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## From MMNP to Project SARAS – Lessons learnt

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Mumbai Maternal Nutrition Project (MMNP) was conceived as a randomised controlled trial of intervention consisting of foodbased micronutrient at least three months before pregnancy and during full pregnancy period in ever married women in slums with the primary outcome of increasing the weight at birth by at least 100 Gms. The supplement consists of GLVs, Milk and Fruit, dehydrated at ambient temperature and converted into an eatable snack supplement.

It was thought that on completion it would give a practicable cost effective public health intervention for use in prevention of CHD and NIDDM in adult life since the trial is food based without any pharmacologic agent. The required sample would be 1500 women including control who would have eaten the food supplement at least thrice a week for at least one year.

Since there were no models available for such a prolonged and extensive trial in urban slums the team decided to take up a pilot study to explore pros and cons of methodologies at every stage from conception to implementation and analysis. The pilot study was conducted in Saitan Chowky slums in the field area of Streehitkarini, a 40 year old women's voluntary organization with an infrastructure of a band of experienced local health workers and a central co ordinating office within the area itself.

This has been done in last two and half years.

Lessons learnt in this pilot study will be used in the full scale trial to be conducted in Bandra East area where a model of Human Development in Urban Slums using Health Clinics as Entry points (WIN Project) is already active. WIN has twenty modular clinics each catering for 1000 families in slums in Bandra, Khar and Santacruz East, in Mumbai providing integrated services.

**The lessons** learnt consist of

1. Change in basic approach from soliciting eligible women after a census done by the health workers to join the trial voluntarily **TO** holding community awareness meetings in slum involving entire community including men, and women who are opinion makers and local social leaders, followed by focus group meetings in each lane to acquaint all women about the Project SARAS. An appeal is made to women to come forward to register in the trial of their own free will and as willing volunteers instead of canvassed subjects.

This will increase reduce Non compliance rate since each and every volunteering woman will be aware of her privileges and responsibilities in the trial fully from the management team itself.

2. Shift in focus of motivation to participate in the trial from eating of Supplement ( and invariably its taste etc) and the Newborn growth **TO** better preparedness for pregnancy including the need to decide the appropriate time for becoming pregnant so that a thorough physical and psychological preparation is undertaken by the couple and the family.( this would ensure that women volunteer to eat the supplements far earlier than they would otherwise done.
3. In the Pilot Project,each Health worker had a dual responsibility of motivating women as well as distributing and keeping record of supplement consumed every day. Non compliance ( for whatever reason ) used to cause some heartburn amongst both the worker and the subject since they knew each other well having worked in the area for many years.The most embarassing situation was when they were told to eat in front of the worker for precise record due to project being a research and not a service project.This would jeopardize the very faith the subjects had in the health worker. This approach has been changed in Project SARAS **TO** appointment of Project clerks (young girls educated upto 12th std and appropriately trained) for day to day fixed time distribution and insistence on eating the supplement in their very presence for precise recording of consumption.Thus health workers remain free both for initial and follow up motivation as well as sympathetically going into reasons for non compliance when reported by the Project clerks.
4. Two channels of supplements have been modified **TO** four channels ( coloured codes ) two for subjects and two for controls so as to improve allocation concealment.
5. From starting supplementation at all 20 centres at the same time, **TO** a staggering pattern has been established where a modular format of Community motivation,Focus group motivation,Road show (of registration,eligibility, Anthropometry,Food Frequency questionnaire filling ) and starting of supplements followed serially in centres one after the other.This will identify local roadblocks and pitfalls in each of the areas undertaken as well as lessons from the centres where this has serial activity has taken place.
6. From manual recording on ledgers **TO** a strong possibility is nearly ready for take off to use a SMART CARD with touch technology for fast and accurate recording of both the data of the subject,compliance pattern and monitoring of implementation of the projects.

In addition to these major modifications many small improvements are expected to improve the taste,size of supplement, smooth distribution , better compliance which will lead to success of Project SARAS in its full scale.



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## Project SARAS – A new Beginning

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Intra-uterine growth retardation (IUGR) is an important cause of neonatal mortality and impaired post-natal growth in developing countries. The risk of IUGR increases in undernourished mothers. The Mumbai Maternal Nutrition Project (MMNP) better known as project SARAS is a randomised controlled trial is to develop an evidence-based nutritional intervention to reduce low birth weight in slums of Mumbai.

Learning from experience of the pilot project in Streehitkarini, the project SARAS has started with new ethos of voluntary participation. The project is based on the already existing structure of "WIN" another project run by CSSC consisting of 20 modular clinics within the slums. Big community meetings with local Community Based Organizations (CBOs) has been organized in each center to get community approval and participation. These meetings focus on profile of CSSC and its activities undertaken by it including this project. Active community members are identified who could help in sensitizing the community. Smaller meeting follows with prospective eligible women to explain about details of the project i.e. need for supplementary food before and during pregnancy, eligibility criteria, how they can join, expectations from the participant and what they can expect from this project. Brochures containing all the information including date and place and time of registration were distributed. The registration camps are organised for 1 week at one place before moving to another area. In the camp, a simple questionnaire is filled up to determine their eligibility. A written informed consent is taken before registration, anthropometry is done and a detailed food frequency questionnaire is administered. A photograph is also taken for making identity cards. A fixed day weekly camp will continue to register the eligible women who wish to join the project on a later date.

We assume daily consumption of a micronutrient-rich food supplement for at least three months prior to conception, and throughout pregnancy, will increase the birth weight at least by 100gm. In order to achieve a sample size of 1500 deliveries based on birth weight approximately 4000 women will be recruited over period of 3-3.5 years. Recruitment into the study will continue, with an ever-expanding base population, until the target numbers are reached. The other parameters we want to observe are improved maternal micronutrient status, reduced maternal infection, reduced fetal loss (miscarriages and stillbirths), improved neonatal body composition and reduction of infant mortality.

The supplementation is given six days a week. Four snacks of supplements, two of which (the intervention group) have green leafy vegetables (GLVs), fruit and milk powder incorporated.

Currently registration of the eligible women is going on in the area and supplementation has already started from 23<sup>rd</sup> January 2006 in Ambedkar Nagar.

The project is currently supported by The Wellcome Trust, The Parthenon Trust, ICICI Bank, USAID and International Life Sciences Institute (ILSI), USA



## Antenatal multiple micronutrient supplementation, birth weight, and infant survival in Nepal

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Two recent randomized trials showed that antenatal multiple micronutrient supplementation increased birth weight in Nepal. Given that the primary aim of supplementation would be to improve infant survival, we examined mortality in the trials. In both cases, multiple micronutrient supplementation did not appear to be associated with reduced neonatal mortality. Indeed, there were non-significant associations with increased neonatal mortality relative to the iron-folic acid group. We therefore pooled the data from the two studies to examine differences in perinatal and neonatal mortality. Our combined data (n=2860 births and n=2751 live births) reveal an increase in perinatal (RR=1.36, 95% CI: 1.02, 1.81) and neonatal (RR=1.52, 95% CI: 1.03, 2.25) mortality associated with multiple micronutrient relative to iron-folic acid supplementation. This finding raises important public health questions and calls for further study.



## Effect of maternal ALA supplementation at marginal protein levels on blood and brain fatty acid profile in offspring

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There is growing interest in the role of essential fatty acids, especially n-3 fatty acids, in promoting fetal growth. Our earlier work has shown that maternal supplementation of fish oil (EPA: Eicosapentaenoic acid and DHA: Docosahexaenoic acid) at marginal protein levels (12%) not only supports growth and cognition but reduces risk of non communicable diseases in pups at adult age. The present study examined whether alpha linolenic acid (ALA), the primary vegetarian dietary omega-3-source and precursor to cellular membrane phospholipid EPA and DHA, can improve birth outcome.

Pregnant dams from 3 groups (7 in each) were fed : control diet (18% protein with 7% soybean oil, normal ALA), or one of two treatment diets at a marginal protein level of 12%; one with 7% soybean oil (SOD, normal ALA), and the other with 3% flax oil and 4% soybean oil (FSD, 4 times normal ALA) during gestation.

There was a significant ( $p<0.05$ ) reduction in both litter size and litter weight at birth in the FSD group as compared to the Control and SOD groups. Flax oil supplementation significantly increased ALA ( $p<0.05$ ) and EPA ( $p<0.05$ ) and reduced AA levels ( $p<0.05$ ) in gastric milk at birth as compared to SOD group. Blood fatty acid profile in pups at the end of lactation also showed significant ( $p<0.05$ ) increase in ALA, EPA levels and lower AA levels. Brain fatty acid levels showed noticeable reduction ( $p<0.05$ ) only in DHA levels in dams but not in pups, from both FSD and SOD, at the end of lactation.

The results suggest that maternal supplementation with flax oil at marginal protein levels did not show beneficial effects either for fetus or for pups. The fact that these effects were seen even after shifting the dams on control diet after delivery indicates the sensitivity of fetus and pups for excess levels of ALA.



## Maternal body water estimation by bioelectrical impedance in pregnancy

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**Background.** Fetal growth is influenced by parental size, maternal parity, gestational age and maternal weight gain during pregnancy. Physiological expansion of body water in pregnancy is directly associated with fetal growth and it confounds measurements of circulating metabolic, nutritional and endocrine mediators which also affect fetal growth. Bioelectrical impedance assessment of body water in pregnancy has been reported to be related to offspring size at birth.

**Methods.** Women with singleton pregnancy who were booked for delivery at KEM Hospital Pune (n=95) and at Vadu rural hospital (n=105) were enrolled in the study for evaluation of nutrition, activity, and biochemical parameters at 17, 27 and 33 weeks gestation. Fetal growth was assessed by serial ultrasound. Body water was measured by bioelectrical impedance analysis on the multi- frequency meter (Bodystat). At delivery cord blood was collected for biochemical and hematological measurements and placental weight was measured. Anthropometry of the newborn and mother was done after delivery.

**Results.** Up till November 2005, 200 women were studied, of whom 173 have delivered live infants. Mean age was 22.6 years, mean weight was 48 kg at enrollment and 70% were primigravid. The mean total body water was significantly higher in rural women compared to urban women at each of the 3 visits – 51.1 v 48.3 litres at 17 weeks, 49.7 v 46.9 litres at 27 weeks, and 49.6 v 46.7 litres at 34 weeks gestation. All further associations were studied adjusting for gestational age and location of the patient. There was no significant relation between total body water and parity, maternal age, maternal hemoglobin concentration, haematocrit, MCV, or any metabolic parameter except fasting glucose at 17 weeks gestation ( $p=0.02$ ). Pulse rate was not related but blood pressure ( $p=0.05$ ) was directly related to total body water at all the visits. Body water predicted birth weight ( $p=0.02$ ) and length ( $p=0.01$ ) but not skin folds or any other anthropometric measurements.

**Conclusion.** Maternal body water is related to aspects of fetal growth. This association needs further investigation.



# Principal Component and Conditional Independence analyses applied to a study of maternal micronutrient status and insulin resistance in the offspring (The Pune Maternal Nutrition Study)

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Data from a sample of 700 women and their children taking part in the Pune Maternal Nutrition Study was used to investigate associations between maternal micronutrient concentrations, especially vitamin B12 and erythrocyte folate, and insulin resistance in the offspring as measured by HOMA. Results are described fully elsewhere; but the main finding was that, in separate regressions, lower maternal vitamin B12 status (18 weeks;  $p=0.03$ ) and higher maternal folate (28 weeks,  $p<0.001$ ) predicted increased insulin resistance in the children.

These associations (and others) were also investigated using Principal Components Analysis (PCA) and Conditional Independence Analysis. PCA was used to condense the information contained in large groups of variables into a small number of 'component' variables. The relevant Principal Components were then included simultaneously in a Conditional Independence Analysis diagram, in which pairs of variables that are significantly correlated are connected with lines. Each correlation was adjusted for all other variables in the model; these included maternal social factors, size, body composition, diet and nutrient status, and offspring size at birth and 6 years.

The PCA of maternal micronutrient status produced a component, interpretable as "low maternal vitamin B12 and high maternal folate", which was significantly and positively correlated with insulin resistance in the child, even after adjustment for other variables ( $p<0.001$ ). Thus the approach confirmed this association, together with other biologically plausible associations demonstrated by the original regression models. In this talk the above techniques will be explained in detail and the value of such an approach compared to that of standard regression techniques will be discussed.



## Plasma vitamin B-12 and homocysteine levels in non-pregnant Indian women

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**Background:** Vegetarianism is common in India and associated with vitamin B12 deficiency and hyperhomocysteinemia. There is little information on vitamin B12 status in Indian women.

**Aim:** To investigate the prevalence of vitamin B12 deficiency in rural and urban Indian women living in and around Pune.

**Method:** We studied 146 women (48 rural, 48 slum dwellers and 50 urban middle-class residents, mean age 34 years). Data on nutrition, physical activity, socio-economic status and medical history were obtained. Circulating levels of Vitamin B12, total homocysteine (tHcy), hematological indices and cardiovascular risk variables were measured on fasting plasma samples.

**Results:** Median plasma vitamin B12 concentration was 127 pmol/l and plasma homocysteine concentration was 9.5 micromol/l . Sixty-one percent of the women were vitamin B12 deficient (<150 pmol/L) and 15% had hyperhomocysteinemia (>15 micromol/L). One third of the women were lactovegetarians; they had higher total homocysteine concentrations ( $p<0.01$ ) than those who ate non-vegetarian food. The frequency of non-vegetarian food consumption was associated with vitamin B12 ( $r=0.29$ ,  $p=0.000$ ) and tHcy concentrations ( $r= -0.29$ ,  $p=0.001$ ). Slum residents had the highest plasma vitamin B12 concentrations. These women's husbands had comparable vitamin B12 status but had considerably higher tHcy concentrations.

**Conclusion:** Low vitamin B12 status is common in Indian women particularly in urban middle-class. Despite this they have lower plasma tHcy concentrations compared to their husbands. This suggests a gender related difference in the vitamin B12- tHcy relationship.



## Follow up Studies in Pune Urban Children's Cohort Physical and Sedentary Activities in Adolescents

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**Background:** Sedentary lifestyle is now recognized as one of the most important factors in the increasing prevalence of obesity in urban areas.

**Objective:** This study was designed to assess the physical activity and inactivity in adolescent (16-17 years) urban Indian children and their association with adiposity.

**Methodology:** A sub group of 141 children (70 girls and 71 boys) from the Pune urban cohort of 477 were included. Their mean age was 16.5  $\pm$  0.66 yrs. Detailed anthropometry (weight, height, waist circumference, skin-folds) was carried out and dietary information was collected during their annual follow up visit. Socioeconomic status was assessed using Kuppuswamy scale. For assessment of physical activity, a questionnaire was designed to collect information regarding physical and sedentary activity in educational institutes, during commuting and at home. In addition, one-day physical activity recall (every 15 minutes) was calculated by interviewing these children. Daily total energy expenditure (TEE) was calculated using physical activity ratios (PAR) and estimated BMR using standard methods. Average activity energy expenditure (AAEE) was estimated from TEE and sedentary activity energy expenditure (SAEE; sleeping, idle sitting and standing). These activities were then graded as light, moderate or vigorous depending on the AAEE and the total duration spent on these activities during the day.

**Results:** *Physical activity in schools and colleges:* Out of 141 children, 5 (4%) were school drop outs and 136 (96%) were studying either in schools (n=65, 68 %) or colleges (n=31, 32%). 37% of the schools and 80% of the colleges had no timetabled PT classes.

*Physical activity during commuting (transport):* 65 (46%) children used cycling or walking. The rest used vehicular transport.

*Patterns of activity:* The mean number of hours spent in various activities were: exercise: 1  $\pm$  0.75 hours, school/ college: 5.4  $\pm$  1.4, tuitions: 3.7  $\pm$  1.8, TV: 2.3  $\pm$  1.9, sleep: 8.4  $\pm$  0.9 and other sedentary activities: 3.2  $\pm$  0.43. Proportions of average energy expenditure per week in vigorous, moderate and light activities were 24%, 48% and 28% respectively (boys) and 4%, 14% and 82% (girls). 20 % of boys and 75% of girls were not doing the minimum WHO recommended physical activity (at least 30 minutes of moderate activity per day)

Exercise hours correlated inversely with sum of skinfolds ( $r = -0.26$ ,  $p = 0.00$ ) and waist circumference ( $r = -0.29$ ,  $p = 0.001$ ). Mean time spent watching TV was 2.3  $\pm$  1.9 hours and

correlated directly with BMI ( $r= 0.236$ ,  $P= 0.002$ ), weight ( $r= 0.180$ ,  $P= 0.033$ ) and subscapular skinfolds ( $r= 0.215$ ,  $P=0.011$ ). Multivariate analyses showed that more TV hours ( $\beta =0.184$ ) and less exercise hours ( $\beta = -0.240$ ) predicted higher waist circumference ( $R^2=9.0\%$ ) and BMI.

**Conclusions:** Physical activity was grossly inadequate in more than 50% of children. Amongst the associations between diet and adiposity, and sedentary habits and adiposity, sedentary habits remained the most important determinant of central and overall adiposity.



# Prevention of lifestyle-related disorders in school children; Proposed study at Symbiosis School, Pune Implementation, problems and solutions

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## Hypothesis

- It is possible to reduce the prevalence of childhood obesity by a school based intervention program.
- It is possible to change life style parameters (e.g. T.V. viewing, physical activity, change in diet) by a multifaceted intervention program.

## Proposed Protocol

School (III and IV standards)

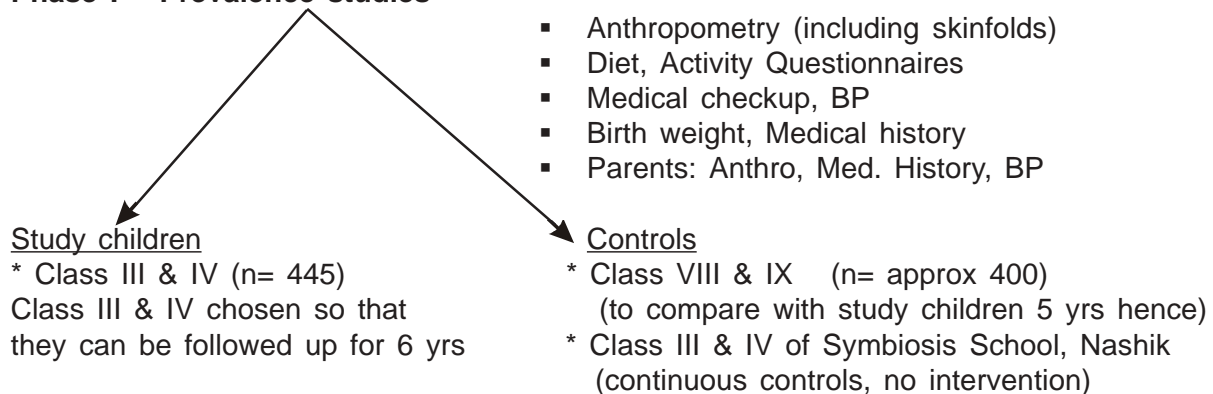
Intervention x 5 years

Outcome measures

## Outcome variables

- BMI, Waist circumference, skinfolds,
- Physical Activity Index, TV viewing,
- Fat, fruit or vegetable intake,
- BP

## Phase I – Prevalence studies



- \* Protocol Meeting *Apr 2005*
- \* Staff Recruited *Aug 2005 (4 Research Assts, 1 Nutritionist, 1 Sports Consultant)*
- \* Screening of study children *Sept to Dec 2005*
  - 15% absent on given date.....*Extra check up dates ?*
  - 77 % mothers & 32% fathers checked.....*Home visits for the rest ?*
  - Questionnaires..... *Will need validation.*
- \* Fitness tests ... *modules prepared by Sports consultant.*
- \* Study of Class VIII & IX ...*require consent for sexual maturity rating (SMR)*  
Nashik controls .....*logistic difficulties*

## Phase II – Intervention Studies (Class III and IV)

1. Health education – Diet, Nutrition, Life styles, Obesity
  - A. Demo / lectures once a week (invited speaker) – Saturday activity 1hr./week
  - B. Incorporate In Science Class (Class Teacher) - 10-15 minutes / daily.\* Problems: Children not interested; teachers too busy for this training.
2. Physical activity / games – 1 hour daily.  
\* Problems: As of now, only two periods per week; ground insufficient  
Sports Specialist – to design special modules.
3. Health & Physical activity – scoring subject  
\* Problem: School has no authority for this.
4. Mid day meal programme – Nutritional modifications  
\* Problems: Finicky tastes of children; only 70% of study group take mid day meal.  
(Many suggestions given by parents to be discussed with dieticians / organizers / contractors, for e.g. uniformity with tiffin meals).
5. No hawkers
6. Newsletter for parents and for children once a month  
(Need to appoint editor and editorial board)
7. Health / Activity day - Saturday morning 2 hrs.  
\* Problems: Place ?; 40% of children already involved in activities Saturday mornings.  
To start Jan 2006
8. Identified overweight and obese children for intensive monitoring, weight monthly, visit to Dietician.  
\* Problem: Children should not be targeted; parents may not come for counseling.
9. Psychometry / Behaviour tests: to be planned next academic year. *Time allotment* by school.

### Methodological / Implementation difficulties

- Co ordination between KEM, Health Centre, School, Sports etc....
- Decentralization of authority!
- Parental co operation, obsession with academics
- School Holidays (nearly 5 months!)
- Sponsorships



# Physical activity and cardiovascular risk factors in young adults

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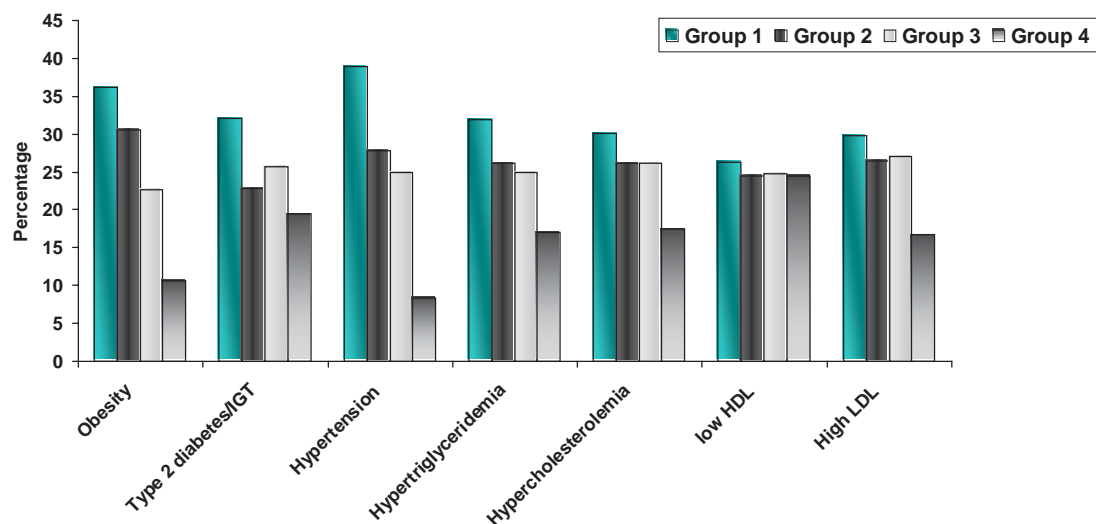
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**Background:** Regular physical activity reduces the risk of obesity, blood lipid abnormalities, hypertension, type 2 diabetes mellitus and coronary heart disease (CHD). Physical inactivity is widespread in India as a result of improved economic conditions and increased mechanization. In this paper we examine the relationship between physical activity and cardiovascular risk factors in young adults.

**Methods:** 2,218 men and women (mean age 28 years) were studied from a population-based birth cohort of 10,670 individuals born during 1969-73 in Vellore town and nearby rural areas. Family history, socioeconomic status, physical activity and tobacco and alcohol use were recorded by questionnaire. Subjects had oral glucose tolerance tests (WHO protocol) including measurements of plasma insulin and lipid levels. Physical activity scores were grouped into quartiles (groups 1 to 4) in men and women separately and were related to the prevalence of cardiovascular risk factors.

**Results:** Women had a higher median physical activity score (median 1858; IQR 1427-2415) than the men (median 1401; IQR 832-1875)( $p < 0.001$ ). The highest percentage of obesity (BMI > 25) ( $p < 0.001$ ), diabetes mellitus or impaired glucose tolerance ( $p = 0.01$ ), hypertension ( $p = 0.01$ ), hypertriglyceridemia ( $p < 0.001$ ), hypercholesterolemia ( $p = 0.04$ ), low HDL ( $p = 0.3$ ) and high LDL ( $p = 0.03$ ) were seen in the lowest quartile of the physical activity score in men. In women, the prevalence of risk factors were similar in all the four groups except obesity ( $p = 0.03$ ) and low HDL ( $p < 0.001$ ).

Prevalence of cardiovascular risk factors among physical activity groups in men



**Conclusion:** Our data points to the high prevalence of cardiovascular risk factors in men who are less physically active. Promotion of regular physical exercise will therefore be a useful public health intervention measure to reduce the risk of cardiovascular disease.



## Physical activity monitoring using accelerometers in Indian children: implications for future plans

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**Aim:** To measure physical activity using accelerometers, test the feasibility and validity of using accelerometers to measure activity, and test and develop interventions to increase activity levels in children.

**Methods:** Physical activity was measured in 105 children (offspring of diabetic mothers/ODM=31, controls=31, high subscapular group=24, low subscapular group=20) using accelerometers (CSA/MTI Actigraph) over 7-day periods. The activity for every 15 minutes was also recorded by their parents/teachers. Total Energy Expenditure (TEE) and Physical Activity Levels (PAL) were calculated from parental diaries using the 'factorial method', by allocating physical activity ratios to each activity based on a published compendium. A separate group of children (not part of the study) of similar age to the study children performed activities of different intensities within a span of one hour (set activity sessions), with Actigraph in place.

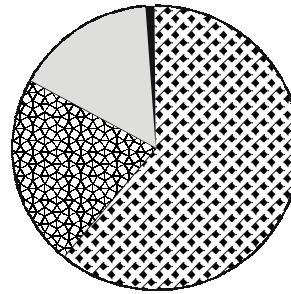
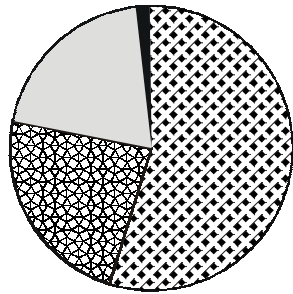
**Validation:** There were statistically significant correlations between Actigraph counts, and TEE ( $r=0.40$ ,  $P=0.007$ ) and PAL ( $r=0.44$ ,  $P=0.002$ ), which was similar to findings from other studies in children. Set activity sessions showed clear-cut differences in counts between activities of varying intensities, and a linear increase in counts with increase in intensity of the task. These counts were used to determine cut-offs for different levels of activity such as sedentary, light, moderate, and hard in the study children.





**Results:** There were no differences in activity measures between boys and girls. Female ODM tended to accumulate fewer counts ( $3.9 \times 10^5$  v  $4.3 \times 10^5$  in controls), and spent less time in moderate and hard activities than control girls. In contrast, male ODM had more counts and spent more time in hard activities than controls. Children with larger subscapular skinfolds at five years had fewer counts, shorter periods for all activities and more time spent on sedentary tasks (Figure).

Pie chart showing average time spent on different activities by girls in a day (1440 minutes, 24 hours)

Low subscapular group

High subscapular group



-  Sedentary
-  Light
-  Moderate
-  Hard

**Conclusions:** Our study showed an inverse association between objectively measured physical activity and body fat. Presently, we are collecting information on behavioural and social factors associated with physical activity in these children by administering questionnaires to parents and teachers. The main objective of our future follow-up is to attempt to achieve sustainable behavioural changes in physical activity in our children.



## **Developmental origins of osteoporotic fracture; the role of maternal vitamin D insufficiency**

*Cooper C*

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Osteoporosis is a skeletal disease characterised by low bone mass and micro-architectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture. The cumulative incidence of fracture from age 50 years is estimated at around 50% among white women and 20% among white men.

Preventive strategies against osteoporotic fracture can be targeted throughout the life course. Thus, modification of physical activity and dietary calcium/vitamin D nutrition in the elderly and during midlife, should complement high-risk approaches entailing appropriate measurement of bone mineral density and targeting of anti-resorptive and formation stimulating drugs.

Prevention of osteoporotic fracture can also be directed earlier in the life course. Environmental influences during early life interact with the genome in establishing the functional level of a variety of metabolic processes which are involved in the pathogenesis of osteoporotic fracture.

The evidence that osteoporosis risk might be programmed in this way stems from four groups of studies: (1) Epidemiological studies which confirm that subjects who are born light and whose growth falters in the first year of postnatal life, have significantly lower bone size and mineral content, at age 60 to 75 years; (2) Epidemiological cohort studies have demonstrated that subsequent lower trajectories of childhood growth are associated with an increased risk of hip fracture among such men and women; (3) Detailed physiological studies of candidate endocrine systems which might be programmed have shown that birthweight and growth in infancy alter the functional settings of the GH/IGF-1, and hypothalamic pituitary adrenal axes; (4) Studies characterising the nutrition, body build and lifestyle of pregnant women which relate these to the bone mass of their newborn offspring, have identified a number of important determinants of reduced fetal mineral accrual (maternal smoking, low maternal fat stores and maternal vitamin D deficiency, intense levels of weight-bearing physical activity in late pregnancy).

Follow-up studies of randomised controlled trials of vitamin D supplementation in infancy suggest persisting benefits in adolescence and young adulthood. These data suggest that undernutrition and other adverse influences arising in fetal life or immediately after birth have a permanent effect on body structure, physiology and metabolism, which might independently influence the later risk of cardiovascular disease and osteoporotic fracture.



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## BMI charts for screening for childhood growth associated with adult metabolic disease

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**Background:** Sustained accelerated childhood body mass index (BMI) gain (crossing centiles upwards) is associated with adult metabolic disorders. Current preventive efforts are primarily focused on obese or overweight children, identified from cross-sectional, 'distance' BMI charts. This strategy, often based on international definitions, ignores relative changes in childhood BMI, and is sub-optimal for developing countries undergoing nutrition transition. For example, young adult Indians with impaired glucose tolerance (IGT) or diabetes were not overweight or obese as children but were characterized by their high rate of BMI gain.

**Objective:** To develop BMI charts for routine use in children to recognize growth associated with adult metabolic disease.

**Methods:** We measured anthropometry, serum lipids and plasma glucose and insulin concentrations in 1492 men and women 26 to 32 years of age who had been measured at birth and at intervals of 3 to 6 months throughout infancy, childhood, and adolescence in a prospective, population-based study (New Delhi birth cohort). Age and sex specific BMI charts were constructed from the cohort data to determine BMI SD scores and the change in BMI SD scores between two or more ages during childhood. The sensitivity, specificity, attributable fraction, and positive predictive value for combinations of childhood BMI SD score and  $\Delta$ BMI SD score, for predicting adult Metabolic Syndrome\*, and IGT or diabetes, were assessed.

**Results:** Point estimates of BMI categories (2/3 SD bands) at ages 5 to 14 years were positively related to adult Metabolic Syndrome (adjusted ORs 1.18 to 1.87;  $P \leq 0.01$  for all). Point estimates of BMI were positively related to adult IGT/diabetes only after 12 years (OR 1.22 to 1.24;  $P < 0.01$ ). Gain in BMI SD score in 3-year intervals from the age of 5 years (5-8, 6-9,.....11-14 years) predicted adult Metabolic Syndrome (adjusted ORs 1.38 to 2.03;  $P \leq 0.001$ ), and IGT/diabetes (ORs 1.25 to 1.97;  $P$  0.06 to  $< 0.001$ ). The best 'screening test' was a combination of 1) an increase in BMI SD score in 3-year intervals between the ages of 5 and 14 years, and 2) a BMI SD score  $> 0$  at the later age of the 3-year interval. Based on BMI measurements at 11 and 14 years, this predicted adult Metabolic Syndrome with a sensitivity of 43%, specificity of 77%, attributable fraction of 21% and positive predictive value of 38%. Corresponding values for IGT/diabetes were: 37%, 72%, 11% and 19%. Similar predictions were obtained using charts constructed from the US-CDC and National Nutrition Monitoring Bureau (NNMB) reference data, with a suitable change in the screening criteria to allow for the larger BMI of US children and smaller BMI of NNMB children.

**Conclusion:** It is feasible to construct BMI charts to enable easy detection of children with accelerated BMI gain. Test criteria performed reasonably well for predicting Metabolic Syndrome in older children and across intervals >3 years, but predictions for IGT/diabetes were relatively poor.

\*IDF definition for South Asians: Waist circumference  $\geq 90$  cm (men),  $\geq 80$  cm (women) plus any two of: Blood pressure systolic  $\geq 130$  mmHg or diastolic  $\geq 85$  mmHg or treated hypertension; Triglyceride  $> 1.7$  mmol/l; HDL-cholesterol  $< 1.0$  mmol/l (men),  $< 1.3$  mmol/l (women); Fasting glucose  $\geq 5.6$  mmol/l or type 2 diabetes



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## Type 2 diabetes and early growth – lessons learned from Helsinki Birth Cohort Study

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**Background:** Both fetal, infant and childhood growth are linked to coronary heart disease (CHD). In general children who later develop CHD are small at birth and thin at around 2 years of age. Between 2 and 11 years of age they put on weight more rapidly than the other children. Low birth weight, thinness at 2 years and rapid gain in body mass index thereafter each contribute to the risk of later CHD. This pattern of growth is also related to insulin resistance in adulthood. Since type 2 diabetes is a well known risk factor for CHD one could assume that similar patterns of growth would be associated with impairment in glucose regulation.

**Study population:** Two birth cohorts consisting of 15846 individuals born at Helsinki University Central Hospital and who grew up in the city of Helsinki have been followed. The older cohort consists of 7086 individuals born 1924-33, with birth characteristics as well as childhood growth data (ages 7 and 15 years). The younger cohort born 1934-44 (n=8760) has detailed data on childhood growth from birth to 12 years of age. Information on growth has been collected from birth, child welfare and school records. Both cohorts have been followed up from 1971 onwards by register linkage to national Finnish registers providing information on both morbidity and mortality. A clinical examination of 2500 individuals has provided more detailed information on metabolic and genetic aspects and their associations with growth and adult health outcomes. In the present sub-study we examined 2003 subjects born in Helsinki from 1934-44. On average they had 11 measurements of height and weight between birth and 2 years of age and another 7 measurements between 2 and 11 years of age. We identified 311 subjects with type 2 diabetes and 496 with impaired glucose tolerance (IGT).

**Results:** Both impaired glucose tolerance (IGT) and type 2 diabetes were associated with low birth weight, after adjusting for current body mass index ( $p < 0.0001$ ). The risk of these conditions was further increased by low weight gain between birth and 2 years of age. A one standard deviation increase in weight at 2 years was associated with an odds ratio for either type 2 diabetes or IGT of 0.76 (95% CI 0.69 to 0.84). This effect was greatest in people who had low birth weight. The odds ratio of IGT or type 2 diabetes was 2.6 (95% CI 1.8 to 3.7), in people who weighed less than 3 kg at birth and less than 11.5 kg at 2 years of age compared with people who weighed more than 3.5 kg at birth and more than 12.5 kg at 2 years of age. Low growth in the first 6 months after birth was a critical period for the development of insulin resistance in later life; other critical periods were associated with slow fetal growth and rapid increase in body mass index between 2 and 11 years.

**Conclusions:** Low weight gain during infancy increases the risks of IGT and type 2 diabetes in adult life. The effect is greater in people who had low birth weight. The first 6 months after birth may be the most critical period for growth, in relation to development of glucose intolerance.



# Metabolic Syndrome in the Pune Urban Cohort; 4 years, 8 years and the proposed follow-up study at 20 years

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On behalf of the Department of Pediatrics and the Diabetes Unit, King Edward Memorial Hospital, Pune, India, and the Medical Research Council, Epidemiology Resource Centre, University of Southampton, UK.

In 1995 we studied glucose tolerance tests in 379 four-year-old children. These included 201 children admitted to the routine postnatal wards and 178 babies admitted to the neonatal intensive care unit. We reported an inverse relationship of birth weight with post glucose load 30 min glucose and insulin in the routine admission group. A similar follow up study of this routine admission cohort in 1999 at 8 years of age with additional 287 children (total n=477) demonstrated that children with the most adverse cardiovascular risk profiles were light at birth and heavy at 8 years. Of the components of body weight, higher fat mass was associated with an increase in all the risk variables. This cohort now aged 17-18 years has been followed annually for growth, anthropometry, sexual maturity (SMR, Tanner staging) for onset and completion of puberty. Skeletal age was done once at 12 years. Insulin resistance and central adiposity at 8yrs in non-obese urban Indian girls predicted earlier menarche. The results related to completion of puberty are awaited.

We propose that this cohort is restudied at 20 years of age to determine the relationship of birthweight, subsequent growth and development and insulin resistance in early adulthood as also tracking of insulin resistance variables from childhood to adulthood.

## Study plan:

- 1. To enroll the entire original cohort at 20 years of age.** This will include the 379 children from the 4 year study and 287 additional children included in the 8 year study. The following parameters will be studied –
  - Anthropometry
  - Insulin resistance, plasma lipids, blood pressure, SGPT, abdominal ultrasound of the abdomen (polycystic ovarian disease and hepatic steatosis), serum leptin, body composition (DEXA), inflammatory markers
  - Assessment of diet and physical activity
  - Assessment of stress level
- 2. To study the parents of the subjects.** These parents have already been studied when the subjects were 8 year old. The following parameters will be studied -
  - Anthropometry, blood pressure
  - Glucose tolerance test

**3.To enroll one sibling of each subject and study for the following**

- Anthropometry, blood pressure
- Glucose tolerance test (for discussion)

This will be the first study with longitudinal data on metabolic syndrome in a children cohort and with data on parents.



## **Association of stunting with high blood pressure levels among adolescent boys from different socio-economic classes**

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An association between low birth weight and adult hypertension has been reported in several populations. Studies examining early life undernutrition and risk of hypertension in adolescents are scarce. In developing countries like India, postnatal nutritional insults leading to growth faltering and stunting are seen in rural and poor communities. In order to investigate their long term effect we examined school children (9-16 yr) from high (n=935) and low (n=1146) socio-economic classes for anthropometry (weight, height, body mass index and body fat %) and blood pressure.

Mean values for weight, height, BMI and body fat percent were significantly ( $p<0.001$ ) higher for children from HSE class for all the age groups compared to those from LSE class. With regard to blood pressure levels, while mean SBP levels were higher in HSE class students, mean DBP levels were higher in LSE students in all the age groups. The prevalence of obesity (27.5%) was significantly higher among the HSE group compared to the LSE (1.7%) class. However the prevalence of high SBP was higher (10.5 % v 2.7 %) among HSE students while that of high DBP was higher among LSE students. In all tertiles of BMI, the prevalence of high DBP was significantly higher among students of LSE group than those in HSE group but this was not the case with SBP. In the case of children in the lowest tertile who were mostly stunted, the prevalence of high DBP increased as BMI increased in both socio economic classes but LSE group showed a steeper increase. Such a trend was not seen in case of SBP prevalence in these stunted students.



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## Life statistics, anthropometry and history

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Historians and economists (\*) have uncovered important resources that give remarkable insights into the recent cultural evolution of our species. Archives of anthropometric and health-related statistics compiled by scientifically minded physicians, public health administrators, army recruiters and veteran's health authorities have been used to address interesting questions about social change. Although such statistics are imperfect historical witnesses, they still provide uniquely revealing reports on the human condition at particular moments. They illustrate objectively the biological consequences of important but diverse historical processes ranging from economic modernisation, to colonisation, emigration and redistribution of wealth in the developed world.

Anthropometric studies reveal the severity of the penalties incurred by the poorest strata of pre-industrial society and of the new society that emerged during industrialisation and urbanisation before the implications of the germ theory of disease were appreciated (e.g. in Britain). Later industrialisations were evidently accompanied by almost continuous improvements in welfare at much lower biological cost. Secular trends in height within particular societies often reflect important changes in nutrition originating in economic circumstances. Times of food scarcity are often apparent in these statistics. In nineteenth century Britain we can chart the "hungry thirties and forties" and the consequences of the "Repeal of the Corn laws" in 1845. A slow upturn in the secular trend in stature in Britain originating in "free trade" had dire consequences in the Indian sub-continent. In land-rich countries that attracted European migrants (USA and Australia), stature initially made impressive advances that were partially lost in the mid-nineteenth century in response to deteriorating diets. Anthropometric data from Scandinavia shows these countries passed a milestone in the 1980s when differences in average growth rate between children from rich and poor homes almost disappeared, perhaps for the first time in the history of humans living in settled communities.

\* This talk is not based on any original research of my own but is a review, based chiefly on material from the following books:

**Robert William Fogel** (2004) *The Escape from Hunger and Premature Death, 1700-2100: Europe, America and the Third World*. CUP ISBN: 0521808782

**Roderick Floud, Annabel Gregory and Kenneth Wachter**. (1900) *Height, Health and History: Nutritional Status in the United Kingdom, 1750-1980* CUP: ISBN: 0521303141

**Richard H. Steckel and Roderick Floud** (1997) *Health and Welfare during industrialization*. Chicago. ISBN: 02206771563

**Mike Davis** (2002) *Late Victorian Holocausts: El Nino Famines and the Making of the Third World* Verso; ISBN: 1859843824

**Hans Waaler** (1984) *Height, weight and mortality. The Norwegian experience*. Acta Med Scand Suppl; 679:1-56.



## Short-term and long-term implications for the offspring of women with gestational diabetes

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There are multiple means to assess fetal growth such as birth weight for gestational age, adjusted for gender and ethnicity. However, in our studies of fetal growth, we have elected to employ estimates of body composition. At birth, the human of all mammalian species has the most amount of body fat, approximately 12-15%, in contrast to less than 5% for most laboratory species. Based on work by Sparks and others, fat free mass is believed to represent genetic factors, whereas, fat mass may be more representative of the intrauterine environment. Although fat mass only represents approximately 12-15% in total weight, it accounts for approximately 50% of the variance in fetal weight. In infants of women with gestational diabetes (GDM), there is a significant increase in fat as compared to fat free mass even though there is no significant difference in weight. Similarly, if one evaluates only those infants who are appropriate for gestational age or large for gestational age, there is a significant increase in fat in the infants of the diabetic mothers and a significant decrease in fat free mass in the infants of GDM mothers as compared to the normal glucose tolerant group.

Although there is an increased trend in obesity in the U.S. in adults, this can also be found in adolescents and based on our own birth weight data from Cleveland, there has been a significant 120g increase in birth weight over the last 30 years. Our strongest correlation with this increase in birth weight has been the 20 pound increase in maternal weight at the time of delivery. Hence, based on our stepwise regression analysis, maternal obesity rather than gestational diabetes has the stronger correlation in fat mass and percent body fat as compared with the other potential covariables such as weight gain in pregnancy and whether or not the woman had GDM.

In the long term (mean 9 years) follow up studies of women of infants with normal glucose tolerance and GDM, infants whose body composition was in the upper tertile at follow up, the mothers had a significant increase in BMI and history of GDM. These infants also had significantly greater systolic blood pressure, increased % fat in their diet, higher triglycerides, lower HDL and greater insulin resistance all consistent with the picture of early metabolic syndrome. Lastly, in the stepwise regression analysis at follow up, factors most strongly correlated with lean body mass included age at follow up, gender, followed by maternal pregravid BMI. In contrast, follow up fat mass correlated most strongly with pre-gravid maternal BMI followed by age at follow up and then percent carbohydrate in the diet. In summary, both maternal pregravid obesity and GDM are significant risk factors for obesity in the offspring of the woman with GDM both at birth and at the time of long term follow up.



## Determinants of maternal hyperglycaemia 6 years after delivery : Pune Maternal Nutrition Study

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**Background:** Gestational diabetes was rare in the young pregnant rural Indian women who took part in the Pune Maternal Nutrition Study (PMNS) and plasma glucose concentrations were relatively low. We have followed these mothers and children and studied their glucose tolerance 6 years later. This provides an opportunity to study the relationship between maternal glycaemia in pregnancy (in normal range) and the future risk of hyperglycaemia.

**Objective:** To study determinants of hyperglycaemia in mothers 6 years after delivery.

**Study Design:** The PMNS has information on the mother's pre-pregnant size, socio-economic status, education and her nutritional and metabolic parameters during pregnancy. An oral glucose tolerance test (OGTT) and other measurements were repeated 6 years after delivery.

**Results:** Of 770 mothers studied during pregnancy, 7 were hyperglycaemic during pregnancy (3 impaired fasting glucose-IFG, 3 impaired glucose tolerant-IGT, 1 Diabetic-DM). Of the remaining 763 mothers, 650 were followed up 6 years later at mean age of 27 years. Fifty-two (8 %) of them were hyperglycaemic. Hyperglycaemic mothers were shorter (leg length 72.0 v 74.0 cm,  $p<0.01$ ), heavier (48.0 v 44.0 kg,  $p<0.001$ ), and more adipose (DXA fat % 30.0 v 25.0,  $p<0.001$ ) compared to normoglycemic mothers. They also had higher levels of other cardiovascular risk factors (cholesterol, triglycerides, HOMA insulin resistance) and had gained more weight since pregnancy (6.0 v 3.0 kg,  $p<0.0001$ ). There was no difference in education and socio-economic status.

Before pregnancy (6 years ago), currently hyperglycaemic mothers were more centrally obese (waist circumference 62.8 v 60.5 cm,  $p<0.001$ ) and more adipose (sum of skinfolds 22.0 v 20.0 mm,  $p<0.001$ ) compared to the normoglycaemic mothers but there was no difference in parity, family history of diabetes and weight gain during pregnancy. During pregnancy, these mothers were physically less active (score 61 v 75,  $p<0.001$ ) and had higher blood pressure (115/64 v 112/62 mmHg,  $p<0.05$ ) and higher 2-hour plasma glucose concentration during OGTT (86 v 78 mg%  $p<0.001$ ). There was no difference in the incidence of pregnancy-related events (anaemia, pregnancy induced hypertension and caesarean section rate) in the two groups.

Children of hyperglycaemic mothers had higher plasma glucose concentrations (fasting 96 vs 90 mg%, 2h OGTT 110 vs 96 mg%) at 6 years compared to children of normoglycaemic mothers.

**Conclusions:** This is the first community-based study of incident hyperglycaemia in rural India. We found a substantial incidence of hyperglycaemia in young rural Indian women; this was predicted by short stature (leg length), lower physical activity and higher glycaemia in pregnancy, and higher weight gain after pregnancy. The predictive anthropometric and biochemical measures are far below any international 'risk factor' categories. These findings have implications for strategies to prevent maternal hyperglycaemia in the Indian subcontinent.



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## Gestational diabetes and the prevalence of diabetes 5 years after the index pregnancy in south Indian women

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It is known that the women with gestational diabetes mellitus (GDM) are at an increased risk of developing type 2 diabetes later in life. We report here the prevalence of diabetes five years after delivery in a group of south Indian women with or without GDM.

During 1997-1998, GDM status was determined (Carpenter and Coustan criteria) for 785 consecutive pregnant women attending the antenatal clinics of the Holdsworth Memorial Hospital (HMH), Mysore. Of the 639 (41 women with GDM) women who delivered at HMH, 526 (35 GDM women) were available for follow-up 5 years after the index pregnancy. At follow-up, the women underwent an oral glucose tolerance test; diabetes was determined by the WHO criteria and the metabolic syndrome was defined by the IDF criteria recommended for south Asian women.

37% (N=13) of GDM and 2% (N=8) of non-GDM women had diabetes at follow-up ( $P<0.001$ ). Metabolic syndrome was present in 60% of the GDM women (waist-hip ratio/WHR 0.91, systolic blood pressure/SBP 117 mmHg, diastolic blood pressure/DBP 70 mmHg, triglycerides 141 mg/dl, HDL 42 mg/dl, fasting glucose 7.7 mmol/l) compared to 26% in non-GDM women (WHR 0.89, SBP 108 mmHg, DBP 66 mmHg, triglycerides 102 mg/dl, HDL 44 mg/dl, fasting glucose 5.4 mmol/l). The differences (diabetes, metabolic syndrome, SBP, DBP, triglycerides, fasting glucose) remained statistically significant independent of current BMI.

GDM women with diabetes had lower insulin concentrations (area-under-the-curve/IAUC,  $P<0.05$ ) at the index pregnancy, and significantly lower insulin increment and higher insulin resistance (HOMA) at follow-up than the rest of the GDM women. They were also more centrally obese (WHR  $P=0.04$ ) than normal glucose tolerant (NGT) women.

Irrespective their GDM status, family history of diabetes in a first-degree relative (OR=4.9,  $P<0.001$ ), high current BMI (OR=1.3,  $P<0.001$ ) and low gestational IAUC (OR=0.999,  $P=0.02$ ) were independent predictors of diabetes in all women. Age, socio-economic status and height did not predict diabetes independent of other risk factors.

Our findings suggest a high cardiovascular risk in women with a history of GDM. Meticulous follow-up of these women after delivery may be crucial to identify a large proportion of women at risk who need more frequent follow-up, and may provide scope to modify lifestyle factors.



## **Effects of Intrauterine Growth Retardation on the development of beta cells and other pancreatic endocrine cells; an autopsy study of human neonates**

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Insulin, acting as growth factor produced by fetal pancreas is of prime importance to fetal growth. Relation of IUGR and Type II Diabetes is well established. How far this is related to impaired development of pancreatic Beta cells is not clear. Development of endocrine pancreas occurs mainly in third trimester of gestation and continues for about three months of postnatal life. So we planned to study pancreas from neonatal autopsies.

- 1) Purpose of this study is to find relationship of birth weight, body parameters and organ weights to gross and microscopic morphometry of pancreas.
- 2) Estimate number of Beta cells and other endocrine cells of pancreas. Compare the data of IUGR babies with normal weight babies

We carried out prospective study of 61 consecutive neonatal autopsies at KEM Hospital Mumbai. The cases were full term neonates dying within 7 days of births and full term fresh stillbirths. Cases with major malformations were excluded. Birth weights and anthropometric measurements were recorded on day 1 of birth. Weights of various organs were recorded at autopsy. Data of all these cases is analyzed.

Of the 61 cases, 32 were female and 29 male. Fourteen were stillbirths. Mean birth weight was 2.43 kg. Thirty two babies weighed below 2.5 kg. Our earlier study on kidneys of these cases, using computer based image analysis system revealed low nephron count in IUGR babies as compared to normal weight babies. Pancreas weight and size was recorded. Sections from head, body and tail region were paraffin processed. Paraffin blocks are preserved. We plan immunoperoxidase staining 'Chromogranin' for quantitative estimation of endocrine cells and 'Insulin' for estimation of Beta cells. Antibodies for detection of glucagon secreting cells and polypeptide secreting cells are also available. Sections stained for specific endocrine cells will be used to study by computerized image analysis procedure. Image analysis equipment is available at Anatomy department of KEM Hospital.

Some of the paraffin blocks, preserved for more than five years appear damaged due to weather. It is difficult to get autopsies on full term babies, so we are adding few more cases which include premature babies 32 wks onwards.



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## Adiponectin and leptin concentrations in cord blood: a spectrum of adipocyte function in Indian and white caucasian newborns

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**Background:** Indians have a higher body fat percent (adiposity) compared to white Caucasians despite a smaller body mass index. We have reported higher cord leptin concentrations in Indian babies compared to white Caucasian babies. There is little information available on adiponectin in cord blood.

**Objective:** To compare adiposity and cord plasma hormonal concentrations (leptin, adiponectin and insulin) in newborns of Indian normal glucose tolerant (NGT) and gestational diabetic (GDM) mothers and those born to white Caucasian normal glucose tolerant (NGT) mothers.

**Methods:** We measured plasma adiponectin, leptin and insulin concentrations in cord blood of babies born to 161 NGT and 108 GDM mothers in Pune, India and 78 babies born to NGT white Caucasian mothers in London, UK. Mothers and babies were measured soon after delivery.

**Results:** NGT Indian mothers were the youngest, shortest, lightest and the least adipose. GDM Indian mothers were the most adipose. Indian babies were lighter and shorter than the white Caucasian babies. Those born to NGT mothers were the least adipose.

Cord plasma leptin levels were higher in the babies of GDM mothers compared to babies of NGT mothers (median 11.6 ng/mL v 5.6 ng/mL,  $p < 0.05$ ) who were similar to the white Caucasian babies (6.4 ng/mL). Cord plasma adiponectin levels were lower in the babies of NGT mothers compared to those in babies of GDM mothers (18.3 mg/L v 21.6 mg/L,  $p < 0.05$ ) who had levels similar to those in white Caucasian babies (25.3 mg/L). In each group leptin concentrations were related to different measurements of neonatal size, while adiponectin levels were related to all measurements except length. When adiponectin concentrations were expressed as a ratio of leptin concentration they were lowest in babies of GDM mothers and highest in white Caucasian babies (Indian NGT 3.0, GDM 1.9, white Caucasians NGT 3.6). Cord plasma insulin concentrations were similar in all the three groups (Indian NGT 5.0  $\mu$ U/L, GDM 6.4  $\mu$ U/L, UK NGT 3.2  $\mu$ U/L).

**Summary:** Indian babies have lower adiponectin concentration in cord blood plasma compared to those in white Caucasian babies. Babies born to GDM mothers have the highest leptin concentrations but lowest adiponectin concentrations. This spectrum of adipocyte function and insulin sensitivity at birth may predict future risk of T2D.



## **Building human capacity in India : the role of biomedical and social science research**

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The urgent need to dramatically improve the public health scenario in India, a country which currently suffers an even greater proportion of the world's disease burden than its 16.8 percent share of the global population can hardly be overstated. This is not only motivated by the belief in health as a fundamental right, but by the understanding that health is a fundamental human capacity that mediates an individual's ability to participate in social, economic and political processes. Public health therefore is a critical dimension of human development and an integral aspect of efforts to reduce poverty and advance a more participatory socio-economic order.

In India, the persistence of a very high incidence of preventable mortality and morbidity, especially the extremely poor health and nutritional status of women and children, demands significantly greater investments in public health and much more effort in the *creation and conversion* of evidence into effective and scaled impact on health outcomes. It is here that public health research has an important role to play and where the capacity to carry out such deeply relevant work within the country currently falls very short. This is not, however, due to a lack of potential within India, where the ability to undertake sophisticated research, especially in the clinical and biomedical domain but even in community-based settings has been amply demonstrated. Rather, it reflects a culture and political-economy of research, both internationally and within the country that has not sufficiently harnessed, developed and positioned the resources to apply interdisciplinary research talent to address the complex and multifaceted determinants of health operating within the households where health is produced and within the health systems designed to serve them.

Good quality research can and must, however, be generated to continuously address critical knowledge and practice gaps to advance innovation in and improve implementation of public health programmes. Such research cannot be viewed as an indulgence in resource-poor settings, but needs to be at its most creative and relevant in precisely those contexts.

Drawing on the analysis and experience emerging from some recent public health research sites and projects, this presentation will try to identify some of the critical constraints in the construction and translation of public health research in the Indian context with a view towards defining directions for strategic collaboration in policy and practice-oriented research.



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# POSTERS

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# Maternal intake of calcium rich foods and circulating micronutrient status during pregnancy predict bone measurements of the offspring; the Pune Maternal Nutrition Study

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**Background:** Bone measurements in offspring are thought to be determined by both genetic factors and the post-natal environment. Recent evidence suggests effects of the pre-natal environment, including maternal nutrition. The Pune Maternal Nutrition Study, a population-based longitudinal study in rural Western India studied maternal nutrition during pregnancy and bone measurements of the children.

**Aim:** To study the relationship between maternal size, nutrition and Bone Mineral Density (BMD) of the offspring at 6y.

**Methods:** We measured maternal anthropometry, nutritional intake, physical activity and circulating nutrients in 797 pregnant women at 18 and 28 weeks gestation. Babies were measured in detail at birth and, infant feeding and weaning practices were recorded. We measured body composition and BMD (Total body, Dual Energy X-ray Absorptiometry) in 695 children (91% of survivors) and their parents, 6y after the delivery.

**Results:** The children's mean height and weight were 109 cm and 16 kg. Total body BMD was 0.78 g/cm<sup>2</sup> in boys (n=369) and 0.77 g/cm<sup>2</sup> in girls (n=326). Children of mothers who consumed milk or milk products frequently (2-3 times /day) had higher BMD (0.79 g/cm<sup>2</sup> Vs rare consumers (<2 times/ month) 0.78 g/cm<sup>2</sup>, p<0.01). Higher maternal erythrocyte folate concentrations at 28 weeks gestation and lower plasma total homocysteine concentration during pregnancy predicted higher BMD in the children (p<0.05 both). Lower maternal parity, higher body fat mass and lower physical activity during pregnancy predicted higher BMD of children (p<0.01 for all). Maternal habit of chewing tobacco was not related to the children's BMD. The child's birthweight, length and placental weight, and current age, height and weight were positively correlated with BMD (p<0.001 for all). Duration of breast-feeding was not related. The height and BMD of both parents and socio-economic status were positively correlated with the BMD of the children (p<0.01 for all). In multiple regression analyses, higher maternal frequency of intake of milk and milk products at 28 weeks (<0.001) and BMD of both parents (Regression slope: Mother 0.21 (95% CI 0.16 to 0.25), father 0.14 (95% CI 0.10 to 0.17) p<0.001 for all) were independent predictors of the child's BMD. Maternal consumption of milk and milk products accounted for around 2% of the total 40% variance.

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**Conclusion:** A sizeable proportion of the BMD of children is determined by 'environmental' factors, with a potential for modification. Higher maternal intake of calcium-rich foods during pregnancy and higher folate and lower homocysteine status predict higher BMD in the child. Intrauterine life is important in determining the bone health.



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# Maternal Nutrition and Paternal Size Affects Age at Adiposity Rebound

## The Pune Maternal Nutrition Study (PMNS)

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**Background:** An early age at adiposity rebound (AR) is a risk factor for later obesity and type 2 DM. It is not known what maternal and paternal factors influence AR in the offspring.

**Objective:** To investigate maternal and paternal factors influencing adiposity rebound in the offspring.

**Study Design:** The PMNS database has information on maternal pre-pregnancy characteristics and her food intake, physical activity and circulating levels of nutrients and metabolites during pregnancy. We also have information on details of breast-feeding and paternal size and socio-economic status. Children were measured serially from birth every 6 months, at last follow up they were 10y old. At 6y body composition was measured by DXA. We defined age at adiposity rebound by age of lowest BMI between 1 and 10 years.

**Results:** The average age of adiposity rebound was 6.6 years (sd 1.8), and was similar in boys and girls. Maternal pre pregnant size and weight gain in pregnancy were not related to AR, nor was maternal intake of macronutrients and frequency of micronutrient rich foods (green leafy vegetables, fruits and dairy products). Higher maternal red cell folate and plasma homocysteine concentrations predicted early AR (standardized  $\beta$  = -0.13,  $p < 0.01$  and standardized  $\beta$  = -0.14,  $p < 0.01$  respectively) but plasma vitamin B12, ferritin, vitamin C concentrations were not related. Lower maternal physical activity during pregnancy predicted early AR (standardized  $\beta$  = 0.08,  $p < 0.05$ ). Birth size measurements and period of exclusive and total breast feeding were not predictive but slower growth of skinfolds in infancy predicted earlier AR. Higher socio economic status (standardized  $\beta$  = -0.087,  $p < 0.05$ ) and higher paternal BMI (standardized  $\beta$  = -0.11,  $p < 0.001$ ) predicted early AR.

Early AR in these children was associated with higher adiposity (standardized  $\beta$  = -0.08,  $p < 0.05$ ) and higher insulin resistance (standardized  $\beta$  = -0.07,  $p < 0.05$ ) at 6y of age.

**Conclusions:** Early AR in rural Indian children is associated with maternal micronutrient status during pregnancy, slower growth in infancy and large paternal body mass. These findings provide important clues to the growing epidemic of obesity and type 2 diabetes in Indians.



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## **Endothelial function; childhood origins, genetic regulation and natural history**

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Endothelium has been regarded as a relatively inert cell layer but over the past 20 years, it has been credited with an extraordinary array of functions that include control of coagulation, fibrinolysis, arterial tone and vascular growth. Endothelial dysfunction is now implicated in the pathogenesis and clinical course of the majority of adult cardiovascular diseases. It has been reported that changes in endothelium dependent relaxation can be detected even in children, and this may represent the very early changes that ultimately lead to atherosclerosis. As a marker of adult heart disease, endothelial dysfunction in children shall give important and meaningful data regarding vulnerability or otherwise.

It is known that endothelial dysfunction in male and female children could differ due to the androgens on males and the protective effect of estrogen in female children respectively. A study is being carried out on 30 children, classified into 3 groups : 1-5 years, 5-10 years and 10-15 years. The data on the children will have complete analysis on family history of hypertension, diabetes mellitus and coronary artery disease (CAD). Environmental factors (e.g) smoking, salt intake, physical activity, childhood stresses and dietary habits will be elaborated upon. Anthropometric measurements including BMI is recorded and blood pressure is being measured with appropriate precaution in every child. A detailed biochemical analysis for blood glucose, lipid profile, homocysteine and C-reactive protein are being carried out. A detailed well informed consent of the parents has been taken in all cases.

The data of the study shall be presented at the conference.



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# Anthropometry and Glucose/Insulin Concentrations in Indian Children - Relationships to Maternal Gestational Diabetes

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**Background:** To test the hypothesis that the environment experienced by fetuses of mothers with gestational diabetes (GDM), and those with higher glucose concentrations even in the normal range, causes increased adiposity, and altered glucose/insulin metabolism in childhood.

**Methods:** Children (n=630), whose mothers were tested for glucose tolerance during pregnancy had detailed anthropometry at birth and annually thereafter. Plasma glucose and insulin concentrations were measured in a 2-hour oral glucose tolerance test in children and in fasting blood samples in fathers at 5 years.

**Results:** At birth, offspring of diabetic mothers (ODM, N=41) were larger in all body measurements than controls (babies of non-GDM mothers and non-diabetic fathers). At 1 year, these differences had diminished and were not statistically significant. At 5 years, female ODM had larger subscapular (7.4 v 6.3 mm) and triceps skinfold thickness (9.3 v 8.1 mm,  $p<0.05$  for both) than control girls. They also had higher 30- (210 pmol/l v 153 pmol/l in controls) and 120-minute insulin concentrations (122 pmol/l v 90 pmol/l in controls,  $p<0.05$ ). IGT was more common in the ODM (11% v 3%,  $p=0.01$ ) than controls. Newborns of diabetic fathers were lighter than controls, but showed no difference in anthropometry at 5 years. They had lower 120-minute insulin concentrations (63 pmol/l v 83 pmol/l in controls). In control children, maternal insulin was positively associated with skinfolds (triceps 7.8 in lowest v 8.7 mm in highest quartile of maternal insulin,  $P=0.02$ ; subscapular 6.1 v 6.6 mm,  $P=0.08$ ) and 30-minute insulin concentrations (152 v 224 pmol/l,  $P<0.001$ ), and paternal insulin was related to skinfolds (triceps 7.6 v 8.1 mm,  $P=0.08$ ; subscapular 6.1 v 6.2 mm,  $P=0.02^*$ ) independent of maternal or paternal skinfolds and socio-economic status.

**Conclusion:** Maternal GDM increases the risk of adiposity and insulin concentrations in female offspring at 5 years. The absence of similar associations in offspring of diabetic fathers suggests a programming effect of the diabetic intra-uterine environment. With increasing levels of obesity and IGT among Indian mothers, these effects may be contributing to the rise of type 2 diabetes in India. Our continuing follow-up aims to determine the long-term effects of higher maternal glucose concentrations in the absence of GDM.



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# Socioeconomic status and cardiovascular risk factors in young adults

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**Background:** In the developed countries, socioeconomic status is shown to be related to the progression of cardiovascular risk factors. This relationship is not clearly evaluated in India. This paper examines the association between socioeconomic status and cardiovascular risk factors in a south Indian population.

**Methods:** 2,218 men and women (mean age 28 years) were studied from a population-based birth cohort of 10,670 individuals born during 1969-73 in Vellore town and nearby rural areas. Past medical history, socioeconomic status, lifestyle factors and anthropometry were recorded using a standard proforma. SES were derived from various social and economic factors in rural and urban groups separately. Subjects had oral glucose tolerance tests (WHO protocol), including measurements of plasma insulin concentration, from which estimates of insulin resistance and secretion were obtained. Socio-economic status score was grouped into 5 groups in rural and urban, and was related to the prevalence of cardiovascular risk factors. Regression analyses were carried out to determine the association between SES and cardiovascular risk factors.

**Results:** A total of 2218 subjects – 1161 men (rural-617, urban-544) and 1057 women (rural-604, urban-453) participated in this study. Higher SES was associated with obesity (BMI > 25) ( $p<0.001$ ), hypertriglyceridaemia ( $p<0.001$ ), hypercholesterolaemia ( $p<0.001$ ), low HDL ( $p<0.001$ ), high LDL ( $p<0.001$ ) and current tobacco use ( $p<0.001$ ) in urban. In rural, higher SES was associated with obesity ( $p<0.001$ ), hypertriglyceridaemia ( $p=0.05$ ), hypercholesterolaemia ( $p=0.04$ ), and high LDL ( $p=0.003$ ). After controlling for age and gender, all cardiovascular disease risk factors were significantly associated with SES status except triglycerides and glucose fasting in rural and HDL cholesterol in rural and urban.

**Conclusion:** Since SES and risk factors for cardiovascular disease were strongly associated, a proper educational intervention program would be beneficial in order to improve the life style of the subjects and thereby prevent worsening of cardiovascular risk factors and an early onset of cardiovascular disease.



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# Abdominal fat distribution and insulin resistance in Indian men

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## CONTEXT

The heightened susceptibility of Indians to insulin resistance and type 2 diabetes is attributed to adiposity, particularly central adiposity. There are few studies of abdominal fat distribution and its association with insulin resistance in Indians.

## OBJECTIVE

To measure abdominal fat distribution in Indian men and study its association with insulin resistance using Magnetic Resonance (MR) imaging.

## SUBJECTS

A total of 144 healthy men (47 rural, 51 slum residents and 45 urban middle class residents) were selected by multi-stage stratified random sampling. They were 39.0 (34.0-45.0) years old, and had a BMI of 21.5 (19.9-25.0) kg/m<sup>2</sup>.

## MEASUREMENTS

Anthropometry, total body fat by bioelectric impedance analysis (BIA) and abdominal fat by serial cross sectional T1 weighted spin echo MR images. Insulin resistance using Homeostasis Model Assessment (HOMA-IR). Cardiovascular risk factors by standard methods.

## RESULTS

Mean total abdominal fat volume was 5.5 (range 3.1-8.1) liters, accounting for 8.5 (5.4-11.2)% of total body weight and 32.5 (26.3-38.9)% of total body fat. Subcutaneous fat contributed 46.7 (40.5-54.1)% and visceral fat 44.7 (38.3-51.2)% to abdominal fat. For a 31% increase in total body fat between rural and urban middle-class subjects, there was a 88% increase in abdominal fat (7.9 vs 4.2 liters,  $p < 0.001$ ), contributed more by the subcutaneous (94% increase) than the visceral fat (70% increase). Total abdominal fat and its subcutaneous and visceral compartments had a comparable association with HOMA-IR ( $r = 0.4$ ,  $p < 0.001$ ,  $r = 0.5$ ,  $p < 0.001$  and  $r = 0.5$  and  $p < 0.001$  respectively). Multivariate analysis showed that non abdominal fat had a larger contribution to the variance of HOMA-IR than that of the abdominal fat ( $R^2$  26.0% and 4.1% respectively).

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## CONCLUSION

Abdominal fat contributes a third to body fat in Indian men and is equally distributed between subcutaneous and visceral compartments. The two compartments have quantitatively similar associations with HOMA-IR. Non-abdominal fat has a stronger association with HOMA-IR than abdominal fat. Subcutaneous fat contributes more than visceral fat to the higher abdominal fat in urban middle class men than in rural men.



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# Relationships of Maternal and Paternal Birthsize to Cardiovascular Disease Risk in the Adult Offspring : An Intergenerational Study in South India

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**Background** Several studies in diverse population have found association between low birthweight and an increased risk of coronary heart disease (CHD) and type2 diabetes in adult life. These associations may reflect programming effects of fetal undernutrition ('fetal origins' hypothesis) and/or common genes causing low birthweight and adult disease ('fetal insulin' hypothesis). We have examined this issue by studying the relationship between maternal and paternal birthweight and CHD risk factors in the adult offspring in South India.

**Methods** We identified 415 mother offspring pairs and 296 father offspring pairs where both the parent and child were born in Holdsworth Memorial Hospital (HMH), Mysore, India, which has preserved birth records since 1934. Investigations in the parents (aged 33-65 years) and offspring (aged 20-46 years) included anthropometry, oral glucose tolerance test, plasma insulin and serum lipid concentrations, blood pressure, electrocardiograph and Rose chest pain questionnaire. Insulin resistance was calculated using homeostasis model assessment. Metabolic syndrome was defined based on WHO criteria.

**Results** Among the offspring, lower birthweight was associated with higher 120-minute glucose, fasting cholesterol and triglyceride concentrations, higher waist/hip and subscapular/triceps ratios, increased insulin resistance and higher combined prevalence of impaired glucose tolerance, impaired fasting glycaemia and diabetes mellitus ( $p < 0.01$  for all adjusted for sex, age and body mass index). Both maternal and paternal birthweight were inversely related to offspring metabolic syndrome (OR 0.4 (95 % CI: 0.2, 1.0);  $p = 0.057$  for mother-offspring pairs; OR 0.3 (95 % CI: 0.08, 0.9);  $p = 0.031$  for father-offspring pairs). Paternal but not maternal birthweight was inversely related to offspring insulin resistance ( $e^{\beta} = 0.9$  (95 % CI: 0.8, 1.08);  $p = 0.3$  for mother-offspring pairs;  $e^{\beta} = 0.8$  (95 % CI: 0.7, 1.01);  $p = 0.06$  for father-offspring pairs). We also found inverse relationships between maternal birthweight and offspring systolic blood pressure ( $\beta = -2.5$  (95% CI: -5.03, -0.07;  $p = 0.04$ ) and WHR ( $p = 0.03$ ).

**Conclusions** In conclusion our study provides support to the fetal insulin hypothesis and indicates that at least part of the association between low birthweight and insulin resistance is genetic in origin. The study also provides evidence for intergenerational effects on vascular disease transmitted through the mother, which are likely to have an environmental component.



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## GUEST SPEAKERS AND HONORARY SNEHA MEMBERS

1. Ehrenberg HM, Mercer BM, Catalano PM. The influence of obesity and diabetes on the prevalence of macrosomia. *Am J Obstet Gynecol* 2004;191:964-8.
2. Catalano PM, Thomas A, Huston-Presley L, Amini SB. Increased fetal adiposity: A very sensitive marker of abnormal in utero development. *Am J Obstet Gynaecol* 2003;198:1698-704.
3. Catalano PM. Obesity and Pregnancy – The Propagation of a Viscous Cycle? *J Clin Endocrinol Metab* 2003;88:3505-3506.
4. Catalano PM, Kirwan JP, Haugel-de Mouzon S, King J. Gestational Diabetes and Insulin Resistance: Role in Short- and Long-Term Implications for Mother and Fetus. *J Nutr* 2003;133:1674S-1683S.
5. Cooper C, Westlake S, Harvey N, Javaid K, Dennison, Hanson M. Developmental origins of osteoporotic fracture. *Osteoporosis International* 2005: online 10.1007/x00198-005-2039-5.
6. Aihie Sayer A, Cooper C. Fetal programming of body composition and musculoskeletal development. *Early Human Development* 2005;81:735-744.
7. Cooper C, Javaid K, Westlake S, Harvey N, Dennison E. Developmental Origins of Osteoporotic Fracture: the Role of Maternal Vitamin D Insufficiency. *J Nutr* 2005;135:2728S-2734S.
8. Javaid MK, Crozier SR, Harvey NC, Taylor P, Inskip HM, Godfrey KM, Cooper, Southampton Women's Survey Study Group. Maternal and Seasonal Predictors of Change in Calcaneal Quantitative Ultrasound during Pregnancy. *J Clin Endocrinol Metab* 2005;90:5182-5187.
9. Tobais JH, Steer CD, Emmett PM, Tonkin RJ, Cooper C, Ness AR. Bone mass in childhood is related to maternal diet in pregnancy. *Osteoporosis Int* 2005;16:1731-1741.
10. Javaid MK, Godfrey KM, Taylor P, Robinson SM, Crozier SR, Dennison EM, Robinson JS, Breier BR, Arden NK, Cooper C. Umbilical Cord Leptin Predicts Neonatal Bone Mass. *Calcif Tissue Int* 2005;76:341-347.
11. Javaid MK, Crozier SR, Harvey NC, Gale CR, Dennison EM, Boucher BJ, Arden NK, Godfrey KM, Cooper C, Princess Anne Hospital Study Group. Maternal vitamin D status during pregnancy and childhood bone mass at age 9 years: a longitudinal study. *Lancet* 2006;367:36-43.
12. Aihie Sayer A, Dennison EM, Syddall HE, Gilbody HJ, Phillips DIW, Cooper C. Type 2 Diabetes, Muscle Strength, and Impaired Physical Function. *Diabetes Care* 2005;28:2541-2
13. Inskip HM, Godfrey KM, Robinson SM, Law CM, Barker DJP, Cooper C, SWS Study Group. Cohort profile: The Southampton Women's Survey. *Int J Epidemiol* 2005; doi:10.1093/ije/dyi202.

- 
14. Hanson M, Gluckman P. Endothelial dysfunction and cardiovascular disease: the role of predictive adaptive responses. *Heart* 2005;91:864-866.
  15. Gluckman PD, Hanson MA, Morton SMB, Pinal CS. Life-long echoes – a critical analysis of the Developmental origins of Adult Disease Model. *Biol Neonate* 2005;87:127-39.
  16. Gluckman PD, Hanson MA. Maternal constraint of fetal growth and its consequences. *Seminars in Neonatology* 2004 *in press*.
  17. Gluckman PD, Hanson MA. Living with the Past: Evolution, Development, and Patterns of Disease. *Science* 2004;305:1733-1736.
  18. Gluckman PD, Hanson MA. Developmental Origins of Disease Paradigm: A Mechanistic and Evolutionary Perspective. *Pediatr Res* 2003;56:311-317
  19. Gluckman PD, Hanson MA. Evolution, development and timing of puberty. *Trends in Endocrinology and Metabolism* 2006;17:7-12.
  20. Gluckman PD, Hanson MA. The developmental origins of the metabolic syndrome. *Trends in Endocrinology and Metabolism* 2004;15:183-187.
  21. Lillycrop KA, Phillips ES, Jackson AA, Hanson MA, Burdge GC. Dietary Protein Restriction of Pregnant Rats Induces and Folic Acid Supplementation Prevents Epigenetic Modification of Hepatic Gene Expression in the Offspring. *J Nutr* 2005;135:1382-1386.
  22. Borghi J, Thapa B, Osrin D, Jan S, Morrison J, Tamang S, Shrestha BP, Wade A, Manandhar DS, Costello AMdeL. Economic assessment of a women's group intervention to improve birth outcomes in rural Nepal. *Lancet* 2005;366:1882-84.
  23. Costello A, Osrin D. The case for a new Global Fund for maternal, neonatal, and child survival. *Lancet* 2005;366:603-05.
  24. Osrin D, Vaidya A, Shrestha Y, Baniya RB, Manandhar DS, Adhikari R, Filteau S, Tomkins A, Costello AMdeL. Effects of antenatal multiple micronutrient supplementation on birthweight and gestational duration in Nepal: double-blind, randomised controlled trial. *Lancet* 2005;365:955-62.
  25. Christian P, Osrin D, Manandhar DS, Khattry S, de Costello AM, West KP. Antenatal micronutrient supplements in Nepal. *Lancet* 2005;366:711.
  26. Knight B, Shields BM, Turner M, Powell RJ, Yajnik CS, Hattersley AT. Evidence of genetic regulation of fetal longitudinal growth. *Early Human Development* 2005;81:823-831.
  27. Barker DJP, Osmond C, Forsen TJ, Kajantie E, Eriksson JG. Trajectories of Growth among Children Who Have Coronary Events as Adults. *N Engl J Med* 2005;353:1802-9.
  28. Kajantie E, Osmond C, Barker DJP, Forsen T, Phillips DIW, Eriksson JG. Size at birth as a predictor of mortality in adulthood: a follow-up of 350,000 person-years. *Int J Epidemiol* 2005;34:655-663.
  29. Barker DJP, Eriksson JG, Forsen T, Osmond C. Infant growth and income 50 years later. *Arch Dis Child* 2005;90:272-273.
-

- 
30. Laaksonen DE, Lindstrom J, Lakka TA, Eriksson JG, Niskanen L, Wikstrom K, Aunola S, Keinanen-Kiukaanniemi S, Laakso M, Valle TT, Ilanne-Parikka P, Louheranta A, Hamalainen H, Rastas M, Salminen V, Cepaitis Z, Hakumaki M, Kaikkonen H, Harkonen P, Sundvall J, Tuomilehto J, Uusitupa M. Physical Activity in the Prevention of Type 2 Diabetes. The Finnish Diabetes Prevention Study. *Diabetes* 2005;54:158-165.
  31. Eriksson JG, Yliharsila H, Forsen T, Osmond C, Barker DJP. Exercise protects against glucose intolerance in individuals with a small body size at birth. *Preventive Medicine* 2004;39:164-167.
  32. Dalziel SR, Lim VK, Lambert A, McCarthy D, Parag V, Rodgers A, Harding JE. Antenatal exposure to betamethasone: psychological functioning and health related quality of life 31 years after inclusion in randomised controlled trial. *BMJ* 2005;331:665-671.
  33. Dalziel SR, Walker NK, Parag V, Mantell C, Rea HH, Rodgers A, Harding JE. Cardiovascular risk factors after antenatal exposure to betamethasone: 30-year follow-up of a randomised controlled trial. *Lancet* 2005;365:1856-62.

## **SNEHIS**

34. Aggarwal D, Sachdev HPS, Nagpal J, Singh T, Mallika V. Haematological effect of iron supplementation in breast fed term low birth weight infants. *Arch Dis Child* 2005;90:26-29.
35. Bajaj M, Dubey AP, Nagpal J, Singh PK, Sachdev HPS. Short term effect of oil supplementation of complementary food on total ad-libitum consumption in 6- to 10-month-old breast fed Indian infants. *J Ped Gastroenterol* 2005;41:61-5.
36. Bhat DS, Yajnik CS, Sayyad MG, Raut KN, Lubree HG, Regge SS, Chougule SD, Shetty PS, Yudkin JS, Kurpad AV. Body fat measurement in Indian men: comparison of three methods based on a two-compartment model. *International Journal of Obesity* 2005;29:842-848.
37. Bhave S, Bavdekar A, Otiv M. IAP National Task Force for Childhood Prevention of Adult Diseases: Childhood Obesity. *Indian Pediatrics* 2004;41:559-575.
38. Fall CHD. Endocrine programming and the fetal and early-life origins of adult disease. In: *Clinical Paediatric Endocrinology*. Eds: CGD Brook, RS Brown, PE Clayton. Blackwell Science, 2005.
39. Ghaffar A. Reddy KS. Singhi M. Burden of non-communicable disease in South Asia. *BMJ* 2004;328:807-810.
40. Hill JC, Krishnaveni GV, Annamma I, Leary SD, Fall CHD. Glucose tolerance in pregnancy in South India: relationships to neonatal anthropometry. *Acta Obstet Gynecol Scand* 2005;84:159-165.
41. Joshi NP, Kulkarni SR, Yajnik CS, Joglekar CV, Rao S, Coyaji KJ, Lubree HG, Rege SS, Fall CHD. Increasing maternal parity predicts neonatal adiposity: Pune Maternal Nutrition Study. *Am J Obstet Gynecol* 2005;193:783-9.

- 
42. Joshi S, Rao S, Girigosavi S, Daware M, Kale A, Hedge M. Differential Effects of Fish Oil and Folic Acid Supplementation During Pregnancy in Rats on Cognitive Performance and Serum Glucose in Their Offspring. *Nutrition* 2004;20:465-472.
  43. Kale SD, Yajnik CS, Kulkarni SR, Meenakumari K, Joglekar AA, Khorsand N, Ladkat RS, Ramdas LV, Lubree HG. High risk of diabetes and metabolic syndrome in Indian women with gestational diabetes mellitus. *Diabetic Medicine* 2004;21:1257-1259.
  44. Kanade AN, Rao S, Yajnik CS, Margetts BM, Fall CHD. Rapid assessment of maternal activity among rural Indian mothers (Pune Maternal Nutrition Study). *Public Health Nutrition* 2005;8:588-595.
  45. Krishnaveni GV, Hill JC, Leary SD, Veena SR, Saperia J, Saroja A, Karat SC, Fall CHD. Anthropometry, Glucose Tolerance, and Insulin Concentrations in Indian Children. *Diabetes Care* 2005;28:2919-2925.
  46. Krishnaveni GV, Hill JC, Veena SR, Leary SD, Saperia J, Chachyamma KJ, Karat SC, Fall CHD. Truncal Adiposity is Present at Birth and in Early Childhood in South Indian Children. *Indian Pediatrics* 2005;42:527-538.
  47. Kumar V, Sachdev HPS, Khalil A. Noninvasive Evaluation of Endothelial Function and Arterial Mechanics in Overweight Adolescents. *Indian Pediatrics* 2004;41:1105-1114.
  48. Reddy KS, Arora M. Tobacco use Among Children in India: A Burgeoning Epidemic. *Indian Pediatrics* 2005;42:757-761.
  49. Sachdev HPS, Fall CHD, Osmond C, Lakshmy R, Dey Biswas SK, Leary SD, Reddy KS, Barker DJP, Bhargava SK. Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurements of body mass index in childhood in the New Delhi birth cohort. *Am J Clin Nutr* 2005;82:456-66.
  50. Sachdev HPS, Gera T, Nestel P. Effect of iron supplementation on mental and motor development in children: systematic review of randomised controlled trials. *Public Health Nutrition* 2004;8:117-132.
  51. Singh PK, Sachdev HPS, Nagpal J, Bajaj M, Dubey AP. Short term effect of complementary feeding frequency on total ad-libitum consumption in 6- to 10-month-old breast fed Indian infants. *J Ped Gastroenterol* 2005;41:422-5.
  52. Solanki I, Matnani M, Kale M, Joshi K, Bavdekar A, Bhave S, Pandit A. Transcutaneous Absorption of Topically Massaged Oil in Neonates. *Indian Pediatrics* 2005;42:998-1005.
  53. Yajnik CS, Deshpande SS, Panchanadikar AV, Naik SS, Deshpande JA, Coyaji KJ, Fall CHD, Refsum H. Maternal total homocysteine concentration and neonatal size in India. *Asia Pac J Clin Nutr* 2005;14:179-181.

## **OTHER**

54. Gopalan C. Towards India's nutritional well-being: the need for a "second wind". *Bulletin of the Nutrition Foundation of India* 2005;27(1);1-3.
55. Gillman MW. Developmental Origins of Health and Disease. *N Eng J Med* 2005; 353:1848-1850.