for both genders. Overweight adults aged 25 to 40 had a significantly higher ANGS than older subjects at the top $HOMA_{IR}$quintile. These results suggest that ANGS may be a useful screening tool for identifying IR status in overweight AA and H but not in WNH. Higher ANGS is a strong indicator of IR independently of glucose tolerance categories, and is influenced in their extent and severity by ethnicity and age.

P432

Insulin Resistance Is Associated with a Clustering of Metabolic Disorders in a South African Community

NAOMI S. LEVITT[1], Krisela Steyn[2], Margaret Hoffman[3], Carl Lombard[2], Andre Daniels[2], Estelle V. Lambert[2], Jean Fourie[2].
[1] Department of Medicine, University of Cape Town, Cape Town, South Africa; [2] Medical Research Council, Cape Town, South Africa; [3] Department of Community Health, University of Cape Town, Cape Town, South Africa

This study examined the association between insulin resistance and the major contributors to the metabolic syndrome in an underprivileged South African community.A cross-sectional survey was conducted on 974 residents >15 yrs of age in a periurban community of mixed ancestral origin. Each participant had a 75g OGTT, blood pressure, anthropometry, fasting lipids and insulin measured. The relationship between insulin resistance as measured by HOMA model or fasting insulin concentration and the number of metabolic disorders (IGT or newly diagnosed diabetes, hypertension (excluding those on anti-hypertensive therapy) and dyslipidaemia (triglycerides >2.3mmol/L or small dense LDL or HDL <1.0mmol/L was examined. Insulin data was log transformed. The mean age of the participants was 37.6 (range 15-86) years. The crude prevalence of diabetes was 7.1%, IGT 8.0%, hypertension 19.2% and dyslipidaemia 28.0%. Obesity was present in 9.3% of men and 31.6% of women. Fasting insulin concentrations (49, 59, 71 and 87pmol/L, p=0.01) and HOMA (1.7, 2.2. 2.9 and 4.3, p=0.001) both increased with increasing numbers of metabolic disorders (0, 1, 2, 3). Using linear regression analysis insulin resistance was independently associated with age (p=0.0001), sex (p=0.0003), BMI (p=0.0001), waist circumference (p=0.0029) and the number of metabolic disorders in women only (p=0.0376) R2 = 38%. In conclusion insulin resistance is associated with a clustering of metabolic disorders in this community.

P433

High Birth Weight Babies Predict Death, Obesity, Glucose Intolerance and Insulin Resistance in Mothers 8 Years after Delivery

CHITTARANJAN S. YAJNIK[1], Charudatta V. Joglekar[1], Jyoti A. Deshpande[1], Ashish Bavdekar[2], Anand Pandit[2], Caroline Fall[3].
[1] Diabetes Unit, KEM Hospital Research Centre, Pune, Maharashtra, India; [2] Department of Pediatrics, KEM Hospital, Pune, Maharashtra, India; [3] MRC Environmental Epidemiology Unit, University of Southampton, Southampton, United Kingdom

Low birth weight is associated with insulin resistance, diabetes and increased cardiovascular risk in later life. Recent reports have linked child's low birth weight with increased maternal mortality. We have shown an association of low birth weight with insulin resistance and other cardiovascular risk factors at 8 years of age in a cohort of 477 children born in our hospital. We now report the relationship between child's birth weight and cardiovascular risk factors and death in the parents 8 years after child's birth.
Six mothers died, of these four belonged to the highest tertile of child's birth weight, paternal deaths were equally distributed (4 in each tertile). Parents who had given birth to heavier babies were more obese (P<0.01, both), more taller (P< 0.01, both) 8 years later. Parents'blood pressure and circulating lipid concentrations were not related to child's birth weight There was an excess of glucose intolerance (DM + IGT) in the mothers of heaviest children (q1 15%, q2 11%, q3 37%). Mothers of heavier children had higher fasting plasma insulin concentration, even after allowing for.current obesity, age and socioeconomic status (P=0.02). Maternal B-cell function ('insulinogenic index') was not related to child's birth weight. There was no relationship between child's birth weight and paternal glycemia and insulin concentrations 8 years later.
Thus, mothers of heavier Indian babies are at increased risk of all cause mortality, obesity, glucose intolerance, and fasting hyperinsulinemia (insulin resistance) within 8 years of delivery of the child. Fathers are at increased risk of obesity. Low birth weight did not predict any cardiovascular risk factors in either parents. The relationship could be due to low grade gestational hyperglycemia in insulin resistant mothers and/or an influence of insulin resistance linked gene(s) on fetal growth. Unlike the reports from the UK, heavier babies predicted more risk to parents than the low birth weight babies.

P434

Predictors of Deterioration of Glucose Tolerance in Indian Subjects

SHAILAJA D. KALE, Kishor M. Shelgikar, Mehmood G. Sayyad, Kondiram N. Raut, Dattatray S. Bhat, Chittaranjan S. Yajnik. *Diabetes Unit, K.E.M. Hospital Research Centre, Pune, Maharashtra, India*

We have studied progression to impaired glucose tolerance (IGT) and diabetes mellitus (DM) over 10 years in 133 subjects with normal glucose tolerance and without family history of diabetes. There were 76 men (mean age 42 y, BMI 23.5 kg/m^2) and 57 women (39.5 y, 23.5 kg/m^2). At 10 year follow-up 22 subjects had progressed to IGT or DM, 73 continued to have normal glucose tolerance and 38 were lost to follow-up.
Men who progressed to IGT or DM were more obese (BMI 26.1 vs. 22.5 kg/m^2, p<0.01), more centrally obese (waist circumference 87.6 vs. 78.1 cm, p<0.01), and had higher central (subscapular skinfold 29.1 vs. 21.8 mm, p<0.05) and peripheral (triceps skinfold 15.3 vs. 11.9 mm, p<0.05) subcutaneous fat. Plasma glucose concentrations were similar in the two groups (fasting 79.9 vs. 82.1 mg/dl and 2h post glucose 114.8 vs.107.7 mg/dl, both n.s.) but fasting plasma insulin concentration (21.0 vs 8.7 mU/L, P< 0.01) and calculated insulin resistance variable (HOMA, 0.60 vs 0.26, P<0.01) were higher (both, independant of obesity) in those who progressed than in those whose glucose tolerance did not deteriorate. Subjects whose glucose tolerance deteriorated continued to be hyperinsulinemic (fasting 16.2 vs. 9.7 mU/L, P<0.05) and more insulin resistant (0.66 vs. 0.30, P<0.01) at 10 y follow up. There were no anthropometric and metabolic predictors of deterioration of glucose tolerance in women. Generalised and central obesity and insulin resistance predict deterioration of glucose tolerance in Indian men. Preventive measures should concentrate on controlling obesity and treating insulin resistance.

P435

Beta Cell Disfunction as the Main Factor To Determine Glucose Intolerance In Insulin Resistance Patients

MARCOS A. TAMBASCIA[1], Bruno Geloneze[1], Sylka O. Rodovalho[1], Jose C. Pareja[2], Daniela M. Franco[1], Enrico M. Repetto[1].
[1] Endocrinology, UNICAMP, Campinas, SP, Brazil; [2] Gastrocirurgy, UNICAMP, Campinas, SP, Brazil

The pathogenesis of type 2 Diabetes Mellitus (DM2) involves both the insulin resistance and deficiency of secretion. Some patients with very high degrees of insulin resistance as seen in obesity, do not show any level of glucose intolerance due to a very efficient ability to secrete insulin leading to a state of compensatory hyperinsulinemia.
To study the relationship of insulin resistance and insulin secretion we have used the HOMA method (Homeostasis Model Assessment) extracting two different scores for insulin resistance (HOMA-IR) and beta cell function (HOMA-b). We studied 347 patients from the Metabolic Syndrome Outpatient Clinic of University of Campinas classified by BMI