

THE GENETIC CHARACTERISTICS OF CEPHALIC ABNORMALITY AND ITS AQUACULTURE IMPLICATION IN CLARIAS GARIEPINUS (BURCHELL 1822)

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ABSTRACTS

Cephalic abnormality in nine mating groups involving *Clarias gariepinus* with cephalic abnormality, non Cephalic and Maiduguri origin were investigated to determine the level and the genetic basis of the occurrences and its aquaculture implication. The highest mean percentage survival in a Cephalic crossed with non cephalic group was 68.6% whereas the last mean percentage survival was 25.7% in the group of Maiduguri cross with Maiduguri parent. Cephalic abnormality was observed in the crosses with both female and male cephalic with a total frequency ranging from 0 % to 70.8. %. The least number of cephalic abnormality was 18.5% which involved a cross of non cephalic with cephalic, and the highest was 70.8%. This level of cephalic abnormality shows that the defect was hereditary. This result implies that, the genetic factor is a major contributor in the feature of cephalic abnormality in *clarias gariepinus*.
Key words cephalic, Non-cephalic, abnormality cephalic hereditary.

INTRODUCTION:

The African Clariid catfish, especially the species of *clarias* and *Heterobranchus*, are candidates for commercial aquaculture in African, they are mostly used under controlled condition in fish hatcheries. In addition, hatchery operators hardly have room for exchange of genes with collections from the wild or other hatcheries, thus encouraging morphological abnormalities in hatchery breed catfish (Aluko et al 2001). Some of the most important problems in fish culture is high rate of body deformation and abnormalities. Deformities represent a considerable problem since the injury may reduce product quality and lead to economic losses for the fish farming industry. Communications describing the incidence and inheritance of morphological trait in commercial valuable fish are increasingly being documented. A lot of studies have been carried out among Cyprinids, *Salmonids*, *Casostomils*, *percids*, *Silurids* as well as in aquarium fish such as the *cepridontidae* and the *peocilidae* (Kirpichnikov, 1987). Aberrations commonly described include; scaling pattern, fin shape, skeletal deformity, eye abnormality and colour differences. Morphological abnormalities affecting pectoral and ventral fins have been reported in some second generation backcross hybrids of *clarias* and *Heterobranchus* species (Aluko, 1998). Other aberrations that affect the dorsal, adipose, caudal and anal fins as well as the cephalic region and the shape of the abdominal region have been observed in hatchery breed African catfish, clariid species (Aluko et al. 2001).

The reasons for this deformation in fishes are barely understood. There is evidence of both environmental and genetic causes. Blaxter et al (1974) induce skeletal abnormalities with nutritional deficiencies experimentally. Environmental factors may affect egg during vitellogenesis or incubation, or hatched larvae in rearing tanks. Malformation is induced when eggs are incubated in excessive densities or exposed to mechanical shock (Porhmaranz 1974). Other environmental factors such as density of eggs, mechanical or thermal shocks, presence of pollutants in the water, radiation, salinity, oxygen depletion and light intensity have also been reported to cause aberration in development Caris and Rice, 1990). Van valen (1962) considered that the presence of asymmetrical abnormalities is an external symptom of a weakness in the buffering power of the polygenetic systems under unfavourable environmental conditions. In some natural populations of fish, frequency of abnormalities has been shown to increase with age. This suggests that unfavourable environmental factor notably toxin pesticides and other polluting agents may induce abnormalities of any stages of the fish life (Valentine, 1975). Finally, the genetic basis of the cephalic abnormality of African catfish; *clarias gariepinus* and its aquaculture implication has not been reported before in the literature.

The aim of this study was therefore designed to determine whether environmental or genetic factor is responsible for cephalic abnormality in *clarias gariepinus* and the implication of cephalic abnormality to aquaculture.

MATERIAL AND METHODS

Induced spawning and incubation: The brood stock of *clarias gariepinus* were obtained from Fish Genetic Experimental tanks (NIFFR), which can be described as cephalic abnormality (CP), Non-cephalic (NC) and Maiduguri Origin (Mai) were selected. Induced Spawning were carried out by injecting only the females of each combinations with ovaprim hormone at a single dosage of 0.5ml/kg body weight.

After latency period of 12hrs the males of each combination were sacrificed and their testes were removed, cleaned and kept in a covered Petri-dishes. Slight-pressure were applied to the abdomen of the females to release their eggs into separate Petri-dishes. These testes were cut opened with a sterilized razor blade and the spermatozoa were diluted with saline. The eggs were then fertilized by mixing them with spermatozoa with the aid of sterilized dry feather.

From those combinations, nine generic groups were formed;

Cephalic (F) x Cephalic (m) (CP x CP)

Non cephalic (f) x Non Cephalic (m) (NCP x NCP)

Maiduguri (f) x Maiduguri (m) (Mai x Mai)

Cephalic (f) x Non Cephalic (m) (CP x NCP)

Cephalic (f) x Maiduguri (m) (CP x Mai)

Non Cephalic (f) x Cephalic (m) (NCP x CP)

Non Cephalic (f) x Maiduguri (m) (NCP x Mai)

Maiduguri (f) x Cephalic (m) (NCP x CP)

Maiduguri (f) x Non cephalic (m) (Mai x Cp)

All these series of combinations were carefully transferred into well aerated glass aquarian of 60cm x 30cm size tanks immediately after fertilization with the following water quality parameters, dissolved oxygen 5mg /l, pH 7.4, and temperature 26.1°C.

Within the 24hrs, the hatchings were observed swimming around the base of aquaria. The percentage hatchability of each the combinations were taken.

REARING INDOOR

Immediately after completion of hatchlings, one hundred numbers (100) of hatchlings were randomly picked out from each combination and stocked in each aquarium with duplication making total of 18 aquaria. The aquaria was filled with water to the depth of 2/3 of the containers. During the indoor rearing for a period of 11 days, these fry were fed once daily with live mixed 200 plankton till the time of stocking out door. Daily survivals were monitored by individual counting of the fry.

At the age of 1 day, 70 numbers from each replicates of the combination were re-selected and stocked in 2m x 2m x 2m concrete tanks, the mean initial weight and length were taken at stocking. During the rearing out-door, the fry were fed with compounded 45% crude protein.

DATA COLLECTION

Data collections were made by taken weight and length on weekly basis for a period of 3 weeks. The length measurements were carried out with the aid of metric ruler while the weight measurements were carried out with Acculab 333 of 0.1g sensitivity.

RESULTS

Table 1 and Appendix 1 shows the percentage hatchability of eggs for nine generic groups. Among these crosses (group 1) which involves CP x MAI CP gave 82.5% hatchability. The cross involving NCP x NCP (group 2) gave 67.4% hatchability. The percentage hatchability in + Mai O Mai (group 3) gave 84.6%, which is the highest percentage of the hatchability. The cross involving CP x NCP (group 4) gave 73.8% hatchability. The cross involving Cp x MAI (group gave 76.3% hatchability. From (group 6) NCP x CP gave 75.4%. Group 7 involving NCP x MAI gave 73.5% while that of group 8 and 9 involving MAI x CP and MAI x NCP gave 70.1%, 72.2% respectively.

Table 1, figure 1, Appendix 2 and 3 shows the indoor and outdoor daily survival and percentage survival of fry for each day for nine generic groups. Group 1 CP x CP gave survival of 70% and 35.7% outdoor survival

value. Group 2 NCP x NCP gave 71.0% indoor survival and 37.1% outdoor survival value. In group 3 MAI x MAI gave 80% indoor survival and 25.7% outdoor survival value. The group 4 gave 88% indoor survival and 68.6% outdoor survival value, which is the highest percentage survival value recorded. From group 5 CP x MAI gave 81% indoor survival value and 54.3% survival value 56% while outdoor percentage survival value was 38.6%. From 7 NCP x MAI gave 63% for indoor survival value and 47.2% gave outdoor survival value. From group 8 the indoor value and outdoor survival value read 71% and 30% respectively. Group 9, the indoor percentage survival value 62% while outdoor survival value was 30%.

Table 1 figure 3 shows the percentage of cephalic abnormality value among the mating groups. The cephalic abnormality recorded in the group involving CP x CP gave 38.5% of cephalic abnormality.

In the group 3 involving MAI x MAI has zero % cephalic abnormality was as high as 70.8% which was the highest number of abnormality occurrence. In group 5 CP x MAI gave 34.2% as cephalic occurrence. The cross involving NCP x CP (group 6) gave 18.5% as the lowest cephalic abnormality. Group 7 NCP x MAI gave 36.4% cephalic abnormality. From group 8 involving MAI x CP give 47.6% cephalic abnormality. From group 9, MAI x NCP has no record of any abnormality.

Statistical Analysis

Student T-test was used to test whether there is significant difference in the indoor and outdoor survival, cephalic and non cephalic. It was observed that there is significant different. The mean is $p > 0.05$ level of significant which implies that there is no significant different between cephalic and non cephalic frequencies.

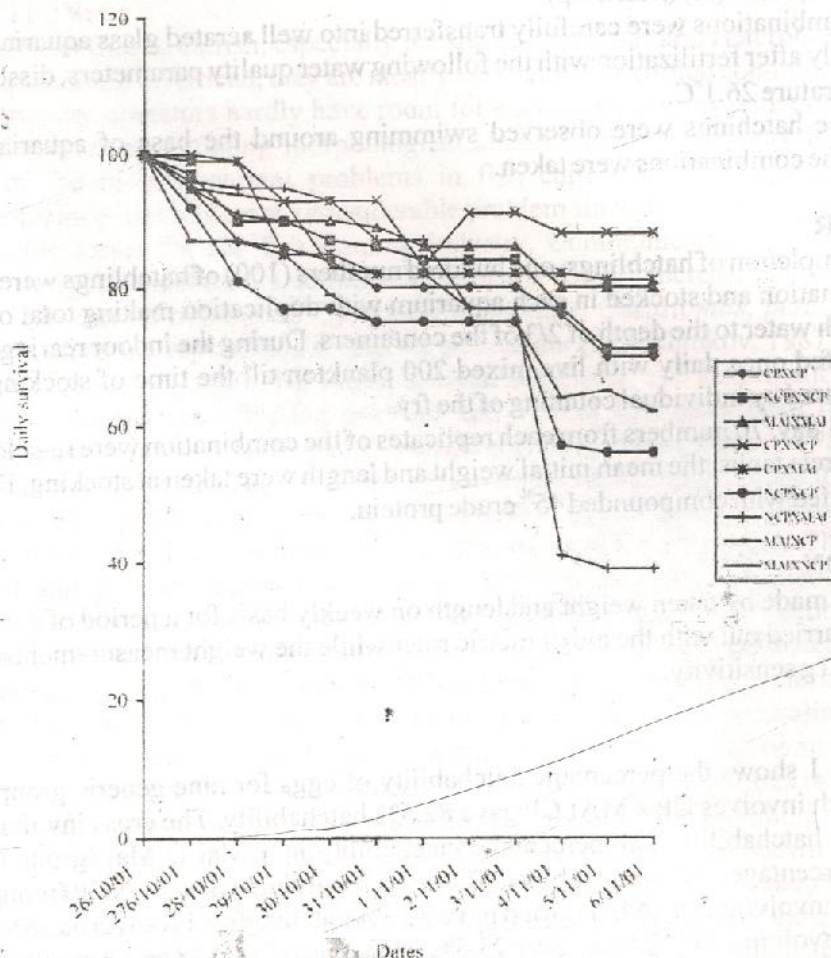


Fig. 1: Indoor monitoring of Cephalic fish and their daily survival

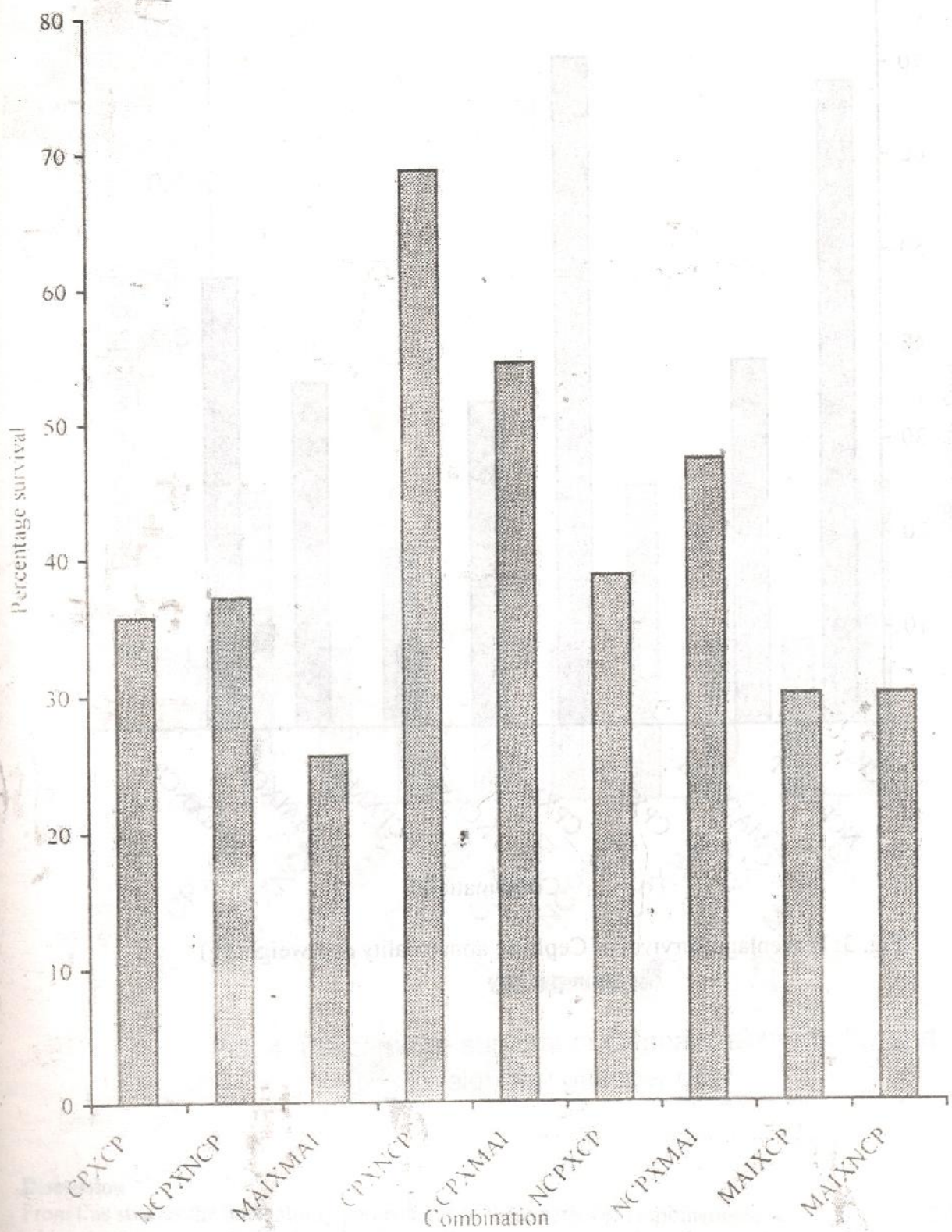


Fig.2: Final percentage survival of outdoor Cephalic fish

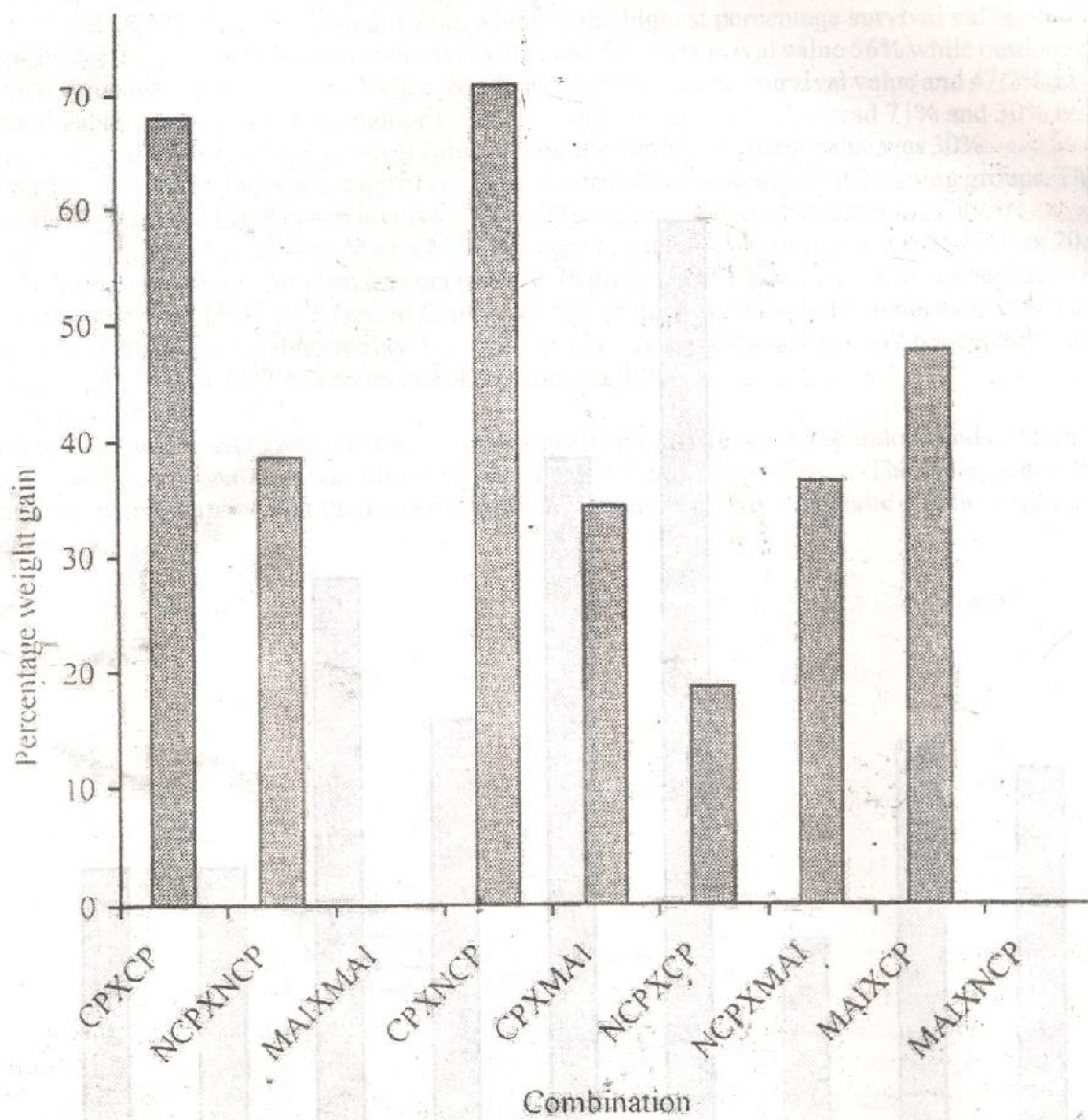


Fig. 3: Percentage survival of Cephalic abnormality and weight (g) gain per day

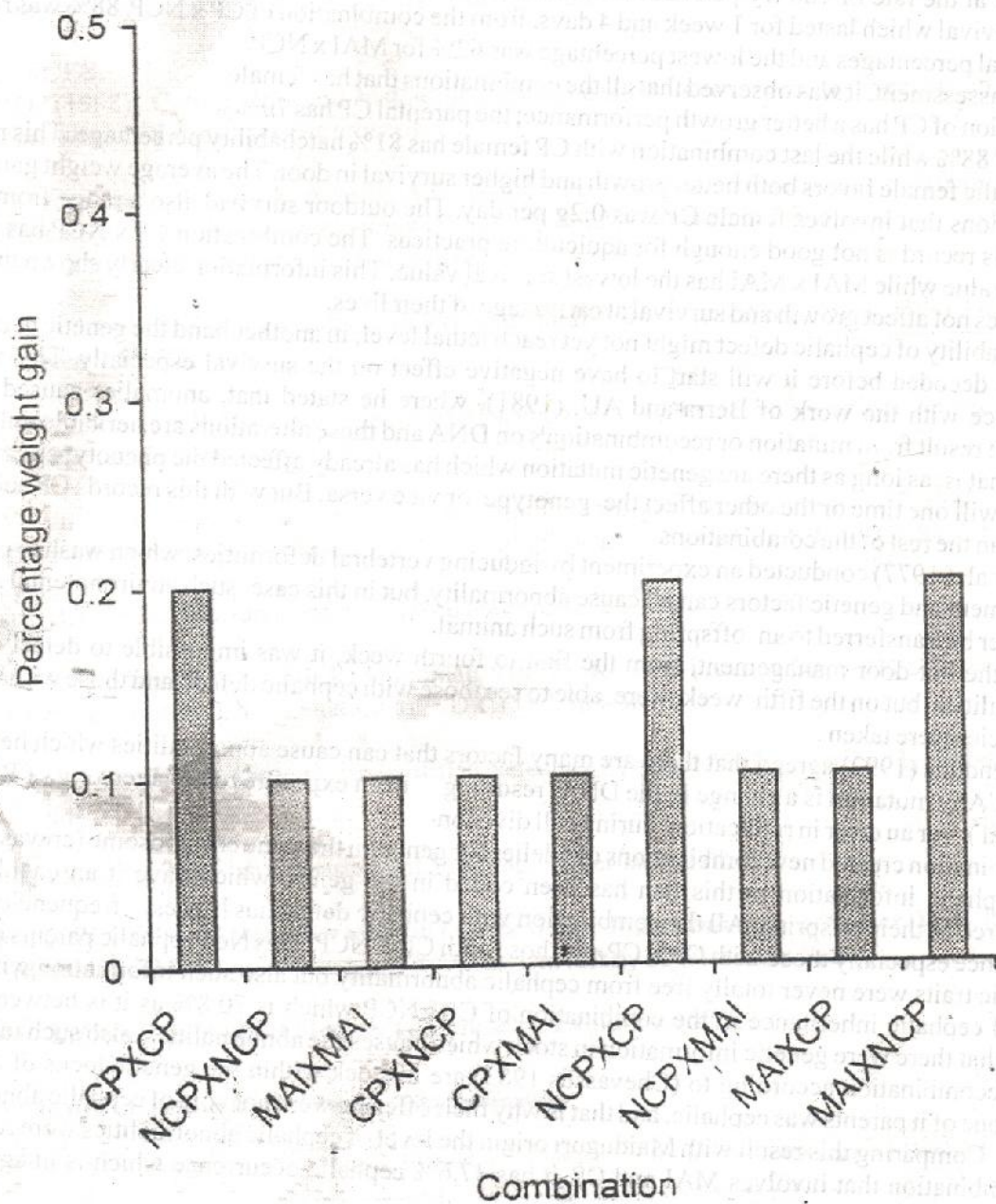


Fig. 4: Percentage survival of Cephalic abnormality and weight (g) gain per day

Discussion

From this studies the hatchability percentages of these series of combinations, Its ranges from 67.4 84.6% which the average was 76%, this records is still acceptable aqua culturally.

In table 1, the combinations that involves cephalic parents did well with the records of more than 70% hatchlings, likewise the other combinations that involves parental female; NCP performed well, but the MAI combines with others did not performed well like when MAI x MAI because from the hatchability results it has the highest percentage of 84.6%.

As regards to heritability of cephalic abnormality, it can not be established at this level or to conclude that the cephalic abnormality does not affect the hatchlings capabilities of each of those combinations that

involves.

After the hatching, the hatchlings were decentralized for proper monitoring and rapid growth in the laboratory at the rate of 100 fry per tank of 60cm x 30cm x 30cm³ in replicates from table 2, as regards to indoor survival which lasted for 1 week and 4 days, from the combination of CP x NCP, 88% was recorded as the survival percentages and the lowest percentage was 62% for MAI x NCP.

From the assessment, it was observed that all the combinations that has female Contribution of CP has a better growth performance; the parental CP has 70%, CP x NCP 88% while the last combination with CP female has 81% hatchability percentage. This result show that cephalic female favors both better growth and higher survival in door. The average weight gains in all the combinations that involves female CP was 0.2g per day. The outdoor survival also a range from 25.7% to 68.6% this record is not good enough for aquiculture practices. The combination CP x NCP has the highest survival value while MAI x MAI has the lowest survival value. This information clearly shown that cephalic defect does not affect growth and survival at early stage of their lives.

The heritability of cephalic defect might not yet reach lethal level, in another hand the genetic code (DNA) is yet to be decoded before it will start to have negative effect on the survival especially. This was also in accordance with the work of Berra and AU, (1981), where he stated that, anomalies caused by genetic alteration result from mutation or recombination's on DNA and these alterations are heritable unless they are lethal. That is; as long as there are genetic mutation which has already affected the phenotypes structurally of the fish will one time or the other affect the genotype or vice versa. But with this records CP and NCP were batter than the rest of the combinations

Couch et al (1977) conducted an experiment by inducing vertebral deformities, which wash to confirm that, environment and genetic factors can all cause abnormality, but in this case such environmental deformities will never be transferred to an offspring from such animal.

During the out-door management, from the first to fourth week, it was impossible to detect any form of abnormalities, but on the fifth week, were able to see those with cephalic defect and those without and their frequencies were taken.

Schapsenduns (1992) agreed that there are many factors that can cause abnormalities which heredity is one of them. Any mutation is a change in the DNA, resulting from exposure to mutagens e.g. (Radiation and chemical) or an error in replication during cell division

Recombination created new combinations of alleles for genes on the same chromosome (crevasses. 1983) This cephalic information in this fish has been coded in the genes, which gave it an enablement to be transferred to their offspring. All the combination with cephalic defect has highest frequencies of cephalic inheritance especially those with CP X CP and those with CP X NCP. This Non cephalic parents but having Cephalic traits were never totally free from cephalic abnormality but also such information with it from the level of cephalic inheritance in the combination of CP&NCP which is 70.8% as it is between CP x NCP shows that there were genetic information in stock which causes the abnormalities; also such information by DNA recombination according to (Chevassus 1983) are in stock within the genetic locus of Non cephalic which one of it parents was cephalic, and that is why their offering were not void of cephalic abnormality.

Comparing this result with Maiduguri origin the level of cephalic abnormalities were reduced. From the combination that involves MAI and CP, it has 47.6% cephalic occurrence which is in agreement with Mendel's experiment.

CONCLUSION

This work shows the comprehensive effect of cephalic abnormality on Africa catfish *Claria ganiepinus* and the possible ways of eradicating it without the use of prophylaxis medication. The aquacultural implication of whatever breeding programme we are carrying out is very important. In this case, in our world today we are so much careful on what we consumes, why because its side effect. Like it is generally believe that diseases are rarely transferred from fish to man, but when the deformity is glaring, everybody will like to run away from it. And let assume that more than 70% of table size fish from a particular fish farm are cephalic, there is a tendency that many of those fish will not be sold, and some of it will still die due to that cephalic abnormality defects. All these will amount to aquaculture loss. In another hands if such fish farm deals in fingerlings productions, there will be reduction in there patronage, because the moment their customer discovered that the fish bought from them are not acceptable and it also goes with mortalities which are all due to cephalic defects, in no time if correction is not done, such fish farm may fold up, which can also leads to many societal problems..

Now, how do we control or eradicate it, looking at the result of MAIXCP gave an indication that

cephalic abnormality can be eradicated and this can be achieved by proper breeding programme couple with understanding of genetic of selective breeding. Crossing the parent cephalic with other parent that does not have a sign of cephalic traces in their generation will reduces cephalic problem, but for total eradication, to break the heritability you dare not cross any fish with cephalic defects together but rather culled them.

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Appendix 1: Hatchability of the series of the combinations

S/No	Combinations	Dead eggs	Hatchling	Total no. of fertile eggs	Dead eggs (%)	Hatchability (%)
1.	CP X CP	80	377	457	17.5	82.5
2.	NCP X NCP	193	47.5	668	28.9	71.1
3.	MAI X MAI	79	435	514	15.4	48.6
4.	CP X NCP	174	490	664	26.2	73.8
5.	CP X MAI	105	330	435	24.1	75.9
6.	NCP X CP	144	350	494	29.1	70.9
7.	NCP X MAI	112	318	430	26.1	73.9
8.	MAI X CP	146	380	527	27.8	72.2
9.	MAI X NCP	101	335	436	23.2	76.8

Appendix 2: indoor monitoring of Cephalic fish and their daily survival.

S/No	Combinations	Day 1 no. stocke d	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12
1.	CP X CP	100	95	87	86	84	80	80	80	80	76	70	70
2.	NCPXNCP	100	97	90	90	87	86	84	84	84	77	71	71
3.	MAI X MAI	100	95	91	90	90	89	86	86	86	80	80	80
4.	CP X NCP	100	100	99	93	93	93	93	91	91	88	88	88
5.	CP X MAI	100	99	99	85	85	82	82	82	82	82	81	81
6.	NCP X CP	100	92	81	77	77	75	75	75	75	57	56	56
7.	NCP X MAI	100	96	95	95	93	87	87	77	77	41	39	39
8.	MAI X CP	100	95	94	93	83	82	82	77	77	77	71	71
9.	MAI X NCP	100	87	87	85	79	75	75	75	75	65	65	62

Appendix 3: weekly sampling and Cephalic Observation in outdoor concrete tank for 42 Days.

S/No	Combinations	1 ST WEEK					2 ND WEEK					3 RD WEEK				
		Sampled no.	Mean total wt(g)	Mean total l(cm)	Freq of CP	Freq of CP	Sampled no.	Mean total wt(g)	Mean total l(cm)	Freq of CP	Freq of CP	Sample d no	Mean total wt(g)	Mean total l(cm)	Freq of CP	Freq of CP
1.	CP X CP	28	0.04	1.5			21	0.4	4.0			19	1.5	5.0		
2.	NCPXNCP	33	0.05	1.5			22	0.4	2.8			23	1.6	4.9		
3.	MAI X MAI	39	0.05	1.6			17	0.3	3.0			14	2.0	5.2		
4.	CP X NCP	56	0.06	1.6			40	0.4	3.5			33	1.4	5.2		
5.	CP X MAI	41	0.06	1.7			36	0.4	2.9			30	1.8	5.4		
6.	NCP X CP	40	0.06	1.7			26	0.4	2.6			27	1.7	5.2		
7.	NCPX MAI	35	0.5	1.6			30	0.4	2.6			34	1.6	4.4		
8.	MAI X CP	36	0.5	1.7			20	0.3	3.2			17	1.7	5.7		
9.	MAI NCP	38	0.5	1.8			16	0.4	2.7			17	2.6	6.2		

Appendix 3: weekly sampling and Cephalic Observation in outdoor concrete tank for 42 Days.

S/No	Combinations	4 TH WEEK					5 TH WEEK					6 TH WEEK				
		Sampled no.	Mean total wt(g)	Mean total l(cm)	Freq of CP	Freq of CP	Sample d no	Mean total wt(g)	Mean total l(cm)	Freq of CP	Freq of CP	Sample d no	Mean total wt(g)	Mean total l(cm)	Freq of CP	Freq of CP
1.	CP X CP	18	2.6	6.0			19	4.6	8.0	6	10	17	5.9	8.2	8	9
2.	NCPXNCP	12	2.3	6.4			11	3.9	6.6	1	9	20	5.6	8.2	2	17
3.	MAI X MAI	22	2.5	4.9			10	3.6	7.4	Nil	10	15	4.5	8.8	Nil	15
4.	CP X NCP	23	1.8	6.0			21	3.3	7.2	3	18	27	4.1	7.7	24	3
5.	CP X MAI	26	2.7	6.9			13	3.8	6.9	Nil	13	31	4.4	8.1	3	28
6.	NCP X CP	25	2.8	6.1			15	4.4	6.9	6	10	20	3.1	7.8	Nil	20
7.	NCPX MAI	27	2.2	6.0			21	3.0	6.1	3	20	28	3.9	7.2	2	26
8.	MAI X CP	14	2.3	5.2			12	3.0	7.4	3	10	16	4.1	7.1	3	13
9.	MAI NCP	14	3.6	7.2			14	5.0	8.7	3	13	19	6.7	9.6	4	17

