

Summer 2007
Volume 5, Issue 2

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This Project is supported in part by funds received from the State of California, Department of Health Services, Maternal, Child and Adolescent/Family Planning Health Branch

SWEET SUCCESS

NEWSLETTER for DIABETES AND PREGNANCY

Notes from New York, 2007

9th Annual Meeting of the Diabetes and Pregnancy Study Group of North America

Leona Dang-Kilduff, RN, MSN, CDE

A Matter of Insulin: Developmental Programming of Body Weight Regulation

Andreas Phegemann, MD

Health and disease are not only genetic but a result of environment. The intrauterine environment with hyperglycemia and hyperinsulinemia results in overfeeding prenatally. This overfeeding leads to a resetting of the hypothalamic response to insulin, leptin and cortisol causing hypothalamic dysregulation. This increases the risk for obesity, and abnormal insulin/leptin responses. Thus, the next generation is setup for diabetes, obesity, metabolic syndrome and all their complications.

Placental Cytokines: A Link Between Maternal and Neonatal Obesity

Sylvi Hauget-de Mouzon, PhD

Dr Hauget-de Mouzon questioned if inflammatory cytokines regulate fetal adiposity. The answer appears to be yes. Adipokines that are in the placenta are leptin, TNF-alpha, and interleukins. Maternal cytokines do not appear to cross the placenta but fetal cytokines pass to the maternal circulation. With maternal obesity a cluster of genes in the placenta changes the inflammatory pathways. These genes are enhanced and results in a 2 fold increase macrophage accumulation in the placenta with obesity. The fetal percent of body fat increases in response to this cytokines/macrophage activity. So, my question is--should we be counseling all women to aim for ideal body weight before pregnancy not just women with DM or risk of GDM?

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Notes from San Diego, 2007

Diabetes and Pregnancy Study Group

M Mark Taslimi, MD **Stanford University**

Investigators from Denver reported on findings from muscle biopsies from GDM patients compared to normal glucose tolerance (NGT) patients. The muscles were tested for Insulin Receptor Substrate 1 (IRS1)-Serine. They found IRS1-Ser a marker of insulin resistance both during pregnancy and post partum.

Investigators from Kaiser, Southern California noted a 10% incidence of

GDM among >18,000 Asians analyzed. There were however, significant differences between Asian Indian nationals, with highest rate of 12%, (among women from India). The lowest rate was 5% among Japanese women.

Investigators from Denver provided a primate model that demonstrated fetal exposure to high lipid-derived fuels results in fetal hepatic re-programming

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GDM: The Forerunner for the Development of Maternal and Childhood Metabolic Syndrome?

Betty Vohr, MD

Women with a history of GDM are at risk for hypertension, hyperlipidemia, EKG abnormalities, and early death (O'Sullivan 1984). The 5 major features of metabolic syndrome are obesity, insulin resistance, dyslipidemia, hypertension and impaired glucose tolerance. The National Cholesterol Education Program added waist circumference for women of >35 inches, in 1999. So in women with GDM histories at 11 years post delivery the hazard of developing insulin resistance syndrome was 26 times higher than women that did not have GDM. Of course obesity was a major risk. These outcomes lead to a point where preventive medicine and lifestyle can impact the potential development of type 2 DM and CV events. In the Pima Indian studies we know that birth weight was associated with maternal glycemia and type 2 DM in the offspring (Franks, et al., 2006). Maternal obesity, birth weight and elevated glucose, predict obesity by age 5 (Scheafer-Graf, et al., 2005). Large for Gestational Age (LGA) offspring were at the highest risk for childhood obesity (Vohr et al, 1999). The metabolic syndrome was seen in 25 % of the LGA infants by age 11 (Boney et al, 2005). The obesity risk was 35 % if they were LGA and GDM infants within a 5-year observation period (Boney et al, 2005). Interestingly maternal obesity, GDM and LGA were independent risk factors for metabolic syndrome.



The Sweet Success Finished Product

Maternal Obesity and GDM: Long Term Consequences for the Offspring

Patrick Catalano, MD

Seventy percent of the fetal growth is in the 3rd trimester. Pregravid maternal weight is a significant predictor of birth weight and long-term risk of obesity. So, GDM is not the only risk factor. GDM women also have more disordered lipids and amino acid metabolism. The lipid and amino acid derangements are greater with obesity. Children of women with both obesity and GDM had higher fat mass in comparison to children of lean or non-GDM mothers, even with the same birth weights. So, maternal obesity and abnormal metabolic environment lead to fetal obesity then to childhood obesity and metabolic syndrome, then to type 2 DM as an adult.

Obesity: Scope of the Problem

F. Xavier Pi-Sunyer, MD

Obesity is an unintended result of economic, social and technologic changes. Obesity is increasing not only in adults but also children. Sixty percent of American adults are obese. Obesity trends are higher in ethnic subgroups. Insulin sensitivity decreases with increasing BMI. With obesity 5-6 times more insulin is needed to maintain normal fasting blood glucose. Abdominal (visceral) fat distribution is a problem since abdominal fat releases free fatty acids so the liver needs to increase uptake and must stop glucose uptake. This affects the muscle uptake of glucose and increases insulin resistance. The rate of metabolic syndrome has paralleled the increase in obesity. Other increased risks are for dyslipidemias, hypertension, cardiovascular disease, some cancers and much more.

Obesity and Diabetes: A Recipe for Obstetric Complications

Barak M. Rosen, MD

Increasing BMI increased c-section rates, instrumental deliveries, shoulder dystocias, postpartum hemorrhage, preterm deliveries, stillbirth rates, neonatal death (<7 days),

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macrosomia, LGA, GDM, preeclampsia, hypertension, wound infection, and intensive care days (Cedergren, 2004 and Sebire et al, 2001). Obesity and diabetes compounded the risk. Good control of obese women with GDM may mitigate some of the risk (Langer et al., 2005). In Langer's work the women and offspring, treated with medications did better, then women treated with diet alone.

Congenital Defects and Links to Obesity

E. Albert Reece, MD, PhD, MBA

Obesity alone is a risk factor for birth defects. Obese women see 3 times more neural tube defects and a doubling of cardiac defects. Folic acid has no effect on the obesity related neural tube defects. The birth defect rate in the normal U.S. population is 2-3%. In the DM population it has remained at 6-10%. In DM pregnancies the most frequent birth defects are neural tube, cardiovascular, gastrointestinal, genitourinary and skeletal. High glucose inflicts injuries by damaging cell membranes and causing biochemical reactions (reactive oxygen species-ROS). Antioxidants are natural scavenging agents and they remove ROS. Animal studies of vitamin E or C and some essential dietary fatty acids lowered the incidence of birth defects. The CDC had a study of 6000 women that demonstrated lowered birth defect rates in early pregnancy with multivitamins use versus those that did not use multivitamins.

Perinatal Outcome in Women with Type 2 DM

Ewa Wender-Ozegowska, MD, PhD

In her studies of women with type 2 diabetes, she saw type 2 women tended to come late to care, when compared with type 1 DM clients. They had higher essential hypertension and had increased preterm delivery rate.

Serum Lipid Profiles During Pregnancy Based on 35,000 Lipid grams

Arnon W Wiznitzer, MD

Total cholesterol, triglyceride, LDL and HDL increase in late pregnancy. Triglycerides above 327 mg%, total cholesterol above 299 mg % and LDL above 180 mg% were associated with adverse obstetrical

outcomes. These outcomes were an increased incidence of preeclampsia, GDM and preterm delivery. The lipid graph followed the insulin graph in these women. LDL increases postpartum. Lipids that were >2 standard deviations above the mean were associated with macrosomia and LGA.

Type 2 Diabetes: Pregnancy Outcome

Oded Langer, MD, PhD

There have been multiple studies with variable outcomes. FBS levels of 105-120 and post meals of 136-143 resulted in a birth defect rate of 1.2% versus FBS of 115-134 and post meal of 142-163 with a defect rate of 10.9 % (Kitzmilller, et al, 1991). Shafer-Graf (2000) found that FBS levels of <115 resulted in no birth defects. FBS of 141 equaled 1 organ affected, and FBS of 166 equaled 2 organs affected. Fuhrmann (1983-4) utilized mean blood glucoses. Means of <85 resulted in SGA, so adequate levels of glucose are needed for growth. A mean blood glucose of 95 resulted in 0.8% rate of birth defects. Mean glucoses of > 98 lead to LGA, macorsomia and stillbirths. Mean values of > 102 delayed lung maturation. Preeclampsia increased after a mean of > 104. Means of > 133 resulted in a 75% mortality and morbidity rate. Again, mean blood glucoses of >148 resulted in increased birth defects in another study. With post-meal values of 140 an increased incidence of LGA and CS occurred. Post meal values of 160 were associated with an increase in both spontaneous abortions and birth defects increased significantly. Type 2 DM had more macrosomia and LGA than Type 1 DM in all of these studies.

The Placenta as Conduit and Barrier Throughout Gestation

Richard K. Miller, PhD

The placenta serves several basic functions that can be disrupted at many stages. Primarily it is an anchor, conduit and controls passage of substances. As an anchor, disruption during implantation can result in increased rates of preeclampsia and poor growth. Diabetes increases the number of villus in

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**Notes From New York, 2007: 9th Annual Meeting of the
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comparison to normal pregnancies. The androgenic factors seen in PCOS can also affect implantation and placental development. Hypoxia affects some of the placental transporters. This changes how some substances cross the placenta and the amounts that cross the placenta. Hyperglycemia increases glycogen production and it is stored in the placenta by the fetus. This process buffers (or modulates) the effect of hyperglycemia as the placenta becomes fibrotic. Macrosomia increased after the placenta's storage capacity of glucagon was exceeded.

Transplacental Transfer of Glyburide, Metformin and Rosiglitazone
Tatiana Nanovskay, DDS, PhD

Many drugs have not been studied during pregnancy due to a lack of information on their safety for the fetus. Drug safety depends on drug concentrations, placental function and drug actions. Most drugs cross the placenta by passive diffusion. Molecular weight will affect passage. Glyburide has a high molecular weight and so does not cross the placenta easily. Plasma binding, placental proteins, and enzymes will affect transport. Both Glyburide and Rosiglitazone bind extensively to human albumin. Rosiglitazone even with binding did pass the placenta. Metformin does not bind, so it easily transports across the placental barrier. Metformin had the highest transfer rate. Both, Rosiglitazone and Metformin passed to the fetal side and could effect the fetus and or newborn.
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Notes from San Diego, 2007
Diabetes and Pregnancy Study Group
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and pediatric NAFLD (non-alcoholic fatty liver disease). These findings provide an explanation for the recent increase in pediatric fatty liver disease.

Investigators from USC and Germany showed a correlation of maternal BG levels with fetal abdominal circumference. They also demonstrated that adequate BG control during pregnancy is effective in slowing fetal overgrowth.

Investigators from Stanford showed that clinical risk factors detectable at the first diabetes appointment to diabetes and pregnancy care and counseling, can predict adverse maternal and fetal outcome.

Higher Maternal BMI increases the rates of macrosomia and un-planned C- section delivery. Type 1 & Type 2 DM are associated with increased rates of IUFD (intrauterine fetal demise) and NICU admission.

Investigators from Case Western provided data

that demonstrated that by 1 year of age infants born to mothers with pre-gravid BMI of <25 gain more fat than infants born to mothers with BMI \geq 25.

In a multi-center American study, linked exposure to maternal diabetes was a risk factor for Type 2 DM at ages 10-24 among Hispanics, Blacks, and Whites. These findings are similar to those seen with the Pima Indian studies.

Investigators from Seattle showed that women with glucose intolerance of pregnancy (with lesser degrees glucose abnormality than that needed to qualify for a diagnosis of GDM), are still at increased risk of developing subsequent diabetes.

In a multi-center American study, future development of metabolic syndrome was lowest if a women never had a pregnancy. This increased to 1.5 times greater in women who had normoglycemic pregnancies and it increased 2.6 times higher with a history of GDM.



Beyond the Numbers: First Trimester use of SSRIs and the Risk of Birth Defects

Charlene Canger, MFT, LCSW

SSRIs are the most frequently prescribed class of antidepressants in the general United States population and are the recommended class for use in pregnancy.

Recently, two large studies published in the *New England Journal of Medicine* (June 27, 2007) suggested further medical assurances that selective serotonin reuptake inhibitors (SSRIs), neither as a group nor individually, are major teratogens in newborns. Although SSRIs slightly increase the risk of birth defects, the chances appear remote and are confined to a few rare defects. The two studies originate from well-established, long-term, multi-site projects. Both studies add to the unfolding story of both a mother's and professional's understanding of medication treatment for major depression in pregnancy and its effects on a developing fetus. Each pregnancy has a 3 per cent risk of a major birth defect regardless of exposure. Likewise a woman's lifetime risk for major depression is 10-25%, with the highest prevalence during her childbearing period of 18-44 years of age.

The Center for Disease Control and Prevention's (CDC) study looked at 9,622 infants with major birth defects and 4,092 infants without major birth defects. They found no increased risk when all SSRIs were studied together. The data was obtained from the CDC funded National Birth Defects Prevention Study, one of the largest epidemiological efforts in the United States to identify causes of birth defects. This study looked at four common SSRIs used to treat depression- fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil), and citalopram (Celexa) and 18 categories of birth defects.

Available data on the overall safety of SSRIs in pregnancy have been limited. The women studied were on antidepressants the month prior to conception and/or during organogenesis. Included in the findings were ventricular septal defects, which were associated with SSRI use, notable paroxetine (Paxil), in previous studies. Researchers did find first time associations between SSRI use and anencephaly, craniosynostosis, and gastroschisis. No comparison was done between the effects of untreated depression compared with depression

treated with SSRIs. CDC cited plans to continue studying the correlation to clarify if a true risk is evident as there were insufficient cases included and a lack of information about duration or dosage of the prescribed antidepressants.

The Slone Epidemiology Center Birth Defects Study authored by Carol Louik et al., examined associations between mothers using SSRIs in their first-trimester of pregnancy and the risk of birth defects among 9849 infants with birth defects and 5860 infants without birth defects. Approximately 10% of the pregnant women were affected by depression and were treated with antidepressants. In this study, defects previously associated with SSRI use were not associated with overall use of SSRIs. However when the risk between specific defects and individual SSRIs were examined, they did find significant associations between sertraline and omphalocele, and between paroxetine and septal defects, as well as right ventricular outflow tract obstruction defects. The authors conclude that their findings do not show increased risk with overall SSRI use although individual SSRIs may have increased risk for specific defects but they are very rare and absolute risks are small.

Michael Greene, MD, from Maternal-Fetal Medicine at Massachusetts General Hospital, wrote an editorial for these two publications and he noted that both women and professionals would prefer clear distinctions between "risk" and "no risk" and would prefer that all studies gave consistent results. Rather "the two reports in the 356 issue of the *Journal* (NEJM), together with other available information, do suggest that any increased risks of these malformations in association with the use of SSRIs are likely to be small in terms of absolute risks".

References

Greene, MF (2007). Teratogenicity of SSRIs—serious concern or much ado about little? *NEJM* 356:2732-2733.

Alwan S, et al. National Birth Defects Prevention Study (2007). Use of serotonin-reuptake inhibitors in pregnancy and the risk of birth defects. *NEJM* 356: 2684-2692

Louik C, et al. The Slone Epidemiology Center Birth Defects Study (2007). Use of selective serotonin-reuptake inhibitors in pregnancy and the risk of birth defects. *NEJM* 356: 2675-83.

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Notes from Chicago: American Diabetes Association 67th Scientific Sessions

Leona Dang-Kilduff, RN, MSN, CDE

Hyperglycemia and Adverse Pregnancy Outcomes (HAPO)

Boyd Metzger, Chair; Lynn Lowe; Jeremy Oats; Bengt Persson; and Moshe Hod

The diagnosis of GDM served as a basis for studies that identified the criteria for future development of type 2 DM. It did not identify what level of glycemia resulted in poor perinatal outcomes. HAPO is a multicentered international observational study, which ultimately followed 23,325 women that had less than the diagnostic levels on a 2-hour 75-gram OGTT. The goal of this study was to identify at what level of maternal glycemia adverse perinatal outcomes occurred. HAPO found that maternal glucoses below the present diagnostic levels were accountable for adverse outcomes.

A continuous association of adverse pregnancy outcomes with maternal outcomes was seen from the lowest (75 mg/dl FBS) to highest levels (<105 mg/dl FBS). These higher levels resulted in large babies (>10% based on population), primary CS and newborn hypoglycemia including cord C-peptide levels. The chance of having a large baby was 10 times higher in the highest glycemic level over the lowest level. Other outcomes that associated with increasing maternal glycemic levels were neonatal anthropometrics, NICU or SIC admission, RDS (Respiratory Distress Syndrome), low 5 minute apgars, the incidence of HTN of pregnancy, including preeclampsia and eclampsia, and maternal hemorrhage. Since the level of complications were continuous, no specific glycemic level was obvious as a cut point for initiating care.

A Conference to translate the results of this study is in the planning stages and is expected to occur in the Spring of 2008.

Prevention of Type 2 Diabetes

Jaakko Tuomilehto

Long-Term Follow-up

Target areas were:

- Weight Reduction--5% wt reduction = 66% reduction in DM
- Increase Fiber to >15 grams per day
- And decrease fat intake
- Increase exercise to 30 minutes per day, 5 days

per week

- Medications for insulin resistance (Metformin) Long-term follow-up demonstrated that when 2 of the above factors were implemented, there was an 8% decreased incidence of DM versus no interventions. Four to five intervention components resulted in no DM. In the DPP intervention group, after 5 years, there was still 40% less DM in the intervention group than in the control group. Under the age of 50 the interventions were less effective versus interventions in the over 50 age group. In our population of younger women, should we be recommending medications also vs. life style alone?

Thiazolidnediones-Emphasis on DREAM Results Hertzl Gerstein

Dream trial- used TZDs / ramrapril

With use of a TZD (specifically Rosiglitazone), there was a decrease: in BG (9 mg/dl FBS and 28mg/dl at 2 hrs PP); in lipids; adipocytes; free fatty acids; beta cell loss or protection; blood pressure; 60% decrease in DM; A1c by 1-1.5%; insulin resistance; albuminuria; CRP (inflammatory marker); endothelial thickness; vascular reactivity; NAFLD (fatty liver); and dementia. There was an increase in adiponectin (good); weight with rosiglitazone but less in visceral fat (central), improvement in waist hip ratio. There are however increases in heart failure; fluid retention; anemia; liver dysfunction; low density lipoproteins (LDL) and triglycerides (TG); and reports of increased macular edema.

How Should Prevention Be Implemented?

Bernard Zinman

Basically treat the causes. If there is a high fasting BG that means there is hepatic insulin resistance, usually with a normal or slight decrease in first phase insulin release. Post meal defect or pre-diabetes has less of a hepatic insulin resistance and has a decreased first phase and possible second phase insulin release. So how should we select medications and interventions to target the associated defects of at-risk women?

First treat elevated lipids. These should be treated with lipid lowering agents and meal plan. Omega-3 fatty acids was the only alternative for women that

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Notes from Chicago: American Diabetes Association 67th Scientific Sessions

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might be planning a pregnancy. Other interventions that should be considered are any that postpone complications, and both micro and macro vascular disease. These interventions that preserve beta cell function such as TZDs; encourage weight reduction; medication treatments (Metformin and or Acarbose); life style-exercise 30 minutes/5 days per week; and screening and diagnosis, so complications do not occur before diagnosis is made. Recommendations are to implement lifestyle changes which are better than Metformin alone. Would the addition of other agents such as Acarbose, or TZDs, or ACEs and/or some combination of these be more effective? Lifestyle is more effective in the populations over 50. For our women should we be adding a medication after they stop breastfeeding? Are we doing them a disservice by not being more aggressive in prevention for this younger group of at-risk women?

Glucose Screening recommendations are:
ADA > age 45 or if high risk, every 3 years
CDA > age 40 and more frequently if high risk
ACE > age 30 or high risk

Average blood glucose to Replace A1C?

The thinking behind this is that if labs express the A1C as an Average Glucose "AG" or mean blood sugar and not a percent, (as it is now), it would make counseling with this value more useful in clinical practice. It would also relate to the CGMS technology and average glucoses seen in meter technology.

Dyslipidemia in Pregnancy—Implications for the Mother and Fetus

Emilio Herrera; Robert Knopp; Patrick Catalano; Gernot Desoye

Changes in the maternal adipose tissue have a significant effect on the fetus, and the mother's metabolism. During the first half of pregnancy the mother is in an anabolic state (storing fat). In the second half she is in a catabolic state (breaking fat down). This is important in feeding the fetus. In early pregnancy the major changes are that the pancreas increases insulin production, insulin sensitivity increases, the synthesis of fatty acids increases, and glycerol from glucose is seen.

In later pregnancy (catabolic state), the stored adipose tissue converts to free fatty acids, lipolytic activity increases. There are decreases in adipose tissue adiponectin, insulin stimulated tyrosine phosphorylation, and receptor substrates (IRS-1), resulting in insulin resistance and breakdown of fat deposits. The fat deposits are broken down mainly as long-chain polyunsaturated fatty acids.

Pregnancy increases TG (2-3 fold), cholesterol increases which peaks at 20 weeks at 20-30%, high-density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) all increase by 40 % or more. The placenta increases the transport of the above fatty acids (FFA), thereby effecting fetal growth. Women with abnormal lipid metabolism may need to modify their meal plan to limit CHO intake which would increase TG production. Fish oil or DHA may also be utilized to control abnormal lipid metabolism. The usual medications utilized for abnormal lipid metabolism can't be used during pregnancy.

GDM alone exaggerates the lipid metabolism and potentates fetal adipose accumulation. Increased fatty acids in maternal diet increased adipose in the fetus. FFA correlates with insulin resistance. The more insulin resistance the more FFA are available. Underfed women had decreased adipose in the fetus and decreased insulin sensitivity.

So what does all this mean? First glucose changes into fatty acids, so higher glucose means higher FFA leading to increased fetal weight. Women who had higher BMI pre-pregnancy had more FFA available so this in turn increased the fetal fat accumulation. All of this leads to babies that are fatter and at risk for obesity and diabetes. Maternal obesity alone caused an over expression of genes in the placenta that effect lipid metabolism and increase the insulin resistance. This is even more exaggerated with GDM when added on top of the maternal obesity effect.



In the next newsletter issue look for continued SUMMARIES of the 67th SCIENTIFIC SESSIONS and the 5th INTERNATIONAL GDM SESSIONS publication.

SWEET SUCCESS
NEWSLETTER FOR
DIABETES AND
PREGNANCY

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CALIFORNIA DIABETES AND PREGNANCY PROGRAM

On the web at:
www.llu.edu/llumc/sweetsuccess

Conferences/Events

Sweet Success Affiliate Training – Preceptorships. July 25, 2007; or August 1, 2007; or August 22, 2007; or September 5, 2007.

Each session can accommodate 3 people of different disciplines. For more information please contact Ramona at 916-208-2811.

August 1 - 4, 2007. American Association of Diabetes Educators. 34th Annual Meeting. St. Louis, Missouri.

September 18-19, 2007. Diabetes and Pregnancy Training. Region 8, California Diabetes and Pregnancy Program. For more information call: 714-456-6706

October 9, 2007. Meeting the Contraceptive Needs of the Woman with Diabetes. For information call 310-222-3651. Los Angeles, CA.

October 12,13,14, 2007. Lactation Professionals Conference 2007: Nurture Mind, Body and Spirit: Breastfeed! Sacramento, CA. For more information please call 925.754.1284 or email LLLConference@yahoo.com

October 25-27, 2007.

7th Annual Diabetes and Technology Meeting. American Diabetes Association. San Francisco, CA. For more information go to: professionaleducation@diabetes.org

November 1-3, 2007. Sweet Success 2007. Sweet Success 2007: Charting a Course for Excellence. Orange, CA For more information go to: www.sweetsuccessexpress.com/conferences.htm

Web Sites of Interest

Organization of Teratology Information Specialist
http://otispregnancy.org/otis_links.asp

For information on Asian Indian clients an excellent site is: www.pamf.org/southasian. Our own Geetha Desai, RD contributed to this sites development.

Are you developing patient education materials? Look at: www.hsph.harvard.edu/healthliteracy/

California Diabetes and Pregnancy Program

