in the night but spread evenly in the pen. Odoya et al [34] noted that transmission is commonly by contact to pen-mate and virus enters through abrasion of the skin. Adebajo et al. [35] reported that aerosols generated from infected birds, or the ingestion of contaminated food or water have also been implicated as a source of transmission. Cockerels produce drier litter than broilers and dry litters are more likely to move into

aerosol with the virus suspended in the air and this could be another factor leading to more potential for infection. We also ruled out the possibility of transmission by biting insects like mosquitoes and flies (which would have transmitted the disease equally to the cockerels and broilers) as the pen was fly proofed.

Another factor could be differences in the antigenicity and host specificity of the infecting virus. Though the host antigen-related viruses known to affect avian species are in general species specific [31], genomic and antigenic heterogeneity among avian pox viruses has already been demonstrated [23]. The authors identified the existence of strain of the virus noting minor antigenic variations among the strains which have been observed by Western immunoblotting technique. The authors argued that this in addition to the emergence of new strains may be responsible for the observed cases or outbreaks of fowlpox that have occurred in previously vaccinated flocks as these vaccines no longer seem to be effective. As stated before during recent years there has been an increase in reported cases of this disease, and cases in new bird species have occurred, suggesting that avian pox may be a re-emerging avian disease [24, 26]. In a study to determine the relationship between immunological variance

and effectiveness of vaccine protection, Singh et al [23] also noted that all the strains of FPV used in the study afforded some degree of protection; the differences in the persistence of primary lesions as well as in the development of secondary lesions indicate antigenic heterogeneity among these strains. Weli and Tryland [16] reported that strains of the virus vary in virulence and host specificity, demonstrating an urgent need for further analyses and characterization of new isolates. Based on the findings of their study, Masola et al, [36] concluded that though, fowl pox is currently prevalent in several regions and geographical locations of Tanzania, caused by FPV which are genetically and phylogenetically closely related, these findings do not rule out the possibility of

existence of genetic divergence among FPV currently prevalent in Tanzania. Based on our observation, it is possible that there may be genetic and antigenic divergence in FPV currently prevalent in Nigeria. Therefore other studies aimed at investigating the molecular and phylogenetic characteristic of the strains or isolates of the virus prevalent in Nigeria is recommended. This observation may also be important epidemiological as cocks may play a vital role in the outbreak, maintenance and spread of the disease in our environment.

We also suggest that this phenomenon of recurring infection could be tried as a method of producing natural infections in cockerels for biological studies in chickens where infection is required at 7 – 11 weeks.

**Table 1: Outbreaks and nature of recurring fowl pox infections in broilers and cockerels**

Batches of birds	Type of birds	Interval before introduction	Outbreak of FP	Age at outbreak of FP	Form of FP seen	Mortality due to FP (%)	Age at disposal
I	Cockerels (100)	3 months	Yes	7 weeks	Cutaneous	0	10 weeks
II	Cockerels (80)	4 months	Yes	8 weeks	Cutaneous	0	10 weeks
III	Broilers (60)	4 months	No	-	-	-	10 weeks
IV	Cockerels (100)	3 months	Yes	8 weeks	Cutaneous	0	11 weeks
V	Broilers (100)	3 months	No	-	-	-	10 weeks
VI	Broilers (80)	4 months	No	-	-	-	10 weeks
VII	Cockerel (120)	3 months	Yes	9 weeks	Cutaneous	0	11 weeks
VIII	Broilers (80)	3 months	No	-	-	-	10 weeks
IX	Cockerel (100)	4 months	Yes	7 weeks	Cutaneous, Mild Diphtheritic	3	10 weeks
X	Cockerel (100)	4 months	No	-	-	-	10 weeks
XI	Broilers (60)	3 months	No	-	-	-	10 weeks



**Figure 1: Arrow showing pox lesion on the comb of an infected cockrel**

## CONCLUSION

This paper therefore reports 83.3% recurring outbreak of fowl pox in white cockerels and no outbreak (0%) in broilers reared at different periods in a particular pen and under the same experimental conditions. Hostilities including fighting among the cocks and possible antigenic variation and host specificity among the viruses were suggested as likely reasons for this observation. It also reports a period between 7 and 9 weeks as the period of outbreak in the study suggesting that this may be the period when maternally derived antibodies would have dropped to a non protective level.

## REFERENCES

[1] G. Isa, K. Pfister, O. R. Kaaden and C. P. Czerny, “Development of a monoclonal blocking ELISA for the

detection of antibodies against fowlpox virus”, *Journal of Veterinary Medicine*, vol. 49, pp. 21-23, 2002.

[2] A. Pledger, “Avian pox virus infection in a mourning dove”, *Canadian Veterinary Journal*, vol. 46, pp. 1143-1145, 2005.

[3] C. Van Riperand and D. J. Forrester, “Avian pox. In: N. J. Thomas, D. B. Hunter, and C. T. Atkinson (editors). *Infectious diseases of wild birds*”. Blackwell, Oxford, pp 131–176, 2007.

[4] P.S. Silva, da, Batinga, T. de B., Sales, T.S., E.F.G. Herval, I. Ramos, P. C. C. Maia and L. M. B. Fernandes, “Fowl pox: identification and adoption of prophylactic measures in backyard chickens in Bahia, Brazil”, *Revista*



- Brasileira de Ciencia Avicola*, vol. 11(2), 115 – 119, 2009.
- [5] D. Boulanger, T. Smithband and M. A. Skinner, “Morphogenesis and release of Fowl Pox Virus”, *Journal of General Virology*, vol. 81, pp. 675 – 687, 2000.
- [6] E. Beytut and M. Haligur, “Pathological, immunochemical, and electron microscopic findings in the respiratory tract and skin of chickens naturally infected with avipoxvirus”, *Turkish Journal of Veterinary and Animal Sciences*, vol. 31(1), pp. 1 – 7, 2007.
- [7] D. N. Tripathy and W. M. Reed, “Pox. In: Y. M. Saif, A.M. Fadly, J. R. Glisson, L. R. McDougald, L. K. Nolan, D. E. Swayne (editors). Diseases of Poultry”, 12th edition. . Wiley-Blackwell, Ames, pp. 291 – 309, 2008a.
- [8] M. Ezeibe, E. Ekeanyanwu, A. Ngene and I. Mbuko, “Aluminum-Magnesium Silicate enhances release of virions of cultured Fowl Pox Virus and inhibits the virus”, *Health*, 5, pp. 1394 – 1396, 2013.
- [9] W. R. Hansen, “Avian pox. In: M. Friend, and J. C. Franson (editors), Field Manual of Wildlife Diseases”, General Field Procedures and Diseases of Birds. U.S.A.:Biological Resources Division Information and Technology Report 1999–2001, vol. pp. 163–170, 1999.
- [10] B. Lawson, S. Lachish, K M. Colvile, C. Durrant and K M. Peck, “Emergence of a novel avian pox disease in British tit species”, *PLoS ONE*, vol. 7(11): pp. 1 – 13. (2012).
- [11] C M. Fauquet, M A. Mayo, J. Maniloff, U. Desselberger and L. A. Ball, “Virus Taxonomy”: VIIIth Report of the International Committee on Taxonomy of Viruses. Elsevier Academic Press; 2005.
- [12] M. Gyuaranex, J T, Foster, A. Dan, H. S. Ip, K. F. Egstad, P. G. Parker, J. M. Higashiguchi, M. A. Skinner, U. Hofle, Z. Kreizinger, G. M. Dorrestein, S. Solt, E. Sos, Y. J. Kim, M. Uhart, A. Pereda, G. Gonzalez-Hein, H. Hidalgo, J. Blanco and K. Erdelyi, “Worldwide phylogenetic relationship of avian poxviruses” *Journal of Virology*, vol. 87(9), pp. 4938 – 4951, 2013.
- [13] C. L. Afonso, E. R. Tulman, Z. Lu, L. Zsak, G. F. Kutish and D. L. Rock, “The Genome of fowl pox virus”,

- Journal of Virology*, vol. 74(8), pp. 3815 – 3831. (2000).
- [14] D. N. Tripathy, W. M. Schnitzlein, P. J. Morris, D. L. Janssen, J. K. Zuba, G. Massey and C. T. Atkinson, “Characterization of poxviruses from forest birds in Hawaii”, *Journal of Wildlife Diseases*, vol. 36, pp. 225 – 230, 2000.
- [15] E. R. Tulman, C.L. Afonso, Z Lu, L. Zsak, G. F. Kutish and D. L. Rock, “The genome of canarypox virus” *Journal of Virology*, vol. 78, pp. 353-66, 2004.
- [16] S. C. Weli and M. Tryland, “Avipoxviruses: infection biology and their use as vaccine vectors”, *Virology Journal*, vol. 8, pp. 49, 2011.
- [17] D. N. Tripathy and W. M. Reed, “Pox. In: A laboratory manual for the isolation, identification and characterisation of avian pathogens”, 5<sup>th</sup> edition. American Association of Avian Pathologists, vol. 5, pp. 116 – 119, 2008b.
- [18] N. D. Maclachlan and E. Dubovi, “Fenner's Veterinary Virology”, 4th edition. Elsevier Academic Press, pp 163, 2009.
- [19] S. S. El-Mahdy, M. H. H. Awaad and Y. A. Soliman, “Molecular identification of local field isolated fowl pox virus strain from Giza governorate of Egypt”, *Veterinary World*, vol. 7(2), pp. 66-71, 2014.
- [20] A. P. A. Mockett, “Fowl Pox and other Avian Poxes”. In: F. T. W. Jordan, and M. Pattison. *Poultry Diseases*. Sun Valley Poultry Limited, Hereford, UK, pp. 166 – 172. (1996).
- [21] D. N. Tripathy and W. M. Reed, Poxvirus. In: Y. M. Saif, H. J. Barnes, J. R. Glisson, A. M. Fadly, L. R. McDougald, D. E. Swayne (editors). *Diseases of Poultry*. Iowa State University Press, Ames, IA; pp. 253 – 269, 2003.
- [22] Office International des Epizooties, “OIE Terrestrial Manual: Fowl Pox”, pp 531 – 537, 2008.
- [23] P. Singh, T. J. Kim and D. N. Tripathy, “Re-emerging fowlpox: evaluation of isolates from vaccinated flocks”, *Avian Pathology*, vol. 29, pp. 449 – 455, 2000.
- [24] V. Shearn-Bochsler, D. E. Green, K. A. Converse, D. E. Docherty, T. Thiel, H. N. Geisz, W. R. Fraser and D. L. Patterson-Fraser, “Cutaneous

- and diphtheritic avian poxvirus infection in a nestling Southern Giant Petrel (*Macronectes giganteus*) from Antarctica”, *Polar Biology*, vol. 31, pp. 569–573, 2008.
- [25] I. Davidson, I. Shkoda and S. Perk, “Integration of the reticuloendotheliosis virus envelope gene into the poultry fowlpox virus genome is not universal”, *Journal of General Virology*, vol. 89, pp. 2456 – 2460, 2008.
- [26] C. Hess, B. Maegdefrau-Pollan, I. Bilic, D. Liebhart, S. Richter and P. Mitsch, “Outbreak of cutaneous form of poxvirus on a commercial turkey farm caused by the species fowlpox”, *Avian Diseases*, vol. 55, pp. 714 – 718, 2011.
- [27] K. Zhao, W. He, S. Xie, D. Song, H. Lu, W. Pan, P. Zhou, W. Liu, R. Lu, J. Zhou and F. Gao, “Highly Pathogenic Fowlpox Virus in Cutaneously Infected Chickens in China”, *Emerging Infectious Disease*, vol. 20, pp. 7, 2014.
- [28] E. C. Okwor, D. C. Eze and I. C. Agbo, “The effects of vaccination, antibiotic and vitamin therapy on some clinical parameters associated with natural outbreak of fowlpox in chickens”, *Journal of Veterinary Medical and Animal Health*, vol. 4(9), pp. 136-139, 2012.
- [29] E. C. Okwor, D. C. Eze and K. F. Chah, “Recurring outbreaks of Fowl pox in a poultry farm in Nsukka, Southeast Nigeria”, *IOSR Journal of Agriculture and Veterinary Science*, vol. 7(4), pp. 104 – 108, 2014.
- [30] V. Srinivasan and D. N. Tripathy, “The DNA repair enzyme CPD photolyases restores the infectivity of UV- damaged fowl pox virus isolated from infected scabs of chickens”, *Veterinary Microbiology*, vol. 108, pp. 215 – 223, 2005.
- [31] M. G. Yoshikawa and J. Alam, “Histopathological studies of fowl pox in bantams”, *International Journal of Poultry Science*, vol. 1(6), pp. 197 – 199, 2002.
- [32] H. Proctor and I. Owens, “Mites and birds: Diversity, parasitism and coevolution”, *Trends in Ecology and Evolution*, vol. 15, pp. 358 – 364, 2000.
- [33] F. M. Medina, G. A. Ramírez and A. Hernández, “Avian Pox in White-tailed laurel-pigeons from the Canary Islands”, *Journal of Wildlife*

- 
- Diseases*, vol. 40(2), pp. 351 – 355, 2004.
- [34] E. M. Odoya, A. Abegunde, B. G. Agyogbo, S. O. Omotainse, E. Gwankat and U. G. Okpara, “Outbreak of turkey pox disease in fowl pox vaccinated poult in Vom Plateau State of Nigeria”, *African Journal of Clinical and Experimental Microbiology*, pp. Vol. 7(2), pp. 136 – 138, 2006.
- [35] M. C. Adebajo, S. I. Ademola and Oluwaseun, A. “Seroprevalence of fowl pox antibody in indigenous chickens in Jos North and South Council Areas of Plateau State, Nigeria: implication for vector vaccine”, *ISRN Veterinary Science*, 2012.
- [36] S. N. Masola, A. Mzula, H. N. Tuntufye, C. J. Kasanga and P. N. Wambura, “Isolation and molecular biological characterization of fowl pox virus from specimen of cutaneous nodular lesions from chickens in several regions of Tanzania”, *British Microbiology Research Journal*, vol. 4(7), pp. 759 – 771, 2014.
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