

SCIENTIFIC SESSION 1

6th February 2004 3.00pm-6.30pm Conference Hall

3.00 pm–3.15 pm: Welcome and Introduction by Dr. S.K. Bhargava

Session 1: Childhood growth and obesity

Chairman: Dr. P. Raghupathy

3.15 pm-3.35 pm: Dr. Sheila Bhave

Epidemiology and Strategies of Prevention of Childhood Obesity with special reference to India

3.35 pm-3.55 pm: Professor David Barker

Early origins of obesity: data from Finland

3.55 pm-4.15pm: Dr. Santosh Bhargava/Dr. H.P.S. Sachdev

The early life origins of obesity: data from the Delhi cohort

4.15pm-4.30pm: Dr. C.S. Yajnik/Dr. Anjali Ganpule

Childhood adiposity – DEXA data from the Pune Maternal Nutrition Study

4.30pm-4.45pm: TEA

4.45pm-5.00pm: Dr. Anita Khalil

Non-invasive evaluation of endothelial function and arterial mechanics in overweight adolescents

5.00pm-5.15pm: Dr. Anand Pandit

Symbiosis: Preliminary plans for an intervention study in school children

5.15pm-5.30pm: Dr. P. Raghupathy

Early plans for a diabetes prevention trial in young adults

SCIENTIFIC SESSION 1 (contd.)

6th February 2004 3.00pm-6.30pm Conference Hall

Free papers from the Sneha group

5.30pm-5.45pm: Dr. G.V. Krishnaveni

Growth, and body composition at birth and in early childhood relationships to glucose and insulin metabolism at 5 years; Parthenon Follow up Study.

5.45pm-6.00pm: Dr. B. Antonisamy

Intergenerational studies covering three generations from Vellore—Part I: Secular trends and intergenerational effects in adult height, weight and BMI

6.00pm-6.15pm: Dr. Agnihotri Biswas

Intergenerational studies covering three generations from Vellore — Part II: Trends and effects of parental birthweight on offspring birthweight

6.15pm-6.30pm: Dr. Geethanjali

Plasma Lp(a) levels in young Indian adults and their relationship with birth measurements

Delegates will be able to go shopping between 6.30 and 8.30pm, before dinner. Transport will be arranged to: Jaipur pottery, Macropol (jewellery, handicrafts, paintings), Sodhi textiles, and Miss India.

Drinks and dinner at the Holiday Inn will start at 8.00-9.00pm (exact time to be announced)

SCIENTIFIC SESSION 2

7th February 2004 9.00am- 12.30pm Conference Hall

Session 2: Maternal Nutrition and Programming

Chairmen: Dr Caroline Fall and Dr. H.P.S. Sachdev

Symposium on the Effects of Maternal Nutrition on Patterns of Health and Disease

9.00am-9.30am: Professor Ricardo Uauy, *London School of Hygiene and Tropical Medicine, UK, and University of Chile, Santiago.*

9.30am-10.00am: Professor Alan Jackson, *Institute of Human Nutrition, University of Southampton, UK*

10.00am-10.30am: Dr Mario Soares, *Department of Nutrition, Dietetics and Food Science, School of Public Health, Curtin University of Technology, Perth, Australia*

10.30am-10.50am: COFFEE

10.50am-11.20am: Dr. Ramesh Potdar, Dr. Nick Brown, Dr. Vijaya Taskar, Dr. Parvez Kosgi, Dr. G.Subbulakshmi

Mumbai Maternal Nutrition Study: Update on progress with the trial

Free papers from the Sneha group

11.20am-11.35am: Mr. Charu Joglekar

Insulin resistance in 6 year old rural Indian children

SCIENTIFIC SESSION 2(contd.)

7th February 2004 9.00am- 12.30pm Conference Hall

11.35am-11.50am: Dr. Niranjan Joshi

Increasing parity predicts fatter babies to thinner mothers in rural India

11.50am-12.05am: Ms. Swapna Deshpande

Vitamin B12 deficiency in India: A possible link between fetal nutrition and later disease?

12.05am-12.20am: Dr. Ashish Bavdekar

A proposed multicentre randomised controlled trial of the effect of Vitamin D supplementation in pregnancy on infant health

12.30pm-14.00pm: SNEHA Annual General Meeting (lunch will be served during the meeting)

2.00pm onwards: Bus leaves Holiday Inn at 14.00 for sight –seeing: Amber Fort

6.00-7.30pm: Shopping

8.00pm departure to Gala dinner at ‘Chowkidani’
including professional Rajasthani Dance performance

SCIENTIFIC SESSION 3

8th February 2004 8.00am-11.00am Conference Hall

Session 3: Genes and gene-environment interactions

Convenor and Chairman: Dr. C.S. Yajnik

8.00am-8.20am: Address by Professor N.K. Ganguly , *Director General, Indian Council of Medical Research, New Delhi*

8.20am-8.50am: Dr. Lalji Singh, *Centre for Cellular and Molecular Biology, Hyderabad*

Human genetic variation and genetic disease: Indian perspective

8.50am-9.20am: Dr. Giriraj Chandak, *Centre for Cellular and Molecular Biology, Hyderabad*

Preliminary genetic data from the Pune Children’s Study

9.20am-9.50am: Professor Andrew Hattersley, *Royal Devon and Exeter Hospital and Peninsular Medical School, Exeter, UK*

Overview and future genetic studies in the Indian FOAD cohorts

DISCUSSION

Closing words from Professor David Barker, and felicitation

END OF SCIENTIFIC MEETING

Buses have been arranged to take you back to Delhi or to Jaipur airport in time for your onward flights – exact timings will be announced by Ameya

Childhood Obesity: Epidemiology & Strategies for Prevention (with special reference to India)

DR. SHEILA BHAVE

Department of Pediatrics, KEM Hospital, Pune

The adverse and serious consequences of childhood obesity are now proven beyond doubt. Psycho-social stigmatization may not be a big problem in our country, and severe complications of obesity (such as obstructive sleep apnea, and pseudo tumor cerebrii) are rare. However, obese children have considerably higher risk factors for cardiovascular disease, such as hypertension, dyslipidemia, insulin resistance and impaired endothelial function. Type 2 diabetes is beginning to emerge in children. Most importantly, 50 to 80% of obese children become obese adults and complications of adult obesity are much worse if the obesity begins in childhood.

Obesity is a global epidemic : At least 50% of adults and 20% children in UK and U.S.A. are currently overweight. Most developing countries too are now reporting unprecedented levels of obesity with trends that are substantially rising every year. Recent urban Indian studies show that more than 30% adults and more than 10% children are overweight, and at least 3.5% children may be frankly obese.

Indian Scenario: India has the largest number of diabetic patients in a single country and the numbers are predicted to rise to 57 million by the 2025. Cardiovascular disease is predicted to be the leading cause of premature death in India by the same time. As an ethnic group, Indians (whether they live in India or abroad) are particularly at high risk for insulin resistance and central obesity, the fore runners of these 'life style' disorders. In transitional economies such as in India, obesity and malnutrition often co-exist ("double burden of disease") causing confusion in public health messages.

Frank obesity may not be as high in India as in the west, but the body composition and metabolism of Indians (and Asians in general) make them especially prone to adiposity and its consequences. South Asians have a 3 to 5% higher body fat for the same BMI as compared to Caucasians. The fat is typically located centrally, and around visceral organs where it is metabolically more dangerous than peripheral fat. Recent Pune studies have demonstrated the 'thin fat Indian Phenotype' with evidence of hyperinsulinemia even at birth. Moreover recent longitudinal studies in India have highlighted the deleterious effect of accelerated weight gain in childhood (crossing of centiles) especially in LBW babies.

CAUSES OF THE EPIDEMIC: In India there is a tremendous 'Urban Rural' and 'Rich Poor' divide, prevalence in the urban rich being much higher than in rural areas and poor communities. The causes include:-

1. Changes in life style ('urbanisation'): With improving standards of living, and availability of food in plenty, the upper class societies of India in recent years have urbanized to western levels. The components of life style changes are:-

□ **Unhealthy eating patterns, wrong choices of food:** Traditional diets replaced by energy dense highly processed, micronutrient poor foods. Increased portions, high calorie snacks, junk food revolution, Cola ("thanda matlab") colonisation, and food as rewards.

□ **Sedentary pursuits:** TV and movie watching, video games, internet gazing and telephone gossip sessions are now important activities of children. TV also affects by heavy marketing of colas and other fatty foods.

□ **'Obesogenic' schools and Tuition classes:** An important factor for obesity in India is the intense competition for admissions to schools and colleges with flourishing tuition classes right from nursery levels! Games or physical training sessions are restricted or non-existent in many schools.

□ **Inadequate play areas:** Motorised vehicles for transport (therefore no walking or cycling to school), **erosion of open spaces for exercises and lack of parental time to supervise play are all part of new obesogenic lifestyles.**

2. Genetic ‘Constitutional’ predisposition: Parental obesity is one of the most important determinants of childhood obesity. The factors responsible could be:-

- Familial pattern of eating, exercise and behaviour.
- Modern environment may have unmasked previously silent obesogenic genes.
- Programming of previously malnourished populations to accumulate fat more intensely in an attempt to store for future starvation (‘early life origins’).
- High rate of gestational diabetes in pregnant women causing higher birth weights in babies leading to intergenerational effects of obesity.

3. Other factors: Prolonged and exclusive breast-feeding is associated with a significantly lower rate of obesity and hypertension in later life. It is not clear if early introduction of energy dense supplements in infancy has contributed to childhood obesity in India.

□ The high glycemic index of our predominantly carbohydrate diet may be responsible for hyperinsulinism, weight gain and eventual type 2 diabetes.

□ Another interesting factor is the obesity rebound phenomenon. But determinants of this factor are not yet well known.

Measurement Of Obesity: The terms overweight and obese are often used somewhat loosely and interchangeably. However,

standardization is necessary for international and secular comparisons. Although there are many limitations of BMI, as of now it appears to be the most practical way of measuring and comparing obesity for clinical and epidemiological purposes and obese are often used somewhat loosely and interchangeably. However, standardization is necessary for international and secular comparisons. Although there are many limitations of BMI, as of now it **appears to be** the most practical way of measuring and comparing obesity for clinical and epidemiological purposes. As per WHO classification (for adults) BMI > 25 = overweight and BMI > 30 = obesity. As per IOTF proposals the standards for Asia are BMI > 23 = overweight, BMI > 25 = obesity.

As BMI values change physiologically (substantially) with age and sex the reference BMI standards for children are:

(i) The **NCHS/CDC BMI charts** from USA with **>85th centile = overweight and >95th centile = obesity** A recent large change in BMI (annual increase of 3 to 4 units) is also an indicator for prompt evaluation.

(ii) New BMI standards in children using a large internationally representative sample (**Cole et al 2000**) with cutoffs analogous to adult BMIs of 25 and 30.

Other markers of obesity are: skinfold thickness, waist circumference, waist hip ratios, bioelectrical impedance analyses Dual energy X-ray Absorptiometry (DEXA) and air displacement plethysmograph (BOD-POD). All these markers have their individual advantages (e.g. waist circumference for central obesity, DEXA for actual fat percentage), but none are really standardized as yet in children for routine clinical practice.

Prevention Of Childhood Obesity (Public Health Approach): **Atleast 1 in 10 urban middle class children in India is overweight. Only community-based approaches can address such large numbers of affected children. Long-term studies have shown that**

interventional programmes in children are far more successful and cost effective than in adults.

Strategies and Aims: As a Public Health Approach, essentially all children, adolescents and families should benefit from counseling to prevent excess weight gain and obesity

• **Life style approach:**

- i) **Healthy eating patterns:** Emphasis should be on nutrition rather than 'dieting'. It is important to maintain healthy components of traditional diets and guard against heavily marketed energy dense fatty and salty foods.
 - ii) **Increase physical activity levels:** A period of atleast one hour a day.
 - iii) **Decrease sedentary behaviour:** Television restricted to no more than 2 hours a day)
- 'Target' Populations: Urban children from higher and mid socio economic status.
 - Address 'Behaviour' Change
 - Focus on involvement of entire family
 - Build supportive infrastructure

**Special Strategies to Target Specific Age Groups
Infants and Young Children:**

- Mothers should prevent excess weight gain in pregnancy; control diabetes or impaired glucose tolerance in pregnancy.
- Promote exclusive breast-feeding for six months.

- Assure appropriate micronutrient intake especially of iron, calcium and vitamins to promote good linear growth.
- Instruct mothers to accept the child's appetite and not to force feed.

Children and adolescents:

The fat rebound age (5-8 years) and adolescence are particularly high-risk periods for accumulating fat.

- Promote active life styles (See strategies)
- Impart health education skills to make healthy food choices
- Modify environments to promote physical activity
- Educate about the evils of alcohol/tobacco to adolescents.

Channels of Interventions / Health Education

School Based Programs:

Schools are probably the ideal medium of intervention as they are central to children's lives and information can be relatively quickly dissipated through this channel.

- Training of teachers in lifestyles
- Introduction of ideal school meals
- Introduction of 'nutrition and physical education' in school curriculum as a 'Scoring subject' with marks.
- Involve parent associations.

Media involvement :

Health centres/Doctors/Other Professionals

- Think 'prevention of obesity' at all visits.

- Incorporate BMI charts and relevant health education in patient records.

Governmental Authorities :

- Devising national strategies
- Providing safe exercise opportunities.
- Consider taxation on ‘fatty food’

Such a Public Health Approach requires strong social and political will with concurrent medical motivation and management.

P.S.: Please see our draft of Recommendations of the IAP Task Force for Childhood Prevention of Adult Diseases: Childhood Obesity.

We would value the discussion and suggestions of the SNEHA group for final recommendations.

□

Parental determinants of body composition in 6 year old rural Indian children.

ANJALI GANPULE, C S YAJNIK

Department of Diabetes, KEM Hospital, Pune 411011.

Introduction: We have reported on the ‘thin fat’ Indian babies and determinants of their birth size in the Pune Maternal Nutrition Study (PMNS). Recently we have completed a follow up of these children at 6 years of age. This provides us with an opportunity to study determinants of body composition in these children.

Aim: To study the relationship between parental characteristics and body composition of 6-year-old rural Indian children.

Hypothesis: Adiposity at 6y of age is related to maternal dietary intake during pregnancy, independent of maternal and paternal size.

Methods: We used correlation analysis to study the relations between maternal and paternal characteristics (size and nutrition) and body composition of the offspring at 6 yrs (measured by Dual energy X-ray Absorptiometry).

Results:

	Boys	Girls
N	374	333
Age (y)	6.1 (6.0, 6.2)	6.2 (6.1, 6.3)
Height (cm)	109.5 (107.2, 113.2)	109.4 (106.5, 112.4)**
Weight (Kg)	16.4 (15.2, 17.6)	15.7 (14.6, 16.9)**
Fat mass (Kg)	2.9 (2.3, 3.5)	3.3 (2.6, 3.9)**
Lean mass (Kg)	13.0 (12.2, 14.2)	12.1 (11.2, 12.8)**
Bone mineral content (Kg)	0.65 (0.59, 0.72)	0.61 (0.55, 0.69)**

Median (IQR), ** p < 0.001, for difference between boys and girls. Adjusted for age

Adiposity in both boys and girls was predicted by the skeletal size and central obesity of father ($p < 0.001$, all). Maternal adiposity predicted adiposity in boys ($p < 0.05$) but not girls. Lean measurements were determined by paternal skeletal size in boys ($p < 0.001$) and maternal skeletal size in girls ($p < 0.05$).

Intake of energy rich foods by the mother during pregnancy predicted adiposity and intake of calcium and protein rich foods predicted lean measurements in boys ($p < 0.05$). Higher intake of protein rich foods by the mother predicted lower adiposity, and higher intake of energy rich foods predicted lower lean mass in girls. Consumption of micronutrient rich foods (GLV and fruits) was not related to body composition of children at 6 years of age.

Conclusion: Body size of both the parents was a strong determinant of body composition in children. There was a sexual dimorphism in the relationships. Maternal intake of energy and protein rich foods influenced adiposity and lean mass. Intake of micronutrient rich foods by the mother during pregnancy was not related to offspring body composition at 6 years of age.

Noninvasive Evaluation of Endothelial Function and Arterial Mechanics in Overweight Adolescents

ANITA KHALIL, VISHAL KUMAR, HPS SACHDEV

Divisions of Cardiology and Clinical Epidemiology, Department of Pediatrics, Maulana Azad Medical College, New Delhi 110002, India.

Objective: To evaluate endothelial function and arterial mechanics in apparently healthy overweight adolescents.

Design: Analytical observational study

Setting: Tertiary hospital.

Methods: 40 asymptomatic, normotensive and non-smoking adolescents (11 to 18 years old) were evaluated. Of these 20 were overweight or obese as per International Obesity Task Force criteria while 20 were controls. High-resolution ultrasonography was performed to measure flow mediated and Glyceryltrinitrate induced dilation in brachial artery, and arterial mechanics in common carotid artery.

Results: Overweight adolescents had significantly lower ratios of flow-mediated dilation to Glyceryltrinitrate mediated dilation (0.40 ± 0.41 versus 0.61 ± 0.17 ; $P = 0.039$). On age and sex adjusted multiple regression analysis, the ratio of flow mediated to Glyceryltrinitrate mediated dilation had a significant negative association with body mass index ($P = 0.012$) and mean skin fold thickness ($P = 0.011$). However for mean skin fold thickness, flow mediated dilation also had a significant negative association ($P = 0.027$). None of the measures of arterial mechanics were significantly different amongst overweights and controls or significantly associated with either body mass index or mean skin Fold thickness.

Conclusions: Endothelial function can be mildly impaired in apparently healthy adolescents who are overweight (assessed by body mass index) or adipose (assessed by skin fold thickness). The use of overweight for screening adolescents likely to develop coronary artery disease is therefore justified. Skin fold thickness is a better indicator than Body Mass Index for predicting endothelial function.

Growth, and body composition at birth and in early childhood - relationships to glucose and insulin metabolism at 5-years - Parthenon Follow up Study.

GV KRISHNAVENI, JC HILL, SR VEENA, CHD FALL

Holdsworth Memorial Hospital Research Unit, Mysore,
MRC Environmental Epidemiology Unit,
Southampton General Hospital, Southampton, UK.

We studied 830 pregnant women attending the antenatal clinics of the Holdsworth Memorial Hospital (HMH), Mysore, South India, as part of a prospective study into the maternal and fetal origins of adult type 2 diabetes. They underwent an Oral Glucose Tolerance Test (OGTT) at 30+/-2 weeks. 674 of these women delivered at HMH. Detailed anthropometry was performed on the offspring at birth, and annually thereafter; 605 mothers returned with their offspring at 4 years of age.

The Mysore babies were small compared to UK neonates, but, as recently shown in Pune neonates, the deficit varied for different body measurements. While birthweight (-1.1 SD) was considerably lower, crown-heel length (-0.3 SD) and subscapular skinfold thickness (-0.2 SD) were relatively spared. At four years, subscapular skinfold thickness was larger than the UK standards (+0.23 SD, $p < 0.001$) despite all other body measurements being significantly smaller. (Fig 1) Findings at 4-years were similar in comparison with another standard, based on Dutch children.

The newborns of mothers with gestational diabetes were larger in all measurements than those of non-diabetic mothers. These differences were considerably diminished during infancy and were not significant at 1 year of age. By 4 years, female Offspring of Diabetic Mothers (ODM) were again significantly more adipose and had bigger arm circumferences. Even in non-diabetic pregnancies, higher maternal fasting glucose concentrations were associated with higher neonatal

of offspring birthweight. Mothers who had LBW were 2.7 times more likely to deliver a LBW baby as compared to mothers who weighed > 3000 g. Every kg increase in maternal birthweight showed a 183 g increase in offspring birthweight. Similarly LBW fathers had 2.8 times higher risk of producing LBW babies as compared to fathers weighing > 3000 g at birth. Each kg increase in father's birthweight resulted in a 218 g increase in offspring birthweight. 26.8% of LBW babies among offspring were associated with maternal LBW and 24.4% with paternal LBW. Heavier and taller parents are less likely to produce LBW babies. Each cm increase in fathers and mothers' height resulted in an increase of 12 g and 9 g in offspring birthweight respectively.

Intergenerational effects on birthweight are partly mediated by parental genetic influence and partly by environmental effects – magnitude of each needs to be seen in further studies. Mother, father and offspring need to be studied in the same model to determine the differential contribution of each parent towards the offspring birthweight.

Intergenerational studies covering three generations from Vellore—Part I: Secular trends and intergenerational effects in adult height, weight and BMI

***B ANTONISAMY, P RAGHUPATHY, CHD FALL,
J RICHARD***

Department of Biostatistics and Child Health
Christian Medical College, Vellore,
MRC Environmental Epidemiology Unit, Southampton UK.

The anthropometry of individuals varies from country to country and within countries there are variations and secular trends. The changes in the prevalence of under and overweight across generations are important in terms of their long-term effect on adulthood obesity and health outcomes. The intergenerational changes were studied by comparing the anthropometry of parents (paternal and pre-pregnant maternal) with that of their adult offsprings.

From a birth cohort enrolled in 1969-73 ($n = 10670$), 4092 subjects with all early life data were targeted and 2218 young adults aged 26-32 years were studied after obtaining their informed consent and institutional ethical review board clearance. Their mean (\pm SD) age was 28.3 ± 1.1 years, which was similar to the mean age of their parents at the time of enrolment (maternal age 26.8 ± 6.0 years).

The mean (\pm SD) adult height ($162.3\text{cm} \pm 6.5$), weight ($51.9\text{ kg} \pm 7.7$) and BMI (19.7 ± 2.8) of fathers showed considerably lower values when compared to those of their sons ($166.5\text{ cm} \pm 6.6$; $57.5\text{ kg} \pm 11.3$; 20.7 ± 3.4 respectively, $P < 0.001$). Similar striking changes were observed when mothers ($150.8\text{ cm} \pm 5.4$; $42.8\text{ kg} \pm 6.1$; 18.8 ± 2.5) were compared with their daughters ($153.6\text{ cm} \pm 5.8$; $49.1\text{ kg} \pm 10.5$; 20.8 ± 4.1 , $P < 0.001$). The percentage of underweight (BMI < 18.5) was 50.1 in mothers and 34.3 in their daughters; 34.6 in fathers and 31.1 in their

sons. The shift in percentage of overweight (BMI >23) was 5.7 in mothers and more than 5-fold higher – 28.5 in their daughters; as compared to 10.7 in fathers and 24.6 in their sons (P <0.001).

Over a period of 30 years, the decline in underweight and rise in overweight was more marked among women and in the urban individuals (P <0.001). As a result, though fetal nutrition and birth weight might have improved, the contribution of obesity to adult disease or the additional effects of parental under nutrition, as well as childhood and adolescent nutritional status needs further evaluation.

Plasma Lp(a) levels in young Indian adults and its relationship with birth measurements

***FS GEETHANJALI, B ANTONISAMY, P RAGHUPATHY,
CHD FALL***

Departments of Biochemistry, Biostatistics and Child Health,
Christian Medical College, Vellore,
MRC Environmental Epidemiology Unit, Southampton, UK.

Lipoprotein (a) is an independent and inherited risk factor for coronary heart disease. Quantitative analysis of Lp(a) concentrations has confirmed large differences in Lp(a) levels among different ethnic groups. Its level cannot be altered by changes in diet, environment, exercise or medication. Okosun et al (1) has shown elevated Lp(a) levels in black and white American children aged 5-11 years who were born with LBW. Pulzer et al (2) also observed similar results in children and adolescents born small for gestational age.

A total of 2,218 young Indian adults aged 26-32 years from an original cohort of 10,670 born in the town of Vellore and the villages nearby during 1969-73 were traced and brought to clinic in 1998-2002 for a detailed clinical evaluation and investigation of cardiovascular risk factors. Socio-economic, nutritional (food frequency), life style, past medical history, anthropometric and biochemical data were recorded using a standard proforma. Lp(a) was measured by immunoturbidimetric method.

Overall mean Lp(a) level reported in this study is very similar to that reported in earlier studies from India. None of the birth measurements correlated with lipid parameters and Lp(a). However the head circumference was negatively correlated with plasma Lp(a) levels (p=0.01) after adjusting with age, sex, sum of skinfold measurements and BMI. Lp(a) level among adults in relation to birthweight has been examined for the first time in India.

References:

1. Okosun IS, Dever GE, Choi ST. Low birth weight is associated with elevated serum lipoprotein(a) in white and black American children ages 5-11 y. *Public Health* 2002 Jan; 116(1): 33-8
2. Pulzer F, Haase U, Kratzsch J, Richter V, Rassoul F, Kiess W, Keller E. Lipoprotein(a) levels in formerly small-for-gestational-age children. *Horm Res.* 1999;**52(5):241-6**

Effect of Maternal Nutrition on Patterns of Health and Disease

RICARDO UAUY.

Public Health Nutrition London School of Hygiene and Tropical
Medicine University of London UK and Institute of Nutrition
(INTA) University of Chile

Nutrition of women throughout the life cycle (before and during the reproductive years) has great significance not only for individual women but also for the health and wellbeing of society. Nutrition of mothers before conception and early in gestation is crucial for normal embryogenesis and for adequate early placental growth and fetal development. In fact cellular growth is most rapid in early fetal development. The role of pre-pregnancy nutritional status can no longer be ignored. Nutrition of the mother throughout gestation can affect the growth and development of the fetus in multiple ways with short term and long term consequences on health and disease patterns at later stages of life. A relative nutritional deficit or excess can mark (program) both the structural and or functional development of the fetus with lifelong impact. The magnitude, timing and duration of the nutritional noxa are important in defining the effect. Thus, the consequences of subtle imbalances are more difficult to evaluate than severe nutritional deficits or toxicity. Most often, it is impossible to fully dissect if the long-term consequences of abnormal fetal growth are the result of maternal nutrition or if they are derived from the multiple other factors that concur in the causal pathway. Thus it is likely that combinations of nutritional, genetic and non-nutritional epigenetic factors are responsible for the observed increased risk in adult chronic disease resulting from abnormal fetal growth. The interaction between genes and environment (including nutrition) occurs from the moment of conception if not earlier as has been defined for some nutrients (iodine, folate, retinol) as well as toxicants.

There are few studies that evaluate the direct effect of maternal

malnutrition on patterns of adult disease. Most epidemiologic studies and controlled interventions have examined early outcomes such as physical growth, body composition, and metabolic indices that are associated the patterns of adult chronic disease (coronary heart disease, hypertension, obesity, diabetes, and some type of cancers). More recently evidence from trans-generational studies has become available demonstrating beneficial effects not only on the first generation born, but also in their offspring. The data for balance food supplementation as reviewed in the recent WHO 2003 technical consultation on optimal fetal growth demonstrate small but significant gains of maternal nutritional supplementation in terms of reduction of fetal growth restriction and LBW risk with little or no effect on preterm delivery. The results from population studies of single nutrient interventions are more elusive to interpret and inconclusive except for populations with high prevalence of specific nutritional deficiencies.

The effects of maternal nutrition on adult health and disease are mediated by effects of nutrition on patterns of gene expression, in some cases affecting transcription factors that control multiple target genes. This may explain the widespread effect of single factors on various developmental indicators. Modern biological techniques such as micro-arrays are permitting the evaluation of how nutrients influence the expression of thousands of genes simultaneously. Parallel observations on how nutrients interact with hormonal receptors, binding proteins and signal transduction processes affecting growth and development will permit a better understanding of how maternal nutrition impacts cell and organ growth at critical periods of development and ultimately define the pathophysiology of fetal growth restriction under different ecological settings. This should ultimately facilitate defining effective approaches to optimize fetal growth not only based on observed birth weight distributions but on maximizing life long health and preventing adult disease and disability. This has significant social and economic implications for societies undergoing a rapid nutrition transition. (Supported by Fondecyt-Chile Grant 1990078)

Nutritional Implications Of Pregnancy In Teenagers.

ALAN A JACKSON,

Institute of Human Nutrition, University of Southampton.

Across a wide range of societies the risk of perinatal complications to the mother and the newborn are substantially increased in those who become pregnant at an early age during the teenage years. The risk is most marked when the mother is under 17 years of age, but even women of 18-19 years have a greater risk than at older ages. An estimated 25% women worldwide have their first child by 19 years of age. The UK has the highest rates of adolescent pregnancy in Europe, about 25 per 1,000 women, with nearly half of conceptions being aborted. The prevalence of adolescent pregnancy by age is not easy to determine, but for many societies it is driven by social factors which overlay the biological competence to become pregnant. There are important intergenerational effects and mothers who are born small have a higher risk of losing their babies, which is particularly marked if the baby is small in comparison with the mother's own birthweight. It is not known whether dietary supplementation during adolescence would support increased growth in the pregnant adolescent or improved lactational performance. In the absence of any direct evidence of the benefit of any practical interventions which might increase birthweight specifically in adolescent groups, one can only draw inferential conclusions, based upon an understanding of the factors of importance which contribute to fetal growth in general, and how these might operate during adolescence.

In a successful pregnancy maternal health is maintained, a healthy baby is delivered, and the mother has adequate ability to nurture her baby. For the mother to be solely dependent upon her dietary intake at any stage of pregnancy would represent a very high risk strategy. Hence, the preparation for pregnancy and the ability to have adequate reserves

to call upon during the different stages is important for a successful outcome. The most readily available measure of pregnancy outcome is the weight of the newborn, although this fails to differentiate important differences in the pattern and pace of growth. Adolescent pregnancy is associated with higher rates of prematurity, low birthweight and perinatal mortality. Although the socio-demographic factors such as unmarried status, poor education and antenatal care which are associated with lower birthweight are often more common among pregnant adolescents, the increased risks of poor birth outcome appear to be independent of these factors. In the USA, for white women from a well to do background, with good education and antenatal care, 13 to 17 year olds had significantly higher risk than mothers aged 20 to 24 years for low birth weight, prematurity, or being small for gestational age. Older teenagers, 18 -19 years of age, also had a significant increase in risk. Continued maternal growth during adolescence makes an important difference to birth weight. For mothers who were still growing, the infants of primips weighed 117 g less than those of mothers not growing and amongst multips the difference was 206 g. For a group of urban, poor women in the USA, the loss of fat from the upper arm was associated with increased birth weight. For women in whom pregravid weight was low, the loss of fat was associated with a birth weight which was lower by 300 g, than for women with higher pregravid weight who also lost fat. It is suggested that in women in whom fat stores were relatively depleted at the start of pregnancy, there was inadequate reserve to enable effective mobilisation. In contrast, birth weight was lower for women who continued to gain fat late in pregnancy. Thus the women who had the largest gestational weight and retained most weight postpartum, bore infants who were smaller, suggesting a block in their ability to effectively mobilise deposited fat for use by the fetus. Lower newborn weight was seen when the mother continued to accrue fat in the subscapular region or on the back of the arm beyond 28 weeks gestation. Despite an apparently sufficient weight gain and the accumulation of apparently abundant fat, it was not used to enhance fetal growth. These mothers needed to achieve larger gestational weight gains for a baby of equivalent weight. A more detailed analysis showed

that the added weight and added fat mass were only beneficial up to a point. Beyond that point increased fat mass no longer carried any advantage as there was no further increase in fetal weight, but rather an increase in excess lipid retention by mother post-partum. Experimental studies in sheep support these observations and provide some evidence for the likely mechanisms involved. The data from Europe appear at variance, as in three studies, the main associate with birth weight was maternal lean body mass, rather than fat mass or fat accretion. When compared with studies in the USA, the data from Europe leave open the question of whether the differences observed are related to the age of the subjects in the study, the wider aspects of social disadvantage, dietary patterns, or some combination of factors.

For any pregnancy any other stressor can lead to a change in the availability of nutrients to the developing fetus. Low grade pelvic inflammation associated with an ascending infection is common and may play a greater role than previously recognised. Behavioural (smoking and alcohol), psychological (physical and emotional violence) and social stressors (poverty and deprivation) can also lead to impaired reproductive performance. Although the evidence is limited these are all likely to be common, and additive for adolescent pregnancies.

Infants of adolescent women appear to have a deficit of up to 200 g, relative to older more mature women, which is not accounted for directly by any specific environmental factor, and might be directly attributed to the relative immaturity of their bodies in general and their reproductive system in particular. Any intervention which ameliorates or removes one or other adverse factor is likely to confer some benefit, but it seems likely that even under excellent circumstances women who enter pregnancy at an age below 18 years, carry an increased risk for themselves and their baby. If this is so then the most important intervention which might confer benefit is to delay the time of the first pregnancy to later than 19 years of age.

Dietary calcium in the regulation of body composition.

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Calcium fulfills a variety of physiological functions in the body from muscle contraction and blood clotting, to mineralization of the skeleton. Recently, a role in the regulation of body weight and composition has been proposed. Cellular studies have demonstrated that fluctuations in intracellular calcium reciprocally regulated enzymes influencing fat synthesis and lipolysis. A rise in intracellular calcium promoted fatty acid synthase expression and activity, while reducing basal lipolysis. Accordingly, the provision of dietary calcium is expected to reduce intracellular calcium via the suppression of calcium regulating hormones. This would decrease fat synthesis and increase lipolysis, and thereby reduce body fat content. Animal studies, as well as retrospective analyses of clinical and epidemiological data, provide some support for the inverse relationship between dietary calcium and adiposity. Our results indicate that dietary calcium acutely stimulates energy expenditure and fat oxidation, while reducing energy and fat intake. Calcium is important to nutrition throughout the lifecycle, yet even in a developed nation like Australia, a sizeable segment of the population has intakes below the RDI. India faces the dual burden of chronic undernutrition and obesity. If the role of calcium in energy balance is confirmed, it could have additional relevance to fetal and post natal nutrition, and the risk of the metabolic syndrome in adulthood.

Mumbai Maternal Nutrition project; background, methodology, logistics and results to date

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Low birthweight is associated with a high infant mortality and long-term morbidity.

Nutritional interventions in pregnancy are a hypothetically attractive solution but trials using protein energy supplements have been disappointing. Micronutrient enhancement trials to date have focused on a pharmacological approach. Observational studies notably the Pune Maternal Nutrition Study suggest a role for a food based approach. Using a novel form of supplementation, the MMNP aims to test the hypothesis that enhancement of women's micronutrient status through food from before conception and through pregnancy improves a number of foeto-maternal outcomes including fetal growth, birthweight, infant mortality and early markers of adult chronic disease.

First outline the methodology, progress, scientific debate; ethical issues and early results will be outlined. This will be followed by a three-part presentation including the philosophy and organisation of a sustainable infrastructure, community sensitisation and data management.

Insulin resistance in 6year old rural Indian children

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Background: Thrifty phenotype hypothesis predicts that improved fetal nutrition will reduce the risk of IRS (Insulin Resistance Syndrome). PMNS (Pune Maternal Nutrition Study) offers an opportunity to test this idea. Details of maternal nutritional intake, physical activity during pregnancy and neonatal size measurements are available in the database. The children have been studied at 6 years of age for insulin resistance markers and other cardiovascular risk factors.

Hypothesis: Higher maternal intake of micronutrient rich foods and higher circulating concentrations of folate and Vitamin C during pregnancy will predict improved insulin sensitivity in the offspring at 6 years.

Methods: We measured IRS variables (HOMA-R, HDL cholesterol, plasma triglycerides, systolic blood pressure) and plasma cholesterol concentration at 6y of age in 707 children born in PMNS. Relation to maternal nutrition and metabolism during pregnancy was studied by regression analysis.

Results: Neither HOMA-R nor other IRS variables in the offspring were related to macronutrient consumption during pregnancy.

Frequency of intake of GLV, dairy products, and fruit by mother at 18 and 28 wks gestation associated with larger size at birth, was not associated with offspring HOMA-R at 6y. However increased frequency of maternal fruit intake was predictive of higher plasma total cholesterol, HDL cholesterol and triglyceride concentrations in the offspring ($p < 0.05$). Frequent consumption of snacks and festival foods by mother

was associated with higher HOMA-R in the offspring ($p < 0.01$, both). Many of these relations were independent of birth size.

Higher maternal circulating Vitamin C and erythrocyte folate concentrations predicted higher HOMA-R in the offspring ($p < 0.001$, both). There was no relation with any other IRS variable.

We defined increased cardiovascular risk for a variable as belonging to either upper (HOMA-R, total cholesterol, triglycerides, systolic blood pressure) or lower quartile (HDL) of distribution. Increasing frequency of maternal consumption of GLV at 28 wks gestation predicted presence of increasing number of cardiovascular risk factors in the offspring ($p < 0.05$).

Increased maternal activity at 28 weeks gestation was not related to HOMA-R but predicted lower plasma total and HDL cholesterol and triglyceride concentrations in the offspring ($p < 0.05$ for all).

Conclusions: Our results do not support the hypothesis that consumption of maternal micronutrient rich foods during pregnancy will improve insulin sensitivity in the offspring at 6 y of age. On the contrary, increasing frequency of consumption of a number of food items and higher circulating micronutrient concentrations during pregnancy predicted increased insulin resistance and higher cardiovascular risk in the offspring. Our data challenges the 'thrifty phenotype' hypothesis and supports the 'fuel mediated teratogenesis'.

Increasing parity predicts fatter babies to thinner mothers in rural India

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Background: Babies born to multiparous mothers are heavier than those born to primis. There is little information on body composition of these heavy babies, nor on changing maternal physiology with increasing parity.

Aim and objectives: 1) To study the relationship between maternal parity and offspring body fat and lean mass measurements in a cross sectional study, 2) To study maternal size, body composition, her metabolism in pregnancy and cardiovascular measurements in relation to parity.

Methods: We compared detailed body size of babies born to 785 mothers in the Pune Maternal Nutrition Study (PMNS) in relation to maternal parity. We also compared maternal characteristics across parity.

Results: Mothers were on average 21 y old and 35% were primiparous, 4.2% were grand-multiparas (parity>3). Parity was strongly related to maternal age ($r=0.67$, $p<0.001$) but not to her educational and socio-economic status. Multiparous women were lighter (46 vs 48 kg, $p<0.01$) and less adipose (body fat 10.8 vs 11.8 %, $p<0.001$) compared to primiparous women. There was no relationship between parity and height. Dietary intake and weight gain during pregnancy were similar but multiparous women worked harder than the primis (total activity score 67 vs 57, $p<0.05$). Circulating concentrations of vitamin C (4.0 vs 4.0 mmol/L) and red cell folate (427 vs 419 ng / mL) were similar in the two groups but plasma ferritin concentration was higher in primiparous (12.0 vs 16.0 mg/L, $p<0.01$). There was no correlation

between parity and fasting plasma glucose, insulin and triglyceride concentrations but multiparous women had lower systolic blood pressure, lower plasma albumin concentration and lower white cell count ($p<0.001$, all). Two-hour plasma glucose concentration during an OGTT was lower in multiparous women (68.0 vs 94.0 mg%, $p<0.01$).

The offspring of multiparous mothers were heavier (2770 vs 2550g) and had thicker skin folds (subscapular 4.2 vs 3.8 and triceps 4.2 vs 4.0, $p<0.001$, both). There was no difference in crown-heel length and head circumference. This difference was independent of baby's gender, gestation at birth, maternal body fat, systolic blood pressure, albumin concentration and white blood cell count and paternal size. At 1 year of age children of primiparous women were heavier than those of multiparous women but there was no difference in their subscapular and triceps skin folds. This trend persisted till 6year of age. There was no difference in glucose and insulin concentrations and other cardiovascular risk variables at 6y in relation to maternal parity.

Conclusion: Multiparous women give birth to heavier and fatter babies. This could be related to larger vascular expansion and a lower inflammatory response during pregnancy in the multiparous compared to the primiparous women.

Vitamin B12 deficiency in India: A possible link between fetal nutrition and later disease?

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Vitamin B12 deficiency has been described in Indians living in India and those living abroad since 1950's. However this fact remained largely unnoticed till recent reports of high tHcy concentrations in Indians predominantly due to vitamin B12 deficiency. More than half of the subjects in our original case control study were vitamin B12 deficient and 3/4th had hyperhomocysteinemia (*Am J Clin Nutr*, 2001). Interestingly, none of the Indians studies including our case control study in Pune have shown an increased risk of CHD in hyperhomocysteinemic subjects.

In a population based study (CRISIS) we investigated determinants of vitamin B12 deficiency and hyperhomocysteinemia in rural and urban men. In addition to increasing age, the two major determinants of vitamin B12 deficiency and hyperhomocysteinemia were place of residence and vegetarianism. Two third of rural, half of slum and three fourth of middle class urban residents were vitamin B12 deficient (< 150 pM). This contributed to hyperhomocysteinemia in half of rural and slum residents and three fourth of urban middle class residents. Surprisingly, folate deficiency was rare in our study subjects.

With this background we investigated maternal vitamin B12 and folate status and circulating tHcy concentration during pregnancy in relation to neonatal size in the Pune Maternal Nutrition Study. Plasma tHcy concentration at 28 wk gestation was higher in mothers who delivered SGA babies (n=30, birth weight < 10th centile) compared to mothers who delivered AGA babies (n=50, ≥ 10th centile). Both vitamin B12 and folate status were strong predictors of plasma tHcy concentration. Seventy percent of mothers were vitamin B12 deficient while none were

folate deficient. Plasma tHcy concentration >6.8 uM increased the risk of delivering SGA baby more than 3 times (OR 3.4, CI 1.1-9.9) compared to mothers who had lower plasma tHcy concentration.

Thus, our results show that vitamin B12 deficiency is very common in Indians in India and that it could contribute to small size at birth. Small Indian babies are thin by conventional criteria (Ponderal Index) but adipose when measured appropriately. We are intrigued by the idea that vitamin B12 deficiency in a folate replete population might contribute to relative adiposity and its associated morbidity.

A Multicentre Randomised Controlled Trial Of The Effect Of Vitamin D Supplementation In Pregnancy On Infant Health

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Vitamin D is essential for growth, skeletal homeostasis, bone mass accrual and prevention of rickets. Wider roles for vitamin D are emerging. Currently, much interest focuses on the role of vitamin D in the pathogenesis of diabetes. Three studies of vitamin D supplementation in pregnancy have shown reduced size at birth or age one year for children whose mothers received placebo. Differences in length were large (0.9 – 1.1 SD scores) at age one year, independent of post-natal vitamin D intake. In separate studies, low vitamin D intake in infancy was associated with reduced later bone mass and an increased risk of type I diabetes. Data from India and China indicate that one third of pregnant women are deficient (serum 25 OH D <11ng/l) and one third insufficient (11-20ng/l) in vitamin D.

We propose a randomised, double blind, controlled trial of vitamin D supplementation during pregnancy in India and China and in Asian populations in Europe. The supplement will be equivalent to 1000 IU/d of vitamin D during the last 3 months of pregnancy. We will recruit 1000 women in each region over a two-year period, allowing within region detection of effects as small as 0.225 SD scores (assuming 20% loss of subjects) and 0.13 SD scores for the whole study with 90% power.

The outcomes to be assessed include growth (length, weight, body proportions), biochemical measures of vitamin D sufficiency (25 OH D, PTH), bone turnover (alkaline phosphatase, urine NTx), body composition (deuterium dilution, dual energy x-ray absorptiometry) and

insulin resistance (fasting glucose:insulin ratio) at birth and one year of age.

The proposal addresses the specific call focusing on micronutrient issues in child health targeting the continuing scourge of rickets and increasing problem of diabetes. The consortium will provide a platform for future studies. Inclusion of a European arm will facilitate comparisons relating to environment and adoption here of any necessary preventative measures.

Human Genetic variation and Genetic Disease : Indian Perspective

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Genetic differences between people and populations affect susceptibility to disease, resistance to infection, and response to drug treatment. Most of the variation in susceptibility to highly prevalent diseases is caused by variations that occur at high frequency in human populations. India represents one of the largest human diversity, consisting of 4635 culturally and anthropologically well-defined populations with little or no gene flow between them. The discovery of human genetic variation is likely to transform our understanding of medical science and the practice of clinical medicine. Hence, study on Indian populations not only provides insight into their complex origin, history and relatedness, but also helps in understanding molecular pathology of genetic diseases. Our interest has been to study both population history and molecular mechanism of diseases.

Our recent study on nearly 4000 individuals belonging to 65 populations revealed that the tribal populations of Andaman Islands are probably the descendents of the earliest migrants from Africa about 60,000 – 1,00,000 years ago. Interestingly, we found a few tribal populations in India, which show close affinities with Andaman tribes. Further, we have also found a novel mtDNA haplogroup - R8 that might be the source of spread of this lineage in Southeast Asia. As each of the Indian populations is unique in its genetic landscape, etiologies of genetic diseases are often different from other global populations as well as from each other. For example, mutations in cationic trypsinogen gene (PRSS1) has been reported to be associated with chronic pancreatitis in many Western populations, but not in Indian population and CAG repeat motif in the androgen receptor gene has not been found to be associated with male infertility in the Indian population. Similarly, we

have found novel mutations in DAZL, SPINK1 and CYP1A1 genes in individuals with infertility, fibrocalculous pancreatic diabetes, and recurrent early pregnancy loss, respectively. These studies suggest that the Indian populations are unique and the genetic attributes, underlying the genetic disease, valid for other global populations may not be valid for Indian populations.

GENETIC STUDIES IN PUNE COHORTS

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There is a rapidly increasing epidemic of type 2 diabetes in India and other Asian countries. The thrifty genotype and the thrifty phenotype are two nonexclusive explanations. Recent evidence suggests that in addition to fetal parameters, postnatal factors must also contribute. Thus, a life-course model of evolution of insulin resistance and type 2 diabetes, incorporating fetal, postnatal and adult components, seems most appropriate. Diabetes Unit at KEM Hospital in Pune has pioneered the Indian studies of the 'fetal origins' of diabetes and CHD. This includes two cohorts: 1) Pune Children's Study (PCS), and 2) Pune Maternal Nutrition Study (PMNS). Between them they offer the best prospective information on determinants of fetal and childhood growth and evolution of insulin resistance syndrome. Pune studies are also empowered with paternal measurements and stored DNA samples for future analysis to look at possible genetic explanations for these relationships. After publication of 'fetal insulin' hypothesis, it was obvious that genetic mechanisms need to be looked at more closely. This set the ground for appropriate collaboration and after lot of discussions, collaboration was set up between the KEM Hospital, Pune, the Centre for Cellular and Molecular Biology, Hyderabad and the Department of Molecular Genetics, Exeter, UK to study genetic determinants of fetal growth and type 2 diabetes which has been formally approved by both CSIR and ICMR. Many candidate genes are being and will be studied for their possible role in the aetiology of type 2 diabetes. In addition to the above two cohorts, there are other cohorts in Pune, which will contribute to these studies: the Wellcome Diabetes Study, the CRISIS study and

the Gestational Diabetes Study. DNA samples and extensive phenotypic information have been collected prospectively over years. A systematic collection of DNA samples and relevant phenotypic information will be started in the Diabetes Clinic of the KEM Hospital and the network of collaborating clinics, concentrating on patients diagnosed before 45 years of age.

In the first phase we plan to look at genetic influences in the triglyceride and cholesterol pathways. PMNS provided a convincing proof that maternal lipid metabolism during pregnancy is a major determinant of fetal growth and its body composition. We are analyzing relevant polymorphisms in various genes, which affect circulating concentrations of lipids, their deposition in (ectopic, i.e. non-adipose) tissues, and growth and body composition of the offspring. Preliminary results on the relationship between APOAV polymorphisms and maternal circulating triglyceride concentrations and fetal growth are really exciting and will be discussed during the meeting.

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