

## **Adult Disease Induction and Life Course Events: Some Perspectives from a Resource-compromised Economy**

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### **Abstract**

#### **Background**

Nigerian newborns face difficult childhood and adulthood with prevalent morbidities and resultant reduced life-expectancy tasking our practice and policy. We need clearer appreciation of disease induction from foetal origins with a 'Critical Window' in the life course for possible interventions.

#### **Disease Induction And Life Course Events**

Life course and events, from conception to death ('womb to tomb'), are highlighted re: roles in disease induction. Foetal growth, early child nutrition/ diet and early postnatal growth represent a 'Critical Window' for modulation re: inferred 'Body Programming' effect.

#### **Specific Life Course Events And Some Nigerian Perspectives**

Birth and its attributes are reviewed. Birthweight is discussed re: determinants, meaning and usefulness in disease induction. We have simple clinical tools for assessing gestational age and body proportion/ composition. We highlight our simplified methods of maturity determination as preterm infants have peculiar postnatal adiposity. Postnatal growth correlates with later disease and exclusively breastfed infants, with observed accelerated growth, are assessed using our new growth standards.

Child diet contributes to adult disease. We highlight observations on exclusive breastfeeding for policy and practice. With BFHI and the 'INAGOSICI Phenomenon', breastfeeding is improving. Two 'growth spurts' are reviewed for child feeding policy and the 'Transgenerational Model'.

#### **Conclusion**

Nigerian newborns face quadruple-barrel survival tragedy. Our perspectives on life course events are highlighted for policy and practice. Institute interventions are highlighted for community impact.

### **Introduction**

The Nigerian newborn infant is faced with 'Quadruple-barrel Survival Tragedy'. Born in a country with a low-birthweight rate of 16% (UNICEF 2005), Neonatal Mortality Rate (NMR) averaging 50/ 1000 live-births and Maternal Mortality Ratio (MMR) over 1000/ 100,000 births (UNICEF 2005), the newborn is off to a bad head start coupled with low exclusive breastfeeding rate (Eregie 1997a). Emerging from this, the newborn advances into the survival tragedy of early childhood laden with a 'morbidity-trap' of prevalent diseases including: diarrhoeal disorders, acute respiratory infections, malaria, measles and malnutrition among others with resultant gory childhood mortality statistics: Infant Mortality Rate (IMR) of 100/ 1000 live-births and Under-Fives Mortality Rate (U5MR) of over 200/ 1000 live-births (UNICEF 2006). Surviving early childhood, the next tragedy is a challenging adolescence and adulthood with emerging prevalent non-communicable morbidities: Obesity 12%, Hypertension 10%, Diabetes Mellitus 8% (with 50% complicated by the Metabolic Syndrome) and Coronary Heart Disease



among others (Akinkugbe and Akinyanju 1997). The final tragedy, a consequence of the preceding ones, is a compromised Quality of Life (QOL) with the resultant unacceptable Life Expectancy estimated at 52 years (UNICEF 2006).

This gory picture of the postnatal difficulties of the Nigerian challenges our clinical practice, public health policy and preparedness/ capacity to make a difference. A proper understanding of the foetal origins of adult disease induction and the nexus with life course events is a sine qua non for the formulation of interventions that will reflect desirable shifts in policy and practice.

### **Life Course/ Life Course Events**

‘Life Course’ defines the span from conception to death (‘Womb to Tomb’). Growth, prenatal to postnatal trajectory, is approximately a continuous process and ‘Birth’ is only an event within the ‘continuum’. The first event in the life course is conception though pre-conceptual factors may contribute to determining subsequent life course events and outcomes. Foetal growth defines intrauterine life and bio-physical changes which culminate in the birth process and associated parameters. The pattern of changes in body dimensions, proportions and compositions after birth reflects the event of early postnatal growth. Early child feeding/ diet, another life course event, influences postnatal growth trajectory. Adolescence, with the attendant pubertal changes, presents a unique event in the life course. Life-style, as a conceptual event, reflects the way of life of a person: active/ ambulant or sedentary, dietary habits, exercise routine, work pattern and social habits including smoking and drinking among others. Infections and several diseases/ disorders may also be events in the life course. Death, the physical end of life, marks the end of life course concluding the ‘Womb to Tomb’ transition.

An appreciation of the various events in the life course is essential for an in-depth discourse of their contributions to adult disease induction and possible strategies for prevention and control. Foetal growth, early postnatal growth and early child nutrition are regarded by some authors as offering a ‘Critical Window’ for interventions concerning policy and practice (McCance and Widdowson 1974).

### **Life Course Events And Adult Disease Induction: The Nexus**

There are several reports which suggest linkage of adult disease induction with life course events. A correlation was reported within different areas in Norway between coronary heart disease and infant mortality some 70 years earlier and this was thought to be due to poverty and nutritional deficit resulting in life-long predisposition to more affluent life-style in adulthood (Forsdahl 1977). Adult blood pressure was reported to be inversely related to birthweights of persons born in 1946 in the United Kingdom (Wadsworth et al 1985). Curhan et al (1996) reported a U-shaped curve relationship between birthweight and obesity. The Dutch Hunger Winter of 1944-1945 was linked to later adult disease in the offspring of afflicted mothers (Ravelli et al 1998). This suggested the influence of maternal pre-conceptual nutritional state



on later adult disease in the offspring. Also, Barker and Osmond (1986), from the observation of the link between deaths from stroke and cardiovascular diseases and neonatal mortality in various parts of England and Wales, concluded that poor health and physique of mothers determined the predisposition to stroke in their offspring. Reasoning further, they suggested that environmental factors affect growth and development in early life and result in amplified risk for coronary heart disease. Other reports have linked prenatal and postnatal growth with later adult disease comprising mostly components of the 'Metabolic Syndrome' including insulin resistance. These observations led to the 'Foetal Origins of Adult Disease (FOAD)' and 'Developmental Origins of Health and Disease (DOHaD)' Hypotheses: 'Environmental factors, particularly nutrition, act in early life to programme the risks for early onset of cardiovascular and metabolic disease in adult life and premature death' (Barker et al 2002).

This presentation intends to highlight some perspectives from Nigeria, a resource-compromised economy and, therefore, will not discuss the various mechanisms explaining the nexus between adult disease induction and the life course events. However, a few points are noteworthy. Genetics and epigenetics are important determinants of preconception/ conception and subsequent foetal growth amplifying the role of 'Maternal Thrifty Phenotype' Hypothesis and the subsequent 'Developmental Plasticity' and 'Programming' (Neel 1962, Lucas 1991, Hales and Barker 1992, Neel 1999). Conception and foetal growth are influenced by the type of conception (natural or assisted/ artificial), singleton or multiple gestation including zygosity/ chorionicity, 'maternal effect', maternal nutritional supplementation and timing in gestation, placental function/ placental nutrition and its determinants and the relevant hormones including insulin, insulin-like Growth Factor I, insulin-like Growth Factor II and Leptin among others.

## LIFE COURSE EVENTS/ ADULT DISEASE INDUCTION AND SOME NIGERIAN PERSPECTIVES

Nigeria, a sub-Saharan economy with Gross National Income Purchasing Power Parity per Capita (GNI PPP/ Capita) about \$2000, lacks the sophisticated and requisite clinical care tools and state-of-the-art research facilities to critically investigate the nexus between life course events and adult disease induction. A few research output from this locale will, however, be highlighted in relation to the events in the perceived 'Critical Window' in the life course to situate our contributions, using appropriate health technology, to the on-going investigations of the nexus.

### **Foetal Growth And Birth: Some Measurable Attributes**

There are measurable attributes at birth which are held as possible surrogates of foetal growth and have presumed implications for adult disease induction.

#### Gestational Age



Gestational age determined at birth defines the presumed duration of foetal life and growth and its accurate determination remains the subject of several scientific investigations as most methods of assessment do not have 95% confidence limits on precision better than +/- 2weeks (Casaer and Akiyama 1970). Accurate maturity determination is relevant to adult disease induction since preterm infants are thought to accumulate intra-abdominal adiposity which predisposes to the insulin resistance syndrome (Singhal et al 2001, Singhal et al 2003a). Accurate gestational age is also required for identification of intrauterine growth restricted infants at birth with its association with altered subcutaneous adiposity and body fat composition together with prediction of future adult disease (Singhal et al 2003b). Several authors have reported various methods of maturity determination at birth (Dubowitz et al 1970, Parkin et al 1976, Finnstrom 1977, Capurro et al 1978, Ballard et al 1979). We have developed several simplified methods from Nigeria with comparable accuracy with the method by Dubowitz et al (1970) (Eregie 1991b, Eregie 1991c, Eregie 2001, Eregie and Muogbo 1991, Eregie 2000). Figures 1 and 2 illustrate some of the simple methods. Our model has been further investigated among Cameroonian newborn infants and, based on ease and speed with accuracy, recommended for use in developing countries with high birth rates and busy delivery/ newborn practice (Sunjoh et al 2004).

### Birthweight

The weight at birth is a more readily measurable parameter which presumably reflects the quality of foetal growth. As a surrogate of foetal growth, it is no longer very meaningful particularly in the prediction of adult disease induction. There are several diverse variables, pre-conceptional and gestational, which determine weight at birth and the various pathways to the birthweight have different predispositions to later disease induction (Lissner et al 1999, Huxley et al 2000, Loos et al 2001, Loos et al 2002, Greenwood and Bell 2003). For a given birthweight, the different body compositions regarding lean mass and fat mass also predict differential predisposition to future adult disease (Singhal et al 2003c). Further, the pattern of adiposity, superficial/ subcutaneous or deep/ intra-abdominal, also suggests different susceptibility to the insulin resistance syndrome and related adult disease. Birthweight is, therefore, no longer very informative in studies investigating 'Body Programming' and adult disease induction. Reports linking low-birthweight with subsequent adult disease may need to further evaluate the different body compositions for more useful correlation.

### Body Mass Index and Age-Weight Relationship

These parameters require the use of birthweight in their classification and have the same drawbacks already discussed in relation to birthweight. While 'Smallness-for-Gestational Age' may reflect intrauterine growth restriction, it does not define the body composition and,



therefore, has limited value in 'Body Programming' studies. Body Mass Index (BMI), on the other hand, has 'twin defects': it does not reflect body composition at birth as a variable for predicting adult disease induction and, as a criterion for diagnosing obesity, it does not reflect the amount and distribution of fat-mass. The critical review of the appropriateness of the BMI as the criterion for the diagnosis of obesity could be the focus of another presentation.

#### Body Composition/ Body Proportion Indicators

Attributes which reflect body composition and body proportionality possibly hold more promise for useful investigation of 'Body Programming' and adult disease induction. We have reported some standards of mid-arm circumference/ head circumference ratio for the assessment of body proportionality/ body composition as simple, rapid and reliable clinical tools in a developing economy (Eregie 1992a, Eregie 1998a) (Figure 3). The first model, in nutritional state assessment reflecting the quality of foetal growth, had a sensitivity of 85.5% and specificity of 92.9% and comparatively better than age-weight classification with sensitivity and specificity of 67.7% and 85.3% respectively. The latter arm/ head standard is independent of precise maturity determination (Eregie 1998a), performed comparatively with the former (Eregie and Abraham 2000) and avoids the difficulty of accurately estimating gestational age. The arm/ head ratio has also been reported to be superior to ponderal index in predicting neonatal metabolic morbidity (Georgieff et al 1988). Additionally, ponderal index uses birthweight in its computation with the attendant drawbacks afore-stated. We have also used the arm/ head ratio standard for the evaluation of infants of diabetic mothers at birth (Eregie 1992b) with implication for practice and follow-up re: adult disease induction. In more developed and affluent economies, more sophisticated investigative tools exist and include, among others, Magnetic Resonance Imaging (MRI) Body Fat Mapping, Dual-energy X-ray Absorptiometry (DEXA), Densitometry, Bioelectrical Impedance Analysis and Deuterium Dilution (Isotope Technology) (Elia et al 2007). Skin-fold thickness and a standard of mid-arm circumference (Eregie 1991a) have also been reported as surrogates of foetal growth.

#### **Early Postnatal Growth**

The postnatal growth trajectory has been investigated for its value in predicting adult disease induction. The growth pattern with the greatest predisposition is the postnatal 'catch-up' growth involving small babies at birth who subsequently become big adults with abnormally increased fat-mass/ lean –mass ratio and huge 'Metabolic Load' for lean-mass with resultant insulin resistance syndrome and the components of the 'Metabolic Syndrome' (Wells 2007). The recognition of the postnatal growth pattern requires the use of appropriate growth standards. We have observed accelerated growth of exclusively breastfed infants possibly making the use of previously available standards to be inappropriate (Eregie 1999). In response to this observation and challenge, we have developed new growth normograms for the proper early postnatal evaluation of exclusively breastfed infants in the first six months of life (Eregie 2001).



Fortunately, one of the normograms (Figure 4) is a standard of arm/ head ratio and, therefore, assesses body composition/ proportionality with its implication for follow-up studies for ‘Body Programming’ and adult disease induction.

### **Early Child Nutrition/ Diet**

There are several detailed reviews of the pattern of early child feeding particularly concerning appetite regulation and Orexigenic-Anorexigenic hormones and will not be the thrust of this presentation (McMillen et al 2005, Mcmillen et al 2006, McMillen and Robinson 2005). The role of the shift from the ‘Hunter-Gatherer’ diet to the ‘Western Toxic’ diet in predisposition to adult disease induction is well known. On the other hand, lactation and breastfeeding (particularly exclusive and optimal breastfeeding) offer a further opportunity for the mother to continue the ‘Body Programming’ of the offspring for the next two years after birth. This is due to the unique content of human breastmilk and the epigenetic benefit of breastfeeding both of which assure normal ‘Body Programming’ and protect against ‘Obesiogenic’ potential (Adams 1995, Alam et al 2003, Wells 2007). Strategies to promote, protect and support exclusive and optimal breastfeeding, i.e. the Baby-Friendly Hospital Initiative (BFHI), should possibly protect against adult disease induction. We have reported several observations towards the promotion of breastfeeding initiative in Nigeria (Eregie 1997, Eregie and Abraham 1997, Eregie 1998c). Inappropriate marketing of Breastmilk Substitutes by Infant Food Manufacturers (IFMs) has possibly contributed to the low exclusive breastfeeding rate in Nigeria. Imaginatively innovative strategies to implement, and ensure compliance with, the International Code of Marketing of Breastmilk Substitutes have been evolved leading to the conceptualization of the ‘INAGOSICI Phenomenon’ to make ‘The Code’ work in Nigeria (Eregie 1998b) (Figure 5).

#### **‘INAGOSICI Phenomenon’**

This phenomenon was conceptualized by the author, as a UNICEF Consultant in 1998, to celebrate the World Breastfeeding Week in Nigeria and to step up the tempo to ensure effective Code implementation and compliance for the promotion and protection of breastfeeding and optimal infant and young child feeding in the country. The term ‘INAGOSICI’ is derived from the factual statement that ‘**I**ndustry **A**nd **G**overnment are **S**imilar **I**n **C**ode **I**mplementation’. The similarity between Industries (Infant Food Manufacturers) and Governments is gleaned from the following and IBFAN State-of-the-Code by Company and by Country (1998):

- i) Both parties (‘Ind-Gov’ Dyad) participated at the 1979 United Nations Meeting on Infant and Young Child Feeding (‘UNMIYCF’) culminating in the production of the draft of the International Code of Marketing of Breastmilk Substitutes
- ii) Both parties contributed to the successive reviews/ revisions of the original draft Code resulting in a significantly weakened draft Code but which must be implemented in its entirety as a minimum to achieve the true spirit and aim of the Code



- iii) Both parties have not strictly discharged their responsibilities within the Code: Most Governments do not have legislations which reflect the Code provisions as a minimum and most Industries do not comply with Code provisions in their entirety as a minimum
- iv) Several Industries are aggressively lobbying most Governments to weaken and dilute their legislations for Code implementation
- v) Both parties are possible contributors to the Code not working with resultant low exclusive and optimal breastfeeding rates.

For effective breastfeeding promotion campaigns, the ‘INAGOSICI Phenomenon’ ensures that Industries and Governments (‘Ind-Gov’ Dyad) must be the specific targets of strategic interventions. A ‘Phenomenon or Factor’ must have effects or outcomes/ impact that are possibly ascribable to it. For the ‘INAGOSICI Phenomenon’, the suggested impact in Nigeria includes the following among others:

- i) Promulgation of Decree 22 of 1999 as an Amendment of Decree 41 of 1990 originally enacted as the Nigerian legislative instrument for Code implementation but very deficient in terms of Code provisions
- ii) Designation of the National Agency for Food and Drug Administration and Control (NAFDAC) as the regulatory authority to enforce Code implementation and monitor compliance and violations
- iii) Establishment of a National Technical Committee to monitor the marketing of Breastmilk Substitutes in Nigeria and develop capacity for Code compliance
- iv) Conduct of several capacity-building trainings on Code implementation, compliance and monitoring for relevant stakeholders involved in infant and young child feeding in Nigeria
- v) Production ,and printing in the official government gazette, of the new NAFDAC ‘Regulations on Infant and Young Children Food and Designated Products 2005’ which largely reflects the Code provisions as a minimum
- vi) From preliminary pilot survey on Code implementation conducted by the National Technical Committee in 2005, exclusive breastfeeding rate is improving but remains unacceptably low.

### **Two ‘Growth spurts’ And The ‘Transgenerational Model’**

There are, at least, two possible ‘growth spurts’ in the life course. The first spurt probably occurs during the first six months of life and coincides with the period of exclusive

breastfeeding. Therefore, strategies to promote exclusive breastfeeding have the additional dividend of optimizing the first growth spurt. This sets a good growth template for the second 'growth spurt' which is pre-pubertal. Starting with the good template offered through the benefits of breastfeeding, and harnessing the growth potential through the pre-pubertal and pubertal years, the desired optimal adult physique is achieved. For the female/ girl-child, this physique determines the pre-pregnancy state which is a critical determinant, through the 'Maternal Thrifty Phenotype' Hypothesis, of foetal growth and birthweight of the offspring. With this 'Transgenerational Model', exclusive breastfeeding impacts positively on birthweight. Breastfeeding promotion has salutary dividend for improved birthweight, optimal child health and growth and possible protection against adult disease induction.

### **Summary Of Nigerian Perspectives And 'Critical Window' In Life Course: Policy And Practice Implications**

This section intends to situate the Nigerian perspectives regarding the 'Critical Window' in the life course for possible policy and practice interventions towards preventing adult disease induction from presumed foetal origins.

#### **Foetal Growth and Birth**

- i) Develop policies, including exclusive breastfeeding promotion/ BFHI, to ensure optimal maternal nutrition and physique, as pre-conceptual factors, to guarantee normal 'Body Programming' of offspring through developmental plasticity
- ii) Adopt policies for appropriate timing of maternal nutritional supplementation in pregnancy
- iii) Use simple, rapid, reliable and accurate clinical tools for gestational age determination to influence subsequent follow-up re: differential predisposition to adult disease induction
- iv) Use simple, rapid, reliable and accurate clinical tools for determination of body composition/ proportionality to reflect the quality of foetal growth and predisposition to later adult disease
- v) Use arm/ head ratio standard as a simple, rapid and reliable clinical tool for assessing infants of diabetic mothers at birth and guide further follow-up practice.

#### **Early Postnatal Growth**

- i) Use appropriate growth standards for the proper assessment of postnatal growth trajectory to recognize 'Catch-up' pattern and inform follow-up practice



- ii) For exclusively breastfed infants, use the newly developed normograms for the first six months of life

#### Early Child Nutrition/ Diet

- i) Promote, protect and support exclusive and optimal breastfeeding to guarantee normal ‘Body Programming’, and prevent ‘Obesigenic’ potential and adult disease induction
- ii) Focus on locale-specific determinants of breastfeeding rates to achieve meaningful promotion campaigns
- iii) Consider the ‘INAGOSICI Phenomenon’ for effective Code implementation to protect and promote breastfeeding and optimal infant and young child feeding
- iv) Build-up and strengthen national capacity for effective Code implementation and monitoring for optimal child health and prevention of adult disease induction
- v) Ensure effective implementation of Child Survival Strategies to assure optimal early child nutrition and protect against later adult disease
- vi) Formulate surveillance strategies to regularly update the accurate situation with prevalent adult diseases particularly components of the ‘Metabolic Syndrome’.
- vii) Develop policies and practice that will amplify the potential synergy and dividends of the two ‘growth spurts’ in the life course and the ‘Transgenerational Model’.

#### **Life Course Events And Institute Interventions: Programmes And Practice For Community Impact**

The Institute of Child Health, University of Benin, Nigeria, where the author is Director, currently discharges several interventions to impact on the community towards the prevention of adult disease induction from possible foetal origins. They include health care provision and appropriate health information dissemination to improve health-seeking behavior and have been detailed in a previous publication (Eregie 2004).

#### Community-based Interventions for Child Health (COBICH)

- i) Project on Integrated Community Child Health Interventions in Nigeria (PICCHIN)
- ii) Expanded Programme on Early Child Care, Health, Education and Development (EPECCHED)
- iii) Household Integrated Initiatives for Child Survival (HIICS)



These interventions ensure possible impact for the largely unreached, unserved and vulnerable children in the community as the health facilities are generally poorly patronized and under-utilized.

#### Public Enlightenment and Mobilization Programme (PEMP)

- i) Code Awareness and Breastfeeding Action Network (CABAN)
- ii) Infectious Disease Control Action Network (IDCAN)
- iii) Integrated Child Survival Action Network (ICSAN)
- iv) Perinatal Action Network for Newborns (PANN)
- v) Fathers' Empowerment and Mobilization Action Network (FEMAN)
- vi) Child Health Awareness and Mobilization Programme (CHAMP)

These domain-related interventions include domain-specific research, capacity-building trainings, appropriate health information dissemination, production of user-friendly information, education and communication materials, advocacy campaigns and networking with relevant stakeholders in child health for cost-effective and sustainable prevention of adult disease induction. It is hoped that data from impact evaluation of these interventions will be available in due course to further guide our strategies.

#### **Conclusion**

This presentation has exposed the ‘‘Quadruple-barrel Survival Tragedy’ faced by the newborn in Nigeria culminating in poor Quality of Life (QOL) and reduced life expectancy. Life course is defined and the events in the life course are reviewed. The nexus between life course events and adult disease induction is discussed and the suggested ‘Critical Window’ for policy and practice interventions include: foetal growth and birth, early postnatal growth and early child nutrition/diet. Some Nigerian perspectives are presented in relation to the events in the ‘Critical Window’. Policy and practice thrusts are highlighted including the consideration of the ‘INAGOSICI Phenomenon’ in Code implementation/ breastfeeding promotion and optimizing the two ‘growth spurts’ with the ‘Transgenerational Model’ for adult disease prevention. The Institute interventions are briefly highlighted for community impact.

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FIGURE 1: Maturity determination using head circumference and mid-arm circumference (Adapted from Eregie 1991b)

TABLE 1  
Gestational age groups and the corresponding mean values, 95 per cent confidence intervals, maturity scores, and their definitions for mid-arm circumference measurements

Gestational age group (completed weeks)	No.	Mean ± SEM (cm)	95% CL (cm)	95% CI (cm)	Definition (cm)	Maturity score
<28	32	6.3 ± 0.2	±0.4	5.9-6.7	<6.9	0
28-30	38	7.3 ± 0.2	±0.4	6.9-7.7	≥6.9 & <7.9	1
31-33	35	8.1 ± 0.1	±0.2	7.9-8.3	≥7.9 & <8.6	2
34-36	41	9.0 ± 0.2	±0.4	8.6-9.4	≥8.6 & <9.9	3
37-39	308	10.1 ± 0.1	±0.2	9.9-10.3	≥9.9 & <10.7	4
≥40	54	10.9 ± 0.1	±0.2	10.7-11.1	≥10.7	5

SEM, standard error of the mean; CL, confidence limits; CI, confidence intervals;  $r=0.856$  ( $P<0.001$ ).

TABLE 2  
Gestational age groups and the corresponding mean values, 95 per cent confidence intervals, maturity scores, and their definitions for head circumference measurements

Gestational age group (completed weeks)	No.	Mean ± SEM (cm)	95% CL (cm)	95% CI (cm)	Definition (cm)	Maturity score
<28	32	23.3 ± 0.4	±0.8	22.5-24.1	<25.4	0
28-30	38	26.4 ± 0.5	±1.0	25.4-27.4	≥25.4 & <28.8	1
31-33	35	29.6 ± 0.4	±0.8	28.8-30.4	≥28.8 & <30.7	2
34-36	41	31.3 ± 0.3	±0.6	30.7-31.9	≥30.7 & <33.4	3
37-39	308	34.0 ± 0.3	±0.6	33.4-34.6	≥33.4 & <34.7	4
≥40	54	34.9 ± 0.1	±0.2	34.7-35.1	≥34.7	5

SEM, standard error of the mean; CL, confidence limits; CI, confidence intervals;  $r=0.867$  ( $P<0.001$ ).

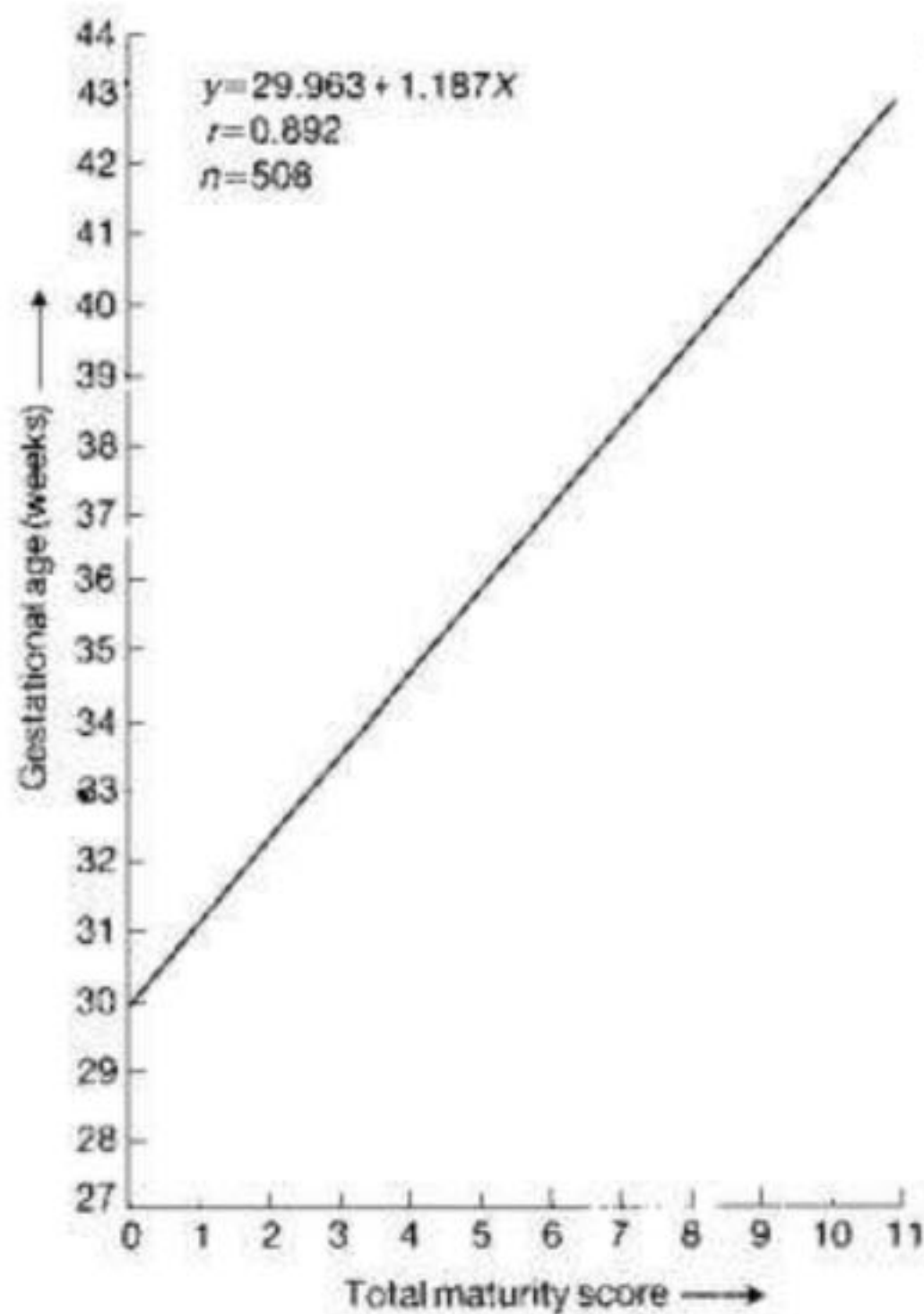

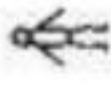
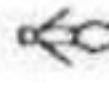
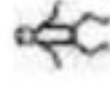
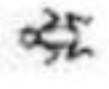






FIG. 1. Regression line of gestational age on total maturity score.



FIGURES 2: Maturity Assessment by an abbreviated and modified clinical tool (Adapted from Eregie 1991c)

TABLE 1  
Features, definitions and maturity scores for estimating gestational age

Features**	Maturity scores*					
	0	1	2	3	4	5
1. Head circumference (cm)	<25.4	≥25.4 and <28.8	≥28.8 and <30.6	≥30.6 and <33.4	≥33.4 and <34.7	≥34.7
2. Mid-arm circumference (cm)	<6.9	≥6.9 and <7.9	≥7.9 and <8.6	≥8.6 and <9.9	≥9.9 and <10.7	≥10.7
3. Posture						
4. Scarf sign						
5. Skin texture	Very thin, gelatinous	Thin and smooth	Smooth, medium thickness, rash or superficial peeling	Slight thickening, superficial cracking and peeling especially hands and feet	Thickening parchment-like, superficial or deep cracking	
6. Ear form	Pinna flat and shapeless, little or no incurving of edge	Incurving of part of edge of pinna	Partial incurving of whole of upper pinna	Well-defined incurving of whole of upper pinna		
7. Breast size	No breast tissue palpable	Breast tissue on one or both sides <0.5cm diameter	Breast tissue on both sides, one or both 0.5-1.0cm	Breast tissue on both sides, one or both >1 cm		
8. Genitalia						
Male	Neither testis in scrotum	At least one testis high in scrotum	At least one testis right down in scrotum			
Female	Labia majora widely separated, labia minora protruding	Labia majora almost cover labia minora	Labia majora completely cover labia minora			

\*If the score for an individual feature differs on the two sides of the body, take the mean. For female genitalia scoring, keep the hips half abducted.

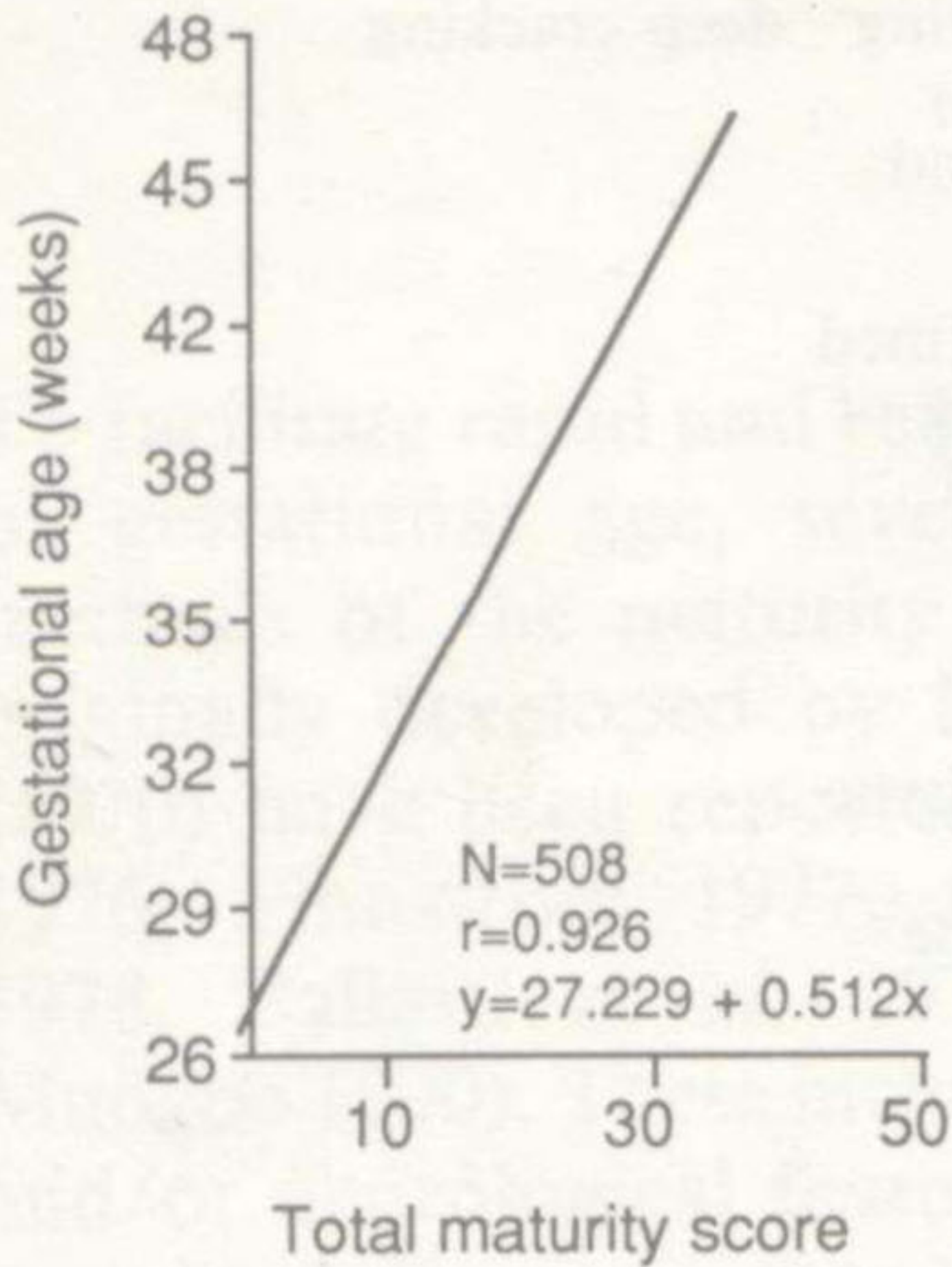
\*\*Features 1 and 2 adapted from previous report (Eregie 1991); 3 to 8 adapted from previous report (Dubowitz *et al.* 1970).

$Y = 25.004 + 0.250x$ ;  $r = 0.928$ ;  $p < 0.001$  (Dubowitz system).

$Y = 26.806 + 0.794x$ ;  $r = 0.911$ ;  $p < 0.001$  (simplified method).

$Y = 27.229 + 0.512x$ ;  $r = 0.926$ ;  $p < 0.01$  (model).





*Fig. 2. Regression line of gestational age on total maturity score for model.*



FIGURE 3: Two standards of arm/ head ratios for determination of body proportionality (Adapted from Eregie 1992 and Eregie 1998)

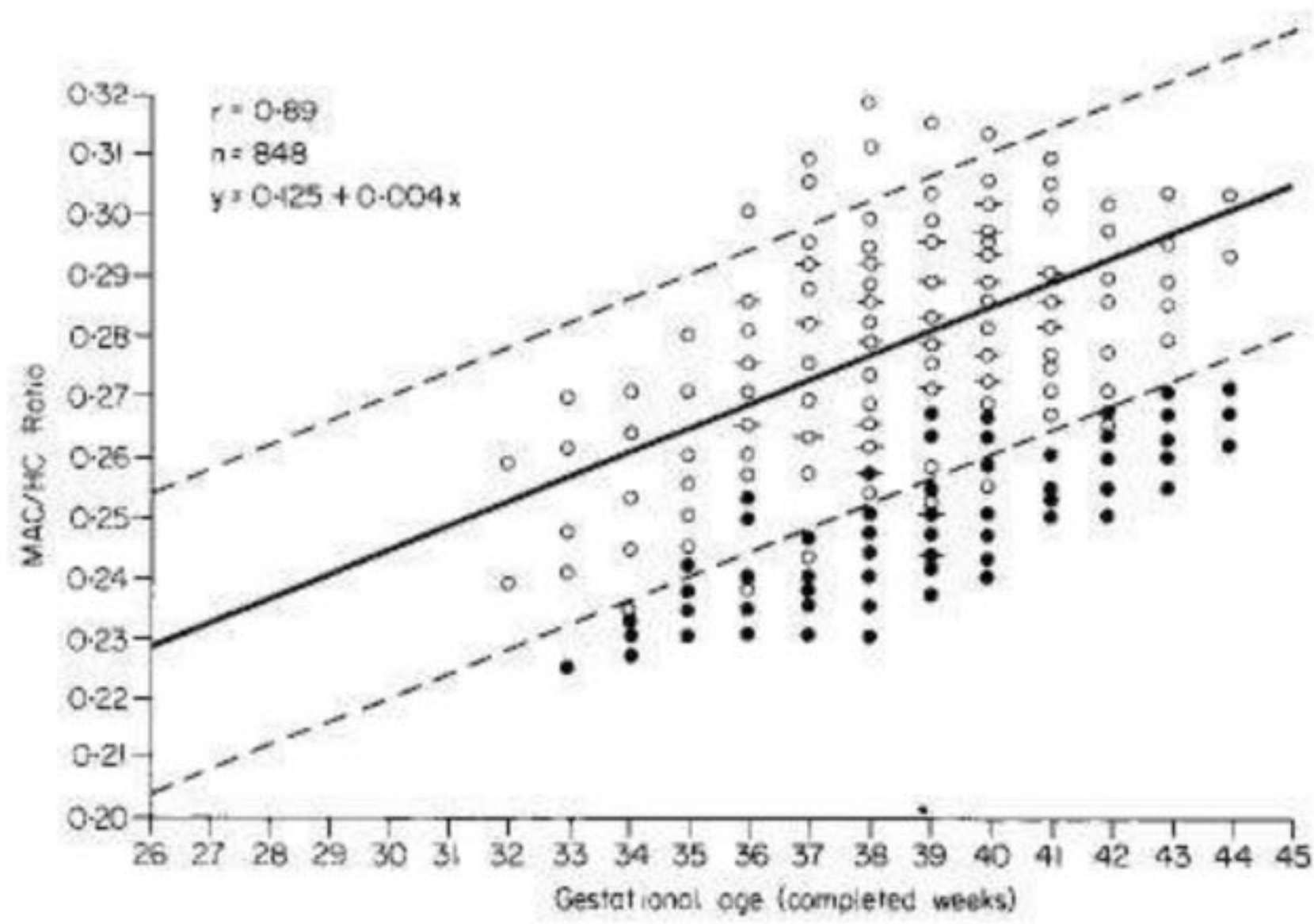


FIG. 1. Scatterplot of 246 infants on the MAC/HC standard. — mean MAC/HC; --- 95% confidence limits; ○ = wellnourished infant (—○— = more than one infant); ● = malnourished infant (—●— = more than one infant).



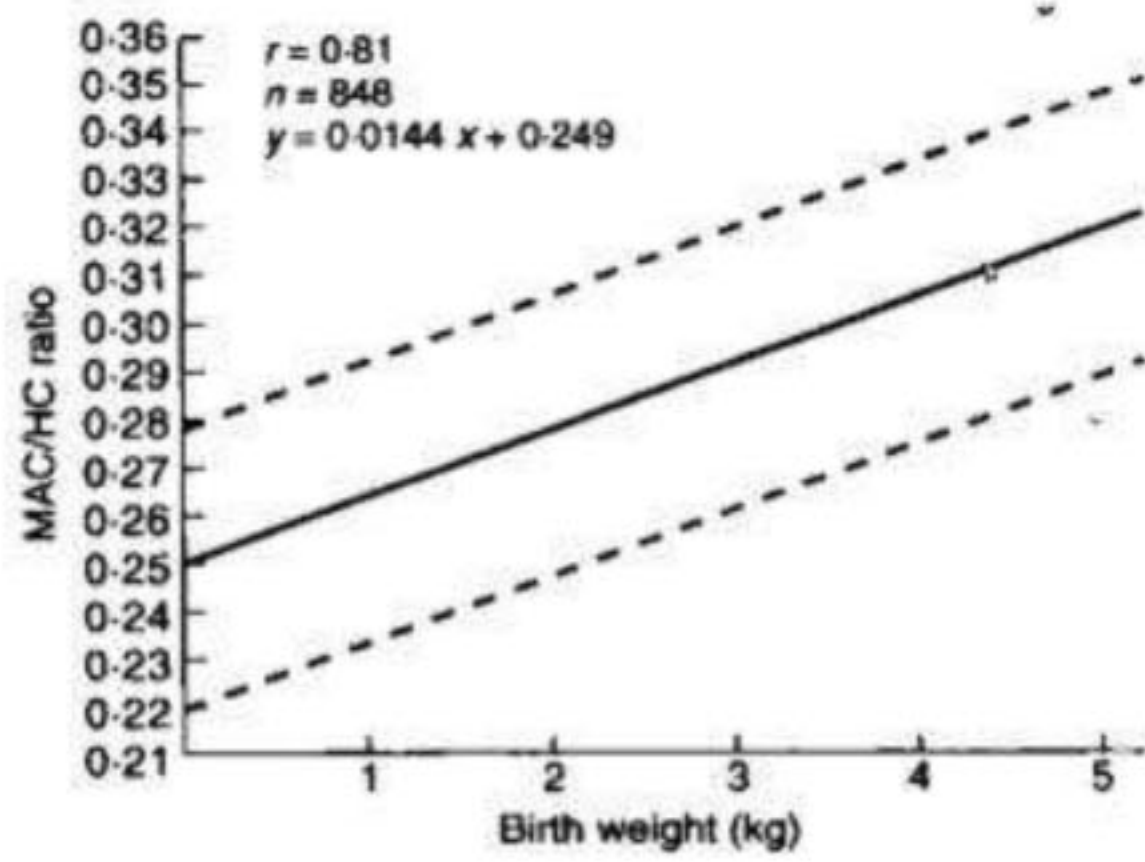


FIG. 1. The new model showing the regression line of MAC/HC\* on birth weight with the confidence belt demarcated by the 95% confidence limits. —, regression line of MAC/HC on birth weight. - - - - -, 95 per cent confidence limits.

FIGURE 4: A new normogram for postnatal growth assessment of exclusively breastfed infants in the first six months of life (Adapted from Eregie 2001)

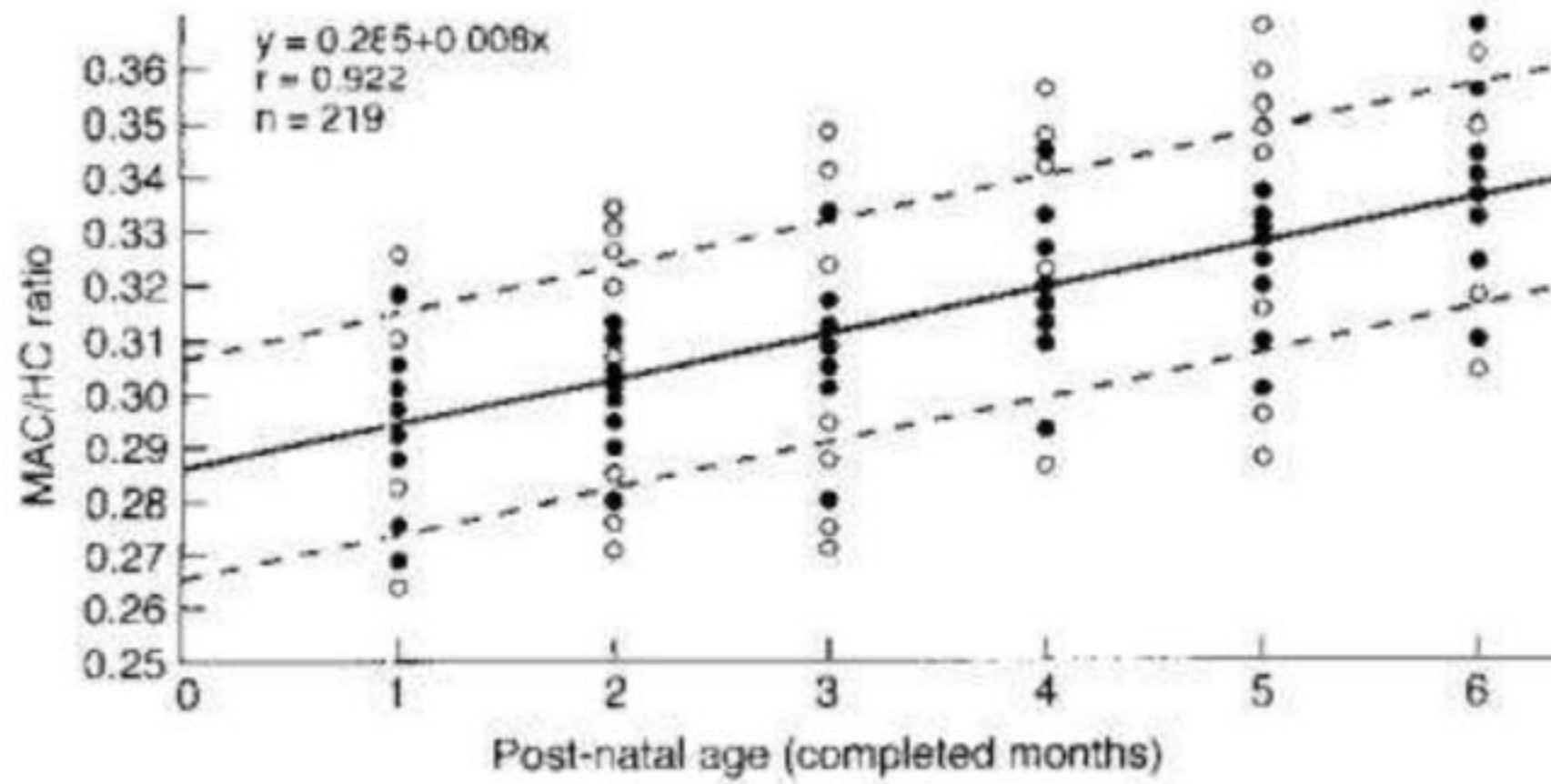
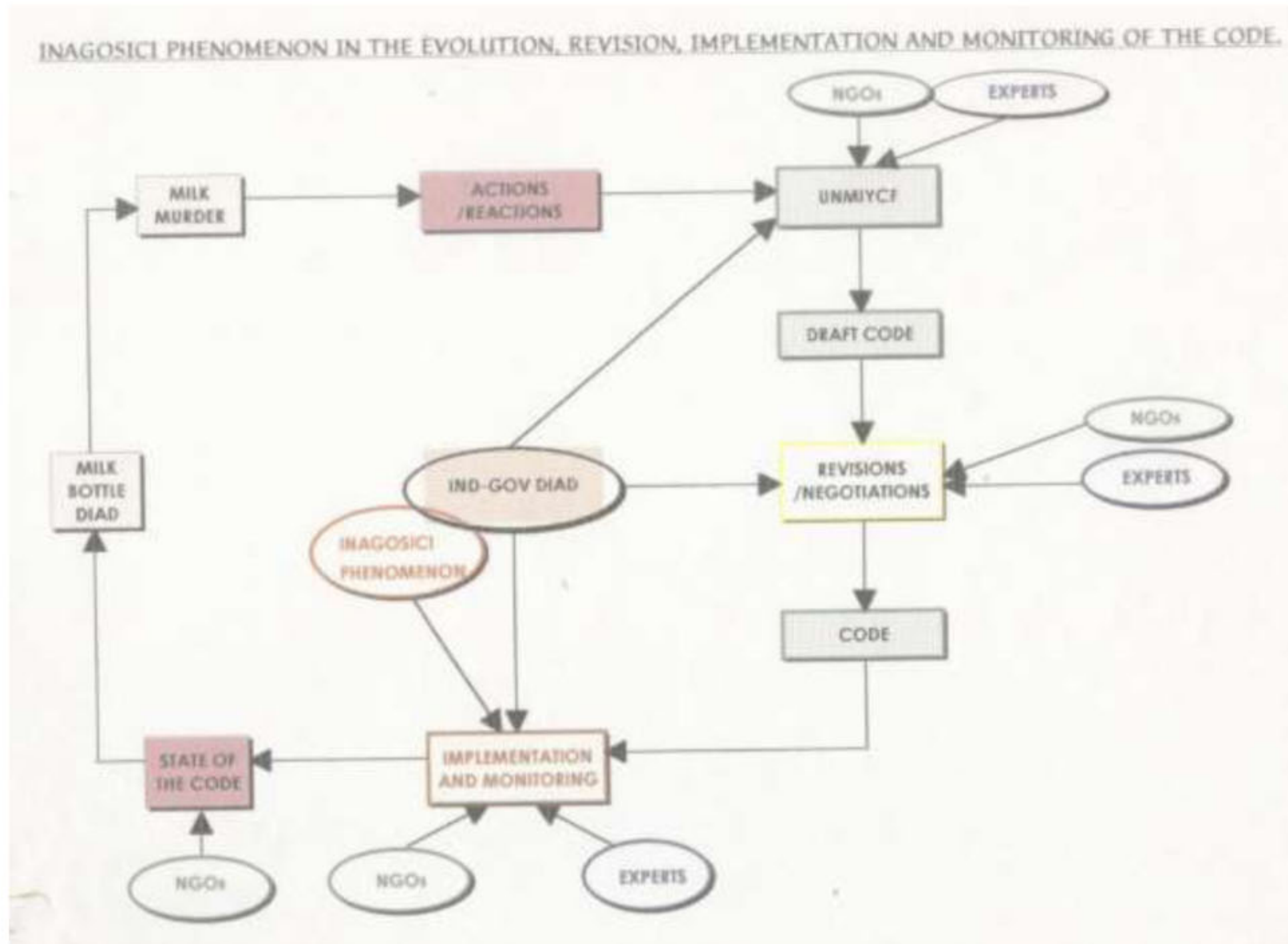


FIG. 3. A scatterplot of 219 infants showing the regression line of MAC/HC ratios on postnatal age with the corresponding 95% confidence limits (— regression line; - - - - - 95% confidence limits; ○ = one infant; ● = many infants).



FIGURE 5: Industry and Government in Code implementation and the 'INAGOSICI Phenomenon'





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