

Improving the Health of Ethiopian Indigenous Chickens under Confinement



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Research Report 69

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Ethiopian Institute of Agricultural Research

Improving The health of Ethiopian Indigenous Chickens under Confinement

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Introduction

For a large number of people living in rural areas, poultry contribute the major source of livelihood in bridging some socioeconomic gaps by supplying food and income and hence should be included in rural development strategies. Such bridging role of poultry is attributed to its fast generation interval and high reproductive rate, prolific nature, easy to raise and their output can be generally expanded more rapidly and easily than that of other livestock.

Ethiopian has 65 million poultry population (FAO, 2000) that comprise of about 99 % of indigenous chickens (Alamargot, 1987). Indigenous chickens in Ethiopia are predominantly raised where traditional family-based free-range scavenging management system is practiced (Alemu and Tadelle, 1997). Thus, the birds are left to depend primarily on what nature offers.

Indigenous chickens are constrained by inadequate information at all stages of research and development. Very limited information is available from the scattered efforts of research and development concerning these genetic resources. Thus, what has been studied so far on these animals is not tangible enough to show the relative effects of genetic and non-genetic factors on performance of indigenous chickens (Alemu and Tadelle, 1997). The cause for inadequate research and development efforts on poultry in Ethiopia could be associated to the lack of priority in development agenda. As a result, efforts on these animals have long been interplayed between “on and off” until poultry research is recognized and established as one of the national research commodity to date.

Generally, despite the fact that indigenous chickens are huge in population and occasionally they have been considered to be disease resistant and adaptive to their environment, their contribution to human nutrition, gross domestic products, and export earnings are

disproportionately low. Such sub-optimal production has been related to delivery of a low standard of management, health care and feeding. There is a general tendency that improvement in management, health care and nutritional status of indigenous chickens could result in increased output per bird. Thus, there were attempts to rear indigenous chickens under confined management with improved management, health care and nutritional status in various geographical areas of the country (Teketel, 1986; Abebe, 1992; Brannang and Pearson, 1990; Solomon, 2003), but all have failed due to serious problem of high morbidities and mortalities.

The cause of failure of native chickens to survive under confined environment as repeatedly proved from high morbidity and mortality is not adequately addressed yet. Therefore, this study was intended to describe the etiology for unsuccessful survival of the chickens under confined management. The study also assessed the prevalence, clinical and pathological manifestations and extent of mortality, mean length of survival, mortality pattern, and incidence of mortality of five chicken ecotypes during the diagnostic phase. The extent and effectiveness of vaccination in reducing mortality to the natural challenge of Marek's disease (MD) during intervention phase was also investigated.

Materials and Methods

Study Sites

The study was conducted at the Debre Zeit Agriculture Research Center 45km southeast of Addis Ababa. The site is located at an altitude of 1900m above sea level. The area receives an average annual rainfall of 851mm and a minimum and maximum temperature of 8.9°C and 26.2°C, respectively. The average humidity level of the site is 58.6 %.

Acquiring Study Animals

During the diagnostic phase eggs of indigenous chickens were purchased from different geographical areas of the country: Tilili, Horro, Tepi, Konso, and Jarso located in the northwest, west, southwest, south, and east of the country, respectively. The chickens were named after their areas of origin. Local eggs collected from Horro, Konso and Jarso were transported to Debre Zeit Agricultural Research Center for hatching. Eggs from Tepi and Tilili were hatched at the Jimma Agricultural College and Andasa Agricultural Research Center, respectively and the chicks were transported to the Debre Zeit Agricultural Research Center at day-old. Fayoumi eggs were collected and hatched from the poultry farm of Debre Zeit Agricultural Research Center. During Marek's Disease (MD) intervention phase, eggs of indigenous chickens were purchased from Horro, Tepi, and Jarso. The chickens were named after their area of origin. The eggs were collected and transported to Debre Zeit Agricultural Research Center for hatching. Fayoumi eggs were collected and hatched from the poultry farm of Debre Zeit Agricultural Research Center.

Study Design

The study was designed and undertaken in two broadly separated but related phases. The first phase was termed as diagnostic phase whereby in-depth investigation was conducted on Tilili, Horro, Tepi, Konso and Jarso ecotypes and Fayoumi to diagnose the cause of high morbidities and mortalities of the indigenous chickens kept under confinement regime.

The second phase was adherent to the first phase and termed as the intervention phase to respond to the diagnosed etiology. In this phase, the study involved four chicken types (Horro, Tepi, and Jarso ecotypes and Fayoumi) that were grouped in to MD vaccinated and non-vaccinated group. In both phases (diagnostic and intervention phase) data on mortality parameter was collected from chickens until 21 weeks of age daily.

Management of Animals during Diagnostic Phase

The collected eggs were selected for physical quality, fumigated for hygiene with 17g potassium permanganate + 100ml of 20% formalin, and incubated for hatching. All chickens were vaccinated against Newcastle disease with (HB1 and LaSota) in accordance with the producer's recommendation and transferred to clean and disinfected brooder house. The house was bedded with tef straw and chicks were placed in pens hatched with infrared bulbs. The baby-chicks were supplied with starter ration and clean potable water. The chicks were fed a commercial starter ration during the brooding period (starter phase), which lasted for 2 months. After the end of the starter phase, the chickens were transferred to a grower ration for about 3 months. Antibiotics and feed additives (vitamins) were supplied for all chick flocks under study when disease was suspected in a pen.

Diagnosis Procedures

Clinical and necropsy examination, Histopathology, Virology and serology techniques were employed to identify the causative agent of high morbidities and mortalities of indigenous chickens under confined management regime.

Clinical and necropsy examination

Daily follow-up was made to the chicken flocks during the study period (November 2003 to January 2004). Clinical observation on disease signs and symptoms observed in sick birds during the study were closely followed, examined, and noted. A macroscopic post mortem (necropsy) examination was conducted on dead birds during the study period. The most obvious abnormality or lesion observed on a number of body organs and/or tissues was examined and recorded.

Histopathology

Sick chickens were submitted to the Pathology Section of the National Animal Health Research Center, Sebeta. Tissue samples were collected according to (Luna, 1968) in 10 % buffered formalin, dehydrated in alcohol, embedded in paraffin, sectioned into 4-5 μm thick sections, and stained with haematoxylin-eosin. The stained samples were examined by light microscope for histological changes.

Virology and serology

Blood samples were collected according to (Alders and Spradbrow, 2001) and submitted to National Veterinary Institute that is located at Debre Zeit. Viral agent isolation from buffy coat cells of centrifuged blood cell samples was done according to (OIE, 1994) by inoculating 0.2ml of sample suspension into monolayer cultures of chicken kidney cells and incubated at 38.5°C in a humidified incubator containing 5% CO₂ and areas cytopathic effects (plaques) were examined for 7 days.

Similarly, blood samples for serological tests were collected from wing veins according to (Alders and Spradbrow, 2001) and the sera

submitted to the National Veterinary Institute. Agar-gel immunodiffusion (AGID) test was employed on serum samples. The test was undertaken according to (OIE, 1994) using Petri dishes coated with 1% Noble agar in phosphate buffered saline containing 8% sodium chloride by filling adjacent wells with test serum and the center well with standard antigen and kept in an incubator at 37°C for 24 hours. The development of radial zones of precipitation around the test serum denotes the presence of antibody in the serum and hence of MD infection of the chickens.

Management of Animals during MD Intervention Phase

The intervention phase was investigated from July 2005 to January 2006 at Debre Zeit Agricultural Research Center in central Ethiopia. The hatchery room was cleaned and disinfected with 1% formalin spray 3 hours before the arrival of the eggs. The eggs were selected for physical quality, fumigated for hygiene with 17g potassium permanganate + 100ml of 20% formalin, and incubated for hatching. Three hours before transfer of eggs from the setter to the hatcher and before each candling 1% formalin was sprayed in the hatchery room to disinfect and avoid infection of the ovo with pathogenic MD virus while in the hatchery. Egg candling was undertaken at 7 and 18 days of setting.

All chickens were vaccinated against Newcastle disease with HB1 at 1st day and LaSota at 21st, 56th days and every 6 months of life in accordance with the producer's recommendation. MD vaccine (1ml) was given on the neck subcutaneously to 96 Fayoumi, 222 Horro, 90 Tepi, and 234 Jarso chickens and equal number of chickens from each type received the same management except lack of MD vaccination to serve as a control.

The poultry house both brooder and grower house with all poultry equipments and beddings were disinfected by 2% formalin one day before the introduction of the chickens. The house was bedded with

Teff straw and had infrared bulbs for heating. The baby-chicks were supplied with starter ration and clean potable water. The chicks were fed a commercial starter ration during the brooding period (starter phase), which lasted for 2 months. After the end of the starter phase, the chickens were transferred to a grower house where they fed a grower ration for about 3 months. Antibiotics and vitamins were supplied for all chick flocks under study when disease was suspected in a pen.

Data Collection and Analysis

Daily records of mortality data from indigenous chicken ecotypes (381 Jarso, 233 Konso, 261 Tepi, 441 Tilili, and 400 Horro) and from exotic chicken (401 Fayoumi chicks) were collected for 21 weeks of life to describe the extent of mortality at different phases and pattern of mortality and length of survival in weeks. Daily mortality data during 14 to 20 weeks were collected from 503 indigenous chickens; Jarso (32), Konso (84), Tepi (88), Tilili (151) and Horro (148) to describe the manifestation of clinical signs. Fatality rate was analyzed from 340 indigenous chickens; Jarso (28), Konso (52), Tepi (67), Tilili (92) and Horro (101). Data for serology was collected from 70 native chickens; Jarso (13), Konso (19), Tepi (11), Tilili (14) and Horro (13) and assessed.

During the intervention phase, daily data were collected from 546 indigenous chickens (234 Jarso, 90 Tepi, and 222 Horro) and 96 Fayoumi that were vaccinated against MD subcutaneously at neck region (1ml/bird) to investigate the extent and effectiveness of MD in reducing morbidity and mortality. Parallel daily mortality data were also collected from equal number of chickens from each ecotype that was kept without MD vaccination as a control.

Mortality prevalence was calculated by dividing the number of animals died by the total number of animals present at day-old. Incidence mortality rate was calculated by dividing the number of new animals died in each week by the total number of animals present in that particular week. **Fatality rate** was calculated by dividing the number of animals died by the total number of sick animals examined. The susceptibility and resistance rate to natural MD challenge was measured by mortality rate. The response of the chickens to MD vaccination was measured by improvement in mortality rate. Measurement of association between mortality prevalence and chicken genotypes, chicken genotype susceptibility/resistance to natural MD challenge and vaccination trial was tested using chi-square (χ^2). Thus, the data were analyzed using simple descriptive statistics and a chi-square test at 95 % confidence interval ($\alpha=5\%$). The Version 12 SPSS software (SPSS Inc., Chicago, Ill, USA) was employed for data analysis.

Results of Diagnostic Phase

The clinical symptoms of the disease were first observed in Jarso chicken genotypes at first week of life. The morbidity, mortality, and fatality rates widely and intensely spread within and among the indigenous chicken genotypes until 14 weeks of age with subsequent ravaging death toll. The overall morbidity, MD antibody prevalence rates, mortality as well as fatality rates in the indigenous chickens over the study period were 67.9% (340/503) (Table 1), 72.9% (Table 3), 66.2% (Table 4) and 97.9% (Table 5), respectively.

Clinical Examination

In this study, an overall acute (65.2%) and chronic /classical form (34.8%) of clinical manifestation of the disease were recorded (Table 1). In this category, the major signs observed in acute form were found death without any observed clinical signs (31.5%) and severe depression (33.8%). Of the total chickens with the acute form of MD 28.8% (64/222) were observed in Horro chicken genotypes. However, in paralytic (chronic) form, clinical signs with paralysis of different body parts were observed during clinical examination and death occurred within a few days to several weeks after showing clinical signs. Of the 118 chickens with chronic (paralytic) form of the disease, studied 38.1% (45/118) were from the Tilili genotypes (Table 1).

Table 1: Manifestations and forms of clinical signs in MD infected intensively managed indigenous chicks in 2003 at Debre Zeit.

| Clinical signs | Indigenous Chicken Genotypes (N=503) | | | | | Total |
|--|--------------------------------------|-----------------|-----------------|-----------------|-----------------|------------------|
| | Jarso | Konso | Tepi | Tilili | Horro | |
| Found dead | 14(50.0) | 11(21.2) | 32(47.8) | 19(20.7) | 31(30.7) | 107(31.5) |
| Severe depression | 11(39.3) | 18(34.6) | 25(37.3) | 28(30.4) | 33(32.7) | 115(33.8) |
| Total acute form | 25(89.3) | 29(55.8) | 57(85.1) | 47(51.1) | 64(63.4) | 222(65.2) |
| Leg and wing paralysis | 1(3.6) | 5(9.6) | 2(3.0) | 15(16.3) | 11(10.9) | 34(10.0) |
| Twisted and distorted neck | - | 1(1.9) | 1(1.5) | 1(1.1) | 4(4.0) | 7(2.1) |
| Fail to stand and walk with split legs | - | 3(5.8) | 2(3.0) | 2(2.2) | 4(4.0) | 11(3.2) |
| Combination of various paralysis | 2(7.1) | 14(26.9) | 5(7.5) | 27(29.3) | 18(17.8) | 66(19.4) |
| Total chronic form | 3(10.7) | 23(44.2) | 10(14.9) | 45(49.9) | 37(36.6) | 118(34.8) |
| Total chicken observed | 28 | 52 | 67 | 92 | 101 | 340 |

Lesions

Gross lesions either in visceral organs and/or peripheral nerves indicative of lymphoid tumors was observed in all 110 chickens autopsied. The lesions in visceral organs took either nodular, diffuse, or mixed forms with varying degree in size of white or grayish coloration with deep, superficial, or mixed depth in parenchyma of the organs. They had firm consistency and smooth surfaces. Of the autopsy examination made on 110 chickens, 71.8% (79/110) showed the acute (visceral) form, 11.8% (13/110) chronic /paralytic form and 16.4% (18/110) mixed form of visceral and paralytic form. The gross lesions were seen in visceral organs including the liver 69.1% (76/110), spleen 64.5% (71/110), lung 59.1% (65/110), heart 49.1% (54/110), etc (Table 2). This shows that, birds with acute form of MD clinical signs had mainly gross lesions in visceral organs. In paralytic form, the involved nerves were pale or grayish in color enlarged in size and lost the characteristic cross-serrations when compared to the non-affected nerves.

Microscopic examination was made on brain, peripheral nerves, liver, spleen, bursa, proventriculus, and heart. In brain, microgliosis and lymphocytes perivascular cuffing were observed. In peripheral

nerves either massive or moderate infiltration of lymphocytes were recorded. In visceral organs, diffuse infiltration or aggregates of lymphocytes were noted.

Table 2: Gross organ changes during autopsy in MD infected intensively managed indigenous chickens in 2003 at Debre Zet.

| Organ/Tissue with diffuse, focal, or mixed tumorous lesions | Chicken Genotypes | | | | | Total (n=110) |
|---|-------------------|--------------|--------------|---------------|--------------|---------------|
| | Jarso (n=25) | Konso (n=20) | Tepi (n=20) | Tilili (n=25) | Horro (n=20) | |
| Liver | 19 (16.5) | 10 (12.0) | 13 (19.4) | 17 (22.7) | 17 (27.0) | 76 (69.1) |
| Spleen | 17 (14.8) | 13 (15.7) | 11 (16.4) | 16 (21.3) | 14 (22.2) | 71 (64.5) |
| Lung | 18 (15.7) | 18 (21.7) | 14 (20.9) | 9 (12.0) | 6 (9.5) | 65 (59.1) |
| Heart | 21 (18.3) | 16 (19.3) | 9 (13.4) | 5 (6.7) | 3 (4.8) | 54 (49.1) |
| Hydro- pericardium | 4 (3.5) | 2 (2.4) | - | - | 1 (1.6) | 7 (6.4) |
| Proventriculus | 8 (7.0) | 3 (3.6) | - | 3 (4.0) | 2 (3.2) | 14 (12.7) |
| Bursa | 3 (2.6) | 1 (1.2) | - | - | | 6 (5.5) |
| Viscera only | 21 (18.3) | 15 (18.1) | 16 (23.9) | 13 (17.3) | 14 (22.2) | 79 (79.8) |
| Paralysis only | 1 (0.9) | 2 (2.4) | - | 5 (6.7) | 5 (7.9) | 13 (11.8) |
| Viscera + paralysis | 3 (2.6) | 3 (3.6) | 4 (6.0) | 7 (9.3) | 1 (1.6) | 18 (16.4) |
| Total lesions | 115 (28.5) | 83 (20.6) | 67 (16.6) | 75 (18.6) | 63 (15.6) | 403 |

Serology and Virology

MD prevalence (Antibody prevalence) using agar gel immunodiffusion (AGID) test was very high, 72.9% (Table 3). In general, chickens under intensive management are not vaccinated against MD in most chicken farms of Ethiopia. However, the present study attested that MD is of comparable importance to the devastating Newcastle disease in intensive poultry farms hence deserving serious attention. The overall picture of this study showed that the extent and

forms of clinical signs (Table 1), the nature of gross lesions (Table 2) and magnitude of mortality (Table 4) among indigenous chicken genotypes were significantly different ($p < 0.01$). Once the chickens manifested clinical signs, the ultimate fate of such sick chickens was death (Table 5). However, serological results (the level of antibody prevalence to MD infection) showed non-significant difference among the chicken genotypes (Table 3). This probably indicated that despite equal chance of acquiring MD infection, the extent of disease development, i.e. becoming sick and fate of diseased individual chicken, to some extent varied with the ecotype. This may imply the presence of a degree of resistance and/or susceptibility to MD among chicken genotypes.

Table 3: MD prevalence as assessed by Agar gel immuno-diffusion (AGID) in intensively managed indigenous chicken genotypes at Debre Zeit

| Serological Event | Indigenous Chicken Genotypes | | | | | Total |
|-------------------|------------------------------|-------|------|--------|-------|-------|
| | Jarso | Konso | Tepi | Tilili | Horro | |
| Positive to MD | 10 | 12 | 9 | 12 | 8 | 51 |
| Negative to MD | 3 | 7 | 2 | 2 | 5 | 19 |
| Total | 13 | 19 | 11 | 14 | 13 | 70 |
| MD Prevalence (%) | 76.9 | 63.2 | 81.8 | 85.7 | 61.5 | 72.9 |

Mortality During 14 to 20 Weeks

The overall mortality of indigenous chickens was 66.2% (333/503). The highest mortality was observed in the Jarso genotypes, 87.5% (28/32) and the least in Tilili genotypes, 58.3% (88/151) ($p < 0.01$). Comparison of mortality among age groups showed highest mortality at 14 weeks age (14.1%=71/503) and 15 weeks age (16.1%=81/503) and then mortality pattern continued at a decreasing rate (Table 4). The reason for high magnitude of death during these ages might show the specific age group when the wave of the attack from MD reaches its climax.

Table 4: Effect of age and genotype on mortality due to MD infection under confined management

| Types | Number and percentage of Age-specific chicken Mortality (in week) | | | | | | | | | | Survivors at 20 weeks | Total chickens observed |
|-------|---|----------|----------|----------|---------|--------|---------|-----------|-----------|-----|-----------------------|-------------------------|
| | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 14-20 wks | | | | |
| Jarso | 11(34.4) | 4(12.5) | 4(12.5) | 3(9.4) | 3(9.4) | 1(3.1) | 2(6.3) | 28(87.5) | 4(12.5) | 32 | | |
| Kono | 8(9.5) | 13(15.5) | 7(8.3) | 8(9.5) | 9(10.7) | 1(1.2) | 5(6.0) | 51(60.7) | 33(39.3) | 84 | | |
| Tepi | 9(10.2) | 17(19.3) | 11(12.5) | 11(12.5) | 3(3.4) | 6(6.8) | 9(10.2) | 66(75.0) | 22(25.0) | 88 | | |
| Tiili | 18(11.9) | 21(13.9) | 14(9.3) | 20(13.2) | 1(0.7) | 9(6.0) | 5(3.3) | 88(58.3) | 63(41.7) | 151 | | |
| Horro | 25(16.9) | 26(17.6) | 19(12.8) | 8(5.4) | 8(5.4) | 9(6.1) | 5(3.4) | 100(67.6) | 48(32.4) | 148 | | |
| Total | 71(14.1) | 81(16.1) | 55(10.9) | 50(9.9) | 24(4.7) | 26(5.) | 26(5.2) | 333(66.2) | 170(33.8) | 503 | | |

Overall Fatality

An overall fatality rate in the present study was high (97.9%) (Table 5), indicating that almost all diseased (sick) chickens ultimately died.

Table 5: Effect of ecotype on fatality rate in MD infected intensively managed indigenous chickens

| Disease event | Indigenous Chicken Genotypes | | | | | |
|---------------|------------------------------|-------|------|--------|-------|-------|
| | Jarso | Konso | Tepi | Tilili | Horro | Total |
| Mortality | 28 | 51 | 66 | 88 | 100 | 333 |
| Morbidity | 28 | 52 | 67 | 92 | 101 | 340 |
| Fatality (%) | 100 | 98.1 | 98.5 | 95.7 | 99.0 | 97.9 |

Mortality during Brooding and Growing Phase

There was interaction between chicken strain and MD-related mortality. Chicken susceptibility to death from MD cause differed significantly between exotic and indigenous chicken strains and among indigenous chicken ecotypes. It was observed that mortality from MD cause was considerably higher in the indigenous chickens than the imported strains. Very high proportion of Jarso ecotypes died (55.9%), Konso (26.6%), Tepi (37.2%), Fayoumi (6.2%), Tilili (27.4%) and Horro (25.3%) during brooder phase whereas the Konso (60.1%), Tepi (54.4%), Tilili (58.3%) and Horro ecotypes (63%) died during the grower phase. Compared to indigenous chicken ecotypes mortality of Fayoumi strain was so low during both brooder (6.2%) and grower phase (8.2%) (Figure 1).

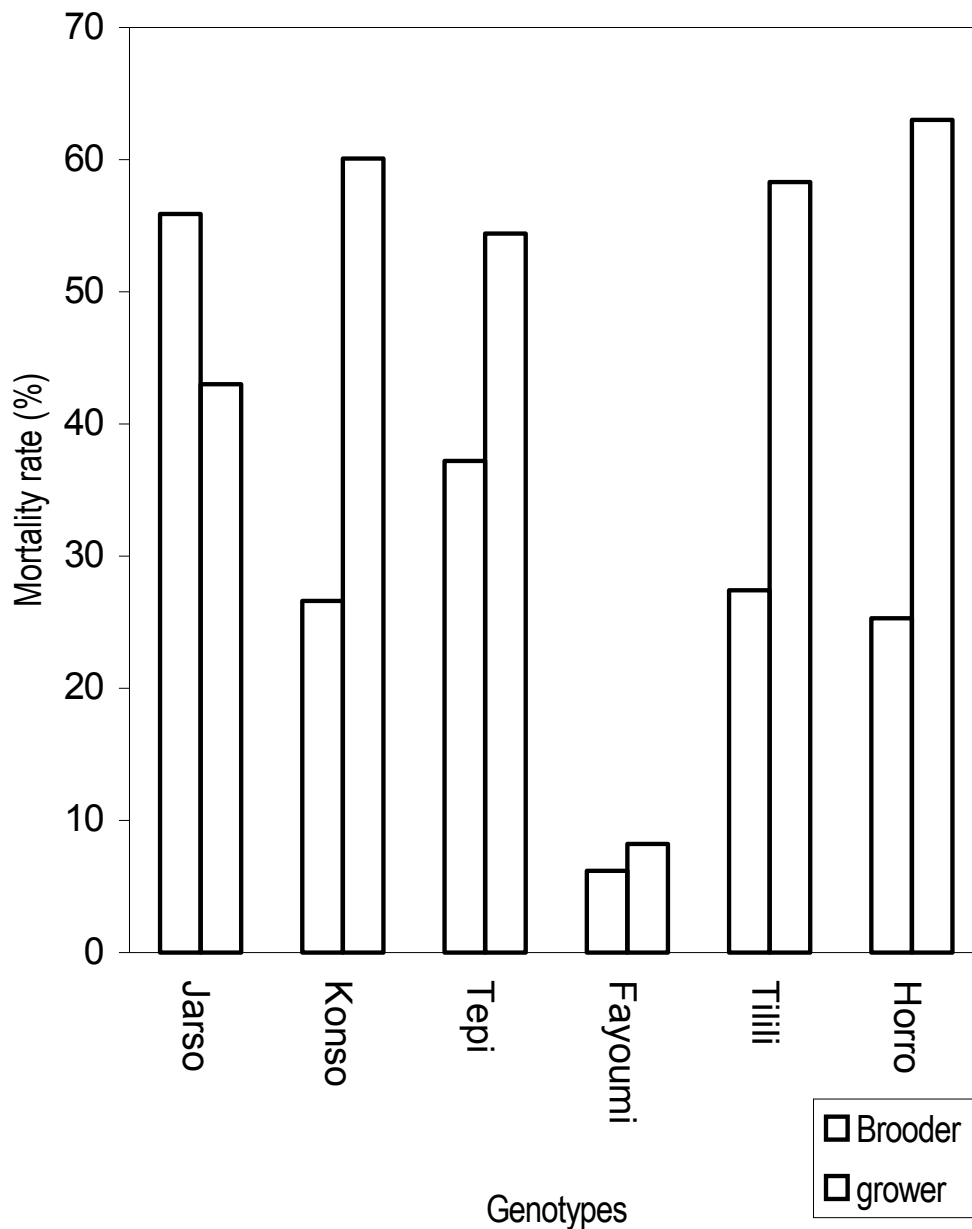


Figure 1: Effect of chicken strain and phase of production on mortality under natural MD challenge under confined management.

Mortality Pattern and Incidence

There was a difference between the indigenous chicken strains and the exotic ones in the pattern and incidence of mortality, which helps in the relatively ranking of the chicken genotypes for survival under confined management.

Mortality pattern of indigenous chicken strains until 20 weeks of study period continued in a wave fashion in undulating amplitudes. Jarso, Tepi, and Konso genotypes more or less followed similar pattern of mortality curve except with varying amplitude for each ecotype. However, Horro and Tilili had somewhat similar but a distinct pattern of mortality curve from the rest three indigenous chicken genotypes. These two ecotypes had a sort of similarity in their amplitude of mortality curve except during the 8-10 weeks of age, where mortality in Tilili ecotype was high but low in Horro ecotype. The exotic chicken strain (Fayoumi) had a different picture of mortality pattern in that the amplitude of mortality curve was constantly very low (<5%) and with low amplitude through out the study period.

Detailed observation of the curve of mortality pattern indicated that the magnitude of chicken population attacked among indigenous chicken strains was considerably different. It was indicated in the mortality curve that majority of the Jarso ecotypes died during the infant stage (brooder phase) unlike Konso, Tepi, Tilili and Horro, which were at grower stage. Thus, incidence of mortality was very high in the first 3-6 weeks of age for Jarso and 9-14 weeks for Tepi and Konso genotypes. The magnitude of mortality was very high and skewed to the grower phase (9-14 weeks of age) in both Tilili and Horro. Fayoumi chicken was less affected by mortality indicating to be better survivors under confined management (Figure 2). The similarity in pattern and magnitude of mortality among some of the indigenous chickens while there is still variation in some of the indigenous and Fayoumi chickens of this study might depict the presence of genetic relatedness and variation among the chickens.

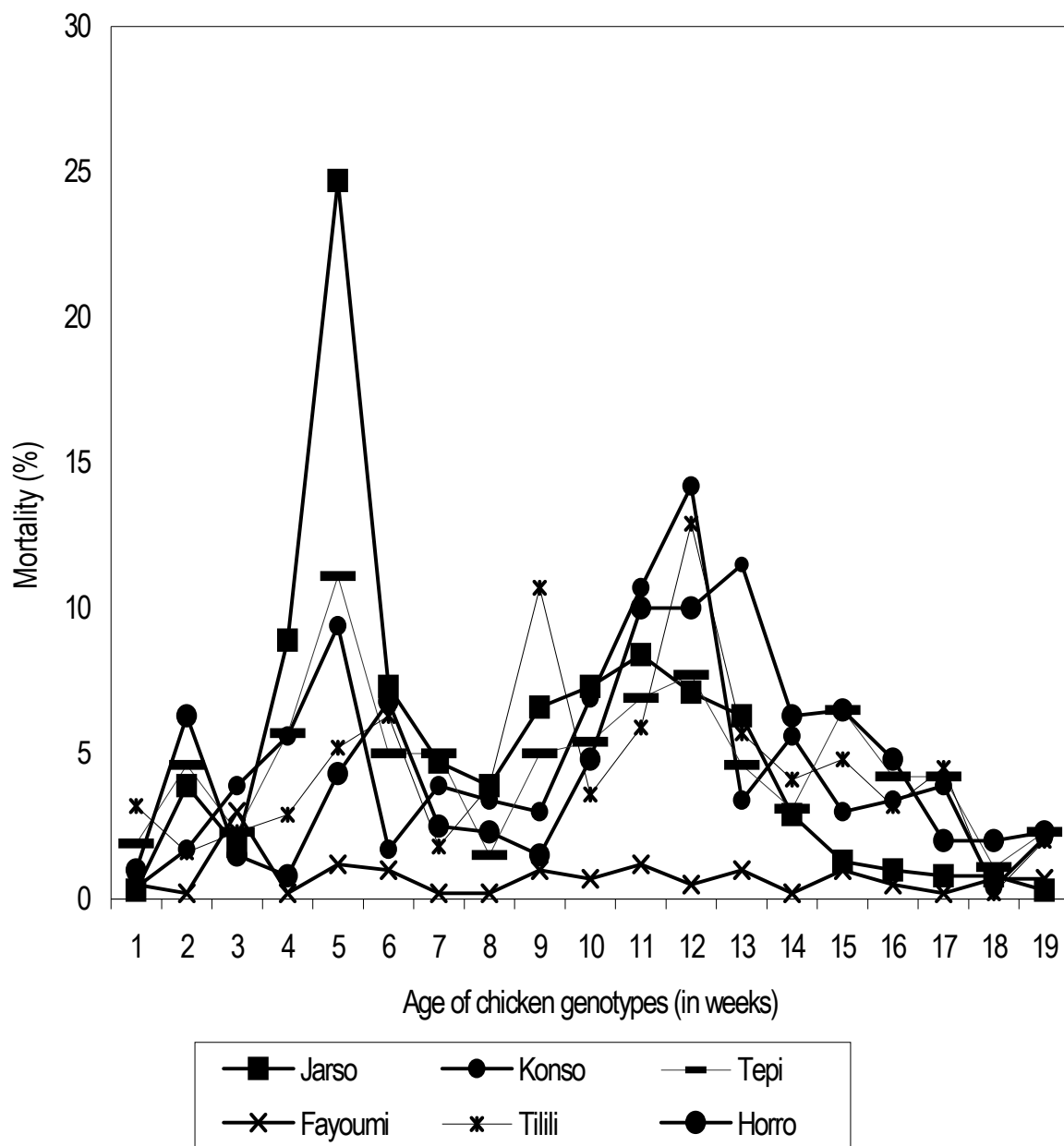


Figure 2: Effect of age and chicken strain on pattern and incidence of mortality under confined management

Chicken Survival

The mean length of survival of Jarso ecotype under confined management under MD natural challenge was so short (8.07 weeks) compared to the other ecotypes. Konso (11.80 weeks) and Horro (11.55 weeks) which seemed to have better life span (Table 6).

However, this variation in mean length of survival did not seem to have economic relevance for those who need to rear indigenous chicks for egg laying under confined management unless mortality situation is reversed.

Table 6: Effect of chicken strain on mean length of survival under confined management

| Chicken | Jarso | Konso | Tepi | Fayoumi | Tilili | Horro |
|---------------------------------|-------|--------|--------|---------|--------|--------|
| Initial chick population | 381 | 233 | 261 | 401 | 441 | 400 |
| Mean length of survival (weeks) | 8.07c | 11.80a | 10.19b | 66.22 d | 10.50b | 11.55a |

The population of indigenous chickens that entered the layer phase was so negligible (Figure 3). The Jarso ecotypes were almost depleted to reach the layer phase due to the ravaging mortality due to MD followed by the Tepi ecotypes. The Horro, Konso, and Tilili ecotypes were also seriously affected. However, the Fayoumi chickens were very hardy in resisting the MD challenge and hence 85.5% reached to commence the layer phase at 21 weeks of age.

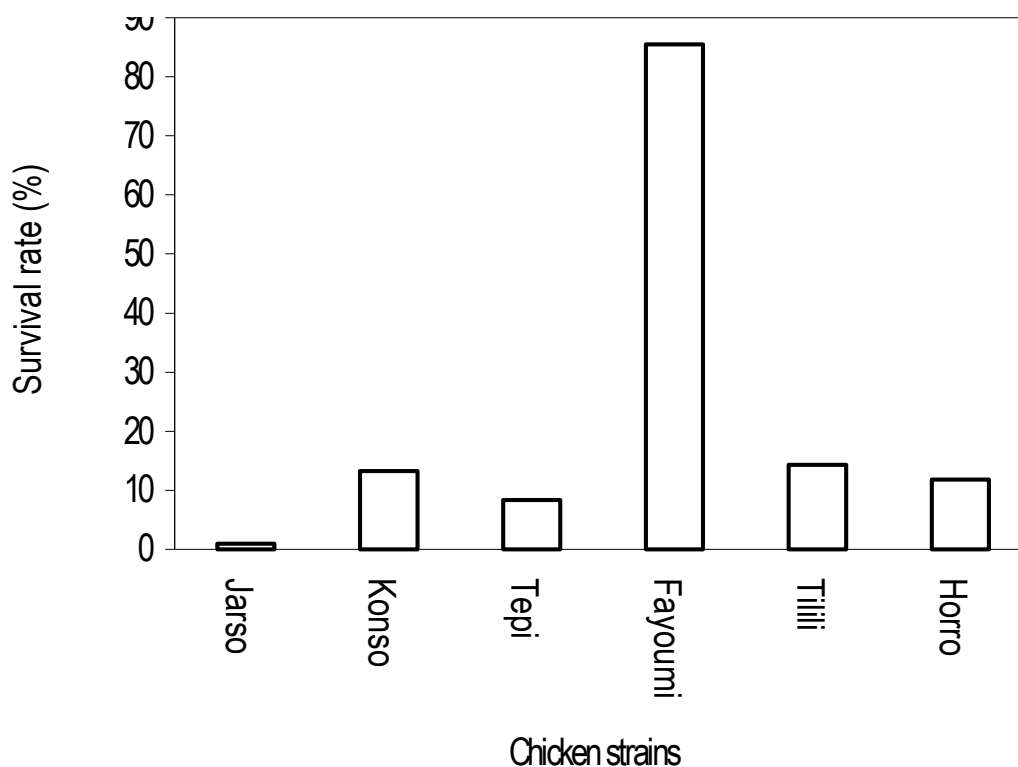


Figure 3: Effect of age and chicken strain on survival rate under confined management until the laying phase

Results of the Intervention Phase

Prevalence of Total Mortality

The prevalence of mortality in MD vaccinated and non-vaccinated ecotypes is presented in (Table 7). The level of susceptibility rate to MD natural challenge was measured by mortality rate and it varied between ecotypes (Table 8). The response of the chickens to MD vaccination varied among ecotypes (Table 9). Mortality variation was also observed within ecotypes for presence or absence of MD vaccination. The study indicated that MD vaccination brought a remarkable survival rate.

Table 7. Prevalence of mortality in four chicken ecotypes (MD Vaccinated and non-vaccinated) reared under confined management in the first 21 weeks of life

| Ecotypes | MD vaccine | | Outcome | | Total |
|----------|----------------|---|----------|-------|-------|
| | | | Survival | Death | |
| Fayoumi | Non-vaccinated | N | 91 | 5 | 96 |
| | | % | 94.8 | 5.2 | 100.0 |
| | Vaccinated | N | 96 | 0 | 96 |
| | | % | 100.0 | 0 | 100.0 |
| Horro | Non-vaccinated | N | 114 | 108 | 222 |
| | | % | 51.4 | 48.6 | 100.0 |
| | Vaccinated | N | 176 | 46 | 222 |
| | | % | 79.3 | 20.7 | 100.0 |
| Tepi | Non-vaccinated | N | 56 | 34 | 90 |
| | | % | 62.2 | 37.8 | 100.0 |
| | Vaccinated | N | 62 | 28 | 90 |
| | | % | 68.9 | 31.1 | 100.0 |
| Jarso | Non-vaccinated | N | 85 | 149 | 234 |
| | | % | 36.3 | 63.7 | 100.0 |
| | Vaccinated | N | 194 | 40 | 234 |
| | | % | 82.9 | 17.1 | 100.0 |

Of the 96 MD non-vaccinated Fayoumi chickens 91 (94.8%) survived and 5 (5.2%) died whereas from equal number of MD vaccinated Fayoumi chickens 96 (100%) survival was observed. Of 222 MD non-vaccinated Horro ecotypes 114 (51.4%) survived and 108 (48.6%) died while 176 (79.3%) survived and 46 (20.7%) died from MD vaccinated group. Of 90 non-vaccinated Tepi ecotypes 56 (62.2%) survived and 34 (37.8%) died while 62 (68.9%) survived and 28 (31.1%) died. Concerning 234 non-vaccinated Jarso ecotypes 85 (36.3%) survived and 149 (63.7%) died whereas of equal vaccinated chicken number 194 (82.9%) survived and 40 (17.1%) died.

Response to MD Natural Challenge

The survival rate of MD non-vaccinated Fayoumi chickens differed significantly from native chickens under MD natural challenge. The response of survival rate of non-vaccinated Tepi, Horro, and Jarso to MD natural challenge also differed significantly from each other. The survival rate of non-vaccinated chickens was in the order of Fayoumi, Tepi, Horro, and Jarso, respectively. In other words, the degree of susceptibility to MD challenge as measured by mortality rate was differed between ecotypes. Fayoumi chickens showed some degree of resistant to MD challenge whereas Jarso was more susceptible to death from MD. Tepi and Horro showed moderate resistance to the death caused by MD in that the survival rate of Tepi and Horro was significantly higher than Jarso to MD natural challenge (Table 8).

Table 8. Response of Health Performance of Chicken Ecotypes to Natural MD Challenge.

| | | Fayoumi | Horro | Tepi | Jarso |
|----------------------------|---------|---------|--------|--------|-------|
| Survival (%) | | 94.8 | 51.4 | 62.2 | 36.3 |
| Death (%) | | 5.2 | 48.6 | 37.8 | 63.7 |
| χ^2 significance test | Fayoumi | — | | | |
| | Horro | 0.000* | — | | |
| | Tepi | 0.000* | 0.081‡ | — | |
| | Jarso | 0.000* | 0.001† | 0.000* | — |

*, †, ‡ Chi-square statistic was significant at the 0.000, 0.001, and 0.1 level, respectively.

Response to MD Vaccination

MD vaccination increased the survival rate of Fayoumi breed that differed significantly ($p < 0,000$) from our native chickens. In presence of MD vaccination, the survival rate of Jarso and Horro was also differed significantly ($p < 0.1$) from that of Tepi ecotype. MD vaccination brought better survival rate of the chickens that ranked in the order of Fayoumi, Jarso, Horro, and Tepi, respectively (Table 9).

Table 9. Response of Health Performance of Chicken Ecotypes to MD Vaccination

| | | Fayoumi | Horro | Tepi | Jarso |
|----------------------------|---------|---------|--------|--------|-------|
| Survival (%) | | 100 | 79.3 | 68.9 | 82.9 |
| Death (%) | | 0 | 20.7 | 31.1 | 17.1 |
| χ^2 significance test | Fayoumi | — | | | |
| | Horro | 0.000* | — | | |
| | Tepi | 0.000* | 0.051‡ | — | |
| | Jarso | 0.000* | 0.322 | 0.006* | — |

* †, ‡ Chi-square statistic was significant at the 0.000, 0.05, and 0.1 level, respectively.

From the result of Table 8 and 9, Jarso ecotype seemed to be an ideal indicator and might serve as a standard susceptible check whereas Fayoumi chickens might serve as standard resistant check to monitor survival in MD related research activities and challenges.

Impact of Vaccination

The survival rate of the chickens improved due to MD vaccination. MD vaccination increased the survival rate of Jarso and Horro significantly ($p < 0.000$) from 36.3% to 82.9% and from 51.4% to 79.3%, respectively. The Tepi ecotype showed improvement in survival rate due to MD vaccination but not significantly. Survival of Fayoumi chickens also increased significantly ($p < 0.05$) from 94.8% to 100% due to MD vaccination (Table 10). Thus, MD vaccination brought a significant change successfully and improved the survival of the chickens dramatically.

Table 10. Impact of MD vaccination and non-vaccination on survival of chicken ecotypes

| | Fayoumi | Horro | Tepi | Jarso |
|----------------------------|--------------------|--------|-------|--------|
| MD vaccinated (%) | 100 | 79.3 | 68.9 | 82.9 |
| MD non-vaccinated (%) | 94.8 | 51.4 | 62.2 | 36.3 |
| χ^2 significance test | 0.023 [†] | 0.000* | 0.347 | 0.000* |

* [†]Chi-square statistic was significant at the 0.000 and 0.05 level, respectively.

Jarso genotype is the best to respond to vaccination; while Tepi responded least. The response of Horro and Fayoumi to MD vaccination was moderate. Therefore, the vaccine-ecotype interaction to reduce MD caused mortality was ranked and more pronounced in Jarso, Horro and Fayoumi chickens than Tepi ecotypes (Table 10).



a). Marek's disease (MD) vaccinated

b). From MD non-vaccinated

Figure 4. Effect of Marek's disease vaccination on survival of native chicken ecotypes

Overall Survival

Comparison of the mortality during the diagnostic and intervention phase was made and summarized in figure 5.

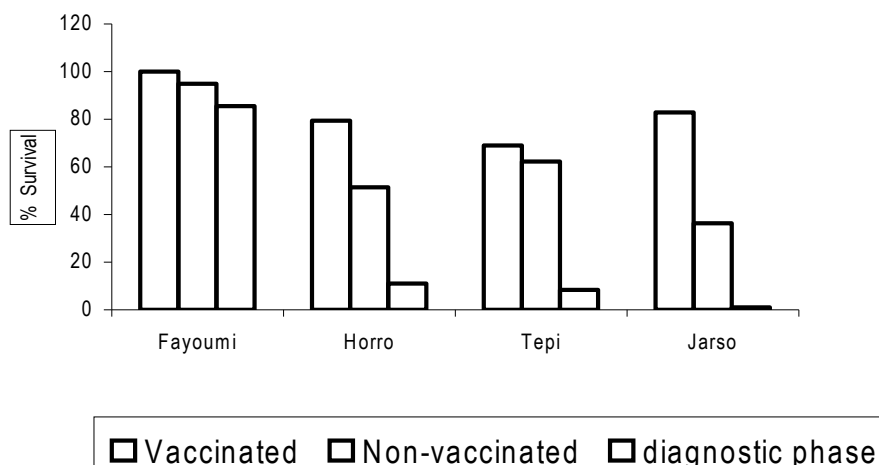


Figure 5: Survival rate of chickens under confinement during intervention phase (MD vaccinated and non-vaccinated) and diagnostic phase.

The survival rate of the Jarso chicken ecotypes changed from 1% to 36.3% and then to 82.9% during diagnosis phase, intervention phase but non-vaccinated and MD vaccinated, respectively. In the Tepi, Horro, and Fayoumi chickens, the survival rate in diagnostic phase was 8.4%, 11%, and 85.5%, respectively. During the intervention phase, survival rate in MD vaccinated chickens dramatically increased in Tepi (68.9%), Horro (79.3%), and Fayoumi (100%) whereas in MD non-vaccinated the survival rate was 94.8% in Fayoumi, 51.4% in Horro and 62.2% in Tepi ecotypes.

Despite absence of MD vaccination both in diagnostic phase and the MD non-vaccinated control group of the intervention phase, the survival rate of the same chicken ecotypes varied. The live MD vaccine can shed life long permanently from the vaccinated chicken

to the environment that might in turn infected via inhalation chicken's of the control group that was closely kept with vaccinated ones. In so doing, the control groups of the intervention phase were partly immunized by the shading live vaccine virus, hence, partially protected showing some degree of improved survival rate. The increased survival rate of the non-vaccinated group during the intervention phase in comparison to the diagnostic phase, in figure 5 above, might show this situation.

Implications of the Study

This study indicated that susceptibility to natural Marek's disease challenge as measured by mortality rate varied significantly among the ecotypes of chickens. The mortality rate recorded was 87.5% in Jarso, 60.7% in Konso, 75.0% in Tepi, 58.3% in Tilili and 67.6% in Horro ecotypes in non-vaccinated indigenous chickens during the diagnosis phase. The magnitude of morbidity and mortality is nearly equal indicating that MD is fatal. However, during the intervention phase in the MD non-vaccinated group the mortality rate due to natural MD challenge was significantly different in Fayoumi (94.8%), Tepi (62.2%), Horro (51.4%), and Jarso (36.3%). Unlike the control group (MD non-vaccinated) of the intervention phase, the birds were dead in the first 21 weeks of life and failed to survive under the confined management regime during the diagnostic phase. The research publications on indigenous chickens pertaining to natural MD disease challenge and mortality attack are scarce to compare with this finding. Nevertheless, the overall non-vaccinated chicken groups that were challenged by natural MD were compared crudely with findings of others. The higher mortality rate observed in MD non-vaccinated group is in agreement with the findings of Blaha, (1989) and Palya, (1991) who reported high morbidity and mortality that range from 20-70 % in intensive production system in non-vaccinated chickens. It also agrees with the observation of Fikre and Moges (2004) who recorded mortality as high as 46 % in intensively managed non-vaccinated exotic chickens in central Ethiopia. This finding is in congruent with the findings that indigenous chickens of Ethiopia reveal high susceptibility (very low survival rate) than the exotic breeds such as Fayoumi, WLH, and RIR under confined management system (Teketel, 1986; Brannang and Pearson, 1990; Abebe, 1992; Solomon, 2003).

The observation made during diagnostic phase showed that the chicken genotypes were more susceptible to the acute forms of clinical signs and visceral form of gross lesions. These

manifestations and forms of clinical signs and gross lesions across chicken genotypes showed significant difference ($p < 0.01$). The course of MD was different among chicken genotypes in such a way that acute form of the disease was observed in 65.2% during clinical examination and visceral form MD was 71.8% during autopsy examination. According to (Palya, 1991) tumor formation in various organs is the characteristics of MD virus sero-type 1.

In the current study, MD attack at 14 and 15 weeks of age was higher than the subsequent age groups. The occurrence of high magnitude of acute form of clinical signs and visceral forms during autopsy with varying degree of mortality rate among age groups were in agreement with the reports of Fikire and Moges (2004), Brannang and Pearson (1990), and OIE (1990). According to Venugopal (2004), there was also a report that acute and/or visceral forms of MD, with recent increasing evolution of virulent viral strain, have been frequently observed currently.

Antibody prevalence in grower indigenous chickens to MD in this study is very high, 72.9 %. According to (OIE, 1990), AGID tests are employed most commonly to detect antibody and the presence of antibodies in chickens above 4 weeks of age is an indication of MD infection. However, the mortality figure is so low in the finding of control group than the findings of diagnostic phase despite both are the non-vaccinated ecotypes. This might be due to exposure of the current control groups to infection via inhalation by shedding vaccinal virus as they were placed randomly near each other within one house. For instance, the control group of Tepi ecotype on average was randomly placed unexpectedly with the three of the MD vaccinated other chicken genotypes. Thus, this might have resulted in slight infection that might have led to partial protection and survival of the control chicken groups in the control group than the diagnostic phase. In this line, Fraser and Mays (1986), OIE (1990), Venugopal (2004) have reported that the live vaccine MD can shed permanently life long from the vaccinated chicken to the environment that might in turn infect other chickens that are closely kept via inhalation.

The intervention phase of this study indicated that Marek's disease vaccination brought a dramatic change through increasing survival rate of chickens from 36.3% to 82.9% in Jarso, 51.4% to 79.3% in Horro, 62.2% to 68.9% in Tepi and from 94.8% to 100% in Fayoumi ecotypes. As research information on indigenous chickens pertaining to MD vaccination trial is scarce, comparison is made with exotic chickens. This finding agrees with the report of Fikre (2003) who observed mortality rate of 5.51 % of MD in vaccinated exotic pullet flocks in Ethiopia. In this line, Blaha (1989) and Palya (1991) have indicated that MD vaccination successfully dropped mortality incidence dramatically from ranges as high as 70 % in non-vaccinated to less than 5 % in vaccinated. However, the mortality figure of the current finding varies from their reports. It may be attributed to the genetic variation of the birds; dosage and strain of the infecting virus or variation in management that in turn interacted (host-agent-environment) to determine the outcome of the mortality status in current study. In this line, Blaha (1989) reported that three sets of factors are related to in disease process that includes infective agent (viral strain, dosage, and route of exposure), host (genetic constitution , age and sex), and environmental factors such as confining and stocking.

According to same author, these complex set of factors could influence and involve in the pathogenesis, and then the incubation period, the age at which an outbreak of MD occur, character and extent of lesions and symptoms, the course of the disease, the difference in rate and duration of morbidity and mortality in a flock.

Considerable resistance to MD challenge is observed in Fayoumi chicken. Better survival rate of the Fayoumi chicken than the indigenous chickens is the indicator to attest this scenario. Such resistance of Fayoumi is revealed in both presence and absence of MD vaccination. The indigenous chickens are generally susceptible to MD but significantly differed. For instance, Jarso seemed more susceptible than others did. Indigenous chickens show lower survival rate than the exotic breeds such as Fayoumi, WLH, and RIR under confined management system (Teketel, 1986; Brannang and Pearson,

1990; Abebe, 1992; Solomon, 2003). The reason for higher susceptibility (the poor survival) of our indigenous chickens might be very velogenic viral strain is involved and/or our chicken might be genetically predisposed to higher susceptibility and might be contributed by immune stressor factors of confining and stocking, as they are naturally adapted to free range scavenging environment and new to confinement. Thus, as it is proved in this work, without full components of package such as MD vaccination our chicken genotypes are very susceptible to MD under confined management. Similarly, variation is observed among the chicken ecotypes to respond to the MD vaccination. In this case, Jarso seemed to show dramatic response to MD vaccination to improve survival rate while Tepi showed less response to MD vaccination as measured by improvement in survival rate.

According to all chicken genotypes are susceptible to MDV infection, but they differ greatly in their resistance or susceptibility to clinical MD (Calnek, 1985). The variation in magnitude of signs, lesions, and mortality rates among chicken genotypes despite similar exposure to MD infection could be related to genetic variations. Similarly, the study on MD natural challenge (control group) and MD vaccination among chicken ecotypes showed the presence of immunogenetic variation to respond to the wild and vaccine virus. In this line based on PCR study on the DNA, Tadelle (2003) indicated the presence of genetic variation and relatedness within and between the Jarso, Tepi, Chefe, Tilili, and Horro ecotypes although the correlation of the genes with MD infection had not been dealt within his study. Fayoumi chickens showed a considerable degree of resistance against MD challenge. They strongly responded to MD vaccination than any of the studied indigenous chickens of Ethiopia. The works of many authors have been cited in the report of [Lui et al., 2001] that chicken genotypes (strains) that were resistant to MD were observed. The utilization of such resistant chicken genotypes can serve as alternative control strategies due to its reliability, long lasting and environmental soundness in the front of continuing evolution of virulent MD viral strains in the field despite presence of MD vaccination.

Conclusion and Recommendations

In conclusion, we have presented four observations that indicate the problems and corrective strategies for rearing indigenous chickens under intensive (confined) management. First, MD is diagnosed in heavily attacked indigenous chickens by present mortality. Second, the level of mortality among various chicken strains is different. Third, lesions of malignant neoplasm (tumors) of visceral organs and other body tissues that seems to be MD virus serotype 1, according to (Palya, 1991), have been frequently encountered. Fourth, MD vaccination dramatically changed the previous routine of mortality scenario and survival rate is increased. In general, this work is in agreement with previous publications from several authors that have implied an association between MD and level of host mortality, genetic variation among chickens and visceral tumors in poultry and role of MD vaccination to control MD challenge in poultry farm.

Thus, the result of the study gives that MD is a serious health problem for indigenous chickens kept under confined management and probably contributed to the collapse of previous attempts. MD vaccination has dramatically improved the survival rate of indigenous chickens, and hence opened the door and put the landmark for the future research efforts on these animals. Therefore, the need for MD vaccination for rearing indigenous chickens under confined management should be practiced as an important management strategy.

The susceptibility/resistance rate of different chicken types to MD is quite different. Still their immunological response to MD vaccination to withstand mortality varies with chicken types. Further detailed study on immuno-genetic basis of variation to respond to the MD challenge and to vaccination among indigenous chicken ecotypes is recommended. As MD is a severe health threat to poultry industry

and yet its vaccine production not started in the country, efforts should be made to isolate the strains circulating in different production systems in the country. Thus, with increasing establishment of large and small-scale poultry farms and with increasing MD prevalence reports, production of MD vaccine in the country should be ultimately recommended. Generally, this work gives a point of reference on which to base further investigations to address mortality problems of indigenous chickens under confined management.

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