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Exclusive Breastfeeding, Maternal Phenotype and Adult Disease Induction ('DOLSOC^{2}PH^{3}I^{2}N^{2}DIMS'): Amplifying the Pre-FOAD Hypothesis and Rekindling the Archetypal Thrusts of an Inaugural Lecture

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Abstract

The Thematic Thrusts of a 2009 Inaugural Lecture are disposed as Archetypal of the Evolving Thrusts of the currently emerging Nutrition and Epigenetics Research Outpourings. The FOAD Hypothesis has a 'Starting Locus' as the Maternal Phenotype (Including Maternal Nutrition) and links Adult Disease Induction (ADI) with Prenatal and Early Childhood Life-Course Events while the Pre-FOAD Hypothesis has its 'Starting Locus' as Exclusive Breastfeeding (EBF) which, intertwining with Child Survival Interventions (CSI) and optimizing 'Two Growth Spurts', shapes the Maternal Phenotype and impacts on ADI through a 'Transgenerational Model'. Several Conceptual and Contextual Technicalizations in the 'Nutrition and Epigenetics Space' are ventilated albeit as Tantalizing Teasers in some aspects. It is hoped that, with better appreciation of the Pre-FOAD Hypothesis, Maternal Phenotype, Foetal Programming, Developmental Plasticity, Nutritional Epigenetics (Nutrigenomics-Nutrigenetics Dyad), Postnatal Metabolic Niche, Epigenetic Mechanisms and Postnatal Physiological Regenerative Medicine, among several others, Adult Disease Induction (ADI) can be prevented or modulated through Exclusive Breastfeeding (EBF), a Low-cost High-impact Intervention as a 'Starting Locus', taking advantage of the Pre-FOAD Hypothesis.

Keywords: Adult Disease Induction; Child Survival Interventions; Developmental Plasticity; Epigenetics; Epigenetic Mechanisms; EPISTLE Matrix; Exclusive Breastfeeding; FOAD Hypothesis; Foetal Programming; Maternal Phenotype; Maternal Thrifty Phenotype Hypothesis; Metabolostat; Nutritional Epigenetics; Nutrigenetics; Nutrigenomics; Postnatal Metabolic Niche; Postnatal Physiological Regenerative Medicine; Pre-FOAD Hypothesis; TEA Triad

Abbreviations: ADI: Adult Disease Induction; CBF: Continued Breastfeeding; CF: Complementary Feeding; CSI: Child Survival Interventions; EBF: Exclusive Breastfeeding; EBATES: Eregie Breastfeeding Assessment Tool Examination Score; EBS: Eregie BREAST Score; EIB: Early Initiation of Breastfeeding; FOAD: Foetal Origins of Adult Disease; LBW: Low Birthweight; TEA: Technology in relation to Ecology not an Apology

Introduction

This Author delivered the 106th Inaugural Lecture of the University of Benin in Nigeria on the 17th December 2009 and entitled: 'Programming the END from before the BEGINNING: Juxtaposing TECHNOLOGY with the TEA Triad' [1]. The Thematic Thrusts of the Inaugural Lecture appear to be Archetypal of the currently emerging Thrusts of the Nutrition and Epigenetics Research Enterprise and Outpourings. In aligning the Thematic Thrusts of the Inaugural Lecture with the current 'Nutrition and Epigenetics Research Trends', this Author may appear to be re-crystallizing the Conceptual and Contextual Essence of the Inaugural Lecture for better appreciation of the Lecture as did Barker[2] too in his original Presentation on the Foetal Origins of Adult Disease Hypothesis (FOAD Hypothesis) in 1986 and a later 'Re-Interpretation on FOAD Hypothesis Presentation'[3] in 2007

also for its enhanced better understanding. The 'Starting Locus' of the FOAD Hypothesis is the Mother with focus on the Maternal Phenotype [2-4]. The Maternal Phenotype reportedly then drives Foetal Programming in the Prenatal Developmental Stages[4] through Developmental Plasticity [5,6] with a resultant Body Composition of the newborn at birth which sets off the Postnatal Health Trajectory promoting or preventing future Adult Disease Induction (ADI) [1,7-8]. On the other hand, the 'Starting Locus' of the Pre-FOAD Hypothesis is Exclusive Breastfeeding (EBF) [1,9,10]. This Feeding Intervention, coupled with Child Survival Interventions (CSI) [11] and optimizing 'Two Growth Spurts' in a 'Transgenerational Model', results in the Maternal Phenotype which is the 'Starting Locus' of the FOAD Hypothesis and, hence, it is the 'Origin' of the Pre-FOAD Hypothesis. This Communication amplifies the relevance of the Pre-FOAD Hypothesis in preventing ADI and also highlights the Thematic Thrusts of a 2009 Inaugural Lecture as possibly the Archetype of the currently emerging Nutrition and Epigenetics Research Realities.

The Life-Course and The Rekindled Thrusts of an Inaugural Lecture

This Author previously highlighted the importance of the Life-Course Events in the Holistic Health Trajectory of man [12]. The Critical Events will be highlighted as Tantalizing Teasers in this Communication and will not be dilated on exhaustively so as not to detract from its main focus. They include: Conception, Foetal Growth, Birth Process, Early Child Nutrition/ Diet, Early Postnatal Growth, Puberty/ Adolescence, Lifestyles, Infections/ Diseases and Death [1,12]. The 2009 Inaugural Lecture highlighted this Author's Research Contributions to elucidating the importance and relevance of the Critical Events to shaping the Holistic Health Trajectory as they dispose promotion or prevention of Adult Disease Induction (ADI) [1]. In the Inaugural Lecture, the Acronym 'DOHIDIMS' was imaginatively, innovatively and creatively disposed to capture the Non-Communicable Diseases (NCDs): Diabetes, Obesity, Hypertension, Ischaemic Heart Disease, Dyslipidaemia, Insulin Resistance and Metabolic Syndrome[1]. Between 2009 and 2024, a lot has changed and evolved in the 'Nutrition and Epigenetics Space' that this Author, taking cognizance of the currently emerging Thrusts in the Nutrition and Epigenetics Research Undertakings and Outpourings, now redisposes another and more appropriate and Change-compliant Acronym to capture the also evolving and expanding 'Non-Communicable Diseases Space' as 'DOLSOC^{2}PH^{3}I^{2}N^{2}DIMS' (Pronounced 'DOLSOKFINDIMS'): Diabetes, Obesity, Lung Disease, Stroke, Osteoporosis, Cancers, Chronic Renal Disease, Psychiatric Disorders, Hypertension, Hypothyroidism, Hyperthyroidism, Ischaemic Heart Disease, Immune Dysfunction, Non-Alcoholic Fatty Liver Disease, Neuropsychiatric Disorders, Dyslipidaemia, Insulin Resistance and Metabolic Syndrome[13-14]. This imaginative, innovative and creative Acronymic evolution and expansion latches on the Dynamically Transmuting Nutrition and Epigenetics Space and Research Outpourings.

The Nexus of the Archetypal Inaugural Lecture with the Thrusts of the Currently Emerging Nutrition and Epigenetics Research Realities

Recalling the 2009 Inaugural Lecture title 'Programming the END from before the BEGINNING: Juxtaposing TECHNOLOGY with the TEA Triad' [1], the Conceptual and Thematic Thrusts relate to the Life-Course Events [12] with the 'END' of Life, the 'BEGINNING' of Life, 'PROGRAMMING' the Foetus and possibly the New born, Infant and Early Child and Impacting on the 'Causes' leading to the 'END' with focus on the importance of 'BEFORE THE BEGINNING' [1]. The Conceptual and Thematic Thrusts of the Lecture and the currently emerging Thrusts of the Nutrition and Epigenetics Research Enterprise and Outpourings also relate to 'Maternal Nutrition' which is 'BEFORE THE BEGINNING' and the Impact on 'PROGRAMMING' and Development of the Foetus with Long-term Health Implications including the Risks of Chronic Diseases in Adulthood. There is, indeed, a great and intriguing nexus between these Conceptual and Thematic Thrusts with those of the 2009 Inaugural Lecture, therefore, possibly being Archetypal of the currently emerging Nutrition and Epigenetics Research Realities.

Juxtaposing Technology with the 'TEA Triad'

In the 2009 Inaugural Lecture [1], 'Technology' was simply defined as the 'Application of Knowledge to solve Practical Tasks' [15]. Technology can also be defined as the 'Application of conceptual knowledge to achieve practical goals, especially in a reproducible way' [16-17]. Man is expected to have dominion over his 'Environment' and bring it under his 'Control' (Gen 1:28). There is, in fact, a 'Bi-directional Relationship and Interaction' between 'Man' and his 'Environment': 'Man' reportedly interacts with and alters his 'Environment' to realize his 'Desired Attributes' (Parameters) through 'Niche Construction' while the 'Environment' reportedly interacts with 'Man' to effect 'Measurable Changes' ('Features') through 'Adaptation' [18]. Niche Construction is reportedly a complex concept and, with its legacy and 'Ecological Inheritance' and 'Selection Pressure/ Natural Selection', reportedly results in 'Adaptation' for 'Man-Environment Fit' with 'Ultimate-Proximate Explanations' for the 'Evolutionary Flow' [18] which is not ventilated further in this Communication. The 'Man-Environment Interaction' is the Foundational Basis for the 'Gene-Environment Interaction' and is Archetypal of Epigenetics which will be amplified a little further vide infra in this Communication. Epigenetics is reportedly the mechanism by which Environment induces programmed changes (Adaptation) in man while man causes changes in his Environment through Technology. The 'Niche Construction-Adaptation Dyad' is reportedly in a dynamic flux to achieve 'Ecological Equilibrium' for Sustainable Development and Well-being of Man at all Ages and Stages of Life [1,18].

Mahatma Ghandi [19] in the 1920s reportedly made 'Appropriate Technology' popular in preparing India for Independence from Britain by advocating 'Rural Technology' for

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'Self Reliance' and 'Self Sufficiency' [1,19]. In 1973, Schumacher [20] defined 'Appropriate Technology' as 'Technology that is suitable for the situation, capable of being maintained without depleting natural resources or damaging the environment and regards/ fits the community economics'. It is 'Technology as if people mattered' and 'Technology that fits' [20]. It is also 'Technology that is Self-Reliant' [19]. Appropriate Technology is 'Technology that is User-, Client-, Resources- and Environment-Friendly' [1] and it reportedly applies to all Human Developmental Spheres and Existential Spaces with a plethora of attributes including among others: Availability, Acceptability, Accessibility, Affordability, Adoptability and Sustainability. It is also Scientific, Effective, Efficient and Safe and disposes Optimal Resource utilization [1]. According to this Author in the 2009 Inaugural Lecture, 'Appropriate Technology' should be 'TEA Triad-compliant' where 'TEA Triad' is an imaginatively innovative conceptualization that 'Technology in relation to Ecology should not be an Apology'[1]. The 'Man-Environment Interaction' which is Archetypal of Epigenetics should amplify the importance and relevance of the 'TEA Triad'.

Again, 'Technology which fails the 'PESTLE Matrix' is an Apology' [1,12] where 'PESTLE' is an Acronym reportedly indicating: Political, Economic, Social, Technical, Legal and Environmental Dimensions [21]. This Author in the 2009 Inaugural Lecture re-conceptualized the Acronym 'PESTLE' to 'EPISTLE' indicating: Economic, Political, Infrastructure, Investments, Innovations and Institutional, Social, Technical, Legal and Environmental [1]. The intellectually infused 4 'Is' were excerpted as the reportedly disposed 'Bane of Developing Economies' from a 1997 World Bank Report [22]. The 'TEA Triad' was then, therefore, also re-conceptualized as 'Technology which fails the 'EPISTLE Matrix' is an Apology'[1]. Indeed, it was previously reported that 'Modern Technology owes Ecology an Apology' [23].

The 'END'

The 'END' is 'Death' which marks the terminus of the Life Course Events [1,12]. Death results from a spectrum of Contributory Causes and Risk Factors in categories: Accidents, Degenerative Lesions, Infections, Inflammatory Lesions, Metabolic Disorders, Neoplasia and Unknown Causes, among others, and these may be Congenital or Acquired; some are Communicable and some others are Non Communicable. Still others may be Iatrogenic, Idiopathic or Idiosyncratic. The Non-Communicable Diseases (NCDs) as a 'Public Health Scourge and Burden' in Nigeria was presented previously: Type 2 Diabetes (8%), Obesity (12%), Hypertension (10%), Ischaemic Heart Diseases, Dyslipidaemia and Insulin Resistance Syndrome (IRS) [24,25]. In the 2009 Inaugural Lecture, the Acronym 'DOHIDIMS' captured these NCDs: Diabetes, Obesity, Hypertension, Ischaemic Heart Disease, Dyslipidaemia, Insulin Resistance and Metabolic Syndrome [1]. With the phenomenal 'Gene-Environment Interactions and Nutrition-Epigenetics Research Advances and Outpourings', the 2009 Acronym has metamorphosed to 'DOLSOC^{2}PH^{3}I^{2}N^{2}DIMS' capturing: : Diabetes, Obesity, Lung Disease, Stroke, Osteoporosis, Cancers, Chronic Renal Disease, Psychiatric Disorders, Hypertension, Hypothyroidism, Hyperthyroidism, Ischaemic Heart Disease, Immune Dysfunction, Non-Alcoholic Fatty Liver Disease, Neuropsychiatric Disorders, Dyslipidaemia, Insulin Resistance and Metabolic Syndrome [13-14]. These NCDs are the Signposts of Adult Disease Induction (ADI) and the Undergirding Thematic Thrust of the 2009 Inaugural Lecture is how Promotive and Preventive Interventions can modulate their 'Prevalence and Public Health Burden, [1]. The importance of the 'Environment' will be amplified further, albeit as Tantalizing Teasers, under Exposome and Exposomics [26-28].

The 'BEGINNING'

The 'Origin' of the Life-Course Events is the 'BEGINNING' and this is 'Conception' with 'Birth' as an 'Incidental Occurrence' in the Life-Course Trajectory [1]. 'Conception' in the Life-Course Events [12] is 'The Process of the Male Gamete (Spermatozoon) penetrating and Fertilizing the Female Gamete (Ovum) to produce a Zygote and 'Signposting' the commencement of Pregnancy [29,30]. Between 'Conception' and 'Birth' is the 'Prenatal Period (Gestational Period)' which reportedly includes three distinct Stages: The Germinal Stage (1st 2 weeks), Embryonic Period (3 to 8 weeks) and Foetal Period (9 weeks to Birth) [5]. In the 'Prenatal Period' and beyond, the 'Environment' reportedly interacts with the Genetic Make-up of the 'Growing and Developing Being' to shape its 'Future Health Trajectory' through Foetal Programming [5,6] and Developmental Plasticity [31-32] with the 'Molecular Biological Phenomenon of Epigenetics [33,34]. For this discourse to be better appreciated, the 'Environment' reportedly includes, but not limited to: 'Internal' and 'External' Factors which interact with and shape the Structure, Function and Metabolism of the Organism and may be Physical, Chemical, Biological and includes 'Nutrition and Metabolites/ Bioactive Substances, [34].

The FOAD Hypothesis

The Foetal Origins of Adult Disease Hypothesis (FOAD Hypothesis) reportedly is the concept that 'Events during early development have a determinant effect on the subsequent risk for future Adult Disease Induction (ADI) [35]. As originally conceptualized by David Barker [2], 'Low Birthweight (LBW)' was conjectured as a 'Marker' for 'Poor Foetal Growth and Nutrition' and linked to Coronary Artery Disease, Hypertension, Obesity and Insulin Resistance. With further studies, the possible 'Adult Disease Profile [35] reportedly increased to include NCDs captured by 'DOHIDIMS'1 in the 2009 Inaugural Lecture and is now further expanded to a myriad of 'Adult Diseases/ Disorders [4,13,14,35] captured by 'DOLSOC^{2}PH^{3}I^{2}N^{2}DIMS' in this Communication. LBW, disposed as a 'Marker', reportedly stands proxy for both Prenatal/ Foetal Health and Predictor of Postnatal/ Later Life and Adult Health [35]. The 'Early Development Events' cover the 'Prenatal Period' and 'Early Childhood' and include, beyond 'Foetal Environmental Exposure and Nutrition', Stress (Nutritional

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and Non-Nutritional), Infection, Inflammation, Toxins, Hypoxia etc during various 'Critical Stages of Development' and, they in various and varied ways, shape the Adult Phenotype [35]. In addition to LBW as a 'Marker' of 'Prenatal Events', other possible 'Markers' reportedly include: Smallness-for-Gestational Age (SGA), Largeness-for-Gestational Age (LGA) and Prematurity [35]. This typifies the Maternal Thrifty Phenotype Hypothesis [36] wherein the 'Maternal Phenotype (Malnutrition as a Case-in-Point)' determines the Structure, Function, Genetic Expression and Metabolism of the Foetus (Foetal Programming) with resultant Body Composition, Behaviour and Cognition that shape future Adult Diseases [36]. Antedating the FOAD Hypothesis, and reportedly as an impetus, is the Epidemiological Report on the 'Dutch Hunger Winter (1944 – 1945)' with the development of later NCDs in the offspring of mothers who survived the assault [36]. The concepts of Maternal Thrifty Phenotype Hypothesis and Foetal Programming [35] will be ventilated further, albeit as Tantalizing Teasers but with nexus to the 2009 Inaugural Lecture Thematic Thrusts, in this Communication.

From 'BEFORE THE BEGINNING' and the Pre-FOAD Hypothesis

While the 'BEGINNING' of the Life-Course Events is 'Conception [1,29,30] Conceptually and Contextually, the 'BEGINNING' of the FOAD Hypothesis is reportedly the Mother and the Maternal Phenotype [1,2-4,14]. The Maternal Phenotype reportedly determines the 'Prenatal Intrauterine Human Environmental Exposures' which contribute to shaping the Foetal Programming [4,14] through the Developmental Plasticity [5,6] with the resultant Body Composition at birth which, among others, reportedly determines the future 'Postnatal Health Trajectory' with promotion or prevention of Adult Disease Induction (ADI) [1-8,14]. From the 2009 Inaugural Lecture, the 'BEGINNING' is, therefore, the Mother and Maternal Phenotype and 'BEFORE THE BEGINNING' reflects determinants which contribute to the Maternal Phenotype and Exclusive Breastfeeding (EBF) is disposed as one such determinant and, specifically, is presented as the 'Starting Locus' of the Pre-FOAD Hypothesis which conceptually encapsulates 'Exclusive Breastfeeding (EBF) assuring the female child experiences the 'First Growth Spurt' in the first 6 months of life and coupled with Child Survival Interventions (CSI) [11] and optimizing the 'Prepubertal Growth Spurt (Second Growth Spurt)' results in a Maternal Phenotype which facilitates the desired Foetal Programming through Developmental Plasticity with the resultant Optimal Body Composition at 'Birth', in a 'Transgenerational Model', protecting against Adult Disease Induction (ADI) [1,9-10]. This is the imaginatively innovative Conceptual and Contextual Technicalization disposed as the 'Pre-FOAD Hypothesis [1,9,10] as its 'Starting Locus' antedates that of the 'FOAD Hypothesis'. With the Pre-FOAD Hypothesis, Exclusive Breastfeeding (EBF) is a Critical Determinant of Maternal Phenotype and, therefore, of Adult Disease Induction (ADI) [1]. In line with the currently emerging Thrusts of Nutrition and Epigenetics Research Outpourings disposing Long-term health implications of Foetal Programming related to maternal nutrition including risks of chronic diseases in adulthood, Exclusive Breastfeeding (EBF) determines Maternal Phenotype (And, therefore, 'Maternal Nutrition') which, in turn, undergirds Foetal Programming through Developmental Plasticity resulting in a Body Composition at 'Birth' reportedly with a specific 'Postnatal Metabolic Niche' which promotes or prevents Adult Disease Induction (ADI).

Exclusive Breastfeeding, Foetal Programming and Developmental Plasticity

With Exclusive Breastfeeding (EBF) and the resultant Maternal Phenotype under the Pre-FOAD Hypothesis [1,9-10], the 'Intrauterine Nutritional Environment and Exposure' presented by the Mother through Developmental Plasticity reportedly alters the Structure, Function, Metabolism and Gene Expression of the Foetus through effects on the Hypothalamo-Pituitary-Adrenal Axis to endow the Newborn at 'Birth' with a 'Postnatal Metabolic Niche' through the complex process of Foetal Programming [4,14,37,38]. With the unique 'Postnatal Metabolic Niche', the baby is endowed with a 'Metabolostat' (Like a 'Thermostat') which enables the Baby and the 'Developing and Growing Child' to 'Adjust and Regulate' its Metabolic Activities to align with what the Maternally-induced Foetal Programming has set for the 'Metabolostat' [1]. The Developmental Plasticity is reportedly the process of 'One Gene', 'Plastic' or 'Sensitive', being able to manifest 'Different Phenotypes' occasioned by 'Various and Varied Environmental Exposures, Stresses or Challenges' [5-6]. So, with Exclusive Breastfeeding (EBF) being a determinant of the Maternal Phenotype which dictates the 'Intrauterine Nutritional Environment Exposure' undergirding the Foetal Programming through Developmental Plasticity, the 'Human Conceptus' is born with a 'Postnatal Metabolic Niche' and the 'Metabolostat' to set off on a Postnatal Health Trajectory that may Promote or Prevent Adult Disease Induction (ADI) [1-8,14,35-37]. With Foetal Programming, the 'Environment (Including Maternal Phenotype and Maternal Nutritional Status)' impacts on the Human Being from Early Life ('Prenatal Period') to Later Life ('Adulthood') [1,4,14].

Exclusive Breastfeeding, Epigenetics, Nutritional Epigenetics and Nutrigenomics-Nutrigenetics

Exclusive Breastfeeding (EBF) is part of the 'Environment' of the Human Being and Nutritional Status and Nutritional Interventions reportedly represent 'Environmental Pressures or Exposures' to which the Human Being responds. Epigenetics [33-34] represents the Conceptual and Contextual Technicalization reportedly disposing the 'Study of heritable traits, including stable change of Cellular structure, Function and Metabolism, which result without Alterations to the DNA Sequence [33,34]. The 'Normal or Traditional Mechanism' of Genetic Inheritance is reportedly 'DNA Sequence-based' but in Epigenetics, the

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Organismal Inheritance leaves the 'DNA Sequence Unaltered [37]. Normal 'Development' reportedly involves the 'Organismal Responses and Changes' to the 'Environment' and these 'Cellular Changes' reportedly affect Structure, Function, Metabolism and Gene Expression and are not eclipsed by Cell Division [37]. Simply, Epigenetics reportedly regulates 'Gene Expression'; it reportedly involves 'Covalent Modifications of Nucleic Acids and Histone Proteins which reportedly regulate Gene Structure, Functions, Metabolism and Expression' [32,37]. The word 'Epigenesis' [39] was reportedly first used in 17th Century but 'Epigenetics', as in contemporary usage, was reportedly disposed in the 1990s [40]. The 'Consensus Concept' of 'Epigenetics' reportedly from the '2008 Cold Spring Harbor Meeting' [41] is 'Epigenetic Trait is Stably Heritable Phenotype resulting from Changes in a Chromosome without Alterations in the DNA Sequence' although some Alternate Definitions conjecture the inclusion of 'Non-heritable Traits [42]. To be sure, an example of 'Non-heritable Trait' is the fact that 'Chromatin Remodelling' is reportedly not always heritable and not all 'Epigenetic Changes' involve 'Chromatin Remodelling ' [43]. A reportedly practical and simple appreciation of Epigenetics is 'Cell Differentiation' [31] with 'Totipotent Stem Cells' reportedly transforming into the 'Pluripotent Cell Lines' of the Embryo resulting in the various 'Differentiated Cells' emanating from 'Different Genes' some of which are reportedly 'Expressed' and others 'Repressed [43,44].

Epigenetic Mechanisms

The Phenomenon of Epigenetics has reportedly been extensively studied with Increasingly Dynamic Transmuting Research Outpourings with what is ventilated in this Communication reflecting some Microcosmic Tantalizing Teasers [1,33,34,45-48]. The 'Epigenetic Mechanisms' reportedly include, among others: DNA Methylation, Histone Modification, Chromatin Remodelling and Non-coding RNA Modulation [33,34,45-48]. These Epigenetic Mechanisms are variously reportedly involved in 'Genomic Stability' and 'Genomic Health' through DNA Maintenance, Damaged DNA Repairs, Gene Expression and Gene Repression [33,34] with 'RNA Splicing' with formation of 'Introns' and 'Exons' reportedly occurring during Transcription [34].

DNA Methylation

One of the reportedly most frequently occurring and greatly studied 'Epigenetic Mechanisms' is the DNA Methylation which reportedly involves 'Methylation' of the Cytosine Base ('5mC') in the 'CpG Sites' and 'CpG Islands' involving the 'Cytosine Base' and the 'Guanine Base' in the 'DNA Sequence' and in the region of Telomeres, Centromeres, Repeat Sequences, Dormant X-Chromosomes among others [33,34]. The 'CpG' reportedly represents '5'-Cytosine-Phosphate-Guanine-3' (5'-C-phospate-G-3')' which reportedly has a 'Gene Repression Effect'. It is reportedly catalyzed by the Enzyme '5 DNA Methyl Transferase (5DNMT)' in a 'One-carbon Metabolism Reaction' [33,34]. 'Hypomethylation', therefore, reportedly results in 'Increased Gene Expression' while 'Hypermethylation' results in 'Increased Gene Repression'. There reportedly may also be 'Hydroxymethylation'.

Histone Modification

Another Epigenetic Mechanism is reportedly Histone Modification. The Lysine (Also, Arginine, Serine and Threonine are 'Targets') is reportedly the Amino Acid that is the most frequent 'Target' of the 'Modification Reactions' which reportedly include, among others: Lysine Acetylation, Lysine Methylation, Arginine Methylation, Serine Phosphorylation, Threonine Phosphorylation, Lysine Ubiquitination, Lysine Sumoylation, Dopaminylation etc. Lately, Lactylation has reportedly been documented as a Histone Modification Reaction [49]. Important Enzymes for Histone Acetylation and Histone Deacetylation are reportedly 'Histone Acyltransferase (HAT)' and 'Histone Deacetylase (HDAC)' respectively [34].

Chromatin Remodelling

Chromatin is reportedly the 'DNA-Histone Complex [33,34]. The 'Spatial Relationship' between the DNA and the Histone Proteins (How the DNA is wrapped around the Histone with regards to the 'Levels of Protein Organization' re: Primary, Secondary, Tertiary and Quaternary Structures) also reportedly has 'Epigenetic Mechanism Effect' with possibilities reportedly including Moving, Sliding, Disruption and Restructuring [33,34]. Incidentally, Friday 16th August 2024 on a 'Television Network Programme', an unnamed Guest, while discussing the Topic 'DNA, RNA, 'Protein Structures', Neuroscience, Transformative Technology and Artificial Intelligence', stated emphatically that 'PROTEINS DO EVERYTHING IN LIFE'; from this Communication, 'Epigenetic Mechanisms' is all about the 'Environment' interacting with 'DNA without altering its Sequence' to 'Alter Protein Synthesis' and impact on 'Appearances and Functions in Life'. The Chromatin Remodelling reportedly may be 'Post-Translational Modification' of the Histone Amino Acids or it may reportedly be the 'CpG Islands Methylation of the DNA' in the 'Chromatin Complex [33,34]. The presence of '5-Methylcytosine' in the DNA of the 'Chromatin Complex' reportedly results in Gene Repression. However, the presence of 'Methylated Cytosines' in the 'Gene Coding Region', excluding the 'Transcription Start Site (TSS)', reportedly results in 'Enhanced Gene Expression' [33]. Gene/ DNA Transcription reportedly requires a 'Transcription Factor' which binds to a 'DNA Recognition Sequence (Reportedly usually not more than 10 Bases)' at the 'Enhancer' which reportedly interacts with the 'Promoter Region'. The presence of a 'Methylated Cytosine' in the 'Recognition Sequence' reportedly inhibits the 'Transcription Factor Binding'. Also, 'Methylated Cytosines' in the 'Gene Promoter Region' reportedly attracts 'Methyl-CpG-Binding Domain (MBD)' Proteins. The MBDs reportedly interact with the 'Nucleosomes Remodelling' and 'Histone Deacetylase Complexes' with resultant 'Gene Silencing'. Additionally, 'Demethylation' reportedly occurs in

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the 'Neurons' in 'Learning and Memory Development' in the brain [33]. There reportedly appears to be a reciprocal occurrence of 'DNA Methylation' and 'Histone Lysine Methylation [33].

Non-coding RNA Modulation

Only about 2% of the Human Genome is reportedly involved in 'Translation' into 'Protein Synthesis' while the vast majority of about 98% is involved in 'Transcription' into 'Non-coding RNAs (ncRNAs)' with diverse Sizes and Functions [34,50]. There are reportedly many Types of the 'Non-coding RNAs (ncRNAs)' determined by their 'Length of Nucleotides': 'Small ncRNA (sncRNA); 18-200 Nucleotides', 'Long ncRNA (lncRNA); more than 200 Nucleotides' and 'Circular ncRNA (circRNA); have 'Closed Loop Structures [50]. The 'sncRNAs' are reportedly sub-classified as: microRNAs (miRNAs), small nuclear RNAs (snRNAs), and piwi-interacting RNAs (piRNAs). The 'ncRNAs' are reportedly not involved in 'DNA Translation'/ 'Protein Synthesis' but possibly in such other Cellular Processes as: Proliferation, Apoptosis, Autophagy etc; they reportedly may also be promising useful 'Disease Biomarkers' [34]. The miRNAs are reportedly involved in pairing 'RNA-induced Silencing Complex (RISC)' with '3'-Untranslated Regions (3'-UTRs)' of mRNAs with resultant Gene Repression, Translation Inhibition and 'HDACs and DNMTs Modifications' with Epigenetic Regulatory Implications [34]. The 'lncRNAs', acting as scaffolds, are also reportedly involved in Enzyme Modifications, DNA Methylation, Histone Modification and Chromosomal Restructuring [34]. The 'circRNAs' are reportedly generated during 'RNA Splicing' with 'circRNA' derived from 'Introns (Intronic circRNAs)', 'Exons (Exonic circRNAs)' or combined 'Introns and Exons'. With 'RNA Splicing', only 'Exons' are reportedly involved in 'Translation'/ 'Protein Synthesis' and 'Introns' may support 'Transcription' and mRNA production [34]. The 'circRNAs' are reportedly involved in the Regulation of Metabolic Processes and may be useful as 'Disease and Therapeutic Biomarkers'.

Nutritional Epigenetics

The concept of Nutritional Epigenetics reportedly disposes the 'Interaction between Food with its 'Bioactive Components' and the 'Genetic Make-up' of the individual [13,45-48]. While Epigenetics explores the 'Gene-Environment Interaction' and 'Phenotype spectra from Alterations which leave the DNA Sequence Unaltered [33,34], Nutritional Epigenetics specifically focuses on 'Food and its 'Bioactive Components' in the Environment [13,45-48]. There are reportedly two equally important aspects of Nutritional Epigenetics in a 'Bidirectional Interaction' viz: Nutrigenomics which covers Conceptual and Conversational Discourse of the 'Effects of Bioactive Components of Food Interacting with the 'Genetic Make-up' to impact on Gene Structure, Functions, Metabolism, Stability, Health and Expression [13,45-48] while on the diametrically opposite disposition, Nutrigenetics covers the Conceptual and Conversational Discourse of the 'Effects of Food and its Bioactive Components on an individual as determined by the 'Genetic Make-up''[13,45-48,51]. Breastfeeding and Breastmilk in Exclusive Breastfeeding (EBF) reportedly present 'Complex Systems Biology' with 'Nutrigenomics-Nutrigenetics Dyad' exposing the 'Growing and Developing Child' to Nutrients Intake ('Nutriome'), Epigenomes and Bioactives which assure 'Personalized and Precision Nutrition [45-48,51]. The 'Epigenomes' in Breastmilk reportedly include, among others: 'Leptin-Ghrelin which modulate Energy Balance-BMI Growth Trajectory', 'Exosome-related miRNAs', 'Stress Modulators which impact on Neurodevelopmental-Mental Stress Status', 'Immunomodulators which determine resistance to Infections'- [45-48,51].

The 'Bioactive Food Components' reportedly yield 'Active Chemical Substances and Species' which participate in Chemical Reactions involved in 'Gene Regulatory Mechanisms' through Gene Maintenance, Damaged DNA Repairs, Gene Structure, Function and Metabolism, 'Gene Health and Stability' and Gene Expression through the Epigenetic Mechanisms: DNA Methylation, Histone Modification, Chromatin Remodelling and Non-coding RNA Modulation [13,33,34,51]. There reportedly are two ways 'Nutrient Bioactives' interact with 'Genetic Make-up': 'Induce or Repress Gene Expression' and/ or 'Yield 'Single Nucleotide Polymorphisms (SNPs)- through altered Metabolic Pathways and mediation of the Nutrients Interactions with the Pathways' [13]. The 'Bioactive Nutrients' reportedly may alter Gene Expression and Structure, Diet alter Risk Factors for Disease, Diet Regulates Genes affecting the Onset, Progress and Severity of Chronic Diseases, Diet affects the Balance between Health and Disease determined by the 'Genetic Make-up' and Dietary Requirements [51]. Nutritional Status and Genetic Make-up undergird the concept of 'Precision or Individualized/ Personalized Nutrition and Dietary Interventions [13].

'Dietary Bioactives' reportedly influence DNA Methylation by yielding 'Methyl Groups' involved in the generation of 'S-Adenosylmethionine (SAM)' involved in DNA Methylation and altered DNMT Activity. The 'Methyl Group Status' reportedly depends on Vitamin B_{12}, Folate and B_6 among others. Folate reportedly mediates 'One-carbon Metabolism' involved in DNA Methylation and dependent on the availability of Vitamins B_{12}, B_6, Riboflavin, Niacin and Minerals (Zinc, Iron, Cobalt among others) [13]. Therefore, as Tantalizing Teasers, Suboptimal Dietary Intakes are reportedly associated with some Health Disorders: Cardiovascular Disorders (Vitamins B and E, Carotenoids), Cancers (Folate, Carotenoids), Neural Tube Defects (Folate), Inadequate Bone Mass (Vitamin D), Coronary Artery Disease (Vitamins B_{12}, B_6 and Folate) [13].

Epigenetics Research Tools

With currently emerging Nutrition and Epigenetics Research Enterprise and Outpourings and increasing advances in 'Research

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Governance and Research Technologies', Epigenetics is reportedly denominated in 'Epigenetic Editing', 'Epigenetic Biomarkers', 'Epigenetic Diagnostics', 'Epigenetic Drugs' and 'Epigenetic Therapeutics'; possibly 'Epigenetic Holistics' too [34]. With Epigenetic Diagnostics and Epigenetic Therapeutics, 'Predictive, Promotive, Preventive, Proactiive, Precision Personalized Medicine (P6M)' is reportedly facilitated. This brief Tantalizing Teaser presents in a 'Snapshot' some 'Research Tools/ Technologies' in Epigenetics and Epigenomics without dilating exhaustively on their Operational Details and, because of their differential Advantages and Limitations, may reportedly usually be deployed in combinations [33,34]:

i. **Chromatin Immunoprecipitation and Sequencing (ChIP-seq):** In 'Genome-wide Studies', this Tool reportedly facilitates investigation of Protein/ DNA-binding and Histone Modification Sites. Specific Antibodies immunoprecipitate DNA Fragments for Purification, Sequencing and Mapping to the Genome to locate the 'Interaction Site' in relation to the 'Transcription Start Site (TSS) [34].

ii. **In situ Hybridization and Proximity Ligation Assays (ISH-PLA):** These reportedly detect Specific Gene Loci of Histone Modifications in Single Cells. It is reportedly not widely used as it is highly Antibody-dependent.

iii. **Deoxyribonuclease I (DNase I)-Hypersensitive Site Sequencing (DNase-seq):** Reportedly evaluates 'Chromatin Accessibility' and its Undergirding Regulatory Mechanism. Huge number of cells are reportedly required limiting the usefulness.

iv. **Assay for Transposase-Accessible Chromatin using Sequencing (ATAC-seq):** This reportedly maps 'Genome-wide Chromatin Accessibility' using little number of cells. Bioinformatics Analysis reportedly limits its use.

v. **Formaldehyde-assisted Isolation of Regulatory Elements (FAIRE) and FAIRE coupled with Deep Sequencing (FAIRE-seq):** These are reportedly useful for 'Chromatin Regions Identification'. High 'Background Noise' and 'Low Signal-Noise Ratio' reportedly limit Results Interpretation.

vi. **Micrococcal Nuclease Sequencing (MNase-seq):** Reportedly used for 'Nucleosome Positions Mapping' but reportedly does not give the 'Genome-wide Picture'.

vii. **Bisulfite Sequencing (BS-seq):** Reportedly a High-Throughput Detection Method particularly for '5mC' but reportedly cannot decipher '5-Methycytosine (5mC) from '5-Hyroxymethylcytosine (5-hmC).

viii. **Oxidative Bisulfite Sequencing (oxBS-seq):** Reportedly developed specifically for detecting '5-Hydoxymethylcytosine (5hmC).

ix. **5-Formylcytosine (5fC) Chemically-assisted Bisulfite Sequencing (fCAB-seq):** The method is reportedly for mapping and sequencing 'Formylcytosine (5fC).

x. **Fluorescence In Situ Hybridization (FISH):** This reportedly locates specific DNA Sequence on a Chromosome.

xi. **Cleavage Under Targets and Tagmentation (CUT&TAG):** Reportedly Highly Antibody-dependent with High-resolution and reportedly for small samples and single cells in 'Transcription Factors Identification' and Histone Modifications in 'Complex Cellular Systems'

xii. **Cleavage Under Targets and Release Under Nuclease (CUT&RUN):** This is reportedly for 'Mapping Protein-DNA Interactions' in situ and is user-friendly [34].

Further research into the Pre-FOAD Hypothesis, Maternal Phenotype and Adult Disease Induction (ADI) can hopefully benefit from these 'Epigenetics Research Tools' albeit disposed as Tantalizing Teasers in a 'Snapshot' in this Communication.

Exclusive Breastfeeding, Exposomics and Adult Disease Induction

With the Pre-FOAD Hypothesis, Exclusive Breastfeeding (EBF) forms part of the 'Nutrition Environment and Exposures' leading up to the development of the Maternal Phenotype and, with Foetal Programming and Developmental Plasticity, the 'Birth Body Composition' and 'Postnatal Metabolic Niche' are determined[1]. The 'Nutritional Environment and Exposures' of the Early Child also include Exclusive Breastfeeding (EBF) as a component of Optimal Infant and Young Child Feeding (OIYCF) recommended by the World Health Organization (WHO) [52] and UNICEF [53]. The Exposome is reportedly the 'Totality of the Exposures of an individual in the Life Time' and Exposomics is reportedly the Study of the Exposome [26-28]. Exposomics is also reportedly the study of the exposome relying on the application of Internal Exposures (Metabolomics, Transcriptomics, Proteomics, Lipidomics, Glucomics, Metallomics etc) and External Exposures (Biomarkers mapping Exposures, their Effects and Modulators) [26-28,54]. Thus, Exclusive Breastfeeding (EBF) is part of the Exposomics which, in tandem with the FOAD Hypothesis and Pre-FOAD Hypothesis, contribute to shaping the 'Human Postnatal Health Trajectory' which either Promotes or Prevents Adult Disease Induction (ADI) [1-8,14,26-28,33,34].

Exclusive Breastfeeding, Postnatal Physiological Regenerative Medicine, 1st 1400 Days of Life, Adult Disease Induction

Exclusive Breastfeeding (EBF) is the 'Starting Locus' of the Pre-FOAD Hypothesis with the determined Maternal Phenotype endowing the Conceptus with Foetal Programming through Developmental Plasticity to have a 'Birth Body Composition' which sets off the New-born, with a 'Postnatal Metabolic Niche', on a 'Human Postnatal Health Trajectory [1-8,14,33,34]. In fact, Exclusive Breastfeeding (EBF) was disposed as a 'Metabolic Niche

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[54] with amplification of the 'Intrauterine Life' and 'Extrauterine Life' forming a 'Growth and Development Continuum [1,55-56]. It was previously suggested that, Conceptually and Contextually with Technicalization, a 'Critical Window [55,56] can be conjectured within the 'Growth and Development Continuum'. With Exclusive Breastfeeding (EBF) being a 'Metabolic Niche [54], coupled with the Breastmilk the baby is fed with given its 'Postnatal Metabolic Niche', the 'Critical Window' in the 'Postnatal Period' can be optimized to continue the 'Body Programming' with future Promotion or Prevention of Adult Disease Induction (ADI). Since Breastmilk reportedly contains 'Stem Cells [57,58] with their 'Totipotency' and 'Pluripotency' of 'Committed Cell Lines', in addition to the 'Breastmilk Epigenomes' and the Epigenetic Mechanisms of the Nutritional Epigenetics (Nutrigenomics-Nutrigenetics Dyad), Exclusive Breastfeeding (EBF) can be conjectured to hold some promise for 'Postnatal Physiological Regenerative Medicine [14] for the 'Human Growing and Developing Infant' in addition to the implications for the Promotion or Prevention of Adult Disease Induction (ADI). This Author previously developed 'Simple Clinical Tools' involving Neonatal Anthropometry Innovations for Neonatal Nutritional Assessments as 'Surrogates' for the 'Quality of Intrauterine Growth and Nutrition [59-60] and has also only recently deployed improved 'Breastfeeding Assessment Tools [61-62] to assure Successful Breastfeeding Practice to galvanize the Benefits and Advantages of Exclusive Breastfeeding (EBF) to the 'Baby-Mother Dyad' including Nutritional Epigenetics (Nutrigenomics-Nutrigenetics) with implications for Promotion or Prevention of Adult Disease Induction (ADI).

Much of Human Development is reportedly completed during the first 1000 days after Conception: Prenatal and Early Postnatal Periods [63]. It is reportedly conjectured that Pregnancy-related Exposures, and Life-Course Events [1,12] (Exposome-Exposomics) and Breastmilk-Breastfeeding-IYCF affect the 'First 1000 Days' re: Health and Development in the first two years of life [64-65]. The WHO [52] and UNICEF [53] recommend Optimal Infant and Young Child Feeding (OIYCF): Early Initiation of Breastfeeding (EIB) within the First Hour of Life, Exclusive Breastfeeding (EBF) for the First 6 months of Life, Introduction of Age-Appropriate, Nutritious and Nutrient-diverse Complementary Feeding (CF) from 6 months while Continued Breastfeeding (CBF) is facilitated until 2 years or Beyond as feasible. With the Subsequent Relevant World Health Assembly (WHA) Resolution 69.9/ '2016 WHO Guidance' on 'Ending Inappropriate Promotion of Foods for Infants and Young Children up to 36 months [66], the 'Early Child Period'/ 'Early Childhood', therefore, can be viewed as the 'First 36 months of Life'. Also, the 'Early Postnatal Period' can, ipso facto, be extended to the 'First 3 years' and, therefore, a new concept of the 'First 1,400 Days of Life' was disposed by this Author [47-48]. The 'Under-Fives Period' has also reportedly been disposed as the 'First 2000 Days of Life [67].

Conclusion

This Communication presents the Thematic Thrusts of a 2009 Inaugural Lecture by this Author as Archetypal of the currently emerging Thrusts of Nutrition and Epigenetics Research Outpourings. The undergirding importance and relevance of the Pre-FOAD Hypothesis is distilled with its 'Starting Locus' as Exclusive Breastfeeding (EBF) which, coupled with Child Survival Interventions (CSI), impacts on Maternal Phenotype (Maternal Nutrition) which effects Foetal Programming through Developmental Plasticity and guiding and shaping the Conceptus to its 'Birth Body Composition' and, with the endowed 'Postnatal Metabolic Niche', sets off the Newborn on a 'Human Health Trajectory' which either Promotes or Prevents the Adult Disease Induction (ADI) encompassing the Chronic Non-Communicable Diseases of Adulthood. In Tantalizing Teasers, several Conceptual and Contextual Technicalizations were briefly ventilated including, among several others: Foetal Programming, Developmental Plasticity, Epigenetic Mechanisms, Epigenetics Research Tools, Exposomics, 'Postnatal Metabolic Niche', 'Postnatal Physiological Regenerative Medicine', 'First 1400 Days of Life' and NCDs captured by the new Acronym 'DOLSOC^{2}PH^{3}I^{2}N^{2}DIMS'. It is hoped that with better appreciation of the Pre-FOAD Hypothesis, and Exclusive Breastfeeding as its 'Starting Locus' with effect on Maternal Phenotype, Adult Disease Induction (ADI) with its 'Public Health Burden' can be prevented or modulated.

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