Sero-Evidence of Infectious Bursal Disease Transmission between Quail (Coturnix coturnix japonica) and Chicken (Gallus gallus domesticus)

SONFADA, M.L.1, EL-YUGUDA, A.A.2, GULANI, I.3, RABO, J.S.4, HENA, S.A.1 and DANMAIGORO, A.1

INTRODUCTION

Chicken have a high potential of becoming a significant source of income and self-sufficiency in meat production in many developing countries (Akoma and Baba, 1995; Adene, 1997; Okoye et al., 1999). The Japanese quail is a small avian species kept for meat and egg production (Minvielle, 1998; Baumgartner, 1994). It is widely being used as a model in research in a variety of disciplines including physiology, nutrition, endocrinology, pathology, embryology, reproduction and immunology (Tilgar et al., 2008; Vatsalya and Arora, 2011).

Infectious Bursal Disease (IBD) is a highly contagious immunosuppressive disease (Kasanga et al., 2008). Chickens between 3 and 6 weeks of age are most susceptible to IBD virus (Kenton, 2008). The disease was first described in 1962 by Cosgrove (Cosgrove, 1962).

The economic losses resulting from infectious diseases such as IBD include not only the heavy mortalities but also the immunosuppression precipitated by damage to the bursa of Fabricius in survivors and sub-clinically infected birds which result in increased susceptibility to other diseases (Akoma and Baba, 1995; Kataria et al., 1998; Oyeduntan and Durojaiye, 1999). Though, Japanese quails are fairly resistant to diseases and also show strong resistance to IBD viral infection (Haruna et al., 1997; Whyte et
al., 1999; Sonfada et al., 2014), the bird could serve as a reservoir of some disease.

As one of the measures aiming at the control of infectious bursal disease, various methods have been developed for its diagnosis such as virus isolation in cell culture, embryonated chicken eggs, or young specific-pathogen-free (SPF) chickens and localization of the virus in infected tissues by electron microscopy, fluorescence assay, antigen-capture enzyme-linked immunosorbent assay (ELISA), or immunohistochemistry. All these methods may have disadvantages, such as being time consuming, labor intensive, expensive, or nonspecific (Barlic-Maganja et al., 2002). It is thus of importance to establish serological evidence of the transmission or infectivity of the disease among chickens and quails as they are commonly raised either together or in close association with each other by some peasant farmers in Nigeria.

**MATERIALS and METHODS**

One hundred and fifty (150) day-old, non-vaccinated quail chicks were obtained from a hatchery at Maiduguri, Borno State. Chickens and quails were bought and managed on deep litter system. Two 100-watt electric bulbs and additional four bush lamps were used alternatively to provide warmth to the birds. The birds were fed *ad libitum*; they were fed with broiler starter from Sander's Company (ChickMash®, Sander's Company) for 5 weeks thereafter grower's mash from the same company was fed.

The quail chicks were raised for two weeks during which six (6) were lost due to management factors, the remaining birds were then divided into two groups (group A and B) with group A having 70 birds and B having 74 birds, these birds were kept separately in different pens, located farther apart.

The group B birds were inoculated per os using a Pasteur pipette, by giving them two drops (0.1ml) of infectious bursal disease viral antigen that was prepared by maceration of bursae from IBD diagnosed birds. The birds were observed for 14 weeks; similarly those in group A were inoculated with two drops of phosphate buffered saline per os, and kept for 14 weeks. In order to test for contact infection, 20 four weeks old non vaccinated cockerel chicks were put together with IBD virus inoculated quails, sharing the same feeders and waterers. They were monitored and observed for symptoms and clinical signs for IBD.

At weekly interval, five birds were randomly sampled from each group (A and B) of the two species and weighed using a beam balance and sacrificed using halal method of slaughter (Anil et al., 2004). The blood from each slaughtered bird was collected for serology.

Agar gel was prepared to carry out the Agar Gel Precipitin Test (AGPT). The central well was filled with the antigen (bursal homogenates) using pastuer pipette. One peripheral well was filled with drops of the test serum samples (one well to one test sample) using a single channel pipette. A similar procedure was adopted for the remaining group of wells. The plates were thereafter incubated in a tray for 24 – 48 hrs at room temperature under moist condition. A 60 watt light bulb was used to illuminate the petri dish in aiding the test to be read accurately. Positive samples showed precipitin lines and were seen between the samples and viral antigen wells. The intensity of reaction was recorded and interpreted following the standard technique outline by Hirai et al., (1972) as:

i. **–** ve: no visible band or line of precipitin

ii. **+**ve: faint, but recognizable diffused precipitin line

iii. **++**: distinct band of moderate opacity and varying width

iv. **+++**: very opaque, of varying width.
RESULTS
The results obtained in this work are as presented in the table and plates below.

**TABLE I: DISTRIBUTION OF PRECIPITIN ANTIBODIES AGAINST IBDV IN CHICKENS AND QUAILS**

<table>
<thead>
<tr>
<th>Species</th>
<th>Number Tested</th>
<th>Number Positive (+ve)</th>
<th>Number Negative (-ve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chickens</td>
<td>20</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Quail</td>
<td>144</td>
<td>74</td>
<td>70</td>
</tr>
</tbody>
</table>

**Fig 1:** Infected quails (B) mixed with non-vaccinated, non-infected chickens (A) x125.

**Fig 2:** AGPT plate showing positive precipitin line (arrow) and negative wells (N) x125.

The chickens mixed with infected quail did not show any clinical sign of the disease (IBD) however, they were serologically positive for IBD. The precipitin line which indicates the positivity of the tested sera is shown in plate II, and in which the negative result is also shown. All the infected birds tested positive serologically. Histopathologically, the infected quail showed hyperplasia of the lymphocytes (Plate IV), while those of the infected chicken showed evidence of lymphocytolysis though slight (Plate III).
Fig 3: A chicken bursa of Fabricius showing increasing number and size of lymphoid follicles (a); less prominent connective tissue septae (b), and slight lymphocytolysis (arrow) H&E  X 100.

Fig 4: Quail bursa showing: Follicular hyperplasia (H). H&E x100.
The infected quail did not show any clinical sign of the disease (IBD). Histologically, the bursa of Fabricius of the infected quail showed hyperplasia of the lymphoid follicles (Plate IV).

DISCUSSION

The cross infection between the quails and the contact chicken showed a different disease pattern. There was no mortality experienced in the contact chickens through infected quails, because there are no clinical manifestation of the disease among the birds. However, they were all serologically strongly positive, as all the infected quails as well as cross-infected contact chickens reacted positively for the presence of infection as shown in the results. Haruna et al., (1997) and Whyte et al., (1999) observed that quails are fairly resistant to viral infection so there are less need for vaccination. The observation in this study is however in contrast.

Infectious bursal disease (IBD) is an acute contagious viral disease of young chickens (Kibenge et al., 1988; Lasher and Shane, 1994). The aetiological agent, IBD virus (IBDV), has a predilection for the cells of the bursa of Fabricius where the virus infects actively dividing and differentiating lymphocytes of the B-cell lineage (Burkhardt and Muller, 1987). Increase in bursal size and weight as observed in this study coupled with lymphoid hyperplasia showed that the organ responded to the viral challenge. The IBD-virus destroys lymphocytes, primarily in the bursa of Fabricius, but also in other organs of the immune system, like the thymus, the spleen and the caecal tonsils. The result is a marked immune-suppressive effect, causing increased susceptibility to other diseases and impaired response to many vaccinations (Katariya et al., 1998). Clinical and sub-clinical types of the disease may occur. However, in this study, none of these clinical signs were observed in both the infected quails and cross infected chicken.

Because vaccination is the principal method of viral disease control in commercial poultry worldwide (Lasher and Shane, 1994), IBDV is being considered as one of the most important viral pathogens of the commercial poultry industry and hence appropriate means of diagnosis and control of the disease should continue to be put into place. It has been discovered in the course of this work that serological means of diagnosis is one of the simple and accurate diagnostic procedures which can not only yield quick result but may even detect a subclinical infection among birds, which may ultimately lead to timely intervention in curtailing the disease spread. Above all the technique does not need sophisticated materials. In this work the chickens mixed with unexposed quail did not show any clinical sign of the disease (IBD) or any sign of infection serologically. Farmers are therefore advised strongly to vaccinate quails as well, so as to avoid circumstance of playing a role of reservoir host thereby shedding the viral loads to other susceptible animals.

REFERENCES


