THE CHICKEN OR THE EGG: NURTURE AND NATURE: NEW GENETIC SPREADSHEETS AND GENE POOLS IN THE BREEDING AND EVOLUTION OF A NEW NIGERIA-MAN

INAUGURAL LECTURE SERIES

10

FEDERAL UNIVERSITY OF TECHNOLOGY, OWERRI
17TH NOVEMBER, 2004

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THE CHICKEN OR THE EGG: NATURE AND NURTURE; NEW GENETIC SPREADSHEETS AND GENE POOLS IN THE BREEDING AND EVOLUTION OF A NEW NIGERIA-MAN.

Inaugural Lecture by

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1.0 PREAMBLE

The occasion of today’s Inaugural marks yet another important milestone in an odyssey which for me started precisely on Friday the 7th of October, 1960; the day I first dreamed of and decided that indeed I will become a PROFESSOR.

That was more than 44 years ago, but I still vividly remember it just as if it was merely yesterday. The occasion on that eventful day was the official opening of the very first, fully-fledged, autonomous university in our country, the University of Nigeria, Nsukka. I was then merely a little over 10 years old, and as was usual for most children of my generation, I helped contribute to my family’s food basket by selling a variety of confectionary items which were baked by my mother. The tray which I carried on my head on that day was luxuriantly emblazoned in full colours with the smiling pictures of Her Majesty Queen Elizabeth II, the Imperial Head of the Commonwealth; His Excellency, the Rt. Hon. Dr. Nnamdi Azikiwe, the then Governor General of the Federation; the red-blue-and-white colours of the Union Jack; and the green-white-green flag of the new Nigerian nation. My tray was filled with a variety of wares such as cup cakes, one-penny loaves of bread, doughnuts, chin-chin, etc. Of course, the large crowd which had gathered to witness the official opening of the University presented a very good market for my wares.

However, a different deja-vu awaited me that fateful morning. Like the Paul of Tarsus, I met with my destiny.
As I stood in the crowd behind the mobile, barricaded, white painted fences; which were manned by policemen who were smartly clad in their starched and neatly ironed khaki shorts and shirts; I was stupendously enraptured, benumbed, stunned, amazed and irrevocably captivated by the galaxy of beautifully robed men and women, who moved with majestic dignity in the academic procession that flowed down the road, towards the Convocation Arena. These men (and few women) wore the most beautifully coloured robes I had ever seen. They processed in measured strides, some with the air of ecclesiastical authority, others with the air of imperial dignity. Each carried what looked like a scroll in their left hand. They would greet or acknowledge one another with a gentle bow and by tipping and doffing their gold tasselled mortar-board caps with dignified elegance. They carried the graceful airs and epitome of intellectual detachment. It was impressive. My young imaginative mind was bowled over.

I had never seen anything like this. When I asked "who are these men and women?" I was told in very respectful whispers, "they are P.R.O.F.E.S.S.O.R.S; Professors of the university!!" As I continued to stare at them, my stupefaction turned into admiration; my admiration turned into daydreaming; and I longed to be like one of them. "it would be nice to be like one of them. Yes, I will be one of them" and there started my dream.

But that was not all

As I flowed with the crowd into the Convocation Arena, I beheld yet another great spectacle. One of these professors was standing on the
central podium; addressing the Convocation in what obviously (judging by the thunderous applauds he received), was indeed a very great speech. I was later to learn that the speech was a citation delivered with the great oratorial panache reminiscent of Marcus Tullius Cicero, the great Roman Orator. The professor spoke with dignity and eloquence, and with a mastery of the English language that I am yet to hear paralleled. He laced his citation with renditions both in Latin, in French, Spanish, German, Creole, and some other East European language. Who was this Professor? He was Professor Edward Blyden, Ill; the very first University Orator at the University of Nigeria.

Those who know Professor Blyden Ill, or have had the privilege of hearing him speak, will attest to this man's great oratorial talents. He inspired me. Now, I didn't want to stop at just being a professor; I also wanted to be a University Orator, just like Professor Blyden. And so started my dream.

Ladies and Gentlemen, I stand here today to witness that precisely thirty years later, both dreams came true for me. With the peer review stamp of the intellectual community of scholars, and by the authority of the Senate and the Governing Council of the Federal University of Technology, Owerri; I was invested with the tenured Professorial Chair of Animal Genetics & Breeding in October 1990, and was later that year appointed as the University Orator—a duty which I very ably discharged in this University for more than 10 years. My dreams had come true.

My dear students, respected ladies and gentlemen: this Preamble has therefore had a purpose. I stand here today in eloquent testimony that
success indeed awaits all those who dare to dream; and who dare to work hard in pursuit of their dreams. I stand here today, with my most heartfelt sincerity, to say to every individual in this assembly, MAY YOUR OWN DREAMS COME TRUE, in Jesus' name, Amen.

With this Preamble, I also pray and hope that maybe, there could be just one person; one young adult; who may be by the way I deliver this Inaugural; or by the way I have been delivering my professorship in this University and in the wider world of learning; would be impressed and inspired to dream like I dreamed 44 years ago; to pursue his destiny to the greatest of his ability. My dreams would then have sired a protégé.
2.0 INTRODUCTION

2.1 The Concept and Origin of Inaugural Lecture.

It has been suggested that the earliest form of the university originated in Egypt and in the early civilizations of the Asia Minor. However, the concept of the university as we know it today owes its origin to the old universities of Europe. It evolved from the medieval schools known as studia generalia which were generally recognized places of study, sequestered in a section of a city, and open to all scholars from various parts of Europe and beyond. The word universitas originally applied only to the scholastic guild (or guilds) that is, the corporation of students and masters within the studium. It was modified as universitas magistrorum or universitas scholarium, or universitas magistrorum et scholarium. In the course of time, probably during the later part of the 14th century, the term began to be used by itself, with the exclusive meaning of “a self-regulating community of teachers and scholars whose corporate existence had been recognized and sanctioned by civil or ecclesiastical authority”.

This early concept of a university, which has been ably narrated by Iloeje (2004) extols a tradition of loyalty of discipleship in which there was a master (i.e. a professor) and his pupils. Learning took the form of an apostolic followership. The master and his pupils formed a guild of scholars. Education was learning through a pupilage or an apprenticeship of training in the scholarship of chosen disciplines in the sciences, art, philosophy, logic, etc. (erziehung durch wissenschaft).

This “self regulating community of professors and scholars whose
corporate existence had been recognized and sanctioned by civil or ecclesiastical authority" was usually domiciled or sequestered in specified location in a city. However, as the number of such guilds of scholars increased, and as the frontiers of knowledge expanded, it became expedient that new professors establish new guilds, or extend their discipleships into new communities or cities. Probably as a way to earn the required civil or ecclesiastical authority to establish such new community of scholars, or to stake a claim to attract new discipleships and apostolic followships, or both; new professors usual took an early opportunity to demonstrate their skills to both townsfolk and scholars alike, in a formal ceremony to inaugurate, induct or commence their scholastic guild within a studium or within a universitas scholarium. This academic tradition is what has metamorphosized into the professorial inaugural lecture as it exists today in the Nigerian university system.

It has been necessary to narrate this history so that the older ones amongst us may not forget, and also to assist our younger colleagues to situate the inaugural lecture in its proper context and form.

2.2 Objective and Purpose of this Inaugural

As ably said by Ugbolue (1996), an Inaugural lecture presents different opportunities to different persons. To some, it offers an opportunity for a newly appointed professor to demonstrate his perception of his new role and share his expertise and vision of his subject and specialism. To others, it may be a time to forge and foster the much desired town-and-gown interaction and encourage greater appreciation and understanding of the professor’s discipline, while stimulating interest in the subject area. To a few, it is simply an age-long ritual in the academic almanac of a university
aimed at promoting intellectual discourse and socialization. Simply put, albeit a little bit hyperbolically, the audience may have come to expect the Inaugural lecture as an opportunity for the lecturer to exhibit a collection of his works and sound-off by telling us “come and hear me justify my various accomplishments which have earned me a professorship”

For me, this Inaugural offers a unique opportunity to use the skills of my training and experience as a Researcher and Professor of Animal Genetics & Breeding to chart a new important vista for our general genetic education and sharpen our awareness for increased genetic counseling by pointing out to both my learned colleagues, to our community of scholars, and more importantly to everyday ordinary townfolks in this great country, that there is a new genetic trend and a shift in the gene pool and in our breeding patterns which almost imperceptibly, are slowly but surely leading to the gradual evolution of a new Nigeria-man.

Yes, a new Nigeria-man is evolving; with a new genetic hardiness, adapted and maybe tailor-fit to cope, survive, harness, and adapt to the demands and consequences of our environment. Is this as a result of a change in our “nature or nurture”; or a consequence of our quest to resolve the age-old dilemma of “the chicken or the egg”?

Certainly, there is a Geneticist in everyone of us, and really we all are animal breeders. It doesn’t matter whether like our colleagues in the Department of Animal Science you breed cows, goats, pigs, chicken etc; or whether as what goes on every night in most bedrooms you breed humans, albeit in perpetuation of the species. If we must harken to the biblical injunction to “increase and multiply”, the genetic concepts are the same, and the rules of engagement, similar!!
3.0 LARGE RANDOM MATING POPULATIONS

I will begin my task this morning by first of all explaining and describing what is generally referred to as a large random mating population. Then I will proceed to show and explain that in genetic definitions, Nigeria indeed is a panmictic population; which obeys the general genetic laws governing large random mating populations; and is therefore subject to its genetic consequences, whether desirous or deleterious.

In order to carry along with me everybody in this audience, even if it is at the expense of boring some of the very learned geneticists and biologists among us; let me commence by explaining in very simple terms some of the elementary rudiments of the mechanisms of heredity.

In searching for the mechanisms of heredity, geneticists at the turn of the 19th century discovered and established the Mendelian principles from studies at the familial level. They limited their investigations to the likeness or unlikeness between specified parents and their offsprings. In a simple Mendelian study, for example, they might cross a purple flowered plant with a white flowered one and examine the proportions of the various kinds of plants arisen from the seeds. However, when we wish to study the genetics of large populations, like Nigeria, population geneticists on the other hand, are concerned with the statistical consequences of Mendelism in a group of families or individuals: they study the hereditary phenomenon on a populational level. Of course, the mechanism of heredity is presumed to be what Mendelian genetics has described. The population geneticist then goes on to investigate, as in the above example, the proportions of purple and white-flowered plants in a given region, the frequencies of the various kinds of plants from each type of cross and the genetic composition.
of one generation as compared with that of the next generation under various circumstances. The same thing in plants, follows, mutatis mutandis, for human populations.

The life of an individual is limited in length of time. Barring mutations his genetic makeup is fixed throughout his life. In contrast, a population is practically immortal, may be large or small in size, may be distributed over a large or limited area, and may change in genetic composition from generation to generation, suddenly or gradually. The study of the genetics of populations is thus inevitably related to that of organic evolution, which, from the genetical viewpoint, is but a process of cumulative change in the heredities characteristic of the species. The human population in Nigeria is typical of this afore described genetic population. It is genetically immortal, large in size, distributed over a large geographical area, and as this lecture will show, is indeed, in the genetic viewpoint undergoing the usual and gradual processes of cumulative changes in the heredities characteristic of our specie.

I will commence my thesis with the simplest but most frequently encountered case ie, that of autosomal genes of diploid organisms practicing bisexual reproduction in a large population where mating is free and random. There is a very important landmark law that governs the genetics of such populations. However, before I go on to explain that law, I think that for the benefit of some of the non-biologists among us, it may be necessary to explain a few of the terms implicit on that law.

3.1 Gene Frequency

In order to explain this term in the simplest and most rudimentary way, I
will take a simple case where there are only two alleles (A, a) at a particular locus. We shall suppose that there are N diploid individuals, of which D are dominant (AA), H are heterozygous (Aa), and R are recessive (aa); where $D + H + R = N$. To designate such population of individuals, I will subsequently adopt, for the sake of brevity, the following symbols: (D, H, R) where the three numbers are always understood to be in the order of AA, Aa, aa. Although there are three types of individuals in this population, there are only two kinds of genes: A and a. Because they are diploid, these individuals have $2N$ genes altogether. Since each AA individual has two A genes, and each Aa individual has one A gene, the total number of A genes in this population is $2D + H$. Therefore, the proportion of A genes in this population is:

$$p = \frac{2D + H}{2N} = \frac{D + 1/2H}{N}$$

This proportion is known as the gene frequency of A in this population. Similarly, the frequency of the gene a in this group is $q = (H + 2R)/(2N) = (1/2H + R)/N$; so that $p + q = 1$. For example, in a population of 40 individuals: (2, 12, 26), $p = (2 + 6)/40 = 0.20$; and $q = (26 + 6)/40 = 0.80$. Frequently, the three genotypes are given in percentages instead of in actual numbers, especially when the population is large. More rigorous treatment of this treatise can be found in Li (1955), Srb et al (1965), Sutton (1975), Falconer (1976) and Lush (1948).

In the genetic sense, the population I have described here is not just any group of individuals, but a breeding group. We are interested therefore not just in the genetic constitution of the individual but also with the transmission of the genes from one generation to the next. In the
transmission, the genotypes of the parents are broken down and a new set of genotypes is constituted in the progeny. The genes carried in the population thus have continuity (i.e., immortality) from generation to generation, but the genotypes in which they appear do not. The genetic constitution of that particular population, referring to the genes it carries, is described by the array of gene frequencies.

3.2 Random Mating.

Many organisms in nature, both animals and plants, seem to mate at random or nearly so. In the case of bisexual organisms, a formal definition of random mating is that any one individual of one sex is equally likely to mate with any individual of the opposite sex. In other words, the frequency of a certain type of mating is random and dictated by chance. Thus, if the mating is completely at random in the population we described above (D, H, R), the frequency of AA X AA would be D² among all matings, assuming that the genotype proportion in both sexes are the same. The various types of mating and their frequencies is shown in Table 1.

<table>
<thead>
<tr>
<th>Females</th>
<th>Males</th>
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<tbody>
<tr>
<td></td>
<td>AA</td>
</tr>
<tr>
<td></td>
<td>D</td>
</tr>
<tr>
<td>AA</td>
<td>D.....</td>
</tr>
<tr>
<td>Aa</td>
<td>H.....</td>
</tr>
<tr>
<td>Aa</td>
<td>R.....</td>
</tr>
</tbody>
</table>

I should however emphasize that the above theoretical frequencies will
be realized only in very large populations. The term “panmixia” is sometimes used as a synonym of random mating, and then the population is said to be panmictic.

3.3 Hardy-Weinberg Law

Continuing our example in 3.1 above, if in our large panmictic population, an allele \( A \) has a frequency \( p \); while the allele \( a \) has a frequency \( q \); where \( p + q = 1 \), then the chance combinations of gametes through random mating in this population will produce in the next generation the frequencies as already shown in Table 1. The distribution of the genotypes is therefore approximated in a single generation of random mating, and it is maintained in all successive generations. A population having this kind of genetic stability of structure is described as being in genetic equilibrium. This phenomenon is often referred to as Hardy-Weinberg law of genetic equilibrium, so named after its co-discoverers.

For the benefit of the engineers among us, and all those who are gifted mathematically, the law of genetic equilibrium can also be best explained by considering a large panmictic population in which the frequency of \( A \) is \( p \); and frequency of \( a \) is \( q \); such that \( p + q = 1 \). If the proportions of these three genotypes with respect to this pair of genes in the population are:

\[
D = p^2; \quad H = 2pq; \quad R = q^2;
\]

the genotypic proportions in the next generation will be the same as those in the preceding generation. The population \((p^2, 2pq, q^2)\) is then said to be in genetic equilibrium under the system of random mating.

By “equilibrium” we mean that there is no change in the genotypic proportions in that population from generation to generation. This implies also no change in the gene frequencies. Table 2 illustrates this phenomenon.
TABLE 2. ESTABLISHMENT OF EQUILIBRIUM UNDER RANDOM MATING

<table>
<thead>
<tr>
<th>Type of mating</th>
<th>Frequency of mating</th>
<th>Offsprings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>AA</td>
</tr>
<tr>
<td>AA x AA</td>
<td>D^2</td>
<td>D^2</td>
</tr>
<tr>
<td>AA x Aa</td>
<td>2DH</td>
<td>DH</td>
</tr>
<tr>
<td>AA x aa</td>
<td>H2</td>
<td>1/4 H^2</td>
</tr>
<tr>
<td>Aa x aa</td>
<td>2DR</td>
<td>-</td>
</tr>
<tr>
<td>Aa x aa</td>
<td>2HR</td>
<td>-</td>
</tr>
<tr>
<td>aa x aa</td>
<td>R2</td>
<td>-</td>
</tr>
<tr>
<td>TOTAL</td>
<td>1.00</td>
<td>(D + 1/2 H)^2 = p^2</td>
</tr>
</tbody>
</table>

3.4 Changes In Genetic Equilibrium

The genetic stability asserted above is of course not a matter of absolute and immovable fixation. Factors act to move, change or modify this particular random equilibrium and to change the relative frequencies of genes and genotypes from generation to generation. The most important three of such factors are mutation, selection and migration. Recurrent mutation from A to a, for example, serves to increase the value of q at the expense of p; and this may be opposed by the pressures of reverse mutation, from a to A. If we were to consider only the values of p and q for many generations, we would find that under the counter balancing pressures of mutations and reverse mutations, an equilibrium for the value of p will be achieved. The point at which this value stabilizes is determined by the relative rates of mutation and reverse mutation for the particular locus (gene) under consideration.

Similarly, selection in which one genotype or phenotype leaves (or is permitted to leave) fewer progeny than others, acts to modify the relative frequency of alleles from generation to generation. For example, if
individuals of blood type \( M \) in Mbaise were sterile or partly sterile, or barred from marrying in Mbaise, then the relative frequency of \( M \) will decrease, while the frequency of \( N \) will increase in later generations in Mbaise. In the same way, migration of itinerant and nomadic Fulanis with blood type \( N \) into Mbaise will, under random mating conditions, produce a relative increase in the frequency of \( N \) in Mbaise. Let us now consider more fully the impacts of mutation, migration, and selection in genetic populations.

**Mutation.**

Gene mutations are naturally occurring sudden and spontaneous changes that take place in the gene from time to time. They are the ultimate sources of new alleles and thus genetic variability in populations. Most mutations are spontaneous although some can be induced. The process of mutation generates allelic variations which make evolution possible. However, new mutations change gene frequencies very slowly. This is because mutation rates are low.

Let us illustrate this by assuming that the gene allele \( A^1 \) mutates into allele \( A^2 \) at the rate of \( m \) per generation, and that at a given time the frequency of \( A^1 \) is \( p \). In the next generation, a fraction \( m \) of all \( A^1 \) allele will become \( A^2 \) alleles. The frequency of \( A^1 \) in the next generation will then be reduced by the fraction of mutated alleles \((pm)\), or \( pl = p \cdot pm = p(l-m) \). After \( t \) generations, the frequency of \( A^1 \) will be \( pt = p(1-m)^t \). If the mutations continue, the frequency of \( A^1 \) alleles will gradually decrease, because a fraction of them changes every generation to \( A^2 \). If the process continues indefinitely, the \( A^1 \) allele will eventually disappear, even though the process is slow. If the mutation rate is \( 10^{-3} \) (i.e. 1 in 100,000) per gene per generation, about 2,000 generations will be required to change the frequency of \( A^1 \) from 0.50 to 0.49; and about 10,000 generation to change it from 0.10 to 0.09.
However, remember that gene mutations are also reversible. The allele A2 may also mutate to A1. As before, assume that A1 mutates to A2 at a rate \( m \); and that A2 mutates to A1 at a rate \( n \) per generation. If at a certain time the frequencies of A1 and A2 are \( p \) and \( q \) respectively; then after one generation the frequency of A1 will be \( p'/p = p \pm m + q \). A fraction \( pm \) of allele A1 changes to A2; but a fraction \( qn \) of A2 changes to A1. The condition of equilibrium will occur when \( pm = qn \) or \( p = m/(m + n) \). Suppose that the mutation rates are \( m = 10^{-5} \) and \( n = 10^{-5} \); then at equilibrium, \( p = 10^{4}/(10^{4} + 10^{5}) = 1/(1 + 10) = 0.09 \), and \( q = 0.91 \). Expatriations of this theme were amply demonstrated by Illoeje (1982), Illoeje (2000) and Illoeje and Iniguez (2003).

I have used the above mathematical illustrations to show that changes in gene frequencies due to mutations occur, albeit very slowly. The slow pace is also further slowed down because forward and backward mutations counteract each other. In any case, allelic frequencies are not in mutational equilibrium, because some alleles are favoured over the others by natural selection. The equilibrium frequencies are then decided by the interaction between mutation and selection, with selection usually having the greater consequence.

**Migration**

Gene flow, or gene migration, takes place when individuals migrate from one population to another and interbreed with its members. Gene frequencies are not changed for the species as a whole, but they change locally whenever different populations have different allele frequencies. Illoeje (2002) demonstrated that generally, the greater the difference in allelic frequencies between the resident and the migrant individuals, and the larger the number of migrants, the greater the effect which the migrants will have in changing the genetic constitution of the resident population. Suppose that a proportion of all reproducing individuals in a
population are migrants and that the frequency of allele $A$ is $p$ in that population, but $pm$ among the migrants. The change in gene frequency, $Dp$, in the next generation will be $Dp = m(pm - p)$. If the migration rate persists for a number $t$ of generations, the frequency of $A$ will be given by $pt = (1 - m) t (p - pm) + pm$. The most obvious effect of continued intergroup migrations is to make the gene frequencies of the various groups more nearly alike, and thus, in the absence of any countermeasures, render the total population more homogenous.

**Selection**

Selection is said to operate when there is inequality in fitness of genotypes. Fitness is to be understood here in a broad sense which is measured ultimately in terms of the numbers of viable offsprings produced. A person may be fit physically and functionally in that he is healthy and may contribute to the survival and perpetuation of the population; but if he fails to leave some offspring, he is scored as "genetically dead".

It is common to speak of positive or negative selective advantage. An allele can be said to have positive selective advantage if it leads to greater fitness than the alternate allele under consideration. It has negative selective advantage if it leads to lesser fitness. Favourable selection are the ones which increase chances of survival and procreation. Selection, whether natural selection or artificial selection ensures that advantageous genes are preserved and multiplied from generation to generation at the expense of the non-advantageous ones.

One of the genetic consequences of selection is the production of an individual that is well adapted to his environment, and evolution often occurs following this consequence. Selection may be due to differences in survival, in fertility, in rate of development, in mating success, or in any other aspect of the life cycle. Darwin maintained that competition for
limited resources results in the survival of the most effective competitor. Natural selection can also occur not just as a result of competition, but also as a result of other environmental factors such as inclement weather, disease outbreak, war, famine etc. Above all, natural selection would also occur even if all the individuals in a population died at the same age, simply because some of them would have produced more offspring than others, before dying.

3.5 Gene Pools And Genetic Spreadsheets
An individual of a sexually reproducing species, such as humans, is not biologically complete in itself. Its biological role is actualized through its membership in a reproductive community, a Mendelian population. A Mendelian population consists of individuals among whom matings may and do occur. An individual is mortal and temporary, however his Mendelian population is immortal and has continuity through time.

Any such Mendelian population is said to have a gene pool. The gene pool is the sum total of the genes carried by the individual members of the population. The gene pool also continues through time. The genes of the individuals of the generation now living come from a sample of the genes of the previous generation. If these individuals reproduce, their genes will pass into the pool of the following generations.

The Mendelian population and its gene pool in humans have a very complex structure. Individuals born and living close together are more likely to meet and to mate than those living far apart. In a widely distributed species such as Homo sapiens, the likelihood of mating individuals born on different continents was, until the development of modern means of travel, very small. The gene pool of the human species is, accordingly, divided into the smaller gene pools of races, and even further into tribes, linguistic groups, and so on. In addition to these
geographical divisions, there are also linguistic, religious, economic, social, and educational barriers that break the gene pools into further, often overlapping, subdivisions. The smallest subdivision is referred to as an isolate panmictic unit; it consists of a relatively limited number of persons that may be regarded as potential mates. Few of these subdivisions may be sharp enough to decide where one gene pool ends and where the other begins. Yet these subdivisions are genetically meaningful.

The limits of the human gene pool are without any doubt specific, since no gene exchange between humans and any other species takes place. The intra-specific differentiation between humans does not impair this unity and integrity of the human gene pool. There may never have been a marriage, for example between an Ngwa woman and an Eskimo; however, the genetic communications between the Eskimo gene pool and the Ngwa gene pool occur through the chains of geographically intermediate populations. The corollary is the same between the tribal gene pools within Nigeria.

Thus, a genetic change arising anywhere in the world, if favourable, may spread throughout humanity. *Ipso facto*, a genetic change arising from any one tribal gene pool in Nigeria will, with time, spread to all the remotest gene pools in every tribe in this country. This is how genetic changes may have transformed the ancestral prehuman species into the present one. So also are how the genetic changes in the various gene pools in this country are gradually, imperceptively, but surely gelling us into genetic unity and evolution of a new Nigeria-man.

Let me illustrate this with an example. Consider, for example, a particular gene such as the one determining the MN blood groups in humans. One
form of this gene codes for the M blood group, while the other form codes for the N blood group. The gene pool of a particular population is specified by giving the frequencies of the alleles M and N. According to a recent World Health Report the M allele in Nigeria occurs with a frequency of 0.539 and the N allele with a frequency of 0.461. In other population, these frequencies are different; the frequency of the M allele is 0.917 in Navajo Indians, and 0.178 in Australian aborigines (WHO, 1998)

A menu or potpourri of gene pools acting in concert within a specified panmictic cognate population is what I have referred to in this discussion as genetic spreadsheet.

3.6 Breeding Patterns

There are very many types of mating systems and breeding patterns available. We will not list all of them here. However, the ones that readily come to mind can be generally grouped as various forms of outbreeding and inbreeding. Outbreeding is the opposite of inbreeding. It includes all systems of mating where the mates are chosen so that they are less closely related to each other than if they were paired at random. On the other hand inbreeding is the mating of individuals who are related to each other more closely than the average relationship within that population. They can occur variously as follows:

Selfing

The simplest and most extreme form of inbreeding is selfing, ie self-fertilization, or identical genotypes. (such as in monozygotic twins). The correlation between mates is thus perfect, ie equal to 1. This sort of breeding is uncommon in humans.
Sib Mating
For dioecious organisms, the most intensive system of inbreeding is the mating between full-sibs, usually known as brother sister mating. The properties of this system of inbreeding have been studied by very many geneticists, and are of importance in both theoretical and applied genetics, especially in animal breeding. Although it is not common in human populations, many reports do document that it was practiced in some royal families or in some human communities who were overtly fixated on purity of bloodlines.

Parent Offspring Mating
Parent offspring mating can occur in any of two broad forms. The first type is the mating between a fixed sire and his daughter, grand daughter, great grand daughter, etc. The second type of parent-offspring mating is that in which each individual is mated successively with his (her) younger parent and with his (her) offspring. Parent offspring mating is not common in human populations. It however is important in animal breeding.

Half- Sib Mating
This involves a situation in which one male (or female) is mated with his half-sisters and half-brothers of each other. Again, this type of mating is not common in human population, although animal breeders find it very useful.

Mating Between Cousins
Such matings could be between first cousins, second cousins, double-first cousins, or even quadruple-second cousins. See figures 1 and 2. In which ever form it takes, the typical point of note
Fig. 1 Mating between double first cousins

Fig. 2 Mating between single first cousins
Is the close consanguinity between mates. Although not very widespread, it cannot be denied that this form of mating occurs, to varying degrees, in human populations.

Mating Between Remote Relatives

This is the mating of individuals from the same gene pool in a population with overlapping familial generations and traceable consanguinity. Despite all pretensions to the contrary, we are sure that this sort of mating occurs in human populations. The only debate might be on their frequency.

One important genetic conclusion from these various types of matings is that geneticists are in uniform agreement that continued matings between relatives more remote than first-cousins cause only an insignificant decrease in the heterozygousity of the population. Genetically speaking, consanguineous matings produce a better chance for the appearance of rare recessive gene than do random mating. Where such a recessive gene is deleterious (or beneficial), society suffers (or enjoys) the consequence. In human populations, the rubicon or the maginot line in the fiat designating whom to mate and whom not to, is usually determined by religious, cultural, or social norms and not by genetic etiquettes.

3.7 Effective Population Number

In a natural population, the total number of individuals of all ages may be very large, but not every individual reaches sexual maturity and not every individual mates. Even those that mate may not necessarily leave offspring that survive to maturity in the next generation. No matter what their genotypes may be, those that leave no offspring will have nothing to do with the genetic composition of the next generation. The number of actual progenitors (breeding size) responsible for the genetic constitution of the
next generation may be less than the number of individuals living at
anytime in a population. In furtherance of this thesis, Illoeje (1974) and
Illoeje and Akhiobare (2000) in their dissertations on Numerical Studies on
the Genetic Properties of Randomly Generated, Finite, Panmictic, Diploid,
Populations, posited that for the perpetuation of the species, the effective
population number can be defined as the average number of individuals in
a population which actually contributes genes to the succeeding
generation.

In the above-going discussions and the corollaries drawn from them, it is
obvious that the Nigerian human population indeed obeys the general
genetic laws governing large random mating (panmictic) populations, and
is therefore subject to the genetic consequences; desirous or deleterious.
Several gene pools marked by tribal, or linguistic, or cultural, or religious
boundaries can be identified. Example, it is undoubted that the nomadic
(or transhumant) Fulani cattlemen constitute a gene pool rarely or most
infrequently intermingled with genetic infusions from outside. However,
it is also true that the frequent inter-marriages between and among the
various population groups in Nigeria, occasioned by increase in education
and improvements in transportation and social mobility, have all
combined to erode the genetic boundaries and produce a new Nigeria-
man, more genetically equipped to adapt to the varied consequences and
vagaries of the new Nigeria.
4.0 APPLICATIONS OF GENETICS TO HUMAN PROBLEMS: IMPLICATIONS IN EVOLUTION OF THE NEW NIGERIA-MAN

In the preceding argument, I have posited that genetically a new Nigeria-man is slowly but surely evolving. In order to properly explain this evolutionary process, I had proceeded to define some genetic parameters and characteristics of large random mating panmictic populations, such as Nigeria. Various genetic phenomena such as gene frequencies, panmixia, genetic equilibrium, gene pools, breeding patterns, and genetic spreadsheets were defined in order to use them to properly explain and witness to this on-going process of evolution.

The concept of the distribution of gene frequencies is indeed very very important in any discussion on biological evolution at the population level, and the change in gene frequency must be considered as an elementary step in that direction. The distribution forms of these frequencies depend upon the relative magnitudes of the various agents which bring changes in gene frequency.

In large random mating populations, all gene frequencies remain at a certain relatively stable equilibrium until they are perturbed by the systematic pressures of mutation, selection and migration. In response to these pressures and to other substantial changes in the environment, the population will continuously seek to re-adjust by continuously evolving. As conditions continue to change, the total population, with its diversified and genetically flexible subgroups, will thus be able to cope with the new situation instead of facing extinction.
It is however clear that there is no all-important single factor in evolutionary change. The type and magnitude of evolutionary change in a population depends on what Sewel Wright (1931 and 1932) called the "breeding structure" of that population. As we had earlier discussed, these include such factors as effective population size, inbreeding, mutation, selection, migration, and the absolute value of the gene frequency. The most favourable condition for evolutionary advance is that in which all these forces act in concert and are properly balanced with one another, in certain ways and under certain conditions, rather than that in which one factor dominates the situation all the time.

The process of evolutionary change is also affected by the average fitness of a population. A change in the environmental condition means a change in the topography of the field of gene combinations. Favourable gene combinations may become unfavourable, and vice versa. The effect of selection will therefore be to lead the selected population and its conucopia of new gene frequencies gradually toward a new equilibrium point determined by the new environment. Hence, the evolutionary advance is assured.

I have not just spent all these time talking about evolution merely for the sake of making an impressive pedagogic argument. The harvest is on how the newly evolving Nigeria- man is taking advantage of his new genetic equipage to reap the dividends of this evolutionary process. For example, what advantages has it given him in his efforts to subdue his environment, or to master and control the tapestry and vagaries of the livability issues that confront him? How have these helped him to understand and take advantage of such genetically influenced issues as sex determination, blood groupings, genetic resistance to diseases, human intelligence,
fecundity traits, and other anatomical traits? These will be the subject of the next discussion.

4.1 Sex determination

Because this is a mixed audience of varied academic and professional backgrounds, I will not bore some of us by discussing sex determination with the usual genetic jargons and cross-diagrams. Instead, I will present it in a way that it will be intelligible (and amusing) even to the least among us.

4.1.1 Let's Talk About Sex.

For the ladies among us, next time your husband bullies you for having only female babies; or harasses you if your son appears girly and your daughter appears macho; tell him to come and buy my mimeograph!!

There is this joke about enlightened 21" century parents. They don't want to raise their son to be macho or their daughter to be girly. So, they gave the boy dolls to play with while the girl is given toy guns. What happens? The girl wraps the gun in blankets and rocks it to sleep. The boy tears the doll apart and assembles its arms and legs into a weapon.

Not so long ago, this punch line would have been dismissed as sexual stereotyping. But today, it doesn't laugh off so easily. In the past decade, research has repeatedly shown that, aside from cultural effects, there are undeniable genetic influences on male and female behaviours. So the gun in the cradle raises some interesting questions. Is the joke about biology? Is it about culture? Nature or nurture? Or is it, as scientists increasingly suspect, about both?
4.1.2 X and Y chromosomes:

Put very simply, whether people are born male or female depends on the sex chromosomes: X and Y. Females get an X from each parent. Males get an X from one and a Y from the other. XX produces the basic female body design. XY orders up the standard male model. The Y-chromosome carries an influential gene called SRY (sex-determining region Y). The SRY gene codes for a protein which triggers male body building. Activation of the SRY gene about 6 weeks after conception triggers the formation of the testes. The testes then start manufacturing the hormone testosterone which floods through the body making it male. Without the SRY gene, the foetus will develop the default body plan which is female!! Now ladies, next time your husband harasses you for not giving him a male child, tell him that his Y-team was too weak to play!! Fig 3 shows the various stages in the development of the human body.

![Fig: 3 Development of the human body](image)
On a more serious note, testosterone has been strongly associated with aggression and competiveness. Testosterone appears fairly directly linked to the rough-and-tumble play in small boys.

Studies

with closely related primate species, such as rhesus monkeys, have found out that if testosterone is deliberately suppressed, the young male monkeys almost instantly quieten down. They stop wrestling and climbing. They begin to act much more like young females, grooming each other and playing gentler games.

If the routine processing of hormones is interrupted, similar behaviour changes are seen in humans. In girls, for instance, there is a well known genetic variation called Congenital Adrenal Hyperplasia (CAH). In CAH, excess testosterone flows into the bloodstream. As a result, CAH-girls tend to have the bulkier muscled, slim hipped, masculine body type. They often play more roughly than their peers do, and excel in the physically hard, rough and sweaty, male dominated professions.

It is worthy to note that in Nigeria, despite the fact that the selective pressure and cultural pressures favour the survival and preference for male babies, our census have continued to record that in our evolutionary growth, a one-on-one equilibrium exists between the number of males and those of females. However, the genetic effective population number is in favour of females.
4.2 Human ABO Blood Groups

The mechanism of the inheritance of ABO blood groups has been explained in almost all textbooks of genetics and need not be given here in detail. Briefly, and in the most rudimentary forms which can hold the interest of everybody here, the explanation is simple and is based on the existence of three alleles (genes) namely: A, a, and a*. The allele A produces antigen- A and is dominant over a which is not capable of producing any antigen. The other allele a* produces antigen B and is also dominant over a. However, A and a* are not dominant over each other (co-dominance) and produce their antigens independently, so that the heterozygote Aa* has both kinds of antigen. Consequently, there are four distinguishable phenotypes (blood groups) in human populations: AB, A, B, and O; corresponding to (Aa*); (AA, Aa); (a*a*, a*a); and aa. The illustration is given in Table 3 below.

<table>
<thead>
<tr>
<th>Genotypes</th>
<th>Blood Group</th>
<th>Antigens on Red Cells</th>
<th>Antibodies in Plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA, Aa</td>
<td>A</td>
<td>A</td>
<td>Anti-B</td>
</tr>
<tr>
<td>a<em>a</em>, a*a</td>
<td>B</td>
<td>B</td>
<td>Anti- A</td>
</tr>
<tr>
<td>Aa*</td>
<td>AB</td>
<td>A, B</td>
<td>None</td>
</tr>
<tr>
<td>aa</td>
<td>O</td>
<td>None</td>
<td>Anti- A, and Anti- B</td>
</tr>
</tbody>
</table>

Genetic consequences of this situation should now be clear to all. An individual of blood group O, for example, might come from a mating of two individuals of blood group A; or from two individuals of blood groups A and B respectively; or from other possible kinds of matings; but definitly
mother of his parents could ever be AB.

This kind of genetic analysis has very important implications in that it forms
the basis of the application of blood grouping to legal medicine; the solution
of cases of possible baby mix-ups and a source of evidence in cases of
disputed paternity. So, next time she accuses you that you are the father of
her child, you can prove that you aren’t, but of course you can’t prove that
you are, except by DNA-typing.

4.2.1 The Rh-System

The antigen-antibody interplay in human blood systems also has very
important applications in human medicine. Rh incompatibility has been the
major contributor to hemolytic diseases of the newborn. By incompatibility
is meant any maternal-fetal combination in which the fetus possesses an
antigen not possessed by the mother. In order to rid the population of such
hemolytic diseases of the newborn, it has become routine in many parts of
the world to legally require intending couples to undergo Rh tests prior to
any nuptials.

4.2.2 Histocompatibility

When tissue is grafted from one person to another, the recipient forms an
immune response against antigens present in the donor cells but not
present in the recipient. As a result, the graft is soon rejected. Subsequent
grafts from the same donor are rejected more rapidly because the recipient
is already sensitized to the specific antigens. If the donor and the recipient
are genetically identical, as in the case of monozygotic twins, the graft is
readily accepted. In humans, grafts between persons un-matched for ABO
type are rapidly rejected. Histocompatibility, or lack of it, is due especially
to an antigen complex, the HL-A region. Again, this kind of genetic analysis
has very important applications in tissue and organ transplants.

Undoubtedly, our present knowledge of, and our applications of the
genetics of human blood groupings, as we have demonstrated in such areas

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as in human medicine and human reproduction, have given the present Nigerian human population a genetic advantage which its predecessor did not possess. We now have the capability to influence and determine, to some extent, the genetic constitution of the next generation. We now have the capability of breaking new genetic frontiers into such areas as genetically modified foods, germ-cell research, and recombinant-DNA technology. Indeed, possessors of some detrimental genes can now be limited from contributing such genes to the gene pool of future generations. Thus, the population is assured that the present individuals are genetically harder and more adapted to combat or even harvest from the challenges of their present environment. A leap in the evolution process.

4.3 Genetic Resistance to Diseases

The production of antibodies provides vertebrates with a very powerful weapon against invasion by bacteria and other microorganisms. The acquisition of such an immune system increases the likelihood for individual survival, and permits evolution to favour those organisms with fewer offspring and longer periods of development, rather than those other organisms that must devote a major portion of their energy to producing vast numbers of their young in order to circumvent the high probability that the young may not survive to reproduce. The details of the origin, acquisition and transference of the present immune system is largely genetic. Functionally, antibodies are defined by their specific reactions with antigens. Chemically, they are classified as globulins. A wider term immunoglobulins has become more appropriate to accommodate all classes of globulins involved in our study of immunogenetics.

Antibodies are a prime example of adaptation in response to environmental stimuli. An organism not previously exposed to a foreign endangering substance (antigen) does not have detectable levels of antibody that react with that antigen. Following exposures, by infection, injection, and so on; there is a build up of antibodies that react with the
specific antigen, but not with other unrelated antigens. The level of antibody may taper off if exposure has not continued, but a second exposure (immunization) causes a prompt rise in antibody tier, usually to higher levels than previously. The immune system thus becomes primed by its first exposure, and the machinery of producing antibodies for body defence is ready to function.

There are about five different classes of immunoglobulin (I,) molecules now known in man, and they can each be distinguished by solubility differences and also on the basis of antigen differences. However, they all appear to have evolved from same primordial gene. There are several inherited defects in I, production in humans. Some of the defects have led to immunodeficiency disorders in which the immune response becomes defective, impaired or dysfunctional. These have often resulted to incidences of immune deficiency diseases.

**Immune Deficiency Diseases**

Some of these immune deficiency diseases are inherited (IIDD) while others may be acquired (AIDS).

Several inherited immune deficiency conditions are known in human. They result in generalized inability to respond to antigen stimulus. They are grouped under the terms dysgammaglobulinemia or agammaglobulinemia. Although their pathogenic mechanisms are however varied; they occur as a result of the presence of an inherited sex-linked recessive gene. The symptoms usually appear between four to twelve months of age when the immunoglobulins passively transferred from the mother to the child during fetal period are exhausted. This leaves the affected child with little or no defence against bacterial or viral infections. Repeated infections usually lead to death before two years of age.

A high percentage of infant mortality in our population is probably caused by the above situation. We probably attributed it to Ogbanje, Juju, or other occultic sources. Happily, this genetic defect, which had hitherto
dessimated our child-population, has now been hemmed into control by recent advances in immunization. Hence, the child is now better equipped to survive, evolve, and contribute its genes to the next generation.

Recently, human population has become vagarized by the incidence of acquired immunize deficiency disorders; now more generally referred to as AIDS. The only difference is that in this case, the immune deficiency is acquired. Recent advances in human medicine trace this condition to the presence of a mutant HIV (+ve) strain in the affected individuals. It is not our purpose here to go into the genetico-histopathology and treatment of AIDS. The most important thing is to underscore that AIDS is acquired not inherited; therefore as the soothsayer would say "it is in your blood, and not in your genes".

In charting out the course of the evolutionary vista of our population, I invite you to pause and wonder the effects which AIDS, in the long term, will have on the effective population number of individuals who survive to contribute their genes to the next generation. Or is it as some fatalists portend, just one more way in which nature tricks and inflicts its vagaries on human populations so as to ensure that only those with the selective advantage survive to contribute their genes to succeeding generations. The viewpoint is yours.

4.4 Genetic Abnormalities

During embryogenesis, many genes become active (or inactive) at regular stages in development. What, you may ask, are the mechanisms that trigger or control the activities of these genes during embryogenesis and fetal development? To what extent are the various stages in this
development programmed by genes, and to what extent are variations in embryonic development due to genetic variation? At a practical level, to what extent are congenital malformations attributed to heredity? Put more mildly, what abnormalities in humans can we say are attributed to genetic defects, and what are their consequences in the evolutionary process of our species?

Congenital malformations constitute a major problem in human health. In a detailed study carried out by the World Health Organization in 1000 centres in 50 countries, a frequency of 12.7 major malformations per 1000 single births was observed (Stevenson et al. 1966). A number of other studies such as Russel (2003) and German (1992) have suggested frequencies in the order of 10 children with malformations per 1000 births. Thus some 1 percent of newborn babies have a readily recognizable congenital defect. These malformations are caused by errors in the genetic apparatus. They may cluster in families and in populations in a manner consistent with genetic origin.

For lack of space and time, some of the abnormalities we will consider here are those that are involved in achondroplasia (dwarfism), albinism, sickle cell anemia, and cancer.

4.4.1 Achondroplasia

Achondroplasia (or dwarfism) is a congenital malformation due to single genes. This disorder has been recognized as genetic for over a century. It is a simple dominant trait occurring with a frequency of approximately 20 per million births. Affected persons (see fig. 4) are characterized by very short long bones, an enlarged head and depressed nasal bridge. The trunk is normal in size. Adults average about 4 feet in height. Affected persons are fertile but reproduction is low. 80% of the cases result in new mutation. Most dominant conditions occur in the heterozygous combination, ie two
normal parents can mate to produce a dwarf. Hall and coworkers (1969) however reported some cases where matings between two achondroplastics gave rise to severely affected children thought to be homozygous.

Fig: 4. Photograph showing typical features of a Achondroplasia
4.4.2 Albinism

A vast number of genes (and enzymes) have as their function the synthesis of body constituents. A block in essential biosynthetic pathway is therefore likely to have profound effects on development. Some such blocks may be embryonic lethal. Some are not, and hence become available for us to study. An example of this is albinism.

Albinism is as a result of such a blockage. Persons with albinism are easily identified, as the familial nature is often obvious. It is due to an inherited enzyme defect which is characterized by a genetically determined lack of melanin, the substance that codes for epidermal pigment. Three types of albinism are known (i) tryosinase-positive oculo-cutaneous albinism, (ii) tryosinase-negative oculo-cutaneous albinism and (iii) ocular albinism. In the first two, pigment is absent both in the eyes and in the skin; whereas in ocular albinism only the eye pigment is missing.

The genetic heterozygosity of oculo-cutaneous albinism was first clearly demonstrated by matings between albinos. For matings between persons homozygous at one locus, all the children should be affected. However, some cases have been observed where two albino parents have produced normal children. In such a case, one parent was tryosinase negative and the other tryosinase positive. Although albinism is not lethal, and pigment is clearly not necessary for normal growth; however, it is a detrimental trait which increases the victim's predisposition to some diseases, e.g. skin cancer.
4.4.3 Sickle Cell Anemia

The disease sickle cell anemia is an inherited condition in which affected persons suffer from anemia resulting from excessive destruction of red cells in the circulatory system. In the presence of copious amounts of oxygen, the red cells essentially operate normally. But if oxygen is low, as may happen in many tissues, the red cells become distorted, in some instances becoming sickle in shape. Formation of sickle-shaped cells grossly impedes circulation, leading to greater oxygen deficit and hence more and more sickling. Interference with circulation leads to a variety of pathological consequences, depending on the tissues involved. Persons with this disease, especially in its acute form, may not survive to adulthood. Sickle cell anemia involves the substitution of valine for glutamic acid at a specific locus in the adult hemoglobin molecule.

In many populations, there is a moderately high frequency of the gene for abnormal hemoglobin, which in the homozygous state causes sickle cell anemia. Persons who are homozygous rarely survive to adulthood; but in some parts of the world heterozygotes seem to have a slight advantage over persons homozygous for the normal allele, because of their increased resistance to malaria.

4.4.4 Abnormal Chromosome and Cancer

Following the advent of techniques for successful chromosomal analysis and recent advances in chromosomal mapping, it became clear that abnormal chromosome-complements were characteristic of malignant tissues. On the one hand, this relates abnormal chromosomes to the origin of cancer. On the other hand, it also implicates the possibility that cancerous cells might be more prone to develop abnormal chromosomes.
In culture media, abnormal chromosomes have indeed shown a tendency to undergo re-arrangements, in contrast to the normal chromosomes which remained quite stable. Relevant to this issue are some inheritable diseases which lead to, or are a result of, chromosome instability, and subsequently to cancer. Some of such diseases include chronic myelocytic leukemia, Bloom's syndrome, Fanconi's anemia etc. In all these cases, there is consistent association between a malignancy and aberrant chromosomes. For example, studies reported in NATURE by Rowley (1973) suggested that the translocation of the missing portion of the long arm of chromosome-22 to one of the larger chromosomes, most often to the long arm of chromosome number-9, is implicated in chronic myelocytic leukemia.

Fig 5 shows the chromosomal map of a human male. Incidences of aberrations due to translocation, deletion or trisomy lead to several kinds of genetic disorders.

Examples:

(a) Deletion of a major portion of the short arm of Chr 5 may result in some type of severe mental retardation and deficiency, a moon-faced appearance, congenital malformations: low set malformed ears, and brain dysfunction. An important distinctive feature of this condition is the plaintive cry in infancy described as similar to a cat's cry. It is more frequent in females than in males, perhaps, reflecting increased fetal mortality in males.

(b) Deletion of a large portion of the short arm of Chr 18 may result in mental retardation, short stature with retarded development, ptosis and severe webbing of the neck. Deletion of a large portion of the long arm of Chr 18 leads to mental retardation, microcephaly, diminitive stature

(c) A chromosomal aberration in which one of the chromosomes of the genome is present in triplicate
Fig: 5 Chromosomal map of the human male
rather than in duplicate (ie trisomic) gives rise to a genetic disorder known as Down's syndrome, or Mongolian idiot. Affected individuals are mentally deficient, dysfunctional, physically retarded, and have such other abnormalities as stubby flat face, slanted eyes, with facial features that are out of alignment. Mongolism is about 100 times more frequent in children of mothers over 45 years of age than in children from mothers 16-28 years of age. It increases with increasing age of the mother.

4.5 Implication of Genes on Some Fecundity and Anatomical Traits

4.5.1 Fecundity Traits

Fecundity traits refer to those traits which have to do with fertility, fruitfulness, prolificity, and ability to reproduce. They include such traits like ovulation rate in females, conception rate; fertilization capability of sperm cells, their morphological intactness or otherwise; embryo survival rates, etc. Undoubtedly, environment plays a large role in the manifestation of these traits. However, there is undeniable important and significant genetic input and predisposition to them.

Using experiments on domestic animals, several reports by Iloeje and co-workers have confirmed various degrees of genetic, environmental and genetic x environmental variations in these traits. For details, see Iloeje and Van Vleck (1978 and 1979) Iloeje et al (1981); Iloeje (1985, 1987 and 1988); Ogundu Iloeje and Herbert (2002).

Several experiments have also been carried out in an attempt to elucidate and quantify the influences of heredity and other manipulative techniques on the sexual repertoire and reproductive efficiency of domestic animals.
A major part of my research work in the past ten years or so have focused on this theme. For some of our results, see: Umesiobi, Okani and Iloeje (1999); Umesiobi, Omalaka and Iloeje (1997); Iloeje (1998); Umesiobi, Iloeje et al (2000); Iloeje (2000); Umesiobi, Iloeje and Berepubo (2002); Umesiobi; Iloeje et al (2002); Herebert, Ezeobi and Iloeje (2002); Umesiobi, Elekwa, Herbert, Iloeje and Ngongoni (2002); Umesiobi and Iloeje (1999), to mention but a few.

4.5.2. Anatomical Traits

In biological evolution, some of the anatomical, and other traits we will consider here have to do with the following:

**Body Shape:**

One of the most striking and "magical" features of identical twins is their appearance. Even close family members often find it difficult to tell them apart. Doesn't this suggest to you that the shape of our bodies is dictated by our genes?

**A Head Start:**

Your height as an adult is determined to some extent by your genes. But of course, your diet and the environment in which you grow will determine exactly where on the tape measure you finish up. The average height of Nigerians has increased dramatically over the last fifty years. This is thought to the partly due to improvements in our diets and in our living environment. The first advertisement for recruitment of Nigerians into the Police and into the former West African Frontier Force demanded for the tall Nigerian male of height 5ft 4 inches. Today, he will be considered too short.

**How Tall Might Your Child Be?**

Your genes have a strong enough effect on your height for you to be able
A major part of my research work in the past ten years or so have focused on this theme. For some of our results, see: Umesiobi, Okani and Iloeje (1999); Umesiobi, Omalaka and Iloeje (1997); Iloeje (1998); Umesiobi, Iloeje et al (2000); Iloeje (2000); Umesiobi, Iloeje and Berepubo (2002); Umesiobi; Iloeje et al (2002); Herebert, Ezeobi and Iloeje (2002); Umesiobi, Elekwa, Herbert, Iloeje and Ngongoni (2002); Umesiobi and Iloeje (1999), to mention but a few.

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One of the most striking and "magical" features of identical twins is their appearance. Even close family members often find it difficult to tell them apart. Doesn't this suggest to you that the shape of our bodies is dictated by our genes?

**A Head Start:**

Your height as an adult is determined to some extent by your genes. But of course, your diet and the environment in which you grow will determine exactly where on the tape measure you finish up. The average height of Nigerians has increased dramatically over the last fifty years. This is thought to the partly due to improvements in our diets and in our living environment. The first advertisement for recruitment of Nigerians into the Police and into the former West African Frontier Force demanded for the tall Nigerian male of height 5ft 4 inches. Today, he will be considered too short.

**How Tall Might Your Child Be?**

Your genes have a strong enough effect on your height for you to be able
to predict what the adult height of your children should be if they are given a good diet and raised in a good environment. Based on figures published by the Child and Family Nursing Services, USA; if you take the father's height (cm), add the mother's height (cm), divide this by two; add 7 cm for a boy or subtract 7 cm for a girl; what you will get will be the expected average height of the child.

Are you Carrying A Heavy Burden?
Come to me all yee that are heavy laden. This is my polite way of saying, "are you fat." Some people stay slim no matter how much they eat. For others, battling the bulge has become their perennial problem, no matter how little they eat. This is because some people are genetically equipped to catabolize excess energy more efficiently, while some others are better equipped to store it as fat. Body weight is therefore one of these traits in humans which is influenced by many different genes and all manner of external influences. Diet and exercise may shed the pounds if you can stick at it. Genetics won't provide you with an excuse to stuff yourself every day with mounds of pounded yam, isi ewu, ice cream, and crates of beer!!

Mirrors Don't Lie:
Before you look at your mirror next time, please look at the picture of an ape. They are our distant relatives you know! The extent to which our facial features have evolved from the apes will be shown by such "beauty clips" as how flattened your nasal bridge is, how flared your nostrils are, how thick and wide your lips are; the epical length of the distance of your forehead from your hair line to your eye-brow line. Beauty they say is in the eye of the beholder; but check out the beauty queens in today's contemporary magazines and you can see the direction of the human
ascent on the evolutionary ladder.

4.6 Genetics and Human Intelligence

Much concern has been expressed over the decades on the roles which genetic equipage plays on human intelligence. Does your intelligence quotient (IQ) depend on your gene, or on your environmental conditions? Is it determined by your nature or by your nurture? Several studies showed that parents with higher IQs also tended to produce children with high IQs. But is this conclusive? Isn't it true for example that high IQ individuals often mate and marry individuals with relatively high IQs also. Similarly, parents with high IQs tend to have smaller family sizes than parents with low IQs. How many children did Einstein have? or Beethoven or Soyinka?

If this follows, therefore high IQ persons would contribute relatively less to the gene pool of the succeeding generation. But is this true? Is it not also true, for example, that individuals with abysmally low IQs may never get a chance to marry or mate, and therefore may leave no offsprings at all. From these axioms, its seems perfectly plausible that higher intelligence is indeed associated with greater reproductive fitness; although this need not be true under all circumstances, since higher IQ persons may be more likely to decrease their family size in response to the economics of the burgeoning population.

Can You Make A Genius?

How do you fancy a nobel prize-winning scientist as the father of your children? Would they inherit his genes and turn into geniuses too?
Research into the inheritance of IQ has been one of the most controversial areas of genetics. Many scientists do believe that a great deal of our intelligence is inherited. Others contend that some people are “born stupid” and that there is no point in educating them. These points of view however are not conclusive and still remain controversial.

There is also great controversy on the lack of objectivity in the way intelligence is measured, using the IQ test. The current measures have been accused of having some cultural biases in them, and may also be guilty of measuring only a small part of a person’s overall ability. More radically, the evidence for variations in intelligence levels may be questioned by challenging the objectivity of the standards relative to which these levels are assessed. It may be argued that conceptions of what constitutes a rational or intelligent response to a situation or to a problem are themselves culturally conditioned, i.e. a product of the way in which the member of a group devising the tests and making the judgments have been taught to think. Such an argument certainly undermines the claims by any one human group to intellectual superiority over others, whether these others are their contemporaries or their own forebears.

However, no matter how well and how transparently objective any new measures of IQ may be, the question that still remains unresolved is whether our intelligence is determined by the genes we carry, or by the environment we grow up in. Is it in our nature or in our nurture. The debate continues. ALUTA CONTINUA.
5.0. NATURE AND NURTURE: THE CHICKEN OR THE EGG?

Some time ago, in the not too distant past, one of my very good friends, Dr. Mrs. Sophie Ogwude, a distinguished Associate Professor of English knocked me out with her dissertation on the WOMAN QUESTION. Today, I have decided to bounce back with the Darwinian dialectic on yet another question: the HUMAN QUESTION nature or nurture; the chicken or the egg?

5.1. Nature or Nurture.

If human nature is understood simply as man’s special form of that which is biologically inherited in all species, there remains the delicate problem of discovering in any given case, exactly what role environment plays in determining the actual characteristics of mature members of the species. As in the above case of IQs, and even in the case of some purely physiological characteristics, this may be far from being straightforward.

For example, the extent to which diet, exercise, and conditions of work determine such things as susceptibility to heart disease and cancer remains very hotly debated, unresolved, and a subject of intensive scientific investigation. In the case of human intelligence quotient IQ, as shown above, the problems have exacerbated so much that they can no longer be answered by purely empirical investigation. There is room for much conceptual debate about what is meant by intelligence, and over what test, if any, can be supposed to yield a direct measure of this capacity, and thus provide evidence that an individual’s level of intelligence is determined at birth (by nature) rather than by subsequent exposure to the environment (nurture) that conditions the development of his capacities.
Man is traditionally thought to be distinguished from and privileged above other animals by virtue of his possession of superior reason, or intellect. When the intellect is valued as purely genetic in origin, and sorely endowed on us by our nature, this confers a concept of genetic stereotyping of man, and the thought that different races of people differ in nature in their intellectual capacities. This has been used as a justification of a variety of racist attitudes and policies, and the fallacy of a superior race.

Similarly, this fallacy of a hierarchy in naturally endowed intelligence quotients has been used to support the jaundiced argument that women are by nature intellectually inferior to men, thus justifying their domination by men.

However, on the other hand, the argument that our intelligence is a product of our nurture, underscores the concept that differences in adult intellectual capacity are as a product of the circumstance in which potentially similar people are brought up; thus all of us are equally human but some as having been more privileged when growing up, than others.

These debates about intelligence provide an example of the complexity of the impact of evolutionary biology on conceptions of human nature and nurture. We all live in different worlds. Nobody can claim that their life has been exactly the same as yours. You might be wealthy or poor, a megastar, or an anonymous face in a city of millions. You may have an older brother, be an only child, have mixed parents, live on the sunny side of the street or in a low down ramshackle dungeon. Your genes make you unique, but so does your life, and it is different from every other human life on earth.
From the moment you were conceived, you have been dealing with unique surroundings. You may have had quite a different nine-months in the womb compared to that of your brother or sister. Do stressed out mothers-to-be produce agitated babies? Scientific studies suggest that there is a link; so this is just one sign that our different experiences, even from the womb, count from very early on. And once you are born, then just about anything can happen to you.

Your genes, in combination with early experiences of life, have an enormous impact on you. Your inherited genetic disposition acting in concert with your environment determine at what levels in the league you may play. Human character, as well as human intelligence, is indefinitely plastic; each individual is given a determinate form by the nature of the genetic endowment he has, by the environment in which he is born, brought up, and lives. Unfortunately, the exact extent of the contribution of each of these components to the totality that is you, is a measure that has continued to elude us. Attempts to study the behaviour of identical monozygotic twins, raised in the same environment versus those raised in different environments have shed some little light on this dilemma.

So, are you a thief because you were born with the genes that irrevocably condemned you to be a thief; or are you a thief because you allowed yourself into the environment of greed that conditioned you as a thief? Which takes the lead, nature or nurture? Some would say, both come first; but in my mind, the real answer should be nature and nurture.

5.2. **The Chicken or the Egg**

Ineluctably linked with the evolutionary question of nature and nurture is the Darwinian dialectic and deadlock dilemma of the chicken or the egg: which came first?
As with most problems, it is important to try to understand the question, and a good place to start is by asking what is meant by "the egg" and what is meant by "the chicken". In common viewpoint, the egg is thought to be a chicken egg. This is a very obvious assumption, given that the question implies that there is a link between the two. If we assume that the egg is a chicken-egg then we have to define what is meant by a chicken-egg. "A chicken will hatch from a chicken egg." True? Yes!! This ofcourse means that an animal that was not a chicken laid the chicken egg which contained the first ever chicken. In this case the egg came first. But if "a chicken's egg is the egg that a chicken lays" this ofcourse implies that a chicken (that already exists) laid an egg (a chicken-egg). In this case the chicken came first.

A less common viewpoint is ofcourse one in which the egg is not thought to be a chicken egg. This in effect changes the question to "What came first a chicken or an egg". From a purely scientific point of view this question can be answered quite easily. It is obvious that there were eggs before there were chickens; since some of the first animals to have ever existed laid eggs. From an evolutionist view, as well, chicken eggs preceded chicken in so far as the first chicken must have been the mutant offspring of a proto-chicken that laid the first chicken's surrounding egg:

There is yet a third view point. Evolutionary biologists believe that in fact chickens, or eggs, for that matter, were not created by God; rather they are the product of a long evolution. We have to specify that we are speaking about chickens and chicken's eggs, not about chickens and any kind of eggs.

Let us call the animal, which preceded the chicken, in the evolutionary ladder, as the primeval chicken, or P-chicken for short. The difference
between this P-chicken and our today's chicken is arbitrary, and so not very critical to this argument.

One day a P-chicken must have laid an egg, out of which a modern chicken emerged. The arrival of this strange chicken has maybe happened on several occasions independently, and the first modern chicken might not be the ancestor of all our chickens, or it might even have died childless; but this is of no importance to our discussion. The only important factor is, whether the egg out of which the first modern chicken was born, was a real chicken's egg. If this egg actually was a chicken's egg, then the EGG existed before the CHICKEN; if not, it can only have originated later. QED Now, we still have to define a chicken's egg. At first sight, there seem to be three possible definitions: (1). A chicken's egg is an egg laid by a chicken. (2). A chicken's egg is an egg out of which a chicken is born. (3). A chicken's egg is an egg laid by a chicken, out of which a chicken is born. Infact, only the first definition can be right, because also the eggs we eat, which most certainly do not contain chickens, are called chicken's eggs. And on top of that, if eggs were named after what they contained, there would also have to exist cock's eggs. No, the definitive and genitive form can only refer to the maker not to the inhabitant of the egg.

So, as the egg, out of which the first chicken emerged, cannot have been a chicken's egg, because it was laid by a P-chicken; and as we have clearly stated that the EGG in the dilemma of the chicken and the egg can only have been meant as a chicken's egg: I beg to conclude that the EGG came into existence after the CHICKEN. True? False!!

Did an egg hatch a chicken before a chicken laid an egg? OR are we all
agreed that the first chicken was hatched from an egg laid by a P-chicken? Then where did this P-chicken hatch from?

The heap paradox in this logic is that it brings us round and round in the evolutionary cycle; not just of chickens or eggs; nor of the dilemma of nature or nature; BUT essentially to the realization that our stance on the evolutionary ladder can best be quantified in finite definitive quanta of time. Therefore, I take the stance to repeat, like I said at the beginning, that as of TODAY, there is an observable new genetic trend and a shift in our gene pool and in our breeding patterns, which almost imperceptibly, are slowly but surely leading to a gradual evolution of a new Nigerian man, genetically natured and environmentally nurtured to combat and harvest the challenges of today's Nigeria.
6.0 CONCLUSIONS AND RECOMMENDATION

6.1 Conclusion

My conclusion will try to encapsulate in SUMMARY all I have toiled to say in the course of this lecture.

I have used the platform of this Inaugural to chart a new vista for our general genetic education and increase our awareness that a new Nigeria-man is indeed evolving; with a new genetic hardiness, adapted and may be tailored to cope, survive, harness and adapt to the demands and consequences of our environment.

I have used explanations of such genetic phenomena of gene frequencies, panmixia, random mating, genetic equilibria, gene pools, breeding patterns and genetic spreadsheets to examine and authenticate the consequences which the emerging new genetic tapestry plays in our continued struggle to survive and conquer the challenges of our environment, in such areas as disease resistance, human intelligence, genetic disorders, fecundity, sex determination, blood groups, anatomical traits and other livability traits. I have utilized the paradox of the Darwinian dialectics as to the question of “nature or nurture: the chicken or the egg”. To domicile our stance on the evolutionary ladder, and qualify in finite, definitive quanta of time the arrival of the new Nigeria-man.

6.2 Recommendation

It is usual to itemize a list of recommendations from such a lecture. The temptation is compelling. However, in order to make the desired impact, and give all of us one single important item which we cannot forget, I have
decided to sum all the possible recommendations into one single important thrust called genetic counseling.

This lecture therefore recommends a clarion call for a public awareness, and our dire needs for appropriate genetic counseling in our process of mating and the pro-creation of the next generation of Nigerians. Every Nigerian, private or public; rich or poor; urban or rural, male or female; should be availed of the opportunity for genetic counseling. What you may ask is genetic counseling.

6.2.1 Genetic Counseling

Human genetics has become a major issue in man's concern for his future. This has developed, largely, as a consequence of man's ability to manipulate the hereditary process, and in part control or influence the genetic constitution of the population. The fact that many heritable genetic disorders can now be managed has been a factor. Man's great success in the conquest of infections, nutritional and heritable disease have increased the contributions of both our nature and our nurture to longevity. In order for each of us to harness and harvest the dividends of this new genetic capability, we must be properly counseled.

Persons desiring genetic counseling do so for a variety of reasons. The obvious one may be to obtain information on the likelihood that a genetic disorder present either in a previous child of the marriage, or in some relatives, or in some intended spouse, will also be present in children not yet born. This information can now be provided by genetic counseling and with increased accuracy, depending on the disorder and the information available on the pedigree.
Genetic counseling has also increasingly become important in the diagnosis of several hereditary disorders, especially where the pedigree analysis may help differentiate among possible diseases.

Certain genetic disorders are masked in the heterozygotes of carrier-individuals. Genetic counseling can now give you accurate probabilities that a relative of an affected person is heterozygous for the genetic disorder.

Occasionally, prospective marriage partners, each with an affected relative, may wish to know the likelihood of their having affected children. Genetic counseling un-masks this wonder; and replaces the agonies of fear, with the calmness and realities of certainty.

Not every genetic disease can be diagnosed at birth. For diseases of late onset, (e.g. Huntington's disease), genetic counseling can reveal the risks of the transmittability of the abnormal allele, even before the potential carrier parent has yet had the opportunity to express the trait.

Sometimes, because of reduced penetrance, a genotype is not expressed, and a particular difficult problem occurs in assessing the status of a person at risk for an inherited disease. Many tumors and malignancy predispositions present such a problem. An a-priori genetic counseling provides an early opportunity to handle such a problem.

A family's response to the threat of inherited disease depends on their perception of, and early warning of, the burdens imposed by the disease.
Such early warning can be provided by genetic counseling. This situation is also applicable for pre-natal diagnosis.

Genetic counseling is indeed not new, even in our traditional village setting. Probably without knowing it, one of the earliest forms of genetic counseling implicit in our traditional custom come in the form of the traditional enquiries which our parents make about the ancestry, family background, and genealogy of your intended spouse. The objective of such pre-nuptial enquiries are usually to find out if an intended spouse comes from a family where there have been incidences of some heritable genetic disorders such as madness, albinism, chronic bareness, criminality and other deviant behaviour. The question they seek to answer is: "Is that trait in their nature, or is it in their nurture?"

When we mate in response to the need to increase and multiply, we are in effect contributing our genes to the next generation of the human population. It is a sacred duty to ensure that such matings produce and perpetuate genes desirable for the effective perpetuation of our species.

Mr. Vice Chancellor Sir, distinguished ladies and gentlemen, there is an old Igbo adage that says, "If you want to see what your new young bride will look like in days after tomorrow, please look at your mother-in-law". For it is in her genes, it is in her nature, and it is in her nurture!!

I rest my case.
7.0 ACKNOWLEDGEMENTS

I started this Inaugural with a preamble on the dream that propelled me into becoming a Professor and a University Orator. The fact is that I really love being a professor and an orator. There is something that comes alive in me in professing a subject matter in which I am well schooled. And the university above everything else, provides me such a setting.

Let me therefore share with you, the words of John Masefield, a British poet and the Orator of the University of Sheffield, who on 24 June, 1946 at the installation of the Chancellor of that University said:

“There are few earthly things more beautiful than a university:
In these days of broken frontiers and collapsing values:
When the dams are down and the floods are making misery:
When every future looks grim and every ancient foothold has become a quagmire:
Wherever a university stands, it stands and shines:
Wherever it exists, the free minds of men, urged on to full and fair inquiry, may yet bring wisdom into human affairs.

There are few earthly things more splendid than a university:
It is a place where those who hate ignorance may strive to know:
Where those who perceive truth may strive to make others see:
Where seekers and learners alike, banded together in the search for knowledge, will honour thought in all its finer ways:
Will welcome thinkers in distress or in exile:
Will uphold ever, the dignity of thought and learning:
And will exact standards in these things.  

There are a few earthly things more enduring than a university;  
Religions may split into sects or heresy;  
Dynasties may fall or be supplanted;  
But for century after century, the university will continue;  
The stream of life will pass through it;  
And the seekers and the learners will be bound together in the undying cause of bringing thought into the world"
who is now residing in Connecticut, I say thank you. I also wish to thank my third brother, Prof. S.O. Iloeje, a Professor of Pediatrics and Chief Consultant Pediatric Neurologist at the University of Nigeria Teaching Hospital. I owe tremendous gratefulness to my fourth brother, Professor O.C. Iloeje, a Professor of Mechanical Engineering, University of Nigeria and Director, Energy Commission of Nigeria Abuja. I say a big thank you to my only sister, the Hon. Chief Mrs. Anayo Justina Offiah, Senior Advocate of Nigeria, a former Attorney General and Commissioner for Justice, Enugu State. My big thanks also go to my youngest brother Arc Chief A.F. Iloeje, a Senior Lecturer in Architecture ESUT, and an Environmental Management Consultant.

My biggest gratefulness go to my four lovely children: Emeka (Electronic & Computer Engineer); Nnamdi (Medical student Univ. of Port-Harcourt); Chuka (final year law student, ESUT) and Ikenna (Engineering student, FUTO). They are undoubtedly the 4-pointed star in my life. In a most special way, I want to remember and shower my eternal gratitude's to my Late wife Dr. Mrs. Eme Iloeje who stood by me, showered me with love, helped me, and saw me through the most difficult periods of my education, from my B. Sc, M.Sc., Ph.D, to my burgeoning years as a budding academic. Thank you for believing in me; and may your gentle soul through the mercy of the Lord rest in perfect peace. Amen. For those who have ever suffered such a loss, it is undeniable that the passing-away of a beloved spouse is in fact the worst kind of loss and tragedy that can ever befall anybody. In this regard, I must express my fathomless gratefulness and eternal love to Chinatu Nwoko for helping me walk again, and for lifting me back into the promise that there will again be a blissful union together with me again. I will like to conclude this part by thanking all my sisters-in-law, my only brother-in-law Dr. E.C. Offiah, my
numerous nephews, nieces, and several other members of the Greater-Iloje family.

If there is a re-incarnation, may God Almighty please bring me back into this family again.

Next, I have to remember my teachers and thank them for their sweat and for the successful "miracle" they performed in pummeling some education into somebody like me. I remember very specially Dr. G.C. Ukaga my English Teacher at Government College Umuahia; Professors Unamba-Opara, C.O. Okafor, Anya Oko-Anyia, Gaius Igboeli, C.C. Nwosu and many others who taught me at the University of Nigeria; Professors L.D. Van Vleck, C.R. Henderson, R.L. Quaas, Bob McDowell, R. H. Foote, Bob Everett and several others who moulded me at Cornell University, Ithaca New York; and of course to Prof. George Wilcox and Dr. Tanya Smith whom I was very privileged to work with during my post-doctoral studies at Oxford; and to my several classmates at the University of Nigeria, Cornell University, Oxford University, and of course my indomitable classmates of the famous class of 1962 of Government College Umuahia. I take this opportunity to remember again my classmate, friend and colleague Professor O.P. Nwamnuo who passed away in May 2004. May his soul rest in the Lord. Amen.

The destination I have reached today would not have been possible without the assistance of many of my colleagues (senior and junior) and the countless friends who put very many extra miles on my behalf. I especially thank the various Vice Chancellors of this University Professor Umaru D. Gomwalk who first employed me in 1981, appointed me the Co-ordinator
and Head of the Animal Production Department in 1983, and gave me my first baptism of opportunity at university administration and management; to Professor Amagh Nduka under whom I served as Dean of the School of Agriculture & Agric. Technology, and whom as history will surely vindicate ran one of the most transparent, accountable, and disciplined administration FUTO has ever had; to Engr. Prof Chuka Obah under whom I served first as Director of Academic Planning and later as Deputy Vice Chancellor (Academic) "if history will liken Gomwalk as our Moses, then Obah was the Joshua who led FUTO into the promised land"; and now to our present Vice Chancellor, Professor Jude Njoku - a dedicated, quiet and peace loving man. I thank them all.

My gratitudes also go to my other mentors, friends and colleagues such as Mr. Joe Anafulu (Uncle Joe), Professors Ed Banigo, I. C. Onwueme, Linus Ogbuji, Mike Oti, Okoro Ogbobe, Chibuzo Ogbuagu, Chuks Ugbolue, S. K. Nnana, E. B. Sonaiya, Felix Madubike, O. C. Onwudike, Placcid Njoku, Eka Briade, Fidelis Ogah, Tom Famula, Louis Iniquez, Martin Nwufo, Rev. Fr. Prof Louis Asiegwu, Dr. U. Herbert, Arc & Dr. (Mrs.) Oti, Dr. Marshall Odii, Dr. Jerry Obi-Okogbue, Dr. Uche & Mrs. Tina Ezem, Mrs. N. Onwudike, Rufus & Cecelia Ugbolue Dr. Kitt Emetarom, Prof. Thelma Obah, Mrs. C. C. Ahiarakwe, Chief and Mrs. Dave Amonu, Engr and Mrs. Evans Uhegbo of OBEV Systems; and very many others too numerous to mention. To Dr. Tom Scott, Dr. Jim Donaldson Dr. Ana Fields, Dr. Dennis Umesiobi, Dr. (Mrs.) Uduak Ogundu and Dr. Friday Iheukwumere you will continue to represent some of the best doctoral students I have ever had. Sometimes we must agree that excellent students contribute to bringing out the best out of good instructors.
There are many other very deserving people I must have missed in this listing. May my apologies never undermine the depths of my gratitudes; and in the words of Lucius Scipio Asiaticus, the great Roman editor may I say to all of you "Gratias eis maximas agit pessimus omnium scriba".

Finally, to God be the glory, honour, majesty, dominion, and authority; before all time, and now ever more. AMEN.
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