

**DEMOGRAPHIC AND PSYCHO-SOCIAL BACKGROUND AS
CORRELATES OF PARENTS' PERCEPTION OF CAUSES OF
MENTAL RETARDATION AMONG SCHOOL CHILDREN IN
SOUTH-WESTERN NIGERIA**

BY

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ABSTRACT

There are contradictory opinions surrounding the causes of mental retardation among parents of children with mental retardation. The causes on one hand may be genetic and on the other may be largely attributed to mystical forces. Previous studies have tried to ascertain the causes of mental retardation in families but they have not been able to establish the actual etiology. However, demographic and psychosocial factors are also important factors worthy of investigation in identifying the causes of mental retardation. This study, therefore, investigated the demographic and psycho-social background as correlates of parents' perception of the causes of mental retardation in 15 special education primary schools in South-Western, Nigeria.

This study adopted a descriptive survey design of the *ex-post facto* type. Purposive sampling technique was used to select 606 respondents, 338 women and 268 men who were parents of children with mental retardation. These parents were met during Parent Teacher Association meeting at different occasions in all the schools. Data were collected using Parents' Perception of Mental Retardation Scale ($r=0.68$) and Mental Retardation Determinant Scale ($r=0.77$). Six research questions were answered and three hypotheses tested at the 0.05 level of significance. Data collected were analysed using multiple regression and analysis of variance.

The psycho-social variables have a multiple correlation with the dependent variable (chances of giving birth to a child with mental retardation) ($R=0.86$). This joint correlation is shown to be significant ($F_{(7,598)}=247.48$; $p<0.05$). The psycho-social variables accounted for 74.3% of the total variances in the chances of giving birth to a child with mental retardation. Three of the psycho-social variables have significant ($p<0.05$) relative contribution to the chances of giving birth to a child with mental retardation. These are cultural practices ($\beta=0.36$; $t=15.92$; $p<0.05$); birth order ($\beta=0.09$; $t=3.65$; $p<0.05$) and birth trauma ($\beta=0.07$; $t=2.921$; $p<0.05$). Socio-economic status and disease during the child's development have no significant relative contribution. It was also discovered that one in every three women that have children with mental retardation is between age 35 and 40. Large proportion of women (77.7%) that have children with mental retardation had no complication during pregnancy and (72.3%) had no difficulties during labour.

Demographic and psycho-social variables were perceived to have jointly contributed to predicting mental retardation in children. Therefore, there is need for public enlightenment on the causes of mental retardation. Efforts should be made to educate and counsel parents of children with mental retardation on the causes and proper management of the condition.

Key words: Mental retardation, Demographic, Psycho-social background and Child's development

Word count: 405

DEDICATION

This work is dedicated to Jesus the King of kings, the Ancient of Day and the
Lover of my soul.

And
To my beloved family

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CERTIFICATION

I certify that this work was carried out by Miss. T.A. Adeyemi in the Department of Special Education, Faculty of Education, University of Ibadan under my supervision.

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CHAPTER ONE

INTRODUCTION

1.1 Background to the Study

Mental retardation is a condition that stands as a source of pain and bewilderment to many families, particularly in developing countries like Nigeria. Its history dates back to the beginning of human's time on earth. The idea of mental retardation can be found as far back in history as the therapeutic papyri of Thebes (Luxor) Egypt, around 1500BC (Biasni, Grupe, Huffman and Bray, 2002). It has also been defined by a collection of symptoms, traits and/or characteristics and renamed many times all through history. For example, feeble-mindedness and mental deficiency were used as labels during the later part of the last century and in the early part of this century.

Consistent across all definitions are difficulties in learning, social skills, everyday functioning and age of onset (during childhood). Mental retardation is a challenge and potential source of stress to the family of an individual with this disorder. From identification through treatment or education, families struggle with questions about the future. The mentally retarded have been the subject of myth and confusion throughout history (Thompson and Grabowski, 1977). Many misconceptions remain, making it difficult to initiate change, particularly in large institutions. Some misconceptions lead to institutionalisation of retarded people who do not belong in such a setting and who suffer deleterious effect.

Societal attitude towards parents of people with mental retardation is negative in the Nigerian society. Society defines them by what they do not have rather than what they have; what they cannot do than what they can do. They are relegated, denigrated and stigmatised on the basis of some retrogressive myth and tradition. They are abused physically and sexually with impunity and in extreme cases; even their right to life itself denied them. As a result of negative societal attitude and the stigma attached to the condition, parents of children with mental retardation tend to develop negative attitude. They even display an attitude of anger, rejection and overwhelming protectiveness of their children.

This development makes parents "shut out" these children from their immediate environment (their peers, visitors and friends). Such attitude does not augur well for the development of these children. Locking them up and preventing

them from interacting with their immediate environment further deteriorates their conditions. Despite the great variation in gross physical appearance and the obvious variability in performance levels of any population of mentally retarded residents, many still refer to them as basically alike. The simple fact is that there are many types of mental retardation which can be grouped into different levels.

Attempts to define varying levels of impairment have tended to rely increasingly on measurement by means of intelligence quotient test. (Heward, 2004). In the American Association on Mental Retardation (2002) definition, for example, the phrase, “significantly subnormal general intellectual functioning” translates directly and officially into an intelligence quotient test score that is more than two standard deviations below the population mean. That is, a cut off representing about the third percentile which is a point below which only some three percent of the population score. This means an intelligence quotient of close to 70, depending on the particular test used.

American Association of Mental Retardation (2000) recognised four levels of retarded mental development as follows: mild (educable) mental retardation (IQ 52-67), moderately mental retardation (IQ 36-51), severe mental retardation (IQ 20-40) and profound mental retardation (IQ 20-25). Individuals with mild mental retardation constitute the largest number of those labeled mentally retarded. Persons in this group are considered educable and their intellectual levels as adults are comparable to those of average 8 to 11 year old children. Their social adjustment often approximates that of the adolescent although they tend to lack the normal adolescent imagination, inventiveness and judgment (Heward, 2004). They do not show signs of brain pathology or other physiological anomalies, but often they require some measure of supervision due to limited ability to foresee the consequences of their actions. With early diagnosis, parental assistance and special educational programmes, the great majority can adjust socially, master simple academic and occupational skills and become self supporting citizens.

Individuals who are moderately retarded with IQ 36-51 are likely to fall in the educational categories of trainable mentally retarded. In adult life, individuals classified as moderately retarded attain intellectual levels similar to those of average 4-7 year old children (Heward, 2004). He went further to say that while some of the brighter ones can be taught to read and write a little, and some manage to achieve a fair command of spoken language, their rate of learning is relatively slow, and the

levels of conceptualising extremely limited. Physically, they usually appear clumsy and ungainly, and they suffer from bodily deformities and poor motor coordination.

A distinct minority of these children is hostile and aggressive. More typically, they present an affable and somewhat vacuous personality picture. In general, with early diagnosis, parental help and adequate opportunities for training most of the moderately retarded can achieve partial independence in daily self-care, acceptable behaviour and economic usefulness in a family or other sheltered environment. Whether or not they require institutionalisation usually depends on their general level of adaptive behaviour and nature of their home situation.

Severely retarded individuals have intelligence quotient scores of 20-40. They may master some basic self-care and communication skills. Many severely mentally retarded are able to live in homes. However, many profit to some extent from training and can perform simple occupational tasks under supervision. (Coleman, Butcher and Carson, 1987).

Profoundly retarded individuals have intelligence quotient range of 20-25 or less. They may be able to develop basic self-care and communication skills with appropriate support and training. Their retardation is often caused by an accompanying neurological disorder. They need a high level of structure and supervision. (Heward, 2004; Beirne-Smith, Patton, Smith and Ittenbach, 1994).

Anything that damages and interferes with the growth and maturation of the brain can lead to mental retardation. There are hundreds of underlying determinants of mental retardation such as specific genetic abnormalities and syndromes or multifactorial conditions. (Black and Scott 2002). These might happen before, during or after the birth of the child.

Some environmental causes of mental retardation may include but not limited to maternal infection during pregnancy, prenatal exposure to certain medications or drugs, prematurity, delivery trauma and brain injury. These determinants are not hereditary and the recurrence risk for other relatives is not increased above the general population risk as long as these environmental triggers are not present (WHO, 2004).

There are other forms of mental retardation caused by genetic abnormalities. Examples are chromosomal anomalies, frequently associated with other physical conditions. Some chromosome anomalies are hereditary and can be inherited through an unaffected parent (Emory, 2004). Other types of mental retardation are multi-

factorial, meaning that both genetic and environmental factors contribute to the condition.

In a family with hereditary type of mental retardation the risk is greater for the first-degree relatives (parents, siblings and children) of the affected individual and is lower for more distant relatives (Burns, 2003). These types of mental retardation cannot be diagnosed prenatally. Socio-economic status encompasses the levels of cultural rank and degree of financial independence of an individual. Ward (2002) and Eggen (2001) define a person's income, his/her occupation, and his/her greatest level of mastered education as a means of calculating the socio-economic status. Socio-economic status directly affects basic needs such as nutrition, quality of life, safety and security at home and in the community (Hartwell, 2002). Brody (2004) states that older mothers from poverty environment also stand the risk of having retarded children. This suggests that age is not the only factor, rather, the mother's overall physical health is probably critical. Older mothers, if they live in poverty are likely to be less healthy but if such women maintain good physical fitness, they are likely to have reduced risks of pregnancy.

Poverty and cultural deprivation are key factors in determining mental retardation in some individuals. Children in poor families may become mentally retarded because of inadequate medical care and environmental health hazards. Also, children in disadvantaged areas may be deprived of many common cultural and day-to-day experiences provided to other youngsters (WHO, 2004). Research suggests that such under-stimulation can result in irreversible damage and can serve as a determinant of mental retardation.

The relationship between mental retardation and parental age at the time of a child's birth has been identified as an important determining factor for mental retardation in a family. Older women are more likely to suffer pregnancy complications; genetic abnormalities are more common in their fetuses and the miscarriage rate rises as the fertility rate falls. The effect of age on men's reproduction is less clear. Freeman (2000) opines that there is a small decline in sperm number and motility in men over 45 years. Though these characteristics are less noticeable in young men, they usually remain within normal limits until about age 70. However, men over 40 are more likely to produce genetically damaged sperm and can transmit these defects to their offspring.

At the other end of the age range, there are also added risks of young teenage mothers having a low birth weight infant, mentally retarded child or even still-birth. These effects are particularly evident for 15 year old mothers or younger. Given the fact that a very young mother has not completed her own growth and thus has extra nutritional needs of her own, one should probably not be surprised that young mothers with pregnancies are at risk of low birth weight or fetal abnormalities such as mental retardation.

Mental retardation knows no boundaries; it cuts across the lines of racial, ethnic, educational, social and economic backgrounds. It can occur in any family. In the light of the foregoing, the researcher will be looking at the demographic and psycho-social backgrounds of parents as correlate of mental retardation in South West Nigeria.

1.2 Statement of the Problem

In Africa, there is an estimation of 10-15 million people with mental retardation (United Nations Population Information Network, 2007). The causes are like the ones found anywhere else in the world, a combination of genetic factor, environmental factors such as malnutrition, poor prenatal care and childhood diseases. In Nigeria however, according to Bolumole (2006) there are as many as 3 million people with mental retardation.

Till date, the causes of mental retardation (MR) in developing countries in general and particularly among the low income group (the poor) and those that reside in rural areas are shrouded in myths and misconceptions. There seem to be shallow and limited understanding of the causes of mental retardation. Regardless of seemingly improved level of education in the country, there is no commensurate enlightenment with respect to the causes. Parents oftentimes see the birth of a child with mental retardation in the family as either a curse or an invocation by an enemy or directly from God. This therefore calls for a study that will bring to the fore relevant and basic knowledge that will stem the ignorance that increases the number of people that have to contend with the causes of mental retardation.

Lastly, the unwarranted stigmatisation of individuals with mental retardation and the poor comprehension of their condition underscore the need to study and unearth what promoted their disturbing state.

1.3 Purpose of the Study

Mental retardation is a source of worry and anxiety for any family with a child with such a problem. Many parents wonder why such a child is born into the family. The purpose of the study is to look at how and why a child develops mental retardation and also look at the correlation between mental retardation and the following psychosocial variables parental age, birth order, socio-economic status, birth trauma, disease, genetic make-up of the parents and cultural practices engaged in by parents.

1.4 Significance of the Study

First, the study has been able to generate sufficient data and information that will reorientate people about unsubstantiated myths and misconceptions. It is believed that the research output will be appropriately and sufficiently disseminated. Arising from this, when parents and other stakeholders are better informed, this will reduce cases of mental retardation and equally facilitate better management of children with mental retardation. Herein, lies the significance and the essence of this study.

Second, the study has made a veritable addition to the growing literature in this scholarly field. Indeed, though marginal, it has certainly bridged the gap in literature, particularly in Nigerian context. Further, determination of an underlying etiology serves to limit additional unnecessary testing and empowers the family by providing a better understanding of a child's problem and the reasons for it. Families need to gather the right form of information about the condition and become knowledgeable about its causes or management of such a child. This study therefore, has been able to significantly increase the awareness of families and the community in general to factors that determine mental retardation in an individual. Families within the child bearing age can then avoid these conditions that are known to cause mental retardation. When parents and stakeholders are better informed this will stem down the erroneous beliefs about the causes of mental retardation.

Lastly, the study's significance can be premised on the likelihood that the expected results should facilitate the determination of the underlying etiology of mental retardation. This will entail early identification and prompt provision of rehabilitation services, which are essential components of service delivery system to children with mental retardation. The knowledge of this fact will equip special education teachers, counsellors and service providers with ways and means of

counselling parents and the community at large on the causes and prevention of mental retardation.

1.5 Research Questions

1. Do the children with mental retardation have common birth order?
2. Is there any significant association between the birth order of children and mental retardation?
3. What is the age range of the parents of children with mental retardation when they had the babies?
4. Did mothers of the children with mental retardation have difficulties during pregnancy and labour?
5. Did these children experience accident or chronic disease at the early developmental stages?
6. What is the perception of parents of children with mental retardation on the following variables as factors responsible for mental retardation?
 - (a) Parental age
 - (b) Birth order
 - (c) Socio-economic status of the parents
 - (d) Birth trauma
 - (e) Disease
 - (f) Genetic make-up of the parents
 - (g) Cultural practices engaged in by parents.

1.6 Hypotheses

HO₁: There is no significant composite effect of (a) parental age (b) birth order (c) socio-economic status of the parents (d) birth trauma (e) disease (f) genetic make-up of the parents (g) cultural practices engaged in by parents on the chances of giving birth to a child with mental retardation.

HO₂: There is no significant relative effect of:

- (2a) parental age on the chances of giving birth to a child with mental retardation;
- (2b) birth order on the chances of giving birth to a child with mental retardation;
- (2c) socio-economic status of the parents on the chances of giving birth to a child with mental retardation;
- (2d) birth trauma on the chances of giving birth to a child with mental retardation;

- (2e) disease on the chances of giving birth to a child with mental retardation;
- (2f) genetic make-up of the parents on the chances of giving birth to a child with mental retardation; and
- (2g) cultural practices on the chances of giving birth to a child with mental retardation.

1.7 Scope of the Study

The study covers all the states in the South-Western part of Nigeria namely: Oyo, Ogun, Osun, Ondo, Ekiti and Lagos. The independent variables are parental age, birth order, socio-economic status of the parents, birth trauma, disease, genetic make-up of the parents and cultural practice. The dependent variable is chances of giving birth to a child with mental retardation.

1.8 Operational Definition of Terms

Mental Retardation: A disability that occurs before age 18. It is characterised by significant limitations in intellectual functioning and adaptive behaviour as expressed in conceptual, social and practical adaptive skills.

Genetic Factor: Any condition affecting the unit of heredity called genes. Anything that goes wrong with the genes during fertilisation and conception and results in a handicapped baby who may be mentally retarded, deaf or blind.

Socio-economic Status: This describes the income, educational achievement and occupational level of an individual or family

Birth Order: This is the way and position in which a child is born into a family.

Environmental Toxins: Pollutants and poisons in the environment leading to the development of mental retardation in an individual. This may happen during the perinatal period and they include alcohol, tobacco, lead, mercury and drugs.

Birth Trauma: Birth related brain injuries caused by deprivation of oxygen, mechanical injuries or disease at birth.

Intelligence Quotient: The level of intelligence estimated on the ratio of mental age to chronological age.

Genetic make-up: The units in the cells of a living thing that control its physical characteristics.

CHAPTER TWO

LITERATURE REVIEW

This chapter covers a review of literature on the following:

- 2.1 Conceptual Clarification
 - 2.1.1 Concept of Mental Retardation
 - 2.1.2 Prevalence of mental retardation;
 - 2.1.3 Causes of mental retardation;
- 2.2 Empirical studies
- 2.3 Theoretical Framework

2.1 Conceptual Clarification

2.1.1 Concept of Mental Retardation

Mental retardation is a term used when a person has certain limitations in mental functioning and in skills such as communicating, taking care of him or herself and social skills. These limitations will cause a child to learn and develop more slowly than an average typical child. Children with mental retardation may take longer to learn, to speak, walk and take care of their personal needs such as dressing or eating. They are likely to have trouble learning in school. They will learn, but it will take them longer than necessary. (Heward, 2004). Indeed, there may be some things they cannot learn (ARC, 2002).

An accurate and consistent definition of mental retardation is critical because of its impact on the prevalence and the number of those with mental retardation. However, despite the importance of consistency, mental retardation is not always defined in the same way across studies or service agencies, even within the same state (Koller, Richardson & Katz, 1984; Brothwick – Duffy, 1994). While some definitions rely on intelligence quotient scores alone to classify individuals with mental retardation, some only use adaptive skills classification and others include both intelligence quotient scores and measures of adaptive skills (Brothwick–Duffy, 1994; and Whitman, Hantula and Spence, 1990). In addition, many studies are based on broad categories of either severity (using labels such as mild, moderate, severe and profound mentally retarded) or etiology (utilising the terms cultural/familial and organic mental retardation).

The most commonly cited definition of mental retardation comes from the American Association on Mental Retardation (AAMR) (1992). The AAMR (1992)

defines mental retardation as the onset of significant limitations in both general and intellectual adaptive functioning during the developmental period (18 years and under). Intellectual limitations refer to an Intelligence Quotient (IQ) which falls two standard deviations below the population mean of 100 (<70), and adaptive functioning limitations refer to impairments in at least two out of ten skill areas (American Association on Mental Retardation, 1992). Mental retardation is also defined in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM – IV) by the American Psychiatric Association (APA) (1994).

Similar to the American Association on Mental Retardation definitions, the DSM-IV has three diagnostic criteria for mental retardation, this includes sub-average intellectual functioning ($IQ < 70$), impairment in adaptive functioning and onset before age 18 (APA, 1994). Although the core criteria for mental retardation are similar between the AAMR and the DSM – IV definition, there are important differences between the two. First, while DSM – IV definition of mental retardation has a strict intelligence quotient cut off of 70, the 1992 AAMR definition suggested that if an individual shows other signs of mental retardation, the intelligence quotient may be raised to 75 (Schalock, Stark, Snell, Coulter Polloway, Luckasson, Reiss, Spitalnik, 1994). Second, although both definitions include a sub-classification system, the bases of the two sub-classification systems differ.

The AAMR definition includes a scale measuring the extent of support needed to function in the environment, focusing on individual's strengths, support systems capabilities and interaction with the environment (King, State, Shah, Davanzo, Dyken, 1997; Schalock Stark, Snell Crulter, Polloway, Luckasson, Reiss and Spitalnik, 1994). In contrast, the DSM-IV definition specifies the degrees of mental retardation severity based on the level of Intelligence Quotient (mild = 50 – 55 to 70, moderate = 34 – 40 to 50 – 55, severe 20 – 25 to 35 – 40 and profound $<20 - 25$) (APA, 1994). Further, although the APA includes mental retardation in the DSM-IV, thereby classifying mental retardation as a mental disorder, the American Association on Mental Retardation however, explicitly states that mental retardation is neither a medical nor mental disorder (AAMR, 2000).

Considerable controversy exists over the use of the 1993 AAMR definition. For instance, while Macmillan, Gresham, Siperstein (1995), Reiss (1994) state that the definition is intended to broaden the definition of mental retardation so that more individuals would be eligible for services, several researchers believe that the

definition compromises the conceptual and psychometric integrity of the 1983 definition of mental retardation. Prior to 1992, for example, the AAMR definition focused on deficits at each developmental stage using a severity scale (similar to that used by the APA) to emphasise intelligence quotient score and expected age-appropriate behaviour (AA MD, 1983). In 1993, however, the AAMR increased the possible upper IQ score to 75, set general adaptive behaviours as a criteria and developed a sub-classification system based on levels of needed supports (Macmillan *et al.*, 1993).

Critics of the new definition believed that setting the intelligence quotient score limit high has been criticised because of the cultural biases inherent in this measure (Zigler, Balla and Hodapp, 1984, Hobbs, 1975). Additional concern revolves around the measurement of adaptive behaviours and needed support which are thought to be poorly defined and to ignore developmental factors, thereby increasing the potential for sub classification. Consequently, some authors believe that a sub classification system of mental retardation should rely on etiology rather than poorly measured levels of support (Macmillan *et al.*, 1993).

In contrast, Zigler and colleagues (1991, 1987, 1986, 1984, and 1967) argue that an appropriate classification of mental retardation employs both intelligence quotient score and etiology of the retardation. Consequently, they suggest categorising mental retardation into cultural/familial organic groups based on the presence or absence of a known organic etiology. This two-group approach is one of the most well documented in the mental retardation literature over the last century.

Cultural/familial mental retardation refers to individuals with intelligence quotient of 50 to 70 who do not have any identifiable physiological or genetic deficit. Although individuals with cultural/familial mental retardation have lower intelligence than individuals without mental retardation, the stages of cognitive development do not vary between these two groups. Those with cultural/familial mental retardation however develop cognitively at a slower rate and do not reach the same cognitive level as the general population. Consequently, individuals with the same mental age (or cognitive ability), regardless of chronological age, should perform similarly on cognitive-linguistic tasks.

Emotional and motivational factors, however, influence the performance of individuals, and may account for certain behavioural difference between those of the same mental age (Zigler and Hodapp, 1991; Zigler and Hodapp, 1986; Zigler 1986).

In contrast, organic mental retardation is attributable to an identifiable physiological deficit. Individuals in this group typically have intelligence quotient below 50, although individuals with intelligence quotient between 50 and 70 can also be classified as having organic mental retardation.

The cognitive development of individuals in this group is generally not thought to be comparable to those either without mental retardation or with cultural/familial mental retardation. The behaviour of individuals in this group then is primarily the result of their physiological deficit (Zigler and Hodapp 1991, Zigler and Hodapp 1986, and Zigler and Hodapp 1984). Some researchers, in fact, believe that all individuals with mental retardation should be classified in the organic group. As science advances, they argue physiological deficits will be discovered even among those with no present known organic etiology (Zigler and Hodapp 1986, Richardson 1981).

The two-group approach, however, may be too broad a classification system to adequately account for the heterogeneity of each group. While Zigler and Hodapp (1986) thought the cultural/familial group to have at least three different sub- types, Grossman (1983), Lubs and Mass (1977) opine that there are hundreds of identified etiologies of organic mental retardation. Burack (1990) concludes that it is inaccurate to view individuals with mental retardation as fitting into one of two homogenous classes particularly because many experts in the area embrace the theory of polygenic inheritance. Although these different definitions of mental retardation do overlap, and are therefore somewhat comparable, multiple classification systems can make comparisons across studies difficult. In addition the consistency of mental retardation classification has been further complicated by the use of imprecise labelling.

In the United States, for example, many individuals with mild mental retardation have adopted the label “learning disabled” in order to avoid the stigma associated with “mental retardation” (Palfrey, 1994). The label “learning disabled” however, technically refers to individuals of normal intelligence who are not performing at their maximum ability level (American Association on Mental Retardation, 1983). Moreover, in England according to Bhrochain (1989), the term “learning disabled” is used to identify individuals with mental handicaps. This term then, has become non-specific and includes individuals with a variety of conditions, including both individuals with and without mental retardation. This type of imprecise labelling can

be problematic, because it can lead to difficulties in conducting needs' assessments and allocating services, as well as interpreting studies that use this classification.

2.1.2 Prevalence of Mental Retardation

The prevalence of mental retardation is affected by many factors, including the definition of mental retardation, the population studied and advances in medical technology. The definition of mental retardation is an integral part of the determination of mental retardation prevalence in the population. In addition, the population studied influences the prevalence found and indicates how generalisable the findings may be. Most research uses either population-based or service use-based (administrative) data. While many European countries maintain registries of individuals with mental retardation (making population based studies common in those countries) no such registry or comprehensive national survey exists in the United States. One national survey of the United States population, the National Health Insurance Scheme (NHIS), did have one question regarding mental retardation, but because of low prevalence found in 1981, the question was dropped in 1988 (Boyle, Decoufle, Yeargin All-Sopp, 1994).

In addition, in 1994 a supplement to the National Health Insurance Scheme (NHIS-D) was employed to collect population-based data on disabilities. The definition of mental retardation used in the National Health Insurance Scheme-D, however, was not consistent with either the AAMR or the APA definition; rather the NHIS-D classification focused on previously diagnosed mental retardation, conditions frequently associated with mental retardation and functional limitations in learning. Further, although mental retardation involves disabilities of development, individuals with mental retardation did not necessarily meet the criteria (three or more functional limitations) to be classified with a developmental disability as defined by Public Law 98-527, in the NHIS-D (Research and Training Centre on Community Living and Institute on Community Integration, 2000).

Since 1990, the Survey of Income and Programme Participation (SIPP), another US population-based survey has documented mental retardation among households randomly selected for participation. It does not, however, make a specific effort to sample households of individuals with mental retardation or other disorders. As a result, given the low probability of identifying individuals with mental

retardation in a randomly selected population, the SIPP cannot be considered a comprehensive account of those with mental retardation (U S Bureau of the Census, 1999). In addition, both the NHIS and the SIPP underestimate the prevalence of disabilities among children and adults because individuals living in institutions or group homes are excluded from the surveys (U S Bureau of Census, 1999). In contrast to many European studies, most research efforts in the U S do not use population-based samples; rather, they rely on the numbers of individuals who utilise special services to estimate the prevalence of mental retardation in the overall population.

Advance in medical technology have had a great impact on the prevalence of mental retardation as well. For a long period, according to Whitman, Hantula, Spence (1990); Primrose (1984), medicine's ability to treat the co morbid conditions of individuals with mental retardation and thus increase their survival time had improved. For example, individuals with down syndrome tend to suffer from thyroid and heart condition, which can be better detected and treated today than in the past (Saenz, 1999; US Preventive Services Task Force, 1996; Singer, Cooper, Levy, Ladenson, Braverman, Daniels, Greenspan, McDongall, Nikolai, 1995). Therefore, the increased life expectancy of those individuals results in higher prevalence at any one point in time.

Further, several factors potentially affect the number of individuals who are actually born with mental retardation. The rise in prenatal care increased genetic screening and improvements in neonatal testing which tend to increase the likelihood that children are born healthy. In contrast, other factors such as increased prenatal use of substance tend to counteract these effects and increase the prevalence of mental retardation (Grossman, Richards, Anglin and Hutson, 2000). In sum, it is difficult to predict how the synergy of these factors affects the ultimate prevalence of mental retardation.

It is estimated that as many as 2.0 to 7.5 million Americans of all ages may have mental retardation, and that 1 in 10 families are directly affected by mental retardation (Grossman *et al.*, 2000). Many reports have suggested that the population prevalence of mental retardation in the U S is as high as 3.0% (Zigler and Hodapp, 1986).

A US study using administrative data, however, found the prevalence among children to range from 0.3% to 3.1% in different regions of the country, with a national average of 0.1% (King, State, Shah, Davanzo, Dyken, 1997). According to Boyle,

Yeargin - Allsopp, Holmgreen, Murphy, Schwendel (1996), the Metropolitan Atlanta Developmental Disabilities Surveillance Programme, a population-based study, which only used IQ score as the criterion for mental retardation, found an overall prevalence of 0.9% among 3 to 10 years old children. Further, although the National Health Insurance Scheme – D used its definition of mental retardation, it reported that 0.78% of the population had mental retardation, with a prevalence of 0.45% for children 0 to 5 years, 2.0% for children 6 to 17 years, and 0.52% for individuals 18 years or older (Research and Training Centre on Community Living and Institute on Community Integration, 2000).

Further, because teachers are often the first to notice child developmental problems most identified mild mental retardation is initially detected during school years. Boyle, Yeargin-Allsopp, Holmgreen, Murphy, Schendel (1996) state that the Atlanta population based study, for example, indicated that while the prevalence of mild or moderate mental retardation was only 0.5% for children 3 to 4 years of age the prevalence rose to 1.2% when older school-aged children were studied. It has been suggested, however, that only 50% of children with mental retardation are identified at a young age because the failure to adapt normally and grow intellectually may not become apparent until later in life. Early identification may be further hampered by the fact that most pediatricians do not generally use standardised instruments to detect developmental delays (Grossman, *et al.*, 2000)

In addition, because of their high level of functioning those with mild mental retardation are often unknown to specific services once they leave school, and so, as adults, these individuals may not be counted as having mental retardation in studies using administrative data. Moreover, many diagnosed children do not meet the criteria when tested later in life. This suggests that either childhood or adult diagnoses are not adequately evaluating adaptive functioning (King, *et al.*, 1997; Dykens *et al.*, 1994; Loveland and Kelley, 1988; Zigler and Hodapp, 1986; Zigler *et al.*, 1984).

The majority of individuals with mental retardation have historically been classified as having mild, cultural/familial mental retardation. In the Atlanta population-based study 0.84% of 10 years olds had IQs between 50 and 70 (mild mentally retarded) and 0.36% had IQs less than 50 (moderate to profound mentally retarded) (Yeargin-Allsopp *et al.*, 1997). In addition, Boyle *et al.*, (1996) reported that two thirds of the children with mental retardation in this study were classified as mild. Further, the prevalence and type of mental retardation found in this study varied with race and

gender, with black males having percentages of mild, moderate and severe mental retardation 3.1 times as high as those for white female. Percentages of profoundly mentally retarded (most likely organic), however, did not vary by race in this study (Boyle *et al.*, 1996).

Part of the variation in US reported prevalence of mental retardation is clearly due to difference between research efforts. For example, researchers making extrapolation based on birth estimates may report a higher prevalence than the number of cases counted in studies using either population- based or administrative data (Tarjan, Wright, Eyman, Reevan, 1973). The results of these latter studies however, consistently indicate a prevalence of 1.0%.

In other developed countries the prevalence of mild mental retardation appears to be lower than it is in the U S Percentage of mental retardation or mental handicap in Sweden for instance, have been estimated to be between 0.3% and 0.7% (Zigler and Hodapp, 1986; Haldin, 1986; Goldin, 1982; Grunewald, 1979). Interestingly, Zigler and Hodapp (1986) state that although the prevalence of mild mental retardation has been found to be lower in Sweden than in the US, the two countries have reported comparable percentages of severe mental retardation. Sweden's low prevalence of mild mental retardation may seem surprising, given that at least some of the Swedish studies use a higher IQ cut-off (<80) to define this condition. However, Sweden has few psychologists, and testing is not as widespread there as it is in the US

Additionally, Zigler (1987) believes that Swedish prevalence estimates of mental retardation are based on the subjective opinions of teachers and clinicians, who are reluctant to label mildly cognitively impaired children. Further, since Sweden keeps a registry of individuals with mental retardation, many Swedish studies are population-based which may lead to more accurate population prevalence than that estimated in the U S In addition, Sweden is a welfare state, and has many programmes available for those with mild mental retardation. As a result many of these individuals are cared for in the community and may never even be thought of as having mental retardation until their IQs (at least males) are formally tested for entry unto military service (Zigler, 1987; Zigler and Hodapp, 1986). When estimates from the community are combined with estimates from armed forces testing, the prevalence estimates for mental retardation increases to 2.21% similar to that found in other countries.

Other developed countries also have registries of mental retardation which makes population-based studies more feasible than in the US The overall prevalence

of moderate and severe mental retardation, arrested development or severe abnormality among children and adults in England had been found to range from 0.3% to 0.5% (Goh and Holland, 1994). A study using a surveillance registry in British Columbia found the overall mental retardation prevalence rate to be similar (0.4%) with 0.1% mild, 0.1% moderate, 0.05% severe, 0.04% profound and 0.01% unspecified mental retardation (Herbst and Baird, 1983). In Ireland, using an intelligence quotient cut off of 50 (severe mental retardation) the rate of mental retardation among adults 20-29 was found to range from 0.4 to 0.6 % (Mallon Mackay, McDonald, Wilson 1999).

In less developed countries, percentages of mental retardation are generally found to be higher, from 1.6% to 3.0% (Islam, Durkin, Zaman, 1993). However, several recent studies have found the prevalence of mental retardation to be quite low. For example, in the People's Republic of China, the use of intelligence test in several districts found prevalence that range from 0.4% to 0.7% (Kuo-Tai, 1998). Similarly, a study in Cape Town, South Africa using administrative data, found the prevalence of severe mental retardation to be 0.3% (Friedlander and Power 1982), and a population-based study of prevalence in Bangladesh found a rate of 0.6% for severe mental retardation and 1.4% for mild mental retardation. Further, a study that went door-to-door in India, using Binet Simon scale to define mental retardation as intelligence quotient <80, has indicated a prevalence rate of 0.4% in the general population and 1.0% among children.

In Nigeria, there is no accurate data and statistics of the population of people with mental retardation. But according to Down Syndrome Association of Nigeria (2001) it is hypothesised that for every 800 babies born one will have mental retardation, bearing in mind the fact that Nigeria has an estimated population of over 140 million a large component of the people will have mental retardation. Indeed, it is further estimated that there are about 200,000 persons with mental retardation in Nigeria. However, the fact still remains that there is no accurate data and statistics of population of people with mental retardation in Nigeria.

Mental retardation is the most common developmental disorder. Centre for Diseases Control (2000) in tracking the number of children with mental retardation in a five-country area in Metropolitan Atlanta (Georgia) found that between 1991 and 1994 an average of 1% of children ages 3to10 years had mental retardation. It was also found that mental retardation was more common in older children (ages 6to10

years) than in younger children (ages 3-5 years). Mental retardation was also more common in boys than in girls and more common in black children than in white children. Centre for Disease Control also studied how many children in Metropolitan Atlanta had mental retardation in the mid 80s, this was found that 12 of every 1,000, 10-year-old children had mental retardation. Mild retardation is four times more common than severe mental retardation. Also, in 1997s in another study Centre for Disease Control found that about 1.5million children and adults (ages 6to64 years) had mental retardation. The highest rate of mental retardation was found in West Virginia and the lowest rate was found in Alaska.

Most prevalence studies then utilise I Q alone to define mental retardation. In the United States while the range of mental retardation prevalence had been reported to be between 0.3% and 3.0%, most studies using administrative or population-based data have found a prevalence of 1.0%. In contrast, international studies using population-based registries and somewhat different definitions of mental retardation report the prevalence to be less than 1.0%. The United States prevalence of severe mental retardation however, is comparable to that of other countries; in fact, some studies have found lower percentage of severe mental retardation in the United States than in other countries. Since most mild or moderate mental retardation is identified among school children in the United States, the discrepancy in the prevalence of these conditions may be due to international difference in school-based testing and services requirement (Palfrey, 1994), as well as mainstreaming practices.

Further, the low prevalence of mental retardation in some countries may be due to socio- cultural factors. In China for example, there is a one child per family policy (Kane and Choi, 1999) and a strong preference for terminating pregnancies with genetic abnormalities both of which may affect the number of children born with mental retardation (Mao and Wertz, 1997).

Bashir, Yaqoob, Ferngren, Gustavson, Rydelius, Ansari and Zaman (2002) studied the prevalence of mild mental retardation in 6to10 years old children in a prospectively followed cohort in Pakistan from areas with different socio-economic conditions. Retarded children were identified by a two-step method, comprising a household screening with the ten questions screening in 649 families followed by clinical investigation and psychometric testing (WISC-R and Griffiths) of the 132 children found by the screening, the overall prevalence of mild mental retardation among 6 to10 years old children was 6.2%. The distribution of mild mental

retardation was uneven with 1.2% among children for the upper middle class, 4.8% in the village, 6.1% in the urban slum and 10.5% in the poor peri-urban slum area. Additional impairment was found in 75% of the children with mild mental retardation, of which speech impairment was the most common.

Conclusively, the prevalence of mild mental retardation was found to be higher in a developing country than in developed countries. It also seemed to be related to poor socio-economic condition, as the prevalence in the upper-middle class was comparable to figures from developed countries while the prevalence in children from poor population groups was much higher.

2.1.3 Causes of Mental Retardation

Mental retardation can be caused by any condition, which impairs development of the brain before birth, during birth or in the childhood years. Scientists can identify a specific cause in 60 to 70% of mental retardation cases but in the remaining 30 to 40% of the people affected the cause remains unknown. (Schroeder, 2004). Three major causes of mental retardation have been identified as down syndrome, fetal alcohol syndrome and fragile X. Sutton (2001) states that many studies had attempted to find out how and why mental retardation occurs. Unfortunately, most were done in the 1980s or earlier, before the recent advances in the ability to identify and test for certain genetic and chromosomal disorders. Chromosomes are the materials within human cells that contain our genes. Damaged or abnormal genes and extra or missing chromosomes are known to be a prime cause of mental retardation.

It appears that the more severe the mental retardation, the more likely the cause is to be genetic. In mild mental retardation, a specific risk factor has been identified in about 43% of the cases studied. (Sutton2001) Only about 24% of those cases yielded a fairly clear or convincing diagnosis. Of those, 14% were classified as genetic and 10% environmental. With moderate - severe mental retardation, a cause is determined in up to 64% of cases with 45% of those being linked to genetic causes and 19% to environmental factors (Sulton, 2001).

Further still, Coon (1983) states that about 25% of all the cases of mental retardation are organic, or related to known physical disorders, including one or more of the following, birth injuries such as lack of oxygen are relatively rare but significant problem; fetal damage is a more common problem. Maternal drug abuse

and disease or infection contracted by the mother before birth, or by the child shortly after birth, can cause retardation. Metabolic disorders, such as cretinism and phenylketonuria could cause retardation. Explaining about the remaining 75% of the cases, Coon notes that in the majority of retardation cases no known biological problems can be identified which is attributed mainly to environmental factors.

As observed by Coon (1983) and Gebremariam (1993) familial retardation as it is called, occurs most often in very low income or impoverished households. In such homes nutrition, early stimulation, medical and emotional support are inadequate. Yet, even where a child's physical needs are being met, the intellectual and educational level of the home is typically low, making the chances of familial retardation high.

The seemingly lesser role of genetics in milder retardation may have something to do with the fact that some that fall into the mild mental retardation category may simply represent the low end of the normal range of human intelligence. In other words, their low IQ may be the result of low family IQ, not a genetic problem or other specific cause. Those in the moderate-severe category however are much more likely to be there because of an environmental or genetic problem that has disrupted normal development (Hunter, 2000). The main causes of mental retardation are classified as follows:

Genetic Causes

The most common genetic disorders that have been shown to cause mental retardation are Trisomy 21, which causes down syndrome deletions or duplications of the ends (Telomeres) of chromosomes and fragile X syndrome. Woods (2005) opines that scientists in 1992 identified fragile X as the most common inherited cause of mental retardation, responsible for up to 10% of cases. People with this condition inherit a defective gene that results in a weak spot on the X chromosome, a sex chromosome. The weak part of the chromosome is susceptible to breaking. Fragile X syndrome is more likely to cause retardation in males than females.

Genetics is the science that studies the principles and mechanics of heredity or the means by which traits are passed from parents to offspring (Glanze 1996). Through genetics, a number of specific disorders have been identified as being genetically caused. One example is fragile X syndrome, a common genetic cause of

mental retardation which is caused by the presence of a single non-working gene called the FMR-1 gene on a child's X chromosome.

Genetics originated in the mid 19th century when Gregor Mendel discovered over a ten year period of experimenting with pea plants that certain traits are inherited. His discoveries provided the foundation for the science of genetics. Mendel's findings continue to spur the work and hopes of scientists to uncover the mystery behind how genes work and what they can reveal about the possibility of having certain diseases and conditions. The scientific field of genetics can help families affected by genetic disorders to have a better understanding about heredity, what causes various genetic disorders to occur, and what possible prevention strategies can be used to decrease the incidence of genetic disorders.

Some genetic disorders are associated with mental retardation, chronic health problems and developmental delay. Because of the complexity of the human body, there are no easy answers to the question of what causes mental retardation. Mental retardation is attributable to any condition that impairs development of the brain before birth, during birth or in the childhood years (The Arc, 1993). As many as 50% of people with mental retardation have been found to possess more than one causal factor (AAMR, 1992). Some researchers have found that in 75 % of children with mild mental retardation the cause is unknown (Kozma and Stock, 1993).

The field of genetics has important implication for people with mental retardation. Over 350 inborn errors of metabolism have been identified most of which lead to mental retardation (Scriver, 1995). Yet, the possibility of being born with mental retardation or developing the condition later in life can be caused by multiple factors unrelated to one's genetic make-up. It is caused not only by the genotype (or genetic make-up) of the individual, but also by the possible influences of environmental factors. Those factors can range from drug use or nutritional deficiencies to poverty and cultural deprivation.

Since the brain is such a complex organ, there are a number of genes involved in its development; consequently, there are a number of genetic causes of mental retardation. Most identifiable causes of severe mental retardation (defined as an IQ of 50 or less) originate from genetic disorders. Up to 60 % of severe mental retardation can be attributed to genetic causes making it the most common cause in cases of severe mental retardation (Moser, 1995). People with mild mental retardation

(defined as an IQ between 50 and 70-75) are not as likely to inherit mental retardation due to their genetic make-up as are people with severe mental retardation.

People with mild mental retardation are more likely to have the condition due to environmental factors, such as nutritional state, personal health habits socio-economic level, access to health care and exposure to pollutant and chemicals, rather than acquiring the condition genetically (Nelso-Anderson and Waters 1995). Two of the most common genetically transmitted forms of mental retardation included down syndrome (a chromosomal disorder and fragile X syndrome (a single-gene disorders).

Over 7,000 genetic disorders have been identified and catalogued, with up to five new disorders being discovered every year (McKusick, 1994). Genetic disorders are typically broken down into three types: Chromosomal, single-gene and multifactorial. Chromosomal disorders affect approximately 7 out of every 1,000 infants. The disorder results when a person has too many or too few chromosomes, or when there is a change in the structure of a chromosome. Half of all first-trimester miscarriage or spontaneous abortions occur as a result of a chromosome abnormality. If the child is born, he or she usually has multiple birth defects and mental retardation. Most chromosomal disorders happen sporadically. They are not necessarily inherited (even though they are considered to be genetic disorders). In order for a genetic condition to be inherited the disease causing gene must be present within one of the parent's genetic code. In most chromosomal disorder, each of the parent's genes is normal. However, during cell division an error in separation, recombination or distribution of chromosomes occurs. Examples of chromosomal disorders include down syndrome, trisomy 13, trisomy 18 and crude chat (Arc, 1996).

Single-gene disorders (sometimes called in- born errors of metabolism or Mendelian disorders) are caused by non-working genes. Disorders of metabolism occur when cells are unable to produce proteins or enzymes needed to change certain chemicals into others, or to carry substances from one place to another. The cell's inability to carry these vital internal functions often results in mental retardation (Scriver 1995). According to Batshaw (1997) in 5,000 children are born with defective enzymes resulting in inborn errors of metabolism. Although many conditions are genetically referred to as 'genetic disorders'. Single-gene disorders are the most easy to identify as true genetic disorders since they are caused by a mutation (or a change) within a single gene or gene pair.

Combination of multiple gene and environmental factors leading to mental retardation are called multifactorial disorders. They are inherited but do not share the same inheritance patterns typically found in single-gene disorders. It is unclear exactly why they occur. Their inheritance patterns are usually much more complex than those of single gene disorder because their existence depends on the simultaneous presence of heredity and environmental factors. For example, weight and intelligence are traits inherited in this way (Batshaw, 1997). Other common disorders, including cancer and hypertension, are examples of health problems caused by the environment and heredity. Multifactorial disorders are very common and cause a majority of birth defects. Examples of multifactorial disorders include heart disease, diabetes, spinal bifida, anencephaly, cleft lip and cleft palate, clubfoot and congenital heart defects.

Genetic disorder can be inherited in much the same way a person can inherit other characteristics such as eye and hair colour, height and intelligence. Children inherit genetic or hereditary information by obtaining genes from each parent. There are three modes of inheritance; dominant, recessive and X-linked (or sex-linked) Dominant inheritance occurs when one parent has a dominant, disease-causing gene which causes abnormalities even if coupled with a healthy gene from the other parent. Dominant inheritance means that each child has a 50 % chance of inheriting the disease-causing gene. An example of dominant inheritance associated with mental retardation is tuberous sclerosis.

Recessive inheritance occurs when both parents carry a disease gene but outwardly show no signs of disease. Parents of children with recessive conditions are called 'carriers' since each parent carries one copy of a disease gene. They show no symptoms of having a disease gene and remain unaware of having the gene until having an affected child. When parents who are carriers give birth, each child has a 25% chance of inheriting both disease genes and not being affected. Each child also has a 25 % chance of inheriting two healthy genes and not being affected, and a 50 % chance of being a carrier of the disorder, like their parents. Examples of disorders which are inherited recessively and are also associated with mental retardation include phenylketonuria (PKU) and galactosemia.

X-linked or sex-linked inheritance affects those genes located on the X chromosome and can be either X-linked recessive or X-linked dominant. The X-linked recessive disorder, which is much more common compared to X-linked

dominant inheritance, is referred to as a sex-linked disorder since it involves genes located on the X-chromosome. It occurs when an unaffected mother carries a disease-causing gene on at least one of her X-chromosomes. Since females have two X chromosomes, they are usually unaffected carriers because the X chromosome that does not have the disease-causing gene compensate for the X chromosome that does. Therefore, they are less likely than males to show any symptoms of the disorder unless both X chromosomes have the disease causing gene.

If a mother has a female child, the child has a 50 % chance to inherit the disease gene and be a carrier and pass the disease gene on to her sons. (March and Dimes, 1995). On the other hand, if a mother has a male child, he has a 50 % chance of inheriting the disease-causing gene since he has only one X chromosome. Consequently, males cannot be carriers of X-linked recessive disorders. If a male inherits an X- linked recessive disorder, he is affected. Some examples of X-linked inheritance associated with mental retardation include fragile X syndrome, Hunter syndrome, Lesch Nyhan syndrome and Duchenne muscular dystrophy.

In the past, only a few genetic disorders could be detected and treated early enough to prevent disease. However, the Human Genome Project, an international project among scientists to identify all the 60,000 to 100,000 genes within the human body, is significantly increasing the ability to discover more effective therapies and prevent inherited disease. (National Center for Human Genome Research, 1995). As more disease-causing genes are identified, scientists can begin developing genetic therapies to alter or replace a defective gene. However, the development of gene therapies is still in the infancy stage.

Gene therapy (also called somatic-cell gene therapy) is a procedure in which 'healthy genes' are inserted into individuals to cure or treat an inherited disease or illness. Although there is a role for gene therapy in the prevention of mental retardation, it will most likely benefit only those people who have single-gene disorders, such as Lesch-Nyhan disease, Gaucher disease and phenylketonuria (PKU) that cause severe mental retardation (Moser, 1995). Gene therapy is far less likely to provide treatment of mild mental retardation which accounts for 87% of all cases of mental retardation (The Arc, 1993).

Down syndrome occurs when people inherit all or part of an extra copy of a pair of chromosomes known together as chromosome 21. Although regarded as genetic disorders, chromosomal disorders are not necessarily inherited (Woods, 2005). Both

parents may have normal genes, with the defect resulting from a random error when chromosomes reproduce.

Shaeffer, Ledbetter and Lupski (2000) state that Down syndrome accounts for 5% of mild mental retardation cases and 30% of severe mental retardation cases, telomere deletions of duplications account for about 7.5% of mental retardation, while fragile X is seen in 5% of mild and moderate – severe mental retardation. However, Wahlstrom (1990) declares that the fact that more than 500 other genetic diseases, mostly very rare, have also been associated with mental retardation suggests that scientists may someday learn that genetics are responsible for all, or nearly all, of the mental retardation cases whose causes is currently in the unknown category.

Other genetic causes of mental retardation are inborn errors of metabolism. They involve inheritance of a defective gene unable to produce enzymes or proteins needed for critical cell functions. Woods (2005) opines that scientists have identified more than 300 gene disorders involving inborn errors of metabolism. Many can result in mental retardation, including phenylketonuria (PKU), Tay Sachs disease, galactosemia, homocystinuria, maple syrup urine disease and biotinidase deficiency. Another common cause of mental retardation, congenital hypothyroidism, occurs in about 1 in every 4000 births. Infants with this disorder are unable to produce enough thyroxin, a hormone secreted by the thyroid gland. Mental retardation and stunted growth result unless they receive thyroid replacement therapy.

External Causes

A variety of problems during a woman's pregnancy can cause mental retardation in her child. These problems include malnutrition; a mother's use of alcohol or drugs; environmental toxins such as lead and mercury; viral infections, including rubella (German measles) and cytomegalovirus; and untreated diseases such as diabetes mellitus. Fetal Alcohol Syndrome results from excessive consumption of alcohol during pregnancy and is the most common preventable cause of mental retardation in the United States (Woods, 2005). It occurs in 1 to 3 out of every 1000 births. Malnutrition during pregnancy is a common cause of mental retardation in developing countries, where many women do not consume adequate amounts of protein and other necessary nutrients.

Some cases of mental retardation result from problems during birth, including premature birth, very low birth weight and stresses to the fetus such as deprivation of

oxygen. Infectious diseases during childhood, which are easily preventable through immunisation, also can cause complications. For example, measles, chicken pox and whooping cough may lead to encephalitis and meningitis, which can damage the brain.

Sutton (2001) opines that studies have shown that a number of environmental factors can cause or contribute to mental retardation. Physical trauma to the brain can cause mental retardation. Brain damage may result from accidental blows to the head, near drowning, severe child abuse and childhood exposure to such toxins as lead and mercury. Experts believe that poverty and lack of stimulation during infancy and early childhood can be factors in mental retardation. Children raised in poor environments are more likely to experience malnutrition, lack of routine medical care and environmental health hazards.

In Nigeria, large families are common among the low socio-economic group and in most cases children with mental retardation from such families sharing limited living space with other members of the family may suffer ill health as a result of overcrowding. Malnutrition from poor feeding is also a common feature in such families (Dubey, Edern, Tuarku, 1979). It should be noted that in most if not all societies, the family is viewed as critical determinant of development of the children. Socioeconomic status has variously been measured by considering the father's or mother's occupational level, family income, social values, types of dwelling and the individuals with whom the family identifies.

Socio-economic Status

Socio-economic status (SES) is typically broken into three categories, high, middle, and low to describe the three areas a family or an individual may fall into. When placing a family or individual into one of these categories any or all of the three variables (income, education, and occupation) can be assessed.

A fourth variable, wealth, may also be examined when determining socio-economic status.

Additionally, income, occupation, and education have shown to be strong predictors of a range of physical and mental health problems, ranging from respiratory viruses, arthritis, coronary disease, and mental retardation.

Income refers to wages, salaries, profits, rents, and any flow of earnings received. Income can also come in the form of unemployment or workers

compensation, social security, pensions, interests or dividends, royalties, trusts, alimony, or other governmental, public, or family financial assistance.

Income can be looked at in two terms, relative and absolute. Absolute income, as theorised by economist John Maynard Keynes, is the relationship in which as income increases, so will consumption, but not at the same rate. Relative income dictates a person or family's savings and consumption based on the family's income in relation to others. Income is a commonly used measure of SES because it is relatively easy to figure for most individuals.

Income inequality is most commonly measured around the world by the Gini coefficient, where 0 corresponds to perfect equality and 1 means perfect inequality. Economic inequality in the US is on the rise, leaving low income families struggling in society. Low income families focus on meeting immediate needs and do not accumulate wealth that could be passed on to future generations, thus increasing inequality. Families with higher and expendable income can accumulate wealth and focus on meeting immediate needs while being able to consume and enjoy luxuries and weather crises.

Educational attainment is preferable to analyse for SES because it can be figured for all individuals. A person's educational attainment is considered to be the highest level (grade or degree) of education they have completed.

Education also plays a role in income. Median earnings increase with each level of education. Higher levels of education are associated with better economic and psychological outcomes (that is more income, more control, and greater social support and networking).

Education plays a major role in skill sets for acquiring jobs, as well as specific qualities that stratify people with higher SES from lower SES. Lareau (2003) speaks on the idea of concerted cultivation, where middle class parents take an active role in their children's education and development by using controlled organised activities and fostering a sense of entitlement through encouraged discussion. Lareau(2003) argues that families with lower income do not participate in this movement, causing their children to have a sense of constraint. A division in education attainment is thus born out of these two differences in child rearing. In theory, lower income families have children who do not succeed to the levels of the middle income children, who feel entitled, are argumentative, and better prepared for adult life.

Occupational prestige as one component of SES, encompasses both income and educational attainment. Occupational status reflects the educational attainment required to obtain the job and income levels that vary with different jobs and within ranks of occupations. Additionally, it shows achievement in skills required for the job. Occupational status measures social position by describing job characteristics, decision-making ability and control, and psychological demands on the job.

Occupations are ranked by the census (among other organisations) and opinion polls from the general population surveyed. Some of the most prestigious occupations are physicians and surgeons, lawyers, chemical and biomedical engineers, computer support specialists, and communications analysts. These jobs, considered to be grouped in the high SES classification, provide more challenging work and ability and greater control over working conditions. Those jobs with lower rankings were food preparation workers, counter attendants, bartenders and helpers, dishwashers, janitors, maids and housekeepers, vehicle cleaners, and parking lot attendants. The jobs that were less valued were also paid significantly less and are more labourious, very hazardous, and provide less autonomy (Scott and Leonhart, 2005).

Occupation is the most difficult factor to measure because so many exist, and there are so many competing scales. Many scales rank occupations based on the level of skill involved, from unskilled to skilled manual labour to professional, or use a combined measure using the education level needed and income involved.

Wealth, a set of economic reserves or assets, presents a source of security providing a measure of a household's ability to meet emergencies, absorb economic shocks, or provide the means to live comfortably. Wealth reflects intergenerational transitions as well as accumulation of income and savings (MacArthur Research Network, 2008). Income, age, marital status, family size, religion, occupation, and education are all predictors for wealth attainment.

The wealth gap, like income inequality, is very large in the United States. There exists a racial wealth gap due in part to income disparities and differences in achievement. According to Shapiro (2004), differences in savings (due to different rates of incomes), inheritance factors, and discrimination in the housing market lead to the racial wealth gap. He claims that savings increase with increasing income, but African-Americans cannot participate in this, because they make significantly less than whites. Additionally, rates of inheritance dramatically differ between African-Americans and whites. The amount a person inherits; either during a lifetime or after

death can create different starting points between two different individuals or families. These different starting points also factor into housing, education, and employment discrimination. A third reason, Shapiro offers for the racial wealth gap are the various discriminations African- Americans must face, like redlining and higher interest rates in the housing market. These types of discrimination feed into the other reasons why African -Americans end up having different starting points and therefore fewer assets.

Krasus and Keltner (2008) found that children of parents with a high socio-economic status tended to express more "disengagement" behaviours than their less fortunate peers. In this context, disengagement behaviors represents actions such as fidgeting with other objects and drawing pictures while being addressed. Other participants born into less favored circumstances tended to make more eye contact, head nods and signs of happiness when put into an interactive social environment. Authors hypothesise that the more fortuitous peers felt less inclined to gain rapport with their group because they saw no need for their assistance in the future.

Virtually all societies are stratified according to socio-economic status and are marked by differences in almost all facets of life, environmental conditions, values, attitudes and expectations too. Further, socio-economic status variables begin to affect a child even prior to his/her entrance into the world. There are ample evidence from literature to show that socio-economic status (SES) is related in a highly positive way to the amount and quality of a person's education. This could be the reason why it is believed that socio-economic status (SES) today is largely a matter of educational and occupational status. Bradley and Corwyn (2002) described socioeconomic status (SES) as one of the most widely used constructs in the social sciences.

Several ways of measuring socioeconomic status have been proposed, but most include some quantification of family income, parental education and occupational status. Families with high socio-economic often have more success in preparing their young children for school because they typically have access to wide range of resources to promote and support young children's development. They are able to provide their young children with high quality childcare, books and toys to encourage children in various learning activities at home. Also, they have easy access to information regarding their children's health, as well as social, emotional, and cognitive development. In addition, families with high socioeconomic status often seek out information to help them better prepare their young children for school.

Crnic and Lamberty (1994) while discussing the impact of socio-economic status on children's readiness for school stated that the segregating nature of social class, ethnicity and race may well reduce the variety of enriching experiences thought to be prerequisite for creating readiness to learn among children. Social class, ethnicity, and race entail a set of contextual giving that dictate neighbourhood, housing, and access to resources that affect enrichment or deprivation as well as the acquisition of specific value system.

Ramey and Ramey (1994) opined that across all socioeconomic groups, parents face major challenges when it comes to providing optimal care and education for their children.

For families in poverty, these challenges can be formidable. Sometimes, when basic necessities are lacking, parents must place top priority on housing, food, clothing, and health care. Educational toys, games and books may appear to be luxuries and parents may not have the time, energy, or knowledge to find innovative and less-expensive ways to foster young children's development.

In families with above-average incomes, parents often lack the time and energy to invest fully in their children's preparation for school, and they sometimes face a limited array of options for high-quality childcare both before their children start school and during the early school years. Families with low socio-economic status often lack the financial social and educational supports that characterise families with high socioeconomic status. Poor families also may have inadequate or limited access to community resources that promote and support children's development and school readiness. Lower socioeconomic status can be a factor in poor health. Studies have shown mental health to be impaired due to the daily stress due to unemployment, economic displacement and housing dislocation, including homelessness. In addition, it is more difficult to provide healthy hood, safe communities and clean work environments in areas of lower socioeconomic status.

Research shows that socioeconomic status is associated with a wide array of health, cognitive and socio-emotional outcomes in children, with effects beginning prior to birth and continuing into adulthood. A variety of mechanisms linking socioeconomic status to child well being have been proposed, with most involving differences in access to material and social resources or reactions to stress-inducing conditions by both the children themselves and their parents.

For children, socioeconomic status impacts well being at multiple levels, including both family and neighbourhood. Its effects are moderated by children's own characteristics, family characteristics and external support system. It is well known that there is a strong relationship between mental retardation and low socio-economic status (Blair and Scott, 2002). They postulated further that whether factors associated with poverty such as limited access to health care or the lack of a supportive environment put these children at great risk for mental retardation is not known but the relationship is clear.

The basic sequence of fetal development clearly is not different, whether children are born to poor mothers or to middle-class mothers. But many of the problems that can affect prenatal development are more common among low socio-economic group. Hartwell (2002) stated that low socio-economic status is related to the incidence of mental retardation and premature babies. For example, the father's occupation has been related to substantial difference in the incidence of mental retardation and infant mortality. Mental retardation has also been associated with ethnicity, although this is probably related to low socio-economic status more than ethnic difference. The percentage of infants born with mental retardation among white Americans is consistently about half the percentage born in non-white Americans (WHO, 2004). 51% of all non-white (low income is more prevalent as a group) have birth complication of some sort whereas only 5% of the white upper class births are so affected (U S Bureau of Census, 1982).

Studies have also found definite links between mental retardation and a number of medical problems chiefly epilepsy, cerebral palsy, blindness and deafness but it is not known for sure whether these diseases and mental retardation have common genetic cause or whether the mental retardation is the by-product of an underlying medical problem (Herbst and Baird, 1982).

In one of such studies, conditions such as seizures and cerebral palsy were strongly connected with mental retardation, though there was no specified reason for this. It was suggested that it could be that some undetected trauma or infection caused both the medical condition and the mental retardation. Another possibility speculated was that in some cases, complications from seizures caused the mental retardation. A third possibility was that a single genetic cause may be responsible for both mental retardation and the accompanying condition. Finally, the answer, it was opined, may

be a combination of two or even all three of the aforementioned possibilities (Sutton, 2001).

Heredity plays an obvious part since the chance for retardation is greater for children born into families with parents who are significantly intellectually limited. Equally apparent is the environmental influence upon the incidence of mental retardation. The environmental factors include cultural, familial or sub-group attitudes; indifferent maternal care; parental rejection; sensory deprivation; and institutionalisation.

In the vast majority of cases, the degree of influence of the familial or environmental factor cannot be determined. Persons with mental retardation with cultural/familial etiology comprise nearly all of the mild and most of the moderate categories. Although the causes are non-specific, it must be emphasised that this etiological group comprises the large majority of the population with mental retardation. There are, however very few individuals with mild mental retardation in institutions. Those persons with mild mental retardation in institutions usually have severe medical or behavioural complications. As previously stated, as other etiological factors, especially genetic, are better recognised, the percentage of persons with mental retardation in this category may decline.

According to World Health Organisation (WHO) (2004) a large sections of the populations in developing countries still believe that mental retardation is caused by bad deeds in the previous life of parents. Some communities perpetuate the myth that if one tries to remedy the illness or take treatment, the suffering will be repeated in one's next life. It is also assumed that mental retardation is caused by cultural practices engaged in by pregnant and lactating women not following restrictions on food and that mental retardation is infectious.

Birth Order

Birth order refers to whether one is perhaps the first child born in a family or may be one of many, or maybe even the last. Many researchers think that where one is, in relationship to his or her brothers and sisters helps influence how he or she develops. Birth order is one way of getting some good clues as to why people are the way they are. There is no way to always accurately predict how one person may turn out; people are all too different, complex and unique.

Birth order is not a simple system stereotyping all first-borns as having one personality, with all second-born another and last-born kids a third. Instead, birth order is about tendencies and general characteristics that may often apply. Other things also influence birth order. Spacing is an obvious factor. Whether there is a gap of five or more years between children, it often means that a second family has begun. So a child born third in a family constellation but whose next order sibling is seven years older, may develop first born tendencies. This does not mean he would not have any characteristics of a middle or last-born child, but is likely to also be quite “adult”-conscientious and exacting –because he had so many older models.

Sex of the child is another way birth order characteristics can change. The first born of any gender is more likely to take on first-born characteristics. Sometimes work or chores are assigned based on sex. In a very traditional home the oldest male usually gets the “manly” chores such as setting the lawn, digging weeds, hauling trash, and helping the father. His younger sister would be assigned the “mother’s helper jobs”: ironing, house cleaning, doing the dishes and so on. In larger families, when sex differences create someone “special” (like three boys and one girl) it can put pressure on the children immediately above or below that special person.

Leman (1985) stated that the physical make up of the children can turn birth order upside down or at least tilt it a bit sideways. Examples include two closely spaced boys with the youngest being significantly bigger; a first born girl who is extremely pretty and a second-born girl who is extremely plain; a child in any birth order who has a serious physical or mental disability. Twins are often an interesting mix of competitor or companion. The “firstborn” often takes the assertive role of leader while the “second-born follows along. In a family constellation, twins are bound to cause pressure especially on any children born after them.

Birth order has been studied for many years as a factor that plays a part in an individual’s intelligence. In general, not much has been looked into concerning birth order and education. Firstborns and children born later have many circumstances, within the family unit, that affects the development of strengths and weaknesses and ultimately influence their personality traits. Parents are often overly anxious about their first child and may be more restrictive with them than with later children (Eisenman, 1992). Birth order theory holds that children develop their behavioural patterns largely as a result of their position within their family (Morales, 1994).

Intellectually, firstborns have been found to have larger receptive vocabularies than later born children, but that later born may have better conversational skills than firstborns (Coates & Messer, 1996). Social interactions experienced by later born children are characterized by less supportive and more directional communication than firstborn. These less supportive interactions may result in later born children having smaller vocabularies (Coates & Messer, 1995). The study of interactions among intellectual performance, family size, and birth order according to Morales (1994), predicts an additive decrease in intellectual performance with increasing family size and birth rank. Studies have shown that firstborns hold memberships in more organisations and demonstrated a significantly higher GPA than later borns (Nelson & Harris, 1995). As such, education would be directly affected by these achievements.

Firstborns tend to be different, some of the time, than children born into other birth orders. This may be due to the early parental treatment received. The firstborn has more time alone with the parents than the later born children, by virtue of having no siblings until the second child is born. The early adult oriented styles learned when they had only the parents and no other siblings would account for the anxiety, achievement, and creativity of some firstborns (Eisenman, 1992). Cooperative learning groups may be one of the best methods teachers can employ to promote the social and psychological development of their students. Birth order theory provides a frame of reference with regard to student's social and psychological characteristics. Therefore, a blending of the information from both of these areas, cooperative learning and birth order theory, provides teachers with the knowledge necessary for developing an effective learning environment conducive to a student's total growth and development (Morales, 1994).

The bias in favour of or against a particular birth position seems to be linked to personality traits found distinctive to that position. Regardless of sex, the firstborn was viewed as the most favoured birth position, followed by the middle, youngest, and only child positions (Nyman, 1995). Problems of adjustment are associated with each of the ordinal positions, and birth order concepts offer teachers a psychological frame of reference to assist them in understanding the children in their classroom. Sibling rivalry, self-esteem, competition, peer relationship and fear of failure are all concepts relevant to a child's birth order (Romeo, 1994). Differences in family size

and birth order were found in adolescent's achievement and perceptions of parenting style and parental involvement but not in parents' perceptions of parenting.

Professionals tend to look at the parents as the primary influence when it comes to different child behaviour. These generalisations may be based on current trends and perceptions about families, when actually some are research based and some are not may influence achievement. Exploration of different variables, such as social relationships with peers and relationships with siblings, may be at least partly responsible for differences in achievement that have been found (Sputa & Paulson, 1995).

Findings that suggest association of psychological birth order and measures of lifestyle but not actual birth order with measured lifestyle, suggest that psychological birth order may be more descriptive of individuals in defining life positions than is actual birth order. The variables are related to the way in which people make meaning of their worlds. These constructs are reflective of early decisions about one so that psychological birth order characteristics should show a pattern of relationships with measures lifestyle characteristics (White, Campbell & Stewart, 1995).

Children's perception of the influence of parental and sibling responsiveness and support differs by birth order. For firstborn children, second born sibling warmth was a stronger predictor of self perceptions than maternal warmth and responsiveness. Mothers' influence on firstborn children's self perception was mainly indirect and occurred by influencing second born siblings' warmth, which then influenced the firstborn children's perceptions of intellectual and physical abilities, self-assurance, and happiness (Barnes, 1995). The order of a person's birth has a lasting impact on personal development. Studies have indicated a strong relationship between birth order and perception of favoritisms, where there is clearly tendency for favoritism to be perceived from the opposite-sex parent (Chalfant, 1994). Findings about the connections between differential treatment and children's sibling relationship also were consistent in showing equal treatment by both parents has the most positive correlates (McHale, 1995). Kristensen and Bjerkedal (2007) stated that the interest in the relation between birth order and intelligence elated back to Sir Francis Galton in 1874. Galton found more firstborn sons in prominent positions than what he attributed to chance. This was the start of numerous studies; one of the most influential was a science publication showing a negative association birth order and intelligence in young Dutchmen. Since then, sociologists, psychologists and demographers have

proposed several explanatory models. The most influential models have emphasised explanations relating to interactions within the family and favourable conditions for intellectual stimulations for low-birth order children. Several researchers have claimed that the relationship between birth order and intelligence is false, confounded by factors relating to family size. Families with low intelligence children tend to be large, and the relation with birth order is an artifact when comparisons between families are made (Rodgers, Cleveland, van der Oord and Rowe 2000).

Another model claims that the relation is explained by prenatal or gestational factors. One hypothesis suggests an effect of maternal antibody attack on the fetal brain. Maternal antibody levels tend to increase by higher birth orders in a suggested mechanism parallel to rhesus incompatibility and erythroblastosis. It has been shown that children of mothers with antimmune disease have an increased risk of learning disabilities (Ross, Sammaritano, Nass and Lockshin, 2003), but there are no empirical data to support immunoreactivity in explaining the birth order effect.

There are some children who have different social and biological ranks in the family. One example is children who grow up in families with decreased elder siblings. A social interaction effect within the family would result in higher scores for a second born who had lost an elder sibling than for subjects ranked second both socially and biologically. On the other hand, if the birth order effect was gestational, second born children who are raised as the eldest would have intelligence score equal to those of other second born children.

Cultural Beliefs

Before the late 1950's culture was defined in terms of patterns of behaviour and customs (Sleeter 1990) Good enough (1984) defined culture as a way of perceiving, believing, evaluating and behaving. Spradley and McCurvy, (2000) focused on the acquired knowledge that people use to interpret experience and to generate social behaviour. It has also been described as the ever-changing values, traditions, social and political relationships, and a worldview shared by a group of people bound together by a number of factors that can include a common history, geographic location, language, social class, and or religion (Neto 2002 in 'O' Connor 2003). Cultural development some argue is never static but evolving.

Olatawura (2005) defined culture as the complex pattern of learned behaviour, values, and belief systems shared by members of a designated group such as a tribe.

These patterns of learned behaviour are generally transmitted through succeeding generation, creating a blue print for experience, thought, and action. The main agent for this transmission as in other cultures all over the world is the family. In Nigerian culture, however, it is the whole extended family that has major influence over a child's development, contrasting with the nuclear family system of the western European cultures Meaney (2004).

One important point about cultural considerations is that culture pervades peoples thinking about disability, defining the ways intellectual ability is dressed, influencing the methods through which intervention and services are provided and determining the attitudes and behaviours with which the society respectively perceive and act with respect to people with disabilities (Meaney 2004).

Westbrook, Legge and Pennay (2003) opined that an emerging literature has developed in the last two decades, particularly concerning differences among cultures in attitudes towards people with mental retardation. Likewise, there is recognition of the importance of language as the communication aspect of cultures and therefore correlated with the way disabilities are perceived by society at large and among cultures.

Culture is certainly an important factor in how families cope with having a member with mental retardation and eventually adapt through modification of individual and familiar behaviours. A rich literature has developed during the last 15 years concerning the stress experienced by families in multiple cultures when a family member has a developmental disability such as mental retardation (Meany, 2004). Data collected have been able to demonstrate substantial evidence of the influence of cultural values and behaviours in adjustment of families.

The word culture then is one that brings with it a number of interpretations and is no longer being seen narrowly (O'Connor 2003). In this sense, culture is more than a singular experience (that is ethnicity) but rather the outcome of a number of key elements (race, ethnicity, gender, disability) that are pertinent to an individual's identity. Culture can be viewed as something very personal as well as something more persuasive that affects entire groups. Much of this way of understanding culture arises in the renewal of interest in concepts such as cultural pluralism, cultural diversity and multiculturalism. Cultural customs and practices can influence the growth and development of an unborn child. Nutritional practices and child rearing practices may

influence the rate of growth and intelligence quotient of infants (Ball and Bindler 2006).

According to O'Connor (2003), culture affords us ways of seeing the world. The word culture then is one that brings with it a number of interpretations and is no longer being seen. In another perspective, Burns (2003) identified different factors that can be implicated in the etiology or cause of mental retardation. She outlined three major categories of etiology of mental retardation; these are psychosocial etiology, polygenic etiology and biological defects.

The psychosocial etiology category includes things such as impoverished environment, substandard living arrangement, low socio-economic status of families, families in which there is a lot of emotional problems, a lack of environmental stimulation, which can be translated into sensory deprivation. There is an increased rate of prematurity, which seems to have a higher incidence in low socio-economic status mothers, and may indeed be due to poor matrimonial status of the mother during her childhood or something that comes back to influence the effect on her offspring later on in her life. Burns noted that there is no pathological etiology known for mild mental retardation, and that looking at the characteristics of families in this group, there is at least one parent or sibling who also has mild mental retardation. Such families are found in the lower socio-economic status of the society. This kind of mental retardation becomes apparent or detectable during the school years when children are challenged with the kind of cognitive tasks that happen in schools.

These children usually get by very well in early childhood, and fit into their environment, but then they cannot handle school-like competencies. Interestingly enough, in many cases, this label of mental retardation disappears in adulthood because they are not being challenged with the kinds of cognitive structures that are acquired in school, so they are able to fit in with a segment of society where they can have job and get along. She then suggested that early intervention programme for children from deprived homes will be very successful in preventing manifestation of mental retardation. Also, an improved standard of living for all families in poverty will bring a quick decrease in the amount of mental retardation that fits this category. About 12 % of all cases of mental retardation fall into the category of polygenic etiology. The etiology of polygenic mental retardation is due to the additive effect of many genes, each of which contributes in a quantitative way to the intellectual or cognitive potential.

The levels of mental retardation that can be explained with polygenic etiology are mostly mild mental retardation. Usually the family members cluster around a low average level of intellectual functioning and it is not limited to low socio-economic families. This type of mental retardation is not detectable until the school years. This is because the level of mental retardation is mild, and these children fit in very well until they start school and are challenged by intellectual functioning.

The polygenic model of inheritance of intelligence quotient is based on a very sound, but somewhat complex model of quantitative genetics, which explains the action multiple genes, which contribute an additive effect to characteristics, which can be measured along the continuum. Some of the examples in which quantitative inheritance is used to understand what is going on are things like skin colour and height.

The third category identified by Burns (2003) is the biological deficits. She split this category into two namely; the genetic causes and environmental/medical causes. Genetic causes account for between 12 and 30 percent of all mental retardation. Specific gene disorders that fall into this category are phenylketonuria (PKU), Tay Sachs diseases, others are chromosomal disorders. In this case either an extra chromosome is present or a chromosome is missing or a piece of a chromosome is missing or there are extra amounts. Sometimes pieces of chromosomes twist around and get inverted and are in different places, and that can cause problem, the kinds of condition like Down syndrome, Klinefelters syndrome, cri du Chat or the cat cry syndrome.

The levels of mental retardation in conditions as such Down syndrome, Klinefelters syndrome are primarily moderate, severe, and profound. No family, depending upon social or economic class is excluded from having a contribution or being involved in this group of etiologies. It crosses all social and economic classes. However, there are no parents or siblings with mental retardation within these families. It is usually a situation where a single incidence happens within the family. In single gene disorders there is a recurrence risk but in most of these situations recurrence for a second time within families is rare. These conditions are almost always detectable at birth because usually these conditions carry with them characteristics that are identifiable either from some form of dismorphological or some biochemical characteristics and these are usually identified shortly after birth. Certainly mental retardation in these cases is evident before children start school.

Lastly according to Burns, the other groups of biological defects are the ones with environmental/medical etiology. Burns stated that of the approximately 25 % of cases of known causes of mental retardation, about half or somewhere between 12 and 15 % are known to be due to environmental /medical insults.

The etiology involved is a whole variety of biological and medical problems. Things that result from birth trauma, lack of oxygen either during pregnancy or at birth time, or even after birth, infections and intoxicants, which can affect the development of a fetus parentally. These can happen during the birth process and affect the individual around the time of birth, or there might be indication or accidents that occur after an individual is born perfectly healthy. Certain vaccines and infection can also cause some kinds of physiological reactions, which may result in mental retardation.

The levels of mental retardation in cases mentioned above are mostly moderate, some are profound and very few are mild. This also cuts across all socio-economic and educational classes, although it has a higher predominance in low socio-economic status families due to poor prenatal care, a higher infection rate and living in poor polluted areas. Mental retardation associated with these conditions is usually at birth.

During the prenatal period certain maternal conditions can cause mental retardation. Older mothers are at higher risk of having babies with Down syndrome. Mothers who are under the age of 16 also have a higher risk of having babies with these kinds of problems possibly due to the immaturity of the mother's own physiology (Burns 2003). Certainly the health of the mother, including nutritional status, even the nutritional status of the mother during her own childhood as well as nutritional status during pregnancy can lead to prematurity and prematurity does carry with it a lot of the possible conditions which may result in damage to the fetus.

Another concern about maternal conditions that predicts mental retardation is the frequency of pregnancy. Burns (2003) opined that mothers should allow approximately two years between pregnancies, simply for the possibility of maternal rebuilding of her own physiological status. Guastello and Guastello (2002) stated that birth order plays an important role in the family. Most importantly is the spacing of children.

From another perspective, Gaulden (1992) opined that the only etiological factor that has been established for the occurrence of Trisomy 21, which causes down syndrome is advanced maternal age. Studies showed that 90 % of down syndrome

children received their extra chromosome from their mother. Hook (2000) also stated that the likelihood that a reproductive cell will contain an extra copy of chromosome 21 increases dramatically as a woman ages. Therefore, an older mother is more likely than a young mother to have a baby with mental retardation.

Parental Age

Age – related shifts in child bearing pattern may have implication on both immediate and longer term pregnancy outcomes. Babies conceived by women at the lower or upper ends of the age spectrum are at higher risk for a number of adverse outcomes, including fatal death, preterm birth; low weight certain types of birth defect and mental retardation. In addition, surviving children of very young or very old mothers may be at disproportionate risk another problem including a low level of cognitive functioning a child being born to a teenaged mother can be hypothesised as a risk factor for diminished cognition ability because of the competing nutritional needs of mother and fetus the mother lack of emotional maturity the lack of a stable family structure an inadequate care environment and disadvantages associated with a lack of economic resources according to Williams and Decoufle (2007) there are some available data on the relation between a woman's age at delivery and her child's level of cognitive functioning. Among children who have reached school age research findings have constitutently shown a positive linear correlation between a mother's age at delivery and her child's measured mental ability.

Furthermore, lower achievement test scores and a higher frequency of retention in kindergarten or first grade have been reported among the children born to teenaged mothers. However, these associations became weaker or disappeared altogether when confronting factors such as maternal education, socio-economic status and family size were taken into consideration.

Older maternal age is associated with meiotic non- disjunction and reduced rates of recombination resulting in aneuploidy. The most commonly detected aneuploidies are those compatible with fetal survival they result in genetic syndromes characterized by multiple birth defects and mental retardation, that is, trisomy 21, 18 and 13 older maternal age is a well known risk factor for Down syndrome (trisomy 21), the most common genetic cause of mental retardation.

Further, several reports have shown that children of mothers aged 40 years or older at delivery have some what lower mental test scores than children of women in their thirties, which raises the question of whether other congenital anomalies that affect the central nervous system maybe associated with older material age (Williams and Decoufle 2007) Conceivably, less commonly detected or more subtitle material age dependant aberrations in genetic recombination events may exist and may adversely affect embryogenesis. If any embryologic development and therefore the cognitive capabilities of children of older mothers may be diminished.

The likelihood that a woman under 30 who becomes pregnant will have a baby with Down syndrome is less than one in 1000, but the chance of having a baby with Down syndrome increases to 1 in 400 for women who become pregnant at age 35. The likelihood of Down syndrome continues to increase as a woman ages, so that by age 42, the chance is 1 in 60 that a pregnant woman will have a baby with Down syndrome, and by age 49, the chance is 1 in 12. According to Hook (2000), using maternal age alone will not detect over 75 percent of pregnancies that will result in Down syndrome.

Relationship of Down syndrome Incidence to Mother's Age

| Mother age | Incidence |
|------------|--------------------|
| Under 30 | Less than 1 in 100 |
| 30 | 1 in 900 |
| 35 | 1 in 400 |
| 36 | 1 in 300 |
| 37 | 1 in 230 |
| 38 | 1 in 180 |
| 39 | 1 in 135 |
| 40 | 1 in 105 |
| 42 | 1 in 60 |
| 44 | 1 in 35 |
| 46 | 1 in 25 |
| 48 | 1 in 16 |
| 49 | 1 in 12 |

Source: Hook E. G., Lindsjo A; Down syndrome in Live Births by Single Year maternal age

Laxova (1989) found that there was a significant correlation between increase maternal and paternal age and the birth of infants with Down syndrome. However, when he patterned his analysis after that employed by Swall Wright to assess the cause of polydactyl in guinea pigs (Wright, 1926), Penrose discovered that there was a highly significant partial correlation between maternal but not paternal age and the occurrence of Down syndrome. To confirm this further Penrose used regression analysis to compare paternal and maternal ages as well as mean maternal and paternal ages at the time of birth of Down syndrome and other off springs within families.

After correction for paternal age he found no significant difference between the means of observed and expected paternal ages of other offspring, whereas the difference of the same parameters between those observed and expected from maternal age was six times the standard error. Penrose showed that birth order, parity and length of interval between pregnancies were not significant etiological factors.

Rives, Langlois, Bordes, Simeon and Maco (2002) have also identified parental age to be the most important etiological factor implicated in human trisomy formation. Advanced maternal age is a predisposing factor for most autosomal trisomies but maternal age effect show considerable variation among chromosomes. Data on human trisomy from spontaneous miscarriages and offspring suggested that non-disjunctions at maternal meiosis 1 were the most common cause of trisomy for acrocentric chromosomes. Two possible mechanisms may be responsible for non-disjunction of acrocentric chromosomes, premature division of sister chromatins and non-disjunction of bivalent chromosomes. The second mechanism increases with maternal age.

Down syndrome was named after John Langdon Down, the first physician to identify the syndrome. Down syndrome is the most frequent genetic cause of mild to moderate mental retardation and associated medical problems and occurs in one out of 800 live births, in all races and economic groups. Down syndrome is a chromosomal disorder caused by an error in cell division that results in the presence of an additional third chromosome 21 or trisomy 21. (National Institute of Child Health and Human Development, 2004).

The structure and function of the human chromosome must be understood so as to know why Down syndrome occurs. The human body is made of cells; all cells contain chromosomes, structures that transmit genetic information. Most cells of the human body contain 23 pairs of chromosomes, half of which are inherited from each

parent. Only the human reproductive cells, the sperm cells in males and ovum in females, have 23 individual chromosomes, not pairs. Scientists identify these chromosome pairs as the XX pair, present in females and the XY pairs, present in males and number them 1 through 22.

When the reproductive cells, sperm and ovum, combine at fertilization, the fertilized egg that results contains 23 chromosome pairs. A fertilized egg that will develop into a female contains chromosome pair 1 through 22, and the XX pair. A fertilized egg that will develop into a male contains chromosome pairs 1 through 22 and the XY pair. When the fertilized egg contains extra material from chromosome number 21, this results in Down syndrome. Three genetic variations can cause Down syndrome in most cases, approximately 92% of the time Down syndrome is caused by the presence of an extra chromosome 21 in all cells of the individual. In such cases, the extra chromosome is repeated in every cell. This condition, in which three copies of chromosome 21 are present in all cells of the individual, is called Trisomy 21.

In approximately 2-4% of cases, Down syndrome is due to mosaic trisomy 21. This situation is similar to simple trisomy 21, but in this instance, the extra chromosome 21 is present in some, but not all, cells of the individual. For example, the fertilized egg may have the right number of chromosome, but due to an error in chromosome division early in embryonic development, some cells acquire an extra chromosome 21 thus, an individual with Down syndrome due to the mosaic trisomy 21 will typically have 46 chromosomes (including an extra chromosome 21) in others. In this situation, the range of the physical problems may vary, depending on the proportion of cells that carry the additional chromosome 21.

In trisomy 21 and mosaic trisomy 21, Down syndrome occurs because some or all of the cells have 47 chromosomes, including three chromosome 21. However approximately 3-4% of individuals with Down syndrome have cells containing 46 chromosomes, but still have the features associated with Down syndrome. In such cases, material from one chromosome 21 gets stuck or translocated into another chromosome, either prior to or at conception. In such situations cells from individuals with Down syndrome have two normal chromosomes 21, but also have additional chromosome 21, material on the translocated chromosome. Thus, there is still too much material from chromosome 21, resulting in the features associated with Down syndrome. In such situations, the individual with Down syndrome is said to have translocation trisomy 21.

Most of the time, the occurrence of Down syndrome is due to a random event that occurred during formation of the reproductive cells, the ovum or sperm. Down syndrome is not attributable to any behavioural activity of the parents or environmental factors. The probability that another child with Down syndrome will be born in subsequent pregnancy is about 1 percent, regardless of maternal age.

For parents of a child with Down syndrome due to translocation trisomy 21, there may be an increased likelihood of Down syndrome in future pregnancies. This is because one of the two parents may be a balanced carrier of the translocation. The translocation occurs when a piece of chromosome 21 becomes attached to another chromosome, often number 14, during cell division. If the resulting sperm or ovum receives a chromosome of 14 (or another chromosome), with a piece of chromosome 21 attached and retains the chromosome 21 that lost a section to translocation, then the reproductive cells contain the normal or balanced amount of chromosome 21. While there will be no Down syndrome associated characteristics exhibited, the individual who develops from this fertilised egg will be carrier of Down syndrome. Genetic counseling can be sought to find the origin of the translocation. However, it is important to realise that not all parents of individuals with translocation trisomy 21 are themselves balanced carriers. In such situations, there is no increased risk for Down syndrome in future pregnancies.

Researchers have extensively studied the defects in chromosome 21 that cause Down syndrome. In 88% of cases, the extra copy of chromosome 21 derived from the mother. In 8% of the cases, the father provided the extra copy of chromosome 21. In the remaining 2% of the cases, Down syndrome is due to mitotic errors, and error in cell division which occurs after fertilisation when the sperm and ovum are joined.

Girirajan (2009) suggested a partial correlation between advanced maternal age and the risk of having a child with Down syndrome. Using a regression analysis, Girirajan compared the paternal and maternal ages of Down syndrome offspring. After regressing out the paternal age effect, the difference between the observed

Fragile X syndrome the most common case of inherited mental retardation, is seen in approximately one in 1,200 males and one in 2,500 females. Males with fragile X syndrome usually have mental retardation and often exhibit characteristic physical features and behaviour. (Hagerman and Silverman, 1991; Warren and Nelson, 1994) Affected females exhibit a similar but usually less severe phenotype.

The diagnosis of fragile X syndrome was originally based on the expression of a foliate sensitive fragile site at XqS 27.3 (FRAXA) induced in cell culture under conditions of foliate deprivation. Cytogenesis analysis of metaphase spreads demonstrates the presence of the fragile site in less than 60% of cells in most affected individuals. The cytogenesis test has limitation especially in testing for carrier status; it exhibits a high degree of variability between individuals and laboratories.

Hagerman and Silverman (1991) found that the fragile X gene was characterized to contain a tenderly repeated trinucleotide sequence near its 5th end. The mutations responsible for fragile X syndrome involves expansion of this repeat segment. The number of trinucleotide sequence repeats in the genes of the normal population varies from six to approximately fifty. There are two main categories of mutation, permutations of approximately 50 - 200 repeats and full mutations of more than approximately 200 repeats.

Males and female carrying a permutation are unaffected. Male carriers are referred to as “normal transmitting” males and they pass on the mutation relatively unchanged in size, to all of their daughters. These daughters are unaffected, but are at risk of having affected offspring. Variable clinical severity is observed in both sexes. Most but not all males with a full mutation are mentally retarded and show typical physical behavioural features. Of females with a full mutation, approximately one third is mentally retarded.

The pre-natal world of the fetus while in the mother’s womb is sustained in a well protected environment although not immune to the influence of its external environment. Fetal alcohol syndrome was first observed by Lemoine (1968) and was later coined by Jones and Smith (1973) in Seattle as the Fetal alcohol syndrome (FAS).

In a broad sense fetal alcohol syndrome may be viewed as a repercussion of an external environmental influence on the internal psychological environment of the developing fetus. Alcohol acts as teratogen (derived from the Greek word tera, meaning monter) an agent, which when prenatally exposed can cause serious risk to pre and post natal human development (Caleekal, 1996).

Fetal alcohol syndrome lies at the extreme end of the continuum of alcohol effects on the fetus with heavy persistent maternal alcohol consumption during pregnancy contributing most significantly to the full blown syndrome. Clinically, three areas are affected (1) prenatal and/or postnatal growth retardation such as infants

being shorter in length and less in weight (2) central nervous system (CNS) damage such as permanent and irreversible brain damage, learning and behavioural disorders, deficits in memory and attention, hyperactivity, speech and language delays, poor coordination (3) head and facial abnormalities such as small head circumference and abnormally small eyes.

Children diagnosed as having fetal alcohol syndrome have deficits in some or one of the above three areas of FAS and their mothers were found to drink smaller daily amounts of alcohol than mothers who had Fetal Alcohol Syndrome offspring. FAS were previously estimated as the third most frequent causes of mental retardation after Down syndrome and certain neural tube effects (Caleekal, 1996). Current prevalence estimates of Fetal Alcohol Syndrome may be the leading most common, preventable cause of mental retardation in North America and Europe.

In France, Sweden and North America, prevalence of FAS is one per 750 live births per year (Goodstadt and Caleekal, 1994). It is expected that FAS children will have some degree of mental impairment ranging from minimal brain dysfunction to severe mental retardation. For every child identified with FAS, there are several others who are affected by alcohol exposure but who lack the full set of characteristic of FAS.

Fetal Alcohol Effect (FAE) is more common than fetal alcohol syndrome and is estimated to be 3-10 times that of those diagnosed with full fetal alcohol syndrome. It must be noted that although many other factors can also cause low birth weight, alcohol is said to account for 2% of the decreased birth weight associated with prenatal alcohol exposure. Another example of FAE might be a case where there is some central nervous system damage, with signs of speech problems or attention deficit disorder, but no apparent facial and head abnormalities. The lack of specific criteria for FAE makes estimation of incidence statistics and diagnosis difficult.

Studies indicate differential susceptibility to FAS occurring on both a racial and genetic basis. According to Caleekal there are ethnic differences in the metabolism of alcohols and there is genetic control over the individual variability on the rate of alcohol metabolism. In comparison to Caucasians, the Chinese, Native American and Japanese have a higher rate of alcohol metabolism.

There is also evidence of racial difference in acute reactions to ethanol in terms of alcohol sensitivity symptoms such as dizziness and hangovers. These were found to be greater among Orientals of Mongoloid heritage and American Indians than in Caucasian subjects when exposed to a mild dose. In this respect it is possible, that

fetal susceptibility to alcohol may also be dependent upon maternal racial and genetic composition interacting with consumption patterns. The basis for these differences remains to be determined.

2.2 Empirical Studies

Abasiubong Obembe and Ekpo (2008) carried out a study on the opinions, beliefs and attitudes of mother to mental retardation in Lagos, one hundred and twenty six mothers of children with mental retardation were invited. 106 that is 84.1% of them decided to take part in the study, the mean age was 40.0 ± 6.6 years, 37 (35.0%) attributed the cause of mental retardation to evil spirits or witchcrafts 19(17.9%) to diabolical powers of father or mother in-laws, 14(13.2%) blamed spouses and 11(10.4%) believed in natural causes while 5(4.7%) viewed it as due to their faults. Majority, 79(74.5%) still preferred to have more children in spite of having children with mental retardation; 9(8.5%) of mothers exhibited depressed feelings, 3(2.8%) thought of doing away with, that is, killing the children with mental retardation.

Shevell, Majnemer, Rosenbaum and Abrahamowicz (2000) carried out a study to determine the etiologic factors of young children with mental retardation. All children below 5 years referred over 18-month period for initial evaluation of suspected mental retardation were enrolled. Diagnostic yield was ascertained after the completion of clinical assessments and laboratory investigations requested by the evaluating physician. Ninety-nine children (71 boys) were found to have mental retardation, 96% had mild or moderate mental retardation. An etiologic diagnosis was determined in 44. Four diagnoses accounted for 34 of 44 (77%) of the diagnoses made.

The presence of co-existing autistic traits was associated with significantly decreased diagnostic yield (0/19 vs. 44/80, $P < .0001$), whereas specific historical features such as family history, toxin exposure and prenatal difficulty; 23/32 vs 21/67, $P < .0002$) and findings on physical examination such as dysmorphism, microcephaly and focal motor findings; 35/48 vs. 9/5, $P < .0001$) were significantly associated with identifying a diagnosis. Multiple logistic regression analysis identified antenatal toxin exposure, microcephaly, focal motor findings and the absence of autistic traits as significant predictor variables for the identification of an etiology. It was then

concluded that an etiologic diagnosis is often possible in the young child with mental retardation.

Also in another etiological survey carried out by Laxova, Ridler and Bowen Bravery (1997) in Hertfordshire one hundred and forty –six children (87 boys and 59 girls) were ascertained out of a total population of 46,960, with a prevalence of 1 in 320 or 3.1 per 1,000. Approximately one third (47) had Down syndrome, 1 per 1,000 population. It was possible to establish a diagnosis in a further 45 cases, which included one additional case of autosomal chromosome abnormality and 7 each of autosomal dominant recessive and X-linked conditions; 17 were associated with presumed multifactor etiological factors; in 6, the condition was thought to have been caused by an environmental agent. It was not possible to establish a cause in the remaining 54 cases. Recurrence risks of severe mental retardation in cases where it was possible to establish a definite diagnosis were discussed and the potential value, for genetic counseling purposes, of categorising such individuals into broad symptomatological groups was suggested.

In Taiwan, Hon Wang and Chuang (1998) did a large –scale cytogenetic study of the determinants of mental retardation in children from special schools and institutions between 1991 and 1996. The screening methods and the identification of subjects with mental retardation consisted of both clinical evaluation, (that is, photographs, questionnaires on family pre-, peri- and post-natal history and hospital records including IQ) and further laboratory studies for diagnosis (that is, standard chromosome analysis and if indicated high resolution banding cytogenetic fragile X study or molecular techniques).

A total of 11,892 persons were enrolled in the study. After excluding the acquired causes of mental retardation such as infections and the sequel of brain insults or the well-known single-gene disorders and other multifactor disease, 4372 (36.8%) cumulative cases were recruited for karyotyping studies according to their phenotypes and medical records.

Abnormal karyotypes were noted in 1889 children (43.2%) of all selected children. Thus the overall incidence of chromosomal aberrations in subjects with mental retardation was estimated as 15.9%. Down syndrome the most common cause of mental retardation accounted for 82.4% of all persons with abnormal karyotypes.

The causes of mental retardation were considered to be prenatal in 55.2% (n=6564) of cases, peri-natal in 9.5% (n=1130), postnatal in 3.3% (n=392) and unknown in 32.0% (n=3805) of cases. Two large groups were classified:

(1) Serious mental retardation (37%), including profound severe and moderate categories and (2) mild mental retardation (63%). The causes (pre,-peri- and postnatal and unknown) in these two population were 70%, 10.5%, 5.4% and 14.1% and 46.5%, 8.9%, 2.1% and 42.5% respectively. Genetic causes accounted for 38.5% (n= 4578) of all cases in the study, including 1557 with Down syndrome 233 with fragile-X syndrome 199 with other various chromosomal abnormalities (that is unbalanced translocation, supernumerary makers and structural rearrangement), 238 with a defined or presumed single-gene defect, and 98 with a recognized contiguous gene syndrome (Prader Willi 56; Angelman 34; Wilhains, 5 and Kallmann 3). 2120 cases had familial mental retardation.

Multiple anomalies of undefined pattern but without chromosomal aberration, infantile autism mental retardation of normal phenotype or family history were of the other categories. Patients with a single-gene disorder or chromosomal aberration especially those with unbalanced translocated or rearranged chromosomes had genetic counseling and family studies.

An etiology and pathogenesis of mild mental retardation (IQ 50-70) were analyzed by Hagberg, Hagberg, Lewerth and Lindberg (1991) in an unselected series of 91 Swedish school children with this condition. The cause was considered to be prenatal in 23% including 5% genetic, prenatal unknown in 10% and alcohol fetopathy in 8%. A perinatal cause mainly asphyxia combined with fetal deprivation was found in 18% and a postnatal in 2%. In 55% the cause was untraceable. Half of these latter children had close relatives with below average intelligence. Exceptionally low birth weights and lengths were revealed among the 8% with alcohol fetopathy. Neurological abnormalities were found in 43% of the children, epilepsy in 12% cerebral palsy in 9% and a clumsy child syndrome in 23%. Psychiatric disturbances were present in 31%.

The types and multitudes of predisposing background factors in mild mental retardation were compared with those in severe mental retardation and with those in the IQ group 71-75. The general conclusion was drawn that in Swedish school children, negative pre and perinatal factors were responsible for a larger proportion of mild mental retardation that has previously been known.

The only well established risk factor for mental retardation is advanced maternal age (Hook 2000) Age specific rates have been well documented. One study found that women who had a reduced ovarian complement (congenital absence of removal of an ovary) were at increased risk of having an infant with mental retardation. This suggests that the increased risk of mental retardation, with increased maternal age may be related to the physiological status of the ovaries or the eggs. (Freeman, 2000; Hassold and Jacob, 2000). Other potential explanation for the association between mental retardation and advanced maternal age include delayed fertilization, changing hormones levels and 'relaxes selection' (Hassold and Jacobs, 2000).

In a few studies advanced paternal age (>49 years) had been associated with increased risk of mental retardation births (Macintosh, 1995) The risk for advanced paternal age had not been large and is considerably diminished with the appropriate adjustment for maternal age. A large number of studies have failed to find evidence of this effect (Stoll, 1990). An association has been found between risk of mental retardation and age of the maternal grandmother at mother's birth Female incisions start in fetal life and non-disjunction in the first meiotic division of a female might be induced during the fetal period especially if her mother is older. Several studies have reported secular trends in mental retardation prevalence; however these trends have not been consistent, with some studies reporting an increase while others a decline (O' Leary, 1996; CDC, 1994).

Zhang (1992) carried out a study and reported that 461 matched cases of mental retardation of unknown aetiology were analysed on the relationship between disease and the parental age, birth order with conditional logistic regression. These cases were accurately diagnosed and selected from the epidemiologic survey of genetic disease of Sichuan, China. The result found that mental retardation of unknown aetiology is related to the age of parent and birth order. The paternal age is the main factor while the maternal age is not of a significant effect and birth order also has no significant effect after readjusting the other factors compared to paternal age group of under 25. There are significant increase of relative risk of age group 30-34 and 45 above, about 1.8 and 2.7 fold increase in univariate analyses and 1.9 and 3.3 fold increase in controlling the maternal age and birth order, a chi square test for trend of distribution of paternal age also indicates a significant close response relationship between increasing risk with age. The significance of result and methods of analysis were discussed.

Singh, Aich, Duttm, Luckman (2001) reported that 36% and 24% of mentally retarded children were respectively first born and second born child. Zahan and Ansari (1999) in their own study stated that first born in 45% of their sample were mentally retarded children. These studies were limited by lack of proper control of population. Despende and Mathur (1991) reported first born being predominated in their mentally retarded population but there was no significant association between the birth order and mental retardation. Thus the controversy regarding the significance of birth order in the causation of mental retardation is still not over.

The study of Singh *et al.*, was conceptualised to probe into the area of research interest. For that purpose they had taken purposive sampling of 100 mentally retarded children from Chetama institution and 50 borderline intelligent children from Ashmuta (a slow learner centre located in Luckman). Age range of these children was 5 to 16 years. Their birth order and other demographic and clinical variables were noted on a specially designed demographic and clinical data sheet. All these children were subjected to a series of psychometric test — Seguin Form Board, Alex Pass A—test, Vineland Social Maturity Scale and Stanford Binet Intelligence Test.

Chapman, Scott and Mason (2002) studied the predictive value of maternal age and education in relation to rates of administratively defined mental retardation in a 3 year birth cohort (N-267, 277). Low maternal education placed individuals at increased risk for both educable mentally retarded and trainable mentally retarded placements. Older maternal age was associated with increased risk of mentally retarded but for individuals with educable mental retardation, this age effect was only seen in the lowest education group. In terms of population level risk, it was younger mothers with 12 years of education or less whose birth was associated with the greatest population of mental retardation. From a public policy viewpoint, children born to mothers with low levels of education are an important group to target for prevention and early intervention efforts.

An association of mental retardation with multiparty tends to disappear when maternal age is taken into account (Chan, 1998; Castilla and Paz, 1994; Haddow and Palomaki, 1994). First born infants may be at higher risk of mental retardation than are those later born, independent of maternal age. However further investigation reported firstborns to be at lower risk of mental retardation (Stoll, 1990). A cluster investigation implicated short pregnancy interval as a risk factor. Other reports had noted that periods of an ovulatory activity followed by conception appear to correlate

with increased occurrence of mental retardation. It is possible that conceptions occurring during the transitional period between an ovulation and the establishment of regular ovulation after childbirth might be more vulnerable to maternal meiotic non-disjunction. Therefore, a short inter pregnancy interval might possibly increase a woman's risk of subsequently bearing a mentally retarded child.

This theory has also been posed as an explanation for the observation that risk of mental retardation is associated with season of child's conception or delivery and season of mother's conception (Chapman, Scott and Mason 2002). However, other investigations failed to identify seasonal variation in the month of last menstrual period or delivery for mentally retarded cases.

Studies have reported an increased risk of mental retardation with higher socio-economic status, although the association may be due in part to maternal age (Vrijheid, 2000). Bashir, Yaqoob, Ferngren, Gustavson, Rydeluis, Ansari and Zaman, (2002) in a study conducted in Pakistan from four areas with different socio-economic condition found mild mental retardation to be higher in a developing country than in developed countries. It also seemed to be related to poor socio-economic condition, as the prevalence in the upper middle class was comparable to figures from developed countries, while the prevalence in children from poor population groups was much higher.

A statistically significant association was identified with fathers working in restaurants. One investigation that examined a variety of paternal occupation reported significantly higher rates of mental retardation with paternal occupation of janitor, mechanic and farm manger/work while another study reported no association between paternal occupation and mental retardation (Doyle, Beral, Botting and Wale, 2000).

Poverty is an underlying condition for many students with disabilities. For example mild mental retardation is consistently reported to be associated with low socio-economic status, and race is highly correlated with socio-economic status (Yeargin-Allsopp *et al.*, 1995). Yeargin Allsopp *et al.*, (1995) suggest that socio-economic status is related to the prevalence of mild mental retardation and may account for some of the disproportionate representation of African American children in that category. The disproportionate representation of African American children in the mild mental retardation category was reduced by nearly half after controlling for sex, maternal age at delivery, birth order, maternal education and economic status.

The research suggests that remaining disparity might be reduced further if other confounding factors, such as maternal intelligence and housing density were controlled. The researchers cited previous studies demonstrating that less advantaged African American children who receive early, structured, and intensive social, medical and educational interventions score higher on average, on tests of cognitive ability than African American children from similar backgrounds who have not received these interventions.

2.2.1 Parents' Perception about the Causes of Mental Retardation

Each culture has its own explanations for why some babies are born with disabilities, how these children are to be treated, and what responsibilities and roles are expected of family members, helpers and other members of the society (Groce, 1999). Understanding and building on a family's cultural interpretations of disability is essential in creating partnerships with parents of children with mental retardation. Parents' beliefs about the nature of disability are related to parent beliefs about and participation in the treatment and intervention.

Lamorey (2002) carried out a study among Euro-American women between the ages of 19 and 25; he discovered responses that uniformly reflected a Westernised biomedical orientation. He discovered that even when these women were specifically urged to relate a family story or folk tale about causes of disabilities, these women could not contribute any non-medical responses other than the fact that disability may be God's will. On the other hand, Lamorey interviewed another group of women representing a variety of cultural backgrounds. Their responses included beliefs that reflect the role of supernatural or cosmic causes, fate, magic and religious beliefs, as well as biomedical reasoning.

According to their responses, if a woman views a person with deformities during pregnancy, she will give birth to a child with deformity or mental retardation. A woman who engages in sexual intercourse during pregnancy produces a child with mental retardation. Their responses also showed that if a woman smells certain food during pregnancy, she must lick her right hand to prevent the food from causing childhood disability; and that God causes disabilities in order to examine a couple's patience.

Several other studies have examined the perception and beliefs of parents about the causes and meanings of their children's disabilities. Cho, Singer and

Brenner (2000) compared the experiences and perceptions of Koreans and Korean American who have children with mental retardation. 80% of the Korean parents attributed causes of disabilities to their own mistakes during the prenatal period and poor parenting attitude while 63% of Korean American parents attributed the causes to a divine plan, that is, God's will for the family and child as well as lack of education during pregnancy.

In Australia, Gray (1995) reported that 25% of the parents of children with mental retardation mentioned religious, magical or psychological reasons for the cause of their child's mental retardation. According to Mardinros (1989) Mexican-American parents of young children with mental retardation perceived the causes of the disability to be either a biomedical etiology, that is, health problem, genetic disease, birth trauma or a socio-cultural view which include marital difficulties, divine intervention, past sins and negative attitude.

In Yoruba society, mental retardation or childhood disability is understood as an indication of family sin that requires punishment by ancestors or gods and the subsequent need for parental atonement (Olubanji, 1981).

According to Lamorey, as this compilation of etiologies of disability shows, there is a wide diversity of attributions that reflect cultural beliefs. With these traditional beliefs, the impact of industrialisation, urbanisation, socio-economic factors, political change, migration and educational opportunities on traditional family systems, and the outcomes can be complex for both parents and practitioners.

Studies have shown that religion plays a significant role in the lives of families whose children are disabled (Rogers-Dulan, 1998). Parents rely on a duality of beliefs as they seek treatment and educational services for their children with mental retardation.

From another perspective, Garcia, Perez and Ortiz (2000) state that parents' perception about the nature of their children's disabilities have been a focal point among different cultural groups – Mexican American, Chinese American, Arab and Jewish communities. Several studies indicate that although parents' perceptions on the nature of a disability may differ from each others to some degree, based on their cultural values, they also hold similar views about the nature of a disability. Common perceptions include seeing their children as developing normally, identifying their children's condition as a temporary or passing condition, and perceiving a disability as God's punishment or, conversely, God's special gift.

According to Garcia et al studies of Mexican American parents of children with mental retardation demonstrated that the parents believe their child's development to be normal. Although children in the studies had mental retardation, the majority of parents did not believe that their children had mental retardation. Some parents defined their child as having both a disability and developing normally. In the exploration of Mexican American mothers' beliefs about their children with language disabilities, Garcia et al state that Mexican American mothers were not concerned about their children's language difficulties. Although they accepted that their children were developing at a different rate, they had expectations that their children would have better communication skills after age of three.

On the other hand, parents in the study carried out by Mardiros (1989) clearly distinguish between being disabled and being normal. Parents believed that each child with a disability was unique, and as such perceived their child's condition as a disability and not an illness or disease.

Garcia et al state that considering the condition of children with mental retardation as a temporary or passing one is a common parental perception among different cultural groups. This perception was reported in several studies among Mexican American parents, Chinese American parents, Druse parents in Arab communities and Jewish oriental mothers. Studies indicate that Mexican American mothers believe that as their children grow older, they would catch up with their peers. Although the parents of Chinese American children with mental retardation and developmental disabilities demonstrate a lack of knowledge and understanding of the diagnoses, they had a tendency to see the disability as temporary problem. Druse parents in Arab communities, who believe in reincarnation and life after death, also consider the disability to be a temporary or passing condition. Jewish oriental mothers were greatly confident in their beliefs that a change would happen suddenly and their child would become normal. One of the mothers from the study stated with reassurance that her daughter will grow and God will be merciful (Stahl, 1991).

Mardiros (1989) states that some parents, especially those coming from strongly traditional cultural groups, perceived God as the agent of disabled children. The studies with Hispanic parents reveal that they view disability as a punishment for wrongdoing, or as divine punishment for sin. The sin, in general, was presumed to be that of the parents. McCallion and Janicki (1997) point out that while some Mexican American parents see their disabled child as God's special gift, others might see the

child as God's punishment. Jewish Oriental mothers also conceptualized the disability as God's punishment (Stahl, 1991).

According to Hanson, Lynch and Wayman (1990) parental perceptions about the causes of disability have a tremendous impact on parents' behaviours in terms of seeking help or intervention for their children. Consequently, many studies investigate the perceptions of parents from different cultural groups about the causation of mental retardation. Studies show that parents have various cultural explanations for the causation of their children's disabilities. Studies also reveal that some parents have a tendency to accept modern explanations that are rational, scientific, or biomedical in nature.

Parents believe medications taken for infections, diabetes, and epilepsy might affect the development of a child. Mexican American parents cited premature birth, oxygen deprivation, forceps delivery, induced labour, and cerebral aneurysm as possible causes for a child's mental retardation. Environmental factors also were commonly cited; air and water pollution, preservatives in food, and toxic waste disposal were some of the reasons given by Mexican American parents to explain causes of mental retardation (Mardiros, 1989).

Studies also show that although parents were aware of and cited scientific and biomedical causes, they also endorsed socio-cultural or folk beliefs. Parents from traditional cultural contexts tended to express more traditional perspectives, including religious, supernatural, or folk beliefs, about the cause of mental retardation. Parents in the studies most frequently mentioned prenatal influences, religious beliefs, fate and evil eye and the role of parents and family.

Some cultural groups viewed prenatal influences as having a great impact on the causation of mental retardation. These were accepted causes of a child's appearance, character, and behaviour. For example, it was commonly believed among Jewish Oriental mothers that meeting a bad or misshapen person or unclean animal during pregnancy might affect the development of the unborn child (Stahl, 1991). Mexican American parents also held to those beliefs to explain the cause of mental retardation in children, and to other beliefs such as practising birth control, having abortion, not following a cultural prescription, seeking retribution against the spouse, and not wanting the child (Mardiros, 1989). Moreover, Mardiros states that parents also believe mistreating family members and prior attitude towards people with disabilities might be causes of disability.

Religious beliefs have been shown to play a role in parents' views on causation of mental retardation. In many Asian and African groups, violating a religious code is believed to be a cause of mental retardation, especially when rational explanations of the disability are not clear (Nicholls, 1993). Among religious perspectives about the causation of disability, God is considered the main cause, especially Mexican American parents. When studying Hispanic families with deaf children Steinberg and Davila (1997) found that most parents referred to God as the essential cause of deafness. Mardiros (1991) describes similar result from his study with Mexican American parents, which showed that parents most commonly believed that having a child with disability was God's will. This will may be conceptualised as God punishing the parent, testing the parent, or selecting the parent for reasons known only to God. Jewish Oriental mothers also showed similar feelings, referring to God as the cause of their children's mental retardation.

Hanson, Lynch and Wayman (1990) state that many cultural group place a strong emphases on the role of fate in determining a person's outcome. Among those cultural groups, Jewish Oriental mothers reveal a strong belief in an impersonal force, such as fate. They saw fate as the primary causal agent for having a child with mental retardation. They had many stories that even God could not do anything to prevent fate (Stahl, 1991). Chinese American parents also pointed to fate as a causal factor. According to Stahl evil eye was accepted as a reality, especially among Jewish Oriental parents, and the causation of all kinds of misfortunes, including having a child with mental retardation.

In some cultural groups, such as Chinese American, Jewish and Arab communities, parents may consider themselves or their families as the causal agent of disability. According to Diken (2006) several Chinese American parents reacted with guilt about their child's condition. These parents believed that the child's condition was a result of something they had done and feared being blamed for causing the disability. Some blamed their spouse for causing the disability. Parents had a propensity to blame each other and gave great importance to the belief that the disability might have been caused by either the father's or mother's side of the family. The child's disability tended to produce feelings of shame and guilt among Arab societies and Chinese American parents.

When considering cultural beliefs on the nature of disability, according to Diken (2006) results showed that some parents did not believe their children had a

disability, although they recognised their children were developing at a slower rate. Interestingly, some parents within the same cultural group defined their children as having both a disability and as being healthy. Several cultures also perceived the child with mental retardation as both a gift and a punishment from God. Some parents from different cultural group also believed that mental retardation was a temporary condition.

Odebiyi (1983) states that in Nigeria, diseases such as mental retardation have been known to have multiple causes among various cultures such as the Tivs, and Yorubas. They believe that there are natural preternatural and supernatural causes. The natural causes include faulty diet, worms, black or watery blood and hereditary factors. Such conditions may be intensified by the activities of the enemy or spirit. The preternatural causes of illness are believed to be malignant magical practices of sorcerers, curse, and witchcraft. These factors reflect hostilities, jealousies and tensions within the community. The supernatural causes on the other hand involve 'heavenly being'. They are either seen as coming directly from God himself or from the ancestors or from the "Orisas" (deities) as punishment for sins.

2.3 Theoretical Framework

The theoretical framework used in this study is based on Zigler's Development Theory (1990). Based on the universal principle of development, the model emphasizes the reciprocal relationship between typical development and the development of individuals with mental retardation. Early formulations of the developmental model focused on classifying children with mental retardation into two groups, those with and those without organic courses for their disability. Children with organic mental retardation, including those with chromosomal disorders, metabolic imbalances, prenatal, perinatal and postnatal insult and neurological problems were posited to have a typical cognitive process. Children with non-organic mental retardation on the other hand, were expected to have slow but normative cognitive development.

Ziegler represented the cognitive development of this latter group of children as a series of coils, some of which were compressed so that the rings were close together, while others were stretched so the rings were far apart. The point being made was that the loops of the coil representing cognitive growth, unfolded in the same sequence for all regardless of the speed at which the growth took place.

There are hundreds of mental retardation etiologies; it is unlikely that children with this diversity of causes for their disabilities can be combined into one homogenous group. A tree classification structure has been proposed by Burack (1990) that children would first be divided by whether the cause of their mental retardation was organic or non-organic. Children with mental retardation due to organic causes would then be further classified by etiology. Mental retardation theory emphasizes that these etiological differences are important and worth of study.

It has then been suggested that families of children with organic causes for their mental; retardation should focus on family relationships and roles, community participation and social mobility while families of children with non-organic mental retardation should concentrate on the pervasive family effect of poverty.

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CHAPTER THREE

METHODOLOGY

This chapter focuses on the research methodology which entails the research design, variables of the study, study population, participants, sample and sampling procedure, research instrument, validity and reliability of research instrument, administration of the research instrument as well as the analysis of data.

3.1 Research Design

The research design is descriptive survey using *ex-post-facto* type. This is a systematic empirical inquiry in which the scientist does not have direct control of independent variables because their manifestations have already occurred or because they are inherently cross-checked and not manipulable. Inferences about relations among variables are made without independent and dependent variables (Kerlinger, 2003). This design is used because mental retardation already exists in pupils whose parents are covered in this study.

3.2 Variables of the Study

The dependent variable of the study was mental retardation while the independent variables are parental age, birth order, socio-economic status, genetic make-up, diseases, birth trauma and cultural practices.

3.3 Study Population

The target population for the study was parents of children with mental retardation drawn from fifteen special education schools in the South Western zone of Nigeria.

3.4 Participants

The participants for this study were parents of children with mental retardation in selected schools from the South-western zone of Nigeria. These schools were: Hisibu Llahi Al-gaalib (HLA) School for the Handicapped Agodi, Ibadan, Oyo; School for the Handicapped Oke-Bola, Ibadan, Oyo; School for the Handicapped, Oniyanrin, Ibadan, Oyo; Omoyeni School for the Handicapped, Orita Aperin, Ibadan, Oyo; School for the Handicapped Ijokodo, Ibadan, Oyo;

School for the Handicapped, Oyo, Oyo;
 Daniel Akintounde School for the Mentally Retarded Adigbe, Abeokuta, Ogun;
 School for the Children with Special Needs Ejirin Road, Ijebu Ode, Ogun;
 School for the Handicapped Ilesha, Osun;
 Ojuwoye Community Primary School Inclusive Unit, Mushin Olosa, Lagos;
 Olisa Primary School Inclusive Unit Papa Ajao Mushin, Lagos;
 Modupe Cole Memorial Child Care and Treatment Home and School, Akoka Yaba, Lagos;
 Igbobi National Orthopaedic Special School, Igbobi, Lagos;
 School for the Handicapped, Otun Ireti, Ikare-Akoko, Ondo; and
 School for the Handicapped Ikere Ekiti, Ekiti.

3.5 Sample and Sampling Procedure

Purposive sampling technique was used to select the schools and subjects for the study. This is because the selected schools were the specialised schools where children with mental retardation can be found. Some 606 parents of pupils with mental retardation were used in this study. The breakdown is as follows

| State | No of Schools | Distribution of Respondents | No. of participants |
|-------|---------------|---|---------------------|
| Oyo | 6 | Hisibu Llahi Al-gaalib (HLA) School for the Handicapped Agodi, Ibadan | 45 |
| | | School for the Handicapped Oke-Bola, Ibadan. | 51 |
| | | School for the Handicapped, Oniyanrin, Ibadan. | 54 |
| | | Omoyeni School for the Handicapped, Orita Aperin, Ibadan. | 30 |
| | | School for the Handicapped Ijokodo, Ibadan. | 21 |
| | | School for the Handicapped, Oyo | 20 |
| | | Total | 221 |
| Ogun | 2 | Daniel Akintounde School for the Mentally Retarded Adigbe, Abeokuta. | 37 |
| | | School for the Children with Special Needs Ejirin Road, Ijebu Ode. | 113 |
| | | Total | 150 |

| | | | |
|-------|---|--|------------|
| Osun | 1 | School for the Handicapped Ilesha | 100 |
| Lagos | 4 | Ojuwoye Community Primary School Inclusive Unit, Mushin Olosa. | 10 |
| | | Olisa Primary School Inclusive Unit Papa Ajao Mushin. | 10 |
| | | Modupe Cole Memorial Child Care and Treatment Home and School, Akoka Yaba. | 11 |
| | | Igbobi National Orthopaedic Special School, Igbobi | 12 |
| | | Total | 43 |
| Ondo | 1 | School for the Handicapped, Otun Ireti, Ikare-Akoko, Ondo. | 36 |
| Ekiti | 1 | School for the Handicapped Ikere Ekiti. | 57 |
| | | GRAND TOTAL | 606 |

In Lagos State only 43 parents were got as participants. This could be attributed to the fact that not many parents with children with mental retardation could afford the cost of sending such children to school. Apart from this, the stigmatisation attached to having a child with mental retardation would make parents to keep these children away from school or the public.

3.6 Research Instrument

The researcher constructed questionnaires of two different scales namely Parental Demographic Scale (PDS) and Mental Retardation Determinant Scale to elicit information from the parents of children with mental retardation. Section A of the first scale contained biodata of the respondents, that is, age, marital status, address and gender. The scale also bore the following captions:

Section B contained questions on parental age while Section C dealt with questions on birth order of the child. Next to this is section D with questions on socioeconomic status of the parents. This was then followed with Section E eliciting information on the genetic make up of the parents of the child.

Section F contained questions on birth trauma the child sustained at birth while Section G contained questions on the diseases the child is exposed to during his

or her growth and development. Lastly Section H contained questions on cultural practices.

The second part of the Mental Retardation Determinant Scale was the Likert – scale type and it consists thirty one items designed to elicit the opinion of the parents on the seven psychosocial variables of the study.

3.7 Validity and Reliability of Instrument

In order to validate the instrument, the researcher ensured that the items on the questionnaire correlated with the objectives of the study so as to ascertain the content validity of the instrument. Draft copies of the questionnaire were given to the experts in the department of special education, evaluation, psychology, nursing and medicine for editing to ascertain the face and content validity of the instruments. Based on their criticism and suggestions on the items on the questionnaire, modifications were made. Suggestions from experts were incorporated into the final draft of the questionnaire in order to enhance the content validity of the instrument.

The instrument was further subjected to empirical validation to determine the reliability. A pilot study was carried out by distributing copies of the questionnaire to a group of respondents that were not used in the final study. The split-half coefficient reliability of the scale was found to be 0.89. The internal consistency of the scale was also established using Cronbach alpha and a reliability coefficient of 0.77 was obtained.

3.8 Administration of the Research Instrument

Three research assistants who were university graduates were recruited and they worked with the researcher on the field for a period of four months. The research assistants were first given orientation about the modalities of administering the questionnaire before data collection began. An initial visit was made to the selected schools at different times to brief the head teachers and fix dates and time to meet with the parents. Some of these dates were fixed for the parents/teachers association meeting days while some other dates were fixed for the end of the year and Christmas party.

Further, some children were followed home to meet with their parents. On these occasions after briefing the parents about the purpose of the study, the researcher and research assistants were able to administer the questionnaire. Those

parents who were educated responded to the questionnaire themselves while illiterate parents had the questions translated and explained by the research assistants. The questionnaires were retrieved immediately.

3.9 Data Analysis

The statistical techniques used in analysing the data in this study were descriptive statistics of frequency count, percentage, mean and standard deviation. Graph was also used where necessary. The data were also subjected to the correlational matrix showing the inter correlation between the independent variables and the criterion measure. Inferential statistics of multiple regression analysis and t-test were employed in testing the hypotheses. This helped in considering the composite and relative effects of the predictor variables on the dependent variable.

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CHAPTER FOUR

PRESENTATION OF RESULTS

This chapter presents the results from the research questions and hypotheses stated in the study. The results are presented in Tables, Graphs and References are made. The statistics/ techniques that are used, methods of data analysis and results obtained are discussed. For each hypothesis, the statistical test of significance selected and applied to the data are stated, followed by a statement indicating whether the hypothesis was accepted or rejected.

4.1 Results

Research Question 1: Do children with mental retardation have common birth order?

Table 4.1: Birth Order of Children with Mental Retardation as Supplied by the Patients

| Birth order | F | % |
|---------------|------------|------------|
| First | 143 | 23.6 |
| Second | 60 | 9.9 |
| Third | 111 | 18.3 |
| Fourth | 157 | 25.9 |
| No indication | 135 | 22.3 |
| TOTAL | 606 | 100 |

Source: From survey.

Table 1 reveals that 606 parents of children with mental retardation were involved in the study. Out of this, 25.9% indicated that their child with mental retardation is the fourth child, 23.6% shows that the child with mental retardation was in the first position while 9.9% of the parents stated that second born child was the one with mental retardation. However, 22.3% of the respondents failed to indicate the position of their children with mental retardation. This reveals that there is no common birth order for children with retardation. Information in Table 4.1 is presented graphically in Figure 4.1.

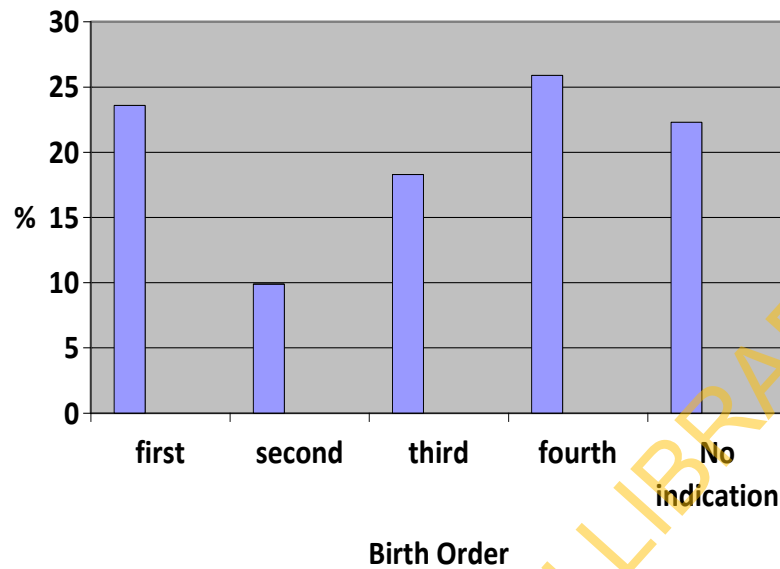


Fig 4.1: Graph Showing Birth Order of Children with Mental Retardation.

Research Question 2: Is there any significant correlation between the birth order of the child and mental retardation?

Table 4.2: Summary of Cross Tabulation and Chi Square Analysis Showing Level of Association between Birth Order of the Child and Mental Retardation

| No of Children in the Family | 1 st | 2 nd | 3 rd | 4 th | X ² | df | Sig (p) | |
|------------------------------|-----------------|-----------------|-----------------|-----------------|----------------|----|---------|-----|
| 1 | 39 | 3 | - | - | | | | |
| 2 | 48 | 15 | - | - | | | | |
| 3 | 63 | 12 | 18 | - | 461.057 | 15 | .000 | sig |
| 4 | 15 | 18 | 36 | 51 | | | | |
| 5 | 9 | 9 | 21 | 42 | | | | |
| 5 and above | 15 | - | 15 | 24 | | | | |

Table 4.2 shows that there is a significant association between the birth order of the child and mental retardation ($X^2= 461.057$; $df= 15$; $P<0.05$). The association

shows that a woman with 1, 2 or 3 children tend to have the first as a child with mental retardation while if the number of children is more than three, the child with mental retardation mostly come as the fourth born. This relation is suggested given that the result of chi-square computation is significant.

Research Question 3: What was the age range of parents when they had the baby with mental retardation?

Table 4.3: Age of Parents of Children with Mental Retardation

| Age range | F | % |
|--------------|------------|--------------|
| 18 – 23 | 128 | 21.1 |
| 24 – 29 | 102 | 16.8 |
| 30 – 34 | 121 | 20.0 |
| 35 – 40 | 255 | 42.1 |
| Total | 606 | 100.0 |

Table 4. 2 shows that the largest proportion of parents, that is, 42.1% had their babies with mental retardation at the age range of 35 years to 40 years, 20% of them had their babies between the ages of 30 and 34 years of age while 37.9% of these parents had their babies before the age 30. This shows that for every three women who have children with mental retardation, women of old age is approximately 1. The graph below depicts this information.

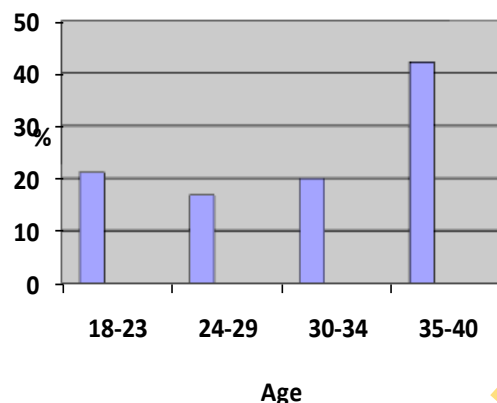


Fig 4.2: Graph Showing Age Range of Women when the Child with Mental Retardation was Born.

Research Question 4: Did the mothers of the children with mental retardation have difficulties during pregnancy and labour?

Table 4.4a: Difficulties Encountered by the Mothers during Pregnancy

| S/N | Statement | Option | Freq | % |
|-----|---|---------------|------|------|
| 1 | How many months was the pregnancy of the child with mental retardation when you encountered difficulties? | 7 months | 27 | 4.5 |
| | | 8 months | 27 | 4.5 |
| | | 9 months | 438 | 72.3 |
| | | 12 months | 6 | 1.0 |
| | | No indication | 108 | 17.8 |
| 2 | Did you use any drug during the pregnancy of the child with mental retardation? | No | 346 | 57.1 |
| | | Yes | 59 | 9.7 |
| | | No indication | 201 | 33.1 |
| 3 | If yes, at what stage of the pregnancy did you use the drug? | 1-3 months | 48 | 7.9 |
| | | 4-6 months | 51 | 8.4 |
| | | 7-9 months | 6 | 1.0 |
| | | No indication | 501 | 82.7 |
| 4 | Were you ill at any stage of the pregnancy? If yes, what type of ailment? | Anaemia | 48 | 7.9 |
| | | Malaria | 78 | 12.9 |
| | | Chicken pox | 3 | .5 |
| | | Jaundice | 6 | 1.0 |
| | | No Indication | 471 | 77.7 |

Table 4.4 a reveals that majority of the parents, that is, 72.3% delivered the child with mental retardation at 9 months, 57.1% of them did not use any drug during the pregnancy while only 9.7% did. Out of those that used drug, 7.9% used the drug during the first three months, 8.4% did between 4 and 6 months while only 1% did so between 7 to 9 months. Some 12.9% of these mothers had malaria during pregnancy, 7.9% of them had anaemia and only 1% had jaundice while 0.5% had chicken pox. From all these, it could be inferred that large proportion of the parents, that is, 77.7% were not affected during pregnancy by diseases and 72.3% of these mothers had no difficulties.

Table 4b: Difficulties Experienced by Mothers of Children with Mental Retardation during Labour.

| S/N | Statement | Option | Freq | % |
|-----|--|-----------------|------|------|
| 1 | How long did it take to deliver the baby? | 0-3 hrs | 416 | 68.6 |
| | | 4-9 hrs | 30 | 5.0 |
| | | 10-14 hrs | 15 | 2.5 |
| | | 15 hrs | 13 | 2.1 |
| | | No indication | 132 | 21.8 |
| 2 | How was the baby delivered? | Normal delivery | 461 | 76.1 |
| | | Breech delivery | 19 | 3.1 |
| | | Vacuum delivery | 24 | 4.0 |
| | | No indication | 102 | 16.8 |
| 3 | Did the child cry immediately after birth? | No | 57 | 9.4 |
| | | Yes | 429 | 70.8 |
| | | No indication | 120 | 19.8 |
| 4 | Did you notice any abnormality immediately the child was born? | No | 357 | 58.9 |
| | | Yes | 102 | 16.8 |
| | | No indication | 147 | 24.3 |
| | If yes, which of the following abnormalities? | Palor | 93 | 15.3 |
| | | Small head | 45 | 7.4 |
| | | Large head | 12 | 2.0 |
| | | No indication | 456 | 75.0 |

Table 4.4b shows that 68.8% of the mothers of children with mental retardation delivered their babies between 0 and 3 hours, while only 9.6% of them delivered the babies above 3 hours of labour. Some 76.1% of them had normal delivery, only 7.1% had abnormal delivery, 70.8% of them noticed that their babies cried immediately after birth. Some 16.8% of these mothers noticed some abnormalities in their babies after birth and the major abnormality was palor of the skin which was 15.3% of the abnormalities. From all these, it could be said that majority of the mothers of children with mental retardation had no difficulties during labour.

Research Question 5: Did the children with mental retardation experience accident or chronic diseases at the early development stage?

Table 4.5: The Chances of Children with Mental Retardation having Accident or Chronic Disease at the Early Development Stage.

| S/N | Statement | Option | Freq | % |
|-----|--|---------------|------|------|
| 1 | Did your child fall at any period of his development? | No | 315 | 52.0 |
| | | Yes | 129 | 21.3 |
| | | No indication | 162 | 26.7 |
| 2 | Did he/she sustain any head injury? | No | 222 | 36.6 |
| | | Yes | 69 | 11.4 |
| | | No indication | 315 | 52.0 |
| 3 | Did your child fall ill at any particular period of his/her development? | No | 177 | 29.2 |
| | | Yes | 246 | 40.6 |
| | | No indication | 183 | 30.2 |
| 4 | If yes, which of the following illnesses? | High fever | 198 | 32.7 |
| | | Meningitis | 21 | 3.5 |
| | | PKU | 12 | 8.0 |
| | | No indication | 375 | 61.9 |

Table 4.5 shows that only 21.3% of the children with mental retardation observed fell during their early developmental stage while only 11.4% of them

sustained head injury. However, about 41% of them fell ill during this period and the common sickness was high fever which was 32.7% of the illnesses observed in the study. From this, only one child out of every five of children with mental retardation fell during early developmental stage and one child out of every three children with mental retardation fell sick during the early developmental stage. Hence, the number of children with mental retardation that fell or became sick during early developmental stage is less than average.

Research Question 6: What is the perception of parents of children with mental retardation on (a) parental age; (b) birth order; (c) socio-economic status of parents; (d) diseases; and (e) genetic make-up of the child, birth trauma, cultural practices engaged in by parents, about the chances of giving birth to a child with mental retardation?

Table 4.6a: Perception of Parents of Children with Mental Retardation on Parental Age as Cause of Mental Retardation.

| S/N | Statement | SA | A | D | SD | Mean | Std Dev |
|-----|---|---------------|---------------|---------------|---------------|------|---------|
| 1 | The older the parents, the more the likelihood of having a child with mental retardation | 294 (48.5) | 192 (31.7) | 78 (12.9) | 42 (6.9) | 3.22 | 1.27 |
| 2 | It is the age of the mother alone that determines having a child with mental retardation | 183 (30.2) | 66 (10.9) | 138 (22.2) | 219 (36.1) | 2.35 | 1.06 |
| 3 | It is the age of both parents that determines having a child with mental retardation | 255 (42.1) | 180 (29.7) | 108 (17.8) | 63 (10.4) | 3.04 | 1.21 |
| 4 | If a woman is 35 years and above and a man 40 years above she is likely to have a child with mental retardation | 240 (39.6) | 135 (22.3) | 63 (10.4) | 168 (27.7) | 2.84 | .99 |

Table 4.6a shows that the parents of children with mental retardation agreed that the older the parents the more the likelihood of having a child with mental retardation ($X=3.22$); they also agreed that it is the age of both parents that determines having a child with mental retardation ($X=3.04$) and they agreed that if a woman is 35 years and above, she is likely to have a child with mental retardation ($X=2.84$) but they disagree that it is the age of the mother alone that determines having a child with mental retardation ($x=2.35$). All these reveal that the parents of these children with mental retardation perceived that parental age could determine having children with mental retardation.

Table 4.6b: Perception of Parents on Birth Order as the Cause of Mental Retardation

| S/N | Statement | SA | A | D | SD | Mean | Std Dev |
|-----|---|---------------|---------------|---------------|---------------|------|---------|
| 1 | Having many children (5 -10) will predispose a woman to having a child with mental retardation. | 216 (35.6) | 99 (16.3) | 198 (32.7) | 93 (15.3) | 2.72 | 1.04 |
| 2 | The number of children one has, has nothing to do with the birth of a child with mental retardation | 279 (46.0) | 228 (37.6) | 78 (12.9) | 21 (3.5) | 3.26 | 0.96 |
| 3 | Children whose birth order falls between 4 and 10 years are likely to have mental retardation | 216 (36.6) | 237 (39.1) | 30 (5.0) | 123 (20.3) | 2.90 | 0.87 |

Table 4.6b reveals that the parents of the children with mental retardation were of the opinion that the number of children one has, has nothing to do with the birth of a child with mental retardation ($X=3.26$) and that children whose birth order falls between 4 and 10 years are likely to have mental retardation ($X=2.9$). This contradiction suggests that these parents find it difficult to say categorically that birth order determines having a child with mental retardation.

Table 4.6c: Perception of Parents on Socio-economic Status as Cause of Mental Retardation

| S/N | Statement | SA | A | D | SD | Mean | Std Dev |
|-----|--|---------------|---------------|---------------|---------------|------|---------|
| 1 | Only poor families give birth to children with mental retardation | 180 (29.7) | 24 (4.0) | 150 (24.8) | 252 (41.6) | 2.22 | 1.61 |
| 2 | Whether a family is rich or poor has nothing to do with the birth of a child with mental retardation in the family | 216 (35.6) | 192 (31.7) | 183 (30.2) | 15 (2.5) | 3.01 | 1.21 |
| 3 | The type of food a woman eats predisposes her to having a child with mental retardation | 288 (47.5) | 231 (38.1) | 66 (10.9) | 21 (3.5) | 3.30 | 0.94 |

Table 4.6c shows that the parents of children with mental retardation agreed that whether a family is rich or poor has nothing to do with the birth of a child with mental retardation ($X=3.01$), but that quality of food and dietary habits predisposes a woman to having a child with mental retardation ($X=3.30$). Further, they expressed the view that families with low socio-economic status are more likely to give birth to children with mental retardation ($X=2.2$). This implies that the parents of these children perceived that socio-economic status of the parents does not determine having a child with mental retardation.

Table 4.6d: Perception of Parents on Diseases and Drug Abuse as Causes of Mental Retardation.

| S/N | Statement | SA | A | D | SD | Mean | Std Dev |
|-----|---|---------------|---------------|-------------|-------------|------|---------|
| 1 | Mental retardation is caused by illness affecting the mother during pregnancy | 312 (51.5) | 240 (39.6) | 39 (6.4) | 15 (2.5) | 3.40 | 0.56 |
| 2 | Drug abuse can be a factor in mental retardation | 447 (73.8) | 120 (19.8) | 24 (4.0) | 15 (2.5) | 3.65 | 0.42 |

Table 4.6d shows that the parents of children with mental retardation were of the view that mental retardation is caused by illness affecting the mother during the pregnancy ($X=3.4$) and that drug abuse can be a factor in mental retardation ($X=3.65$). This shows that the parents of the children with mental retardation perceived that disease and drug abuse could be factors in determining having a child with mental retardation.

Table 4.6e: Perception of Parents on Genetic Make-up as the Cause of Mental Retardation

| S/N | Statement | SA | A | D | SD | Mean | Std Dev |
|-----|---|---------------|---------------|--------------|---------------|------|---------|
| 1 | Mental retardation could be due to inheritance rather than the age of parents | 309 (51.0) | 114 (18.8) | 72 (11.9) | 111 (18.3) | 3.63 | 0.84 |

Table 4.6e shows that the parents of children with mental retardation were of the opinion that it could be due to inheritance rather than the age of the parents ($X=3.63$). This implies that the parents perceived that mental retardation has to do with genetic make-up of the parents than the age of the parents

4.2 Testing the Null Hypotheses

Hypothesis 1: There is no significant composite effect of (a) parental age; (b) birth order; (c) socio-economic status of parents; (d) birth trauma; (e) diseases; (f) cultural practice; and (g) genetic make-up of the parents on the chances of giving birth to children with mental retardation.

Table 4.7: Correlation Matrix of the Independent Variables and the Dependent Variable

| Independent Variables | Parental Age | Genetic Make-up | Socio-Economic Status | Birth Order | Birth Trauma | Disease | Cultural Practices |
|-----------------------|--------------|-----------------|-----------------------|-------------|--------------|---------|--------------------|
| Parental Age | 1 | | | | | | |
| Genetic Make-up | -.347** | 1 | | | | | |
| Socio-economic Status | .257** | -.321** | 1 | | | | |
| Birth Order | .247** | -.296** | .152** | 1 | | | |
| Birth Trauma | .018 | -.332** | .033 | .283** | 1 | | |
| Disease | -.052 | -.313** | .175** | -.014 | .271** | 1 | |
| Cultural Practices | -.107** | .239** | -.101* | .161** | -.136** | -.225** | 1 |
| Mental Retardation | -.297** | .773** | -.260** | -.061 | .200** | -.340** | .540** |
| Mean Score | 3.17 | 1.98 | 2.88 | 2.96 | 3.53 | 2.18 | 3.13 |
| Standard Deviation | 1.160 | 1.130 | 1.161 | 1.124 | 1.56 | 1.161 | 1.212 |
| N | 606 | 606 | 606 | 606 | 606 | 606 | 606 |

** Correlation is significant at the 0.01 level

* Correlation is significant at the 0.05 level

The result in Table 4.7 showed that there is a significant correlation between mental retardation and parental age ($r=-.297$, $P<0.01$), socio-economic status ($r=-.260$, $P<0.0001$), genetic make up ($r=.773$, $P<0.001$), birth trauma ($r=-.200$, $P<0.001$), disease ($r=-.340$, $P<0.001$), cultural practices ($r=.540$, $P<0.001$) while birth order is not significantly related to mental retardation.

Table 4.8a: Summary of Multiple Regression Analysis Showing Composite Effect of Seven Independent Variables

| Model | R | R ² | Adjusted R ² | Std Error |
|-------|-------|----------------|-------------------------|-----------|
| 1 | 0.862 | 0.743 | 0.740 | 8.502 |

Table 4.8a reveals that the seven independent variables are related to the dependent variable (chance of having mental retardation ($R=0.862$)) and that the independent variables accounted for (74%) of total variance in the dependent variable (Adjusted $R^2 = 0.740$).

Table 4.8b below shows the significant status of composite effects.

Table 4.8b: Multiple Regression ANOVA Showing Significant Status of the Composite Effect

| Model | Sum of squares | Df | Mean square | F | Sig (p) | Remark |
|------------|----------------|-------|-------------|---------|---------|-------------|
| Regression | 125210.59 | | 1788.227 | 247.476 | .000 | Significant |
| Residual | 43222.597 | 7,598 | 72.279 | | | |

Table 4.8b shows that the composite relationship given by Table 4.9a is significant ($F=247.476$); $df = 7,598$ $p < 0.05$). This implies that there is a composite effect of the seven independent variables on the chances of giving birth to children with mental retardation. Therefore, the null hypothesis 1 is rejected.

Hypothesis 2: There is no significant relative effect of: (a) parental age; (b) birth order; (c) socio-economic status of parents; (d) birth trauma; (e) diseases; (f) cultural practices engaged in by parents; and (g) genetic make-up of the parents on the chances of giving birth to a child with mental retardation.

Table 4.9: Summary of Multiple Regression Analysis Showing Relative Effects of the Independent Variables on the Chances of Giving Birth to a Child with Mental Retardation.

| Model | Unstandardized Coefficient | | Standardized Coefficient | t | Sig | Rank | Remark |
|----------------------------|----------------------------|-----------|--------------------------|--------|------|-----------------|--------|
| | B | Std Error | Beta (β) | | | | |
| Constant | 9.795 | 1.986 | | 4.933 | .000 | | |
| Socio-economic status | 1.054 | .208 | .000 | .005 | .996 | 7 th | NS |
| Birth order | .948 | .260 | .087 | 3.648 | .000 | 3 rd | Sig |
| Birth trauma | 1.508 | .516 | .067 | 2.921 | .004 | 4 th | Sig |
| Disease during development | -.386 | .491 | -.018 | -.786 | .432 | 6 th | NS |
| Genetic make-up | 10.288 | .365 | .720 | 28.158 | .000 | 1 st | Sig |
| Parental age | -.428 | .357 | -.028 | -1.201 | .230 | 5 th | NS |
| Cultural practices | 8.060 | .506 | .357 | 15.921 | .000 | 2 nd | Sig |

Dependent variable: chances of giving birth to a child with mental retardation

Table 4.10 shows that

1. Parental age has no significant relative effect on the chances of giving birth to a child with mental retardation ($\beta = 0.028$; $t = -1.201$; $p > 0.05$). Therefore, the null hypothesis 2a is not rejected.
2. Birth order has a significant relative effect on the chances of giving birth to a child with mental retardation ($\beta = 0.087$; $t = 3.648$; $p < 0.05$). Therefore, the null hypothesis 2b is rejected.
3. Socio-economic status of the parents has no significant relative effect on the chances of having a child with mental retardation ($\beta = 0.000$; $t = 0.005$; $p > 0.05$). Therefore, the null hypothesis 2c is not rejected.
4. Birth trauma has a significant relative effect on the chances of giving birth to a child with mental retardation ($\beta = 0.067$; $t = 2.921$; $p < 0.05$). Therefore, the null hypothesis 2d is rejected.
5. Diseases the child experienced during the developmental stages have no significant relative effect on the chances of having a child with mental

retardation ($\beta = -0.018$; $t = 0.786$; $p > 0.05$). Therefore, the null hypothesis 2e is not rejected.

6. Cultural practices of the mother while pregnant have a significant relative effect on the chances of giving birth to a child with mental retardation ($\beta = 0.357$; $t = 15.921$; $p < 0.045$). Hence, the null hypothesis 2f is rejected.
7. Genetic make-up of the parents has a significant relative effect on the chances of giving birth to a child with mental retardation ($\beta = 0.720$; $t = 28.158$; $p < 0.05$). Hence, the null hypothesis 2g is rejected.

4.3 Summary of Findings

Based on the analysis presented above, the following are the summary of the findings:

1. Children with mental retardation have various birth orders, but the fourth born and above children have a greater tendency of having mental retardation.
2. For every three women that have children with mental retardation, women of older age (above 35) are approximately 1.
3. Majority of the mothers of children with mental retardation were not affected by diseases and had no difficulties during pregnancy
4. Majority of the mothers of children with mental retardation had no difficulties during labour.
5. Only one child out of every five children with mental retardation fell and sustained injury, during developmental stage and only one out of every three children fell sick during this period. This implies that the children with mental retardation that fell and sustained injury or became ill during their early developmental stages are less than average.
6. Parents of children with mental retardation perceived that socio-economic status of parents could not predispose them to having children with mental retardation.
7. Parents of children with mental retardation perceived that diseases and drug abuse as well as genetic make-up of the parents could determine giving birth to children with mental retardation.
8. There is a significant joint effect of parental age, socio-economic status, birth order, birth trauma, diseases, cultural practices and genetic make-up of the parents on giving birth to children with mental retardation.

9. Genetic make-up of the parents, cultural practices engaged in during pregnancy, birth order and birth trauma are the factors that could significantly determine giving birth to children with mental retardation.

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CHAPTER FIVE

DISCUSSION OF FINDINGS, CONCLUSION AND RECOMMENDATIONS

In this chapter, the discussion of findings based on the research questions and hypotheses, conclusion and recommendations are presented.

5.1 Discussion of Findings

Research Question 1: Do children with mental retardation have common birth order?

The result showed in Table 4.1 and Figure 4.1 that there is no common birth order in children with mental retardation. The parents (25.9%) indicated that the child born into the fourth position had mental retardation while 23.6% stated that their first born had mental retardation, 18.3% of them had the child with mental retardation as the third born and 9.9% had their second born with mental retardation. From this result, mental retardation could not be overwhelmingly associated with any particular birth order in the family. This suggests that a child with mental retardation can be born into any order in the family. This fact lends support to Clayton (2008) findings that birth order, even though plays an important role in an individual's intelligence and that birth order theory holds that children develop their intelligence and behavioural pattern as a result of their position within the family but that the position a child is born into in the family has nothing to do with having mental retardation.

Research Question 2: Is there significant positive relation between the birth order of the child and mental retardation?

The result in Table 4.2 showed that there is a significant relation between the number of children a woman has and the birth order of the child with mental retardation. Considering the trend of the findings as encapsulated in this question and as depicted in Table 4.2, a woman is likely to have a child with mental retardation as her fourth born if she has had between one or three children. In the same vein, when the number of children of a woman increases to four and above, there is high potency of having the child with mental retardation as subsequent children. This is buttressing Williams and Decoufle's (2007) study that the older a woman is and the more the number of children she has, the higher the risk for having a child with either birth defects or mental retardation. Experts believe that in older women, errors occurs more in their ova and the resultant effect of this is having a child with mental retardation

(Giri rajan 2003). From the foregoing therefore, there is a significant relation between the number of children a woman has and the possibility of having a child with mental retardation.

Research Question 3: What is the age range of parents of children with mental retardation when they had their babies?

From the result of the analysis in Table 4.2 and Figure 4.2, the response of the parents show that parental age is a factor to be reckoned with in determining whether a child is born with mental retardation or not. Some 42.1% of the mothers had their children with mental retardation between the age of 35 and 40.

This result reveals further that for every three women who had children with mental retardation, one of them must be between the age range of 35 and 40. This is in agreement with the study of Hollier, Leveno, Kelly, Mcutire and Curningham (2000); they linked advanced maternal age, beyond 25 years, with significant increase in fetal abnormalities such as mental retardation.

In addition, parents who had their children with mental retardation at a young age, 18 to 23, were 21.1%, closely followed by parents whose ages were between 30 and 34 years. From all indication, mental retardation cuts across all ages, though it may be more pronounced in older parents because of the change occurring in their reproductive cells.

According to the report of National Institute of Child Health and Human Development (2004) researchers have established that the likelihood that a reproductive cell will contain an extra copy of chromosome 21 increases dramatically as a woman ages. Therefore, an older mother is more likely than a younger mother to have a baby with mental retardation. However, of the total population, older mothers have fewer babies; about 75% of babies with mental retardation are born to younger women because younger women more than older women have babies. Only about 9% of total pregnancies occur in women 35 years or older each year but about 25% of babies with mental retardation are born to women in this age group.

Research Question 4: Did mothers of children with mental retardation have difficulties during pregnancy and labour?

From the results in Tables 4.4a, among women that carried the pregnancy to full term, 12.9% of them had malaria during pregnancy, 7.9% had anaemia, negligible

percentage, that is, 0.5 had chicken pox and 1% had jaundice. Medically, malaria and anaemia are common problems encountered during pregnancy and this does not necessarily have to be a factor that can be associated with mental retardation except in severe cases. The percentage of those who used drug during pregnancy also experienced short period of labour and ended up with normal deliveries. Considering the trend of these findings, none of the mothers had difficulties during these conditions which indicate that their having children with mental retardation are not related to any condition during pregnancy or labour. There are other causes of mental retardation that can make the woman to have such a child. This is buttressing the findings of Gurnee and Silvestre (2005) that not all malformations or cases of mental retardation can be attributed to pregnancy, labour or drugs. They attribute the cause of approximately 40% of malformations to unknown causes. About 12 to 25% of these congenital malformations are purely genetic defects. Down syndrome is the most common of this group. They also state that another 20% are due to interactions between hereditary and environmental factors, the latter of which are largely unknown.

Research Question 5: Did these children experience accident or disease at the early developmental stages?

Judging from the outcome of the findings in Table 4.5, only 41% of these children fell ill at a particular period of development while 21.3 fell, sustaining injury. From this, the cause of their mental retardation can not be associated with accidents or disease in their infancy.

Research Question 6a: What is the perception of parents of children with mental retardation on parental age as determinant of mental retardation?

From Table 4.6a, parents perceived that the age of parents could be a factor in having children with mental retardation. They express the notion that the older the parents, especially the women the more the likelihood of having a child with mental retardation. This perception has been further stressed by Rives, Langlois, Bordes, Simeon and Mace (2002); they link mental retardation with advanced parental age. They observe that advanced parental age especially the maternal has been known to be the most important etiological factor implicated in mental retardation. Their study shows that the tendency to have a child with mental retardation tend to increase

significantly in elderly women. Further, Brody (2004) opines that older women have a higher incidence of genetically damaged embryo and are five times more likely to have chromosomal disorders.

Similarly, in this study parents agreed that it is the age of both parents that determines having a child with mental retardation. Their responses are in consonance with the finding of Rives' et al (2002) study that there is a possible relationship between parental age and giving birth to a child with mental retardation. Their study shows that older fathers with mothers of 35 year have tendency of having children with mental retardation and for paternal ages of 41 years upwards, the age effect is quite strong. In this same vein, Dzurova and Pikhart (2005) support this viewpoint that most children with mental retardation are born to parents above 35 years of age and that significant risk levels for mental retardation are not only in advanced maternal categories but also in paternal age, indicating that the risk of births of children with mental retardation significantly increases with increasing paternal age, too. From the foregoing, advanced parental age, that is, both father and mother has been perceived as a determinant of mental retardation.

The parents also express the viewpoint that it is not only the age of the mother that determines mental retardation (Table 4.6a). This is not surprising in that for a child to be born, it has to be a joint effort of both the man and the woman. Both of them play key roles in contributing to the genetic make-up of the child, so mental retardation as a result of advanced age could not have been from the mother alone in all cases.

Research Question 6b: What is the perception of parents of children with mental retardation on birth order as determinant of mental retardation?

Table 4.6b shows the perception of parents on birth order as a determinant of mental retardation. The parents were of the opinion that the number of children one has, has nothing to do with the birth of a child with mental retardation but they believed that when the interval between one child and the other is between 4 to 10, years there is the likelihood of having a child with mental retardation.

Research Question 6c: What is the perception of parents of children with mental retardation on socio-economic status as determinant of mental retardation?

Table 4.6c shows parental perception of the socio-economic status of parents as determinants of mental retardation. The parents expressed the notion that whether a family is rich or poor has nothing to do with the birth of a child mental retardation but that the quality of food and dietary habits predispose a woman to having a child with mental retardation. In other words, parents could be rich but if they do not eat what is right and beneficial for the growing fetus, their unborn child may be predisposed to being mentally retarded. It is a known fact that in a family with low socio-economic status, poverty is bound to be present and therefore it will be difficult for such a family to have good quality food and maintain good dietary habits. In this study majority of these families come from the rural setting with poor background. Their knowledge about the quality of food and dietary habits is shallow; therefore the pregnant mothers do not know in most cases the right type of food to eat. This in turn affects the unborn child, some of these women might even believe that certain food which under normal circumstances are expected to enhance the life of the unborn child should be forbidden. According to Bradley and Corwiyn (2002) one of the ways of measuring socio-economic status is parental education. A well informed family though may not be buoyant financially is able to use what they have to maintain a healthy family.

Research Question 4.6d: What is the perception of parents of children with mental retardation on disease as determinant of mental retardation?

Parental opinion on disease as a determination of mental retardation as depicted in Table 4.6d indicates that illness affecting the mother during pregnancy can predispose her to having a child with mental retardation. This opinion has been medically supported, in that certain illnesses in the mother while pregnant could cross the placental barrier and affect the unborn child. An example of such a condition is rubella also known as German measles. Likewise, illnesses such as meningitis or encephalitis affecting the child in infancy may affect the development of the brain leading to mental retardation. In the same vein, parents expressed the viewpoint that drugs can be a factor in mental retardation. Drug abuse indulged in by a pregnant woman predisposes her unborn child to mental retardation. A typical example of this is a drug called thalidomide which is a sleep inducing drug.

This is consonance with the work of Woods (2005) that infectious diseases during pregnancy or childhood which are easily preventable through immunisation, can cause mental retardation when they result in complications. Examples are measles, chicken pox, meningitis, encephalitis and whooping cough which can damage the brain.

Research Question 6e: What is the opinion of parents on genetic make-up as the determinant of mental retardation?

Table 4.6e shows that the parents were of the opinion that mental retardation could be inherited. This result corroborates Coughlin and Marhgam's (2004) assertion that genetic variation can cause mental retardation. They argue further that in most cases, approximately 92% of the time, mental retardation is caused by the presence of an extra chromosome which originates in the development of either the egg or sperm. Consequently, when the egg and the sperm unite to form the fertilized egg, three, rather than two chromosomes 21 are present. As the embryo develops, the extra chromosome is repeated in every cell. American Association of Mental Retardation (2002) also identifies quite a number of genetic causes of mental retardation. Most identifiable causes (defined as an intelligence quotient of 50 or less) are noted to originate from genetic disorders. Up to 60% of severe mental retardation can be attributed to genetic causes, making it the most common cause in cases of severe mental retardation.

Next, the hypothesis will be discussed to show both the composite and relative effects of the seven independent variables impact on the chances of giving birth to children with mental retardation.

Table 4.8a depicts the multiple regression analysis of the component effects of the seven independent variables as related to the dependent variable, ($R = 0.862$) and the independent variables accounted for 74% of total variance in the dependent variable. Adjusted $R^2 = 0.740$.

Hypothesis 1 states that there is no significant composite effect of parental age, birth order, socio-economic status, birth trauma, cultural practices, diseases and genetic make-up on the chances of giving birth to children with mental retardation

The result from the analysis of testing the hypothesis reveals that there is a significant composite effect of the seven independent variables on the chances of giving birth to children with mental retardation. The total variation accounted for by the linear combination of the independent variables is 74%. This means that all the independent variables jointly predicted the chances of giving birth to children with mental retardation in parents covered in this study. Some of the independent variables may not have individual significant relationship with mental retardation but putting them together with other variables makes them to have joint composite effect. The presence of two or more variables increases the chance of predicting mental retardation. For instance where a woman is advanced in age and at the same time does not engage in good dietary habit or does not know what to eat the chances becomes higher.

Birth trauma may not be significantly correlated with mental retardation on its own but where poverty on the side of the parents does not allow the child to receive medical attention the chance of mental retardation becomes stronger. Many studies have demonstrated this composite effect. For example, Dzurova and Pikhart (2005) compared the relationship between parental age, birth order, socio-economic status and education of parents and mental retardation in two culturally and socially contrasting population settings, California and Czech Republic. Their findings show that the four independent variables were highly correlated, more in Czech Republic than in California, but all of them were highly significant. Maternal age was however associated very strongly with down syndrome. This effect as shown in this study is also in agreement with the work of Bradley and Corwyn (2002) showing that socio-economic status of parents is associated with a wide array of health, cognitive and socio-emotional outcomes in children. Further, according to Gurnee and Silvestre (2005) the composite effects of drugs used by the mother in pregnancy, disease and genetic make-up of parents on the chances of having children with mental retardation show that about 12% to 25% of congenital malformations are purely genetic defects in which Down syndrome is the most common of the group. Maternal disease or

infection, chemical and drug abuse accounted for between 5% and 9% of the malformations.

Most drugs cross the placenta barrier by simple diffusion. Many actually reach 50% to 100% of the concentration in fetal blood as that in the maternal blood thereby causing fetal malformation. From the foregoing, it could be stated that all the seven psychosocial variables when taken together have a significant composite effect on predicting mental retardation.

Hypothesis 2 states that there is no significant relative effect of (a) parental age, (b) birth order, (c) socio-economic status of parents (d) birth trauma (e) disease (f) cultural practices and (g) genetic make-up of parents on the chances of giving birth to a child with mental retardation.

There is no significant relative effect of parental age on the chances of giving birth to a child with mental retardation.

This hypothesis is not rejected. Parental age has no significant relative effect on the chances of giving birth to a child with mental retardation. This means a child with mental retardation could be born to parents who are either advanced in age or still within the childbearing age of life. This is in consonance with Brody's (2004) findings that women at both extremes of life could give birth to children with mental retardation. He opined that a teenage mother or a young woman is at the risk of having a child with mental retardation, attributing this to under development in the girl child. Such a young girl who suddenly gets pregnant will probably not have adequate nutrition to support the growing baby and on the other hand a woman who is advanced in age is likely to have a child with mental retardation because of change in her body mechanisms.

Rives, Langlois, Bordes, Simeon and Mace (2002) also agree that parents who are advanced in age could have children with mental retardation. They identified maternal age as a predisposing factor for autosomal trisomies but maternal age effect shows considerable variation among chromosomes. Their study also show that the tendency to have a child with mental retardation increased significantly in elderly male by the age of 39 but more significantly after 41 years.

Conclusively, and as stated earlier, mental retardation cannot be associated with a particular parental age, it can occur at any age of the parents.

There is no significant relative effect of birth order on the chances of giving birth to a child with mental retardation.

This hypothesis is rejected since birth order has a significant relative effect on the chances of giving birth to a child with mental retardation. In this study, children born in the fourth position accounted for 25.9% (Table 4.2) of the total sample used. This could be attributed to multiparity, that is, the number of children the mother has had before the one with mental retardation. In other words, the age of the mother keeps increasing with each child she has and this could predispose the later born child to mental retardation. This is further buttressed by Aboulgher (2001) who linked the relationship between birth order and mental retardation to pregnancy interval. This means the longer it takes for the mother to have a child after another could eventually result in mental retardation. Adler (2004) postulates that the order into which a child is born in a family has direct implication on whether a child is a special child or not. He stated that birth order plays a role in determining the child's level of cognitive functioning.

There is no significant relative effect of socio-economic status on the chances of giving birth to a child with mental retardation

This hypothesis is not rejected since socio-economic status of parents has no significant relative effect on the chances of giving birth to a child with mental retardation. The implication of this is that both parents of low and high socio-economic status have the chances of having children with mental retardation irrespective of their quality of life. The work of Mugno, Ruta, D'Arrigo and Mazzone (2007) lends support to the findings of this study in that they describe socio-economic status as the quality of life of parents of children with mental retardation and this encompasses the house they live in, type of occupation the parents have and their income. These authors opine that the quality of life of parents has no correlation with the birth of a child with mental retardation except where other causes of mental retardation present.

There is no significant relative effect of birth trauma on the chances of giving birth to a child with mental retardation.

This finding as shown in Table 4.9 indicates that birth trauma has a significant relative effect on the chances of having a child with mental retardation. Therefore, the null hypothesis is rejected. This implies that a child who is subjected to trauma while being born can on the long run develop mental retardation. This is further buttressed by the result of Woods (2005) which shows that physical trauma to the brain at birth can cause mental retardation. He further stresses that brain damage may result from birth trauma while the baby is passing through the birth canal. Further, Izuora (1985) identifies high percentage of cases of mental retardation due to birth trauma in Enugu, stating that this only reflects the quality of maternal and child welfare services as well as the obstetric care in the area. Brain damage results from birth trauma because of the immature brain and skull of the child at birth.

There is no significant relative effect of disease on the chances of giving birth to a child with mental retardation.

The results shows in Table 4.9 that disease experienced during developmental stages of life and in the mother have no significant relative effect on the chances of giving birth to a child with mental retardation. Therefore, the null hypothesis is accepted.

The implication of this is that disease cannot be used to predict mental retardation in these children. However, Sutton (2004) finds that there is a definite relationship between mental retardation and a number of medical problems such as cerebral palsy, epilepsy, encephalitis and meningitis. This shows that there are some undetected traumas or infection which may be responsible for mental retardation.

There is no significant relative effect of cultural practices of the parents on the chances of giving birth to a child with mental retardation.

Table 4.9 shows that the null hypothesis is rejected, given that evidence suggests that cultural practices contributed to the etiology of mental retardation in terms of choice of local medication, dietary attribution and traditional beliefs. According to Lambo (1986), several compelling forces, internal and external in health related decisions, compete within the individual to trigger certain behaviours.

Moreover, and undoubtedly such initiated behaviour oftentimes could be risky or detrimental to both mother and child.

There is no significant relative effect of genetic make-up on the chances of giving birth to a child with mental retardation.

Considering the trend of the findings as encapsulated in this study and as depicted in Table 4.9 it shows that the hypothesis on genetic make-up of parents has a significant relative effect on the chances of giving birth to a child with mental retardation. Therefore, the null hypothesis is rejected. Sutton (2004) asserts that the cause of mental retardation is genetic. The most common genetic disorders that have been shown to cause mental retardation are Trisomy 21 and Fragile X syndromes. According to the report from National Institute of Child Health and Human Development, for parents of a child with Down syndrome due to faulty gene, there may be an increased likelihood of Down syndrome in future pregnancies. This is because one of the two parents may be a balanced carrier of the faulty gene. This is further buttressed by Sutton (2004) that the risk for the brother or sister of someone with mental retardation is much higher than that of the general population. According to him, a couple who had one child with mental retardation is ten times more likely to have another. It can therefore be said that outside factors such as disease, injury and social environment play some roles in determining intelligence quotient genetics which is significant, if not the most significant cause of mental retardation.

5.2 Conclusion

Determination of an underlying etiology of mental retardation is an essential part of providing special programmes for the children with mental retardation and a way of reducing stigmatisation among parents of such children. Nigerians still lack adequate a surveillance system that targets incidence and prevalence of mental retardation in the society, the existing surveillance is inadequate for identifying people with mental retardation. The onus is therefore on both the government and stakeholders to find a means of identifying this group of people in the society so as to provide adequate essential services for them as well as programmes that will meet their needs. When parents have an idea about the causes of mental retardation, they are better equipped to accommodate these children in the family without much

stigmatisation attached to them. The fact remains that individuals with mental retardation can live full productive life and quality lives with help from modern medicine and lifetime educational/support programmes.

This study examined the perceived determinants of mental retardation among parents of children with mental retardation in some selected schools in South-Western states of Nigeria. Seven psychosocial variables were used as correlates of mental retardation. These variables are parental age, birth order, socio-economic status of the parents, birth trauma, genetic make-up of the parents, diseases and cultural practices. Four of them that is birth order, birth trauma, cultural practices and genetic make-up of the parents were found to be significant in predicting mental retardation while three others, parental age, socio-economic status of parents and disease were found not to have any significant relationship with mental retardation. However, all these independent variables had a joint composite effect in predicting mental retardation. It is therefore crucial for special educators and parents in particular to be aware of the factors that predispose an individual to mental retardation so that early intervention can be instituted.

5.3 Implication for the Study

The implication of this study was that the independent variables used have high potency for predicting mental retardation.

The result showed that age of both parents at the birth of a child is crucial to determining the mental capabilities of the child. Advanced parental age has a high potency of predicting mental retardation. Married couples should start early enough to have children so as to avoid having children that might have mental retardation.

As part of the implication for this study, genetic make-up of both parents may be relied upon in the attempt to understand the possible determinants of mental retardation. This factor, that is genetic make-up of the parents has been able to show that when one or two parents are carriers of genetically mutilated chromosomes, it definitely affects the outcome of the pregnancy resulting in one form of mental retardation or the other. There is therefore need for parental counselling in order to forestall such situations.

The study is of great value to persons working with children with mental retardation. These include special educators, rehabilitators, social workers or other professionals in related fields. Since the study has been able shed light on the causes

of mental retardation, these professionals can then utilise the knowledge in counselling parents, on their children with mental retardation. Such families need to develop positive attitude towards these children as much as possible and place them in schools where they can learn to become independent in life.

It is of utmost importance that there should be public enlightenment about the causes of mental retardation. This could be done through seminars, workshops, mass media and other forms of publicity.

Finally, the findings have undoubtedly showed that the determining factors of mental retardation, as predictive as they are in the study are part of a new research focus which should be embarked upon in the nearest future to reduce the risk of mental retardation.

5.4 Recommendations

Early marriage should be encouraged especially for women so as to avoid problems that can occur in childbearing as a result of advanced age. Parents especially those living in the rural setting should endeavour to engage in practices that will enhance their quality of life. Pregnant women should avoid taking unprescribed drugs or concoction that maybe detrimental to their health or the life of the unborn child.

Pregnant women should go for early antenatal care so that any abnormality either in the mother or the unborn child can be detected early so that early management can be instituted. Where the abnormality cannot be prevented early intervention can be given to the child.

There is need for parental counselling, public enlightenment programmes, seminars on metal retardation and its etiology. It should be noted that prevention is better than cure; more so, that it is a known fact that mental retardation cannot be cured but can be prevented. The order into which a child is born in the family has been found in the study to have a significant relationship with mental retardation; therefore, parents should make concerted efforts not to compare the developmental progress of siblings in the family. Government should advocate programmes that will enhance health and mental well-being of the general populace thereby boosting the socio-economic status of an average individual.

Further, people working with persons with mental retardation such as special educators, counsellors, resource persons and other professionals should have an in-depth knowledge of the etiology of mental retardation in order to handle adequately

questions, concerns and worries of parents of these children. Not only this, individuals with special needs educators will be able to teach themselves as independent members of the society. Going to school is essential for them to learn not only academic skills but also discipline, social/interactional skills and practical skills for community living.

Early intervention programmes should be commenced for children with mental retardation. This includes an array of specialised programmes and related resources made available to professionals such as special educators, speech therapists, occupational therapists, given that such programmes will optimise development of individuals with mental retardation. Government and other stakeholders should develop and implement a balanced community based education system for the persons with mental retardation.

Policy makers should ensure that national policy on special education is upheld. With the current national reforms in every sector in Nigeria, special education should not be left out of the reforms. Government has the responsibility to provide optimum services to adequately address the problem of mental retardation. This entails strengthening and effective utilisation of existing services in the health, education and welfare sectors. New infrastructure should be created in various states and zones where necessary in Nigeria to facilitate effective services for individuals with mental retardation. The government should build partnership with non-governmental organisations (NGOs) in order to encourage and promote activities that will benefit the children with mental retardation and their families.

The government should allocate resources and develop services in the area of mental retardation needs to be expressed in the form of policy statements and enactment of legislation at the national level. A surveillance system that specifically targets the people with mental retardation and their needs should be developed and implemented by the government. The Federal Government should be more responsive to the unique challenges and needs of persons with mental retardation.

5.5 Suggestions for further studies

This study was carried out in six states in south west of Nigeria, therefore there is need to replicate this empirical study for a larger population in other states of the federation to facilitate a wider generalisation of the determinants of mental retardation. Also, more variables should be included in the study. The groups of

children such as educable, trainable, severe or profound can be compared along with various variables. In this study only the educable ones were explored.

5.6 Contribution to knowledge

This study has contributed to the existing body of knowledge in that it has revealed that advanced parental age in both parent, or in one of them can predispose a family to having a child with mental retardation. A man of above 40 years or a woman above 35 years have a high risk of giving birth to a child with mental retardation. This is contrary to peoples belief that mental retardation is caused by an evil spirit or that it is an invocation from an enemy within or outside the family. This knowledge about the causes of mental retardation will help in modifying the attitude of the general populace towards children with mental retardation, thereby removing the misconception about them.

The study has also shown that as the number of children a woman has increases to four and above, the chances of having a child with mental retardation also increases. It further demonstrated the fact that a woman does not have to undergo difficult pregnancy, labour and delivery before she can have a child with mental retardation. A woman whose pregnancy, labour and delivery are uneventful can equally give birth to a child with mental retardation. Contrary to the view that socio-economic status of the parents can predict mental retardation the study has ascertained that socio-economic status of the parents cannot predict mental retardation. However, on the long run, the study has shown that the joint effect of the variables increases the risk of having a child with mental retardation.

5.7 Limitations of the Study

In this study, scanty literature and very few studies were obtained from Nigeria to back up the literature review. Most of the literature review were from foreign countries. It is therefore hoped that this study will form part of literature base for further studies in the Nigerian context

Further, the study is limited because of the number of subjects covered. The research is limited to only six states in Nigeria, not only this, tracing the homes of these children in order to speak to their parents was difficult. Some of the parents who were illiterates were unwilling to be interviewed.

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APPENDIX

DETERMINANTS OF MENTAL RETARDATION QUESTIONNAIRE

This questionnaire is designed to find out your views about factors responsible for giving birth to a child with learning problems in the family. This information will be treated as confidential. Please, complete the questionnaire properly.

Section A

Personal Information

1. Sex: Male Female
2. Marital Status: Single Married Widowed Divorced

Section B: Parental Age and Chances of Giving Birth to a Child with Mental Retardation

3. Age: 25-29 30-34 35-39 40-and above
4. How old were you when you got married?
18-22 23-27 28-32 33-37 38-42
5. How old were you when you had your first baby?
18-22 23-27 28-32 33-37 38-42
6. How old were you when you gave birth to your child with mental retardation?
18-22 23-27 28-32 33-37 38-42

Section C: Birth Order and Chances of Giving Birth to a Child with Mental Retardation

1. How many children do you have? 1 2 3 4
5 more than 5
2. What position is the child with mental retardation?
1st 2nd 3rd 4th
3. Was there any delay before you got pregnant for the child with mental retardation?
4. Sex of the baby: Girl Boy
5. Is there any other child in the family with mental retardation?
Yes No
6. If yes what position?

Section D: Socioeconomic Status Scale and Chances of Giving Birth to a Child with Mental Retardation

1. Level of Education: Illiterate [] Primary School Leaving Certificate []
WASC [] Teacher Training [] First Degree []
Others _____
2. Occupation: Civil Servant [] Trading [] Self-employed []
Not employed [] Others (please specify) _____
3. Monthly income:
4. What type of house is your family living in?
Flat/Self contained (Personal) []
Flat/Self contained (Rented) []
Duplex building (Personal) []
Duplex building (Rented) []
Shared Apartment (Personal)/ (Rented) []
Single room []
5. Toilet Facilities: Pit Latrine [] Water closet [] Bush []
6. What is the source of your water supply?
Well [] Borehole [] Tap water [] Stream []
7. Do you or your spouse have a car? Yes [] No []
8. If yes how many? 1 [] 2 [] 3 []

Section E: Genetic Make Up and Chances of Giving Birth to a Child with Mental Retardation

1. Is there anybody in your family and that of your spouse that has mental retardation?
2. If there is what is the relationship to you? Brother [] Sister []
Auntie [] Uncle [] Distant relation []
3. Is such a child still alive? Yes [] No []
4. If he/she is alive, how old is he/she
5. Where is he/she?
Attending school [] learning a trade []
cannot learn anything []
6. What in your own opinion is responsible for his/her condition?

Section F: Birth Trauma and Chances of Giving Birth to a Child with Mental

Retardation

1. How many months was the pregnancy before delivery?
7 months [] 8 months [] 9 months [] 12 months []
2. Did you register for pre natal care in a hospital? Yes [] No []
If yes where did you register? State Hospital [] Private Hospital []
Traditional delivery home []
3. How long did it take to deliver the baby?
0-3 hours [] 4-9 hours [] 10-14 hours []
15 hours and above []
4. How was the baby delivered? Normal delivery [] Bottom first []
Forceps delivery [] Vacuum delivery [] Caesarean section []
5. Did the child cry immediately after birth? Yes [] No []
6. If no to question 5, when did the child eventually cry?
5 minutes after [] 10 minutes [] more than 10 minutes []

Section G: Diseases and Chances of Giving Birth to a Child with Mental

Retardation

1. Did you notice any abnormality/abnormalities immediately the child was born? Yes [] No []
2. If yes which of the following abnormalities?
Bluish colour of the baby (pale) [] Small head [] Large head []
Convulsion []
3. Did your child fell ill at any particular period of his/her development?
Yes [] No []
4. If yes which of the following illness
High fever [] Head and neck stiffness []
Inability to digest milk (PKU)[] Unstable head and neck []
Collection of fluid in the head []
5. Did your child fall at the developmental period?
Yes [] No []
6. If yes, how many times did you remember that he/she fell?
Once [] Twice [] Thrice [] Several times []
7. Did he/she sustain any head injury? Yes [] No []

**Section H: Cultural Practices and Chances of Giving Birth to a Child with
Mental Retardation**

1. Did you use any concoction during the pregnancy? Yes [] No []
2. If yes, what type of concoction? Insertion of peccary [] Ingestion []
Bathing concoction []
3. At what stage of the pregnancy did you use the concoction?
1-3 months [] 4-6 months [] 7-9 months []

| S/N | | SA | A | SD | D |
|-----|---|----|---|----|---|
| 1 | The older the parent, the more the likelihood of having a child with Mental Retardation. | | | | |
| 2 | It is the age of the mother alone that determines having a child with Mental Retardation. | | | | |
| 3 | It is the age of birth parents that determines having a child with Mental Retardation. | | | | |
| 4 | If a woman is 35 years and above she is likely to have a child with Mental Retardation. | | | | |
| 5 | A woman between the age of 18 and 35 also can give birth to a child with Mental Retardation. | | | | |
| 6 | The age of the woman has nothing to do with having a child with Mental Retardation. | | | | |
| 7 | Children with the following birth order or position in the family have mental retardation *First Born *Second born *Third born *Fourth born *Last born | | | | |
| 8 | Adequate child spacing of 2-3 years interval will not make the woman to give birth to child with mental retardation. | | | | |

| | | | | | |
|----|---|--|--|--|--|
| 9 | Having many children (5-8) predisposes a woman to having a child with me | | | | |
| 10 | The number of children a woman has,, has nothing with having a child with Mental Retardation. | | | | |
| 11 | Only poor families give birth to children with Mental Retardation. | | | | |
| 12 | Whether a family is rich or poor has nothing to do with birth of a child with Mental Retardation. | | | | |
| 13 | Parents who are uneducated give birth to children with Mental Retardation. | | | | |
| 14 | The type of food a woman eats predisposes her to having a child with mental retardation. | | | | |
| 15 | Geographical location is a factor influencing the birth of a child with a Mental Retardation. | | | | |
| 16 | An educated family can also give birth to a child with Mental Retardation. | | | | |
| 17 | Lack of parental care disposes a child with Mental Retardation. | | | | |
| 18 | The incidence of Mental Retardation is more pronounced among males than female. | | | | |
| 19 | Mental retardation runs in family. | | | | |
| 20 | Mental retardation could be due to inheritance rather than the age of parents. | | | | |
| 21 | A child can be burn with mental retardation if it runs in the family of the woman | | | | |
| 22 | In the family of man | | | | |
| 23 | Inheritance has nothing to do with mental retardation | | | | |
| 24 | Mental retardation is as a result of the injury the baby sustained during or immediately after birth. | | | | |
| 25 | Delay in delivery of the child can lead to Mental Retardation. | | | | |

| | | | | | |
|----|---|--|--|--|--|
| 26 | The following diseases affecting the child during development stages, can lead to Mental Retardation: <ul style="list-style-type: none"> *High fever *Inability to digest milk *Accumulation of water in the head | | | | |
| 27 | Going out at night during pregnancy can make a woman o have a child with Mental Retardation. | | | | |
| 28 | Going out when it is sunny can make a woman to have a child with mental retardation. | | | | |
| 29 | Mental retardation could be caused by evil spirit. | | | | |
| 30 | The use of concoction by the pregnant woman can make her to have a child with Mental Retardation. | | | | |
| 31 | A child with mental retardation is a blessing from God. | | | | |

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