253 What is the association between insulin sensitivity and microarray differential gene expression in omental tissue of healthy non-obese pregnant women?

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OBJECTIVE: To determine correlations between insulin sensitivity and gene expression in omental tissue of non-obese pregnant women.

STUDY DESIGN: Microarray gene profiling using Illumina HumanHT-12 V4 Expression BeadChips was performed on omental adipose tissue obtained during non-laboring cesarean section in a fasting state from 11 non-obese non-diabetic pregnant women. Measuring transcript levels of selected genes using quantitative real-time PCR validated findings. Matusda-Insulin sensitivity Index (IS) was calculated from glucose and insulin levels obtained from a frequently sampled oral glucose tolerance test. homeostasis model assessment of insulin resistance (HOMA-R) was calculated and correlated with IS. Linear regression analysis was performed and list was filtered at $P<0.01$.

RESULTS: 510 genes significantly correlated with IS; of these 325 matched to known genes in NIH David. Twenty-three genes were determined to be involved in immune/inflammatory, vascular or metabolic pathways. Two insulin signaling pathways genes: insulin receptor (INSR) ($r=0.84$), which stimulates glucose uptake, and mitogen-activated protein kinase 3 (MAPK1) ($r=0.78$), which is anabolic, were strongly positively correlated with IS. Leptin which has been shown to suppress insulin gene expression and glucose transport was negatively correlated with IS. Of 13 genes with immune/inflammatory; 10 were positively correlated and 3 negatively correlated with IS.

CONCLUSION: Our findings suggest differential gene expressions that may be associated with cellular mechanisms modulating insulin sensitivity and resistance in visceral adipose tissue in healthy pregnancy. The unexpected finding of most immune/inflammatory genes correlating positively with insulin sensitivity indicates the need for further studies in clarifying relations between functional variants and local protein expressions.

Association between insulin sensitivity and microarray differential gene expression in omental tissue of healthy non-obese pregnant women

Gene Name	Gene Symbol	(ISI matsuda index)correlation	p-value(ISI matsuda index)	HOMA r, correlation	Function
CD48 molecule	CD48	0.75	0.007	-0.48	Facilitate interaction between activated lymphocytes.Regulation of T-cell activation
Killer cell immunoglobulin-like receptor, two domains, long cytoplasmic tail, 3	KIR2DL3	0.74	0.010	-0.48	Inflammatory cells activation
CD19 molecule	CD19	-0.74	0.010	0.42	Facilitate interaction between activated lymphocytes. regulating T-cell activation
Leptin	LEP	-0.80	0.003	0.58	Suppresses insulin expression, secretion & glucose transport into the β-cell. Signaling pathway inhibit food intake &/or regulate energy expenditure to maintain adipose mass. Linked to type 2 diabetes mellitus. Mutations cause obesity & hypogonadism
Chemokine (C-C motif) ligand 19	CCL19	0.87	0.000	-0.64	Inflammatory and immunological responses; normal lymphocyte recirculation and homing
p21 protein (Cdc42/Rac)-activated kinase 2	PAK2	0.79	0.004	-0.45	Cell survival and cell growth
Chemokine (C-X-C motif) ligand 11	CXCL11	0.78	0.004	-0.55	Proinflammatory
Granzyme B (granzyme 2, cytotoxic T-lymphocyte-associated serine esterase 1)	GZMB	0.78	0.004	-0.61	Proinflammatory, target cell lysis in cell-mediated immune responses,involved in stroke, Alzheimer's disease, myocardial infarction, cancer, inflammatory disease.
Mitogen-activated protein kinase 3	MAPK1	0.78	0.004	-0.52	Protein synthesis;cell growth, adhesion, survival

254 Gestational weight gain in excess of IOM guidelines associated with macrosomia in obese women with GDM

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OBJECTIVE: To determine whether adherence to Institute of Medicine (IOM) weight gain guidelines modifies risk of macrosomia among women with GDM.

STUDY DESIGN: We conducted a retrospective cohort study of singleton term pregnancies diagnosed with GDM (50g 1-hr >140 mg/dL; 100g 3-hr OGTT, NDDG criteria) that delivered at UNC Hospitals, January 2002 - May 2010. We calculated body mass index (BMI) using height and self-reported pre-pregnancy or measured first prenatal visit weight <20 weeks. We used last visit weight > 37 weeks - baseline weight to calculate gestational weight gain (GWG). We modeled the odds (OR, aOR) with 95% confidence intervals (95%CI) of macrosomia >4000g by BMI category and 2009 IOM guidelines. Women meeting IOM guidelines were the reference group. Final adjusted models include ethnicity and gestational age at delivery.

RESULTS: Among 577 eligible women, 85% (466/548) comprised the cohort with complete data, and 19% (89/466) delivered infants >4000g. Obese women were more likely than overweight or normal weight women to gain above IOM guidelines (52% v 41% v 31%). Overweight women were more likely than obese or normal weight women to meet IOM guidelines (31% v 20% v 15%). Normal weight