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ASSESSMENT OF EMBRYO GROWTH IN RELATION TO PARENTAL LIFE STYLE USING MULTI LEVEL MODEL

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ABSTRACT: *Parental life-style or behavior is believed to largely influence the growth of the embryo as evident from previous works of literatures. Thus, spurring the aim of this study as to assess the embryo growth in relation to parental lifestyle and experience using an advanced statistical modelling procedure. The study sample comprised one hundred (100) pregnant women that are considered for the study, drawn from Maitama General Hospital, Abuja. Participants answered a general health questionnaire in gestational. We employed the use of statistical structural equation technique to model the relationship between the parental lifestyles (environmental factors, parental stress, parental life style, parental responsiveness and parental allocation of nutrients) and the embryo growth. The findings of the study analysis rejected the null hypothesis on environmental factors, parental stress, parental lifestyle and parental responsibilities with the reasoning that all items of the questionnaire greatly affect the growth of the embryo. While, accepting the null hypothesis the "increase in nutrition" is a necessary factor for the growth of the embryo. In conclusion, the study opines is that parental life styles greatly affect the growth of the embryo. As a consequence, a first-order implication relating to this study is for parents and health care providers to concentrate on most of the externalities that "destroys" the unborn child. This involves environmental factors, parental stress and responsibilities, nutrition and many others that may need to be addressed for the sake of the unborn generations.*

KEYWORDS: Embryo Growth, Parental Life Style, Multi-Level Model, Pregnant Women

INTRODUCTION

Developmental psychologists consider the process of human development as it relates to physical, mental, cognitive and psychological development as an integral part of human existence. This lifespan development is organized into different stages based on age. Usually, prenatal development is the process that occurs during the 40 weeks prior to the birth of a child, and is heavily influenced by genetics (Engl, 2014). There are three stages of prenatal development. These are germinal, embryonic and foetus. These prenatal developments are also organized into trimesters: the first trimester ends with the end of the embryonic stage; the second trimester ends at week 30, and the third trimester ends at birth (Engl, 2014).

Every human being is made up of cells containing chromosomes, which are the genetic materials that determines many things about a person, such as the eye and hair colour, biological sex and personality traits. Gene expression in organisms is carefully regulated to allow the organism to adopt to differing conditions. Genes can either be dominant or recessive, meaning that they can either be expressed or hidden. Gene regulation is the process by which cells differentiate: while some cells develop into brain cells, others develop into liver cells,

intestinal cells, or the sexual reproductive organs. In all of these developmental stages, the DNA (deoxyribonucleic acid) is responsible for the transmission of genetic materials. A mother's father's (parents') DNA are passed on at the moment of conception, DNA is a ladder-like structure that contains genetic material.

Every human being has a total of 23 pairs of chromosomes. The developing zygote gets half of its chromosomes from one parent and half from the other parent. The first 22 pairs of chromosomes are known as autosomes and determine things such as the eye and hair colour. The last part known as the sex chromosomes determine a person's biological sex: females have XX chromosomes, while males have XY chromosomes.

In mammalian development, the mother transduces environmental information such as nutritional status to her embryo or fetus through the placenta or to her infant through lactation. Fetal growth is genetically matched to the mother's body size (rather than to genetic potential) through what is termed maternal constraint (Foley and Hughes, 2018). Maternal constraint may be mediated, in part, by the limiting effects of placenta size in utero or perfusion on fetal nutrition, but imprinted genes, particularly those linked to the expression of growth factors, may also play a role in the allocation of nutritional resources (Engl, 2014). Maternal constraint is increased with short maternal stature, young or old maternal age, first pregnancy, or multiple pregnancy; in addition, the effects of unbalanced maternal diet or excessive maternal thinness or fatness influenced fetal nutrition in the absence.

In the development of the embryo, the stage lasts from implantation (2 weeks after conception) of the germinal stage until the 8^{th} week of pregnancy. During this stage, rapid growth occurs and organ systems develop. The fetal stage lasts from week 9 up until the child's birth (usually between 38 and 40 weeks). Throughout this stage, the brain continues to grow and develop at a rapid pace.

At the embryonic stage (2 – 8 weeks of pregnancy). After the zygote divides for about 7-10 days and has 150 cells, it travels down the fallopian tubes and implants itself in the lining of the uterus. Upon implantation, this multi-cellular organism is called an embryo (Ward, 2007). At this level, the blood vessels grow forming the placenta. The placenta is a structure connected to the uterus that provides nourishment and oxygen from the woman's body to the developing embryo through the umbilical cord (Ward, 2007).

During the germinal stage of prenatal development, the cells necessary for the placenta, umbilical cord, and amniotic fluid will differentiate to form the embryo (Ward, 2007). At the first week of embryonic period, the embryonic disk separates into three layers: the ectoderm, mesoderm and endoderm (Ward, 2007). The ectoderm is the layer that will become the nervous system and outer skin layers; the mesoderm will become the circulatory system, skeleton, muscles, reproductive system and inner layer of the skin; and the endoderm will become the respiratory system and part of the digestive system, as well as the urinary track (Ward, 2007).

The first part of the embryo to develop is the neural tube, which will become the spinal cord and brain. As the nervous system starts to develop, the tiny heart starts to pump blood, and other parts of the body – such as the digestive tract and backbone – begin to emerge. In the second half of this period, growth is very rapid. The eyes, ears, nose and jaw develop; the heart develops chambers; and the intestine grow (Ward, 2007).

These stages in the growth of the embryo are largely influenced by parent's environmental factor. Certain parenting behaviours are associated with specific adolescent internalizing and externalizing outcome. The extent which this is true forms the crux of this study.

LITERATURE REVIEW

Your dictionary.com (2019) defined an embryo in the reproductive cycle as the stage after the fertilization of the egg that precedes the development into a fetus. It is also defined as an organism in the earlier stages of development before it emerges from the egg or before metamorphosis. In viviporous animals, an embryo is the young animal's earliest stages in the mother's body. Hence, the cells in humans usually grow up to the end of the seventh week in the mother's body.

Some embryo does not survive to the next stage of development. When this happened naturally, it is called spontaneous abortion or miscarriage (Liddell and Scott, 2013). There are a number of reasons why this may occur. According to Stopper, Shiel Jr. and William (2004), the most common natural cause of miscarriage is chromosomal abnormality in animals, or genetic load in plants (Karkkanen, Savolainen and Kaski, 1999).

In some species which produce multiple embryos at the same time, miscarriage or abortion of some embryos can provide the remaining embryo with a greater share of maternal resources. This can also disturb the pregnancy, causing harm to the second embryo. Genetic strains which miscarry their embryos are the main source of commercial seedless fruit. Hence, abortion is the process of artificially (non-naturally) removing the embryo through deliberate pharmaceutical or surgical methods.

In the field of medicine, an embryo is an animal in the early stages of growth and differentiation that are characterized by cleavages, the laying down of fundamental tissues, and the formation of primitive organs and organ systems especially; the developing human individual from the time of implantation to the end of the eight week after conception (Harper, 2010). Hence, it is an animal in the early stages of development following cleavages of the zygote and ending at birth or hatching. It is also seen as the human product of conception up to approximately the end of the second month of pregnancy.

The scientific definition of embryo according to the American Herritage Stedman's Medical Dictionary (2002, 2001, 1995) is an animal in its earlier stage of development, before all the major body structures are represented. In humans, other placental mammals and other viviparous animals, young born as embryos cannot thrive. In marsupials, the young are born during the embryonic stage and complete their development outside the uterus, attached to a teat within the mother's pouch.

In the New Dictionary of Cultural Literacy (2005), an embryo is an unborn or unhatched offspring in the process of development, in particular a human offspring during the period from approximately the second week to the eight weeks after fertilization (after which it is usually termed a fetus). In some instances, the term "embryo" is occasionally used to denote a new or developing idea or perfect.

METHODOLOGY

Research Design

A survey-based design is adopted for this study. It begins with a selection of multilevel (hierarchical) model and contrast it with the traditional linear regression model, using an example of a simulated observational study to illustrate how embryo growth can be statistically assessed in relation to parental life style as well exploring the consequences of ignoring data clustering. Additionally, the type of outcome data and design will be discussed, together with the effects of clustering on sample size and power. Given the enormity of the task to be achieved, this study considers the use of multilevel model for data analysis.

Multilevel models are statistical models of parameters that vary at more than one level (Bryk and Roudernbush, 2002). They are associated with a nested data structure of a hierarchical Bayesian models which are general models with multiple levels of random variables and arbitrary relationship among the different variables. One of the major purposes of multilevel models is to deal with cases where the assumptions of independence is violated. According to Hox (2010), multilevel models are designed to simultaneously analyse variables at different levels by properly include various dependences. This can be achieved by randomly choosing samples with a view to bringing out the fact of the hierarchies of the model, not their proven ones.

Population of the Study

A total of one hundred (100) pregnant women are targeted for the study, drawn from Maitama General Hospital, Abuja. The choice of this target population is to avoid the problem of data clustering in order to focus on a well-defined population from whom the study will randomly choose the respondents until the final households (pregnant) patients are selected. Due to the geographical clustering that would be exhibited by some altitudinal measures, special procedures will be developed to produce valid statistical inference to provide adequate values for a fitted regression models for the study.

Sampling Size and Sampling Techniques

A Simple Random Sample will be adopted for this study. It is a technique of sampling in which every member of a given population has an equal chance of being selected. Using the Donner and Klar (2000) formula for variance inflation factor (VIF) which Murray (1998) also referred to as the design effect, this study can determine the adjusted sample size requirements in the presence of clustering. As a result, if an uncluttered design for a randomized controlled trial requires "n" patients per group to detect the desired effect size with adequate power and a level significance, $\alpha = 0.05$ (5 per cent), then the VIF allows the study to adjust the sample size for a positive Intra class Correction Coefficient (ICC) which measures the extent to which individuals within the same group are more similar to each other than they are to individuals in different groups. If a sample of "m" patients per cluster clinic is sampled, then the study will have to inflate the sampled size by a factor of $1 + (m - I)ICC$ where m = designator for number of patients per cluster to be used in the calculation; ICC = Intra class Correlation Coefficients; VIF = Variance Inflation Factor.

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Instrumentation

A questionnaire will be developed and administered to the target respondents. The questionnaire is made up of two sections (A and B). section A deals with the biodata of the respondents. Section B consisted of items that were grouped into three (3) clusters of age bracket (18 – 30), (31- 45) and (46 and over) on the assumption that these age brackets are within the legitimate child-bearing age, the instrument was weighed on a 4-point rating scale for respondents, to make the respondents providing answers by ticking (√) appropriately on phenomenon such as: Strongly Agreed (SA), Agreed (A), Disagree, (DA) and Strongly Disagree (SD).

Method of Data Analysis

This study employs the method of Structural Equation Modelling (SEM) technique. The SEM is thus an extension of the Confirmatory Factor Analysis, where the vector **y** is understood to represent an arbitrarily chosen observation from the population, maybe the ith. We will have a similar situation with the vector **x** that is a q by 1 column vector.

In SEM (Structural Equation Model) terms, we say that **y** contains the *endogenous variables* and **x** contains the *exogenous variables*. An endogenous variable is one that appears at least once as the dependent variable in an equation. On the other hand, variables that do not appear on the left-hand side are exogenous, or "given." In other words, all variances of, and covariances between, exogenous variables are determined outside of the system. They are not at issue. The variances and covariances of the endogenous variables are being modeled as a function of the exogenous variables. The basic model looks like

$$\begin{bmatrix} y_1 \\ y_2 \\ \cdots \\ y_p \end{bmatrix} = \begin{bmatrix} 0 & \beta_{12} & \cdots & \beta_{1p} \\ \beta_{21} & 0 & \cdots & \beta_{2p} \\ \cdots & \cdots & \cdots & \cdots \\ \beta_{p1} & \beta_{p2} & \cdots & 0 \end{bmatrix} \begin{bmatrix} y_1 \\ y_2 \\ \cdots \\ y_p \end{bmatrix} + \begin{bmatrix} \gamma_{11} & \gamma_{12} & \cdots & \gamma_{1q} \\ \gamma_{21} & \gamma_{22} & \cdots & \gamma_{2q} \\ \cdots & \cdots & \cdots & \cdots \\ \gamma_{p1} & \gamma_{p2} & \cdots & \gamma_{pq} \end{bmatrix} \begin{bmatrix} x_1 \\ x_2 \\ \cdots \\ x_q \end{bmatrix} + \begin{bmatrix} \zeta_1 \\ \zeta_2 \\ \cdots \\ \zeta_p \end{bmatrix}$$

$$\mathbf{y} = \mathbf{B}\mathbf{y} + \mathbf{\Gamma}\mathbf{x} + \zeta. \quad (3.1)$$

So, we have p simultaneous equations. Note that for each of the causal parameters, the γ's and the β's, the subscripts follow the same pattern. The first subscript refers to the equation, in other words the y variable which is the effect. The second subscript refers to the cause.

The p by p **B** matrix contains the coefficients of the regressions of y variables on other y variables with 0's on the diagonal which implies that a variable cannot cause itself. The p by q matrix **Γ** contains the coefficients of the y's on the x's. The error vector, **ζ**, is p by 1. These errors are different than factor analysis errors, they represent *errors-in-equations*, in the way that these equations are specified. Thus, they are also called *specification errors*.

In order to get to a point where we can estimate the model, we need to add some assumptions. To start off innocuously enough, we assume that E(**y**) = **0** and E(**x**) = **0**, which has absolutely no impact on the variances or covariances of these variables [see Equation (4.8)]. We then assume that the **x** and **ζ** vectors are independent,

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$$\text{Cov}(\mathbf{x}, \boldsymbol{\zeta}) = \mathbf{0} \quad (0.2)$$

which is to say that the covariances between the x's and the ζ's consist of a q by p rectangular array of zeroes. We will also need to assume that the determinant

$$|\mathbf{I} - \mathbf{B}| \neq 0 . \qquad (3.3)$$

Now let us define

$$\text{V}(\mathbf{x}) = \text{E}(\mathbf{x}\mathbf{x}') = \boldsymbol{\Phi} \quad \text{and} \qquad (3.4)$$

$$\text{V}(\boldsymbol{\zeta}) = \text{E}(\boldsymbol{\zeta}\boldsymbol{\zeta}') = \boldsymbol{\Psi} . \qquad (3.5)$$

Note that we have "reused" the $\boldsymbol{\Psi}$ matrix from Chapter 9. In confirmatory factor analysis, $\boldsymbol{\Psi}$ was used for the factor covariance matrix. In fact, the use of $\boldsymbol{\Psi}$ as the covariance matrix of the ζ's is actually consistent with its Chapter 9 meaning. At this point we are ready to deduce what is known as *reduced form*. Reduced form requires that we solve for the **y** vector, as below:

$$\mathbf{y} = \mathbf{B}\mathbf{y} + \boldsymbol{\Gamma}\mathbf{x} + \boldsymbol{\zeta}$$

$$\mathbf{y} - \mathbf{B}\mathbf{y} = \boldsymbol{\Gamma}\mathbf{x} + \boldsymbol{\zeta}$$

$$(\mathbf{I} - \mathbf{B})\mathbf{y} = \boldsymbol{\Gamma}\mathbf{x} + \boldsymbol{\zeta}$$

$$\mathbf{y} = (\mathbf{I} - \mathbf{B})^{-1}\boldsymbol{\Gamma}\mathbf{x} + (\mathbf{I} - \mathbf{B})^{-1}\boldsymbol{\zeta}$$

$$\mathbf{y} = \mathbf{G}\mathbf{x} + \mathbf{e} . \qquad . \qquad (30.6)$$

The matrices $\mathbf{G} = (\mathbf{I} - \mathbf{B})^{-1}\Gamma$ and $\mathbf{e} = (\mathbf{I} - \mathbf{B})^{-1}\boldsymbol{\zeta}$ are defined merely for convenience, but **G** does highlight the fact that we can go from the structural parameters in **B** and $\boldsymbol{\Gamma}$ to the classic regression parameters with some algebra.

What is the variance of the y variables? We can use the reduced form derived above to simplify our explanation,

$$\boldsymbol{\Sigma} = \text{E}(\mathbf{y}\mathbf{y}') = \text{E}\left[(\mathbf{G}\mathbf{x} + \mathbf{e})(\mathbf{G}\mathbf{x} + \mathbf{e})'\right]$$

$$= \text{E}(\mathbf{G}\mathbf{x}\mathbf{x}'\mathbf{G}') + \text{E}(\mathbf{G}\mathbf{x}\mathbf{e}') + \text{E}(\mathbf{e}\mathbf{x}'\mathbf{G}') + \text{E}(\mathbf{e}\mathbf{e}') . \qquad (0.7)$$

The 2nd and 3rd terms vanish. To see this, we look at the 2nd component which is given by

$$\text{E}(\mathbf{G}\mathbf{x}\mathbf{e}') = \text{E}\left\{\mathbf{G}\mathbf{x}\left[(\mathbf{I} - \mathbf{B}')^{-1}\boldsymbol{\zeta}\right]'\right\}$$

which, using the fact that the transpose of an inverse is the inverse of the transpose, and passing the constants in **G** and $(\mathbf{I} - \mathbf{B})^{-1}$ through the Expectation operator, is equivalent to

$$\text{E}(\mathbf{G}\mathbf{x}\mathbf{e}') = \mathbf{G}\,\text{E}(\mathbf{x}\boldsymbol{\zeta}')\,(\mathbf{I} - \mathbf{B})^{-1} .$$

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Here we note that E($\mathbf{x}\boldsymbol{\zeta}'$) that appears immediately above is another way to express the Cov($\mathbf{x}$, $\boldsymbol{\zeta}$), and that covariance must be zero by previous assumption. The 3rd term is just the transpose of the 2nd. What the cancellation of the 2nd and 3rd components in equation (0.7) means is that we end up with the following expression for $\boldsymbol{\Sigma}$,

$$E(\mathbf{y}\mathbf{y}') = \mathbf{G}E(\mathbf{x}\mathbf{x}')\mathbf{G}' + E(\mathbf{e}\mathbf{e}') \quad (3.8)$$

At this point we might pause and note the similarity between this expression and it's equivalent for factor analysis, Equation (9.3)! Now, to further flesh out this last equation we need to remember that we had previously defined V($\mathbf{x}$) = E($\mathbf{x}\mathbf{x}'$) = $\boldsymbol{\Phi}$, and V($\boldsymbol{\zeta}$) = E($\boldsymbol{\zeta}\boldsymbol{\zeta}'$) = $\boldsymbol{\Psi}$. Proceeding along those lines we see that

$$E(\mathbf{y}\mathbf{y}') = (\mathbf{I}-\mathbf{B})^{-1}\boldsymbol{\Gamma}\boldsymbol{\Phi}\boldsymbol{\Gamma}'(\mathbf{I}-\mathbf{B}')^{-1} + (\mathbf{I}-\mathbf{B})^{-1}\boldsymbol{\Psi}(\mathbf{I}-\mathbf{B}')^{-1}$$

$$= (\mathbf{I}-\mathbf{B})^{-1}[\boldsymbol{\Gamma}\boldsymbol{\Phi}\boldsymbol{\Gamma}' + \boldsymbol{\Psi}](\mathbf{I}-\mathbf{B}')^{-1}.$$

How about the covariance between $\mathbf{x}$ and $\mathbf{y}$? That would be

$$E(\mathbf{x}\mathbf{y}') = E\left[\mathbf{x}(\mathbf{G}\mathbf{x}+\mathbf{e})'\right]$$

$$= E\left[\mathbf{x}\mathbf{x}'\mathbf{G}' + \mathbf{x}\mathbf{e}'\right]$$

$$= E(\mathbf{x}\mathbf{x}')\mathbf{G}' + E(\mathbf{x}\boldsymbol{\zeta}')(\mathbf{I}-\mathbf{B}')^{-1}$$

$$= \boldsymbol{\Phi}\mathbf{G}' + \mathbf{0} = \boldsymbol{\Phi}\boldsymbol{\Gamma}'(\mathbf{I}-\mathbf{B}')^{-1}.$$

The null matrix appears above because we have previously assumed that E($\mathbf{x}\boldsymbol{\zeta}'$) = $\mathbf{0}$ [in equation (0.2)], that is to say the x variables are not correlated with the errors in the equations. Putting all the pieces together,

$$\boldsymbol{\Sigma} = \left[\begin{array}{c|c} \boldsymbol{\Sigma}_{yy} & - \\ \hline \boldsymbol{\Sigma}_{xy} & \boldsymbol{\Sigma}_{xx} \end{array}\right] \quad (3.9)$$

$$= \left[\begin{array}{c|c} (\mathrm{I}-\mathrm{B})^{-1}[\Gamma\Phi\Gamma' + \Psi](\mathrm{I}-\mathrm{B}')^{-1} & - \\ \hline \Phi\Gamma'(\mathrm{I}-\mathrm{B}')^{-1} & \Phi \end{array}\right].$$

The structure above constitutes H_0 and H_A: $\boldsymbol{\Sigma} = \mathbf{S}$ is as before in Chapter 9.

Structural Equation Models with Latent Variables

It is possible to combine the latent variables models of with the structural equation models of this chapter. In other words, we can have path models between factors. While we have already shown we can always get by with just y-variables, here, if only for notational clarity, we will assume we have two sets of variables, an x set and a y set, and therefore we need two measurement models,

$$\mathbf{y} = \Lambda_y \boldsymbol{\eta} + \boldsymbol{\varepsilon} \quad (3.10)$$

$$\mathbf{x} = \Lambda_x \boldsymbol{\xi} + \boldsymbol{\delta} \; . \; (3.11)$$

The y-variables are a function of certain latent variables, the η's, while the x-variables are a function of other latent variables, the ξ's. The next step would be that we can have structural equation models amongst these latent variables as below:

$$\boldsymbol{\eta} = \mathbf{B}\boldsymbol{\eta} + \Gamma\boldsymbol{\xi} + \boldsymbol{\zeta} \; . \quad (3.12)$$

Needless to say, there are a set of assumptions that we must make before we can use these models. These are listed now

$$\text{Cov}\,(\boldsymbol{\eta}, \boldsymbol{\varepsilon}) = \mathbf{0} \quad (3.13)$$

$$\text{Cov}\,(\boldsymbol{\xi}, \boldsymbol{\delta}) = \mathbf{0} \quad (3.14)$$

$$\text{Cov}\,(\boldsymbol{\xi}, \boldsymbol{\zeta}) = \mathbf{0} \quad (3.15)$$

$$\text{Cov}\,(\boldsymbol{\varepsilon}, \boldsymbol{\delta}, \boldsymbol{\zeta}) = \mathbf{0} \quad (3.16)$$

$$\text{Diag}\,(\mathbf{B}) = \mathbf{0} \quad (3.17)$$

$$|\mathbf{I} - \mathbf{B}| \neq 0 \; . \quad (3.18)$$

The first two assumptions in equations (3.13) and (3.14) are that the common factors and the unique factors are independent. In the structural equation model, the independent variable and the error must be uncorrelated [assumption (3.15)]. Each of the three types of errors are mutually uncorrelated [assumption (3.16)]. The diagonal of the **B** matrix is a set of p zeroes, and the expression $(\mathbf{I} - \mathbf{B})$ must be nonsingular, meaning that its determinant cannot be zero so that it can be inverted.

To review, we have now introduced four parameter matrices: Λ_y which contains factor loadings for y variables, Λ_x which contains loadings for the regression of x variables on their factors, the ξ's, Γ containing regression coefficients for $\boldsymbol{\eta}$ on $\boldsymbol{\xi}$, and **B** with the regression coefficients for η's on other η's. To round out the picture, we have four variance matrices. The variance of all inputs must be specified, and that includes

$$V(\boldsymbol{\xi}) = \boldsymbol{\Phi}, \quad (0.19)$$

$$V(\boldsymbol{\zeta}) = \boldsymbol{\Psi}, \quad (0.20)$$

$$V(\boldsymbol{\varepsilon}) = \boldsymbol{\Theta}_\varepsilon \text{ and } (0.21)$$

$$V(\boldsymbol{\delta}) = \boldsymbol{\Theta}_\delta \; . \quad (0.22)$$

Our first illustration involves a longitudinal study in which a group of customers is asked the same two items on four different purchase occasions. These two items are hypothesized to be unidimensional. Here, we have to admit that this is just an illustrative example since any two items are unidimensional! You need more than two items to create a scale, otherwise you are

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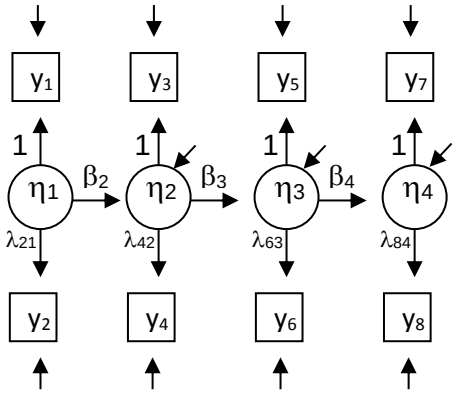
just modeling a plain household variety correlation. But, proceeding anyway, here is the path diagram:



Perhaps we are interested in the persistence of the attitude towards the brand over time. All of the variables have been labeled in such a way as to illustrate an all-y model. The measurement model is

$$\begin{bmatrix} y_1 \\ y_2 \\ y_3 \\ y_4 \\ y_5 \\ y_6 \\ y_7 \\ y_8 \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 & 0 \\ \lambda_{21} & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & \lambda_{42} & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & \lambda_{63} & 0 \\ 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & \lambda_{84} \end{bmatrix} \begin{bmatrix} \eta_1 \\ \eta_2 \\ \eta_3 \\ \eta_4 \end{bmatrix} + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \varepsilon_3 \\ \varepsilon_4 \\ \varepsilon_5 \\ \varepsilon_6 \\ \varepsilon_7 \\ \varepsilon_8 \end{bmatrix}$$

with the structural model

$$\begin{bmatrix} \eta_1 \\ \eta_2 \\ \eta_3 \\ \eta_4 \end{bmatrix} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ \beta_{21} & 0 & 0 & 0 \\ 0 & \beta_{32} & 0 & 0 \\ 0 & 0 & \beta_{43} & 0 \end{bmatrix} \begin{bmatrix} \eta_1 \\ \eta_2 \\ \eta_3 \\ \eta_4 \end{bmatrix} + \begin{bmatrix} \zeta_1 \\ \zeta_2 \\ \zeta_3 \\ \zeta_4 \end{bmatrix}.$$

To this we add the variance matrices of **ε** and **ζ**, respectively,

$$\boldsymbol{\Theta}_\varepsilon = \begin{bmatrix} \theta_{11} & 0 & \cdots & 0 \\ 0 & \theta_{22} & \cdots & 0 \\ \cdots & \cdots & \cdots & \cdots \\ 0 & 0 & \cdots & \theta_{88} \end{bmatrix} \text{ and}$$

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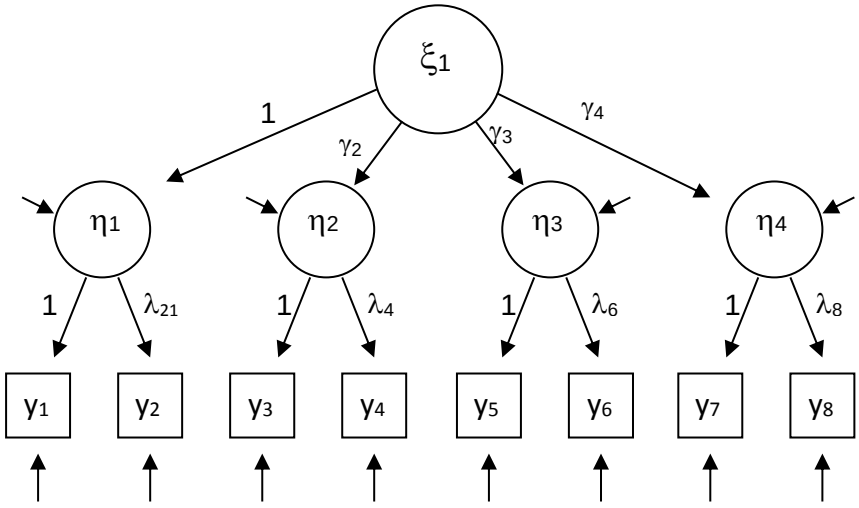
$$\mathbf{\Psi} = \begin{bmatrix} \psi_{11} & 0 & 0 & 0 \\ 0 & \psi_{22} & 0 & 0 \\ 0 & 0 & \psi_{33} & 0 \\ 0 & 0 & 0 & \psi_{44} \end{bmatrix}.$$

In the $\mathbf{\Psi}$ matrix, the parameter ψ_{11} is exogenous.

It is important to be able to calculate the degrees of freedom for this or any other model you are working on. The raw data for the model, given that there are eight observed variables, is given by the expression 9(8)/2 = 36. From this we must subtract the four free elements in the loading matrix, three β's, eight elements in $\mathbf{\Theta_\varepsilon}$ and then four elements on the diagonal of $\mathbf{\Psi}$. This leads to 15 degrees of freedom.

Second Order Factor Analysis

In effect, the factors themselves may form a higher order factor. In other words, if the correlations amongst the factors have the right structure, these may be the result of a latent variable. A path diagram of this model appears below:



Note that the η's have their own loadings and their own unique factors. Here, the variable ξ_1 serves as the higher order factor. In general terms, the second order factor analysis model can be written as

$$\mathbf{y} = \mathbf{\Lambda}_y \boldsymbol{\eta} + \boldsymbol{\varepsilon} \text{ and} \quad (0.23)$$

$$\boldsymbol{\eta} = \mathbf{\Gamma}\,\boldsymbol{\xi} + \boldsymbol{\zeta}, \quad . \quad (0.24)$$

which the reader will recognize as a special case of a SEM with latent variables. We can write the model more compactly as

$$\mathbf{y} = \mathbf{\Lambda}_y\left[\mathbf{\Gamma}\boldsymbol{\xi} + \boldsymbol{\zeta}\right] + \boldsymbol{\varepsilon} \; . \quad (0.25)$$

We need to assume that Cov($\boldsymbol{\varepsilon}$, $\boldsymbol{\zeta}$) = **0** and Cov($\boldsymbol{\xi}$, $\boldsymbol{\zeta}$) = **0**. Here we also have V($\boldsymbol{\varepsilon}$) = $\boldsymbol{\Theta}_{\varepsilon}$, V($\boldsymbol{\zeta}$) = $\boldsymbol{\Psi}$ and V($\boldsymbol{\xi}$) = $\boldsymbol{\Phi}$. The variance matrix of y, $\boldsymbol{\Sigma}$, takes on a particularly aesthetic form with this model,

$$V(\mathbf{y}) = \boldsymbol{\Lambda}_y\left[\boldsymbol{\Gamma}\boldsymbol{\Phi}\boldsymbol{\Gamma}' + \boldsymbol{\Psi}\right]\boldsymbol{\Lambda}_y' + \boldsymbol{\Theta}_{\varepsilon} \quad , \quad (0.26)$$

with the internal part in the brackets being the V($\boldsymbol{\eta}$). Again, students should make certain they can calculate the degrees of freedom for this model.

DATA PRESENTATION, ANALYSIS AND INTERPRETATION

Presentation of Data

Data obtained from the respondents are presented on the tables below:

Table 1a: Demographic characteristics of the Respondents

Variable	Category	Frequency	Percent	Cumulative Percent
Gender	Male	4	2.22	2.22
	Female	175	97.22	99.44
	Unidentified	1	0.56	100
	Total	180	100.00	
Status	Married	171	95	95
	Single	0	0	95
	Divorced	3	1.67	96.67
	Widowed	6	3.33	100
	Total	180	100.00	
Education	Primary	3	1.67	1.67
	Secondary	12	6.67	8.34
	Tertiary	165	91.66	100
	Total	180	100.00	
Years of Married	< 10	13	7.22	7.22
	11 – 20	135	75.00	82.22
	21 – 30	21	11.67	93.89
	31 – 40	11	6.11	100
	41 – over	0	0.00	100
	Total	180	100.00	
Age	< 20	4	2.22	2.22
	21 – 30	10	5.56	7.78
	31 – 40	72	40.00	47.78
	41 – 0ver	94	52.22	100
	Total	180	100.00	

Source: Field Survey, 2019.

Table 1a above presented the demographic characteristics of the respondents as obtained from the field survey.

In terms of the gender characteristics, 175 of the respondents, representing 97.22 per cent were females while 4 of them, representing 2.22 per cent were males. One of the respondents, representing 0.56 per cent did not indicate the gender class. The overriding number of females in this study was due to the fact that the study concern itself more on female characteristics – embryo. A few male respondents were sampled because of the need to observe variations in the responses.

Table 1b: Grade Point and the Range of Inference on Linkert Scale

Variable	SA	A	DA	SD
Grade-Point	4	3	2	1
Range	3.5 – 4.5	2.5 – 3.4	1.5 – 2.4	0.5 – 1.5

Source: Own Computation based on Cronbach Scale
Key: SA = Strongly Agree; A = Agree; DA = Disagree; SD = Strongly Disagree.

Based on this method, survey statement is either agreed or disagreed with

cut-off point of 2.5 derived as follows:

$$\frac{4+3+2+1}{4} = \frac{10}{4} = 2.5$$

As a result, responses ranging from 2.5 and overs are tagged “Agreed” While those less than 2.5 are tagged “disagreed”. Based on this reasoning, 30 out of the 50-point questions representing 60 percent agreed with the object of the study while 20 out of 50 respondents disagreed with the propositions of this study. On the basis of this, issues relating to parental life style affects the growth of the embryo.

RESULTS

Research question 1: Do environmental factors in the life of parents affect the growth of an embryo?

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Table 1: Presents the mean rating of environmental factors as they affect the growth of the embryo.

	QUESTIONS RELATING TO ENVIRONMENTAL FACTORS								
S/N	**Item**	**Measures**	**SA**	**A**	**D**	**SD**	**Total**	**Mean**	**Decision**
1	Too busy to take full parenting responsibility	F X Fx	96 4 384	48 3 144	16 2 32	20 1 20	180	580/180 =3.22	Agree
2	Financial challenges compound issues of good parenting	F X Fx	88 4 352	44 3 132	40 2 80	8 1 8	180	572/180 = 3.18	Agree
3	Long standing health conditions affect embryo development	F X Fx	96 4 384	50 3 150	18 2 36	16 1 16	180	586/180 =,3.26	Agree
4	Neighborhood inconveniences affect the development of the embryo	F X Fx	108 4 432	31 3 93	29 2 58	12 1 12	180	595/180 = 3.31	Agree
5	Non-conducive environment affects the development of the embryo	F X Fx	105 4 420	62 3 186	8 2 16	5 1 5	180	627/180 = 3.48	Agree
6	Attitudes from neighbors affect the mother carrying the embryo	F X Fx	104 4 416	61 3 183	10 2 20	5 1 5	180	624/180 = 3.47	Agree
7	Lack of personal help affect the mother with pregnancy	F X Fx	110 4 440	41 3 123	19 2 38	10 1 10	180	611/180 = 3.39	Agree
8	Lack of rest or leisure affects the embryo	F X Fx	97 4 388	64 3 192	11 2 22	8 1 8	180	610/180 = 3.39	Agree
9	Discrimination with respect to un wanted pregnancy can distort hormones which in turn affect the embryo	F X Fx	99 4 396	41 3 123	30 3 90	10 1 10	180	589/180 = 3.27	Agree
10	Changes in environmental conditions affect the development of the embryo	F X Fx	95 4 380	64 3 192	12 2 24	9 1 9	180	605/180 = 3.36	Agree

Source: Own Computation based on Responses.

Respondents agreed that all the environmental factors as listed in the section B of the questionnaire affects the growth of the embryo. This is because the mean responses were above the cut-off point of 2.5. Consequently, it could be inferred that environmental factors greatly affect the growth of the embryo.

Research Question 2: Does parental stress affect the growth of an embryo?

Table 2: presents the mean rating of parental stress as they affect the growth of the embryo

QUESTIONS RELATING TO PARENTAL STRESS								
Item	**Measures**	**SA**	**A**	**D**	**SD**	**Total**	**Mean**	**Decision**
Anxiety or un necessary happiness can affect the embryo in my role as a parent	F X Fx	101 4 404	51 3 153	18 2 36	10 1 10	180	603/180 =3.35	Agree
Over pampering can affect the deployment of the embryo	F X Fx	82 4 328	63 3 189	21 2 42	14 1 14	180	513/180 = 3.18	Agree
Worrying over meeting family works affect the growth of the embryo	F X Fx	102 4 408	60 3 180	11 2 22	7 1 7	180	617/180 = 3.43	Agree
Hard labor affects the deployment of the embryo	F X Fx	99 4 396	58 3 174	14 2 28	9 1 9	180	607/180 = 3.37	Agree
Spending a lot of times carrying out family duties can affect the growth of the embryo	F X Fx	104 4 416	61 3 183	5 2 10	10 1 10	180	619/180 = 3.44	Agree
Depression can affect the growth of the embryo	F X Fx	103 4 412	49 3 147	19 2 38	9 1 9	180	606/180 = 3.37	Agree
Peer pressure can affect the growth of the embryo	F X Fx	51 4 204	102 3 306	16 2 32	11 1 11	180	553/180 = 3.07	Agree
Pressure from other children can affect the growth of the embryo	F X Fx	13 4 52	11 3 33	86 2 172	70 1 70	180	327/180 = 1.82	Disagree
Exposure to pollution during pregnancy affect the development of the embryo	F X Fx	16 4 64	21 3 63	91 2 182	52 1 52	180	361/180 = 2.01	Disagree
Inadequate social life can affect the growth of the embryo life.	F X Fx	60 4 240	97 3 291	14 2 28	9 1 9	180	568/180 = 3.16	Agree

Source: Own Computation

A total of 8 out of 10 responses to the above questions, representing 80 percent were of the opinion that parental stress greatly affects the growth of the embryo while only 1, representing 10 percent disagreed. Stress greatly affect the growth of the embryo because it distorts the arrangement of the embryo which consequently causes hormonal imbalance which affect the embryo. This is why the mean marks exceeded the cut off mark of 2.5 as stipulated.

Research question 3: Does parental life style affects the growth of embryo?

Table 3: Presents the mean rating of the respondents as they relate to parental life styles as they affect the growth of the embryo

QUESTIONS RELATING TO PARENTAL LIFE STYLES								
Item	**Measure**	**SA**	**A**	**D**	**SD**	**Total**	**Mean**	**Decision**
Smoking (or secondary smoking) during pregnancy can affect embryo development	F X fx	72 4 288	71 3 213	17 2 34	20 1 20	180	555/180 = 3.08	Agree
Frequent intake of alcohol during pregnancy can affect embryo development	F X fx	72 4 288	70 3 210	21 2 42	17 1 17	180	557/180 = 3.09	Agree
Abuse or misuse of drugs during pregnancy can affect embryo development	F X fx	101 4 404	63 3 189	9 2 18	7 1 7	180	618/180 = 3.43	Agree
Lack of proper communication with the embryo affects its growth	F X fx	11 4 44	10 3 30	60 2 120	99 1 99	180	239/180 = 1.63	Disagree
Excessive intake of coffee during pregnancy can affect the growth of embryo	F X Fx	21 4 84	15 3 45	96 2 192	48 1 48	180	369/180 = 2.05	Disagree
Lack of involvement in the affairs of children affects the growth of the embryo	F X fx	11 4 44	19 3 57	101 2 202	49 1 49	180	352/180 = 1.96	Disagree
Low level of vitamin D during pregnancy can affect the growth of the embryo	F X fx	97 4 388	60 3 180	13 2 26	10 1 10	180	606/180 = 3.36	Agree
Being autocratic or authoritarian can affect the growth of the embryo	F X fx	41 4 164	86 3 258	24 2 48	29 1 29	180	499/180 = 2.77	Agree
Difficulty in balancing different responsibilities affects the growth of the embryo	F X fx	42 4 168	91 3 273	31 2 62	16 1 16	180	519/180 =2.88	Agree
Exposure to all kind of stress can affect the development of the embryo	F X fx	109 4 150	50 3 150	11 2 22	10 1 10	180	618/180 = 3.43	Agree

Source: Own Computation from field survey

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There are a 70-30 reactions to the responses of the respondents to research question 3. While 7 of them, representing 70 percent agreed that parental life style affects the growth of the embryo, the other 3 of the respondents disagreed that life style of the parents does not affect the growth of the embryo

Research Question 4: How do variations in parental responsibilities affect the growth of an embryo?

Table 4: Presents the responses in reaction to how variations in parental responsibilities affects the growth of the embryo

QUESTIONS RELATING TO PARENTAL RESPONSIBILITIES								
Item	**Measure**	**SA**	**A**	**D**	**SD**	**Total**	**Mean**	**Decision**
Circumstances of parental responsibility affect the growth of the embryo	F X Fx	14 4 56	70 3 210	71 2 142	25 1 25	180	433/180 =2.41	Disagree
Over bearing affects parental responsibility	F X Fx	12 4 48	72 3 216	70 2 140	26 1 26	180	430/180 = 2.39	Disagree
Family position affects parental responsibility	F X Fx	34 4 136	12 3 36	92 2 184	42 1 42	180	398/180 = 2.21	Disagree
Administration of care affects parental responsibility	F X Fx	21 4 84	65 3 195	66 2 132	28 1 28	180	439/180 = 2.44	Disagree
Restrictions on freedom affects parental responsibility	F X Fx	22 4 88	40 3 120	98 2 196	41 1 41	180	424/180 = 2.36	Disagree
Only the married are vested with parental responsibility	F X Fx	21 4 84	19 3 57	99 2 198	41 1 41	180	380/180 = 2.11	Disagree
Parental responsibility can be affected by divorce	F X Fx	87 4 348	42 3 126	30 2 60	21 1 21	180	555/180 = 3.08	Agree
Other relations can affect or influence parental responsibility	F X Fx	43 4 172	88 3 176	30 2 60	19 1 19	180	427/180 = 2.37	Disagree
Un involved or excessive involvement can affect parental responsibility	F X Fx	10 4 40	88 3 176	80 2 160	2 1 2	180	378/180 = 2.10	Disagree
Limiting roles can affect parental responsibility	F X Fx	20 4 80	88 3 176	61 2 122	11 1 11	180	389/180 = 2.16	Disagree

Source: Own Computation from field survey

9 out of the 10 respondents, representing 90 percent disagreed with all the questions relating to parental responsibilities as they affect the growth of the embryo while only 1 of them, representing 10 percent agreed with the fact that parental responsibilities can affect the growth of the embryo. As a matter of fact, the responses agreed that divorce can affect parental responsibilities due to issues relating to "divided home" and "divided attention". This consequently led to hormonal imbalances which can distort the growth of the embryo.

Research Question 5: Do increased allocation of nutrients affect the growth of an embryo?

Table 5: Presents the responses in relation to how nutrition can affect the growth of an embryo.

	QUESTIONS RELATING TO NUTRITION								
S/N	**Item**	**Measure**	**SA**	**A**	**D**	**SD**	**Total**	**Mean**	**Decision**
41	Total parental nutrition can affect the growth of the embryo	F X Fx	59 4 236	80 3 240	26 2 52	15 1 15	180	546/180 = 3.06	Agree
42	Embryos do not need adequate nutrition	F x Fx	28 4 112	20 3 60	51 2 102	81 1 81	180	355/180 = 1.97	Disagree
43	Embryos receives total parental nutrition through the umbilical cord	F X Fx	101 4 404	51 3 153	17 2 34	11 1 11	180	602/180 = 3.34	Agree
44	No difference between eternal feeding and total parental nutrition	F X Fx	21 4 84	27 3 81	91 2 182	41 1 41	180	388/180 =2.16	Disagree
45	There are risks associated with total parental nutrition	F X Fx	19 4 76	53 3 159	87 2 174	21 1 21	180	430/180 = 2.39	Disagree
46	Total parental nutrition makes the embryo feed well	F X Fx	102 4 408	51 3 153	18 2 36	9 1 9	180	606/180 = 3.37	Agree
47	A child can be on total parental nutrition as long as it remains an embryo	F X Fx	101 4 404	50 3 150	19 2 38	10 1 10	180	602/180 =3.34	Agree
48	Total parental nutrition don't work for the embryo	F X Fx	10 4 40	19 3 57	51 2 102	100 1 100	180	299/180 = 1.66	Disagree
49	Issues involving intestinal transplant can affect the growth of the embryo	F X Fx	99 4 396	43 3 129	18 2 36	20 1 20	180	581/180 = 3.23	Agree
50	Embryos do not really need total parental nutrition	F X Fx	5 4 20	12 3 36	62 2 124	101 1 101	180	281/180 = 1.56	Disagree

Source: Own Computation from field survey

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There are a 50-50 reactions to how issues of increased nutrition can affect the growth of the embryo. While 5 of the respondents, representing 50 percent were in support of the issues raised, 5 of them, representing 50 per cent disagreed. The inconclusiveness of this finding can be subjected to test of hypothesis in order to determine the direction of inferences to be drawn from the survey.

Test of Hypotheses

Hypothesis one: Environmental factors in the life of parents have no significant effect on the growth of the embryo.

Model 1: Impact of environmental factors on embryo growth.

$$EMBRYO = \beta_0 + \beta_1 (ENVIRONMENTAL\ FACTORS) + \mu_{IJ}$$

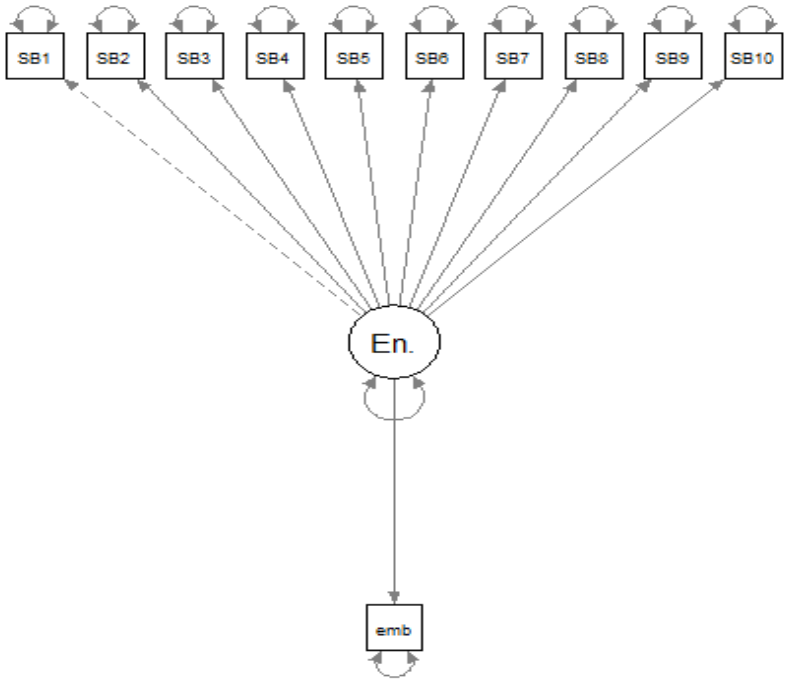


Figure 4.1: SEM for the Impact of Environmental Factors on Embryo Growth

> summary (tofit.model_m1, fit.measures = TRUE)
lavaan 0.6-5 ended normally after 49 iterations

Estimator	ML
Optimization method	NLMINB
Number of free parameters	22
Loglikelihood and Information Criteria:	
Loglikelihood user model (H0)	-3178.835
Loglikelihood unrestricted model (H1)	-3113.628
Akaike (AIC)	6401.670

Bayesian (BIC)	6472.158
Sample-size adjusted Bayesian (BIC)	6402.481
Root Mean Square Error of Approximation:	
RMSEA	0.104
90 Percent confidence interval - lower	0.084
90 Percent confidence interval - upper	0.125
P-value RMSEA <= 0.05	0.000
Standardized Root Mean Square Residual:	
SRMR	0.074
Parameter Estimates:	
Information	Expected
Information saturated (h1) model	Structured
Standard errors	Standard

Latent Variables:

	Estimate	Std.Err	z-value	P(>|z|)
Environmental.factors =~				
SB1	1.000			
SB2	0.723	0.188	3.841	0.000
SB3	0.999	0.201	4.958	0.000
SB4	1.097	0.215	5.114	0.000
SB5	1.404	0.258	5.449	0.000
SB6	1.143	0.232	4.931	0.000
SB7	1.137	0.218	5.210	0.000
SB8	1.062	0.207	5.122	0.000
SB9	1.089	0.216	5.044	0.000
SB10	0.667	0.174	3.839	0.000

Regressions:

	Estimate	Std.Err	z-value	P(>|z|)
embryo ~				
Envrnmntl.fctr	12.891	9.101	1.416	0.015

Discussion

The model result reveals the influence of environmental factors on Embryo Growth (EG). A critical inspection of the result also shows that all the latent variables (SB1 – SB10) of environmental factors has a positive and significant impact on the Embryo Growth (EG), since their respective p-values is less than 0.05 (5% level) ($p<0.05$). Also, the Standardized Root Mean Square Residual value of 0.074 was observed to be relatively low, suggesting the adequacy of the model.

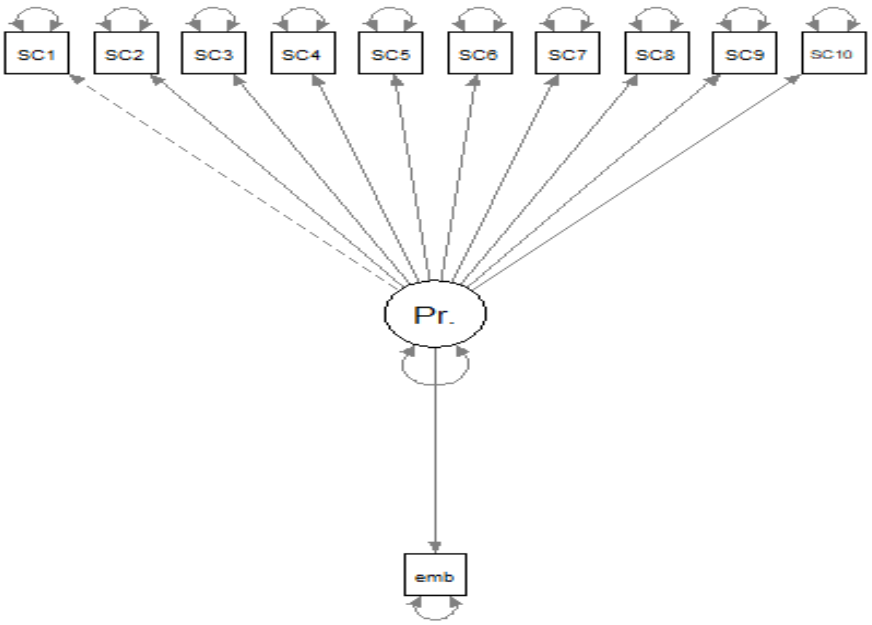
The overall impact of the environmental factor on embryo growth was seen from the beta coefficient of 12.891 with standard error of 9.101 (z-test = 1.416, $p<0.05$) which signifies a

significant impact of the environmental factor on the growth of embryo. Invariably this implies that a unit change in the environmental factors will bring a corresponding improvement on the embryo growth (EG). This means that the parameter is statistically significant in explaining the embryo growth (EG). Hence, the null hypothesis is rejected and alternative hypothesis is accepted which implies that there is significant effect of environmental factors on Embryo Growth (EG).

Hypothesis Two: Parental stress has no significant effect on the growth of the embryo.

Model 2: Impact of Excessive Parental Stress on the Growth of the Embryo.

$$EMBRYO = \beta_0 + \beta_1 (Excessive\ Parental\ Stress) + \mu_{IJ}$$



> summary (tofit.model_m2, fit.measures = TRUE)
lavaan 0.6-5 ended normally after 53 iterations

Estimator	ML
Optimization method	NLMINB
Number of free parameters	22
Loglikelihood and Information Criteria:	
Loglikelihood user model (H0)	-3061.288
Loglikelihood unrestricted model (H1)	-2954.991
Akaike (AIC)	6166.577
Bayesian (BIC)	6237.065
Sample-size adjusted Bayesian (BIC)	6167.388
Root Mean Square Error of Approximation:	
RMSEA	0.145
90 Percent confidence interval - lower	0.126

90 Percent confidence interval - upper	0.165
P-value RMSEA <= 0.05	0.000
Standardized Root Mean Square Residual:	
SRMR	0.103
Parameter Estimates:	
Information	Expected
Information saturated (h1) model	Structured
Standard errors	Standard

Latent Variables:

	Estimate	Std.Err	z-value	P(>|z|)
Parental.stress =~				
SC1	1.000			
SC2	1.229	0.281	4.367	0.000
SC3	1.526	0.315	4.842	0.000
SC4	1.151	0.264	4.363	0.000
SC5	1.621	0.325	4.986	0.000
SC6	0.796	0.213	3.730	0.000
SC7	1.655	0.330	5.008	0.000
SC8	1.514	0.306	4.949	0.000
SC9	0.858	0.222	3.866	0.000
SC10	1.233	0.283	4.358	0.000

Regressions:

	Estimate	Std.Err	z-value	P(>|z|)
embryo ~				
Parental.strss	1.244	11.585	0.107	0.091

Discussion

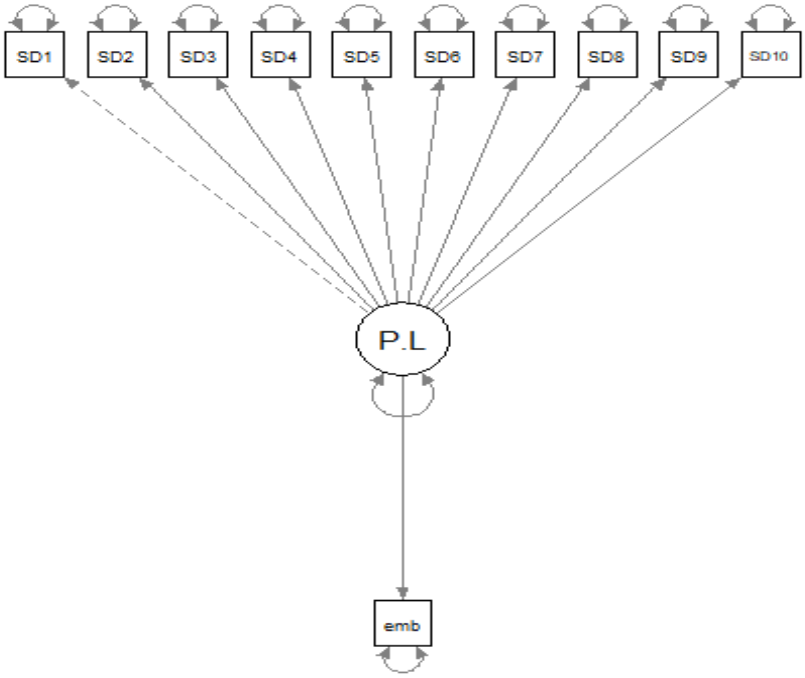
The model result reveals the influence of parental stress on Embryo Growth (EG). A critical inspection of the result also shows that all the latent variables (SC1 – SC10) of parental stress has a positive and significant impact on the Embryo Growth (EG), since their respective p-values is less than 0.05 (5% level) ($p<0.05$). Also, the Standardized Root Mean Square Residual value of 0.103 was observed to be relatively low, suggesting the adequacy of the model.

The overall impact of the parental stress on embryo growth was seen from the beta coefficient of 1.224 with standard error of 11.585 (z-test = 0.107, $p<0.1$) which signifies a significant impact of the parental stress on the growth of embryo at 10% level of significance. Invariably this implies that a unit change in the parental stress will bring a corresponding improvement on the embryo growth (EG). This means that the parameter is statistically significant in explaining the embryo growth (EG). Hence, the null hypothesis is rejected and alternative hypothesis is accepted which implies that there is significant effect of parental stress on Embryo Growth (EG).

Hypothesis Three: Parental Life Style have no significant effect on the growth of an embryo.

Model 3: Impact of Parental life style on the growth of embryo.

$EMBRYO = \beta_0 + \beta_1 (Parental\ life\ style) + \mu_{IJ}$



> summary (tofit.model_m3, fit.measures = TRUE)

lavaan 0.6-5 ended normally after 46 iterations

Estimator	ML
Optimization method	NLMINB
Number of free parameters	22
Loglikelihood and Information Criteria:	
Loglikelihood user model (H0)	-2911.867
Loglikelihood unrestricted model (H1)	-2728.342
Akaike (AIC)	5867.734
Bayesian (BIC)	5938.222
Sample-size adjusted Bayesian (BIC)	5868.545
Root Mean Square Error of Approximation:	
RMSEA	0.201
90 Percent confidence interval - lower	0.182
90 Percent confidence interval - upper	0.220
P-value RMSEA <= 0.05	0.000

Standardized Root Mean Square Residual:
SRMR 0.175
Parameter Estimates:
Information Expected
Information saturated (h1) model Structured
Standard errors Standard
Latent Variables:

	Estimate	Std.Err	z-value	P(>|z|)
Parental.LifeStyle =~				
SD1	1.000			
SD2	0.964	0.114	8.423	0.000
SD3	0.907	0.096	9.411	0.000
SD4	0.073	0.146	0.499	0.618
SD5	0.790	0.129	6.113	0.000
SD6	0.151	0.140	1.081	0.280
SD7	0.623	0.126	4.943	0.000
SD8	0.028	0.157	0.181	0.856
SD9	0.277	0.144	1.926	0.054
SD10	0.657	0.110	5.994	0.000

Regressions:

	Estimate	Std.Err	z-value	P(>|z|)
embryo ~				
Parentl.LfStyl	11.955	8.739	1.368	0.017

Discussion

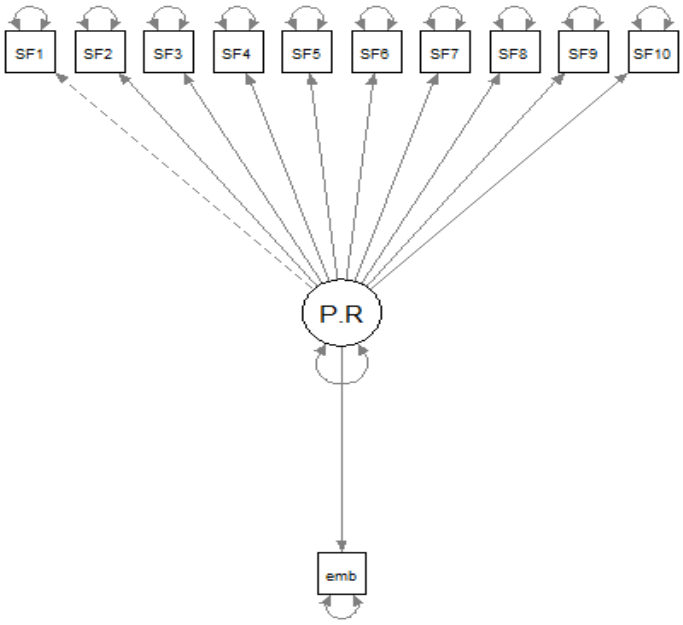
The model result reveals the influence of parental lifestyle on Embryo Growth (EG). A critical inspection of the result also shows that all the latent variables (SD1 – SD10) of parental lifestyle has a positive and significant impact on the Embryo Growth (EG), since their respective p-values is less than 0.05 (5% level) ($p<0.05$). Also, the Standardized Root Mean Square Residual value of 0.175 was observed to be relatively low, suggesting the adequacy of the model.

The overall impact of the parental lifestyle on embryo growth was seen from the beta coefficient of 11.955 with standard error of 8.739 (z-test = 1.368, $p<0.05$) which signifies a significant impact of the parental lifestyle on the growth of embryo at 5% level of significance. Invariably this implies that a unit change in the parental lifestyle will bring a corresponding improvement on the embryo growth (EG). This means that the parameter is statistically significant in explaining the embryo growth (EG). Hence, the null hypothesis is rejected and alternative hypothesis is accepted which implies that there is significant effect of parental lifestyle on Embryo Growth (EG).

Hypothesis Four: Variations in parental responsibilities have no significant effect on the growth of an embryo.

Model 4: Impact of Parental responsibilities on the growth of the embryo.

$EMBRYO = \beta_0 + \beta_1$ (*Parental responsibilities*) $+ \mu_{IJ}$



> summary (tofit.model_m4, fit.measures = TRUE)
lavaan 0.6-5 ended normally after 51 iterations

Estimator	ML
Optimization method	NLMINB
Number of free parameters	22
Loglikelihood and Information Criteria:	
Loglikelihood user model (H0)	-3067.255
Loglikelihood unrestricted model (H1)	-2973.227
Akaike (AIC)	6178.510
Bayesian (BIC)	6248.998
Sample-size adjusted Bayesian (BIC)	6179.321
Root Mean Square Error of Approximation:	
RMSEA	0.134
90 Percent confidence interval - lower	0.115
90 Percent confidence interval - upper	0.154
P-value RMSEA <= 0.05	0.000

Standardized Root Mean Square Residual:

SRMR	0.107
Parameter Estimates:	
Information	Expected
Information saturated (h1) model	Structured
Standard errors	Standard

Latent Variables:

	Estimate	Std.Err	z-value	P(>|z|)
Parental.Variations =~				
SE1	1.000			
SE2	0.749	0.192	3.897	0.000
SE3	0.885	0.209	4.233	0.000
SE4	0.963	0.200	4.821	0.000
SE5	0.861	0.184	4.679	0.000
SE6	0.452	0.193	2.337	0.019
SE7	1.142	0.223	5.111	0.000
SE8	1.154	0.205	5.640	0.000
SE9	1.372	0.238	5.766	0.000
SE10	1.360	0.239	5.692	0.000

Regressions:

	Estimate	Std.Err	z-value	P(>|z|)
embryo ~				
Parental.Vrtns	11.929	10.500	1.136	0.025

Discussion

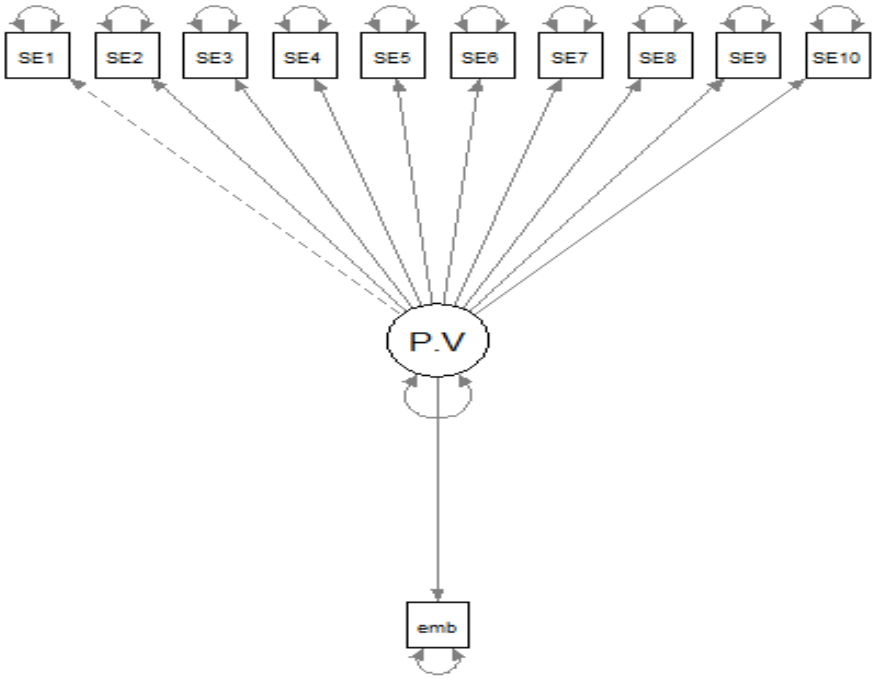
The model result reveals the influence of parental responsibilities on Embryo Growth (EG). A critical inspection of the result also shows that all the latent variables (SE1 – SE10) of parental responsibilities has a positive and significant impact on the Embryo Growth (EG), since their respective p-values is less than 0.05 (5% level) ($p<0.05$). Also, the Standardized Root Mean Square Residual value of 0.107 was observed to be relatively low, suggesting the adequacy of the model.

The overall impact of the parental responsibilities on embryo growth was seen from the beta coefficient of 11.929 with standard error of 10.500 (z-test = 1.136, $p<0.05$) which signifies a significant impact of the parental responsibilities on the growth of embryo at 5% level of significance. Invariably this implies that a unit change in the parental responsibilities will bring a corresponding change in the embryo growth (EG). This means that the parameter is statistically significant in explaining the embryo growth (EG). Hence, the null hypothesis is rejected and alternative hypothesis is accepted which implies that there is significant effect of parental responsibilities on Embryo Growth (EG).

Hypothesis Five: Increased nutrition do not affect the growth of an embryo.

Model 5: Impact of Nutrition on embryo growth.

$EMBRYO = \beta_0 + \beta_1 (Parental\ nutrition) + \mu_{IJ}$



> summary(tofit.model_m5, fit.measures = TRUE)

lavaan 0.6-5 ended normally after 79 iterations

Estimator	ML
Optimization method	NLMINB
Number of free parameters	22
Loglikelihood and Information Criteria:	
Loglikelihood user model (H0)	-3169.382
Loglikelihood unrestricted model (H1)	-3036.590
Akaike (AIC)	6382.764
Bayesian (BIC)	6453.252
Sample-size adjusted Bayesian (BIC)	6383.575
Root Mean Square Error of Approximation:	
RMSEA	0.166
90 Percent confidence interval - lower	0.147
90 Percent confidence interval - upper	0.186
P-value RMSEA <= 0.05	0.000

Standardized Root Mean Square Residual:

SRMR	0.156
Parameter Estimates:	
Information	Expected
Information saturated (h1) model	Structured
Standard errors	Standard

Latent Variables:

	Estimate	Std.Err	z-value	P(>|z|)
Parental. Nutrition =~				
SF1	1.000			
SF2	-0.495	0.380	-1.304	0.192
SF3	2.216	0.898	2.468	0.014
SF4	0.176	0.366	0.482	0.630
SF5	0.639	0.419	1.525	0.127
SF6	2.494	1.001	2.490	0.013
SF7	2.145	0.884	2.426	0.015
SF8	-0.735	0.425	-1.730	0.084
SF9	2.129	0.890	2.393	0.017
SF10	-0.535	0.371	-1.442	0.149

Regressions:

	Estimate	Std.Err	z-value	P(>|z|)
embryo ~				
Prntl.Nutrition	-25.663	23.163	-1.108	0.268

Discussion

The model result reveals the impact of increase in nutrition on Embryo Growth (EG). A critical inspection of the result also shows that of all the latent variables (SF1 – SF10) of "increase in nutrition", only SF2, SF5, SF6 & SF9 that have a significant impact on the Embryo Growth (EG), since their respective p-values is less than 0.05 (5% level) ($p<0.05$), leaving all other latent variables not significant in influencing the embryo growth. Also, the Standardized Root Mean Square Residual value of 0.156 was observed to be relatively high as it not tending to zero as such, suggesting the a relatively low performance of the predictors in the model.

The overall impact of the "increase in nutrition" on embryo growth was seen from the beta coefficient of -25.663 with standard error of 23.163 (z-test = -1.108, $p>0.05$) which signifies a no significant impact of the "increase in nutrition" on the growth of embryo at 5% level of significance. Invariably this implies that a unit change in the "increase in nutrition" will not bring a corresponding change in the embryo growth (EG). This means that the parameter is not statistically significant in explaining the embryo growth (EG). Hence, the null hypothesis is upheld, which implies that there is no significant effect of "increase in nutrition" on Embryo Growth (EG).

SUMMARY OF FINDINGS

This study was able to established the fact that the growth of an embryo is largely influenced by the parental life style. This is manifested in the responses from the respondents. Out of the 50 questions in the questionnaire, 30 of the them agreed that the object of the study can greatly affect the growth of the embryo while only 20 of them disagreed. This was premised on the Cronbach basis of assessment in which the average Linkert as a bench mark for agreeing or disagreeing was calculated to be 2.5. As a result, the mean responses of above 2.5 are considered to agree while those below 2.5 are considered to disagree. On the basis of the findings, the study rejected the null hypothesis on environmental factors, parental stress, parental lifestyle and parental responsibilities with the reasoning that all items of the questionnaire greatly affect the growth of the embryo. While, accepting the null hypothesis the "increase in nutrition" as a necessary factor for the growth of the embryo.

DISCUSSION OF FINDINGS

Although, little empirical studies abound on the relationship between parental life styles and the growth of the embryo, this study is in line with other studies such as Landry (2014) who found that parental role affect the growth of a child. In a related development, this study conforms with Ward (2007) who found that mother's stress harms fetus at its early stage of development. Also, the study conforms with Almoud (2013) who found that parental investment arising from the staged developmental nature reinforce the initial endowment differences in fetal origins.

SUMMARY

A modest attempt has been made in this study to assess the growth of an embryo in relation to parental life style. The study allowed for different dimensions of parental life style within the framework of environmental factors, parental stress, parental responsibilities and nutrition, among others.

Empirical evidence in this study found that about 97 percent of the respondents were females who are mostly above 40 years of age and whose marital experience (that is ages of marriage) fell within 11 – 20 years. Majority of these respondents had tertiary level of education, the bulk of whom (95 percent) were married.

In descriptive term, the respondents agreed that all the environmental factors that were listed in the questionnaire directly affect the growth of the embryo. Additionally, 90 percent of the respondents were of the opinion that parental stress greatly affect the growth of the embryo while it was about 50 percent agreed that parental life style affects the embryo growth. In terms of variations in parental responsibilities, 90 percent of the respondents disagreed with all the questions that related to parental responsibilities as they affect the growth of the embryo. The study also found a 50-50 percent reactions to nutrition as they contribute to the growth of the embryo.

On the whole, when the study was subjected to hypotheses testing, all the null hypotheses were rejected with the reasoning that all the variables for assessing parental life style and the parameters for evaluation were statistically significant in influencing the growth of the embryo.

CONCLUSION

How parents engage in different life style greatly affects the growth of the embryo. This position may generate inherent reactions by confluence of researchers who may be interested on working on this subject matter. However, the study invites a balanced approach of a theoretically informed and design- based analysis to empirically estimate parental life styles as they affect the growth of the embryo. While this study expects such areas of research to focus on the nature of the behavioral patterns of parents to have long lasting effects on embryo growth, it could be concluded that parental life styles affect embryo growth. Indeed, this study seemed to tilt towards all those variables that were listed in the questionnaire as greatly affecting the growth of embryo. Given the lens this study have provided on the parents life style – embryo relationship, those outside the "fetal origin" camp can follow developments set by this study.

A major implication of this study is that parental life styles greatly affects the growth of the embryo. As a consequence, a first-order implication relating to this study is for parents and health care providers to concentrate on most of the externalities that "destroys" the unborn child. This involves environmental factors, parental stress and responsibilities, nutrition and many others that may need to be addressed for the sake of the unborn generations.

RECOMMENDATIONS

Based on the findings of this study, the following recommendations are made:

1. There is the need to address all environmental factors that affect the growth of the embryo. These include addressing issues relating to finances, health conditions, neighborhood inconveniences and changes in environmental conditions among others.

2. Issues relating to parental stress as well as parental life styles needed to be greatly addressed. Stress factors such as depression, family responsibilities, anxieties as well as inadequate social life among others should be addressed to enhance the growth of the embryo.

3. Related to parental stress, life styles such as smoking, alcohol intake and inadequate vitamin D among others should be addressed through enlightenment during antenatal period so that prospective mothers could live healthy.

4. Issues of parental responsibilities such as over bearing, family positions, other relations' influence should be addressed. This may entail allowing couples to live harmoniously and see themselves as partners in family up bring with little or no "external" influence from extended families.

5. Finally, adequate nutrition is key in the growth of an embryo. This entails intake of balanced diets during pregnancy to allow for healthy living of the unborn child.

REFERENCES

Almond, D. & Currie, J. (2011), Human Capital Development before ages Five. *Handbook of Labour Economics,* Vol. 46.

Almond, D. & Mozunder, B, (2012), Fetal Origin and Parental Responses. *Federal Reserve Bank of Chicago.* Revised 2013 WP 2012- -14. Pp. 1 – 32.

Altitude to Baby During Pregnancy may have Implications for Child's Development. *Journal of Development Review.* 61 – 73.

American Heritage Dictionary of English Language (2016), Dictionary. Houghton Mifflin Harcourt Publishing Company.

American Heritage Stedman's Medical Dictionary of English Language (2002), Dictionary. Houghton Mifflin Harcourt Publishing Company.

American Heritage Stedman's Medical Dictionary of English Language (2001), Dictionary. Houghton Mifflin Harcourt Publishing Company.

American Heritage Stedman's Medical Dictionary of English Language (1998), Dictionary. Houghton Mifflin Harcourt Publishing Company.

Baumrind, D. (1991), Effective Parenting During Early Adolescent Transition. *In*

Baumrind, D. (1996), The Discipline Controversy Revisited. *Fam. Relat.* 45, 405 – 414.

Baumrind, D., Larzelere, R.E. & Owens, E.B, (2010), Effects of Preschool

Behaviour in African American Families of Preschool Children. *Journal of Clinical Child Psychology*. 31: 272 – 277.

Behaviour: A 12-year Prospective Community Study. *Journal of Clinical Child Adolescent Psychology.* 40, 468 – 478.

Bercker, G. & Tomes, N. (1976), Child Endowments and the Quantity and Quality of Children. *Journal of Political Economy,* 84(4): S143 – S162

between Parental Authoritative Parenting and Early Adolescent Subsistence use. Journal of Early Adolescent 465 – 487.

Blechsmidt, E. (1977), The Beginning of Human Life. Springer-verlag, P. 32.

Bryk, A. & Raudanbush, S.W. (1992), Hierachical Linear Models: *Newbury Park, California, sage*.

Bryk, A. & Raudanbush, S.W. (2002), Hierachical Linear Models: *Applications and Data Analysis Methods.* 2nd ed. Thousand Oaks, Calif sage publications.

Conservation or Divergence. *Trends in Ecology & Evolution.* 27.(7): 385-393.

Datar, A., Kilbum, M.R., Loughran, D.S. (2010), Endowments and parental Investments in Infancy and Early Childhood, *Demography.* 47(1):145 – 162

Developing Seeds: Bad Offspring or Bad Marternal Genotypes. Evolutionary Ecology. 13(3): 305 – 317.

Dickinson, E. (1972), Complete Poems. New York: Little Brown.

Dickson, L.M. & AninbanBasu, B. (2005), Multilevel modeling and practice-based Research. *Family Medicine.* May 3 (Suppl 1): s52 – s60.

Donner, A. & Klar, N. (2000), Design and Analysis of cluster Randomization Trials in Health Research. London, England: Oxford University Press.

Eckert, J.J. & Fleming, T.P. (2011), the effect of nutrition and environment on the preimplantation embryo. *The Obstetrician & Gynaecology,* 13: 43 – 48.

Ehrlich, P. Holm, R. Parnell, D. (1963), The Process of Evolution: New York. McGraw Hill. Pp. 66.
Engl, N. (2014), Effect of in Utero and Early-Life Conditions on Adult Health and Disease. HHS Public Access. Pp. 61 – 73.
Family Transitions: Cowen,P.A. Hetherhington, H. Eds. Eribaum: Hillsdale. NJ. USA. Pp. 11 – 164
Fietcher, A.C. & Jefferies, B.C (1999), Parental Mediators of Association
Foley, S. (2018), Mothers whose Response to infants' Facial Increase Report Stronger bonds with Babies. December. Unpublished Ph,D. Thesis.
Foley, S.D. & Hughes, C. (2018), Great expectations? Do Mothers' and Fathers'
Gerris, J.R. (2009), The Relationship between Parenting and delinquency, *J. Aboorm Child Psychology*. 37, 749 – 775.
Ginsburg, G.S. & Bronstein, D. (1993), Family Factors related to Children's intrinsic/extrinsic motivational orientation and academic Performance. *Child Development.* 64, 1461 – 1474.
Gluckhum, P.D., Hanson, M.A., Cooper, C. & Thornburg, K.L. (2018), Mother's
Goldson, H, (1998), Non-linear Multilevel Models with an Application to Discrete Response Data. *Biometrika,* 78(1), 45 – 51.
Gonzalez, A., Holbern, M. & Quitter, S. (2002), School Students' goal
Harper, D. (2010), Online Etymology Dictionary Medicine Definition for Embryo. (1-2)
Heckman, J. (2007), The Econometrics, Technology and Neuroscience of Human Capability Formation. *PNAS* 104(33): 13250 – 13255.
Hoeve, M. Dubas, J.S., Eichelsheim, V.I. Van der Laan, P.H. Smeenik, W. &
Hoskins, D.H. (2014), Consequences of Parenting on Adolescent Outcomes. *Societies.* 4, 506 – 531.
Hox, J.I. (2010), Multilevel Analysis, Techniques and Application (2nd ed). *New York Routledge*. *ISBN 978-1-84872-845-5.*
Hsin, A. (2012), "Is Biology Destiny? Birth Weight and Differential Parental Treatment" *Demography*, 49(4): 1385 – 1405.
Kalinka, A.T. & Tomancak, P. (2012), The Evolution of Early Animal Embryos:
KarKKanen, K. Katri, S. & Oati, K.Y. (1999), Why do Plants About so many
Landry, S.H. (2014), The Role of Parents in Early Childhood Learning. *Children Learning Institute of Texas Science Center.* U.S.A. Revised ed. Pp. 1 – 22.
Liddel, H.G. & Scott, R.A. (2013), At the Way Back Machine. Greek-English Lexicon, on Perseus.
Luyckx, K. Tildeley, E.A., Soenens, B., Andrews, J.A., Hampson, S.E. Peterson,
M. & Duriez, B. (2011), Parenting and Trajectories of Children's Maladaptive
Maccoby, E.E. & Martin, J.A. (1983), Socialization in the Context of the Family:
McPherson, m. Arango, P., Fox, H. Lauver, C., McManus, M. & Newacheuv,
Milevsky, A., Schlechter, M., Netter, S., & Keehn, D. (2007), Maternal and
Murray, D.M. (1998), Design and Analysis of Group-Randomized Trials. New York, NY. Oxford University Press.
New Dictionary of Cultural Literacy (2005), Dictionary. (3rd edition). Houghman Mifflin Harcout Publishing Company.
Nowick, S. IIes-caven, Y. Gregory, S., Ellis, G. & Golding, J. (2017), The Impact
of Prenatal Parental Lows of Control on Children's Psychological Outcomes in Infancy and Early childhood; A Prospective 5-year Study. *Front Psychology Development Psychology* 8:546.

orientations and their relationship to perceived parenting styles. *Contemporary education Psychology. 27, 450 – 471.*

P.M. (1998), A New Definition of Children with Special Health Care Needs. *Pediatrics.* 102: 137 – 140.

Parent-Child Interaction. *In handbook of Child Psychology.* Mussen. P.H. Ed. Wiley: New York, NY, USA Volume 4 PP. 1 – 102.

Parenting to a Typology of Family Parenting Styles and Adolescence Outcomes. *Journal of Family Issues.* 28: 212 – 241.

Parents' Power: Assertive Patterns and Practices on Adolescent Development. *Parenting*, 10, 157 – 201.

Paternal Parenting Styles in Adolescent: Association with self-esteem, depression and Life-satisfaction. *Journal of Child Family Studies.* 73, 39 – 47.

Prenatal thoughts and feelings about the infant predict parent-infant interaction quality? A Meta-analytic review. *Developmental Review*. 48: 40.

Querido, J. G. & Warner, J, D., Eyberg, S.M. (2002), Parenting Styles and Child

Simons, L.G. & Conger, R.D. (2007), Linking Mother-Father Differences in

Steinsberg, L. & Suk, J.S. (2002), Parenting Adolescents, *In Handbook of Parenting* (2nd ed) Bostein, M.H. ed. Evlhaum, Mahwah N.J. USA. Vol. 1.

Stopper, C, Melissa, S. Jr. & William, C. (2004), Miscarriage (Spontaneous Abortion). Medicine Net.com.

Ward, L. (2007), Mother's Stress Harms Foetus, Research Shows. BST. *Social Affairs Correspondent.*

Wright, L., Ross, K. & Daar, A.S (2008), Organ Transplantation. In Swinger P.A. Views, A.M. editors. The Cambridge Textbook of Bioethics. Cambridge. U.K. Cambridge University Press. P. 147.