

SHORT COMMUNICATION

OBSERVATION ON CONGENITAL LOCO, A HEREDITARY DEFECT IN THREE FLOCKS OF CHICKEN AT IBADAN

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Received 9 May 1996; Accepted 06 March, 1998

ABSTRACT

Congenital loco, a monogenic, sex-linked hereditary defect was observed in three flocks of chicken at Ibadan. Mortality at the end of the first 4 days of life was unexplainably high. Noticeable signs were birds thrown off balance with necks thrown backwards, chicks unable to feed nor drink. Subsequently, they got weak, slumped and died.

Key Words: congenital loco, hereditary defects, lethal factor, sex-linked, pullets.

INTRODUCTION

Hereditary defects which are essentially lethal within the first few days of life usually pass unnoticed or are reported as disease outbreak (epidemics) in livestock species in this part of Africa. This is because more emphasis is placed on the disease rather than on genetic pre-disposition to disease or death in man and animals. This paper reports on congenital loco, a simple, monogenic, recessive, sex-linked defect, in poultry species as found in 3 egg production farms at Ibadan.

Case Description

2600 day old chicks (pullets) were purchased by 3 different farmers from a highly reputable hatchery on the 18th July, 1994. By the end of the first 48 hours of life, 190 birds

were lost to a strange disease in these herds. Another 97 were similarly lost under the same circumstances in the next 48 hours.

However the effect got stabilized within the following 48 hours when only 58 and 46 birds were lost respectively (Fig 1).

Noticeable signs were, birds thrown off balance, with necks thrown backwards. Chicks were unable to feed or drink. They subsequently got weak, slumped or got turned over by other chicks as they brushed past affected chicks. They were unable to get up after several hours of struggling on their back or sides and they subsequently died.

DISCUSSION

Congenital Loco is a monogenic, sex-linked hereditary defect of poultry transmitted by heterozygous males having chromosome XX^{10} (Cole, 1957). Although the condition is caused by recessive gene in the homozygous state (Knowlton, 1929), the defect is more pronounced in the female chick because females in birds are hemizygous X^0 or X^- . Since the gene is carried as X^{10} on the sex chromosome in the female it has no other protective dominant allele. The x^{10} allele is therefore expressed in the hemizygous carrier and being a lethal effect, all females afflicted are eliminated.

Although the gene is fully expressed in the hemizygous female, it is expressed as a recessive defect in males. Therefore, males are carriers of congenital loco. The effect of congenital loco gene is only expressed in males homozygous for its allele (X^{10}). Carrier males are not recognized phenotypically: they behave as normal males and may even exhibit other traits of economic importance that may make it impossible for farmers to eliminate them such as, rapid feathering, fast growth, and good fleshing, which happen to be qualities

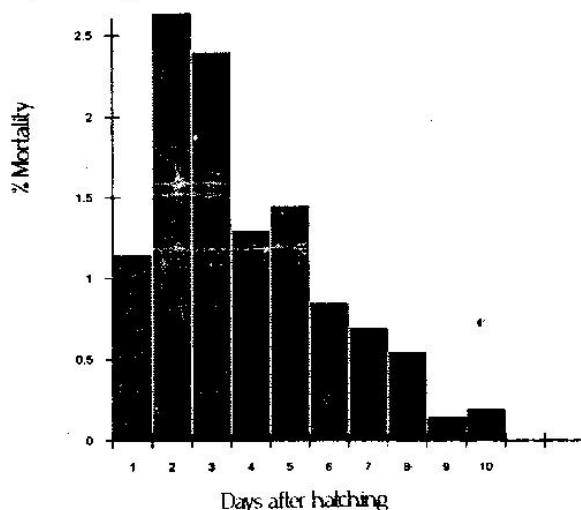


Figure Percentage mortality in affected chicks after hatching

When these heterozygous males are mated to a normal female and the gene is transmitted to his female offsprings, the eggs will hatch normally at 21 days but all affected pullets will die within the first 24 hours of hatch or shortly after. However, when transmitted to the sons

Identification of offending males

In poultry, this is a very simple and inexpensive exercise in terms of time and money (Hutt, 1961). Individually, males are test-mated to normal females at the rate of 1 male to 10-20 female; the fertile eggs collected for every male are set for hatching. All normal eggs with or without this defect will hatch normally after 21 days in poultry and 28 days in turkeys (unhatched eggs may carry other defects or disease to be discussed later). All female chicks that inherit this gene will die within the first 3 days after hatching. Hence a total of 30-40 days is required for this experiment (5-7 days egg collection 21 - 28 days incubation and 3 days observation in both chicken and turkeys). From the mating that yields a loco chick, two-thirds of the unaffected surviving chicks must also be carriers. Hence none of these supposedly normal birds should be used for breeding.

Males that record very high early mortality in his offspring with these observed mode of expression should therefore not be used in producing commercial layers. For example, in these flocks 2600 day old pullets were purchased at the cost of ₦30 per pullet (as at July 1994) with about 300 pullets lost within the first 3 days, about nine thousand naira (₦9,000.00) had been lost to only one genetic defect. In addition to the cost of drugs used at this stage, and the fact that other disease defects might still be encountered, this is definitely an avoidable economic loss to the farmer. Within large flocks with as high as 12% mortality in the first 3 days one can imagine the multiplicity of the loss to the poultry industry.

Time of lethal action

Lethal genes may kill at various stages during the life span but there are usually characteristic modal ages for onset and peak of mortality associated with each type. The range in time of lethal action is illustrated by some of the lethals known in the domestic fowl. In general, the greater the abnormality caused by the lethal gene, the earlier the age at death.

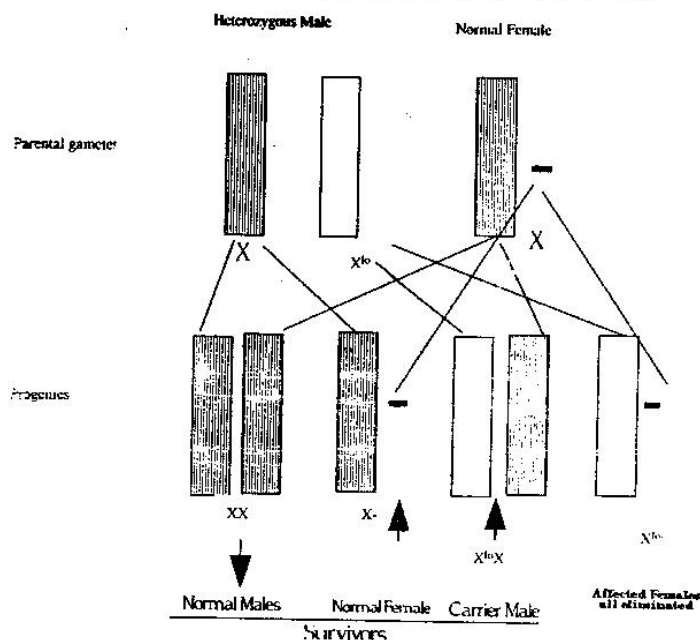


Figure 2. Parental Transmission of Congenital loco

they also become carriers because they have the protective gene on the X chromosome from their normal mothers which along with the gene X¹⁰ from their fathers make them heterozygous XX¹⁰ again (Fig. 2).

As discussed above, homozygous males can not be produced since female carriers which are expected to be used to produce them can not survive till re-productive age however good is the management. At the same time, heterozygous males can not be recognized phenotypically. For this reason, these males are carriers and they could be used unnoticed for a very long time in the flock of poultry breeders. Because males contribute 50% of the breeding value in any flock carrier male remain an unrecognized source of unreported economic loss in poultry hatchery operation. In most cases, helpless farmers are made to bear the brunt by pumping drugs into their birds not knowing that this defect does not respond to any form of treatment. The only treatment which in this case is very cheap and easy is to identify the offending male in the flock for elimination.

CONGENITAL LOCO IN POULTRY

The gene Cp causing the form of incompletely dominant achondroplasia, that is a breed characteristic in Creepers is lethal to most homozygous CpCp or Cp- at 3 to 4 days of incubation while the "Talpid" mutation causes a peak of mortality around the 8 - 10 day of incubation. The sex-linked "naked" gene 'na' is lethal to almost half of affected chicks during the last two days of incubation while the chicks afflicted with Congenital loco hatch quite normally but cannot feed or drink so they die within a few days. It is to be noted that eggs with the first two lethal cases mentioned above will not hatch.

Two sex-linked disorder apparently of the nervous system are not evidenced at hatching and may appear at two to six weeks thereafter and are usually fatal before twelve weeks of age. These are the shakers syndrome 'sh' and Congenital tremor 'tr'. The hereditary atresia of the oviduct is fatal after affected hens have begun to ovulate.

Thyrogenous dwarfism 'dw' is fatal to most chicks at six weeks of age and sometimes it is delayed until the hen is about 18 months. Paroxysm 'pm' which makes afflicted birds to react to unusual noise, sudden bright light or other alarms by making it run and fall over in a tetanic seizure with its legs rigidly extended, head thrown back, wing beating violently and the body in a state of tremor for almost 10 seconds after which the bird relaxes, lie quietly

and eventually stagger off, kills these types of birds before 15 weeks of age. Hence birds affected in this way are not useful for production and are therefore sources of economic losses to the poultry industry.

Semi-lethal genes are fatal only to some of the individuals having the dangerous genotype but not necessarily to all. In such cases, the proportion that escapes may depend largely on whether or not the environment is favourable. Other genes lower physiological efficiencies wither temporarily or permanently and so traits induced by such genes are termed SUBVITAL.

The fact remains that a genetic defect occurring in one species is likely to appear in modified form in other species hence there is the need for breeders to report lethal effects in their beloved species.

REFERENCES

- COLE, R.K. (1957) Congenital Loco in turkey: A thorough genetic study of the abnormality J. Heredity 48: 173 - 75.
- HUTT F.B. (1961) Identification and elimination of defect in animals. In: Germplasm resources, Washington Amer. Assoc. Adv Sci. Pp. 355 - 369.
- KNOWLTON F. L. (1929) Congenital loco in chicks. Oregon Agric. Expt. Sta. Bull 253.