Malnutrition and intellectual development : a comparative follow-up study on the effects of early dietary supplementation in a population at risk.

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TO MY PARENTS

"The greatest evil and the most enduring of all tragedies for the individual and his society lies in the difference between what he was capable of becoming and what he has in fact been caused to become."

Ashley Montagu.
(Devel. Med. child. Neurol., 13: 600, 1971)

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ABSTRACT

This investigation attempted to examine the relationship between malnutrition and retardation of intellectual development by comparing children drawn from the same families who presented varying degrees of protein-calorie malnutrition during infancy. From each of 14 families 4 children were drawn and assigned respectively to the following groups:

a) kwashiorkor group composed of children who had been admitted to hospital during the first 4 years of life for treatment of acute PCM;

b) kwashiorkor control group consisting of the nearest sibling in age to the kwashiorkor child having no history of acute PCM;

c) the fed group consisting of a later sibling in the family in whom malnutrition had been prevented by supplementary feeding during the first 3 years of life; and

d) the fed control group consisting of the sibling in each family just preceding the child who was given supplementary feeding.

A battery of psychological tests was administered when the mean age in the kwashiorkor group was 13.0 years, and 8.9 years in the fed group, with the two control groups taking intermediate positions. On mean full-scale intelligence score the fed group showed a significantly higher difference (p less than 0.01) when compared with the other three groups. The same trend was found for verbal score, although the difference was not quite significant statistically. On non-verbal score the fed group was not significantly higher than its own control group, but was significantly above the kwashiorkor group and kwashiorkor control group. The fed control group was significantly higher than the kwashiorkor group.

Analysis of the sub-scale intelligence scores suggested that whilst nutritional factors play an important part in the determination of the verbal score, an equally important part is played in the determination of the non-verbal score by medical care and environmental stimulation. The sub-test results suggested a relationship between malnutrition and a specific deficit in abstract reasoning and learning abilities, but the conventional measures of 'brain damage' did not discriminate between the four groups. The deficit in intellectual development appeared to be related to the duration of chronic malnutrition rather than to the severity of the insult as indicated by an acute episode of kwashiorkor.

The results are discussed in terms of the failure of compensatory educational programs to provide sustained elevation in the IQ scores of under-privileged children, and the suggestion is made that this failure could at least in part be attributed to the operation of nutritional factors.

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This project owes its inception and completion to the dedicated work of Miss Aileen D. Moodie who has maintained contact and rapport with 123 families over a period of almost 15 years in spite of extremely difficult conditions of accessibility aggravated by the particular mobility and social instability of this population. Her collaboration with this project has made the experience an enjoyable one, due to her enthusiasm and conscientiousness which has made arrangements run smoothly. Her contribution included the compilation of the social and physical data.

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Prof. John D.L. Hansen first stimulated my interest in this field and has continued to do so over the past six years.

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PART I

LITERATURE REVIEW

CHAPTER 1

BACKGROUND AND SIGNIFICANCE OF THE PROBLEM

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BACKGROUND AND SIGNIFICANCE OF THE PROBLEM

In a world in which population is increasing at an enormous rate, we find that 50 - 70% of the population is inadequately nourished. Furthermore, it is this very section of the population that is multiplying most rapidly. Several hundred thousand children are born each day, the majority of whom will be exposed to severe undernutrition during their most formative years. In some areas only half will survive the period of childhood, and malnutrition will be a major contributing cause of death in most of these children. Others will die of infectious diseases which are relatively harmless in well-nourished populations, but which are fatal to children rendered vulnerable by undernutrition (Winick, 1970; Mönckeberg, 1972). Over the past 15 years a better understanding of the bio-chemical pathology of malnutrition has resulted in more effective treatment, and a consequent pronounced reduction in the mortality rate. However, over the same period evidence has been accumulating that an infant who survives a period of severe malnutrition may be seriously handicapped not only in his physical development, but also in intellectual development. This is most likely if the nutritional insult occurs during the period of maximum brain growth (Dobbing, 1970).

Several reviewers have called attention to the self-perpetuating cycle which is set in motion, and which continues from generation to generation with a spiral effect (Cravioto, 1970; Montagu, 1971; Birth, 1972). The malnourished child, developing in a culture of

poverty, is unable to acquire the skills and knowledge necessary for satisfactory adaptation to the increasing complexities of modern society. He remains poor for the rest of his life, and joins a large pool of survivors whose functional level is sub-optimal and who become the victims of their own poor socio-economic environment. It is likely that he will choose a mate from a similarly impoverished background, and his children will be born into and reared under the same conditions. The family does not have the resources to adequately nourish a new infant, who is at birth already at risk under the insult of maternal undernutrition during foetal life (Winick et al., 1973). He in turn becomes seriously malnourished, and if able to survive, is handicapped in a way which may prevent him and his off-spring from extricating themselves from the plight of their parents. The culture of poverty contributes to the poverty of culture, and the condition of poverty is perpetuated and passed from one generation to the next. The genetic endowment is presumably normal, but the environment prevents achievement of the genetic potential (Montagu, 1971). A pool of such handicapped people not only exists but is increasing in almost geometric proportions with the present day population explosion. The socio-economic implications are obvious - the section of the population which contributes least to its own and society's maintenance and development is on the increase both by proportion and in gross number.

DEFINING THE LIMITS OF MALNUTRITION:

In its most specific sense, malnutrition refers to a broad

clinical spectrum of nutritional disease referred to as protein-calorie malnutrition (PCM). The opposite ends of the continuum are represented by kwashiorkor and marasmus respectively, the calorie deficiency in kwashiorkor being not as severe as the protein deficiency. The majority of cases of PCM do not show either the pure syndromes of kwashiorkor or marasmus; they present a mixed picture and occupy an intermediate position on the PCM spectrum, the typical diagnosis being that of marasmic-kwashiorkor.

Whilst the nutritional aetiology of PCM is of paramount importance in the development of the syndrome, it is clear that a complex of interrelated non-nutritive factors contributes significantly to its occurrence. In general the synergism of malnutrition and infection is well recognised. Because of poverty, tradition, or lack of parental understanding, the quantity and/or quality of the food available to the child is marginal or at best inadequate. Since the physical environment is unsatisfactory, the child suffers from repeated and, often, almost continuous episodes of infection. Illness decreases his appetite and undernutrition itself contributes to anorexia. Malnutrition renders him more susceptible to a variety of infections and there is again vicious cycle which accentuates his already precarious nutritional state. Added to this is a frequent lack of timely and appropriate therapeutic intervention, and the illness is prolonged and severe. Finally because the child is ill a large proportion of the time he has little opportunity to learn, and may miss out on critical periods for the development of various motor and cognitive skills (Hegsted, 1972). Psychological and

intellectual stimuli in his environment are minimal, and whether or not permanent intellectual disabilities are imposed by malnutrition per se or by the impoverishment of non-nutritive stimuli is at present an unanswered question.

Emphasis on the relative importance of the various contributory factors to the PCM syndrome has passed through various phases since the nutritional aetiology was realised in the 1940s. Recognition of the nutritional aetiology was naturally followed by a preoccupation with the nutritional factors, and led to the postulation of a direct causal relationship between nutritional deficit and the sequelae of physical retardation and intellectual dysfunction (Stoch and Smythe, 1963, 1967; Mönckeberg, 1968). Most authors begin by discussing 'deprivation' but end up referring only to malnutrition. Cravioto (1970) still questions any emotional basis for the behavioural regression and apathy which is a constant feature of the clinical syndrome, and regards consideration of these as a distraction to research progress. However there appears now to be a tendency among workers in the field towards a more balanced recognition of the interrelationship between nutritive and non-nutritive factors in the aetiology of a wide syndrome of cultural deprivation in which malnutrition is but one aspect coinciding with a lack of usual middle-class benefits. This awareness probably arises out of the difficulties in controlling the non-nutritive variables experienced by all attempts to study the problem in human populations. For example, Mönckeberg who in 1968 wrote with a degree of certainty that permanent brain damage results from early malnutrition, states in 1972 that

"it is obvious that the mental and motor retardation observed in this under-privileged group is not only due to nutritional factors but perhaps to many others, i.e., cultural, educational and socio-economic that affect the intelligence and developmental capacity of the child." (MUnckeberg, 1972).

Except in the instances of cystic fibrosis, coeliac disease and other malabsorption syndromes, malnutrition never exists as an isolated problem. Its co-existence with other debilitating social and environmental factors is now well recognised. The relative aetiological significance of non-nutritive factors in the development of the syndrome is emphasised by Hepner and Maiden in the conclusion of their report on under-privileged children as follows: "An organised, functioning maternal - child relationship promotes normal nutrition even with the rigors and handicaps of poor diet and welfare existence in the slum, and ... a disorganised, poorly functioning maternal - child relationship negates the advantages of adequate nutrient intake to the vulnerable rapidly growing child" (quoted by Hegsted, 1972). This statement harks back to the original description of the causes of kwashiorkor given by Cecily Williams which included not only the loss of the mother's milk, but the loss of the mother herself. The name kwashiorkor meaning 'displaced child', which she gave to the syndrome, speaks for itself (Trowell, Davies and Dean, 1954).

In an investigation of the factors related to the aetiology of undernutrition, Chase and Martin (1970) found a significant relationship between parental separation during pregnancy or soon after birth and undernutrition in the child. These authors suggest that the term

'psycho-nutritional deprivation' could be used for the syndrome in which rejection of the child may be an important aetiological factor. Again Gopalan (1968) failed to demonstrate a significant difference in the diets consumed by children who subsequently developed kwashiorkor or marasmus and those who did not. The supposition that kwashiorkor results from a restriction in total caloric supply is probably an over-simplification of a complex situation, and the development of marasmus on the basis of anaclitic depression in institutionalised infants has received ample documentation by Spitz, Goldfarb, Levy, Ribble, Bowlby, and Dennis, and was reviewed by Yarrow in 1961. Bakwin (1949) and Talbot (1963) found the evidence conclusive for emotional and stimulus deprivation as causes of infants' failure to thrive, and grow and develop normally in childhood. Senn (1961) recognises that the absence of individual personalised nurture has the effect of a disease. The studies of Powell, Brasel and Blizzard (1967) demonstrated growth retardation closely resembling idiopathic hypopituitarism as a consequence of emotional and social deprivation. Even here however it is necessary to heed the warning of Whitten et al., whose study of the relationship between emotional deprivation and growth failure indicated that the parents' perception of the diet fed to their children at home was not always accurate. The children were thus underfed as well as unloved; "the mothers' affects were so consumed by the emotional or social problems that they had not accurately perceived what they usually offered their infants." The perceptual distortion is not unlike that found in anorexia nervosa. (Nutr. Rev., 1970).

A view point similar to that of Chase and Martin is expressed by Montagu (1971) who proposes the term 'socio-genetic brain damage' for the functional expressions in the terms of deficits in behaviour, learning ability and intelligence, of a syndrome characterised by maternal and infantile malnutrition, social deprivation and finally, damage to genetic potential of ensuing generations.

WHY STUDY MALNUTRITION AND INTELLECTUAL DEVELOPMENT?

From the foregoing a connection is apparent between conceptions concerning the aetiology of the syndrome of deprivation and the postulation of its effects. Those who see malnutrition as the most important factor in the aetiology of the syndrome tend to postulate malnutrition as the direct cause of the physical and intellectual deficits demonstrated in its victims. Those who give equal weight to undesirable emotional and social factors in the aetiology of the total deprivation syndrome recognise the relative importance of lack of adequate social, emotional and environmental stimulation in the production of cognitive deficits. Questions relating to the value of attempting to separate the relative strengths of the various contributory factors may be raised.

The first concern in studying the relationship between malnutrition and intellectual development is its implication for prevention and treatment. If malnutrition as such is the single most important causative factor in the development of cognitive deficits in underprivileged populations, then a relatively simple solution to a wide spread problem of enormous consequence would be provided in large scale dietary supplementation. One reviewer comments on this aspect

of the problem as follows:

> "If we are to know whether dietary deficiencies *per se* are the cause of subsequent poor mental performance, then studies will have to be conducted which will separate the nutritional variable from genetic factors and from many environmental variables. High priority should be given to research of this kind because the results have great practical importance for under-privileged people everywhere." (Nutr. Rev., 1969).

Mönckeberg (1972) is pessimistic about achieving this:

> "Because human malnutrition is never an isolated problem, it is almost impossible to analyse separately the importance of each factor and thus obtain a definitive, clear cut answer. The important thing seems to be, not so much to know one mechanism that produces psychological retardation in the lower socio-economic group, but the fact that this situation exists and that it interferes severely with the individual's development and therefore upon society's development."

In 1969 Jensen stirred a controversy by suggesting that the failure of compensatory education programmes in reducing the achievement gap between disadvantaged ('majority') children and middle-class ('minority') pupils was predictable on the basis of genetic, racial differences in intelligence (Jensen, 1969; Eysenck, 1971). The role of nutrition in the production of the phenotype may have been underestimated in the past; perhaps the benefits of compensatory education could be rendered more stable and permanent if combined with more adequate nutrition.

Academic concerns also justify any attempt to single out the variables. It has often been stated that the value of any theory lies not so much in whether it is valid or invalid, but in the amount of research that it stimulates and generates. Another reviewer comments on this aspect of the problem as follows:

> "Research workers should ... be concerned about the false assumptions that are made which indicate that all the facts are known regarding the relationship of malnutrition to mental development. This could stifle or preclude the necessary research being undertaken which will help on so many difficult questions. There is clearly still a need for comprehensive longitudinal studies using a variety of strategies before we can really estimate the role of dietary deficiencies per se in the retardation of intellectual development of children."
>
> (Nutr. Rev., 1971).

Research in this field has already resulted in a large body of information about the normal bio-chemical and morphological development of the central nervous system in man and several animal species. Furthermore there is at present a large group of mentally retarded children whose condition is 'without known aetiology'. Present and future studies may well provide insight and clarification of aetiology leading to more effective treatment and prevention. Only fifty years ago phenylketonuria was an untreatable and unpreventable cause of severe mental subnormality.

The whole field of mental subnormality and 'brain damage' represents common ground for the disciplines of psychology, psychiatry, neurology and paediatrics. In such multi-disciplinary areas of study, progress is fostered by mutual interdependance and contributions from any single discipline have beneficial effects on inter-disciplinary relationships and enrich the entire field. Within the discipline of psychology alone, as in other human sciences, the reciprocal relationship between the experimentalist and the clinician through mutual involvement in a common research area contributes to the development and progress of the science. Theories tested in the laboratory can

be applied in the field, and those developed from direct observation in the field can be tested in the laboratory. In this way unwarranted inter-species extrapolation can be minimised.

Current world-wide research in the field of malnutrition and intellectual development addresses itself to the problem of whether the association between protein-colorie malnutrition in early childhood and subsequent retardation in intellectual development is one of direct cause and effect. If the findings prove to be positive, it will be important to know at what age malnutrition has its most disturbing effect, and what degree of malnutrition in terms of duration and severity results in intellectual retardation. The results of this study will hopefully contribute towards the formulation of answers to these questions.

CHAPTER 2

HUMAN STUDIES OF BRAIN FUNCTION IN NUTRITIONAL DEPRIVATION

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Hypotheses considering an association between early infantile malnutrition and later impairment of intellectual development and function have two sources of origin. Firstly, even the earliest descriptions of kwashiorkor laid stress on symptoms of central nervous system dysfunction during the acute stage of the disease. The descriptions typically include apathy, misery, peevishness, lethargy and anorexia. Improvement in the child's mental state has long been considered a favourable prognostic sign, and is generally regarded as the turning point in the course of the illness. This observation has been strengthened by reports of generalised EEG dysrhythmia during the acute stage of the illness suggesting an encephalopathy, and persistence of focal dysrhythmia long after recovery (Engel, 1956; Nelson, 1959; 1963).

The second source from which these hypotheses derive is the accumulation of animal experimental evidence for reduction of total brain size as consequence of the occurrence of malnutrition during the period of maximum brain growth. For a long time it was accepted on the basis of evidence available from animals studies that post-natal nutritional deprivation that led to an appreciable reduction in the weight of the body and most of its organs, 'spared' the brain in the sense that its weight and chemical composition remained unaffected (Donaldson, 1911). The idea of 'sparing' of the brain has gradually been questioned on the supposition that in

terms of physical development, the brain and central nervous system is not uniquely different from any other organ or system and is likely to follow a biologically comparable sequential development. Abundant evidence indicates a reduction in physical development in terms of final body size as a consequence of malnutrition imposed during the developmental process, and this could logically apply to the brain also. Human studies over the past decade have therefore sought evidence of impairment of intellectual function as an expression of reduced brain size or of structural damage related to previous nutritional insult. Some of these studies have correlated head circumferences as an index of brain size with IQ as an index of brain function.

The relationship between physical growth and intellectual development was reviewed in 1930 by Patterson. A relationship between height and intelligence has since been generally accepted (Tanner, 1966). Fourteen years later Kugelmass, Poull and Samuel (1944) made a clear formulation of the relationship between malnutrition and intellectual function, suggesting a difference between the reversible effects of temporary starvation and possible permanent retarding effects of malnutrition whereby anatomic and biochemical lesions are produced. These authors analysed the effects of nutritional improvement on psychological test performance in 182 children, aged 2 - 9 years. One half of the sample were institutionalised, and the sample was divided into two groups each consisting of 50 normal and 41 mentally retarded children. One group was malnourished at the time of first testing but well nourished at initial and final testing. The groups were equated for chronological

age, IQ and interval between Kühlman-Binet or Stanford-Binet tests. In the previously malnourished group an average improvement of 10 points for retarded children and 18 points for normal children was noted, while no change was found in the initially well-nourished group. A correlation was found between age at first testing and IQ rise: the younger the malnourished child when treatment was instituted the greater the chance of improvement. A sharp decline in the improvement of IQ was found when treatment began after the age of 4 years, suggesting that irreperable damage had occurred. The flexibility of IQ change before this age and the relative lack of IQ change in the older children denotes irreversibility in mental development following prolonged malnutrition.

The resurgence of research interest in the relation of nutritional factors to intelligence and learning over the past decade is a reflection of the fact that, whilst malnutrition remains a major world public health problem, its most significant consequence is no longer death but the survival of massive populations of intellectually handicapped subjects. The socio-economic and political implications have already been discussed. One reviewer goes so far as to suggest that whereas physical stunting of growth is not undesirable because subsequent nutritional needs are reduced without apparent reduction in adult physical function, and death of preschool children due to malnutrition is the most widely used method of population control, the possibility that malnutrition in childhood has lasting effects on human mental potential would call for a re-assessment of economic priorities in that nutrition programmes may prove to show a greater return on developmental capital through

the creation of a more adaptive and capable work force (Klein et al., 1971).

FIELD STUDIES:

The pioneering and most frequently cited field study was semi-longitudinal and begun by Stoch and Smythe in Cape Town in 1955. The authors selected 20 of the most grossly undernourished (marasmic) Cape Coloured children that could be found. Birth weights were said to have been normal apart from 3 children who were below 2.5 kg. The subjects were matched for age and sex with 20 children whose early nutrition had been supervised in a creche. The groups were comparable in respect of parental intelligence, but there was a very marked disparity in their living conditions: "alcoholism, illegitamacy and broken homes were the rule in the undernourished group, whereas the control group lived under more stable home conditions. Only six of the parents of the undernourished group were gainfully employed." Intelligence, head circumference, height and weight were recorded approximately every two years. Significant differences on all these variables were found consistently through to the 11th year of follow up reported in 1968. The authors regard the reduced head circumference together with the defects of visuo-motor and pattern perception on the non-verbal sub-tests as evidence that lower intelligence scores in the undernourished group may be due to organic brain damage resulting from gross undernutrition in early infancy.

A follow up study of marasmic children in Yugoslavia was reported by Cabak and Najdanvic in 1965. The children had been admitted to hospital for marasmus between the ages of 4 and 24 months of age during

the years 1951 - 1957. Chronic diseases of the CNS and tuberculosis were excluded, and no child was more than 73% of the expected weight at the time of admission to hospital. One-third of the group had parents who were army officers or in the professions; the rest were children of skilled or unskilled workers living in an urban area. Thirty-six children aged 7 - 14 years were tested on an adapted Binet-Simon scale. Fifty percent of the group scored within the 'normal' limits of 91 - 110. One-third scored in the 'below normal' range of 71 - 90, and the remaining 6 children had IQ scores of 70 or below. Thus half the group were below the limits of normal intelligence and the difference between the number of children having normal and sub-normal intelligence was significant. No local standards for the dispersion of IQ were available, so the results were compared with those of Serbian children, of whom 21% were below normal intelligence as against 50% of the undernourished group; and 32% of Serbian children were above the range of the undernourished group. The mean IQ of the undernourished group was 88, compared with a mean of 93 for children from families of 'non-qualified' workers. The undernourished group did not differ significantly in height or weight from the local healthy school children. No correlation was found between the age of illness and the subsequent IQ, but there was a correlation between the deficit in the expected weight for age on original admission and subsequent test performance.

A similar study on Indonesian children reports comparable results in 1967 (Liang, et al., 197). The subjects ranged in age from 5 - 12 years and were from families of the lower socio-economic level, living in rural surroundings. Of the 107 children in the sample, 46 had been

classified as malnourished during an earlier investigation in 1957 - 1959; of these, 17 had shown in addition symptoms of Vitamin A deficiency. The remaining children were regarded as healthy. Intelligence quotient was assessed using the WISC and the Goodenough technique. It was found that children's intellectual development, as well as their physical development, could be predicted with a high degree of accuracy on the basis of their nutritional status during the pre-school years. The lowest IQ values were found in children who had been malnourished and had shown clinical signs of Vitamin A deficiency during the 2 - 4 year age period, and the higher scores were attained in those who had never been diagnosed as malnourished. The average IQ for the whole sample was low on both tests, the scores on the WISC and Goodenough being 75 and 77 respectively. The coefficient of correlation between the two tests was 0.58. There were 10 cases where the Goodenough score was more than 10 points lower than the WISC score, and 13 cases with the reverse situation. When the results were analysed with reference to nutritional status at the time of testing there was a good correlation between I.Q. and clinical condition. The group rated 'poor' was much below the group rated 'fair'; the mean IQ for the 'satisfactory' group was the highest but was not statistically different from the 'fair' group. Analysis of the data with reference to expected height for age revealed a 12 point difference on the Goodenough test in favour of the children above the 95th percentile of the Indonesian standard. There was little variation in IQ up to the 95th percentile of standard weight, but above that weight were a significantly

larger number of children with IQ above 90 on the Goodenough test. There was a significant difference in the average score of the groups below and above the 95th percentile of standard weight in both WISC and Goodenough tests. A more significant difference was observed if the children were grouped according to their weight in 1957 - 1959. At the time one-third of the children were at expected weight for age, and this group was an average of 14 IQ points higher than those who were below their expected weight for age at the beginning of the study. EEGs were performed on 43 children and no relation between EEG and nutritional condition was found. Thirty per cent of the total had a mildly abnormal pattern, but the proportion was no higher in the malnourished than in the healthy children.

Perhaps the most complete studies were those that have emanated from Cravioto and co-workers in Mexico since 1960 (Cravioto and Robles, 1965). Reports by Geber and Dean in Uganda, Berrera-Moncada in Venezuela and Robles in Mexico all indicated that undernourished children tested with the Gesell scales showed a marked retardation in all four areas of behaviour sampled by the technique. A further study by Berrera-Moncada showed that intelligence tests given 2 years after discharge to 20 rehabilitated cases who were all older than 2 years 10 months at the time of admission showed normal IQ scores. On the basis of these findings Cravioto and co-workers hypothesised that the effect of severe malnutrition on the mental development of human infants would vary as a function of the period of life at which malnutrition was experienced. To test this hypothesis all severely malnourished children admitted to the nutrition ward of the Hospital Infantil de Mexico during 1959 were studied.

Twenty infants hospitalised for severe PCM were examined on the Gesell scale every two weeks during treatment and rehabilitation. On the first examination, conducted just after the acute electrolyte disturbance and infectious processes had been corrected, all infants scored considerably below age norms. During rehabilitation 14 children who had been admitted for treatment between 15 and 42 months of age came progressively closer to age expectation. However, the developmental deficit shown on admission by the 6 infants whose malnutrition led to hospitalisation before the age of 6 months was still present after complete nutritional rehabilitation. Cravioto (1970) also quotes unpublished data of Brockman and Ricciuti who studied cognitive functioning in a group of 20 Peruvian children aged 12 - 42 months who had been hospitalised because of severe marasmus when they were between 2½ and 42 months of age. The test performance of the infants recovered from malnutrition was significantly below that of a control group of children who had no history of severe malnutrition and who were taller than the children recovered from severe marasmus. Further evidence cited is from a study by Pollitt and Granoff who compared 19 marasmic children and 8 siblings on the Bayley Infant Scales of Mental and Motor Development. All were below 3 years of age at the time of study and the siblings were of similar age but with weights, heights and head circumference within normal limits. It was found that while the siblings were developing according to age expectations, 17 of the 19 children recovered from marasmus had severe mental and motor retardation.

Cravioto (1970) derives the general conclusion from these studies that "protein calorie malnutrition occurring in the first year of life, if severe enough to markedly retard physical growth and to force the infant to be admitted to a hospital, may have adverse effects of mental development. If the duration of the untreated episode is longer than 4 months, particularly during the first months of life, the effects on mental performance may be so intense that it will produce severe mental retardation incompletely corrected by nutritional rehabilitation." In subsequent studies Cravioto (1970) compared children who had been hospitalised because of severe malnutrition before the age of 30 months with the sibling closest in age to the index case. Viewing the human organism as an agent that processes information, and recognising the importance of learning to read and write, and that these skills are dependant on an adequate development of auditory-visual and visual-kinesthetic intersensory integration, Cravioto studied these functions according to the techniques of Birth and Belmont rather than making use of conventional tests of intellectual function. The functions of intersensory integration had the additional advantage of showing clear-cut developmental cause in normal children during the first school years. The group was stratefied according to social class into high, middle, and low urban class, and rural low class. On auditory-visual integration the siblings in the low urban social class had scores below those corresponding with children of the same age but of a better socio-economic condition. The improvement in auditory-visual competence with age was obvious for all social classes and for rural and urban environments. When the performance

age by age of siblings and index cases was compared, it became apparent that children recovered from severe malnutrition were well below their siblings, thus scoring well below their expected values for their class. Equivalent results were obtained in a study of visuo-kinesthetic intersensory integration.

In 1966 Cravioto and associates published the results of a study in Mexico which related height to inter-sensory organisation (Cravioto, Delicardie and Birch, 1966). A cross-sectional study was carried out on all primary school children aged 6 - 11 years living in a rural village of Guatemalia. Malnutrition was defined retrospectively on the basis of height for age: a child who showed significant diminution of stature relative to his age in the total village population was assumed to have an increased likelihood of having been at risk of early malnutrition. In this way an experimental group was identified representing the lowest quartile of the height distribution and was compared with the group consisting of all children in the tallest quartile for age. Information on the stature of the parents of both groups was obtained to control for genetic factors. A second control sample drawn from an upper class urban population was selected consisting of children of the same age who exhibited the equivalent differences in height but who had little or no likelihood of ever having been at nutritional risk. Broad details on the social, economic and educational state of all families was collected. For the rural children a difference in height was accompanied by a difference in the inter-sensory integrative ability. This relationship was not found in the upper class urban sample. The authors

discussed the findings in terms of two possible schemes, viz., 1) that both inter-sensory development and malnutrition arise out of social impoverishment where poor stature is an incidental sequel to the malnutrition, and 2) that both poor inter-sensory development and low stature are sequelae of malnutrition, itself arising out of poor social conditions. On the basis that poor inter-sensory functions and low stature were not significantly associated with adverse social conditions, the authors favour the second scheme, thus relating reduced inter-sensory ability directly to the possibility of early malnutrition as guaged by reduced height for age.

The Wechsler Intelligence Scale for Children in its Mexican form was administered to 37 index cases and 37 siblings. Full scale IQ's averaged 13 points lower for the index cases than for their siblings. Verbal and performance IQ were also significantly lower in the previously malnourished group. Cravioto concludes as follows:- "irrespective of the presence or absence of a previous admission to hospital because of severe malnutrition children developing in this milieu have a higher probability of showing poor performance on intelligence testing, as well as in other types of testing relating to basic mechanisms for learning. The presence of an episode of malnutrition occurring early in life and of enough severity to force the child into the hospital increases the chance of scoring in the low range" (Cravioto, 1970).

Using the same methods for the assessment of inter-sensory integration together with a suitably constructed intelligence test battery, Champaken et al (1968) studied Indian children who had been

treated successfully for kwashiorkor in infancy. Nineteen children between the ages of 8 and 11 years were tested, having been admitted during the period 1959 - 1962 at the ages of 18 - 36 months. For every experimental child three matched control children were selected taking account of age, sex, religion, caste, socio-economic status, and educational background. In addition the intelligence test was standardised on a group of 50 children of the same age range and derived from the same socio-economic group and geographical area as the experimental and control groups. This difference was particularly marked in the younger age group (8 - 9 years) and tended to diminish in the older age group (10 - 11 years). Inter-sensory organisation was poorer in the experimental subjects - markedly so in the younger age group, with a tendency to improve in the older age group. The deficits were particularly marked with regard to perceptual and abstract abilities. Heights, weights, and head circumferences of the experimental children were generally similar to those observed in the matched controls, though the weights tended to be somewhat lower in the experimental group. Intelligence score, head circumference and height were correlated in the control subjects aged 8 - 9 years, but no other significant correlations were found. Although the findings are clear cut, the authors are extremely cautious in their interpretation and question whether they are directly attributable to the earlier episode of PCM. Further factors they suggest are 1) whether the prolonged immobilisation before and after hospitalisation resulted in a loss of 'learning time', together with the emotional stress and anxiety incidential to the hospitalisation.

2) Whether the poor performance of the experimental group is merely a reflection of the low level of intelligence, motivation and resourcefulness - factors which determine the development of full-fledged kwashiorkor in the children.

In 1968 Bothe-Antoun reported an investigation of 22 children aged 4 - 5 years, whose early nutritional status had been well defined. Both experimental and control groups were drawn from a large sample who took part in a longitudinal growth study from birth to 3 months of age. Children in both groups had normal births and normal neo-natal periods. The nutritional status of most children was satisfactory while they were breast-fed in the first 3 months of life. Thereafter PCM appeared in the experimental group and all children in this group fell below the third percentile for height and weight at some stage during the first 18 months of life. Mean differences of IQ on a revised Standford-Binet Test were found between the experimental and control groups and the difference was reflected in both verbal and performance scores. The groups also differed significantly in age of starting to walk and talk, but the scores were within the normal age range on the Gesell Scale. Although intelligence scores of the two groups of parents did not differ significantly, the authors recognise that the experimental and control groups were exposed to qualitatively different environments and maternal attitudes.

Sibling controls were used in a study of the effects of kwashiorkor and intellectual development in Cape Coloured children in 1968 (Evans, 1969; Evans, Moodie and Hansen, 1971). Forty children between the ages of 9 and 14 years were studied, having

been admitted to hospital with kwashiorkor between the ages of 10 - 49 months. The control group consisted of 40 siblings aged 8 - 15 years, each being the nearest in age and of the same sex as the experimental subject but without any history of acute nutritional disease. On the New S.A. Individual Scale (South African equivalent of the WISC) no significant differences were found between the experimental subjects and controls on full scale intelligence score, verbal score or non-verbal score. There was a positive correlation of 0.62 between subjects and siblings on the full scale scores. A low negative correlation between intelligence score and degree of social pathology was found. A significant difference was found between subjects and controls on the Harris revision of the Goodenough Drawing Test. (p less than 0.02). Of 14 drawings selected as indicating marked emotional disturbance by Machovers criteria, 9 were from the experimental group and 5 from the control group. A low negative correlation occurred between the age of the child on admission to hospital and the full scale intelligence score in the experimental group. When the data were analysed according to age of admission to hospital, the difference between the experimental and control groups was not significant in either of the two sub-samples being admission age 10 - 15 months and admission age 16 - 48 months respectively. There was however a significant difference between both the subjects and siblings of the 2 sub-groups, with significantly lower scores in the late onset group. Because the sub-samples were not paired or matched, comparisons were not possible. Inter-group differences in test scores were therefore sought and

significant differences between the late-onset kwashiorkor subjects and their siblings were found on the Harris-Goodenough score and the Arithmetic Problems sub-test of the NSAIS. This finding correlated with significantly poor scholastic achievement in the late onset sub-sample when compared with the control group. This study concluded that whilst an acute episode of PCM did not significantly lower intelligence score, the possibility that prolonged nutritional deprivation retarded intellectual development had not been excluded. This retardation could have been caused by interference of emotional factors in the children's cognitive processes.

Another sibling study was reported by Hertzig et al. in 1972. Seventy-four Jamaican boys who had been treated in hospital for severe infantile malnutrition during the first 2 years of life were tested on the WISC at the age of 5 - 10 years. These were compared with 38 male siblings (i.e. children having the same biological mother as the index case). In addition to the sibs a class-mate or index case being the nearest in age of the same sex was selected. This resulted in 71 matched pairs of index and comparison children. For full scale, verbal and performance IQ the index cases have the lowest scores, the sibs occupied intermediate position, and the comparison children had the highest scores. The index children were 7 - 9 points lower than the comparisons and each of the differences was statistically significant (p less than 0.001). For full scale IQ, 23% of the index cases compared with 7% of the comparison children performed at or below the floor of the test. (p less than 0.01). The proportion of minimally

functioning cases in the sib group was not significantly different from either the index or comparison groups. No correlation was found between age of hospitalisation and IQ, and Pearson correlations indicated a random relationship between the age at hospitalisation and the IQ at school age. There was no significant difference in the IQ between the two groups of children admitted between the ages of 8 - 12 months and 13 - 24 months respectively.

Another prolific writer on the subject is Mönckeberg, who in 1968 expressed certainty in his conclusion that permanent brain damage results from early malnutrition. Fourteen Chilean children admitted to hospital between the ages of 3 - 11 months were followed after treatment, correction of the undernutrition being attempted by an adequate free supply of milk to the families. The children were tested by Binet and Gesell methods when the age range of the group was 3 - 6 years. The average Binet IQ was found to be 62 and in no case above 76. This was significantly less than the average of Chilean pre-school children of low socio-economic level. On the Gesell test, only 1 child attained the 42nd month in all 4 areas. The best development was usually in the personal-social area; the most retardation was seen in language. Head circumferences were definitely below normal; heights were all below average (3rd percentile) whereas weights all were found to be above average (3rd percentile). The relation of weight to height was above normal, in some cases so much as to give the impression of obesity. The author attributes the low test scores of the group to brain damage directly resulting from a disturbance of protein synthesis during brain growth, and likens the effect of

malnutrition during the first month of life to that of hypothyroidism, galactosaemia, and phenylketonuria. The irreversibility of the brain damage is seen in the lowness of the scores despite improved nutrition as indicated by the above average body weights.

Mönckeberg derived further evidence from a study of the relative influence of social class and malnutrition on psychological deficit. He tested 153 pre-school children within three social groups. Group A included middle-class children; Group B and Group C were from the lower class with the same average income per capita and similar educational levels of parents. Group B was from a population with no malnutrition, due to free distribution of milk and medical assistance over the period of 10 years. The proportion of children with normal intelligence was essentially similar in Groups A and B, whereas in Group C only 50% were normal. The author recognises that a supplemental feeding programme affects the environment, maternal motivation, and nutritional state, so that the differences in intelligence level cannot be ascribed entirely to the nutritional factor.

A further study of the epidemiological type was published by Mönckeberg and co-workers. The group consisted of a random sample of 220 children aged 1 - 5 years drawn from the slum area of Santiago where living conditions and housing were sub-standard. Under-nutrition in this group was prevalent with different degrees of growth retardation, but none of the children had oedema or severe malnutrition and none had a weight of less than 60% of standard for age. Group B consisted of 90 children from the

Chilean middle-class with a similar age range to Group A. Their nutritional status was considered normal according to the Iowa standards with weight and height not less than 80% of the standard. Developmental quotients were derived on each child from scores on the Gesell and Termen-Merrill scales, and the intellectual levels of the mothers was obtained from an abbreviated form of the Wechsler-Bellevue Scale. A nutritional survey was made in every home, with a chemical analysis of samples of food consumed during a seven day period. There were substantial differences between the two groups in terms of family income, educational status of the parents, and especially in the intellectual level of the mothers, which showed a significant difference (p less than 0.001). The percentage of the children with an IQ within normal limits for their age was also significantly different (p less than 0.001). Group A children had significantly lower developmental quotients in all areas of function tested. In order to determine whether a relationship existed between the DQ and nutritional conditions, two sub-groups from Group A were allocated according to their nutritional status: those whose weight for age was below the 3rd Iowa percentile and those whose weight for age was over the 10th Iowa percentile both showed a marked difference in DQ in every area. Even children with an acceptable nutritional condition (weight above the 10th percentile) presented lower values in all areas than pre-school children belonging to Group B. However a significant correlation was observed between growth retardation and low intellectual level in Group A and also between the frequency of retardation of psychomotor development and the amount of animal protein consumed by each

child. There was also a positive correlation between deficit in head circumference and DQ level. This correlation ceased to be significant when cranial growth was normal. A further significant correlation was noted between the maternal IQ and nutritional status of the child (β less than 0.001). Mönckeberg states that: "we cannot assert definitely that undernutrition is a factor in the low development quotient" and "it is possible that malnutrition could be a concomitant factor and not the cause of an impaired development quotient." He concludes by noting that "the degree of retardation was directly related to the amount of animal protein consumed and also to the retardation of physical and cranial growth. The results, although they do not allow the formulation of definite conclusions, suggest that chronic undernutrition in the child acts in a negative way upon mental and psycho-motor maturation." It appears therefore that Mönckeberg accepts a statistical correlation between two variables as indication of a causal relationship, and does not allow for the possibility of the operation of a third independent variable - with the statement that "it is almost impossible to analyse separately the importance of each factor and thus obtain a definitive, clear-cut answer."

By far the most penetrating study to date is that of Chase and Martin in 1970. Nineteen urban slum children aged 2 - 3½ years were studied having been admitted to Denver General Hospital between 1962 and 1967 at less than 1 year of age with a primary diagnosis of generalised undernutrition. The sample included two sets of twins, one dizygotic and one monozygotic. Children with any

evidence of organic disease leading to undernutrition, and those with neo-natal difficulties were excluded. With the exception of one child from each pair of twins, birth weights were all above 2 kgs. Nineteen children of similar socio-economic status to the test children were chosen as controls on the basis of birth date, weight, sex, race, and having been born and followed at the same hospital. In addition the siblings of both control and test children were screened on the Denver Developmental Screening Test. Maternal intelligence was estimated with the vocabulary sub-test of the Wechsler Adult Intelligence Scale. Child/parent interaction was evaluated in the children's homes using the Home Stimulation Inventory Scale, and social conditions were evaluated in the home by means of the San Mateo County Family Social Functioning Scale. All families were rated by the same social worker on the basis of direct observation and enquiries. The index and control children were tested blindly on the Yale Revised Developmental Examination. At a mean of $3\frac{1}{2}$ years after admission for both control and test children the developmental quotient was significantly lower in the test children (p less than 0.01). The test children were lower in all five areas of the developmental quotient, but were particularly low in language. Control children were also low in language but not as impaired as the test children. These children admitted to hospital with a duration of under-nutrition exceeding the first 4 months of life were mostly severely impaired with a mean DQ of 70. All 5 areas of development were statistically lower than either the control children or the children admitted with undernutrition before the age of 4 months (p less than 0.01). The DQ of the test

children admitted to hospital within the first 4 months of life was not significantly different from that of the control children. Physical growth also correlated negatively with the duration of undernutrition in the first year of life, with the children admitted to hospital after the age of 4 months of age particularly low in all indices on follow up (Height, weight, head circumference). The group admitted to hospital after the age of 4 months contained significantly more children below the 3rd percentile in weight and height ($\bar{p}$ less than 0.001). Differences were not significant for weight or head circumference between children admitted prior to 4 months of age and the control children. In social functioning the families of the test and control children differed only in the category "health status of family other than the patient" ($\bar{p}$ less than 0.025). A similar number of families in both the control and test groups demonstrated severe problems in community relations, family relations, and economic functioning, but the Home Stimulation Inventory Scale discriminated between the two groups and the difference was significant ($\bar{p}$ less than 0.01). There was no statistical difference between the values for mothers of children admitted prior to the age of 4 months and mothers of children admitted after that age. The proportions of control and test mothers with low intelligence was not significant. Thirty-three siblings of the test group and 19 control siblings were examined. No significant differences between the two siblings groups were found for height, weight and head circumference. Of 27 pre-school test siblings assessed on the Denver Developmental Screening Test, three failed and in two of these

cases other siblings in the family besides the index case were known to have been undernourished. Of 17 pre-school control siblings assessed on the DDST there was one failure. Apparently no comparisons were made between the nourished or control children and their siblings.

The authors address themselves to the investigation of factors relating to the aetiology of undernutrition and attempt to discover why the test children were allowed to become undernourished to the point of requiring hospital treatment. Maternal age, parental height and educational attainment, maternal low IQ, and social deviant behaviour occurred with similar frequency in the two groups. However the number of siblings under the age of 2 years at the time that the index case was born was greater in the test than in the control families. Parental separation during pregnancy or soon after birth occurred with a higher frequency in the test families, and in four out of eight cases the index child bore the exact name of the absentee father. There was also a higher evidence of unwanted pregnancies in the test families than in the control families. The authors conclude that: "five factors were found that probably resulted in environmental stress to the mother at the time of the undernutrition: parental separation; alcohol-related problems; inadequate finances; larger family; and the stress of one or two additional children under 2 years of age." The eight mothers of test children whose husbands deserted them during the pregnancy or soon after the birth were all believed to have been emotionally upset at the time that the child became undernourished. The authors recognise an analogy between

child-battering and infantile undernutrition in a select proportion of cases, and in view of the multi-factorial problems in the production of undernutrition suggest that the term 'psycho-nutritional deprivation' could be used to describe these children.

METHODOLOGICAL PROBLEMS

The investigations reviewed above illustrate the difficulties attending the study of the relationship between malnutrition and intellectual development. Cobos (1972) conceptualises the difficulties under three headings as follows:

1. Lack of precision in the definition of malnutrition.
2. Occurrence of malnutrition within the context of massive deprivation.
3. Lack of precision in the measurement of psychological deficit.

Nutritional status is generally established anthropemetrically by measuring deficits in physical growth. However, the variations in anthropometric measurements and their relation to the effects of nutrition are not fully understood. Some studies (Cravioto, 1966; Stoch and Smythe, 1963) have selected children who showed poor growth and were therefore assumed to have been chronically malnourished. Cobos suggests that a more accurate way of assessing nutritional status would be to measure food intake. This however, particularly in a retrospective study such as Monckeberg's, is likely to be unreliable. Protein-calorie malnutrition is usually blamed for deficits in psychometric scores, but it is recognised that it is extremely difficult to isolate pure forms of protein deficit without associated deficits in other nutrients. The study

of Liang (1967) is the only one which singled out a specific vitamin deficiency, and it is interesting to note that the subjects deficient in Vitamin A showed a greater deficit in IQ score compared with those who had suffered from PCM alone.

All authors have recognised that malnutrition does not occur in isolation but exists as part of a wider syndrome of deprivation. No-one has succeeded in controlling the multiple variables that independently affect both orthodox tests of intelligence and other psychometric tests of cognitive function. The use of siblings is probably the most satisfactory way of achieving this control, but as noted introduces the problem of chronic sub-clinical malnutrition in the siblings. Furthermore, the study by Chase and Martin (1970) has clearly shown that the status quo within the family is not necessarily maintained from sibling to sibling, and that psychodynamic variations within the family are in fact among the very factors which determine the occurrence of an episode of acute malnutrition in a particular child within a chronically sub-nourished family.

Lack of precision in the measurement of psychological deficit refers to the possibility that malnutrition affects differentially several areas of psychological function. This differential impact may be further complicated by the possibility that the severity of the insult as well as the age at which it occurs in relation to critical periods of behavioural and/or organ development may determine the significance and the reversibility of the psychological deficit, and also the particular area of behavioural function affected. Apart from the usual difficulties in the definition of

intelligence and the recognised intricacies of the methodology of measurement, the populations under consideration are generally sub-cultural and trans-cultural, and the tests applied are in general designed for, and standardised on, culturally different populations. To avoid the necessity of applying test norms derived from a culturally different population, various types of control groups have been selected which in turn have introduced the problems of the effects of socio-cultural background on test performance, as for example in the Stoch and Smythe study where the control group had the benefit of nursery school experience. The scientific value of this and other studies is further reduced by the fact that assessments were not conducted blindly. Canosa (1971), reporting on two pilot studies, observed that tests of short-term memory which had previously discriminated between well- and undernourished children failed to do so when the children's group assignment was not revealed to the psychologists administering the tests.

The need for a comprehensive model of malnutrition is stressed by Cobos who proposed that the multi-factorial causation of an unbalanced food intake, that leads to the identifiable alterations in body measurements by which malnutrition is diagnosed, should be thought of as a complex system of interactions or a network of subsystems. This could be represented in a simplified manner as follows:-
Nutritional status (food need/food intake) = f ((FA),(H),(Ps.F),(F.F.),(S.F))
This indicates that the perceived nutritional status is the outcome of biological processes depending on the equilibrium between food needs and food intake, the latter being a function of, at least, food availability (FA), physical health (H), psychological (Ps.F)

and familial (F.F.) and sociological (S.F.) factors.

The elements in this system are related to each other and to the total system in ways not yet well specified. Each element in itself is also a sub-system, the exact composition of which is generally unknown. For example the availability of food is usually linked to factors such as economic status, family size and food habits, which in turn may be related to aspects of social disorganisation such as alcoholism, parental separation and unemployment. Malnutrition is thus the result of the mutual interaction of many factors, some or all of which are present at various levels in varying degrees.

A detailed analysis of the functions of food and the effects of decrease in such functions is provided by Cobos, in terms of which food has mechanical, biochemical, physiological, psychological, and sociological significance. In terms of this analysis, psychological test performance may reflect the effects of food reduction at mechanical level as poor development of perception; at a biochemical level as impairment and alteration in CNS function and structure; at a physiological level as increase in the risk of infection and illness with consequent reduction of concentration ability; at intra-individual psychological level as the activation of mechanisms to increase survival with heightened attention to the 'self' and less attention to the extra-individual factors; at an inter-individual psychological level as maternal deprivation with consequent damage to all areas of psychological development and primary socialisation; at the intra-familial psychological level as syndrome of family deprivation leading to damage of psycho-social

development and secondary socialisation; and the sociological level as identification with a marginal group and impoverishment of psychological and social environment and performance.

THE EFFECTS OF FOOD AND STIMULATION DURING REHABILITATION

Cobos (1972) describes the design of a proposed study known as the Harvard-I.C.B.F.-Cornell Project. The project aims to explore the question of whether malnutrition causes deficit in defined psychological areas, and if possible to clarify the extent to which the psychological deficit may be prevented or reversed by food supplementation. An attempt is being made to discover whether the possible nutritional causes of psychological deficit can be distinguished from other causes by determining the nature of the interaction between nutritional and other non-nutritional factors affecting psychological function. A basis for the design is the well-known fact that siblings have highly correlated psychological scores, a fact which has been confirmed in the pilot study. A further basis for the design is that in the local area in which the project is being conducted, it is a common finding that a child who is malnourished has one well-nourished sibling. Food supplementation of the family should lead to a recovery of the malnourished child and the prevention of malnutrition in the well-nourished child. This allows a comparison to be made between the sibs in order to identify the effect of a history of malnutrition. In order to separate the effect of birth order from nutritional effects, different groups of siblings will be studied. The Griffiths Scale of Mental Measurement will be used to derive a "Cognitive Developmental Quotient."

Preliminary results of the pilot study published in 1972 indicated that various combinations of the social variables significantly accounted for a considerable part of the variance in the individual psychological test scores, and suggested a considerable overlap between the nutritional scales and the combinations of sociological variables. However even after the measured social variables had been accounted for, nutritional factors had an impact on the psychological test scores. The study promises to be the most definitive one so far undertaken.

A study of novel design and with particular relevance to the present study is that of Yaktin and McLaren (1970, 1971). Preliminary findings of the study conducted on Arab children in Beirut were published in 1970. The study aims to assess the improvement in behavioural development during recovery from marasmus, with and without the added effects of stimulation during the rehabilitation period. Thirty children in the study all suffered from acute and severe nutritional marasmus at the time of their admission to hospital and ranged in age from 2 - 16 months. These children were divided into 2 experimental groups matched for age and sex. One group consisting of 17 children was provided with an enriched environment and referred to as the stimulated group. They were treated in decorated wards and provided with toys and music. Nursing staff were instructed to play with them, sing to them, and establish a warm nurse-child relationship. In contrast the 13 children in the unstimulated group were treated in an ordinarily furnished hospital ward of about the same size. Both groups of children received identical medical and dietary treatment. At the beginning of the

study the mean age of the stimulated group was 33 weeks and of the unstimulated group 30 weeks. The mean weights were 33.0% and 52.9% of the 50th percentile of the Boston standards respectively. All children were tested over a period of four months at 2 week intervals on the Griffiths Mental Development Scale. The stimulated group had a mean DQ of 51 at the beginning of the study, and the unstimulated group a mean DQ of 46; the difference was not significant. Significant correlations however were found between the DQ and weight ($p < 0.01$) and between DQ and head circumference ($p < 0.001$). Both groups showed improvements in the DQ scores as they recovered physically, the stimulated group to a significantly greater extent than the unstimulated group. At the seventh testing session the mean DQ was 70 and 75 for the stimulated and unstimulated groups respectively. At the eighth session the mean for the unstimulated group dropped to 66 whereas the mean for the stimulated group rose to 78. The authors do not explain this change in direction, which raises questions about the reliability of the test employed. Over the entire period the greatest improvement was in the area of personal-social function in both groups. At the end of the observation period the greatest remaining deficit was in the area of hearing and speech.

In a more recent paper (Yaktin et al., 1971) the authors report on a stimulated group of 28 children, an unstimulated group of 27 children and a control group of 35 healthy children from the same community and matched in age and sex, but significantly different from the experimental groups in terms of weight, being 106% of the standard. At the initial test session the control group had a

mean DQ of 106 in contrast to 52 for the stimulated group and 49 for the unstimulated group. Analysis of variance was carried out using 20 subjects from each group. The performance of the control group was significantly superior to both experimental groups (p less than 0.01). A difference between stimulated and unstimulated groups became significant only from the third session (p less than 0.05); at the 4th to 7th session the difference increased (p less than 0.01) and at the 8th session the difference became less pronounced (p less than 0.05). Again the greatest deficit appeared in the language area. There was no significant difference between sessions in the control group. When analysed in terms of age on admission to hospital, 30 children admitted before the age of 6 months had a mean DQ of 82 on discharge, 18 children admitted over the age of 12 months had a mean DQ of 61 on discharge. The differences between the three age groups were significant (p less than 0.01) indicating a relationship between duration of malnutrition and severity of impairment.

Follow-up experiments of 33 experimental children and 20 controls are being continued at 3 monthly intervals. The results indicate that the two experimental groups became progressively more similar, the control group remaining more or less constant. At one year follow-up, four previously stimulated children had a mean DQ of 83, and five previously unstimulated child had a mean DQ of 81, compared with 5 control children with a mean DQ of 105. The significant difference in mean DQ between children who were less than 6 months of age on admission and children from the older age group remained significant. The results suggest that any benefits accrued from stimulation are lost when the child returns to the home environment.

A similar study is reported in abstract by Vitale (1970). Over a three year period there was a significant increase in the Gesell DQ of children between 1 - 4 years of age fed food supplements and provided with medical care. A comparable age group provided with only medical care showed no significant increase. Coupled with the increase in DQ was an increase in head circumference, statistically greater in the food supplement group. Medical care and medical care plus food supplement had varying effects on the reduced DQ of children aged 6 - 16 years on Porteus, Goodenough, and Raven Tests. There was no increase, a slight decrease and a significant increase for medical care and medical care plus food supplement groups respectively. However only the medical care plus food supplement group had a significant increase in the DQ on Dearborn Scale. The infant mortality rate and the death rate for the 1 - 4 year group fell significantly without any apparent effect of food supplement.

CHAPTER 3

ANIMAL EXPERIMENTS IN NUTRITIONAL DEPRIVATION AND NEURAL DEVELOPMENT

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More than 50 years have elapsed since Jackson and Stewart (1920) published their findings on the effect of varying periods of inanition and subsequent nutritional rehabilitation on new-born and weanling experimental animals of several species. One of their main conclusions was that nutritional deficiency imposed shortly after birth had permanent and obvious effects on the subsequent growth of the animal, whilst transient malnutrition occurring at a later age depressed growth only for the duration of deprivation without affecting ultimate size after nutritional rehabilitation. The stage of development at which the young animal ceased to be vulnerable to permanent impairment of the growth potential could not be precisely defined and evidently varied amongst species; however it usually occurred shortly after the end of the normal suckling period. Since then it has been demonstrated experimentally in many species that if an animal is deprived of the essentials for growth during critical stages of post-natal development, it will remain physically retarded, notwithstanding later correction of the dietary deficiency. (Waterlow, Cravioto and Stephen, 1960; Widdowson, Dickerson and McCance, 1960). McCance has shown in experimental animals that whether malnutrition has temporary or permanent effects on body size depends on the severity, duration, and timing of the insult. Differences exist between species, but in general "the earlier in its development an animal receives a

nutritional set-back, the less likely it is to reach its full genetic stature." (McCance, 1968). The critical phase of nutritional vulnerability can thus be identified with the period of maximum growth, which in many infrahuman multiparous species takes place predominently during the pre-natal period of development.

Hypothetically this knowledge can be applied specifically to the growth of the brain and development of the CNS. The concept of 'sparing' of the brain from the effects of nutritional insult based on the work of Donaldson (1911) was challenged in a series of pioneering investigations during the 1960's by Dobbing, Widdowson, and Davison which demonstrated that both severe and modest degrees of nutritional deprivation experienced by the animal at a time when its nervous system was developing most rapidly resulted in reduced brain size and in deficient myelination. The deficits were not made up by subsequent nutritional rehabilitation (Birch, 1972).

Two main overlapping hypotheses about the vulnerability of the developing brain have already been extensively tested. Dobbing (1970) states that "the brain is likely to be most vulnerable to permanent restriction from undernutrition during the period of the 'brain growth-spurt', the transient period during which it is passing through the rapid phase of its sigmoid growth trajectory." Dobbing adds two corollaries, firstly, that the nearer the growth-spurt, the milder need be the undernutrition to produce permanent restriction, and the further away, the more severe until in the adult there is no great effect on brain composition from very

severe starvation. Secondly, in view of the different rates of growth in different brain regions and along different parameters, undernutrition at any one stage will produce different effects in relation to the regional velocity obtaining at the time. Another main hypothesis is proposed by Winick and Noble (1966) that "undernutrition during the growth period in any tissue when cell division is taking place will lead to permanent reduction in the numbers of cells attained. Later undernutrition, at a time when growth consists mainly of an increase in the size of cells, will result in a reduction only of cell size and this will be recoverable on restoration of normal nutrition." This would explain the phenomenon of 'catch-up' on rehabilitation subsequent to relatively late onset deprivation.

Dobbing (1972) sees a superficial resemblance between these hypotheses and the hypotheses relating to critical periods in the development of motoric and cognitive skills during which experience and environmental stimulation have an optimal effect. The critical periods for imprinting, socialisation, and acquisition of cognitive skills are described by the developmental behaviourists as periods of increased activity in that the organism is more susceptible to developmental progress. By contrast, the use of the term by neurobiologists refers to sensitivity in terms of vulnerability of developmental phases to interference with developmental processes. The field of teratology has accumulated knowledge of very precise moments of susceptibility during periods of organ development at which specific insults may result in gross disturbances of final shape and form. In experimental animals, viral disease, drugs,

specific nutritional deficiencies, X-irradiation, and even a short episode of mild pyrexia, will produce deformities in the CNS depending on the precise timing of the insult in relation to embryological events.

The growth and maturation of an organ at cellular level comprises four stages:- proliferation, migration, differentiation and death. This sequence of events is observed in all parts of the brain, but the timing differs from one cell type to another, and from region to region. The cellular composition of the brain is relatively heterogeneous, and this contributes to an extremely complex pattern of development when the brain is regarded as a whole (Herschkowitz and Rossi, 1972). Velocity curves for brain growth have been constructed for a variety of animal species using whole brain weight, total brain cholestrol and tissue deoxynucleic acid - phosphorus (D.N.A.-P) content as indices of development by Dobbing (1970, 1972). The major brain growth-spurt may be said to begin in all species at about the time the adult number of neurons is virtually achieved: by the end of the second trimester of gestation in humans (25 weeks), or at birth in the rat. This point however cannot be regarded as the end of cell multiplication, and the heterogeniety of brain cells, and the different times at which they divide causes difficulty with the cell division hypothesis. Neurons multiply very early, mainly before the oligodendroglia begin to do so. Thus adult members of neurons are virtually achieved before the growth-spurt begins. However, the greatest number of brain cells are the glia, and these begin their own mitotic growth-spurt after neuronal division has ceased and concurrently with the growth of dendritic processes and

connectivity. Experimental testing of both hypotheses so far has imposed nutritional restrictions at a stage later than the period of neuronal division, and the cell deficits achieved are therefore likely to be almost exclusively glial. If glial number is a functionally important brain parameter, this would be important and might account for subsequent specific deficits of myelination; but if the period of rapid bio-synthetic activity concerned with myelination is in itself vulnerable, this would open up the further possibility that other non-mitotic processes will be vulnerable simply because they are occurring rapidly at the time. The consequent establishment of synaptic connections, which are not directly concerned with cell division, but are important in the growth of neuronal processses, may be interfered with as a direct consequence of interferences with the previous division of glial cells.

The broad generality of the vulnerable period hypothesis has been effectively tested and proved in animals; and its corrolaries that a vulnerability related to the rate of growth implies a comparative invulnerability before the brain growth-spurt, and an inviolate adult brain, have been well demonstrated. Furthermore it can be shown that growth retardation in animals does not alter the timing of the growth-spurt in the brain, but causes a reduction in the extent of the process, which nevertheless occurs at its proper ordained time. Thus none of the individual velocity peaks of wet weight, DNA or cholestrol accumulation are shifted; they are reduced in height, and because no 'catch-up' occurs on the restoration of ad libitum food, the effect is a permanent reduction. It seems clear then that the brain is an organ which has a once-and-for-all

opportunity to accomplish certain important processes of development. If this opportunity is lost it can never be fully recovered (Dobbing, 1970).

Whilst Zamenhof (1968), Winick (1968) and Dobbing (1968) have shown that permanent alterations in the number of cells and their distribution among brain structures are produced only when the organism is malnourished during the period of rapid growth of the CNS, biochemical and physiological abnormalities have also been reported. Enzymatic maturation and development in the brain is also affected; Chase et al. (1967) have demonstrated defective enzyme organisation in the brains of malnourished animals.

In order to avoid confusion in the interpretation of evidence from different species and to allow speculative extrapolation to man, it is important to consider that the critical periods of maximum organ growth occur at different times in different species but demonstrate an identical pattern. Thus in pig's brain, growth and differentiation occurs most rapidly in the period prior to birth, whereas in the rat the most rapid growth occurs when the animal is suckling. Deprivations that are experienced at the same chronological ages and life stages will therefore have different effects in different species. Thus deprivation during early post-natal life will have little or no effect on the brain's size and structure in an organism whose brain growth has largely been completed during gestation. Conversely intrauterine malnutrition is likely to have only minor effects in species in which the most significant period for brain growth occurs post-natally (Dobbing, 1970).

Attempts have been made in experiments to select an animal species where the brain growth characteristics are known, and nutritional deprivation has been imposed at the supposed vulnerable time for that species. The majority of experimental work has attempted to exploit the post-natal timing of the growth-spurt in rats during their suckling period. The most widely used design retards growth by increasing the size of the suckling litter so that when weaned, the rats are about half normal weight. They are then given ad libitum food until maturity, and the mature brains are then examined and compared with the brains of animals fed well until weaning in an average size litter, but undernourished later by restriction of food.

An alternative experimental design involves the underfeeding of the mother during the pregnancy. Stunted offspring as well as normal control newborns are then cross fostered at birth to well-fed and undernourished lactating females, the litter size being standardised. Thus four groups are achieved according to whether the gestational period or the suckling period or both or neither was nutritionally restricted. Any of these groups can be weaned to a restricted or adequate diet, and in this way all combinations and permutations of growth restriction can be imposed in the pre-growth spurt, the growth spurt, or the ppst-growth spurt periods.

Numerous experiments based on the above two designs indicate clearly that deficits in brain growth are confined to those animals underfed in the suckling period. Growth retardation confined to the bodily growth-spurt which occurs later than that of the brain does not induce permanent stunting of either the whole body or the brain.

It has been recently shown that the timing of the several major components of the brain growth-spurt is determined by chronological rather than developmental age (Dobbing, 1972). Vulnerability of the growth programme may thus reside in failure in growth-retarded animals to accomplish proper growth at the fixed chronological age which is their only opportunity.

The effect of nutritional deprivation during gestation have been demonstrated by Nelson and Evans in 1953. A significant decrease in reproductive performance of rats occurred when dietary protein was reduced to 5%. If the protein restriction was commenced after a delay of seven days after mating, the effects were not as severe. In 1964, Venkatachalam and Ramanathan showed that a 7% protein diet during gestation only, resulted in reduced growth and a 40% post-natal mortality. Taking the above findings a step further, Zeman and Stanbrough (1969) demonstrated that a 6% protein diet during gestation resulted in reduced DNA, RNA/DNA and Protein/DNA ratios in 16 and 20 day foetuses, and newborns. Compared with controls fed a 30% protein diet a primary reduction in cell number in most tissues occurred in the malnourished animals. Similar results were obtained by Zamenhof et al. in 1968. An 8% protein diet fed from before mating through gestation resulted in reduced DNA concentration at birth compared with control animals fed a 27% protein diet.

The work of Winick and co-workers (1966, 1967, 1968, 1969) has demonstrated clearly the effects of nutritional deprivation during infancy in animals. In general the findings in rats have demonstrated that increase in litter size reduces cell number (total size) in all organs. Refeeding after weaning has been ineffective in

improving the DNA content. A restricted protein diet from weaning to 42 days of age resulted in reduced weight, total protein, DNA and RNA in all organs except lungs and brain, where cell size was smaller but total number of cells was unaffected (i.e. total DNA was not reduced). Refeeding of such animals allowed the brain and lungs to regain normal size, but other organs showed no recovery. Restricted diet from 65 to 86 days of age reduced total protein and RNA but had no effect on DNA, indicating that cell size was affected but that cell number remained unchanged. These findings lead to the general conclusion that the brain appeared to be irreversibly damaged only by nutritional deprivation prior to weaning.

The effects of nutritional deprivation after weaning was further investigated by Dickerson and Walmsley (1967), who confirmed that rats undernourished from weaning to 11 weeks of age could be rehabiliated successfully in 2 - 8 weeks, and thereafter showed no reduction in DNA/protein concentration in the brain, indicating normal cell size. Guthrie and Brown (1968) found that brain cholestrol levels were normal if nutritionally deprived animals were rehabiliated immediately after weaning, but remained irreversibly depressed if undernutrition extended to 5 weeks of age. This suggests a second vulnerable period during myelination after which the brain becomes resistant to dietary deprivation. Dobbing in 1968 confirmed that a 45% reduction of body weight in mature rats achieved by imposing a 5 week starvation diet had no effect on brain weight. These findings suggest that the mature brain, unlike the developing brain is indeed 'spared' under severe nutritional deprivation.

The experiments so far reviewed have not taken into account regional differences in the relative timing of the growth-spurt. Although overlap occurs, it is well recognised that development of the brain stem is completed relatively early and development of the cerebrum relatively late with the cerebellum taking an intermediary position. If it is accepted that the periods of increased neuronal hyperplasia, glial multiplication, and myelination represent periods of increased susceptibility, then timing of the nutritional insult takes on a selective significance. The cerebellum passed through its growth-spurt at the same time as the rest of the brain, but its relative rate of growth is very much faster. Thus the effect of generalised growth restriction is imposed selectively upon the especially fast growing cerebellum. This has been well demonstrated by Dobbing (1970) who exposed the heads of infant rats of varying ages to X-radiation.

Regional brain differences in vulnerability to malnutrition have been studied by Culley and Lineberger (1968), Howard and Granoff (1968), Cheek et al. (1969), and Chase et al. (1969). Restricted feeding affected most severely the weight and DNA of the cerebellum: when body weight was improved by refeeding from 57% below normal to 17% below normal, the cerebrum achieved a weight 7% below normal, whereas the cerebellum remained 22% below normal in weight. This indicates that a reduction of cell size occurred in the cerebrum, but that cell number in the cerebellum had been reduced. Fish and Winick (1969) demonstrated that the brain stem was little affected at 6 - 21 days, but the cerebellum was again more affected than the cerebrum and hippocampus in rats reared in large litters.

Altman (1971) suggests the distinction of three critical phases in the development of the mammalian nervous system after birth. These three phases are related to the formation of three major classes of cells which arise from three proliferative sources during three successive periods of growth. The three major cell types are 1) macroneurons, 2) microneurons, and 3) neuroglia. Macroneurons are long-axoned nerve cells that form the afferent, relay, commissural, and efferent elements of the peripheral and central nervous system. As such they are responsible for conveying sensory messages from the receptors to the CNS, for relaying afferent impulses from one brain region to another, and for transmitting motor commands from the brain to the effector organs. These macroneurons form the gross circuitary or 'course wiring' of the nervous system. The microneurons are short-axoned interneurons with regional or local input and output connections. It is presumed that their function is primarily modulatory or associative in nature, and they represent the 'fine wiring' of local brain regions. The neuroglia are supportive elements which are responsible for the myelination of axons and for linking the differentiated neurons with the fine capillaries of the brain. As such they are responsible for the sustenance and fixation of the developed brain circuitry. Macroneurons, microneurons and neuroglia tend to differentiate in any brain region sequentially, and it is possible that a hierarchic inductive relationship controls their successive regional differentiation. For example, the microneurons of the cerebellar cortex migrate and differentiate after the Purkinje cells with which they establish synaptic contacts, have begun to differentiate. The differentiation of neuroglial cells, as judged by myelination of

the cerebellum, is the last event in the maturation of this brain region.

It is of great practical importance that in altricial mammals (e.g. the mouse, rat, cat, or dog) the macroneurons differentiate largely during the period of embryonic development; the bulk of the microneurons come into existence post-natally during the suckling period; the bulk of the neuroglial cells are formed during the early post-weaning period. Accordingly, nutritional deprivation during the suckling period could be expected to inflict less harm on the elements already formed, viz. the long-axoned neurons, but would interfere with the formation of the microneurons and thus the 'fine wiring' of the brain. The great reduction produced in the cell population of the cerebellar cortex by early malnutrition in animals supports this view. Nutritional deprivation during the early post weaning period would interfere with the final stage of the maturation of the brain such as myelination, by preventing the formation of neuroglia.

The use of chemical indices for components for brain structure provides a comparatively simple method of assessment and is now widely used. Thus total tissue DNA provides an index of total cell number, and DNA concentration per gram of fresh weight is an index of cellularity. Cholestrol content represents myelination; RNA/DNA ratio and protein/DNA ratios are used as an index of cell size. Difficulties, however, occur in the interpretation of these indices, and their use is sometimes of questionable value. Thus Granoff (1969) indicates that a deficit in cholestrol per mg. of tissue might reflect reduction in either myelin or total cell

surface area associated with a selective suppression of certain neuron types, and is not necessarily an irreversible interference with myelin synthesising cells as suggested by Winick (1969). Similarly Wigglesworth (1969) suggests that impairment of myelination may be due to a decrease in the number of cells available and lowered activity of enzymes involved in myelin synthesis. Furthermore Dobbing (1970) points out that there are many areas in the brain (including the brain stem) where much of the protein content is related to the axons of cells in a completely different region. Protein/DNA ratio is therefore difficult to interprete in a tissue where cell processes are numerous and where some cells measure a metre in length. Nevertheless the use of the various indices has made experimental investigations in animals possible and must therefore be applied to defining normal human brain growth and the investigation of the effects of nutritional deprivation on the development of the human CNS.

CHAPTER 4

HUMAN STUDIES OF BRAIN GROWTH IN NUTRITIONAL DEPRIVATION

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The first detailed description of the organs of undernourished children was given by Parrot, a French paediatrician who studied the famine victims of the Paris Commune in 1870. He reported fatty degeneration of the brain, and abnormalities of other organs (Naeye, 1970). More sophisticated studies of early childhood undernutrition have only recently been undertaken, and are still incomplete, probably due to the fact that the arrival of severely malnourished children on the autopsy table is now occurring with a diminishing frequency. As noted previously, the fact that more and more undernourished children survive has given rise to interested concern about long-term effects of early malnutrition. The emphasis of research is no longer on death but on possible long-lasting effects of survival.

Hypotheses considering the possibilities that malnutrition in infancy may have an effect on the structural development on the brain and CNS either quantitatively or qualitatively derive from an increasing body of information on the effects of malnutrition on physical growth.

Physical growth retardation following infantile malnutrition has been thoroughly documented in numerous populations. There remains however some doubt about its permanence, and in human studies at least, there is some evidence for the occurrance of 'catch-up' growth which is self-stabilising and occurs with high velocity during rehabilitation until the child's natural trajectory is

reached. A pre-pubertal growth-spurt may also occur (Garrow and Pike, 1967; Moodie et al., 1971).

For many years the concept of 'sparing' of the brain and CNS was generally held, but more recently it has been suggested that the brain is not fundamentally different in its growth pattern from other organs; the CNS is not a perfectly insulated system which grows well independently of its particular needs for nutritional support (Birch, 1970).

However the brain is a peculiar organ in that eventual neuronal cell number is defined early in life. Thus the brain reaches its final cell population number whilst most other tissues, organs and systems are continuing to engage in cell replication for considerably longer periods of time (Birch, 1970). A further distinctive feature of the brain is its relative functional indispensibility in comparison with other organs. Whole sections of gastro-intestinal tract or large proportions of liver may be structurally damaged or surgically removed without serious consequences for the functional capacity of the individual. In contrast, it is virtually impossible to interfere significantly with brain structure without producing pronounced alterations in the functional integrity of the organism (Coursin, 1967). Studies of human brain growth by Dobbing (1968, 1970, 1972) have demonstrated clearly the occurrance of a human brain growth-spurt with essentially the same sequential characteristics as those found in other mammals.

Data (Dobbing, 1970) from the analysis of 200 normal human brains have allowed three main conclusions: 1) DNA estimations reveal a separate growth-spurt from 15 - 20 weeks of gestation

which is distinct from the main period of cell division. The latter extends from about 30 weeks of gestation to at least the end of the first post-natal year, and probably until 18 months of age. The early growth period is almost certainly neuronal; the later glial. 2) Regional differences in growth rate and the general sequence of events in human brain growth are identical with those in all animal species studied except for the timing of events in relation to birth. The wide species variation in this has already been noted and must always be considered in the interpretation of the effects of nutritional insult. 3) The major growth-spurt period in the human brain is predominantly post-natal and applies particularly to the bulk of myelin which has to be laid down before the arrival of the glial cells which make it. These findings indicate that in the human infant neuronal cell number is most probably fully defined before the end of intra-uterine life. Thereafter cell replication is that of glial cells through the first 9 months of life, the process terminating by the end of the first year. However, myelination continues for many years thereafter, as does the proliferation of dendrite branchings and other features of brain organisation, and adult brain weight in humans is achieved during adolescence (Dobbing, 1970; Winick, 1970).

Jackson pointed out in 1909 that in atrophic infants the brain continues to grow, so that emaciated infants can have normal brain weight. Donaldson (1911) formulated the concept of 'brain sparing' on the basis of evidence which indicated that brain weight was unaffected in the presence of an appreciable reduction of body weight achieved by post-natal nutritional deprivation. Supporting evidence

for the concept of 'brain sparing' was provided by Brown who showed in 1965 that although the brains of malnourished children at post-mortem weighed absolutely less than those of the well-nourished children, when the brain weight : body weight was calculated the malnourished group had higher ratios. Alleyne and co-workers (1969) reported similar findings in a post-mortem study of 11 children who died of severe PCM. The brain contributed a larger percentage to the total body weight in the more severely malnourished children, and the authors repeat the suggestion that the brain is relatively well preserved in malnutrition. However, the water content of the brain was higher than in other tissues, and the brain : body weight ratio may be a reflection of over-dydration of the brain, or could be explained by relatively greater wasting of the body. Thompson (1970) points out that changes in body or organ weight may reflect changes in body water and fat and have nothing to do with growth; the above results may therefore be misleading.

The most striking evidence against the concept of 'brain sparing' is provided by Winick and Rossd (1969) who compared the brains of 9 children who had died of severe malnutrition before the age of 12 months with the brains of 10 well-nourished children who had died acutely of accidents or poisoning, and brains of two foetuses obtained from therapeutic abortions. The children who had died from severe malnutrition were all below the 3rd percentile for both height and weight and showed clinical evidence of severe nutritional marasmus. The brains of the 9 severely marasmic children all had reduced weights and reduced quantities of DNA, RNA and protein. In three cases the DNA content was only 40% of that expected,

but these three had weighed less than 2 kilograms at birth. In all cases the DNA/RNA and DNA/protein ratios were normal, suggesting a reduction in the number of brain cells in the malnourished children. It appeared that the younger the child when he developed malnutrition, the more marked the effect on the brain. The data indicate that very little cell division took place from the time these children were exposed to severe malnutrition.

A study of the brains of four malnourished infants aged 2 - 22 months is reported by Fishman et al. (1969). In three of the infants birth weights ranged from 2.5 to 2.8 kilograms and the effect of intra-uterine malnutrition can therefore not be separated from malnutrition during infancy. Estimations of cerebral lipids closely linked to myelin membrane were consistently reduced in the white matter of malnourished brains when compared with controls.

Studies of brain growth and prematurity provide further relevant information reviewed by Birch (1972), Thomson (1972), and Winick (1973). In populations of undernourished women placentas are smaller and contain fewer cells, and some of the tissue changes associated with undernutrition in animals have been found to occur in the placentas of undernourished women. Data concerning the effect on the foetus are meagre and indirect, but show that in small-for-dates infants from undernourished populations who died shortly after birth the cell number was reduced in all organs including the brain. In infants who died during the first year of life of severe undernutrition, by far the greatest deficit in brain cell number was present in the group weighing less than 2 kilograms at birth. When placentas from infants with intra-uterine growth

failure from well-nourished populations are studied, they can be divided into two groups: in one the placentas are normal in size and contain a normal number of cells even though the infant is small; all of these infants had a major congenital malformation. In the second group the placentas were small and had a reduced number of cells; all these infants were physically normal except for their small size.

The first situation is caused by intrinsic abnormalities which manifest in both growth failure and congenital defects of structure and form; the placenta is not affected. The second was caused by extrinsic factors (other than malnutrition), possibly by placental vascular insufficiency. The cellular defects in these infants having small placentas were similar to those produced in rats by ligation of a branch of the uterine artery with consequent reduction of blood supply to some of the foetuses. A 20% reduction in body weight at term was accompanied with a 50% reduction of liver weight whereas brain weight was not reduced at all. The brain contained its full complement of cells whereas liver cell content was reduced to 50% of expectation. Only mild transient changes in tissue enzyme activity occurred in the brain in contrast to marked lasting changes in placenta and foetal liver. Similar effects have been demonstrated in monkeys when placental vascular insufficiency was achieved by partial placental ablation (Winick, 1973).

A study by Davies and Davis (1971) divided 120 infants with birth weights below 1.5 kilograms into two groups, viz. those who were small-for-dates (SFD) and those whose birth weights were

appropriate-for-dates (AFD). A comparison of food intake and body temperature while in the nursery showed that under more optimal post-natal environmental conditions, the growth of the AFD group was capable of 'catch-up', while the retardation (produced in utero) of the SFD group was not reversible. This clearly focuses attention on the foetal environment during the second and third trimesters of pregnancy. In this study the indices of growth were height, weight and head circumference, but no psychological test evidence was provided for the assertion that "reversibility of retarded brain growth in infants of low birth weight depends on gestational age at the time of birth and the vigour of post-natal care." Another study by Naeye and co-workers (1971) of organ and cellular growth in different groups of infants born in New York City showed that the growth of the foetus of the poor mother had been impaired. A racial classification did not account for the differences in organ sizes between poor and non-poor groups, except that the mean brain weight of infants born to poor Black mothers was significantly less than that of the infants of the Puerto Rican or White mothers. The cellular abnormalities observed were similar to those reported as resulting from placental insufficiency, and maternal undernutrition was identified as the important probable factor in the insult to the foetus. This may be related to reduction of pituitary growth hormone (Nutr. Rev., 1972). The effect of gross maternal starvation such as occurred in Leningrad and Holland and Japan in the Second World War was a significant fall in infant birth weight, but there was no apparent effect on the length of gestation. Birth weight increased to pre-warclevels with post-war economic improvement (Altman, 1971).

This evidence indicates that the foetus is not a 'perfect parasite'; foetal growth as indicated by birth weight is impaired by maternal nutritional deprivation, and the foetus is not entirely spared (Thomson, 1970). Nor can it be stated with confidence that the brain is fully spared over and above other organs and systems when the organism is exposed to nutritional deprivation during pre-natal or early post-natal growth.

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CHAPTER 5

ANIMAL EXPERIMENTS IN THE STUDY OF BRAIN FUNCTION IN NUTRITIONAL DEPRIVATION

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Whilst the evidence of biochemical and structural alternations in the brains of experimental animals that have been malnourished during critical developmental periods is clear-cut, the importance of these changes in terms of CNS function and the behaviour of the animal has not been clearly established. In human studies very little data are available concerning the effects of malnutrition on physico-chemical structure, but there is considerable evidence which suggests that brain function, in terms of performance on conventional tests of intellectual functioning and specific tests of perceptual motor functioning, is interfered with in children who experienced malnutrition during early infancy. Some of the evidence so far suggests that there is a relationship between the age at which the nutritional insult occurred and the degree of functional deficit, but a definite causal relationship between the malnutrition and the subsequent functional handicap has been difficult to establish. Apart from difficulties in interspecies extrapolation there are major difficulties in forming cause-effect relationships between deficits of brain structure and deficits in brain function, and between the occurrance of malnutrition and subsequent deficits in cognitive function.

The majority of investigations so far have been conducted either by paediatricians with an interest in psychological functioning or by psychologists with a bias towards behaviourism. Both favour a mechanistic viewpoint. This explains the attempts of

most workers to find a direct cause-effect relationship in mechanistic terms between deficient nutrition and structural deficit on the one hand, and between structural deficit and functional deficit on the other. If we accept Birch's (1964) definition of 'brain damage' as referred to "a behaviour syndrome and not to the fact of 'brain damage' as such", then we can hypothesise that the functional expressions of this type of brain damage are deficits in behaviour, motivation, learning ability, and intelligence, produced not only by the organic, biochemical or somatic aspects of malnutrition. (Montagu, 1971). The paremeters of this type of 'brain damage' are difficult to delineate in humans; behavioural expression of cortical and even sub-cortical functions in man is both qualitatively and quantitatively far removed from some of the animal species studied indicating a need for the utmost caution in interpretation and extrapolation. Clarke (1968) states: "It is possible that, with rapid maturation in a hostile natural world, early learning may have a very different function in animal as opposed to human development." Added to this is the difficulty introduced by the well recognised fact that severe growth retardation together with the syndrome of behavioural, intellectual, and cognitive retardation which closely mimics the syndrome frequently associated with malnutrition, can result from social and emotional deprivation in the presence of an adequate diet (Bakwin, 1949; Powell et al., 1967). Interpretation of the animal experimental work is further confounded by the well known work of Rosenzweig, Krech, Bennett and Diamond (1968) and Levine (1968) demonstrating the effects of early stimulation on CNS development.

Experiments aimed at examining the influence of early drive deprivations upon later behaviour have been conducted by psychologists using several animal species over a long period of time. Investigations of this type reviewed by Bronfenbrenner in 1966 have shown in general that the deprived animals have an increased drive for food or water, which is particularly noticeable if the animals are not satiated at the time of testing (Barnes, 1967). The tendency towards an increased drive for food or water lessened or even disappeared completely on resumption of an ad libitum diet if the food deprivation had been initiated twelve days or more after weaning. Several investigations led to the conclusion that the drive deprivation, rather than altered learning capacity, enhanced the animals general activity level and its ability to solve drive-relevant problems. Another type of drive deprivation involves an increase in the size of litters being nursed by a lactating female rat so that the suckling pups are relatively deprived of milk. Adult animals that were exposed to this feeding frustration were found to be more successful in competition for food, and hoarded significantly more food than control animals. D'Amato reared rats in litter sizes of 4, 8, and 16 per nursing female and using rewards of drinking water and sucrose solution found that litter size had no effect on either discrimination or discrimination-reversal learning when tested soon after weaning or in early adulthood. Lát et al found that rats raised in litters of three were physically more active, moved about and groomed themselves more frequently, than rats reared in litters of 15 - 20.

Attention was given to the specific nutritional effects of

early food deprivation in 1926 by Anderson and Smith, who fed a lysine deficient diet to a group of rats from 45 days of age, and artificially underfed another group with a diet adequate in protein. Both the protein restricted and the underfed rats made more errors in a maze than the controls. After refeeding with a good diet, the differences in maze performance disappeared. Bevan and Freeman (1952) fed a diet low in lysine from 30 days of age and found no difference in maze performance with a group of rats whose food intake had been restricted so as to provide a body weight equal to that of the lysine-restricted group. A study by Andriasov in 1952 indicated that when the regular diet and the reward diet were the same, there were no demonstrable differences in maze performance between rats fed low protein and normal protein diet. In 1954 Griffiths and Senter reported that rats receiving a protein-free diet were superior in maze performance to rats being fed a protein-rich diet when the protein-rich diet was used for reinforcement. If the protein-free diet was used as a reward, the deficient rate showed inferior performance.

An early study by Cowley and Griesel (1959) has provided one of the clearest demonstrations of impairment of learning behaviour in rats following on nutritional restriction. These investigators fed groups of male and female rats on diets of nutural food ingredients which differed in percentage of total protein; the high protein diet contained 20.1% protein, the low protein diet contained 12.9% protein. The rats were tested in Hebb-Williams dry maze which employed a food reinforcement. No significant difference in the maze performance was found between the original groups of rats, but

after breeding, the first filial generation showed a highly significant lowering of scores in the male rats receiving the low protein diet. No significant differences were found in the females. Because the two diets were fed throughout the life span of the animals, it is possible that the effects on behavioural performance were due in part to maternal deprivation, although this was minimised by ad libitum feeding throughout the preservation of litter configuration. It is also possible that the differences in maze performance were due to differences in drive: the rats receiving the higher protein diet not only showed better maze performance scores, but their lapsed time to reach the goal was much shorter and there were fewer signs of emotionality, which suggests that an increased drive in these animals may have led to their superior performance.

In order to avoid the confusion of the use of a food reinforcement in animals that have an altered drive for food as a consequence of early nutritional deprivation, some investigators have made use of the water maze for measuring learning behaviour. In 1936 Berhardt fed diets containing casien in proportions varying from 0 - 17% to groups of rats. When tested in a water maze, the rats receiving the 0% casien diet made more errors than those receiving the 17%, but the differences were not significant. Pilgrim et al. (1951) compared rats fed on low and high protein diets from the 34th to the 50th day of life. No significant difference in performance was found on water maze trials at 74 days of age.

A further approach to the investigation of the effect of early malnutrition on later behaviour has been the attempts to stimulate

human phenylketonuria by feeding excessive phenylalanine to young animals. A review by Karrer and Cahilly in 1965 indicates that attempts to develop a permanent behavioural abnormality by feeding phenylalanine to rats for short periods of time after weaning have been unsuccessful; the results have been variable when the phenylalanine was fed during early post-natal life, but a positive result was obtained when excessive phenylalanine was fed to female rats both before and after parturition. This result was in agreement with the hypotheses of increased vulnerability during critical periods of development reviewed in Chapter 3. However, the behavioural changes resulting from a dietary excess of phenylalanine in rats could be attributed to specific toxic effects of phenylalanine metabolites rather than reflecting an influence of PCM.

The relationship of specific vitamin and mineral dietary deficiencies to CNS performance has also been investigated (Coursin, 1967). Particular attention has been given to the B6 group of vitamins because of their vital enzymatic function in amino-acid metabolism. Experimental vitamin B6 deficiency results in gross behavioural abnormalities including hyper-irritability with a lowered threshold of stimulation, and convulsive seizures. Behavioural changes are reversible provided sufficiently large doses of the vitamin are administered before the onset of irreversible secondary alterations in cerebral function occur. The preferential protection of the brain at the expense of other tissues was illustrated in 1963 by Weber and Wiss who demonstrated that experimental vitamin B6 deficiency in rats may produce a reduction of the vitamin content in liver to 9% of normal values, while the level in brain

persists at 76%. In spite of this evidence of 'brain sparing' it is important to note that the control and low-protein diets used by Cowley and Griesel (1959) differed also in concentrations of vitamins and minerals, type and amounts of fat, carbohydrate, and bulk, and differed in protein quality. Specific amino-acid deficiencies can cause structural and functional lesions of the CNS. Scott in 1964 reported that a synthetic valine-deficient diet fed to young male rats for 22 days, and to older male rats for 70 days produced inco-ordination and circling behaviour as functional manifestations of CNS alterations demonstrated histologically as myelin degeneration, and neuronal deterioration of the deep nuclei.

Functional signs of CNS damage which was verified by histology have been observed by Platt, Stewart, and co-workers in rats, pigs and dogs, weaned from well-nourished mothers and then allowed ad libitum feeding of low-protein diets (Stewart, Platt, 1968). In pigs, this regime retarded physical development and progressively reduced the appetite until a marasmic condition developed. This could then be converted by assiduous feeding of carbohydrate or fat to one resembling kwashiorkor with oedema. After only four days of restricted diet, the rats developed spasmodic trembling of head and fore-paws. The pigs began to walk on tip-toe with a 'hobble-skirt' gait with evidence of inco-ordination of the hind legs. The dogs became hyper-irritable; EEG changes were demonstrated, and periods of loss of consciousness and convulsive seizures were reported. The clinical condition of these animals improved after a short period of rehabilitation.

Similar observations are reported by Barnes (1967). CNS function was evaluated using classical Pavlovian conditioned-response development followed by extinction of the response in pigs. No difference in the speed of development of the conditioned response in malnourished and control pigs was noted, but when attempts were made to extinguish a well established conditioned-response, it was found that the control animals extinguished quite rapidly, whereas the previously malnourished animals had a greatly prolonged period of trials before the conditioned-response disappeared, and some animals did not distinguish at all. (The conditioned-stimulus was a change of light intensity and an electric shock provided the unconditioned stimulus). In order to refine the test procedure and obtain a more quantitative estimate of conditioning and extinction, a modified Skinner box was used to establish a degree of conditioning quantitatively. The results clearly confirmed the earlier investigation; the early-restricted and control pigs developed the conditioned response at the same rate, but the control pigs extinguished the conditioned response much more rapidly than the experimental animals. Interpretation of this behavioural change is not clear, but the author notes a more pronounced subjective response to the shock in the experimental animals which could not be accounted for by differences in pain threshold, although difference in electrical resistance of the body to the imposed voltage did occur between the experimental and the control animals. The elevated emotionality of these animals could be expected to have had an effect on the quantitative aspects of the conditioning; it is tempting to suggest that the resistance to extinction in these

animals is akin to the 'experimental neuroses' produced in animals by Masserman and others in the 1940's. Reference to Dobbing's (1972) demonstration that the timing of the pig brain growth-spurt paralleled that in humans more closely than any other animal species highlights the significance of this work.

Barnes and Frankova (1967) have reported on an investigation using rats that has provided evidence that nutritional deprivation in early life may result in long lasting and possible permanent learning-behaviour abnormalities. An experimental design for the production of nutritional growth retardation which provided four experimental groups was carried out as follows: litter size was adjusted to 8 and 16 neonates per lactating female respectively. Immediately following weaning both groups were split and given diets containing 3.5% and 22% casien respectively for a period of 8 weeks, after which the nutritionally deprived groups were rehabilitated completely. Control groups were suckled normally and fed a normal diet after weaning. Males and females were studied separately but exposed to identical treatment. Growth curves of the rats showed that some permanent retardation resulted from the early nutritional deprivation. Visual discriminative learning-behaviour was assessed using a Y-shaped water maze. A significant difference in error scores was found in the male rats representing the two extremes of treatment, i.e. controls versus those deprived both pre- and post-weaning; the pre-weaning restricted rats also made more errors than the controls. No significant difference was found between the groups in female animals supporting the findings of Cowley and Griesel. Increased expression of emotionality was

observed in the rats nutritionally deprived prior to weaning only; the two groups that had been fed the protein-deficit diet post-weaning were, in contrast, calm when exposed to new test procedures. The same pattern of emotionality was observed in male and female animals, although the females appeared to be less affected. Since there was a motivational difference between the two groups, the authors conclude that early nutritional deprivations in these animals have resulted in a decreased learning capacity as well as changes in other behavioural characteristics. The clear-cut sex differentiation in effect is interesting and awaits interpretation.

More recently these authors have extended their investigations using a similar design combining and separating the variables of pre-natal and post-natal nutritional deprivation, followed by complete rehabilitation (Barnes and Frankova, 1969). Evidence of exaggerated anxiety on the basis of drive deprivation was again found to occur to a significantly greater degree in male rats that were nutritionally deprived both pre- and post-weaning compared with females subjected to the same dietary regime. All rats suckled by protein deficient mothers showed increased exploratory behaviour; after weaning the exploratory activity in the deprived males was intensified over that of the controls while protein or calorie restrictions were still being imposed. The intensification was

even greater in females, and higher in the calorie-restricted than in the protein-deprived rat. After rehabilitation, exploratory activity of all previously malnourished groups tended to decrease, the tendency towards reduced exploratory activity being greater in the males. At the age of 95 days, rehabilitated rats representing all four nutritionally deprived groups were not significantly different from the controls in learning of avoidance responses in an aversive conditioning situation. However the malnourished rats exhibited increased excitability, and inability to delay or extinguish the conditioned-response.

The more recent results of Cowley and Griesel (1963) using their trans-generational design to establish malnutrition and retardation of physical development in rats, are similar. The offspring of previously malnourished female rats were less active after weaning than control animals being the offspring of rats fed stock diet. Even after a long period of post-weaning rehabilitation on stock diet, the offspring of previously malnourished females showed significantly higher error scores on the Hebb-Williams maze than did the controls. Multigenerational effects of inadequate nutrition were reported, and the data indicated that the nutritional environment which the mother had experienced in utero and during the first five post-natal weeks of her life, influenced the rate

of early physical development of her offspring. The study demonstrated that correction of growth retardation proved an unreliable indicator of behavioural rehabilitation.

As in the human studies investigating the relationship between functional deficit and previous nutritional deprivation, so also in animal studies does the deprivation of food produce behavioural alterations which are not related to the nutritional deficiency *per se*. Thus alterations in drive and motivational factors complicate the interpretation of the functional differences between well nourished and previously malnourished animals. Whilst the evidence so far is inconclusive, it does suggest that the behavioural alterations in previously malnourished animals are at least in part due to deficiency of specific elements in the diet. Protein appears to be significant, and the evidence of 'coarse' CNS dysfunction in the form of seizures and loss of consciousness is striking in spite of the difficulties in the demonstration and evaluation of impairment of higher CNS function. Complementary evidence is provided by Flewner et al. (1962) who demonstrated inhibition of learning, loss of memory, and disorientation in mice when protein synthesis in the brain was inhibited by puromycin.

In discussing the disappointing results achieved by experimental animal studies investigating the effects of nutritional deprivation

in early life upon adult learning behaviour Barnes (1967) comments that: "It would appear that a major difficulty has been a lack of nutritional knowledge on the part of the experimental psychologists who have conducted most of the studies in this area and a lack of knowledge of experimental psychology on the part of the nutritionists who have been responsible for some of these studies. This is certainly one area of research in which we need inter-disciplinary team work, and hopefully in the future there will be more contributions reflecting the joint effort of both psychologist and nutritionist."

Two main questions arising out of these studies require clarification: What is the significance, in terms of function, of the structural CNS deficits demonstrated in animals; what meaning does the decreased exploratory behaviour, reduced maze learning ability, and defective extinction of conditioned responses have in terms of the overall learning capabilities of the animal involved and what relevance do these have forhuman intellectual functioning? No one can afford to ignore the important body of evidence emanating from animal experiments with their superior techniques of control, and the possibilities that they offer for the isolation of single variables (Dobbing, 1972a). However, interpretation must consider the fact that many experiments impose a severity of insult which would hardly allow survival if analogously applied to human infants (Clarke, 1968).

CHAPTER 6

THE EFFECTS OF FEEDING, REFEEDING, AND OVER-NUTRITION

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The direct causes of irreversible growth retardation due to malnutrition are not clear. The presence of regulatory systems which are involved in controlling the rate of cell division and growth in various tissues make the simple explanation of inadequate supply of structural materials seem unlikely. For example, in contrast to earlier theories that protein synthesis cannot take place at the synapse, Gordon and Deanin (1968) have shown that, *in vitro* at least, this is possible. All the requirements for the operation of a transport mechanism for protein destined to be the source of amino-acids for new protein at the synaptic endings are present. Potentially a mechanism is provided whereby neuronal structures are protected against at least short term variation in the supply of essential amino-acids to the whole organism.

Winick and Noble (1966) found that raising rats in large litters from birth to weaning resulted in a proportional decrease in the weight, protein, RNA and DNA content of all organs, including the brain, indicating a reduced cell number without alteration in cell size. These animals failed to recover normal growth when re-fed after weaning. Rats raised on a restricted diet from weaning to

42 days of age showed a proportional reduction in weight, protein, RNA, and DNA in most organs except the lungs and brain. In these two organs, the protein and DNA ratios were reduced but not total DNA, indicating that the cells were smaller in size but their total number was unaffected. Re-feeding failed to restore the number of cells in those organs in which cell number was reduced, whereas the lungs and brain regained normal size. Dietary restrictions from 65 - 86 days of age led to a reduction in protein and RNA concentration without affecting DNA in all organs. These studies indicate that nutritional deprivation differentially affects cell size and cell number depending on the developmental phase of the organ at the time of the insult. The brain appeared irreversibly damaged only by nutritional deprivation prior to weaning in rats; when cell size only was affected, recovery took place on re-feeding, whereas a reduction of cell number was not improved during rehabilitation.

Evidence for the effects of re-feeding on functional changes in animals following nutritional deprivation are complimentary. The gross signs of CNS dysfunction produced in animals by post-weaning dietary restriction by Stewart and Platt (1968) were reversible after a short period of re-feeding. However studies of learning behaviour using food reinforcement are difficult to

interpret because long-term effects of early nutritional deprivation on feeding behaviour and food motivation have been demonstrated to persist beyond rehabilitation. The use of learning situations based on aversive reinforcement which do not involve food, do not remove the difficulties in interpretation since early malnutrition modifies sensitivity to such negative stimuli (Levitsky and Barnes, 1970).

Guthrie and Brown (1968) showed that depressed cholestrol levels in malnourished rats were irreversible if undernutrition was extended until 5 weeks of age, but were restored to normal if rehabilitation occurred immediately after weaning. This suggests another vulnerable period occurring after weaning at about 3 weeks of age that relates to myelination of the brain, after which the brain becomes resistant to deprivation. Dickerson and Walmsley (1967) reported that rats undernourished from weaning until 11 weeks of age could be rehabilitated totally if re-fed for 2 - 8 weeks, after which no reduction in DNA-P concentration of the brain was found. Evidence of compensatory mechanisms was provided by Dobbing and Widdowson in 1965 (quoted by Altman) who demonstrated that undernourished 11 week old rats acquired three times the amount of cholestrol found in well-nourished control animals over an 8 week rehabilitation period.

Studies by Chow and Lee (1972) help in explaining the complex systems controlling the rate of cell division. These workers observed that the 'permanent' growth retardation in the offspring of rats subjected to dietary restriction during gestation and lactation could not be corrected by subsequent refeeding, but was restored to normal if the animals were given injections of pituitary extract or growth hormones. Small pituitary glands with a low concentration of growth hormone were reported in the malnourished animals. The hypothesis that pre-natal malnutrition could cause irreversible undersecretion of growth hormone and thereby growth failure of offspring has been further tested using radioimmunoassay and bioassay techniques . The weight of the pituitary in malnourished rats was 60% of the controls and the growth hormone content per pituitary was about one third that of the control group. Plasma growth hormone levels were also found to be lower in the underfed group. The authors suggest that a deficit of cellular growth may be a primary effect of undernutrition of the adrenohypophysis, or that pituitary growth hormone content may be reduced due to abnormal development of the hypothalamus and undersecretion of growth hormone releasing factor. By means of similar mechanisms malnutrition may serve to alter the production and secretion of other hormones, and since relationships between hormone

secretions and abnormal behaviour are well established, these studies may contribute to an understanding of the functional and behavioural abnormalities following malnutrition in animals.

Consideration of the role of growth hormone and the effects of depressed secretion on growth and development may also provide a link between, and elucidate the similarity between the syndrome of retardation related to malnutrition and the almost identical syndrome associated with emotional deprivation and environmental under-stimulation. Rosenzweig et al. (1968) and Bennet et al. (1969) have shown that rats exposed to different environments show characteristic changes in brain weight, enzymatic activity and depth of cerebral cortex. Impoverished animals were caged singly, colony animals were housed 2 or 3 per cage, and enriched animals 10 or 12 per large cage with toys. Dry weight, depth of cerebral cortex, enzymatic activity, and problem solving behaviour were increased by exposure to the enriched environment as compared with standard colony and impoverished conditions.(Henderson (1970) found that an enriched environment resulted in increased brain weight in mice. Thompson and Heron in 1954 showed that pet-reared dogs behaved more intelligently in a variety of situations than their litter mates who had been caged for the first 8 months of life. Cooper and Zubek (1968) have shown using rats that different environments in different genetic

lines may serve to either develop or depress problem solving capacities. Two lines of animals whose maze solving ability had been developed progressively by selective breeding were studied. When rats from the 'bright' and 'dull' lines were raised for a whole generation in a restricted environment which differed from the normal laboratory environment, no differences between the genetic strains could be detected, and both performed almost equally well when both were raised in the same stimulating environment. When both groups were raised in a normal environment the difference in error score was significant.

The work of Spitz, Goldfarb, Bowlby and Dennis concerning the long term effects of early deprivation on cognitive development has been examined critically by Clarke (1968) who stresses the resilience of children moved from an impoverished into an enriched environment. In 1967 Powell, Brasel and Blizzard described the syndrome of pseudo-hypopituitarism occurring in children from an emotionally disturbed background in the presence of an adequate diet confirmed by normal serum proteins. Growth and intellectual functions improved during rehabilitation in hospital, but lapsed on return to the home environment. Engel et al. (1956) described growth failure in hospital in infants fed by gastrostomy, contrasted by significant weight gain in an adequate home environment.

An important ongoing study on the effects of stimulation during rehabilitation of malnourished children (Yaktin et al., 1970, 1971) shows that the benefits of increased stimulation fade after return to the home environment (see chapter 2). It seems clear from these studies that the effects of adverse early experience are reversed at least in part by improvement in environmental conditions but lasting gain is dependent on subsequent reinforcement.

Whilst there has been a preoccupation with the effects of reduced nutrition, some studies of the effects of early over-nutrition have been carried out. Extensive studies by Widdowson and McCance, and Dobbing have demonstrated that increased consumption of maternal milk during the first days of life would accelerate physical growth and development of the CNS (Nutr. Rev., 1971). Animals raised in small litters weighed twice to three times normal at weaning and maintained their superiority throughout life. Winick and Noble described in 1966 an increased number of brain cells in rats reared in small litters compared with those reared in normal sized litters. Lat, Widdowson and McCance (1961) found an increased level of exploratory activity in new environment in fast growing rats compared with slow growing rats between the 21st and 49th day of age.

More recently in 1970 Frankova investigated the behavioural activities of rats derived from litters of 4, 8, and 17 animals. Animals from litters of 8 were regarded as controls for those from small litters (over-nourished) and those from large (under-nourished). One-half of the animals from each group were fed a low protein,

high fat diet from weaning to 56 days of age, thereby imposing a period of protein restriction. These animals were then fed the same basal diet as the rest. Spontaneous exploratory activity, habituation of the exploratory activity, and ability to develop conditioned responses were examined at various times over a period of one year. No permanent behavioural superiority was demonstrated in over-nourished rats. Increased exploratory activity in the over-fed animals was recorded during the early phases of growth coinciding with development of CNS and motor structures, but this was reduced after the 19th day of life. The adult behaviour of the over-nourished animals was in many respects similar to that of the undernourished animals and usually inferior to the controls. In another paper Frankóva (quoted by Winick, 1970) reported that rats super-nourished for the first 21 days of life showed subsequent learning deficits. Thus both undernutrition and over-nutrition in pre-weaning rats will result in persistent functional alterations of the brain, and over-nutrition prior to weaning did not protect against the consequence of protein deprivation after weaning. The response to certain pharmacological and psychological stresses was similar in the over-nourished and under-nourished animals.

The results of these studies raise important questions. The structural and biochemical changes in the brain following neo-natal nutritional deprivation are quite different from those following over-feeding. In terms of cell number, they are opposite. In contrast, the behavioural changes are quite similar. It may then be possible that the biochemical changes in the brain that have been reported to follow on malnutrition imposed in pre-natal and

early post-natal life have little functional significance (Nutr. Rev., 1971). On the other hand the environmental changes brought about by manipulating litter size may have a significant effect on the structure and function of the brain and CNS which is being reflected in these investigations.

CHAPTER 7

OVERVIEW

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OVERVIEW

An attempt to gain perspective in the study of malnutrition and intellectual development must focus attention on two inter-related but distinctive aspects of the problem:
Firstly, what are the methodological problems, and what limits do they place on the reliability and validity on recent research findings; how can the difficulties be expected to effect results of future studies and how can these difficulties be overcome?
Secondly, what conclusions can be drawn from the mass of diverse research approaches to the problem, and what further research directions are suggested which are likely to produce meaningful and useful information?

METHODOLOGICAL PROBLEMS

Studies attempting to relate malnutrition during early life to later intellectual development have been both retrospective and prospective in design (Nutr. Rev., 1970). Retrospective studies define children who have been malnourished at an earlier age, and compare them with children who were not malnourished. Such studies have the advantage of being able to collect relatively large numbers of children in a short period of time, but suffer from the disadvantage of poor definition of the nutritional state during the critical period being examined. Frequently the nutritional status data are only available from historical information on hospital charts and will vary in reliability. Control populations must also be selected on the basis of nutritional history but must match the

experimental group in all other respects. The difficulties attending the selection of such control groups are obvious in all studies so far undertaken; so far no study has successfully achieved complete control over all the non-nutritive variables which have an effect on the development of intellectual functions. Attempts to overcome the difficulty of control by the use of siblings suffer from the danger of controlling out the independant variable. In such studies it is probable that the control siblings suffer from a similar degree of chronic sub-optimal nutrition ("dietary sub-nutrition" defined by Brock in 1961 as any impairment of functional efficiency of a body system which can be corrected by better feeding) and differ only from the index cases in the absence of any acute episode of nutritional disease. Why some children in a chronically sub-nourished population experience an acute episode of nutritional illness, and why others who appear equally malnourished when compared in terms of deficit in expected height and weight do not succumb, is an interesting question introducing a further set of variables which are difficult to account for.

In contrast to retrospective studies, prospective studies usually have fewer children available for study, but the nutritional status is evaluated by the investigator himself in both the malnourished and control groups. The advantage of this type of approach is that better nutritional information is available and the selection of controls is made at the time that the study population is defined. Both groups can then be followed concomitantly. The problem with this type of study is the economic one - a longer period of time is required to study a smaller number of children.

The serious methodological problems to be faced in clinical studies of intellectual and motor development in children treated for malnutrition have been referred to in Chapter 2. The many variables that have to be considered and, if possible controlled, include:

1. The conditions and duration and hospitalisation to which the child was exposed. Geber and Dean (Courrier, 1956) stressed the importance of the mother/child interaction during hospitalisation as a crucial factor in the rate and degree of physical and mental recovery shown by children suffering from kwashiorkor. Whilst this problem applies more specifically to those studies conducted during the immediate recovery period, possible long-term effects of hospitalisation may provide variables in long-term follow-up studies which are difficult to control.
2. The age of the child at onset of malnutrition has varied considerably in the populations studied, and in terms of hypotheses of critical periods of vulnerability in brain and behavioural development may represent a crucial condition of the effect of the independent variable. In retrospective studies it is sometimes impossible to establish the exact age of onset. Whilst age on admission to hospital is used to indicate the age of onset of an acute episode of PCM, it provides actually an approximation of the duration of chronic untreated malnutrition. Cravioto and Robles (1965) found that there was a greater persistence of low performance scores on adaptive behaviour on Gesell Scales in those infants who were under 6 months of age at the time of treatment compared with those who were over

6 months of age and this was confirmed by Yaktin (1971). Conversely Chase and Martin (1970) found that children admitted to hospital with severe undernutrition after the age of 4 months were more severely impaired than those admitted within the first 4 months of life, who did not differ significantly from their controls. Similarly Evans (1969) found a significantly lower mean intelligence score in both ex-kwashiorkor subjects and the siblings of the group admitted to hospital aged 16 - 48 months when compared with the group admitted between the ages of 10 - 15 months. These findings suggest that both the age at which the major nutritional insult occurred as well as the duration of malnutrition are significant independent factors.

3. The social, economic, and family ecology from which the child comes are perhaps the most difficult factors to control, and their effects on the development of intellectual functions, cognitive and motor skills have been abundantly demonstrated in psychological, socio-demographic and anthropological studies. Mönckeberg's (1972) study illustrates this difficulty: even children with acceptable nutritive conditions (as evidenced by optimal growth) in the low socio-economic group showed lower IQ values than preschool children belonging to groups drawn from a better socio-economic level. The use of intra-familial controls does not overcome this problem entirely because of temporal variations in the economic stability and internal psycho-dynamics of the family. Additonal variations may be introduced by geographic mobility.

4. The intellectual and educational level of the parents is supposedly controlled by selection of comparable groups in some studies. At best this method cannot hope for more than minimal control, and intra-familial control provides the only answer to this difficulty with a known expectation for between-sibling IQ correlations.

5. The type of malnutrition present in the subjects studied has varied across the entire range of the PCM spectrum. Different effects may result from a diet deficient mainly in protein as opposed to one deficient mainly in calories. Variations in other specific mineral and vitamin dietary elements are also likely to have occurred in previous studies, and the role of infection or other supervening factors in precipitating the acute episode of malnutrition may also be relevant but uncontrolled. Liang's (1967) study is the only one which singled out a specific vitamin deficiency in addition to PCM. The type of malnutrition prevalent is likely to be related to availability of various food types and will therefore vary from one geographical region to the next. Furthermore the degree of urbanisation and industrialisation will also influence attitudes to breast feeding and the degree of change in traditional child feeding and rearing practices. Thus in less developed areas where breast feeding remains relatively prolonged the onset of malnutrition is likely to be relatively late and the type more typical of kwashiorkor, whereas the urban slum is likely to impose weaning and poor nutrition from an earlier age resulting in an earlier and more marasmic type of illness. Severity of

illness has not been controlled, and in some histological studies the children had died of malnutrition (Winick and Rosso, 1969). A further uncontrolled variable is the degree of maternal malnutrition. Few studies have taken birth weight into account, but the significance of maternal malnutrition has been suggested by Witkop (1967), Birch (1972) and Winick (1973). pg. 95

STATUS OF CURRENT KNOWLEDGE

An evaluation of the current knowledge in the field of malnutrition and intellectual development can be addressed to two main questions, viz. 1) what consequences does protein-calorie malnutrition in early infancy have for subsequent intellectual functioning of the individual and 2) can the mechanisms or processes be identified whereby the effects of early protein-calorie malnutrition and later intellectual functional deficit are mediated.

The studies reviewed in Chapter 2 leave little doubt of a relationship between PCM in infancy and later deficit in performance on tests of psycho-motor and intellectual functions. The causal relationship between the antecedent malnutrition per se and the functional deficit has been inferred but never conclusively demonstrated in human studies because of the difficulty in isolating malnutrition as an independent variable. The extent to which malnutrition on its own is an effective factor in interfering with the development of intellectual functions remains an open question. Attempts to resolve this question using animal studies have achieved more success in isolating malnutrition as an independent variable but do not offer a total immunity to the methodological difficulties of controlling other significant independent variables.

Drive deprivation alone influences the animals' behaviour, and functional and structural changes that have been shown to result from increased handling and tactual stimulation may confound the issue when litter size is modified. However these studies indicate that there are critical periods in the development of the CNS and brain as a cellular and biochemical system during which its morphological and biochemical structure is vulnerable to the effects of malnutrition. Conversely, opposite morphological and biochemical effects can be produced by inverting the independent variables viz. by overfeeding or supernutrition. The problem arises from the fact that these two opposite extremes of treatment in animals produce similar functional, behavioural deficits. Thus inference only relates cell size and cell number to functional disability in maze performance or conditioned response formation; the consequences, in terms of behavioural function, of the morphological, biochemical and enzymatic alterations in the brain produced by malnutrition during critical developmental periods have not been fully established.

Some parallels exist between the behaviour of previously malnourished animals and previously malnourished children. Barnes (1967) demonstrated repeatedly that previously malnourished animals showed no difference in rate of developing a conditioned reflex, but the rate of extinction of the conditioned reflex was significantly prolonged. In humans, Klein et al. (1972) using heart rate as the dependent variable, demonstrated a significantly different pattern and slower rate of habituation to pure tone stimuli between normal infants and those rehabilitated after severe nutritional

marasmus. Similar differences in habituation have been noted between normal children and those suspected of brain damage, but differences in orienting reflex and habituation have also been noted by Levine and Mullins (1968) to occur between manipulated and non-manipulated animals, and between maternally deprived monkeys and normally reared controls. These differences have been related to adrenal steriod secretion.

In overview it is apparent that severe deprivation of either or both nutritional and non-nutritional stimuli is capable of producing a degree of 'damage' to the brain in humans and animals. No structural lesion, focal or diffuse can be identified; the 'damage' is a damage to growth and function (Dobbing 1970, 1972, 1972a). A specific biochemical impairment may yet be found, and there is a need for research in this direction (Witkop 1967).

Difficulties in relating demonstrated morphological and biochemical changes to observed functional deficits in malnourished animals has stimulated a search for mediating mechanisms or processes operating between the two. The search is still in its infancy and as yet no direct functional significance can be attached to such variables as brain cell size, cell number, degree of brain myelination, as assessed by DNA, RNA, and cholestrol indices for these parameters.

An interesting line of research is reported by Witkop (1967) based on the observation that response of children with kwashiorkor to phenylalanine and histidine loads are somewhat like the response in children with phenylketonuria and histidinemia respectively. Evidence for actual placental transport of these amino

acids suggests that children of malnourished mothers may be under considerable risk in intra-uterine exposure to high levels of histidine and phenylalanine. Children with histidinemia present with a particular type of speech disorder based on inability to pronounce certain words, and associated with an auditory 'scramble' manifested as an inability to repeat words in sequence added one at a time, together with a short auditory memory span. A possible mechanism for a large component of mental retardation in children following PCM is the result of nutritionally induced enzymatic deficiencies in histidine and phenylalanine.

Studies such as described by Witkop are rare, but indicate the availability of research approaches which are promising because they suggest testable hypotheses. For example, Witkop suggests that the type of mental retardation expected would have clinical features of histidonemia and phenylketonuria, and that children born to protein malnourished mothers would have an intellectual defect quantitatively and qualitatively different from the child who had only post-weaning PCM. Several studies have indicated that verbal and language functions are consistently more seriously implicated and failed to respond to rehabilitation to the same extent that motoric and non-verbal functions do (Waterlow, Cravioto and Stephen, 1960; Cravioto, 1970; Yaktin, 1970, 1971). This observation has been assumed over the past to reflect the effects of maternal deprivation and of class differences in verbal interaction between mother and child, but it may be related rather to a deficit of auditory memory. Some support for this comes from the observation of Canosa (1971) that short-term memory and the

availability of short-term stimuli discriminated between well- and previously malnourished children on psychological testing. Future studies should therefore be directed towards identifying more precisely the nature of the functional deficit occurring as a consequence of PCM in both animals and children.

PART II

THE PRESENT STUDY

CHAPTER 8

BACKGROUND, RATIONALE AND HYPOTHESES

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CHAPTER 8

BACKGROUND, RATIONALE AND HYPOTHESES

This study is the logical extension of an investigation reported under the title "Kwashiorkor and Intellectual Development" in 1969. When, in 1968, that investigation was undertaken, few studies of its kind had been reported in the world literature, and the study was unique in that it was the first reported use of intra-familial subjects and controls (siblings) in an attempt to control the many non-nutritive variables which act independently in the development of intellectual functioning. The results of the study indicated that an acute episode of protein deficiency disease of sufficient severity to necessitate in-patient hospital treatment did not significantly lower the subsequent functional intellectual level. The study however did not exclude the possibility that long-term chronic undernutrition did retard intellectual development, in both the subjects and their sibling controls. Whilst the study minimised the influence of genetic, social and environmental factors on the determinatin of intellectual development by the use of sibling controls, there was also to some extent a cancelling out of the independent variable. Thus, a similar degree of physical growth retardation was found in both groups, and the group with antecedent kwashiorkor differed only from the sibling control group with the intervention at some stage of one or more factor(s) which precipitated an acute phase of protein-calorie malnutrition sufficient in degree to produce unmistakable signs and warrant in-patient hospital

treatment. In retrospect it is impossible to identify the intervening factor; this may have been nutritional in the form of a deficiency of a specific nutritional element e.g. protein or vitamin, or it may have been infective, or it may have been psychosocial or psychodynamic or any combination of these. The suggestion was therefore made that a study of children who had been nutritionally deprived by virtue of an intestinal malabsorption syndrome or fibrocystic disease should be undertaken in which their intellectual functioning could be compared with that of their siblings.

The impractability of such a study because of the lack of a sufficiently large number of subjects satisfying the experimental requirements led to the idea of approaching the problem from a different angle viz. by studying the effect of inverting the independent variable. Stoch and Smythe (1963) suggested that the effects of PCM on intellectual development could only be demonstrated by the comparison of children representing opposite extremes in nutritional condition, and used this argument to justify their comparison of grossly marasmic children with children whose early nutritional status had been maintained at an adequate level through attendance at a creche. The present study aims to stimultaneously exploit the concept of studying extreme cases of the influence of the independant variable in two directions, and also to include the advantages of the use of sibling controls. The design basically operates by the comparison of different children from within the same family who respectively

a) suffered an acute clinical episode of PCM,

b) suffered PCM evidenced by significant physical growth

retardation but at no time presented with acute clinical symptoms and

c) in whom PCM was prevented by dietary supplementation during the first 2 years of life.

The main hypothesis underlying this study states that if early malnutrition per se causes retardation of intellectual development, then dietary supplementation during the same developmental period should prevent the occurrence of such intellectual retardation in a population at risk. A basis for this research approach is the known fact that siblings have highly correlated scores on intelligence tests, reflecting the fact that siblings belong to the same genetic pool, have been exposed to similar physical and social environments, and have been exposed to similar epidemiological hazards.

Because provision of dietary supplement to an infant within a family is likely to provide other non-nutritive stimulatory effects on that child and the entire family directly, or indirectly via the mother, the design included the selection of a control sibling just preceding the supplemented sibling who was exposed to the same social stimulatory effects but did not receive specific nutritional attention. Because of the improbability that the index child only would benefit from supplement issued to the mother, an attempt was made to ensure that the control sibling was beyond the age when dietary supplementation should, in theory, have any significant beneficial effect on his intellectual development, i.e. after the age of 18 months according to Dobbing (1972). In an attempt to magnify the effect of improving nutrition on intellectual development, the

other extreme situation was studied in each family by testing an older sibling who had been malnourished to a degree sufficient to cause an acute episode of kwashiorkor necessitating hospitalisation, and treatment in a metabolic ward, together with a further sibling whose physical growth had been retarded to a similar extent but who had not succumbed to acute PCM.

The design of this study is unique and was made possible by the existence in Cape Town of an unusual group of subjects who have been under study over the past 15 years by the M.R.C. Clinical Nutrition Research Unit at U.C.T. The feed-up project was initiated in 1963 when the new-born sibling in a group of Cape Coloured families with a known incidence of kwashiorkor was selected for dietary supplementation (Moodie et al., 1973). Apart from the incidence of acute PCM in the family, the main criterion for selection was a poor growth record in all antecedent siblings (below the 3rd percentile in weight and height for age) in spite of normal birth weight.

The project was conducted by a medical social worker who paid weekly visits to the family for 2 years from the birth of the feed-up siblings. Initially, supervision only was given to ensure that breast feeding was well established. When breast feeding failed, or declined to insufficient levels, feeding was supplemented by an adequate supply of branded full-cream powdered or evaporated milk. When each child was aged $2\frac{1}{2}$ months, a fortified cereal was introduced regardless of the status of breast or bottle feeding. (Pronutro: protein content 21.5%, 365 calories/100 grams). In addition, a vitamin and iron syrup was supplied for the duration of the program.

Weights and heights of the infants were recorded regularly, and any tendency towards failure to thrive was investigated medically. Similarly, any incidence of illness or infection was treated promptly. Control siblings received the same medical care; they also possibly benefitted from a proportion of the dietary supplement, the likelihood of which increased only after the first 6 to 12 months of supplementation when they were more able to obtain the cereal for themselves without the mother's knowledge. Apart from this, general improvement in nutrition on account of improved maternal motivation and the general focus of attention on food within the family is likely to have occurred. The actual quantity of food dispensed per child per week was determined by the child's weight gain, the limiting factor being only a regard to excess which could be put at the disposal of the sibling-control child. The feeding was only supplementary - the child otherwise participated in the usual eating habits of the family. The dietary supplementation was discontinued when a mean duration of 28 months had been attained. Thereafter routine care was continued at the local Health and Welfare Centres.

The present study aims to investigate the following postulates:-

Proposition 1: If malnutrition as such causes retardation of intellectual and cognitive development, then the fed group should show a significantly higher intelligence score when compared with each of the other three groups.

Proposition 2: If social and stimulus deprivation (including the effects of recurrent illness and infection) rather

then malnutrition as such is responsible for the intellectual and cognitive retardation, then no significant difference in intelligence score would be expected between the fed group and the fed-control group, but these two groups should differ significantly from the kwashiorkor and kwashiorkor control group.

Proposition 3: If the severity of PCM is related to the degree of intellectual retardation then the kwashiorkor group should show a significantly lower intelligence score when compared with the kwashiorkor control group, and the fed-control group.

Proposition 4: If the duration of chronic PCM is related to the degree of retardation of intellectual development then no significant difference in intelligence score should be expected to occur between the kwashiorkor group and the kwashiorkor control group, but both groups should show significantly lower intelligence scores than the fed group.

Whilst the above propositions are formulated in terms of quantitative differences in intellectual development, qualitative differences should be shown by differences between the groups on the subscale intelligence scores, and by differences on the Bender Gestalt and Harris Drawing Tests (See Chapter 9).

CHAPTER 9

METHODS AND MATERIALS

CHAPTER 9

METHODS AND MATERIALS

THE SAMPLE

In August 1972, 14 of the feed-up children from an original group of 18 were available for study, together with 13 sibling controls. Within 9 of these families, kwashiorkor rehabilitees and their sibling controls had been tested in 1968 during the previous investigation. By including the remaining 5 kwashiorkor rehabilitees and their sibling controls, a sample of 54 children from 14 families was composed as follows: from each of 12 families 4 children were drawn and assigned respectively to kwashiorkor group, kwashiorkor control group, fed group, fed control group; in 2 additional families 3 children were available: assigned to kwashiorkor group, kwashiorkor control, and fed group in the one family; and as kwashiorkor group, fed group, and fed control group, in the other family. Both of these gaps were created by the death of a control child, one due to a motor vehicle accident and one due to accidental poisoning.

THE EXPERIMENTAL GROUPS

Two experimental groups were formed each containing 14 subjects, and each representing an extreme of early nutritional status:

Kwashiorkor Group

The criterion for selection to this group was that the child had been hospitalised at least once during infancy for treatment of acute kwashiorkor as defined by Hansen (1968). The subjects in this

group were all males, reflecting the known unequal distribution of the disease between the sexes.

Fed Group

This group consisted of 14 subjects being all the available subjects from the feed-up project described in Chapter 8. Of the original 18 feed-up children, 2 had died from infectious diseases and 2 were known to have moved to other areas beyond reasonable access. The group consisted of 9 males and 5 females.

THE CONTROL GROUPS

The sample contained two control groups each consisting of siblings of the other, and each matched to one of the experimental groups which were also sibling groups:

Kwashiorkor Control Group

This group was composed by the selection of the sibling nearest in age to the kwashiorkor child, but having no history of acute protein-calorie malnutrition. No attempt was made to match the groups for sex in the present investigation, but the 9 pairs of subjects included from the previous study were sex matched. The group consisted of 13 children (one having died due to alleged poisoning), 10 of whom were males and 3 females.

Fed Control Group

This group consisted of the sibling nearest in age just preceding the fed child in each family. The group consisted of 13 children (one child had been killed in a motor vehicle accident), 7 of whom were males and 6 females. This group was a mean of 21 months (1.8 years) older than the fed group (range of age gap

0.95 - 3.0 years) so that by the time supplementary feeding was commenced the group had a mean age which exceeded the theoretical critical period of vulnerability in brain growth and were therefore not susceptible to significant acceleration in intellectual development resulting from improved nutrition.

No child was included in the study if there was any doubt about maternal or paternal parentage. In all cases children had lived within the same family environment except for periods of admission to hospital. This absence from the family affected mainly the kwashiorkor group in which it was a constant factor throughout the group. A small number of families made moves of residence (generally to improved conditions) during the feed-up period. In 8 of the families a further sibling was born within the feed-up period. The supplementary feeding was extended to this new sibling, but he was not included in the sample.

TEST BATTERY

The battery of tests was essentially the same as that applied in the previous investigation with the addition of the Bender Visual Motor Gestalt Test in the present study. The aim in selecting the battery of tests was to provide a profile of the children's present overall intellectual performance, and the Bender Gestalt Test was included because of its value in discriminating 'brain damaged' and 'non-brain damaged' groups (Koppitz, 1964). No intelligence scale has been standardised for use on Cape Coloured children in this age group, and consideration was therefore given to the possibility of making use of a method for the evaluation of

neuro-integrative competence by examining features of neurological maturation which follow a known clear-cut development course in normal children, and upon which the influence of particular cultural experiences is relatively small (Cravioto, 1970; Birch, 1970). However, more recent research (Bridger, 1970) has demonstrated that poor performance on tests of inter-sensory integration is related to inability to employ abstract verbal labels rather than to perceptual deficit (quoted by Klein, 1972 in discussion). In spite of disadvantages, it seems that the conventional intelligence scale remains, at this stage, the most useful technique of evaluation. The New South African Individual Scale, although standardised on a White South African school-going population remained the most appropriate test for application in this study, and its use provided the additional advantage of making the results of the present study comparable with those of Stoch and Smythe (1968) and with those of our previous study of kwashiorkor rehabilitees and their siblings.

THE NEW SOUTH AFRICAN INDIVIDUAL SCALE (NATIONAL BUREAU OF EDUCATIONAL AND SOCIAL RESEARCH, 1964)

This scale is similar in construction to the Wechsler Intelligence Scale for Children (the Psychological Corporation, New York, 1949). The battery comprises nine sub-tests, 5 of which are verbal and 4 non-verbal, as follows:-

Vocabulary - multiple choice picture-type test using a total of 30 vocally presented stimulus words; the test thus requires a perception and integration of an auditory stimulus with a visual stimulus, but does not require a verbal response. The test is

essentially a measure of abstract verbal labelling.

Comprehension - ten questions relating to everyday life situations and customs are presented vocally. Responses are scored on 3 levels of qualitative differentiation of increasing abstraction, and are said to indicate 'judgement' or 'common sense' performance in which emotional and intellectual functions play a part.

Verbal reasoning - a similarities test of abstract reasoning, the first 2 items of which are in analogical form. Responses are scored according to Rappaport's levels of reasoning, viz. concrete, functional, abstract. Performance on the test depends on ability to distinguish between essential and non-essential similarities, to generalise, and to categorise.

Problems - 15 verbally presented (the first 8 presented vocally) arithmetic problems, considered mainly a test of ability to concentrate. Successful performance is dependent on training, and the score therefore reflects the level of school achievement.

Memory - test of immediate recall of meaningful verbal material presented vocally.

Pattern Completion - performance requires a free response in the completion of 16 partially completed designs; time may be taken into account in scoring. Visual orientation as well as ability to concentrate is involved in performance.

Block design - a Koh's type test which requires analytico-synthetic ability and especially the ability to solve problems in spatial relationships. Scoring may include a time factor.

Absurdities - similar to Wechsler's Picture Completion sub-test but containing incongruities as well as omissions.

Form Board - an adaptation of the Leake-Smith Form Board and consisting of 6 figures: 3 of 3, and 3 of 4 loose coloured pieces. Scoring is based on time taken to complete each figure. The test is regarded as an indication of qualitative aspects of intelligence and reflects the effect of temperament factors on the solution of concrete problems.

The battery uses point scales, so that the same test item can be applied all the way through to all age groups, and the standard deviation - IQ method replaces the mental age method. Performance is indicated in terms of standard scores or normalised scales scores. In the present investigation, 'power-plus time' scores were used throughout.

Because this test has not been standardised for the population to which it was applied in this investigation, the terms 'verbal quotient', 'non-verbal quotient', and 'full scale quotient' are replaced by the terms 'verbal score', 'non-verbal score', and 'full scale intelligence score'. These 'scores' represent the child's performance in terms of the White standardisation sample; significance can only be attached to this score in terms of differences between the scores obtained by the various groups and the scores cannot be interpreted normatively in 'IQs' with reference to a particular population.

THE BENDER GESTALT TEST (Bender, 1938)

This test, consisting of nine geometric figures presented one at a time and which the subject is asked to copy on a blank sheet of paper, was used originally as a visual-motor test by Bender who pointed out that the perception and the reproduction of the Gestalt

figures is determined by biological principles of sensory-motor action and varies according to a) the growth pattern and maturational level of the individual, and b) his pathological state, either functionally or organically induced. The test remains one of the standard and most frequently used in clinical practice with both adults and children.

In the present investigation the administration and scoring of the test was carried out according to the method of Koppitz (1964) who developed a developmental scoring system standardised on a population aged 5 - 10½ years. Koppitz validated the system on a group of children aged 5 - 10 years with known brain injury and showed that the test has some value in discriminating between 'brain-injured' and 'non brain-injured' children scoring at various IQ levels on the WISC. A lack of correlation between IQ and Bender score in the 'brain-damaged' group was demonstrated. In the 'non brain-damaged' sample significant correlations were found between the WISC sub-test score and Bender score on all except Information, Comprehension, Similarities, and Coding sub-tests. The Koppitz Developmental Scoring System evaluates the reproduction of each design in terms of errors of integration, rotation, distortion of shape and perseveration. The error score is then converted to a visual perceptual developmental age level.

In the present study the age range of the group extended beyond the upper limits of the original standardisation sample, and the range of error scores obtained extended beyond the lower limits of those obtained in the original standardisation sample. A smoothed and extended error score curve was therefore constructed using

Koppitz's original standardisation data (Koppitz, 1964). The original Bender scores as reported in the normative data were plotted. The plot points started at 5½ years of age and went to 10½ years of age. From these points the smoothed curve was drawn to extend out towards 20 years of age. The original Bender standard deviations as reported were included in the graph from 8 years of age to 10½ years of age and these standard deviations were likewise extrapolated in the smoothed curve to 20 years of age. The means and standard deviations resulting from this extrapolation are reported in Table X (Appendix III). For each child scored on the Bender in the present study a Z score was formed from his Bender score; the Z score being the difference between his score and the mean on the extrapolated line divided by the extrapolated standard deviations. The Z scores were then related to a curve built up from the distribution of the Student t to two degrees of freedom. The effect of this was to attempt to give the child a percentile rank. The ordinary use of the Z score i.e. as related to the normal curve was not satisfactory because a number of children had such high Z scores (of the order of 5 and 7), that no area was left under the normal curve. The percentile rank approached zero after a Z score of approximately three. To attempt to give a meaningful percentile rank to children with high Z scores, the Z scores were treated as though they were t scores with 2 degrees of freedom. Thus, for example, a child with a so-called Z score of 4.3 had a percentile rank of 5%. A graph was drawn - the points of t at 2 degrees of freedom were taken as 0.0, 1.0, 1.88, 2.92, 4.3, 6.96 and 9.92. The

relative percentiles were taken as 50, 34, 20, 10, 5, 2 and 1. The children's Z scores were then read onto the graph and their percentile ranks were read off from the graph.

THE HARRIS-GOODENOUGH DRAWING TEST

This test was included for its value in evaluating 'intellectual maturity' in contrast to the IQ notion of unitary intelligence. Harris's concept of intellectual maturity includes the ability to form concepts of an increasingly abstract nature. The process of concept formation comprises three functional steps, viz.

1. The ability to perceive i.e. to discriminate likenesses and differences;
2. The ability to abstract i.e. to classify objects according to perceived likenesses and differences;
3. The ability to generalise i.e. to assign a newly experienced object to a correct class, according to the discrimination made.

A child's drawing of any object will reveal the discriminations he has about that object as belonging to a class i.e. the level of his conceptual maturity, and his concept of such a frequently and universally experienced object as a human being provides an index of a growing complexity of his concepts generally, particularly as it requires integration of affective and cognitive elements.

The child is asked to make the best picture he can of 1) a man and 2) a woman, and the formal and structural elements of the drawings are scored on 3 levels viz. presence, dimensions, and proportion. Raw scores are converted to standard scores for the

age group 3 to 5 yeers taking account of the sex of the child; standard scores on both scales are averaged to derive a total drawing score. A validation study of this technique using the NSAIS was carried out on White South African school children by Strumpfer and Mienie (1966). Reliability co-efficients of 0.88 for the Man Scale and 0.92 for the Woman Scale were obtained by the split halves method. Low, but statistically significant correlations were found between the Harris scores and the NSAIS subtest scores; the highest co-efficients of .61 was obtained between the man's scale and the verbal IQ.

An attempt was made in the present study to assess the interference of emotional factors in cognitive functioning by recording the difference obtained by each child in the standard score on the man and the woman scales. In addition each drawing was rated for the presence of evidence of emotional disturbance on a 5 point scale based on the conflict indicators of Machover (1948) viz. omissions of parts, excessive erasures, density of shading, transparencies, and the presence of sexual organs.

PROCEDURE

Testing of the children was begun in August 1972 and continued through May 1973.

Conditions of the test situation were kept as constant as possible. All testing was carried out in a suitably furnished play room at the Psychiatric Clinic, Red Cross War Memorial Children's Hospital. Each child was tested individually by the same examiner in 2 or 3 sessions not exceeding 45 minutes each. Test sessions were interspersed by periods of free play of at least 45 minutes duration

in a well equipped playroom. No incentives were suggested or offered before the completion of testing, but the majority of children assumed on the basis of past experience that a reward would be forthcoming on completion of the test program. Refreshments in the form of fruit juice cordials, milk, coffee, sandwiches, biscuits and sweets were provided during the break periods. All test administration and scoring was carried out blindly. In no case was the examiner aware of the group to which any particular child was assigned until his final test scores had been calculated. The physical stature of these children is such that it is difficult to estimate their age.

The 9 kwashiorkor children and their controls who were tested during the previous investigation were recalled for the administration of the Bender Gestalt Test, at which time the Harris-Goodenough Test was repeated. Of these subjects 2 were tested on a Saturday afternoon in order to avoid interference with their employment; for the rest all sessions were held in the morning. An average of 4 children were tested per morning, and with a few exceptions, these were representatives of at least 2 different families. The order in which the children were taken into the test room was determined randomly by tossing a coin.

Standing height and body weight of each child were measured on the day of testing. In addition the heights and weights of the fed children and their controls were available at an early age (during dietary supplementation) and at a mid-term age (approximately 2 years after the sessation of dietary supplementation). Mid-term and present heights and weights were available for the kwashiorkor

group and their controls. The percentages of expected height for age, and weight for age as at near comparable ages as possible were calculated using the 50th percentile of the Boston Growth Charts as standard.

A Multiple Social Index was available for each family composed of 4 factors as follows:

a) general environment and occupational density of living conditions;

b) economic status calculated with reference to the Poverty Datum Line;

c) diet judged by weekly intake of key protein foods;

d) family stability including illegitamacy, alcoholism and alcohol-related problems, conflict with the law, and marital breakdown.

The families had been rated in 1969, and the rating represented a mean for the families over the 10 year period 1959 - 1969. In addition, income ratings as percentage of the Poverty Datum Line (where 100% represents the Poverty Datum Line, and 150% represents the effective minimum standard) were available on each family at 5 year intervals viz. in 1959, 1964, and 1969. The 1964 figure therefore reflects the situation into which the feed-up control sibling was born, whereas the 1969 figure provides some indications of conditions in the family during the infancy of the feed-up child, A relationship between the income rating and food availability within the family is assumed; the income rating also reflects the degree of social disorganisation as dependent on the presence or absence of unemployment and degree of stable employment.

Birth weights in the case of the fed group and their controls, and the age on admission to hospital in the case of the kwashiorkor

group were recorded, as also was the number of siblings in the family at present, and the ordinal position in each family of the 4 children included in the study.

THE ANALYSES OF VARIANCE (ANOVA's)

The data were subjected to analysis of variance and multiple comparisons of means were computed. Both matched techniques using 12 sets and unmatched techniques using 14 sets employed where appropriate. This anomaly was created by the deaths of two of the sibling control children; the particular technique used will be indicated by the degrees of freedom. Correlation matrices were also computed.

Two-way analyses: Most of the two-way analyses computed were 2-way ANOVA's with repeated measurements on both factors (Kirk, 1968). These are referred to by Kirk as "randomised block factorial designs". Where appropriate 2-way ANOVA's with repeated measures on a single factor were used (Kirk, 1968: Chapt 7). Comparisons between the means of the various treatment levels were carried out using the Scheffé multiple comparison test. For comparisons of means on the NSAIS sub-test scores, Tukey's HSD technique was used (Kirk, 1968: Chapt 3). In cases where significant interaction effects occurred in the 2-way ANOVA's, simple main effects were calculated and their means compared according to the techniques laid out by Kirk (1968: Chapt 7).

One-way analyses: Where appropriate one-way ANOVA's with repeated measurements were computed.

CHAPTER 10

RESULTS

CHAPTER 10

RESULTS

The raw data which were subjected to statistical analysis are presented in Appendix I. A summary of results is given in Tables 1 and 2 and Figure 1.

The results obtained on each of the variables examined were as follows:

TABLE 1: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS

	ANOVA Significance		Kwash	Sib K	Sib F	Fed
Age	***	Mean	13.0	12.0	10.7	8.9
		Sd	1.9	2.1	1.0	0.5
Full Scale Intelligence Score	**	Mean	70.0	72.0	72.2	81.8
		Sd	13.5	14.6	13.0	13.0
Verbal	0.01>p>0.05	Mean	74.3	75.0	70.8	81.3
		Sd	16.1	17.3	13.9	14.0
Non-Verbal	***	Mean	71.3	74.4	80.5	86.3
		Sd	8.8	9.2	11.2	10.4
Harris	NS	Mean	81.3	83.7	75.1	81.0
		Sd	17.6	16.9	11.1	13.3
Bender (PQ)	NS	Mean	71.2	62.5	65.7	63.5
		Sd	30.5	21.4	18.5	19.6
Bender (t' Scores)	NS	Mean	29.7	28.2	18.7	17.6
		Sd	22.2	20.5	18.8	17.6

*p<0.05
**p<0.01
***p<0.001

TABLE 2: SCHAFFE MULTIPLE COMPARISONS BETWEEN MEANS

	Kwash	Sib K	Sib F	Fed
Age				
Kwash		NS	**	***
Sib K			NS	**
Sib F				*
Fed				
Full Scale Intelligence Score				
Kwash		NS	NS	**
Sib K			NS	*
Sib F				NS
Fed				
Verbal				
Kwash		NS	NS	0.05<p<0.10
Sib K			NS	NS
Sib F				NS
Fed				
Non-Verbal				
Kwash		NS	*	***
Sib K			NS	*
Sib F				NS
Fed				

*p<0.05
**p<0.01
***p<0.001

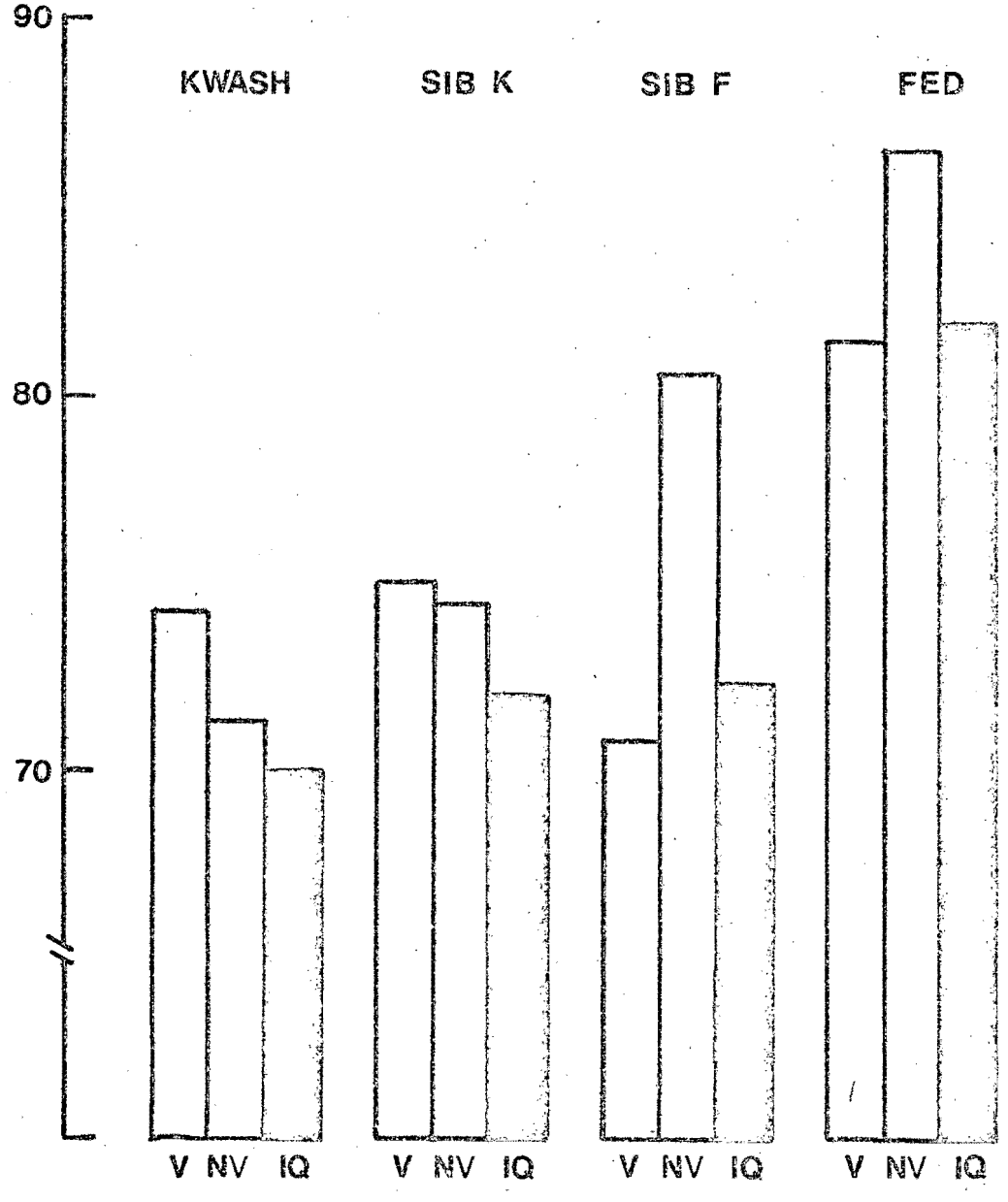


FIGURE 1 INTELLIGENCE SCORES (MEANS)

AGE VARIABLES

The mean age at the time of intellectual testing (NSAIS) for each of the four groups is shown in Table 3. The data analysis is presented in Tables 4 and 5. The fed group differed significantly from the other three groups. The kwashiorkor group did not differ significantly from its control group, nor did the two control groups differ from each other. The significant difference between the fed group and its control group was an essential feature of the experimental design, the purpose being to ensure that the control child was too old to achieve a brain growth benefit from overflow of the dietary supplement provided to his younger sibling.

The age of each child at the time of this investigation is shown in Table 56 (Appendix I). In the kwashiorkor group and its control group the mean ages at present (Table 1) differed from the mean ages on intellectual testing because some of the subjects included were tested as part of an earlier project. The present age is the age at which all children performed the Bender Gestalt and Harris Drawing Tests.

The ages of the fed control group at the start of the feed-up project, and at termination of dietary supplementation are shown in Table 56 (Appendix I). The mean age of the fed control at the start of the project was 21 months (1.8 years).

With the exception of 3 cases, all the fed group of children were brought into the project at birth. The three exceptions were

TABLE 3: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON AGE ON TESTING (NSAIS)

	Kwash	Sib K	Sib F	Fed
Mean	13.0	12.0	10.7	8.9
Sd	1.9	2.1	1.0	0.5

TABLE 4: ANOVA SUMMARY TABLE

One-Way Analysis without repeated measures.

Means for the four groups on Age on Testing (NSAIS)

Source	Sum of Squares	df	Mean Squares	F ratio
Between subjects	135.0	3	45.0	19.0***
Within subjects	118.0	50	2.4	

*** $p < 0.001$

TABLE 5: MULTIPLE COMPARISONS BETWEEN MEANS OF AGE ON TESTING (NSAIS)

Scheffe Multiple Comparisons

Degrees of freedom (3,50) in all cases

	Kwash	Sib K	Sib F	Fed
Kwash	-	NS	5.1**	19.1***
Sib K		-	NS	9.5**
Sib F			-	3.2*
Fed				-

*$p < 0.05$
**$p < 0.01$
***$p < 0.001$

included in the project at the ages of 6 months (case 6), 10 months (case 2), and 4 months (case 14). In all cases breast feeding had been adequately maintained up to the time of admission to the study. Birth weights of the fed group children are shown in Table 56 (Appendix I); in no case was birth weight below 12.5 kg. The duration of breast feeding of the fed group children is shown in Table 56 (Appendix I). The fortified cereal was introduced routinely in all cases at the age of 2.5 months. The program terminated when the mean age of the fed group was 28.1 months (2.3 years).

The age on admission to hospital for the members of the kwashiorkor group are shown in Table 56 (Appendix I). The median was 21 months. In terms of the division used in the previous study, the present kwashiorkor group was predominantly 'late-onset', with only 2 cases being admitted to hospital before the age of 16 months. The age range was 10 - 48 months. In the population under investigation the age of 21 months represents the age of greatest vulnerability to PCM, and the nutritional supplementation of the fed group extended for 7 months beyond this age. On the other hand the fed control group had reached this age of vulnerability by the time the feeding project was started. No significant correlations between age on admission to hospital and full scale, verbal, or non-verbal intelligence scores occurred in the kwashiorkor and kwashiorkor control groups (Table 6).

TABLE 6: CORRELATION COEFFICIENTS FOR NSAIS INTELLIGENCE SCORES AND AGE ON ADMISSION TO HOSPITAL

(df 11)

		F.S.IQ Kwash	F.S. IQ Sib K	VERB.IQ Kwash	VERB.IQ Sib K	NON-VERB.IQ Kwash	NON-VERB.IQ Sib K	AGE ADM. HOSP
F.S. IQ	Kwash	-						
F.S. IQ	Sib K	.71**	-					
VERB.IQ	Kwash	.97**	.70**	-				
VERB.IQ	Sib K	.74**	.98**	.72**	-			
NON-VERB.IQ	Kwash	.83**	.56*	.68**	.60**	-		
NON-VERB.IQ	Sib K	.57	.92**	.59**	.83**	.40	-	
AGE ADM.HOSP.		.17	-.06	.17	-.04	.17	-.07	-

*p<0.05
**p<0.01

SOCIAL VARIABLES

Multiple Social Index

The Multiple Social Index (MSI) for each family is shown in Table 56 (Appendix I). A significantly high correlation (Appendix II, Table 67) between MSI and non-verbal intelligence score occurred in the kwashiorkor group only ($r = .54$, $p<0.05$).

Income

Income ratings (percentage of Poverty Datum Line) other than those included in the MSI are also shown. The 1959 rating corresponds with the period of early childhood (0 - 3 years) of the kwashiorkor group and its control group and the fed control group, whereas the 1964 rating assesses the financial status of the family into which the fed group child was born. Income is an important variable as it will partly determine and reflect food availability in the family. The means show a downward trend for the period 1959 (64.7%) to 1964 (58.3%), and this combined with a steady increase in family size suggests a deterioration in socio-economic and hence nutritional conditions over this period. In this respect the risk of PCM was clearly higher for the fed group control child than for the kwashiorkor control child, but maximal for the children of the fed group in these families. The chances of succumbing to acute PCM were great in both the fed and the fed control groups without the intervention of the supplementary feeding program, which reduced inter-current infection in the fed control group by the concurrent supervision and medical care. The marked rise in income to 88.5% in 1969 is probably a reflection of the

country's general economic inflation, combined with the fact that many of the older siblings were engaged in some form of gainful 'unskilled' employment by this time and were contributing to the family income. In the combined fed and fed control groups significant positive correlations occurred between Income and Pattern Completion ($r = .40$, $p<0.05$), and Block Design ($r = .41$, $p<0.05$) sub-tests of the NSAIS (Table 71, Appendix II). The difference in income between the fed group and control group did not correlate significantly with group differences in verbal, non-verbal and full scale intelligence scores (Table 74, Appendix II).

Position in Sibship

The ordinal position of each child in the sibship is shown in Table 56, (Appendix I), together with the total number of children in the sibship at the time of the present investigation. Position in sibship did not correlate with scores on the intellectual tests in the kwashiorkor and fed groups (Table 7). In 8 families one or more further births occurred during the period of dietary supplementation. The full supplement was extended to all of these new family members, but they were not included in the fed group sample. When an 'Index of Sibship' (Table 57, Appendix I) was calculated for each child taking into account both position in sibship and size of sibship, significantly high positive correlations (Table 66, Appendix II) occurred with full scale intelligence score ($r = .29$, $p<0.05$), non-verbal intelligence score ($r = .37$, $p<0.01$), and the arbitary 'Food Index' ($r = .68$, $p<0.001$). Thus the younger children in the smaller families had the higher full scale and non-verbal intelligence scores.

TABLE 7: CORRELATION COEFFICIENTS BETWEEN INTELLIGENCE SCORES FOR FED GROUP AND KWASHIORKOR GROUP AND PROXIMITY IN SIBSHIP

(df 12)

	F.S. IQ Kwash	F.S. IQ Fed	VERB. IQ Kwash	VERB. IQ Fed	NON-VERB. IQ Kwash	NON-VERB. IQ Fed	FED (POS)-KWASH (POS)
F.S. IQ Kwash	-						
F.S. IQ Fed	.37	-					
VERB. IQ Kwash	.97**	.42	-				
VERB. IQ Fed	.35	.98**	.42	-			
NON-VERB.IQ Kwash	.82**	.13	.64*	.0	-		
NON-VERB.IQ Fed	.37	.96**	.39	.90**	.21	-	
FED (POS)-KWASH (POS)	-.10	.05	-.03	.06	-.27	.06	-

*p<0.05
**p<0.01

Food Index

As an additional technique for exploring the data and for educing meaningful correlations, an empirical 'Food Index' was assigned to each group. The hypothetical values given were as follows:

Kwashiorkor group	- 1
Kwashiorkor-control group	- 1
Fed control group	- 2
Fed group	- 5

These numerical values were based entirely on assumptions concerning the nutritional status of the groups during the first 2 years of life, and are not objectively verifiable. Nevertheless, significantly high positive correlations occurred between 'Food Index' and height at midterm ($r = .51$, $p<0.01$) and weight at midterm stage ($r = .46$, $p<0.01$). Neither height nor weight at present correlated significantly with 'Food Index'. (Table 66, Appendix II).

Significantly high positive correlations were also found between 'Food Index' and full scale intelligence score ($r = .35$, $p<0.01$), and non-verbal score ($r = .55$, $p<0.01$). 'Food Index' also showed a significantly high positive correlation with the Index of Sibship ($r = .68$, $p<0.01$). (Table 66, Appendix II).

In the combined fed and fed control groups (Table 71) the 'Food Index' showed significantly high positive correlations with vocabulary ($r = .40$, $p<0.05$), and memory ($r = .42$, $p<0.05$) sub-tests of the NSAIS. The significantly high negative correlations occurring between the 'Food Index' and age on testing, age on starting and stopping must be regarded as artifacts.

FULL SCALE INTELLIGENCE SCORE

The distribution of full scale intelligence scores over the four groups is shown in Fig. 2.

Table 8 shows the means and standard deviations for the four groups on the full scale scores on the NSAIS. The analysis of the data is presented in Tables 9 and 10. The fed group was significantly higher than the fed control group ($p<0.05$), the kwashiorkor group ($p<0.01$) and the kwashiorkor-control group ($p<0.05$). No significant differences were found between the kwashiorkor group and its control group, or between the two control groups, or between the kwashiorkor group and the fed control group.

Correlations between the four groups on full scale intelligence score are shown in Table 11. The pattern is that of significantly high positive correlations occurring between all groups except between the kwashiorkor group and the fed group.

Significantly high positive correlations were found consistently in all groups between full scale intelligence score and verbal ($r = .96$, $p<0.001$) and non-verbal ($r = .88$, $p<0.001$) scores (Tables 66, 67, 68, 69, 70, Appendix II).

Correlations between full scale intelligence score and other variables are shown in Tables 66 - 76 in Appendix II.

The full scale intelligence score did not correlate significantly with ordinal position in sibship (Table 7) in the kwashiorkor and fed groups, but a significantly high positive correlation ($r = .29$, $p<0.05$) occurred with the 'Index of Sibship' taking

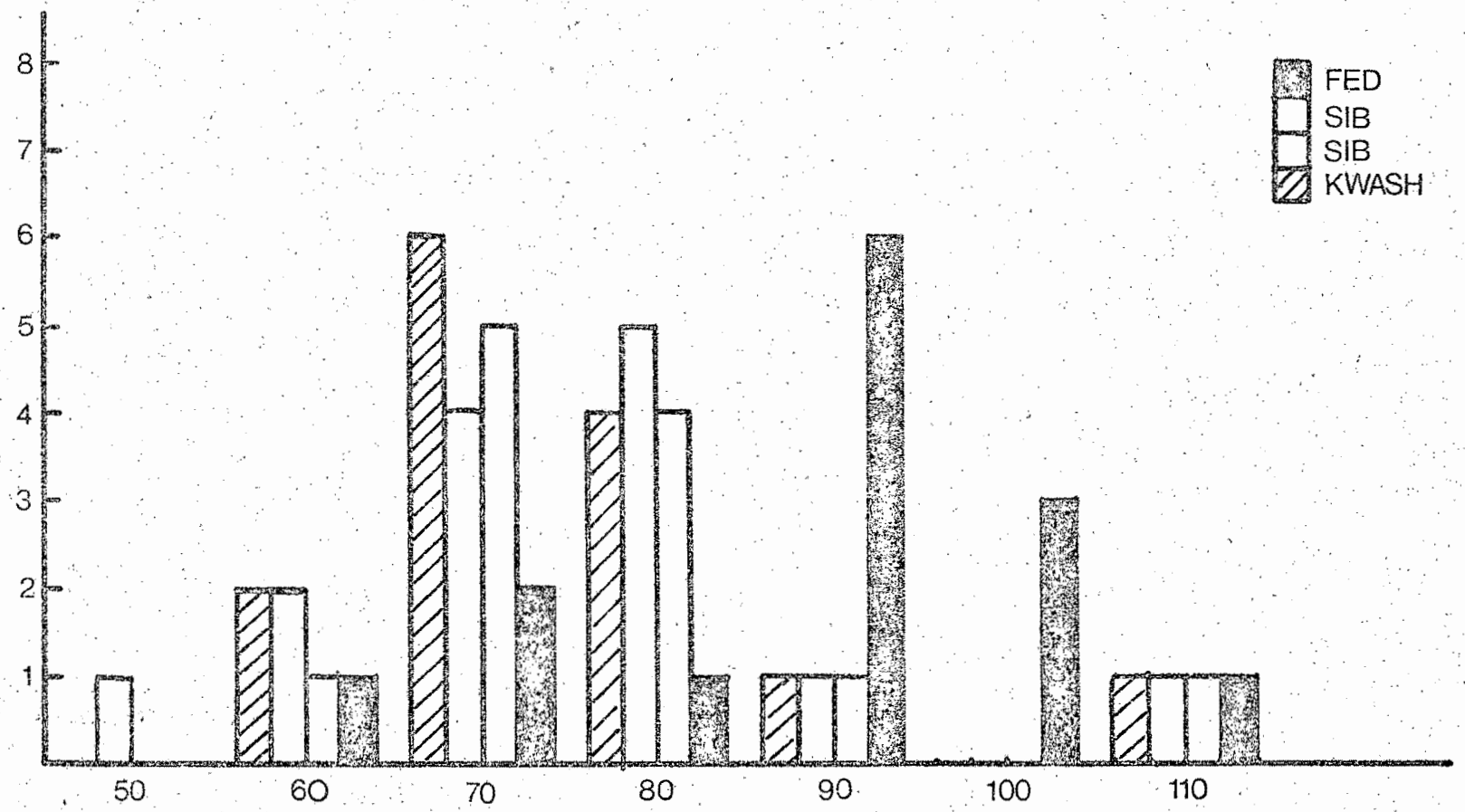


FIGURE 2 FULL SCALE INTELLIGENCE SCORES

TABLE 8: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON FULL SCALE INTELLIGENCE SCORES (NSAIS)

	Kwash	Sib K	Sib F	Fed
Mean	70.0	72.0	72.2	81.8
Sd	13.5	14.6	13.0	13.7

TABLE 9: ANOVA SUMMARY TABLE

One-Way Analysis with repeated measurements

Mean Full Scale Intelligence Scores for the four groups

Source	Sums of Squares	df	Mean Squares	F ratio
Between subjects	6434.7	11	584.0	
Within subjects	2860.3	36	79.5	
Full Scale Intelligence Score	1013.0	3	337.7	6.03**
Residual (error)	1847.3	33	56.0	

**p<0.01

TABLE 10: MULTIPLE COMPARISONS BETWEEN MEANS

Scheffe Multiple Comparisons

Mean Full Scale Intelligence Scores

Degrees of freedom (3,33) in all cases

	Kwash	Sib K	Sib F	Fed
Kwash	-	NS	NS	5.0**
Sib K		-	NS	3.5*
Sib F			-	3.1*
Fed				-

*p<0.05
**p<0.01

TABLE 11: CORRELATION COEFFICIENTS BETWEEN THE FOUR GROUPS ON FULL SCALE INTELLIGENCE SCORE (NSAIS)

(df 13)

	Kwash	Sib K	Sib F	Fed
Kwash	-			
Sib K	.75*	-		
Sib F	.74*	.78*	-	
Fed	.40	.75	.80*	-

p = 0.05*

account of family size. No significant correlation occurred between the intelligence score and the age on admission to hospital in the kwashiorkor group.

In the kwashiorkor group and in the fed control group the intelligence score correlated significantly with the Bender Gestalt test score ($r = .54$, $p<0.05$; $r = .79$, $p<0.01$).

In all groups except the kwashiorkor group the intelligence score showed a significantly high positive correlation with the Harris Drawing Score ($r = .73$, $p<0.01$ for fed group; $r = .56$, $p<0.05$ for fed control group; $r = .65$, $p<0.01$ for the kwashiorkor-control group).

Significantly high correlations occurred between intelligence score and the arbitary 'Food Index' ($r = .35$, $p = 0.01$), and this was substantiated by significantly high correlations between intelligence score and mid-term weight ($r = .32$, $p<0.05$) and between intelligence score and mid-term height ($r = .33$, $p<0.05$). In the fed control group only, full scale intelligence score showed significantly high positive correlations with early height ($r = .57$, $p<0.05$), and early weight ($r = .64$, $p<0.01$). (Table 76, Appendix II).

No significant correlations occurred between intelligence score and -

- Age on testing,
- Age at start of the supplementary feeding program,
- Age at the end of feeding program,
- Duration of the supplementary feeding program,
- Income,
- Multiple Social Index.

A significantly high positive correlation occurred between intelligence score and elapsed time from the end of the supplementation period to the time of testing in the fed group only ($r = .76$, $p<0.01$).

These results indicate that Proposition I and Proposition 4 should be accepted, whereas Proposition 2 and Proposition 3 must be rejected for overall intellectual developmental level.

VERBAL INTELLIGENCE SCORE

The means and standard deviations for the four groups on the Verbal sub-scale of the NSAIS are shoun in Table 12, Figure 3 shows the distribution of the scores. The analysis of the data are shown in Tables 13 and 14. The difference between the fed group and the fed control group did not quite reach statistical significance ($p>0.05, >0.10$). No significant differences occurred between the kwashiorkor group and its control group, or between the two sibling-control groups, or between the kwashiorkor group and the fed-control group.

Correlations between the four groups on verbal score are shown in Table 15. The pattern of significantly high inter-group correlations between all groups except between the fed group and the kwashiorkor group is identical to that found for full scale intelligence score.

Significantly high positive correlations occurred between verbal and non-verbal scores ($r = .72$, $p<0.001$) and between verbal and full scale intelligence scores ($r = .96$, $p<0.001$). This similar correlations were found consistently in all groups (see Appendix II).

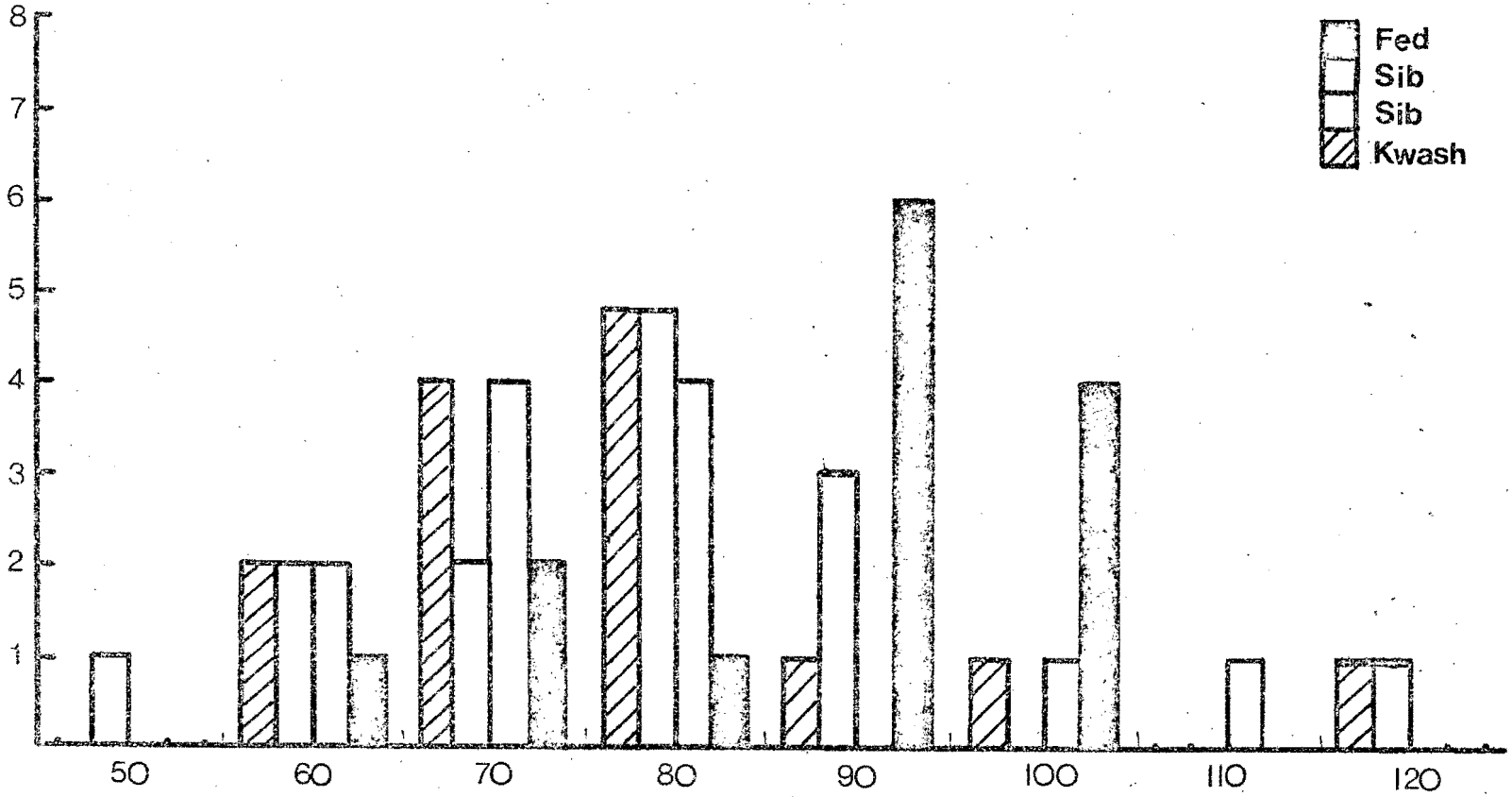


FIGURE 3

VERBAL INTELLIGENCE SCORES

TABLE 12: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON VERBAL INTELLIGENCE SCORE (NSAIS)

	Kwash	Sib K	Sib F	Fed
Mean	74.3	75.0	70.8	81.3
Sd	16.1	17.3	13.9	14.0

TABLE 13: ANOVA SUMMARY TABLE
One-Way Analysis with repeated measurements
Means for the four groups on Verbal Score NSAIS)

Source	Sums of Squares	df	Mean Squares	F ratio
Between subjects	7516.7	11	683.3	
Within subjects	3598.0	36	99.9	
Verbal Intell-igence Score	699.5	3	232.2	2.65
Residual (error)	2898.5	33		

TABLE 14: MULTIPLE COMPARISONS BETWEEN MEANS

Scheffe Multiple Comparisons

Verbal Score (NSAIS)

Degrees of freedom (3,33) in all cases

	Kwash	Sib K	Sib F	Fed
Kwash	-	NS	NS	NS
Sib K		-	NS	NS
Sib F			-	2.55*
Fed				-

* $0.05<p<0.10$

TABLE 15: CORRELATION COEFFICIENTS BETWEEN THE FOUR GROUPS ON VERBAL SCORE (NSAIS)

(df 13)

	Kwash	Sib K	Sib F	Fed
Kwash	-			
Sib K	.72*	-		
Sib F	.66*	.63*	-	
Fed	.44	.70*	.64*	-

p = 0.05*

Correlations between verbal score and other variables are shown in Appendix II.

In the fed-control group a highly significant correlation occurred between the verbal score and the time elapsed between the end of the supplementation period and the time of testing ($r = .74$, $p<0.01$). In this group also, verbal score correlated with early height ($r = .57$, $p<0.05$), and early weight (r 2 .66, $p<0.01$). (Table 76, Appendix II).

No significant correlations occurred between verbal score and:

- Age on testing,
- Age at start of the feeding program,
- Age at termination of the feeding program,
- Duration of feeding program,
- Food Index,
- Income,
- Multiple Social Index,
- Mid-term weight and height.

In the kwashiorkor group, verbal score did not correlate significantly with age on admission to hospital.

These results indicate that Proposition I should be accepted whereas Proposition 2 and Proposition 3 must be rejected for verbal score.

NON-VERBAL INTELLIGENCE SCORE

The means and standard deviations for the four groups on the non-verbal sub-scale of the NSAIS are presented shown in Table 16. Figure 4 shows the distribution of the scores. The analysis of data is presented in Tables 17 and 18. No significant differences occurred between the fed group and the fed-control group, or between the kwashiorkor group and the kwashiorkor-control group. The fed group was significantly higher than the kwashiorkor group ($p<0.001$) and significantly higher than the kwashiorkor-control group ($p<0.01$) on non-verbal score. The kwashiorkor group was significantly lower than the fed-control group ($p<0.01$).

Inter-group correlations for the four groups on non-verbal scores are shown in Table 19. Although the correlations between the kwashiorkor group and the two control groups do not quite reach statistical significance, the pattern is essentially the same as that found for full scale and verbal intelligence scores. The correlations of .51 and .52 are still of the order expected for siblings, and the low correlation of .23 between the kwashiorkor group and the fed group is contrary to expectation for siblings.

Significantly high positive correlations were found consistently in all groups for non-verbal and verbal ($r = .72$, $p<0.001$) and full scale intelligence scores ($r = .88$, $p<0.001$). (See Appendix II).

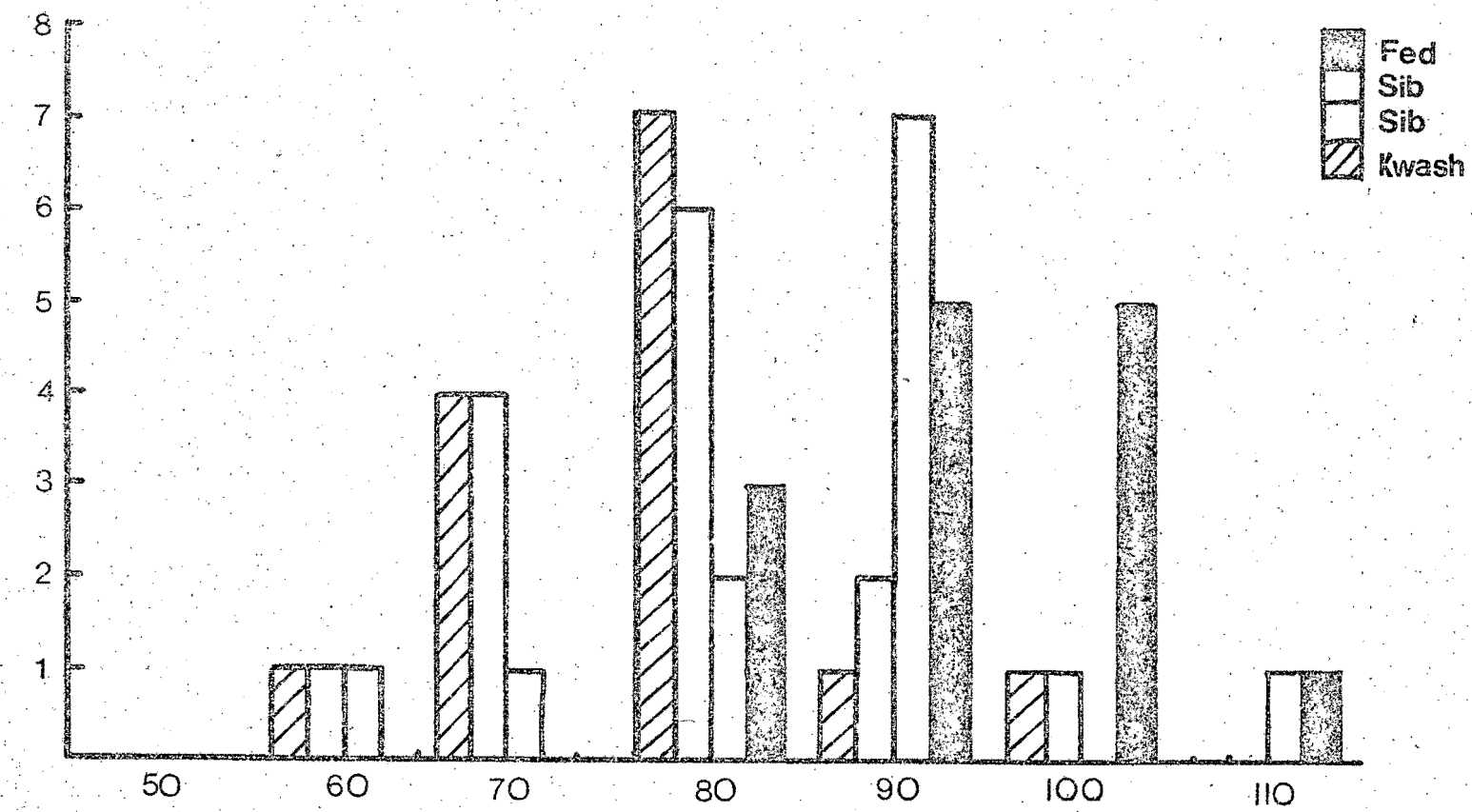


FIGURE 4 NON-VERBAL INTELLIGENCE SCORES

TABLE 16: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON NON-VERBAL INTELLIGENCE SCORE (NSAIS)

	Kwash	Sib K	Sib F	Fed
Mean	71.3	74.4	80.5	86.3
Sd	8.8	9.2	11.2	10.4

TABLE 17: ANOVA SUMMARY TABLE
One-Way Analysis with repeated measurements
Means for the four groups on Non-Verbal Score (NSAIS)

Source	Sums of Squares	df	Mean Squares	F ratio
Between subjects	2972.2	11	270.2	
Within subjects	2965.7	36	82.4	
Non-Verbal Intelligence Score	1594.7	3	531.6	12.79***
Residual (error)	1371.1	33	41.4	

TABLE 18: MULTIPLE COMPARISONS BETWEEN MEANS

Scheffe Multiple Comparisons

Non-Verbal Score (NSAIS)

Degrees of freedom (3,33) in all cases

	Kwash	Sib K	Sib F	Fed
Kwash	-	NS	5.0**	10.8***
Sib K		-	NS	7.0**
Sib F			-	NS
Fed				-

*p<0.05
**p<0.01
***p<0.001

TABLE 19: CORRELATION COEFFICIENTS BETWEEN THE FOUR GROUPS ON NON-VERBAL SCORE (NSAIS)

(df 13)

	Kwash	Sib K	Sib F	Fed
Kwash	-			
Sib K	.51	-		
Sib F	.52	.65*	-	
Fed	.23	.72*	.81*	-

p = 0.05*

Correlations between non-verbal intelligence score and the other variables are tabulated in Appendix II.

In the kwashiorkor group, a significantly high positive correlation was found between non-verbal score and Bender Gestalt Test score ($r = .57$, $p<0.05$). When the fed group and the fed-control group were combined this positive correlation was also significantly high ($r = .43$, $p<0.05$).

Non-verbal score correlated positively at a significantly high level with the Harris Drawing Score in the fed group ($r = .73$, $p<0.01$) and in the kwashiorkor-control group ($r = .77$, $p<0.01$). The correlation was also positive at significant level when the groups were combined ($r = .47$, $p<0.01$).

A significantly high positive correlation occurred between non-verbal score and the empirical 'Food Index' ($r = .55$, $p = 0.01$). Non-verbal score also show significantly high positive correlations with mid-term weight ($r = .37$, $p<0.05$), and mid-term height ($r = .39$, $p<0.05$). In the fed-control group, weight at present showed a significantly high positive correlation with non-verbal score ($r = .57$, $p<0.05$). In this group the elapsed time between the end of the supplementation and testing showed a significantly high positive correlation with non-verbal score ($r = .64$, $p<0.05$).

In the kwashiorkor group, Multiple Social Index correlated at a significantly high positive level with non-verbal score ($r = .54$, $p<0.05$).

In the kwashiorkor group no significant correlation occurred between age on admission to hospital and non-verbal score.

No significant correlations occurred between non-verbal intelligence score and:

- Age on testing,
- Age at start of supplementation,
- Age at termination of the feeding program,
- Duration of supplementation,
- Income.

These results indicate that the requirements for the acceptance of Proposition I and Proposition 2 and Proposition 4 are satisfied in part for non-verbal intelligence score. Proposition 3 must be rejected.

NSAIS SUB-TESTS

The mean scaled scores for each of the four groups on each of the nine NSAIS sub-tests are shown in Table 20. The analysis is presented in Table 21. The main effect of group assignment ($p<0.001$) and NSAIS sub-tests ($p<0.001$) was significant, and the inter-action was significant ($p<0.01$) Simple main effects are shown in Table 22. Group effects were significant for Vocabulary, Verbal Reasoning, Pattern Completion and Form Board sub-tests. The effect of sub-test was significant on all four groups.

Tables 23 to 26 show multiple comparisons between the sub-test means within each group.

Significantly high differences within the four groups occur most frequently between Vocabulary, Comprehension, Verbal Reasoning, Pattern Completion, Block Design, and Form Board sub-tests.

TABLE 20: MEANS FOR THE FOUR GROUPS ON THE NINE NSAIS SUB-TESTS

	Kwash	Sib K	Sib F	F
Vocabulary	7.34	7.81	4.58	7.08
Comprehension	6.91	6.41	6.83	8.33
Verbal Reasoning	8.00	7.66	8.58	10.67
Arith. Problems	6.08	7.34	6.67	7.92
Memory	5.16	4.75	4.17	5.25
Pattern Completion	5.33	6.00	7.08	9.08
Block Design	6.58	6.50	8.25	9.17
Absurdities	6.50	6.00	5.67	6.33
Form Board	5.92	7.58	8.50	8.25

TABLE 21: ANOVA SUMMARY TABLE

Two-Way Analysis with repeated measurements

Means for the four groups on NSAIS sub-tests

Source	Sum of Squares	df	Mean Squares	F
Between subjects				
Subj. within group	947.8	11		
Within subjects				
A: Group of child	165.5	3	55.2	14.6***
B: NSAIS sub-test	444.2	8	55.5	14.7**
A x B	244.0	24	10.2	2.7**
B x subj. within Group	1463.0	385	3.8	

**$p < 0.01$

***$p < 0.001$

TABLE 22: ANOVA SIMPLE MAIN EFFECTS SUMMARY TABLE

(Analysis taken from Table 21)

Source	Sum of Squares	df	Mean Squares	F ratio
Group at Vocabulary	75.7	3	25.2	6.7**
Group at Comprehension	25.0	3	8.4	2.2
Group at Verbal Reason.	65.2	3	21.7	5.7**
Group at Arith. Problems	22.8	3	7.6	2.0
Group at Memory	8.8	3	2.9	0.8
Group at Patt. Comp.	96.7	3	32.2	8.5**
Group at Block Des.	61.4	3	20.5	5.4**
Group at Absurdities	4.9	3	1.6	0.4
Group at Form Board	48.7	3	16.2	4.3**
Sub-test for Kwash	80.7	8	10.1	2.7**
Sub-test for Sib K	99.6	8	12.4	3.3**
Sub-test for Sib F	255.9	8	32.0	8.5**
Sub-test for Fed	252.1	8	31.5	8.3**
		385		

*$p < 0.05$
**$p < 0.01$

TABLE 23: MULTIPLE COMPARISONS BETWEEN MEANS FOR KWASHIORKOR GROUP ON NSAIS SUB-TESTS

TUKEY HSD

Degrees of freedom (9,385) in all cases

	Mem-ory	Patt. Compl.	Form Board	Arith. Probs.	Absurd-ities	Block Des.	Compre-hension	Vocab-ulary	Verbal Reas.
Memory	-							?	*
Patt. Compl.		-							*
Form Board			-						?
Arith. Probs.				-					
Absurdities					-				
Block Des.						-			
Compreh.							-		
Vocabulary								-	
Verbal Reas.									-

*p<0.05

TABLE 24: MULTIPLE CONPARISONS BETWEEN MEANS FOR KWASHIORKOR CONTROL GROUP ON NSAIS SUB-TESTS

TUKEY HSD

Degrees of freedom (9,385) in all cases

	Mem-ory	Patt. Compl.	Absurd-ities	Compre-hension	Block Des.	Arith. Probs.	Form Board	Verbal Reas.	Vocab-ulary
Memory	-					*	*	*	**
Patt.Compl.		-							?
Absurdities			-						
Compreh.				-					
Block Des.					-				
Arith. Probs.						-			
Form Board							-		
Verbal Reas.								-	
Vocabulary									-

*p<0.05
**p<0.01

TABLE 25: MULTIPLE COMPARISONS BETWEEN MEANS FOR THE FED GROUP ON NSAIS SUB-TESTS

TUKEY HSD

Degrees of freedom (9,385) in all cases

	Memory	Vocabulary	Absurdities	Arith. Probs.	Comprehension	Patt. Compl.	Block Des.	Form Board	Verbal Reas.
Memory	-			*	**	**	**	**	**
Absurdities		-					*	**	**
Vocabulary			-						*
Arith.Probs				-					*
Form Board					-				
Compr.						-			
Patt.Compl.							-		
Block Des.								-	
Verbal Reas.									-

*p<0.05
**p<0.01

TABLE 26: MULTIPLE COMPARISONS BETWEEN MEANS FOR THE FED CONTROL GROUP ON NSAIS SUB-TESTS

TUKEY HSD

Degrees of freedom (9,385) in all cases

	Memory	Vocabulary	Absurdities	Arith. Probs.	Comprehension	Patt. Compl.	Block Des.	Form Board	Verbal Reas.
Memory	-			*	*	**	**	**	**
Vocabulary		-				*	**	**	**
Absurdities			-				*	*	**
Arith.Probs.				-					
Comprehension					-				
Patt.Compl.						-			
Block Des.							-		
Form Board								-	
Verbal Reas.									-

*p<0.05
**p<0.01

Table 27 shows the significance of difference between the means of the four groups on Vocabulary, Comprehension, Verbal Reasoning, Pattern Completion, Block Design and Form Board sub-tests. The mean sub-test scores are plotted in Figure 5 and may be summarised as follows:

1) The fed group showed a significantly higher score than each of the other three groups on Verbal Reasoning and Pattern Completion sub-tests. The same trend was shown in Comprehension, but the greatest difference (between fed group and kwashiorkor-control group) did not quite reach statistical significance.
2) On Vocabulary the kwashiorkor-control group was significantly below each of the other three groups.
3) On the Form Board the kwashiorkor group was significantly lower than both the fed and fed-control groups, but the difference between kwashiorkor group and kwashiorkor-control group was not quite significant although the trend was similar.
4) On Block Design the fed group had a significantly higher score than both the kwashiorkor and kwashiorkor control groups. The difference between the two control groups was not quite statistically significant.
5) Arithmetic Problems, Memory and the Absurdities sub-tests did not discriminate between the four groups.

In order to examine the sub-test scatter the standard deviations obtained for each group are shown in Table 28. No significant differences in sub-test scatter were found between the groups.

TABLE 27: MULTIPLE COMPARISONS BETWEEN MEANS FOR NSAIS SUB-TESTS ON THE FOUR GROUPS

TUKEY HSD

(Degrees of freedom 4,385 in all cases)

VOCABULARY

	SF	F	SK	K
SF	—	**	**	**
F		—		
SK			—	
K				—

COMPREHENSION

	SK	SF	K	F
SK	—			?
SF		—		
K			—	
F				—

VERBAL REASONING

	SK	K	SF	F
SK	—			** **
K		—		**
SF			—	*
F				—

*p<0.05
**p<0.01

Continued on pg. 151.

TABLE 27: MULTIPLE COMPARISONS BETWEEN MEANS FOR NSAIS SUB-TESTS ON THE FOUR GROUPS

TUKEY HSD

(Degrees of freedom 4,385 in all cases)

PATTERN COMPLETION

	K	SK	SF	F
K	-		?	**
SK		-		**
SF			-	*
F				-

BLOCK DESIGN

	SK	K	SF	F
SK	-		?	
K		-		**
SF			-	**
F				-

FORM BOARD

	K	SK	F	SF
K	-	?	*	**
SK		-		
F			-	
SF				-

*p<0.05
**p<0.01

TABLE 28: GRAND STANDARD DEVIATIONS ON NSAIS SUB-TEST SCORES FOR THE FOUR GROUPS

	Kwash	Sib K	Sib F	Fed
Sd	2.42	2.74	2.72	2.72

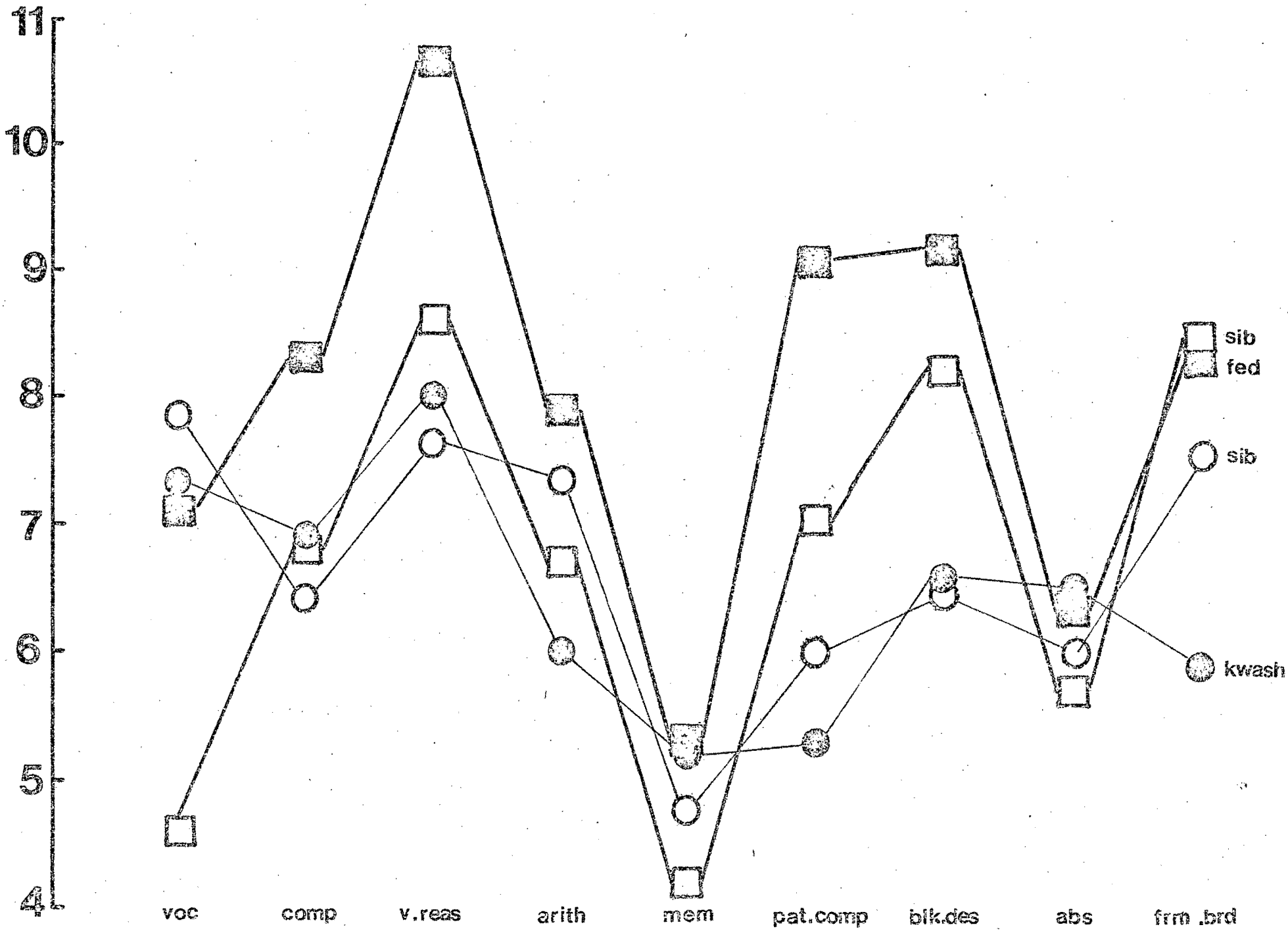


FIGURE 5 MEAN SUB-TEST SCORES

Correlations between the NSAIS sub-test scores and other variables are shown in Tables 71, 72 and 73 (Appendix II).

'Food Index' showed a significantly high positive correlation with both Vocabulary ($r = .40$, $p<0.005$) and Pattern Completion ($r = .42$, $p<0.005$).

Duration of feeding showed a significantly high positive correlation with Arithmetic Problems ($r = .56$, $p<0.01$), Memory ($r = .54$, $p<0.01$), Verbal Reasoning ($r = .48$, $p<0.05$) and Absurdities ($r = .49$, $p<0.05$).

Income showed a significantly high positive correlation with Pattern Completion ($r = .40$, $p<0.05$) and Block Design ($r = .41$, $p<0.05$).

The length of time between termination of supplementation and testing showed a significantly high positive correlation with Arithmetic Problems ($r = .44$, $p<0.05$). Significantly high positive correlations occurred between the age at which feeding started and Pattern Completion ($r = .60$, $p<0.01$), Form Board ($r = .50$, $p<0.01$), and Block Design ($r = .46$, $p<0.05$). Negative correlations of a significantly high level occurred between the age at termination of supplementation and Arithmetic Problems ($r = -0.39$, $p<0.05$), and Pattern Completion ($r = -0.45$, $p<0.05$).

Significantly high negative correlations also occurred between all of the verbal sub-tests and the verbal/non-verbal discrepancy (Table 71, Appendix II).

In the fed group only a significantly high positive correlation occurred between Comprehension and age on testing ($r = .58$, $p<0.05$), and duration of feeding ($r = .54$, $p<0.05$).

In the fed control group the length of elapsed time between termination of feeding and testing showed significantly high positive correlations with Vocabulary ($r = .82$, $p<0.01$), Comprehension ($r = .70$, $P<0.01$), Arithmetic Problems ($r = .68$, $p<0.05$), Memory ($r = .63$, $p<0.05$), and Pattern Completion ($r = .58$, $p<0.05$).

HARRIS DRAWING TEST

Table 29 shows the means and standard deviations for the four groups on the Harris revision of the Goodenough Draw-a-Man Test. Table 30 shows the analysis of data. No significant differences were found in the means between the four groups.

The inter-group correlations on Harris Score are shown in Table 31. Significantly high positive correlations occurred between the fed control group and both the fed group ($r = .54$, $p<0.05$) and the kwashiorkor group ($r = .49$, $p<0.05$).

Co-efficients of correlation between Harris Score and other variables are shown in Tables 66 to 73 in Appendix II.

Harris Score did not correlate at a significantly high level with the Index of Sibship.

Significantly high positive correlations occurred between Harris Score and full scale intelligence score ($r = .54$, $p<0.01$), Verbal Score ($r = .50$, $p<0.01$), non-verbal Score ($r = .47$, $p<0.01$) and the Bender Gestalt Test ($r = .50$, $p<0.01$), in the kwashiorkor group these significantly high correlations did not occur, and in the fed control group Harris Score did not correlate significantly with verbal and non-verbal scores.

In the fed control group Harris Score correlated at a significantly high level with Verbal Reasoning ($r = .59$, $p<0.05$), and Arithmetic Problems ($r = .60$, $p<0.05$). In the fed group Harris Score correlated significantly with Vocabulary ($r = .66$, $p<0.01$) and Comprehension ($r = .74$, $p<0.01$), and Absurdities ($r = .75$, $p = 0.01$).

TABLE 29: MEANS AND STANDARE DEVIATIONS FOR THE FOUR GROUPS ON HARRIS DRAWING SCORE

	Kwash	Sib K	Sib F	Fed
Mean	81.3	83.7	75.1	81.0
Sd	17.6	16.9	11.1	13.3

TABLE 30: ANOVA SUMMARY TABLE

One-Way Analysis with repeated measures

Means for the four groups on Harris Drawing Score

Source	Sum of Squares	df	Mean Squares	F ratio
Between subjects	4973.4	11	452.1	
Within subjects	5366.5	36	149.1	
Harris Drawing Scale	488.1	3	162.7	NS
Residual (error)	4878.4	33	147.8	

TABLE 31: CORRELATION COEFFICIENTS BETWEEN THE FOUR GROUPS ON HARRIS DRAWING SCORE

(df 10)

	Kwash	Sib K	Sib F	Fed
Kwash	-			
Sib K	.41	-		
Sib F	.49*	.19	-	
Fed	.19	.32	.54*	-

*p<0.05

When fed and fed control groups were combined additional significantly high positive correlations occurred with Memory ($r = .44$, $p<0.05$).

In the fed group Harris Score showed significantly high positive correlations with:

- Age on testing ($r = .66$, $p<0.01$),
- Duration of feeding ($r = .78$, $p<0.01$),
- Age at start of feeding ($r = .60$, $p<0.05$),
- Age at termination of feeding ($r = .68$, $p<0.01$).

These significant correlations did not occur in the fed-control group or when the two groups were combined.

The means for the four groups on the Machover rating are shown in Table 32. Table 33 shows the mean discrepancy between Man and Woman scales of the Harris Drawing Test for each of the four groups. No significant differences were found between the groups on the Machover rating or on the discrepancy between Man and Woman Scales, and these variables did not show significant correlations with the Multiple Social Index, or with total Harris Score.

The Harris Drawing Test had been administered previously to nine of the kwashiorkor subjects and their nine controls as part of an earlier investigation. The means and standard deviations on Harris Score for these kwashiorkor subjects and their controls in 1968 and 1973 are compared in Table 34. Table 35 presents the analysis of data: no significant differences were found between mean scores in 1968 and 1973.

TABLE 32: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON MACHOVER RATING

	Kwash	Sib K	Sib F	Fed
Mean	0.71	0.7	0.8	0.8
Sd	0.8	0.8	1.0	0.7

TABLE 33: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON MAN-WOMAN SCALE DISCREPANCY
(Accounting for direction of difference)

	Kwash	Sib K	Sib F	Fed
Mean	-4.6	0.5	1.6	1.7
Sd	7.7	3.2	6.2	9.0

TABLE 34: MEANS AND STANDARD DEVIATIONS FOR KWASHIORKOR SUBJECTS AND THEIR CONTROLS ON HARRIS DRAWING TEST IN 1968 AND 1973

(N = 9 pairs)

	Kwash(N=9)		SibK(N=9)	
	1968	1973	1968	1973
Mean	80.4	85.0	83.8	88.3
Sd	11.9	18.5	10.8	15.8

TABLE 35: ANOVA SUMMARY TABLE

Two-Way Analysis with repeated measurements Harris Drawing Scores in Kwashiorkor subjects and their controls in 1968 and 1973

Source	Sums of Squares	df	Mean Squares	F ratio
Between subjects:				
A: Kwash at Sib K.	103.3	1	103.3	
Error B	2024.9	16	314.1	
Within subjects:				
B: 1968/1973	182.2	1	182.2	NS
A x B	0.0	1	0.0	
Error		16	110.4	

BENDER GESTALT TEST

The means and standard deviations for the four groups on Perceptual Quotient are shown in Table 36. The analysis is presented in Table 37. No effects were found to be significant. Inter-group correlation co-efficients are shown in Table 38. The fed group showed a significantly high positive correlation with both the fed control group ($r = .60$, $p<0.05$), and the kwashiorkor control group ($r = .55$, $p<0.05$).

The means and standard deviations on the Bender t scores (See Appendix III) are shown for the four groups in Table 39. The analysis is presented in Table 40. No effects were found to be significant. Inter-group correlation co-efficients are shown in Table 41. The fed group correlated positively with the fed control group at a significantly high level ($r = .63$, $p <0.05$).

Co-efficients of correlation for the Bender Gestalt Test with other variables are shown in Appendix II, Tables 66 to 73.

Significantly high positive correlations occurred with full-scale intelligence score ($r = .36$, $p<0.01$), Verbal Score ($r = .38$, $p<0.01$), and Harris Drawing Score ($r = .50$, $p<0.01$). These correlations did not occur in the fed group.

When the fed and fed control group were combined the Bender Gestalt Test showed significantly high positive correlations with full-scale intelligence score ($r = .53$, $p<0.01$), Verbal Score ($r = .54$, $p<0.01$), Non-Verbal score ($r = .43$, $p<0.05$), Comprehension ($r = .51$, $p<0.01$), Arithmetic Problems ($r = .61$, $p<0.01$), and Memory ($r = .62$, $p<0.01$).

These correlations did not occur in the fed group.

TABLE 36: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON BENDER GESTALT TEST (PERCEPTUAL QUOTIENT)

	Kwash	Sib K	Sib F	Fed
Mean	71.2	62.5	65.7	63.5
Sd	30.5	21.4	18.5	19.6

TABLE 37: ANOVA SUMMARY TABLE

One-Way Analysis with repeated measurements

Means for the four groups on Bender Gestalt Test (Perceptual Quotient)

Source	Sum of Squares	df	Mean Squares	F ratio
Between subjects	10596	11	963.3	
Within subjects	13253	36	368.1	
Bender Gestalt Test (Perceptual Quotient)	550.5	3	183.5	NS
Residual (error)	12702.5	33	384.9	

TABLE 38: CORRELATION COEFFICIENTS BETWEEN THE FOUR GROUPS ON PERCEPTUAL QUOTIENT (BENDER GESTALT TEST)

(df 10)

	Kwash	Sib K	Sib F	Fed
Kwash	-			
Sib K	.13	-		
Sib F	.34	.37	-	
Fed	-.0	.55*	.60*	-

p = 0.05*

TABLE 39: MEANS AND STANDARD DEVIATIONS FOR THE FOUR GROUPS ON BENDER GESTALT TEST (t' SCORES)

	Kwash	Sib K	Sib F	Fed
Mean	29.7	28.2	18.7	17.6
Sd	22.2	20.5	18.8	17.6

TABLE 40: ANOVA SUMMARY TABLE

One-Way Analysis with repeated measurements

Means on Bender Gestalt Test for the four groups

Source	Sum of Squares	df	Mean Squares	F ratio
Between subjects	8223.2	11	747.5	
Within subjects	10516.7	36	292.1	
Bender Gestalt Test (t'-Score)	1418.1	3	472.7	NS
Residual (error)	9098.7	33	275.7	

TABLE 41: CORRELATION COEFFICIENTS BETWEEN THE FOUR GROUPS ON BENDER GESTALT TEST (t' Score)

(df 10)

	Kwash	Sib K	Sib F	Fed
Kwash	-			
Sib K	-.07	-		
Sib F	.23	.47	-	
Fed	.17	.51	.63**	-

p = 0.05*

HEIGHT

The height of each child expressed as percentage of normal expected height for age is shown in Table 59 in Appendix I, together with the age at which the measurements were taken. Heights were recorded at as near as possible comparable ages within each family at two points in time called mid-term and present. Where possible the mid-term heights were taken within 2 years following the termination of dietary supplementation. Early height in the fed and fed control groups was recorded approximately half-way through the supplementation program.

The means for the four groups on mid-term and present height are shown in Table 42. The analysis of the data are presented in Table 43. Significant effects on groups, measurements and inter-action were found to be significant. Table 44 shows the simple main effects. Multiple comparisons between means are shown in Table 45 and Figure 6.

The results indicate that the fed group children were significantly taller than the members of the other three groups at mid-term stage. At present stage there was no significant difference in height between the four groups, the kwashiorkor group and both the control groups having caught-up with the fed group. The kwashiorkor group and both control groups showed a significant increase in height from mid-term to present stages.

Table 46 shows the mean heights in the fed and fed control groups at three developmental levels. The analysis of the data is presented in Table 47. Significant group, stage of measurement,

TABLE 42: MEANS FOR THE FOUR GROUPS ON HEIGHT AT MIDTERM AND PRESENT AGES (% normal for Age)

	Kwash	Sib K	Sib F	Fed
Midterm	82.5	81.9	84.7	90.3
Present	90.4	90.5	89.7	90.5

TABLE 43: ANOVA SUMMARY TABLE

Two-Way Analysis with repeated measurements

Means on height at midterm and present ages for the four groups

Source	Sum of Squares	df	Mean Squares	F ratio
Between Subjects				
Subj. within Groups	999.6	11		
Within subjects				
A: Group	196.8	3	65.6	5.4**
B: Stages of Measurement	694.8	1	694.9	65.7***
			91.8	7.5**
A x B	275.5	3	12.3	
B x subj. within Groups	943.4	77		

TABLE 44: ANOVA SIMPLE MAIN EFFECTS ON HEIGHT SUMMARY TABLE

(Analysis taken from Table 43)

Source	Sum of Squares	df	Mean Squares	F ratio
Group at midterm	465.9	3	155.3	12.7**
Group at present	6.3	3	2.1	0.17
Kwash at stage meas'd	378.4	1	378.4	30.9**
Sib K at stage meas'd	442.9	1	442.9	36.2**
Sib F at stage meas'd	149.0	1	149.0	12.2**
Fed at stage meas'd	0.03	1	0.03	0.002

**p< 0.01

TABLE 45: MULTIPLE COMPARISONS BETWEEN MEANS ON MIDTERM AND PRESENT HEIGHT

TUKEY HSD

(Data extended from Analysis Table 43)

Midterm	Kwash	Sib K	Sib F	Fed
Kwash	-	NS	NS	**
Sib K		-	NS	**
Sib F			-	*
Fed				-
Present				
Kwash	-	NS	NS	NS
Sib K		-	NS	NS
Sib F			-	NS
Fed				-

*p<0.05
**p<0.01

TABLE 46: MEANS FOR FED AND FED CONTROL GROUPS ON HEIGHT AT THREE STAGES (% normal for Age)

	Sib F	Fed
Early	86.2	91.8
Midterm	84.7	90.3
Present	89.7	90.5

TABLE 47: ANOVA SUMMARY TABLE

Two-Way Analysis with repeated measurements

Mean Early, Midterm and Present height for Fed and Fed control groups

Source	Sum of Squares	df	Mean Squares	F ratio
Between subjects				
Subj. within Groups	552.1	10		
Within subjects				
A: Group	260.7	1	260.7	33.0***
B: Stage of Measurement	68.6	2	34.3	4.3*
A x B	75.3	2	37.3	4.8*
B x subjs. within Groups	394.7	50	7.9	

*$p<0.05$
***$p<0.001$

TABLE 48: ANOVA SIMPLE MAIN EFFECTS SUMMARY TABLE

Mean Early, Midterm and Present height for Fed and Fed control groups

(Analysis taken from Table 47)

Source	Sum of Squares	df	Mean Squares	F ratio
Group at Early stage	168.0	1	168.0	21.3**
Group at Midterm	163.0	1	163.0	20.7**
Group at Present	4.9	1	4.9	0.6
Stage meas'd for Fed	13.2	2	6.6	0.8
Stage meas'd for Sib F	130.8	2	65.4	8.3*

*$p<0.05$
**$p<0.01$

and inter-action effects were found. ANOVA simple main effects are shown in Table 48. Multiple comparisons of the means were computed and revealed significant differences between the two groups at early and mid-term levels. The difference between the groups in height at present stage was not significant.

The means for the four groups at the selected developmental stages are shown graphically in Figure 6. The results suggest that the growth of the fed control child did not benefit significantly from any overflow of food from the fed child.

Correlation co-efficients between height at the three developmental levels and other variables are shown in Tables 66 to 70 in Appendix II. Mid-term height showed significantly high positive correlations with:

- Mid-term weight ($r = .77$, $p<0.01$),
- Index of Sibship ($r = .38$, $p<0.05$),
- Full scale intelligence score ($r = .33$ $p<0.05$),
- Non-verbal intelligence score ($r = .39$, $p<0.05$).

Several significantly high positive correlations occurred between height and weight which are irrelevant to the present investigation.

In the fed control group only, significantly high positive correlations occurred between early height and verbal score ($r = .57$, $p<0.05$), and between early height and full scale intelligence score ($r = .57$, $p<0.05$). (Table 76, Appendix II).

WEIGHT

The weight of each child (expressed as percentage of normal expected weight for age) is shown in Table 59 in Appendix I, together with the age at which the measurements were made. These were recorded at as near as possible comparable ages within each family at two points in time called mid-term and present. Whenever possible the mid-term weights were taken within 2 years following the end of the program of dietary supplementation. In the fed and fed control groups, early weight was recorded approximately half-way through the supplementation program.

The means for the four groups on mid-term and present weight are shown in Table 49. The analysis of the data are presented in Table 50. Significant effects were found for both groups and inter-action . ANOVA simple main effects are shown in Table 51. Multiple comparisons between means (Tukey HSD) were computed on data extended from Table 50 and are shown in Table 52 and Figure 7.

The results indicate that whilst the fed group children weighed significantly more at the end of the supplementary nutritional program than the children of the other three groups, this advantage was not maintained for long after supplementation of diet ceased. The fed children are at present not superior in weight to their unfed siblings.

The mean weights in the fed and fed control groups at three developmental stages are shown in Table 53. The analysis of data is presented in Table 54. Significant group and inter-action effects were found. ANOVA simple main effects are shown in Table 55.

TABLE 49: MEANS FOR THE FOUR GROUPS ON WEIGHT AT MIDTERM AND PRESENT AGES (%normal for Age)

	Kwash	Sib K	Sib F	Fed
Midterm	75.4	71.6	77.0	86.2
Present	76.5	75.3	78.2	78.5

TABLE 50: ANOVA SUMMARY TABLE

Two-Way Analysis with repeated measurements

Midterm and Present weights in the four groups

Source	Sum of Squares	df	Mean Squares	F ratio
Between subjects				
Subjs. within Groups	5611.3	11		
Within subjects				
A: Group	1011.8	3	337.3	6.0**
B: Stage of measurement	5	1	5	0.9
A x B	454.6	3	151.5	2.7*
B x subjs. within Grps.	4291.5	33	55.7	

*p<0.05
**p<0.01

TABLE 51: ANOVA SIMPLE MAIN EFFECTS ON WEIGHT SUMMARY TABLE

(Analysis taken from Table 50)

Source	Sum of Squares	df	Mean Squares	F ratio
Groups at Midterm	1386.6	3	462.2	8.3*
Groups at Present	79.7	3	26.6	0.48
Kwash at stage meas'd	7.9	1	7.9	0.14
Sib K at stage meas'd	81.4	1	81.4	1.5
Sib F at stage meas'd	8.3	1	8.3	0.15
Fed at stage meas'd	361.9	1	361.9	6.5

*p<0.05

TABLE 52: MULTIPLE COMPARISONS BETWEEN MEANS ON MIDTERM AND PRESENT WEIGHT

TUKEY HSD

(Data extended from Analysis Table 50)

Midterm	Kwash	Sib K	Sib F	Fed
Kwash	-	NS	NS	**
Sib K		-	NS	**
Sib F			-	**
Fed				-
Present				
Kwash	-	NS	NS	NS
Sib K		-	NS	NS
Sib F			-	NS
Fed				-

**p<0.01

Multiple comparisons of the means were computed and showed significant differences between the two groups at early ($p<0.01$) and mid-term stages ($p<0.01$). In the fed group, present weight was significantly lower than mid-term weight ($p<0.01$). In the fed control group mid-term weight was significantly higher than early weight ($p<0.01$). These results indicate that the fed control child gained some immediate benefit from the dietary supplementation of the fed child as long as the supplementation continued, but not sufficient to allow him to catch-up to the fed child. (It should be noted that all groups fall below the 3rd percentile at present). The means for the four groups at the three developmental stages are represented graphically in Figure 7.

Correlation co-efficients for the four groups on weight at the three developmental stages are shown in Appendix II, Tables 66 to 70. Mid-term weight showed significantly high positive correlations with:

- Full scale intelligence score ($r = .32$, $p<0.05$),
- Non-verbal intelligence score ($r = .37$, $p<0.05$),
- Index of Sibship ($r = .30$, $p<0.05$).

Several significantly high positive correlations occurred between weight and height which are not regarded as relevant to this investigation.

In the fed control group only early weight showed significantly high positive correlations with verbal score ($r = .66$, $p<0.01$), and with full scale intelligence score ($r = .64$, $p<0.01$). (Table 76, Appendix II).

TABLE 53: MEANS FOR FED AND FED-SIBLING GROUPS ON WEIGHT AT THREE STAGES (%normal for Age)

	Fed	Sib F
Early	86.0	72.0
Midterm	85.7	77.4
Present	79.6	77.9

TABLE 54: ANOVA SUMMARY TABLE

Two-Way Analysis with repeated measurements

Mean Early, Midterm and Present weight for Fed and Fed-Sibling groups

Source	Sums of Squares	df	Mean Squares	F ratio
Between subjects				
Subjs. within Groups	2271.4	10		
Within subjects				
A: Group	1057.8	1	1057.8	25.0***
B: Stage of Measurement	104.6	2	52.3	1.2
A x B	419.9	2	209.9	5.0**
B x subjs. within Grps.	210.5	50	42.2	

**p<0.01
***p<0.001

TABLE 55: ANOVA SIMPLE MAIN EFFECTS SUMMARY TABLE

(Analysis taken from Table 54)

Source	Sum of Squares	df	Mean Squares	F ratio
Group at Early stage	1079.4	1	1079.4	25.6**
Group at Midterm	383.1	1	383.1	9.1*
Group at Present	15.2	1	15.2	0.4
Stage meas'd for Fed	293.8	2	146.9	3.5
Stage meas'd for Sib F	230.8	2	115.4	2.7

*$p<0.05$
**$p<0.01$

FIGURE 6 HEIGHT Z normal

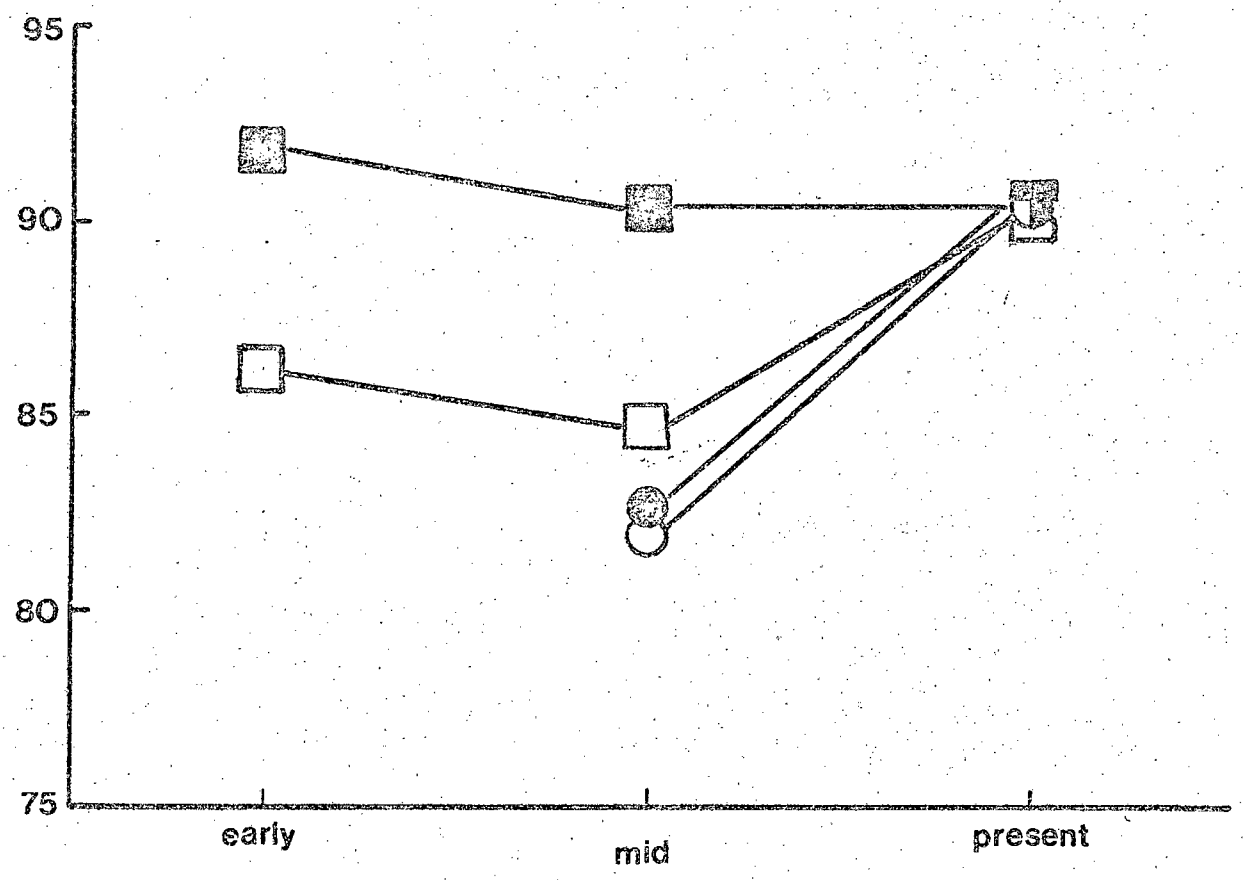
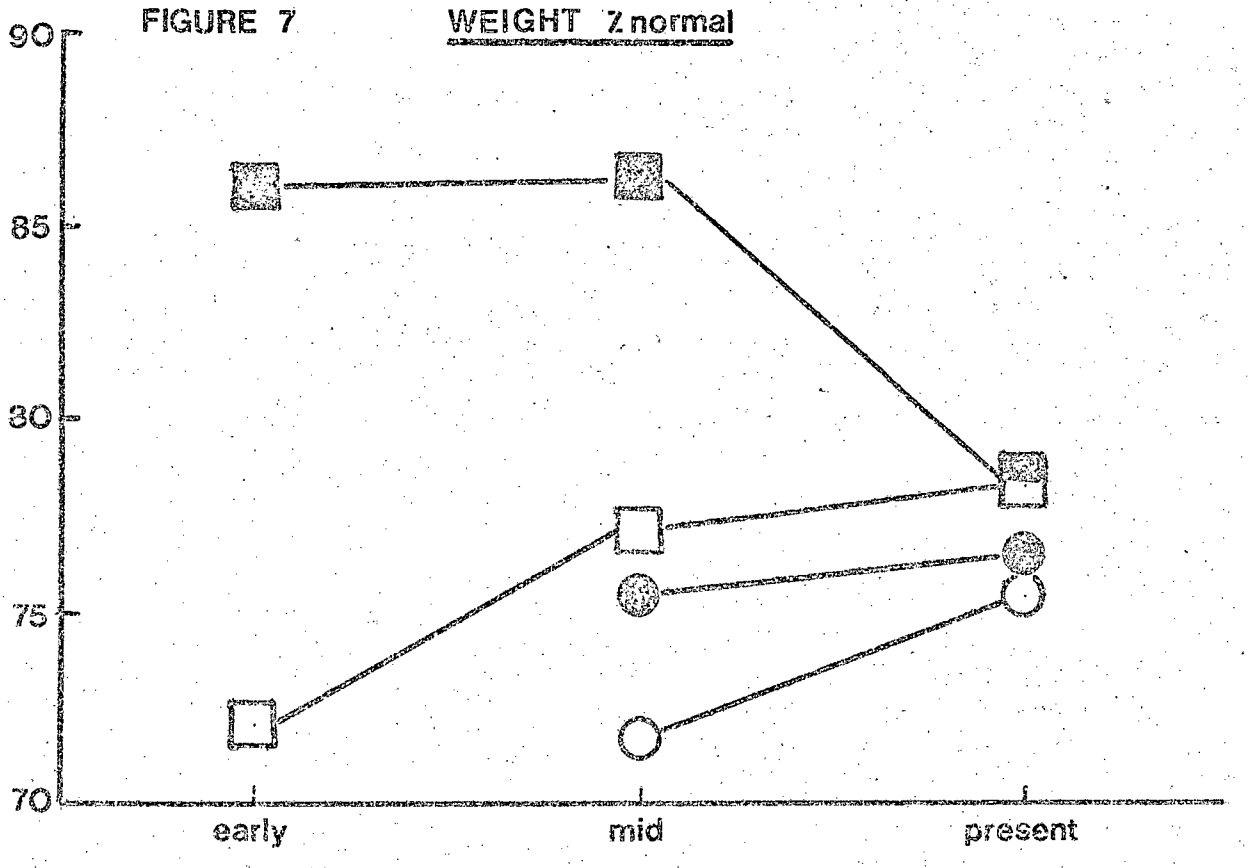


FIGURE 7 WEIGHT Z normal



CHAPTER 11

DISCUSSION AND CONCLUSION

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The results of this study confirm the central hypothesis: the fed group was significantly superior on full-scale intelligence score to each of the other three groups (Table 10). Whilst it is recognised that an 'IQ' does not describe everything about an individual's intellectual or cognitive functioning, it remains the most frequently used composite index of the intellectual status of an individual or a group. Where individual, and especially group differences are the subject of investigation, the assessment of intelligence by the use of standard IQ tests is the most efficient procedure. The results show that early dietary supplementation significantly increased intelligence test scores of children over and above that of their siblings whose life-history had been characterised by chronic malnutrition with or without an acute episode of nutritional disease (PCM). The physical data on these children provide evidence of the fact that the nutritional status of the fed children was significantly superior to that of their siblings for at least the first 3 years of life (Tables 48 and 55). No significant difference in expected weight or height for age occurred between the child who succumbed to acute PCM when compared with his chronically malnourished siblings (Figures 6 and 7).

A more detailed discussion of the intelligence test scores is facilitated by separate consideration of each of the two experimental groups compared with its own control group. Mean intelligence scores for the fed group and their control group are

shown graphically in Figure 8. The difference between the full-scale intelligence score means was significantly high in favour of the fed group. The difference between the means on verbal score did not quite reach statistical significance, but the trend shown suggests that this difference was nearly so. However the mean difference in non-verbal score between these two groups was not significantly different (Table 18). This suggests that mal-nutrition early in infancy has its strongest effect on verbal rather than non-verbal skills. This finding is in accordance with the consistent results obtained by several other studies since the observation was first made by Waterlow and co-workers in 1960 that, on the Gesell Scale malnourished children showed a relatively greater deficit on the language sub-scale than on the other sub-scales when compared with normal children (Waterlow, Cravioto and Stephen, 1960). Both fed and fed control groups received the same amount of environmental stimulation coincidental to the feed-up program, and the same degree of medical care. The non-significant difference between the two groups in non-verbal score can be attributed to this stimulation which was common to both groups. However the fed group was significantly superior to its control group in early nutritional status (Figures 6 and 7), and the elevation of verbal score in the fed group may thus be attributed to the nutritional advantage.

When the kwashiorkor group is compared with its sibling control group, no significant differences are noted between mean full-scale, verbal, or non-verbal scores (Tables 10, 14 and 18). This indicates that an episode of acute PCM had no particular detrimental effect

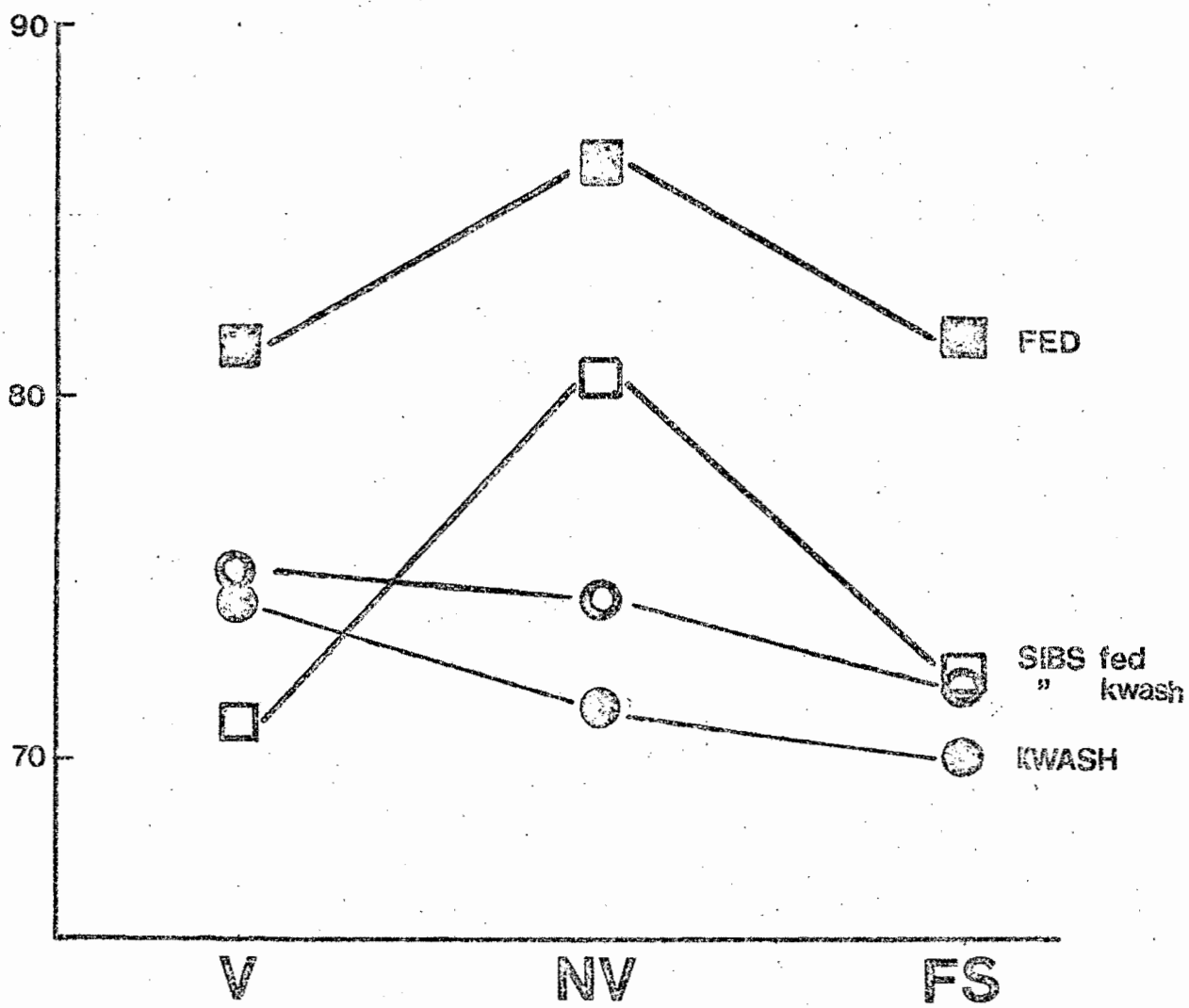

FIGURE 8 MEAN INTELLIGENCE SCORES

on the intellectual functioning of a group of chronically malnourished children. That the early nutritional status of these two groups of children was equivalent is demonstrated by the percentage of expected weight and height for age shown in Figures 6 and 7. It should be noted that the kwashiorkor group and its control group were comparable in height and weight for age with the sibling control group of the fed children at mid-term age level; the fed children differed significantly in weight and height from each of the other three groups until some time after termination of supplementary feeding.

When the four groups of children are compared with each other, Figure 8 illustrates that each pair of experimental and control groups show a distinct trend in the pattern of their intelligence scores. Because of the small size of the sample, these differences in trend must be considered as important differences which may show higher statistical significant in larger samples of the populations. Superiority of the fed group in mean full scale intelligence scores over each of the other three groups was statistically significant on Scheffé multiple comparisons (Table 10). This finding is in accordance with expectation, and can be attributed to the significant superiority of the fed group in early nutritional status when compared with the other three groups (Figures 6 and 7). The non-verbal scores show a similar trend, but show relatively larger differences between the fed control group compared with the kwashiorkor and kwashiorkor control groups.

The comparison of the means for the four groups on non-verbal intelligence score (Table 18) indicates that the fed group was significantly higher than both the kwashiorkor group and the kwashiorkor control group. The fed control group however, was also significantly superior to the kwashiorkor group. This finding suggests that whilst nutritional factors were important, a significant contribution to the elevation of the non-verbal score was made by the general social improvement which coincided with the dietary supplementation, together with improved medical care. It seems plausible that both the fed group and its control group were less frequently immobilised on account of illness and infection than were the kwashiorkor and its control group. This finding would lend some support to the suggestion that loss of 'learning time' on account of chronic ill-health makes a significant contribution to the functional retardation of the chronically malnourished child. It should be noted that the mean weight of the fed control group increased significantly during the feed-up period (Figure 7), although at mid-term it remained significantly below that of the fed group. The findings suggest that whilst increased environmental stimulation and improved social conditions brings about a significant increase in non-verbal skills, early nutrition appears to provide an added beneficial effect. Pg. 191

A deviation in the trend of patterning of intelligence scores of the four groups on the verbal score was contrary to expectation. In verbal intelligence score the means for the kwashiorkor group and its control group took up an intermediate position between the fed group and its control group (Figure 8). The problem posed was why

the trend for full-scale and non-verbal scales in which the kwashiorkor and kwashiorkor control groups had scored below the fed group and its control group, was not consistent also on the verbal scale. Both in terms of the underlying postulates being examined, and in terms of the results on full-scale and non-verbal scale, the fed control group should be expected to score at a higher rather than lower level when compared with the kwashiorkor and kwashiorkor control groups. The possibility that the high verbal score in the fed group is a reflection of an improved mother/child relationship from birth was considered, but the fact that verbal score did not correlate significantly with age of onset of stimulation for the combined groups (Table 71), points against this explanation. In the total sample height and weight at mid-term and the arbitary food index showed significantly high correlations with both full-scale and non-verbal scores, but not with verbal score (Table 66). This suggested that the verbal scores were influenced by some other factor. A significantly high positive correlation occurring in the combined fed and fed control groups between verbal score and the elapsed time between termination of the supplementation program and testing suggested that a time factor may be involved. In terms of this it could be proposed that the verbal score of the kwashiorkor group and its control group had been boosted above that of the fed control group by a relatively longer period of school experience.

Support for the argument that the mean verbal scores in the kwashiorkor and kwashiorkor control groups were boosted by a longer duration of school experience may be seen in their significantly

higher mean age at the time of testing (Table 5). The majority of these children also had progressed beyond the sub-standards in school at the time of testing, by which time also they had about 6 years of attendance at school. On the other hand the majority of children in the fed group and its control group were at sub-standard school level at the time of testing, and had only 2 years of scholastic experience at the time of testing. It should be stressed that the severity of deprivation by Western standards in this population is such that even minimal school attendance represents major cognitive stimulation. The power of school attendance is thus relatively far greater in this sample as environmental stimulation than it is for the average middle class child. The ability of environmental factors to compensate in even severe and acute malnutrition is suggested in the results of our previous study (Evans et al., 1971). At the ten-year follow-up, children with an earlier onset of kwashiorkor and their sibling controls showed significantly higher verbal and full-scale intelligence scores when compared with late-onset kwashiorkor subjects and their siblings. (The differences held for both the subjects and their sibling controls). In the present study the kwashiorkor group was a predominantly 'late-onset' sample: the early-onset sample in the previous study achieved a verbal level even higher than that of the present fed group. It seems apparent that the intervention of active rehabilitation of even rudimentary proportions may promote an appreciable compensatory effect. The implication appears to be that early adversity of short duration was more successfully overcome than prolonged (later) adversity. The present results suggest that

the verbal deficit in the kwashiorkor group and its control group has at least partially been compensated for by 6 years of schooling. If this 'catch-up' phenomenon is operating, it is anticipated that the fed control group (which at present ranks lowest on verbal score) will progress to at least the level of the kwashiorkor and kwashiorkor control groups. On this basis it also seems reasonable to assume that the difference between the fed group and the fed control group might have been greater at an earlier age than at present. Alternatively, the fed group may maintain its advantage, and these proposals will need to be re-tested in about 3 years time.

A review by Clarke (1958) and the observations of Kagan (1972) have demonstrated that the effects of stimulus deprivation and restriction in infancy are by no means permanent or immutable. The effects of early experience, positive or negative, will fade unless they are reinforced. In a recent preliminary report of an on-going study of rehabilitation of malnourished children in Beirut, a relatively stimulating environment increased the DQ of children compared with those rehabilitated in a conventional hospital environment (Yaktin and McLaren, 1970; 1971). Of all the sub-scales measured the least improvement occurred in speech and language functions. The gains however were not maintained after the children returned to their usual home environment. These findings are particularly relevant to the present study because of the reversal of the independent variable: in the Beirut study both groups received equivalent nutritional rehabilitation but only the experimental was exposed to additional environmental stimulation. In contrast the fed and fed control groups in the present study received equivalent environmental stimulation but only the experimental group

received additional nutritional supplementation. The Beirut results confirm our findings in that in both studies environmental stimulation elevated the non-verbal scores. The relative lack of speech and language in the Beirut study contrasts sharply with the mark verbal elevation in the present study. If we discount the nutritional factor, the circumstantial evidence suggests that early stimulation and medical care have a beneficial effect on non-verbal functions, whereas later stimulation has a more selectively positive effect on verbal functions.

In contradiction it could be argued that the fed group's increment in verbal score is due to a closer mother/child interaction at an earlier age. Be this as it may , it is also reasonably likely that this improved relationship was determined at least in part by the child. As Rutter and co-authors (1964) point out, environmental influences are to some extent shaped and modified by the child. The mothers frequently commented during the feeding program on the differences in alertness and brightness between the fed infant and their other children. Even allowing for substantial Hawthorne effect and the possibility of the mother feeding back the appropriate expectations to the investigator it seems reasonable to postulate that the fed child was less disabled by apathy, and therefore more able to contribute to the mother/child feed-back cycle. Even if theobserved improvement in verbal scores is accounted for on a basis of stimulation or secondary interpersonal gains, this was still partly dependent on nutritional improvement, and the benefits can therefore be attributed to nutritional factors indirectly, if not directly.

As an alternative it could be suggested that the non-verbal and, more especially, the verbal scores of the fed control group have undergone a more rapid 'fading' process than in the fed group. Again, however, it would seem to be a reasonable assumption that better nutrition in the fed group promoted a stabilising effect. Whatever the interpretation, it seems clear that the children of the fed group gained an appreciable 'head-start' over their under-nourished siblings.

Significantly high positive correlations in a matrix combining the fed group and the sibling control group (Table 71) between an arbitrary food index assigned to each group and the score attained by each child on the Vocabulary and Pattern Completion sub-tests, and almost significant Comprehension and Verbal Reasoning sub-tests, suggest that conceptual reasoning and learning (Level II) as opposed to associative learning (Level I) may indeed be related to nutritional factors. This would suggest that nutrition plays a role in Jensen's finding that these two levels of functioning are related to socio-economic status rather than to genetic factors. That nutritional factors may be important in determining functional Levels I and II has important relevance to Jensen's belief that different neural structures underly these two different functional levels. Perhaps the full significance of nutritional factors has been overlooked by Jensen.

In spite of the qualitative functional differences described in terms of these two levels, in the present study no significant inter-group differences were found in degree of sub-test scatter, to be the most reliable index of 'brain damage' (Table 28).

The absence of a significantly high sub-test scatter (as indicated by standard deviation of sub-test scores) in any of the four groups, together with the findings that the four groups did not differ significantly on the Bender Gestalt Test indicates that the observed intelligence score differences does not support 'brain damage' as the underlying factor in these differences. However a high standard deviation on Bender Gestalt test scores in the kwashiorkor group must be noted (Table 36), suggesting a higher variability in perceptual motor functioning within this group when compared with the other three groups, in spite of the fact that this group had a significantly higher mean age.

The application of the traditional verbal/non-verbal dichotomy to the sample of children used in this study is probably of limited validity and reliability. A number of the sub-tests included in the NSAIS are unsuitable in concent for this socio-cultural population, and their use was therefore inappropriate. It was decided however not to omit the more culture-bound tests. The application of these tests to this group was justified, firstly, in order to allow for comparison of the results with those obtained by Stoch and Smythe (1968) on a different sample of undernourished and control children and secondly, so as not to interfere with the internal structure of the Scale. The mean differences between the groups on each sub-test (Table 27) indicate that the more dependent a sub-test is on socio-cultural or scholastic experience, the less it discriminates between the four groups. Thus equivalent scores with no significant inter-group differences were found on Arithmetic, Memory, Absurdities, and Form Board sub-tests.

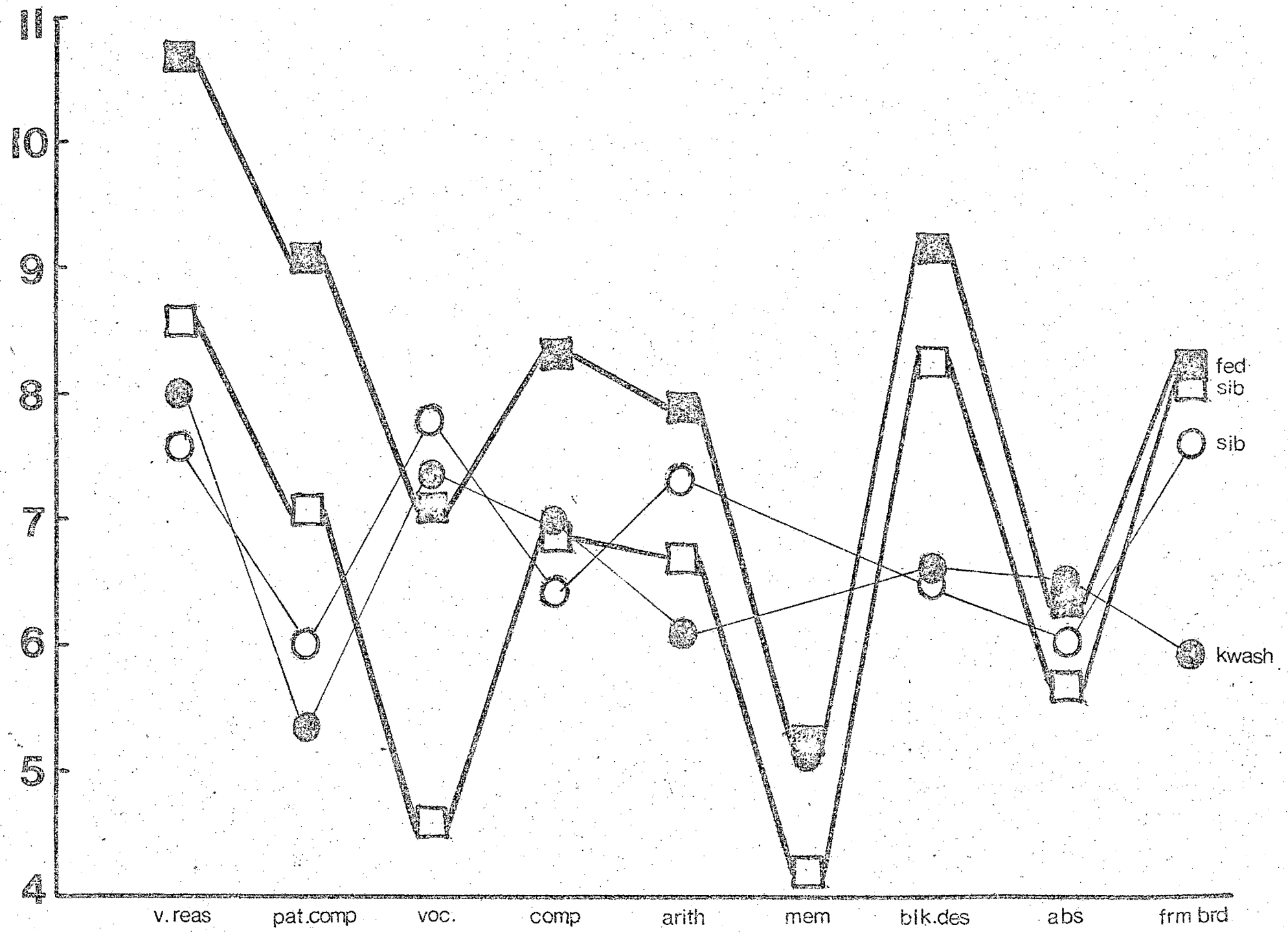
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FIGURE 9

Jensen (1969) observed differences in learning and problem solving ability in children related to socio-economic status. On tests involving associative learning ability (Level I), children from low and middle- socio-economic groups perform equally well, and performance is independent of IQ. However in tasks involving conceptual learning and problem-solving ability (Level II), large social class differences show up: the children of middle socio-economic group show a significantly higher level of performance. Bearing this in mind it is striking that the fed group in this study showed significantly higher scores on all of the sub-tests involving conceptual learning or problem-solving ability when compared with its control group, viz. Vocabulary, Verbal Reasoning and Pattern Completion. Figure 9 presents the means for the four groups on the nine NSAIS sub-tests when placed in rank order according to the degree of abstraction required. The tendency is also present in the Comprehension sub-test which is less abstract and more concrete than the Verbal Reasoning and Pattern Completion sub-tests. The Pattern Completion sub-test was the only non-verbal sub-test which discriminated between fed and control groups: it is the only non-verbal sub-test in which the child is required to contribute something which he is not given. Availability of cues is reduced and it therefore differs considerably in the degree of abstraction required from the Block Design sub-test in which all the elements required for the correct solution are provided in concrete form, and have only to be manipulated correctly. The same applies to the Form Board sub-test which, in addition includes a temperament factor on an inhibition-impulsiveness continuum. On this sub-test

the fed group and both the sibling control groups performed at a comparable level so that the kwashiorkor group's performance was significantly lower. The kwashiorkor control group had an intermediate position, and the trend in this sub-test parallels that found for the total non-verbal scores (Figures 8 and 9). A study by Pedersen and Wender (quoted by Kagan, 1970) demonstrated that a child's performance on the Block Design, Picture Completion, and Maze sub-tests of the WISC was related to his earlier social behaviour on four clusters:- physical contact with adults, seeking attention from adults, oral behaviour, and sustained involvement in a play sequence with objects. Children who sought physical contact and attention did poorly on the performance tests, while the children who displayed sustained involvement with the toys did well on the performance tests. Children who sought physical contact were more likely to use relational (i.e. Level I, associative) concepts of a sorting test, whereas the subjects who became more involved in play showed a preferred use of categorical (i.e. Level II, conceptual) concepts. In the present study the Block Design sub-test also reflects a pattern of the means for the total non-verbal scores; the fed group differed significantly from both the kwashiorkor group and the kwashiorkor control group, and the two control groups showed an almost significant difference (Table 27).

A report on a study of malnutrition and reasoning ability in Zambian school-children has just come to hand (Fisher et al., 1972). The authors compared the performance of 74 children who had been malnourished during the first 2 years of life with 143 control children on two non-verbal tests of general reasoning ability.

Although the authors do not provide any statistical analysis, their graphs show clear-cut differences between the two groups on both tests. It appears from the graphs that the differences only become apparent after the age of 12 - 13 years, a finding which probably can be explained by the ontogenetic developmental sequence of reasoning ability from concrete (Level I) to abstract (Level II). The results of this study on Zambian children together with the results of the present study on Cape Coloured children are in accordance with Witkop's (1967) expectation that the type of intellectual retardation found in malnourished children may have specific identifiable features similar to the mental retardation resulting from phenylketonuria and histidinemia, and suggests a promising avenue for new research as well as re-analysis of previous findings.

The analysis of the sub-test scores in the present study in terms of abstract versus associative functions helps to interprete the apparent inconsistencies in the sub-scale scores when it is recognised that abstract-conceptual functions contribute far more strongly to the so-called 'verbal' score than to the 'non-verbal' score.

The lack of significant differences between the four groups on mean Harris drawing score suggests that the differences between the four groups in intellectual functioning is a quantitative rather than a qualitative one. Both correlation drawing scores occurred between the fed control group and the other three groups, and this finding combined with the finding that in the fed control group the Harris score did not correlate at a significantly high level with

verbal and non-verbal intelligence scores suggests a possibility that this group could show a greater degree of emotional disturbance than the other three groups. However this postulate is not borne out by the ratings of Machover conflict indicators, the means of which showed no significant differences for the four groups. These findings are contrary to those found in the previous study in which a significantly lower Harris score in the kwashiorkor group was associated with a greater degree of emotional interference in cognitive functioning in the kwashiorkor group compared with its sibling control group.

The findings on the Bender Gestalt and Harris Drawing Tests are in agreement with Dobbing's (1972, 1972a) assertion that the effects of malnutrition during the vulnerable period of brain growth are qualitatively different from the results of tissue destruction or of the focal brain lesions familiar to the paediatric neurologist.

CONCLUSION

The results of this study have extended from the results of the previous study on the effect of malnutrition on intellectual development in the Cape Coloured population. By combining these studies greater clarity of interpretation of the findings of both has been achieved. The results can also be meaningfully related to work in other parts of the world dealing with the development of intelligence and specifically with the influence of nutritional and other environmental variables on intellectual development.

The results indicate that malnutrition has a significant detrimental effect on intellectual development. There appears to be a specific effect on the emergence of higher order, abstract-conceptual learning and reasoning processes in contrast to lower order, associative processes. It is possible that functional alterations in biochemical or enzymatic processes may provide the mediating link between the nutritional deprivation and the cognitive deficit. Although the effect of early environmental stimulation on intellectual development cannot be excluded in this study, the results indicate that nutrition per se has a significant effect on intellectual development. Medical supervision and social supervision however are also important factors, the effect of which was amplified by improved nutrition. It seems reasonable to conclude that adequate nutrition during the first two years of life enabled the children in this study to fulfil to a greater degree the functional intellectual developmental level set by their genetic endowment. Although the adverse effects of early nutritional deprivation tended to be at least partially compensated for in time, and school experience promoted some 'catch-up' in intellectual development,

the chronically malnourished children remained inferior to their fed siblings at the age of testing; this, in spite of the fact that subsequent poor nutrition has caused the fed children to lose their substantial early gain in physical development. The intellectual 'head-start' given them by adequate early nutrition has been maintained, even though they are at present no different physically from their siblings.

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APPENDIX I

RAW DATA

TABLE 56 RAW DATA FOR AGE AND SOCIAL VARIABLES

Fam. No.	MSI	No. in sibship	Position in sibship				Present Age (yrs)				Age Tested NSAIS (yrs)				Birth Wght (Lbs.oz)	Durat. Breast Fed (mths)	Age Start (mths)		Age Stop (yrs)		Durat. Fed (yrs)	Age Adm. Hop. (yrs)
			K	SK	SF	F	K	SK	SF	F	K	SK	SF	F			F	SF	F	SF		
1	31.5	13	2	3	6	8	15.6	14.3	13.1	9.6	15.6	14.3	13.1	9.6	6-11	5	0	3.5	2.4	5.9	2.4	1.6
2	30.0	17	8	7	13	14	16.0	16.75	11.3	10.0	11.8	12.5	11.3	10.0	7-11	10	10	1.3	3.5	5.6	3.5	1.5
3	47.0	18	11	10	17	18	17.2	18.3	9.6	8.4	12.3	13.4	9.6	8.4	7-8	1	0	1.2	2.0	3.2	2.0	2.75
4	29.0	16	6	5	11	12	18.7	19.5	10.0	8.25	13.8	14.3	10.0	8.25	7-0	2	0	1.75	1.9	3.65	1.9	4.0
5	33.5	12	4	5	7	8	15.75	14.75	9.8	8.5	11.0	10.0	9.8	8.5	7-12	2	0	1.3	2.1	3.4	2.1	2.75
6	42.5	8	1	2	5	6	15.75	14.2	10.7	9.75	15.75	14.2	10.7	9.75	7-8	6	6	1.75	3.0	4.75	3.0	2.25
7	22.5	7	4	5	6	7	14.2	12.9	10.75	8.7	14.2	12.9	10.75	8.7	6-8	2	0	2.05	2.2	4.25	2.2	2.2
8	41.5	7	2	1	4	5	15.7	17.0	11.0	9.0	13.4	11.5	11.0	9.0	7-0	6	0	2.0	2.4	4.4	2.4	1.3
9	66.0	6	2	3	5	6	16.5	14.3	10.4	8.9	11.9	9.6	10.4	8.9	5-13	3	0	1.5	2.5	4.0	2.5	2.25
10	58.0	11	5	6	9	10	15.7	13.5	10.6	8.6	10.9	5.9	10.6	8.6	6-0	3	0	2.0	2.25	2.25	2.25	2.5
11	45.5	15	9	10	12	13	15.3	13.7	10.1	8.25	15.3	13.7	10.1	8.25	9-13	0	0	1.85	1.75	3.6	1.75	1.2
12	21.5	15	10	11	14	15	15.4	12.8	9.9	8.9	10.3	8.75	9.9	8.9	6-12	0	0	1.0	2.5	3.5	2.5	0.8
13	66.0	13	5	-	10	11	15.4	-	12.4	8.6	15.4	-	12.4	8.6	5-0	3	0	3.8	2.25	6.05	2.25	1.75
14	55.5	11	3	2	-	7	14.75	17.1	-	9.2	11.3	12.75	-	9.2	7-0	4	4	-	2.9	-	2.9	2.4

TABLE 57

INDEX OF SIBSHIP

Fam. No.	KWASH	SIB	SIB	FED
1	-9	-7	-1	+3
2	-1	-3	+9	+11
3	+4	+2	+16	+18
4	-4	-6	+6	+8
5	-4	-2	+2	+4
6	-6	-4	+2	+4
7	+1	+3	+5	+7
8	-3	-5	+1	+3
9	-2	0	+4	+6
10	-1	+1	+7	+9
11	+3	+5	+9	+11
12	+5	+7	+13	+15
13	-3	-	-1	+9
14	-5	-7	-	+3

TABLE 58

INCOME

(% Poverty Datum Line)

Fam. No.	1959	1964	1969
1	0	65	65
2	49	26	54
3	51	75	106
4	40	35	110
5	32	38	48
6	101	0	55
7	53	66	53
8	144	52	57
9	110	92	92
10	47	54	139
11	82	69	79
12	33	49	71
13	66	112	233
14	98	83	57
	64.7	58.3	88.5

TABLE 59

PHYSICAL DATA

FAM. NO.		EARLY			MID-TERM			PRESENT		
		AGE MOS	WHT % EN	HT % EN	AGE MOS	WHT % EN	HT % EN	AGE MOS	WHT % EN	HT % EN
1	K				27	69.8	82.8	187	85.1	92.0
	S				27	69.8	77.9	172	63.2	83.7
	S	37	73.5	82.8	37	73.5	82.8	156	81.8	90.5
	F	26	92.1	84.4	26	92.1	84.3	115	66.6	81.8
2	K				33	94.6	82.6	192	93.6	92.0
	S				32	93.7	79.2	201	90.5	95.3
	S	15	76.3	88.7	31	77.8	86.4	136	88.2	95.7
	F	10	98.9	95.0	31	101.9	94.6	120	87.4	93.1
3	K				55	67.0	80.4	206	78.3	90.3
	S				55	71.6	80.5	162	80.0	92.2
	S	23	73.0	87.0	50	80.4	89.3	115	76.5	99.6
	F	23	76.5	89.2	59	79.5	89.7	101	71.7	88.7
4	K				66	46.0	68.9	224	59.0	83.0
	S				66	50.0	75.5	172	44.1	77.1
	S	29	68.5	79.9	57	70.4	81.0	120	61.3	82.3
	F	22	71.1	86.0	60	65.0	79.1	99	62.6	82.3
5	K				39	86.7	82.8	189	87.7	96.5
	S				38	65.2	79.5	177	76.9	94.9
	S	19	57.9	79.2	40	68.1	83.6	118	73.1	93.0
	F	24	96.2	94.2	39	93.0	95.3	102	78.9	94.8
6	K				38	73.4	87.5	169	66.9	88.8
	S				44	62.7	80.2	170	65.7	88.2
	S	21	68.4	90.3	33	84.8	86.8	128	74.9	91.1
	F	12	85.0	98.9	36	93.0	95.2	117	89.2	96.3
7	K				43	90.7	85.3	170	81.4	92.9
	S				46	73.0	89.0	155	79.7	95.0
	S	24	65.4	85.5	37	72.0	83.3	129	81.8	89.7
	F	25	83.2	90.5	42	84.2	91.1	104	73.8	90.0
8	K				44	95.7	93.0	188	81.1	94.3
	S				44	75.0	83.1	211	75.9	88.4
	S	25	80.5	90.5	48	81.8	83.6	132	81.4	91.2
	F	29	94.8	91.3	46	89.6	91.4	108	90.7	93.2
9	K				56	63.5	84.6	198	73.8	90.7
	S				58	71.5	83.2	172	88.3	92.5
	S	27	70.4	83.8	55	71.6	83.4	125	71.2	87.0
	F	29	82.9	92.8	44	83.7	91.2	107	84.5	94.2
10	K				34	77.1	83.3	188	61.6	82.4
	S				35	78.7	88.0	162	82.3	91.7
	S	16	82.6	89.4	34	83.3	89.2	127	93.8	93.7
	F	27	78.2	91.0	35	78.1	87.9	103	76.2	89.8
11	K				23	76.2	83.9	184	80.1	93.3
	S				23	79.0	85.1	162	80.4	98.2
	S	16	74.4	85.9	23	81.1	80.5	121	81.4	86.1
	F	20	91.8	90.5	20	91.8	90.5	99	93.3	91.6
12	K				41	63.4	74.5	185	69.4	87.7
	S				39	69.5	81.8	154	76.9	88.9
	S	13	75.0	88.3	42	79.7	86.4	119	73.2	86.2
	F	15	87.9	89.4	43	83.1	87.4	107	66.8	82.0
13	K				34	78.6	81.3	185	74.4	89.2
	S				-	-	-	-	-	-
	S	31	82.4	89.2	31	82.4	89.2	148	86.6	94.6
	F	27	114.3	92.9	27	114.3	92.9	102	81.7	87.0
14	K	41			41	62.1	75.8	177	77.3	86.3
	S				41	97.8	98.4	153	94.0	98.5
	S	-	-	-	-	-	-	-	-	-
	F				42	95.8	88.5	110	77.9	86.6

TABLE 60

INTELLIGENCE SCORES

(NSAIS - IQ)

Fam. No.	VERBAL IQ				NON-VERBAL IQ				FULL SCALE IQ			
	K	SK	SF	F	K	SK	SF	F	K	SK	SF	F
1	58	61	71	93	55	69	81	92	52	61	73	92
2	75	76	65	81	72	88	78	85	71	80	68	81
3	89	72	71	91	65	71	83	88	76	68	74	88
4	64	54	61	54	71	69	58	71	63	56	55	57
5	72	74	51	68	76	65	80	71	71	67	60	66
6	52	82	57	86	67	76	81	94	54	77	64	88
7	75	75	67	85	64	76	80	88	67	73	69	85
8	74	82	91	95	76	78	85	95	72	78	87	95
9	91	82	72	82	80	80	87	95	84	79	76	87
10	110	116	102	99	90	90	106	102	102	105	104	101
11	60	47	67	60	69	57	69	72	60	46	64	61
12	71	79	74	82	71	74	78	83	68	74	73	81
13	62	-	69	86	74	-	72	97	64	-	68	90
14	69	57	-	76	76	60	-	81	69	54	-	76

TABLE 61

NSAIS SUB-TESTS

		VOCAB.	COMPR.	VERB. REAS.	ARITH. PROB.	MEMORY	PATT. COMPL.	BLOCKS	ABSURD.	FORM BRD.	STD. DEV.
Fed	1	10	9	12	9	7	11	8	7	10	1.7
	2	7	8	10	7	6	6	10	9	7	1.6
	3	8	8	13	8	8	8	11	5	10	2.3
	4	2	5	6	4	2	5	6	4	9	2.2
	5	4	8	10	6	1	7	6	6	5	2.5
	6	9	12	11	7	3	10	9	9	9	2.6
	7	7	7	11	9	7	10	9	8	7	1.5
	8	11	11	10	9	7	9	11	8	9	1.4
	9	9	10	12	11	7	12	15	4	7	3.3
	10	10	9	13	12	7	11	10	8	13	2.1
	11	4	5	9	4	1	9	6	4	6	2.5
	12	4	8	11	9	7	11	9	4	7	2.6
	13	9	9	11	8	5	10	13	9	7	2.3
	14	2	9	7	10	7	6	8	5	11	2.7
Sib.F	1	7	8	8	5	3	7	7	8	8	1.7
	2	3	5	10	6	3	6	8	9	5	2.5
	3	4	5	8	9	5	8	7	4	12	2.7
	4	4	6	6	5	3	6	4	3	4	1.2
	5	2	7	2	3	2	7	10	4	8	2.9
	6	3	4	7	4	3	4	9	5	12	3.1
	7	4	7	9	4	4	8	10	5	6	2.3
	8	8	11	11	8	7	9	7	5	11	2.1
	9	4	7	10	7	4	9	10	5	9	2.4
	10	9	10	12	14	8	10	11	10	13	1.9
	11	3	6	9	7	4	4	8	4	7	2.2
	12	3	6	11	8	5	7	8	6	7	2.2
	13	7	4	6	2	2	6	5	3	4	1.8
	14	-	-	-	-	-	-	-	-	-	-

TABLE 61
continued

NSAIS SUB-TESTS

	VOCAB.	COMPR.	VERB. REAS.	ARITH. PROB.	MEMORY	PATT. COMPL.	BLOCKS	ABSURD.	FORM BRD.	STD. DEV.
Kwash 1	6	4	7	4	1	4	4	4	3	1.7
2	7	7	10	4	6	4	6	10	5	2.2
3	8	7	9	9	11	6	6	5	4	2.2
4	9	7	5	3	3	5	7	6	6	1.9
5	6	9	8	5	4	5	9	7	6	1.8
6	5	5	4	4	0	3	5	6	8	2.2
7	8	7	7	9	3	5	4	5	6	1.9
8	8	7	8	5	5	8	6	7	6	1.2
9	9	9	10	9	9	7	9	7	6	1.3
10	12	10	13	13	11	7	14	6	8	2.9
11	3	5	6	4	5	4	5	8	6	1.5
12	8	6	9	4	4	6	4	7	7	1.8
13	3	6	8	8	0	6	5	8	7	2.7
14	9	6	7	4	4	6	7	7	7	1.6
Sib K 1	4	7	7	5	1	4	5	6	8	2.1
2	7	7	9	6	6	6	9	13	6	2.3
3	7	4	8	10	3	4	17	5	8	2.3
4	5	6	2	2	4	3	7	4	9	2.3
5	13	4	6	7	3	1	7	6	7	3.4
6	7	6	8	13	5	7	6	4	10	2.7
7	7	8	9	8	2	6	6	7	8	2.0
8	8	9	10	5	7	7	6	8	7	1.5
9	9	7	8	9	6	8	7	6	8	1.1
10	13	11	13	15	11	11	9	5	10	2.8
11	4	2	5	1	2	5	5	3	3	1.5
12	10	6	7	7	7	10	4	5	7	2.0
13	-	-	-	-	-	-	-	-	-	-
14	5	8	8	5	4	8	7	7	3	1.9

TABLE 62

HARRIS DRAWING TEST

Man Scale	K	SK	SF	F	Women Scale	K	SK	SF	F	Total Score	K	SK	SF	F
1	70	75	75	80	1	82	73	70	76	1	76	74	73	78
2	63	97	87	99	2	65	96	90	97	2	64	97	89	98
3	76	79	82	81	3	77	78	84	76	3	77	79	85	79
4	64	93	50	63	4	62	96	49	57	4	63	95	50	60
5	81	62	73	65	5	85	62	65	69	5	83	62	69	67
6	60	68	67	100	6	67	66	70	101	6	64	67	69	101
7	58	77	58	63	7	68	80	69	86	7	63	79	64	75
8	117	104	72	100	8	114	105	87	89	8	116	105	80	95
9	83	96	74	72	9	83	90	72	82	9	83	93	73	77
10	105	102	84	95	10	94	102	85	96	10	100	102	85	96
11	81	57	81	69	11	85	51	80	70	11	83	54	81	70
12	100	98	86	75	12	107	97	84	76	12	104	98	85	76
13	74	-	70	82	13	93	-	72	87	13	84	-	71	85
14	67	67	-	78	14	81	62	-	84	14	74	65	-	81

TABLE 63

HARRIS DRAWING.

TOTAL SCORES

	1968		1973	
	K	S	K	S
1	-	-	-	-
2	73	78	64	97
3	82	82	77	79
4	66	78	63	95
5	80	78	83	62
6	-	-	-	-
7	-	-	-	-
8	76	91	117	104
9	85	93	83	93
10	108	106	100	102
11	-	-	-	-
12	82	78	104	98
13	-	-	-	-
14	72	71	74	65

TABLE 64

MACHOVER RATING

ON

DRAW-A-PERSON

CONFLICT INDICATORS

	K	S	S	F
1	0	2	0	0
2	1	1	0	0
3	1	0	3	1
4	1	1	1	1
5	1	1	0	1
6	1	2	1	1
7	0	0	2	2
8	0	0	1	2
9	3	2	0	1
10	0	0	0	0
11	1	0	2	1
12	0	0	0	0
13	1	-	1	1
14	0	0	-	0

TABLE 65

BENDER GESTALT TEST

RAW SCORES

NO.	KWASH S	SIB S	SIB S	FED S
1	6	8	5	7
2	11	2	4	9
3	1	6	8	12
4	6	0	13	16
5	1	7	14	20
6	7	2	11	9
7	4	6	9	9
8	0	0	1	1
9	0	1	8	5
10	0	2	1	11
11	1	6	8	18
12	4	2	3	3
13	1	4	-	5
14	1	0	-	13

APPENDIX II

CORRELATION MATRICES

TABLE 66: CORRELATION MATRIX

ALL GROUPS COMBINED

(df 52)

	SIBSHIP INDEX	F.S. IQ	VERBAL IQ	NON-VERBAL IQ	BENDER GESTALT	HARRIS DRAWING	WEIGHT-MIDTERM	HEIGHT-MIDTERM	WEIGHT-PRESENT	HEIGHT-PRESENT	FOOD INDEX
SIBSHIP INDEX	-										
F.S. IQ	.29*	-									
VERBAL IQ	.22	.96***	-								
NON-VERBAL IQ	.37**	.88***	.72***	-							
BENDER GESTALT	-.20	.36**	.38**	.21	-						
HARRIS DRAWING	.04	.54**	.50**	.47**	.50**	-					
WEIGHT-MIDTERM	.30*	.32*	.25	.37*	.04	.18	-				
HEIGHT-MIDTERM	.38*	.33*	.27	.39**	-.00	.16	.77***	-			
WEIGHT-PRESENT	.09	.19	.16	.18	.05	.14	.61**	.52**	-		
HEIGHT-PRESENT	-.06	.00	-.00	.00	-.07	.04	.40**	.50**	.82***	-	
FOOD INDEX	.68***	.35**	.22	.55**	-.22	-.03	.46**	.51**	.08	-.12	-

* P < 0.05
** P < 0.01

TABLE 67: CORRELATION MATRIX

KWASHIORKOR GROUP

(df 12)

	F.S. IQ	VERBAL IQ	NON-VERBAL IQ	WEIGHT-MIDTERM	WEIGHT-PRESENT	HEIGHT-MIDTERM	HEIGHT-PRESENT	MULT. SOC. INDEX	HARRIS DRAWING	BENDER GESTALT
F.S. IQ	-									
VERBAL IQ	.96**	-								
NON-VERBAL IQ	.82**	.65*	-							
WEIGHT-MIDTERM	.09	.08	.06	-						
WEIGHT-PRESENT	-.23	-.16	-.35	.66**	-					
HEIGHT-MIDTERM	.09	.09	.05	.77**	.39	-				
HEIGHT-PRESENT	-.32	-.26	-.36	.66**	.83**	.60*	-			
MULT. SOC. INDEX	.42	.33	.54*	-.13	-.23	.20	-.23	-		
HARRIS DRAWING	.43	.36	.47	.20	-.11	.30	.05	.20	-	
BENDER GESTALT	.54*	.47	.57*	.11	.10	.27	.12	.71**	.55*	-

r = 0.46, P = 0.10
r = 0.53, P = 0.05*
r = 0.65, P = 0.01**

TABLE 68: CORRELATION MATRIX

KWASHIORKOR CONTROL GROUP

(df 11)

	F.S. IQ	VERBAL IQ	NON-VERBAL IQ	WEIGHT-MIDTERM	WEIGHT-PRESENT	HEIGHT-MIDTERM	HEIGHT-PRESENT	MULT. SOC. INDEX	HARRIS DRAWING	BENDER GESTALT
F.S. IQ	-									
VERBAL IQ	.98**	-								
NON-VERBAL IQ	.91**	.82**	-							
WEIGHT-MIDTERM	.07	.04	.08	-						
WEIGHT-PRESENT	.24	.25	.16	.85**	-					
HEIGHT-MIDTERM	.00	.07	-.16	.67**	.65*	-				
HEIGHT-PRESENT	.00	.04	-.12	.75	.89	.65*	-			
MULT. SOC. INDEX	.21	.27	.07	.31	.44	.43	.32	-		
HARRIS DRAWING	.65**	.57*	.77**	-.06	-.05	-.20	-.41	-.05	-	
BENDER GESTALT	.29	.26	.34	.10	.01	.21	-.25	.31	.59*	-

P = 0.10
P = 0.05*
P = 0.01**

TABLE 69: CORRELATION MATRIX

FED GROUP

(df 12)

	F.S. IQ	VERBAL IQ	NON-VERBAL IQ	WEIGHT-MIDTERM	WEIGHT-PRESENT	HEIGHT-MIDTERM	HEIGHT-PRESENT	MULT. SOC. INDEX	HARRIS DRAWING	BENDER GESTALT
F.S. IQ	-									
VERBAL IQ	.98**	-								
NON-VERBAL IQ	.96**	.90**	-							
WEIGHT-MIDTERM	.20	.19	.21	-						
WEIGHT-PRESENT	.0	.0	.16	.53*	-					
HEIGHT-MIDTERM	.23	.24	.21	.66**	.75**	-				
HEIGHT-PRESENT	.11	.09	.15	.26	.82**	.82**	-			
MULT. SOC. INDEX	.33	.23	.47	.29	.40	.21	.27	-		
HARRIS DRAWING	.73**	.71**	.73**	.40	.51	.50	.43	.26	-	
BENDER GESTALT	.32	.30	.30	.26	.05	-.02	-.23	.26	.17	-

r = 0.46, P = 0.10
r = 0.53, P = 0.05*
r = 0.65, P = 0.01**

TABLE 70: CORRELATION MATRIX

FED CONTROL GROUP

(df 11)

	F.S. IQ	VERBAL IQ	NON-VERBAL IQ	WEIGHT-MIDTERM	WEIGHT-PRESENT	HEIGHT-MIDTERM	HEIGHT-PRESENT	MULT. SOC. INDEX	HARRIS DRAWING	BENDER GESTALT
F.S. IQ	-									
VERBAL IQ	.95**	-								
NON-VERBAL IQ	.87**	.68**	-							
WEIGHT-MIDTERM	.44	.47	.29	-						
WEIGHT-PRESENT	.66**	.62*	.57*	.55*	-					
HEIGHT-MIDTERM	.45	.32	.50	.59*	.43	-				
HEIGHT-PRESENT	.34	.16	.50	.31	.75**	.60*	-			
MULT. SOC. INDEX	.39	.34	.36	.36	.27	.38	.24	-		
HARRIS DRAWING	.56*	.49	.54	.56*	.63*	.47	.44	.16	-	
BENDER GESTALT	.79**	.84**	.53	.53	.59*	.40	.27	.08	.61*	-

r = 0.48, P = 0.10
r = 0.55, P = 0.05*
r = 0.66, P = 0.01**

TABLE 71: CORRELATION COEFFICIENTS

FED GROUP AND FED CONTROL GROUP COMBINED

(df 24)

	F.S. IQ	VERB. IQ	NON-VERB. IQ	BENDER	HARRIS	AGE TEST	DUR. FED	INCOME	AGE START	AGE STOP	AGE (T-S)	FOOD IND.	(NV-V)	VOCAB.	COMPR.	VB. REASON	ARITH.	MEMORY	PATT. COMP.	BLOCK DES.	ABSURD.	FRM. BRD.
F.S. IQ	-																					
VERB. IQ	.97**	-																				
NON-VERB.IQ	.93**	.81**	-																			
BENDER	.53**	.54**	.43*	-																		
HARRIS	.68**	.64*	.66*	.37	-																	
AGE TEST	-.12	-.14	-.08	.00	-.01	-																
DUR. FED	.18	.12	.24	.19	.52	.36	-															
INCOME	.35	.31	.37	.10	.16	-.44*	-.31	-														
AGE START	-.28	-.28	-.23	-.12	-.18	.94**	.12	-.40*	-													
AGE STOP	-.30	-.32	-.24	-.13	.01	.90**	.53**	-.45*	.84**	-												
AGE(T-S)	.42*	.42*	.38	.30	-.07	.20	-.40*	.03	.21	-.24	-											
FOOD IND.	.36	.38	.29	.09	.26	-.76**	.03	.41	-.89**	-.68**	-.16	-										
(NV-V)	-.45*	-.65**	-.08	-.36	-.23	.13	.11	-.04	.18	.23	-.23	-.26	-									
VOCAB.				.27	.54**	-.09	.05	.35	-.23	-.26	.38	.40*	-.43*	-								
COMPR.				.51**	.52**	-.05	.17	.20	-.22	-.20	.34	.38	-.45*	.77**	-							
VB.REASON				.33	.54**	-.18	.48*	.33	-.30	-.27	.21	.38	-.60**	.65**	.48*	-						
ARITH.				.61**	.55**	-.21	.56**	.25	-.29	-.39*	.44*	.27	-.50**	.57**	.56**	.68**	-					
MEMORY				.62**	.44*	-.14	.54**	.18	-.27	-.27	.30	.27	-.54**	.59**	.54**	.61**	.82**	-				
PATT.COMP.				.34	.30	-.30	-.09	.40*	.60**	-.45*	.36	.42*	-.26	.70**	.62**	.63**	.60**	.54**	-			
BLOCK DES.				.32	.44*	-.16	.22	.41*	.46*	-.18	.06	.24	.17	.52**	.45*	.41*	.46*	.51**	.49*	-		
ABSURD.				.23	.68**	.20	.49*	.14	.38	.11	.22	.18	-.16	.59**	.44*	.47*	.38	.27	.26	.32	-	
FRM. BRD.				.23	.35	.04	-.03	.06	.50**	-.13	.36	-.05	-.02	.31	.26	.16	.50**	.48*	.17	.20	.15	-

r = 0.33, P = 0.10
r = 0.39, P = 0.05*
r = 0.50, P = 0.01**

TABLE 72: CORRELATION COEFFICIENTS

FED GROUP

(df 12)

	F.S. IQ	VERB. IQ	NON-VERB. IQ	BENDER	HARRIS	AGE TEST	DUR. FED	INCOME	AGE START	AGE STOP	AGE(T-S)	VOCAB.	COMPR.	VB. REASON	ARITH.	MEMORY	PATT. COMP.	BLOCK DES.	ABSURD.	FRM. BRD.
F.S. IQ	-																			
VERB. IQ	.98**	-																		
NON-VERB. IQ	.96**	.90**	-																	
BENDER	.32	.30	.30	-																
HARRIS	.73**	.71**	.73**	.17	-															
AGE TEST	.38	.38	.34	.12	.66**	-														
DUR. FED	.30	.30	.29	.22	.78**	.91**	-													
INCOME	.22	.13	.36	.10	.02	-.41	-.32	-												
AGE START	.05	.05	.07	-.30	.60*	.74**	.80**	-.27	-											
AGE STOP	.23	.22.	.22	.04	.68**	.89**	.98**	-.32	.91**	-										
AGE(T-S)	.18	.20	.12	.12	-.33	-.17	-.51	.04	-.66**	-.59*	-									
VOCAB.				.06	.66**	.32	.15	.23	.12	.15	.25	-								
COMPR.				.42	.74**	.58*	.54*	-.06	.23	.46	.04	.66**	-							
VB.REASON				-.0	.40	.11	.02	.19	-.00	-.00	.21	.76**	.44	-						
ARITH.				.50	.45	.24	.29	.10	-.19	.13	.14	.51	.58*	.56*	-					
MEMORY				.51	.39	.29	.31	.03	-.08	.18	.12	.47	.39	.50	.83**	-				
PATT.COMP.				.21	.24	-.00	-.09	.26	-.09	-.08	.17	.63	.42	.75**	.57*	.37	-			
BLOCK DES.				.42	.45	.15	.25	.43	.04	.19	-.14	.65*	.55*	.56*	.62*	.61*	.51	-		
ABSURD.				-.07	.75**	.51	.46	.15	.51	.50	-.17	.63*	.50	.32	.20	.15	.13	.24	-	
FRM. BRD.				.11	.39	.13	.10	.13	-.11	.03	.17	.27	.29	.14	.51	.48	-.03	.07	.13	-

r = 0.46, P = 0.10
r = 0.53, P = 0.05*
r = 0.66, P = 0.01**

TABLE 73: CORRELATION COEFFICIENTS

FED CONTROL GROUP

(df 10)

	F.S IQ	VERB. IQ	NON-VERB. IQ	BENDER	HARRIS	AGE TEST	DUR. FED	INCOME	AGE START	AGE STOP	AGE (T-S)	VOCAB.	COMPR.	VB. REASON	ARITH.	MEMORY	PATT.COMP.	BLOCK DES.	ABSURD.	FRM. BRD.
F.S. IQ	-																			
VERB. IQ	.95**	-																		
NON-VERB. IQ	.88**	.68*	-																	
BENDER	.79**	.85**	.55	-																
HARRIS	.56	.49	.53	.62*	-															
AGE TEST	.18	.16	.16	.09	.06	-														
DUR. FED	.0	-.0	.20	.17	.35	.40	-													
INCOME	.31	.35	.20	.01	.16	-.02	-.51	-												
AGE START	.14	.16	.07	-.02	-.08	.94**	.15	.04	-											
AGE STOP	-.32	-.33	-.27	-.24	-.05	.77**	.61*	-.24	.65*	-										
AGE (T-S)	.76**	.74**	.64*	.49	.15	.22	-.38	.33	.34	-.45	-									
VOCAB.				.63*	.17	.46	-.12	.22	.51	-.11	.82**	-								
COMPR.				.64*	.13	.29	-.24	.33	.33	-.19	.70**	.87**	-							
VB.REASON				.72**	.59*	.20	125	.31	.12	-.03	.33	.45	.36	-						
ARITH.				.74**	.60*	-.14	-.12	.32	-.11	-.57	.68*	.60*	.46	.70**	-					
MEMORY				.84**	.45	-.06	-.08	.25	-.08	-.47	.63*	.75**	.68*	.72**	.86**	-				
PATT.COMP.				.52*	.19	.03	-.13	.53	-.02	-.35	.58*	.66*	.72**	.40	.56	.72**	-			
BLOCK DES.				.12	.32	-.04	.18	.13	-.13	-.25	.31	.11	.17	.14	.19	.20	.32	-		
ABSURD.				.56	.57	.51	.54	-.08	.48	.20	.50	.50	.31	.54	.49	.38	.32	.37	-	
FRM. BRD.				.35	.38	-.00	-.00	-.05	-.09	-.33	.44	.50	.30	.22	.54	.60*	.37	.40	.18	-

r = 0.51, P = 0.10
r = 0.58, P = 0.05*
r = 0.69, P = 0.01**

TABLE 74: CORRELATION COEFFICIENTS

DIFFERENCES BETWEEN FED AND FED CONTROL GROUP

IN INTELLIGENCE SCORES, AGE AND INCOME

(df 10)

	F.S. IQ (FED-SIBF)	VERB. IQ (FED-SIBF)	NON-VERB. IQ (FED-SIBF)	AGE TEST. (FED-SIBF)	INCOME (FED-SIBF)	EARLY WEIGHT (FED-SIBF)	EARLY HEIGHT (FED-SIBF)	DURATION FED	AGE START	AGE STOP	AGE (T-S)
F.S IQ (FED-SIBF)	-										
VERB. IQ (FED-SIBF)	.92**	-									
NON-VER. IQ (FED-SIBF)	.53	.16	-								
AGE TEST. (FED-SIBF)	.25	.14	.33	-							
INCOME (FED-SIBF)	-.13	-.01	-.29	.22	-						
EARLY WEIGHT (FED-SIBF)	.28	.44	-.26	.05	-.05	-					
EARLY HEIGHT (FED-SIBF)	.08	.25	-.32	-.29	-.19	.65*	-				
DURATION FED	.55	.51	.28	.15	-.38	.22	.09	-			
AGE START	.25	.14	.34	1.0**	.23	.05	-.29	.15	-		
AGE STOP	.68*	.53	.60*	.65*	-.10	.45	.01	.61*	.65*	-	
AGE (T-S)	-.43	-.36	-.38	.34	.38	-.49	-.43	-.38	.34	-.45	-

r = 0.51, p=0.10
r = 0.58, p=0.05*
r = 0.69, p=0.01**

TABLE 75: CORRELATION MATRIX

FED GROUP

(df 11)

	EARLY HEIGHT	EARLY WEIGHT	VERBAL IQ	NON-VERBAL IQ	F.S. IQ
EARLY HEIGHT	-				
EARLY WEIGHT	.33	-			
VERBAL IQ	.10	.12	-		
NON-VERBAL IQ	.20	.12	.90**	-	
F.S. IQ	.14	.12	.98**	.96**	-

** P < 0.01

TABLE 76: CORRELATION MATRIX

FED CONTROL GROUP

(df 11)

	EARLY HEIGHT	EARLY WEIGHT	VERBAL IQ	NON-VERBAL IQ	F.S. IQ
EARLY HEIGHT	-				
EARLY WEIGHT	.50	-			
VERBAL IQ	.57*	.66**	-		
NON-VERBAL IQ	.51	.29	.70**	-	
F.S. IQ	.57*	.64**	.94**	.87**	-

* P < 0.05
** P < 0.01

APPENDIX III

MODIFICATION OF KOPPITZ SCORING

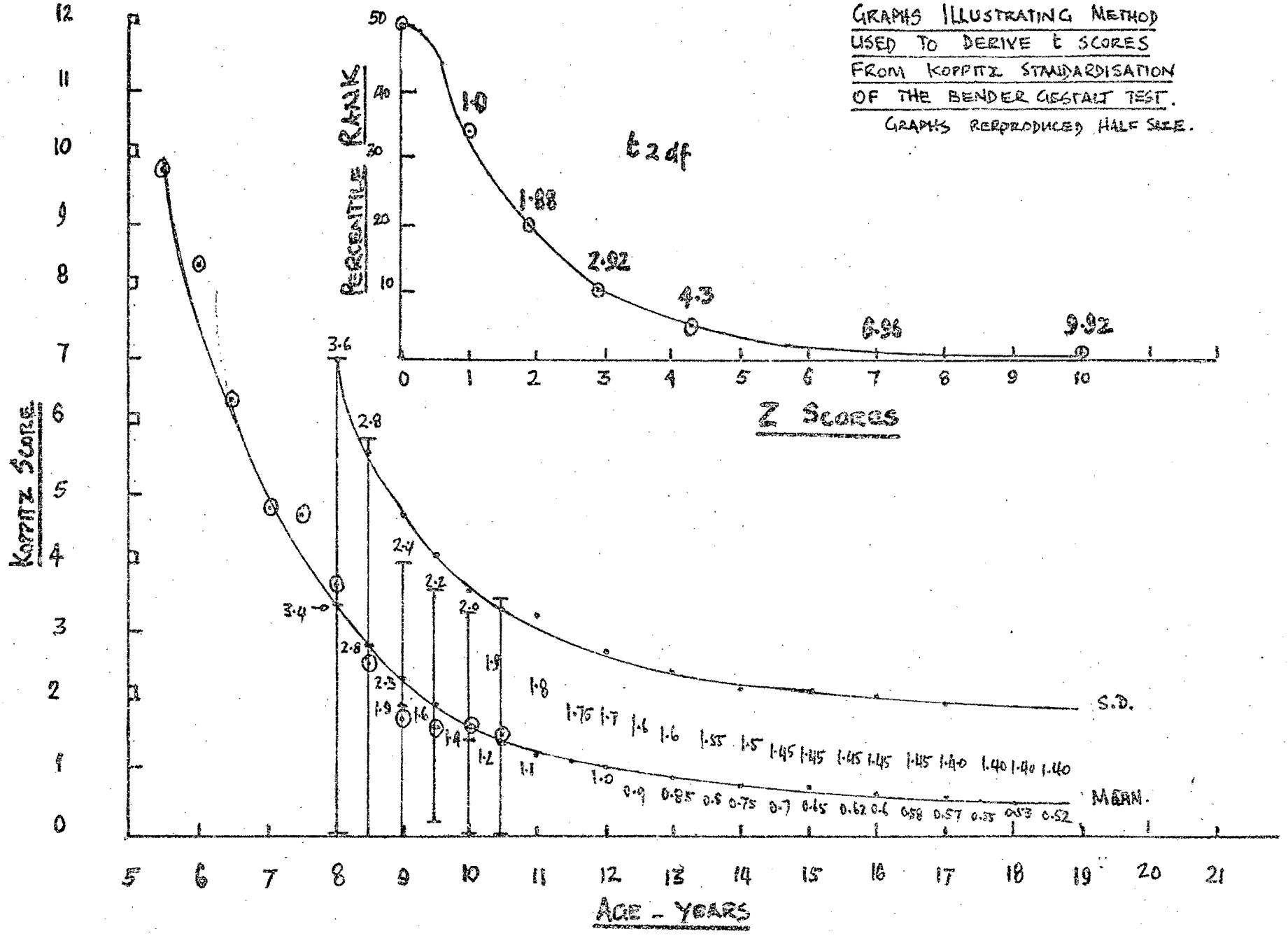
ON BENDER GESTALT TEST

TABLE X

EXTRAPOLATED MEANS AND STANDARD DEVIATIONS

For Koppitz Scores on Bender Gestalt Test.

MEAN	S.D.
3.4	3.6
2.8	2.8
2.3	2.4
1.9	2.2
1.6	2.0
1.4	1.9
1.2	1.8
1.1	1.75
1.0	1.7
0.9	1.6
0.85	1.6
0.8	1.55
0.75	1.5
0.7	1.45
0.65	1.45
0.62	1.45
0.6	1.45
0.58	1.45
0.57	1.4
0.55	1.4
0.53	1.4
0.52	1.4
0.51	1.4
0.50	1.4


GRAPHS ILLUSTRATING METHOD USED TO DERIVE t SCORES FROM KOPPITZ STANDARDISATION OF THE BENDER GESTALT TEST.
GRAPHS REPRODUCED HALF SIZE.
PERCENTILE RANK
50
40
30
20
10
t 2 df
1·0
1·88
2·92
4·3
6·96
9·92
0 1 2 3 4 5 6 7 8 9 10
Z Scores
KOPPITZ SCORE
12 11 10 9 8 7 6 5 4 3 2 1 0
3·6
2·8
2·4
2·2
2·0
3·4
2·8
2·5
1·9
1·6
1·9
1·8
1·4
1·2
1·1
1·0
1·8
1·75 1·7 1·6 1·6 1·55 1·5 1·45 1·45 1·45 1·45 1·45 1·40 1·40 1·40 1·40
S.D.
0·9 0·85 0·8 0·75 0·7 0·65 0·62 0·6 0·58 0·57 0·55 0·53 0·52
MEAN.
5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
AGE - YEARS