dependent uptake of [3H]-paclitaxel and [3H]-estrone sulfate strongly suggests the involvement of efflux transporters in its transfer across the placenta. This work was supported by NICHD Obstetric-Pharmacology network grant U10HD047891.

234 Inhibition of autophagy by sera from pregnant women

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OBJECTIVE: Autophagy is a process that maintains homeostasis by eliminating senescent or damaged intracellular organelles and proteins. In addition, autophagy participates in regulation of innate and acquired immunity. A role for autophagy in pregnancy has been scarcely studied. We compared the influence of sera from pregnant and non-pregnant women on autophagy induction. p62 is a cytoplasmic protein essential for induction of autophagy. Its concentration in the cytoplasm is inversely proportional to the level of autophagy induction.

STUDY DESIGN: Peripheral blood mononuclear cells (PBMCs) from female donors were incubated with sera from 35 women in the second trimester of pregnancy or 35 non-pregnant reproductive age women. After 48 hours cells were collected, lysed and assayed for p62 concentrations by ELISA. PBMCs were also incubated with the autophagy inducer, rapamycin, in the presence or absence of sera. Sera were tested for concentrations of immune mediators by ELISA. Clinical data and source of sera were accessed only after completion of all experiments.

RESULTS: Median (range) p62 concentrations were 6.7 ng/ml (1.1-22.7) for PBMCs incubated with pregnancy sera vs. 2.5 ng/ml (0.8-7.7) for non-pregnant sera ($p<0.0001$). Even in the presence of rapamycin, median p62 levels were elevated in the presence of pregnancy sera, 1.3 ng/ml (0.06-4.9), as compared to non-pregnant sera, 0.7 ng/ml (0-3.3) ($p=0.0191$). Among the pregnant subjects, the p62 level was inversely proportional to the results of a 50 g glucose challenge test (GCT) ($r = -0.5630$, $p=0.0005$). Insulin-like growth factor-1 and interleukin-13, inhibitors of autophagy, were elevated in sera from pregnant women.

CONCLUSION: Sera from healthy pregnant women inhibit autophagy to a greater extent than sera from non-pregnant women. Autophagy inhibition during pregnancy may function to decrease insulin resistance.

235 A preconception nomogram to predict preterm birth

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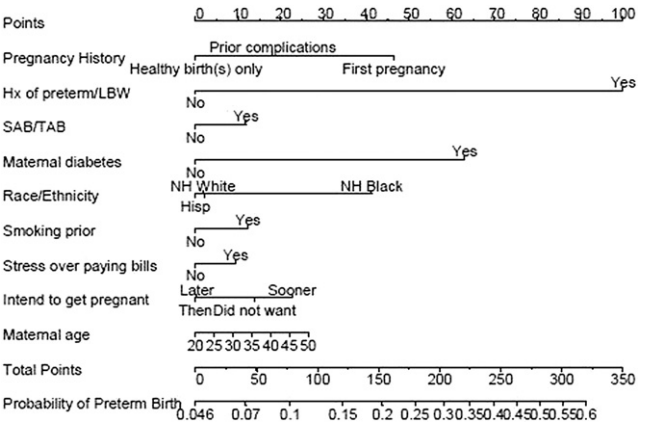
OBJECTIVE: Preterm birth is a leading cause of perinatal morbidity & mortality. Prevention strategies rarely focus on preconception care. We sought to create a preconception nomogram identifying women at highest risk for preterm birth using the Pregnancy Risk Assessment Monitoring System (PRAMS) surveillance data.

STUDY DESIGN: PRAMS data from 2004-2009 was utilized. Odds ratios (OR) of preterm birth for each preconception variable were estimated and adjusted analyses were conducted. A validated nomogram predicting the probability of preterm birth was created using multivariate logistic regression model coefficients.

RESULTS: 192,208 cases met inclusion criteria. Demographic/maternal health characteristics and associations with preterm birth and ORs are reported (Table). After validation, significant predictors of preterm birth among all women, were prior history of preterm birth or low birthweight baby, prior SAB/TAB, maternal diabetes prior to conception, maternal race (e.g., NH black), intention to get pregnant prior to conception (i.e., did not want or wanted it sooner), and smoking prior to conception ($p<0.05$). Overall, our preconception preterm risk model correctly classified 76.1% of preterm cases with a negative predictive value (NPV) of 76.7%. A nomogram using a 0-100 scale illustrates our final preconception model for predicting preterm birth (Figure).

CONCLUSION: This preconception nomogram will give providers a tool to assist in predicting a woman's individual preterm birth risk and to triage high-risk women to preconception care. Future studies are needed to validate the nomogram in the clinical setting.

Summary nomogram for predicting the probability of preterm birth according to selected preconception risk factors using the PRAMS surveillance 2004-2009 data



Instructions: using a pencil and ruler, draw a line vertically up to the top 'Points' axis to get an estimate of the points associated with each risk factor level for a given individual woman. Sum the points across all risk factors for that individual to create a 'Total Points' score. Draw a line vertically down from the 'Total Points' axis through the 'Probability of preterm birth' axis to obtain an approximation of the individual's baseline risk of preterm birth prior to conception.

Sample characteristics–Estimated preterm birth prevalences by subgroup

Categorical variables		Preterm birth		Total	p-value
		Unweighted n	Weighted %	n[1]	
Total		47,654	9.2%	192,110	
Race/Ethnicity	NH White	26,385	8.5%	103,326	.0000
	NH Black	11,558	13.2%	33,351	
	Hispanic	5,668	8.0%	29,087	
	Other	4,043	8.6%	26,346	
Income – grouped	<10k	11,025	10.4%	39,748	.0000
	10 - 14k	4,489	9.3%	17,338	
	15 - 19k	3,089	9.7%	11,982	
	20 - 24k	3,301	8.8%	13,173	
	25 - 34k	4,404	9.4%	18,134	
	35 - 49k	4,423	8.5%	18,900	
	50k+	12,942	8.4%	57,587	
Other Insurance (not including Medicaid)	Not on Other Insurance	20,851	9.4%	80,081	.0179
	On Other Insurance	26,685	9.0%	111,506	
Medicaid	On Medicaid	8,345	11.0%	31,189	.0000
	No	39,174	8.9%	160,260	
Education	0-8 yrs	1,666	8.7%	7,913	.0000
	9-11 yrs	7,394	10.4%	27,142	
	12 yrs	15,148	9.8%	57,924	
	13-15 yrs	11,134	8.9%	44,698	
	16+ yrs	11,659	8.4%	52,123	
Urban/Rural Category	Urban	14,612	9.4%	62,174	.0000
	Rural	5,618	8.9%	27,247	
Prior live birth complications (prior live preterm/LBW birth)	No	35,990	8.1%	161,639	.0000
	Yes	9,970	20.5%	22,978	
Prior SAB/TAB	No	33,747	8.8%	139,856	.0000
	Yes	13,411	10.2%	50,878	
Any Pregnancy History	First pregnancy	17,716	9.49%	66,397	.0153
	Second Pregnancy	29,976	9.05%	125,811	
Intention to get pregnant	Sooner	9,541	10.8%	10.8%	.0000
	Later	14,684	9.0%	9.0%	
	Then	16,968	8.2%	8.2%	
	Did not want	5,649	11.1%	11.1%	
Diabetes before pregnancy	No	45,118	9.0%	185,075	.0000
	Yes	1,800	19.2%	4,467	
Smoke – 3 months before pregnancy	No	34,137	8.9%	141,936	.0085
	Yes	12,671	10.1%	47,019	
Drink – 3 months before pregnancy	No	25,485	9.9%	96,870	.0000
	Yes	21,146	8.5%	91,150	
Abuse – 12 months before pregnancy	No	43,517	9.1%	176,875	.0000
	Yes	3,250	10.7%	11,509	
Stressors	1. I was in a physical fight	2,511	2,511	10.9%[2]	8,759
	2. My husband/partner was in jail	2,732	2,732	10.8%	9,758
	3. I lost my job even though I wanted to continue working	5,473	5,473	10.6%	19,371
	4. I couldn't pay my bills	12,583	12,583	10.5%	45,737
	5. My husband/partner said he did not want me to be pregnant	4,548	4,548	10.6%	16,610

Continuous variables	No Preterm birth	Preterm birth	Total	p-value
	Weighted Mean (CI)		n	
Age	28.37 (28.31, 28.45)	28.48 (28.31, 28.65)	192,110	.1263
BMI	25.54 (25.47, 25.61)	25.96 (25.77, 26.15)	181,349	.0000

[1]Weighted n may not add up to 100% due to missing data; [2]Individual stressor variables only shown for "yes" responses.

236 Ectonucleotide pyrophosphatase phosphodiesterase 1 (ENPP1) expression in adipose tissue of women with excessive versus normal gestational weight gain during pregnancy

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OBJECTIVE: ENPP1, a transmembrane glycoprotein, has been shown to modulate adipocyte maturation and insulin receptor signaling. These effects have been associated with systemic insulin resistance and increased risk for type 2 diabetes. Our objective is to measure adipocyte ENPP1 expression in response to gestational weight gain (GWG).

STUDY DESIGN: Women scheduled for elective repeat cesarean at term and who fasted at least 6 hours were recruited. Blood was obtained before the initiation of intravenous fluids and subcutaneous fat was biopsied after the skin incision. Adipose cell size was analyzed using 3D multi-photon imaging. Tissue expression level of ENPP1 and phosphorylation of Akt (pAkt) for insulin signaling were measured by Western Blot. Using IOM guidelines, excessive vs normal GWG were compared. Statistical Analysis Software was utilized.

RESULTS: Fifteen subjects with excessive GWG were compared to 9 with normal GWG. Maternal age, EGA at delivery and pregestational body mass index (BMI) were not significantly different. Delivery BMI, birthweight, tissue expression level of ENPP1, adipocyte cell size and phosphorylated Akt were significantly different (Table).

CONCLUSION: Increased expression level of ENPP1 in women with excessive GWG is associated with decreased adipocyte cell size and phosphorylation of Akt, indicating impaired maturation of adipocytes and insulin signaling. These findings suggest women with excessive GWG may be at risk for future systemic insulin resistance and type 2 diabetes.

Excessive versus normal gestational weight gain

	Normal GWG 9 subjects		Excessive GWG 15 subjects		p value
Pregestational BMI*	25.9	± 3.3	27.3	± 5.4	0.55
Delivery BMI*	29.7	± 3.1	34.4	± 4.7	0.01
Birth weight (gm)	3306	± 392	3656	± 358	0.03
Expression level tissue ENPP1	0.35	± 0.18	0.57	± 0.20	0.01
Adipocyte cell size (micron)	111	±15	95	±15	0.03
Ratio p tAkt	0.77	± 0.20	0.52	± 0.23	0.01

* BMI [weight (kg)]/[height (m)]^2.