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The Differential Susceptibility of Yoruba Ecotype Nigerian Indigenous Chicken Varieties To Newcastle Disease

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ABSTRACT
This experiment was conducted to assess and compare the susceptibility of the varieties of Yoruba indigenous chicken ecotype: Yoruba Smooth Feathered (YSF), Yoruba Frizzled Feathered (YFF) and Yoruba Naked Neck (YNN) to Newcastle disease. A total of sixty (60) 16 weeks old chickens were used comprising of twenty (20) chickens per variety. The assessment of their susceptibility to Newcastle disease was by evaluation of clinical signs, humoral response, mortality, pathology following contact with Newcastle disease infected chickens. The experimental chickens developed clinical signs of Newcastle disease from day 9 after infection. Haemagglutination inhibition (HI) titre was determined on days 0, 21 and 28 post-infection. On day 0, the HI titre of the 3 genotypes were below 3log2, on day 21, there were significant differences within the group (P<0.05) where in YNN had the highest mean HI titre of 7.5. There was decrease in the mean HI titre in all on day 28 with the YNN having the least reduction (P<0.05). The antibody titre against Newcastle disease was higher in the YNN than all the other varieties. The proventricular, enteric and caecal tonsilar haemorrhages associated with the disease were more frequent and severe in YSF. This finding indicated that YFF and YSF may possibly be more susceptible to Newcastle disease than the YNN. It was concluded that indigenous chickens of Yoruba ecotype in Nigeria differ in their susceptibility to ND with YNN being possibly the most resistant to Newcastle disease followed by YFF and the least, YSF. Keywords: Haemagglutination inhibition, Newcastle Disease, indigenous Chicken, Ecotype, Varieties

INTRODUCTION
Rural poultry is a major source of readily available protein in the form of meat and eggs, as well as cash money for about 90% of the rural household in African countries (Mbugua, 1990). In rural areas where subsistence peasantry farming is the main occupation, the indigenous chicken assumes great importance economically, nutritionally and socially (Minga et al., 1989). According to Sonaiya (1992) more than 80% of the poultry population is found in the rural household. These animals are known for their adaptation, probable superiority in terms of resistance to endemic diseases and other unfriendly environmental condition (Nwakpu et al., 1999, Fayeye et al., 2006). The indigenous chicken has remained largely genetically uncharacterized and unimproved (Oluyemi and Roberts 2000).
They assumed various names such as local chicken, the family poultry, the traditional chicken and the backyard chicken. The indigenous chicken in the developing world has not been subjected to purposive selective breeding for any particular trait instead they have been subjected to natural selection imposed by endemic diseases, climate, nutrition and other stresses. This has created diversity in plumage type and colour, productivity, and body size. This immense biodiversity has ensured their survival in diverse ecological zones by naturally being selected for survival fitness. Certain major genes have been found potentially useful to the tropical production environment either because of their direct effect on production or because of their indirect effect on quantitative trait loci. Among these major genes with indirect effects are the feather distribution (Naked neck, Na) and feather structure (Frizzle gene). In Nigeria, Sonaiya and Olori (1989) reported that of the rural chickens, 75% were the smooth multicoloured type, 15% had frizzled feathers, 6% had naked necks and 4% were the dwarf chickens. The major production constraint in free-ranging local chickens in developing countries had been disease with Newcastle disease (ND) being ranked as the number one killer disease of free-ranging local chickens (Minga et al., 1989). Fowl typhoid and Newcastle disease (ND) were the most prevalent disease of indigenous chicken (Minga et al., 1989, Ohore et al., 2002). Newcastle Disease (ND) is an acute, mild to severe, highly infectious and pathogenic disease of domestic poultry, caged and aviary birds as well as wild birds (Sonaiya, 2009). The outbreaks of this disease were more common in layers (Abdu et al., 2005b) and during the dry harmattan (November-March) (Saidu et al., 1994; Halle et al., 1999; Abdu et al., 2005a). Clinical signs associated include: sudden drop in egg production often accompanied by production of abnormal eggs, loss of appetite, fever, weakness; respiratory signs, which may include increased respiratory rate, respiratory distress, coughing and a high-pitched sneeze, nervous signs, which include loss of balance, circling, backward progression and convulsive, stiff and wry neck, wing and leg paralysis.

Investigation into the various poultry diseases commonly encountered in indigenous chicken had been the focus of research for quite some time, however fewer investigations have been conducted to establish the disease resistance within the Nigerian indigenous chicken especially the varieties within the ecotype. Testing of disease resistance potential can be direct by infecting the host with the virulent pathogen (Okoye and Aba-Adulugba 1998, Mdegela et al., 2002) directly or by comingling, and response of the host towards the pathogen is evaluated.

Disease resistance is a trait often controlled by multiple genes as well as interactions between several factors (Hartmann, 1997). The chicken Major Histocompatibility complex (MHC) is one genetic system which control disease resistance (Bacon, 1987). In chickens the MHC is called the B complex; it was originally described as a system controlling blood group antigen (Briles et al., 1950). One of the most interesting features of the chicken MHC is the strong influence it exerts on resistance to a variety of viral, parasitic, and bacterial diseases (Bacon, 1987; Lamont, 1998). MHC control of disease resistance has been established for Marek's disease (MD) (Briles et al., 1983) with very little information on the possible disease resistance associated with Nigerian indigenous chicken. The need to assess
and compare resistance to Newcastle disease within varieties of Yoruba ecotype of Nigerian indigenous is paramount as improvement of the indigenous chicken is an essential tool for poverty alleviation.

**MATERIALS AND METHODS**

**Experimental Site:** The experiment was carried out at the Teaching and Research Farm, University of Ibadan, Ibadan, Nigeria. The site is located on latitude 7°20’N, 3°50’E, 200m above sea level.

**Experimental Chicken:** Twenty 16 weeks old indigenous chickens each from YSF, YFF and YNN were used for the experiment. On arrival, the chickens were given a dose of antibiotics and anticoccidal in accordance with the manufacturer recommendation. The chickens were put on deep litter and fed commercial layers mash containing 16% crude protein and 2600MEKcal/kg with water ad libitum.

**Experimental Design:** Completely randomized designed (CRD) was used. The chickens were allocated into three treatments of YSF, YFF and YNN, having four replicates each of five birds.

**Inoculation:** Each bird was naturally exposed to Newcastle disease by coming in contact with already infected chickens which were mixed with them in their different compartments.

**Degree of Disease Susceptibility**

**Clinical Signs and Mortality:** The chicks were observed twice daily for clinical signs and mortality up to 28 days post-infection. (-) was assigned for no clinical signs noticed, (+) was assigned for depression, ruffled feather, loss of appetite and weakness, (++) was assigned for nervous signs, which include loss of balance, circling, backward progression and convulsive, stiff and wry neck, wing and leg paralysis. Percentage mortality was calculated using:

\[
\text{\% Mortality} = \left( \frac{\text{Number of dead birds}}{\text{Total number of birds}} \right) \times 100
\]

**Blood Sampling and determination of Newcastle Disease Antibody Titres:**

Before infection and on days 21, 28 post infection, blood samples was collected from randomly selected birds and Newcastle disease antibodies titre were determined. Haemagglutination Inhibition (HI) test was used to determine specific antibodies against Newcastle disease virus by the method described by Allan and Gough (1974).

**Pathology**

The lesion observed in the proventriculus, jejunum, cecal tonsils, lungs and follicles were graded; (-) was assigned for no obvious lesion, (+) was assigned for mild lesion, (++) was assigned for moderate while (+++) for severe lesion.

**Statistical Analysis:** All data collected from the experiment were analysed using one way analysis of variance procedure of Statistical Analytical System (SAS, 1990).

\[
Y_{ij} = \mu + \alpha_i + e_{ij}
\]

Where \( Y_{ij} \) = Individual observation assumed to be random elements
\( \mu \) = Population means fixed and unknown, \( \alpha_i \) = Treatment effect effect of ecotype varieties assumed fixed
\( e_{ij} \) = error associated with each record assumed random and normally distributed.

**RESULTS**

**Clinical Signs:**

The clinical signs observed in each variety are presently in Table 1, on day nine post-infection (PI), eight out of twenty chickens from YSF showed depression, ruffled
feathers, loss of appetite and weakness while other did not show any clinical signs. On day ten, clinical signs were observed in all the three varieties in which nine out of twenty from YFF and seven out of twenty chickens from YNN showed clinical signs mentioned above. Four YFF chickens showed nervous signs which include loss of balance, circling and backward progression on day eleven PI. On day twelve and thirteen PI, four from YFF, two from YNN showed the same clinical signs respectively. Signs were observed in YNN and YFF till seventeenth day post-infection while in YSF continued up to the eighteenth day. No clinical sign was observed in the three varieties from day nineteen till the end of the experiment.

**Antibody Titre:**
Table 2 shows the mean HI titres (expressed as log 2<sup>+</sup>). In the three genotypes, it was observed that on day zero before inoculation, there was similarity in the mean HI titres and they were below 3log2. On day 21 post infection YSF had the least HI titre mean (4.75) which differed significantly P < 0.05 from YNN but similar to YFF. The YNN had the highest mean HI titre (7.5) similar to YFF mean HI titre (6.25) but differed significantly P < 0.05 from YSF. On day 28 post infection there was slight decrease in the antibody titre and of the three genotypes and still YNN had significant higher (p<0.05) HI titre than the other genotypes.

### Table 1: Clinical signs of the infected chickens

<table>
<thead>
<tr>
<th>Days post infection</th>
<th>YSF</th>
<th>YFF</th>
<th>YNN</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 8</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>+ (8)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>+ (14)</td>
<td>+ (9)</td>
<td>+ (7)</td>
</tr>
<tr>
<td>11</td>
<td>+ (12) ++ (4)</td>
<td>+ (11)</td>
<td>+ (8)</td>
</tr>
<tr>
<td>12</td>
<td>+ (13) ++ (5)</td>
<td>+ (7) ++ (4)</td>
<td>+ (9)</td>
</tr>
<tr>
<td>13</td>
<td>+ (9) ++(4)</td>
<td>+ (10) ++(5)</td>
<td>+ (8) ++(2)</td>
</tr>
<tr>
<td>14</td>
<td>+ (5) ++(5)</td>
<td>+ (7) ++ (4)</td>
<td>+ (3) ++(3)</td>
</tr>
<tr>
<td>15</td>
<td>+ (5)</td>
<td>+ (6) ++ (2)</td>
<td>+ (5)</td>
</tr>
<tr>
<td>16</td>
<td>+ (3)</td>
<td>+ (5)</td>
<td>+ (4)</td>
</tr>
<tr>
<td>17</td>
<td>+ (1)</td>
<td>+ (2)</td>
<td>+ (1)</td>
</tr>
<tr>
<td>18</td>
<td>+ (1)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>19</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>20</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>21</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

(-) : no clinical signs noticed
(+): respiratory signs (depression, ruffled feathers, loss of appetite and weakness)
(+ +): nervous signs (twisting of head and neck, circling and loss of balance)
YSF= Yoruba Smooth Feathered chicken
YFF= Yoruba Frizzled Feathered chicken
YNN=Yoruba Naked neck chicken
Table 11: Hemagglutination Inhibition Antibody Titres (GMT) of the infected chickens

<table>
<thead>
<tr>
<th>Days post infection</th>
<th>Genotypes</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>YSF</td>
<td>YFF</td>
</tr>
<tr>
<td>0</td>
<td>1.00</td>
<td>1.50</td>
</tr>
<tr>
<td>21</td>
<td>4.75\textsuperscript{b}</td>
<td>6.25\textsuperscript{a}</td>
</tr>
<tr>
<td>28</td>
<td>4.25\textsuperscript{b}</td>
<td>5.73\textsuperscript{b}</td>
</tr>
</tbody>
</table>

Means in the same row with different superscripts are significantly (P<0.05) different from each other.

Mortality and Necropsy findings:
Table 3 shows mortality pattern of the experimental chickens, mortality started on day ten post-infection, there were two chickens from YSF and one from YNN while there was none in YFF. On day eleven there were five in YSF, four in YFF and two in YNN. Mortality continued up to 13\textsuperscript{th} day post-infection in both YSF and YFF but there was none in YNN. In table 4 % mortality in YNN was the least (30) followed by YFF (40) and YSF had the highest % mortality (70). All the dead chickens were examined and the observations were presented in table 4.

Table 11: Daily mortality of the infected chickens

<table>
<thead>
<tr>
<th>Days post infection</th>
<th>YSF</th>
<th>YFF</th>
<th>YNN</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 9</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>5</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>13</td>
<td>4</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>14 to 21</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>40%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Table 1V: Pathology score of the infected chickens

<table>
<thead>
<tr>
<th>Varieties</th>
<th>Lung consolidation</th>
<th>Enteric haemorrhage</th>
<th>Proventriclar Haemorrhage</th>
<th>Follicular atresia</th>
</tr>
</thead>
<tbody>
<tr>
<td>YSF</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>YFF</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>YNN</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
DISCUSSION
This investigation further showed that indigenous chicken including the various varieties are susceptible to ND which further substantiated the report of Okoye and Aba Adulugba (1998) that this group of chicken do experience fulminating ND. In this experiment, the clinical signs were observed in all the three groups of chicken from day nine up to eighteen post-infection with severe signs observed in YSF. It also showed that comingling infected chicken with uninfected can result to infection, this observation further showed the possible means of transmission of the virus through aerosol or contact. Six of the YSF chickens, twelve of the YFF chickens, fourteen of the YNN chickens, some of which showed nervous signs survived the infection, which is in agreement with the literature that birds showing nervous signs have a tendency to recover and there is variation in susceptibility within the ecotype (Yongolo 1990; Bell 1992).

From this study, chickens with major genes recovered faster than the YSF and mortality was 30% to 40% but in YSF it was 70%. This low mortality could be attributed to major gene effect which could have helped the chicken to recover faster and combat the virus. The findings corroborated the work of Auernez et al.(2003) who reported that Mexican indigenous naked chicken showed higher survival rate than the commercial line. Based on the major gene effect, it could therefore be reasonably assumed that Naked Neck gene contributed to the resistance to disease than the Frizzle gene.

The mean HI titres for the three genotypes on day zero before infection were below 3log2 which had been reported to be protective (Allan and Gough, 1974, Musa et al. 2009) which showed that local chickens are also susceptible to Newcastle disease, the number one killer of the free range local chickens in Africa (Minga et al., 1989; Bell 1992; Yongolo 1996). The mean HI titre on day 21 post-infection (Table4) shows that the three genotypes had mean HI titre >3log2, this indicates that the survived chickens seroconverted and had attained the protective level. This observation corroborated the reports of Mtambo et al. (1999) and Waihenga et al. (2002) that high levels of HI titre contributed towards the recovery of the infected birds. The significant difference in the HI titres (P<0.05) at day 21 between YSF and YNN is an evidence that the different major genes within Yoruba Ecotype differ in their humoral response to the viral challenge which in turn account for the differing disease resistance capability. The difference in percentage mortality between YSF (70) and YNN (30) further reiterate this observation. The differing susceptibility and immunological responsiveness to Newcastle disease vaccination among ecotypes had also been reported by Gwakisa et al. (1994) who tested four Ecotypes namely Arusha, Coast, Mbeja, Singida and Rhode Island Red where they found some to be high responders while other are low responders. Msoffe et al. (2001) also postulated greater variability in immune response within Ecotypes than between Ecotypes. The similarity between YNN and YFF was indicated by the high immune response further suggests a genetic influence. This variation was also observed in the rate of decay of antibody as observed on day 28 post-infection with a decrease in the HI titre level with YNN chicken having the least. Although there have been differences in the opinion as to whether there is correlation between disease resistance and major genes, this investigation further showed that there is variation in disease resistance of major
genes within Yoruba Ecotype.

In conclusion, the current study revealed that variation in resistance to Newcastle disease exists among major genes within Yoruba Ecotype of the Nigerian indigenous chicken with YNN chickens having the least mortality rate and highest mean HI titre hence are superior in terms of resistance to Newcastle disease. Hence it will be advantageous if these major genes that are gradually going into extinction can be conserved for future breeding programme. A cross-breeding between YNN chicken and YFF chicken may give better performance in terms of resistance to Newcastle disease.

Further work is required in order to ascertain the basis and mechanisms of this observed resistance using genetic method of characterisation such as Major Histocompatibility Complex (MHC) Typing and identification of Quantitative Trait Loci (QTLs).

REFERENCES


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