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| Impaired Carbohydrate Metabolism, Insulin Resistance,  Improving our Health and Normalizing Bodyweight |  | For Rog.....  Bon Appetite & remember, it too,   is a work in progress and desperately needs subtitles and hyperlinks! |
| Title: New Information on Adult/Gestational Diabetic -- hypothesis -- it may be possible to prevent non-genetic macrosomic (too big unborn babies) and its pregnancy complications by identifying GDM / impaired carbohydrate metabolism and helping childbearing women (and others) learn how to manage better carbohydrate consumption. author ~ faith grunow gibson, LM,CPM | | |

 Important Quote: "Inadequate calories puts the body in a starvation mode. This induces a rise in blood sugars due to glycogen release by the liver. It also initially stimulates the release of the neurotransmitter seratonin which, unfortunately, is followed by a �crash�  -- that is, a "high" or rush followed by an intense deficiency of seratonin, which makes the dieter feel really bad. In addition, it resets the body's caloric thermostat to a lower setting so that the dieter either gains weight or fails to loose weight while restricting themselves to a much lower calorie diet "

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Navigational Shortcut: To [skip the medical stuff & get right to the heart of the matter](#gjdgxs)

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| Synopsis:  This information may permit us to reduce the number of neonates experiencing shoulder dystocia (SD) at birth and associated perinatal morbidity or mortality. A significant number of SD babies will suffer from respiratory and cardiac depression, admission to the NICU, or brachial nerve injury with Erbs palsey. Reduction of macrosomia is a goal worth pursuing. This new understanding of normal and impaired carbohydrate metabolism is also useful to the non-pregnant population (including ourselves, children, spouses and others).  It is an all too familiar story for home birth midwives -- a midwife we know and like and whose experience and skills we consider to be excellent -- had a bad outcome in what should have been a normal low risk home birth. One of the most frequent scenarios of this type is the 9, 10, or 11 pound baby with a pernicious shoulder dystocia, requiring massive intervention by the midwife to resolve and resulting in a baby born with a really low apgar (0 or 1). Resuscitation was necessary, often without success or perhaps the baby was kept on life support for several days before being determined to be in a vegetative state. Everyone involved is just devastated.  A frequent theme of these stories is that the mother had her last baby/babies at home without incident (good size but not huge) and so the family and the midwife are all understandably shocked. This shouldn�t have happened (but it did!). Numerically this is a rare occurrence but of that small number of adverse events for home birth families, a surprising number seem to involve macrosomic babies and its subsequent problems.  Obstetrical literature concludes that  shoulder dystocia can not be reliably predicted. More than 50% of cases occur in normal weight babies. For babies predicted to be macrosomic, more than 90% do not have SD. Ultrasound estimates of fetal weight can be wrong in both directions, by as much as 15%. This means a 10 pound baby may be estimated to be anywhere between 8 1/2 pounds to 11 and 1/2 pounds. Clearly screening is not able to reliably identify SD before the fact.  While midwives know many useful techniques for freeing the baby without damage, a small number of cases cannot be satisfactorily resolved. Being in the hospital would not have helped. There is no realistic surgical cure for SD (Zanvanelli maneuver/CS not withstanding!).  Further more, routine interventions of hospital care may make matters worse, especially if the mother is given drugs or anesthesia or forced to deliver on her back. Certainly the Gaskin Maneuver is a useful tool for resolving SD irrespective of the location of the birth, but it is not a magic bullet.  Consider what a blessing it would be if we could rather predictably prevent the very type of macrosomia that seems so often to be associated with SD. I don�t know for sure that what I am going to say will be able to live up to that promise but I am convinced that it has a high likelihood of helping, at the very least, to lower the number of undiagnosed and/or subclinical GDM macrosomic babies (the group most particularly at risk for SD). It also would permit us to identify and offer help to mothers who have impaired carbohydrate metabolism and therefore are at high risk for developing diabetes and its many complications sometime later in life.  The difficulties and cumulative dangers of Impaired Carbohydrate Metabolism   which eventually will lead to adult-onset or Type II diabetes  The medical and personal consequences of diabetics are legion. It is the leading cause of blindness in the US and it contributes to heart disease, kidney disease (and the resulting need for dialysis), amputation of lower limbs and for men, erectitle dysfunction and impotence. Childbearing women with insulin-dependent diabetes have greatly increased stillbirth rates. For women with GDM the major maternal complication during pregnancy is hypertension. For their babies it includes a small increase in stillbirth, macrosomia, shoulder dystocia, brachial nerve injury and higher rate of otherwise sick neonates. However, better identification of clinical GDM and good management of it antepartally has improved the circumstances for these babies.  With good care (maintaining of near-normal blood glucose levels) the rate of stillbirth is now equal for GDM mother to that of normoglycemic women.  Traditionally all forms of diabetics have been assumed to arise from insulin insufficiency. In juvenile onset diabetic this pathological condition is ascribed to the death, dearth or damage of beta cells in the  pancreas. In adult onset diabetes, medical authorities have promoted the theory of beta cell exhaustion, often due to overweight and an intemperate diet. In gestational diabetics the pathogenesis is assumed to be, in part, the result of the hormones of pregnancy which are antitheses to the action of insulin, in combination with some underlying genetic propensity to develop non-insulin dependent (NID) diabetics later in life.  The idea that some diabetics in both groups have or develop �insulin resistance� has been observed but that concept was poorly understood and thus had little effect on current treatment or managements of diabetics or its understanding by the public.  Classically midwives do not provide primarily care to insulin dependent diabetics. Most midwives also do not continue to provide primary services (planned labor and birth at home) to those mothers identified as having clinically diagnosed GDM. As for routine screening of all women for GDM, midwives have had a lot of resistance to doing this routinely, in part because it seemed to us that its identification resulted in a form of medical overkill. The definition of GDM appeared rather arbitrary (diagnosis and treatment for a woman with a postparadial blood glucose level of 141 but no Tx if her BG was 139 -- only one or two points lower).  Since the treatment of GDM is primarily calorie restrictions (which can be done independently of a diagnosis) this category appeared to accomplish little except to make everybody anxious. In fact, tight caloric restriction for GDM women often results in small for gestational age baby at birth and so seems to merely trade one possible problem for another.  Identification of GDM seemed to provide little  benefit to mother or baby and so midwives have had little interest in the subject. I�d like  propose that new information makes this a topic  one of great interest which may will permit is to make a very positive contribution to women. This benefit would surpasses the period of pregnancy and can even be useful to the general population (both male and female). In particular, this new understanding of non-insulin difficent diabetes (NIDD) would permit us to substantially reduce the previlence of macrosomic babies and the complications that accompany  these bigger babies. It would also help is to be assured that a mother with a big baby is genuienly (ie. genetically) appropriate rather than reflecting ICM/GDM.  The following information is presented as my personal opinion (as opposed results of RCT or other forms of research). This theory and expanded understanding as presented here was developed after becoming aware of some exciting new research by an endrocrenologist at Stanford University (Dr. Reagen), in conjunction with other sources of new material on adult diabetes by physician experts.  Taken together, this material dramatically changes the theory behind NID diabetes and its management, especially the identification of imparied carbohydrate metabolism (ICM) and suitable dietary and life-style recommendations to prevent it from turning into active type II diabetes/GDM.  In addition to very different dietary choices i also recommend the liberal use of self-monitoring of blood sugar levels at home by women, including antepartum, postpartum and for well-women gyn clients with risk factors for impared carbodrate metabolism (ICM).  The glycometer marked by Bayer (Glycometer Elite) is one of  the best currently available as it requies the least blood and it calibrates to same numbers as plasma so blood glucose values do not have to be adjusted.  The consenses of several authors) is a target level of blood sugars significany lower that those currently used by clinical labs to define �normal� for GDM screening. Last but not least, to properly uutilize these suggestions, each midwife must educate herself  independently about ICM/GDM and the glycemic index of foods.  A reading list is included at the end of this article. Many midwives and/or their family members also have characteristics of  ICM. One of the best ways to develop a thourgh understanding of these principles may be to keep track one�s own blood sugars and follow the dietary recommendation for 90 days and monitor the effect on blood glucose levels and general wellbeing. Reports of benefit include normalized blood sugars and reversale of frequent urination that accompanies hyperglycemia, weight loss specifically around the middle of the body (waist and upper hips) without any calorie restriction or calorie counting, improved health.  Topics to be covered and new information provided:  Pathogenesis of Impared Carbohydrate Metabolism/pre-diabetes/Gestational Diabetes M-----------  Updated understanding carbohydrate, lipid and protein metabolism  Phase I and Phase II insulin production, the Dawn Phenomonon  Glycemic Index and how it relates to ICM  Ideal blood sugar levels -- normal,  ICM and GDM  Techniques for hunger management without pain or strain  Dietary recommendations for ratio and timing of carbohydrates, protein and fats comsumption  Identifying importance and sources for �good� dietarly lipids, such as olive oil and real butter  Other symptoms of ICM -- Increased urination, intermittent visual problems, subjective feelings of hypoglycemia and gastroparesis (gastric distention, pain, sometimes progressing to intractable vomiting)  Self-testing for blood glucose, equipment and methods  The story unfolds  ~ Some basic background facts  Healthy adults with normal carbohydrate metabolism will have fasting blood sugars between 75 and 90. Even a high carbohydrate meal will only raise it by 20-40 points. At 2 hours the blood sugar of normoglycemic individuals will be under 120. The longer these people fast (for instance, not eating overnight), the lower their blood sugars will go but without becoming hypoglycemic (below 70). This occurs because they have an intact phase I insulin production in which small quanities of insulin are constantly present in the blood stream. In response to a carbo load phase II insulin production is triggered to meet the high need relative to intense carbohydrate metabolism.  Adults with lowered glucose tolerance/or impaired carbohydrate metabolism do not have or have lost the phase I insulin production. This results in increasing blood sugars overnite (often peaking at 4 in the morning) as their livers release the small quanities of glycogen that are part of normal physiology but the pancreas fails to do the same for insulin.  Their fasting blood sugars are typically in the high 90s or 100s. After eating, blood sugar levels rise very rapidly (absent phase I insulin already in blood stream) to greater than 140 (perhaps 180 or higher) and do not returned to under the 140 mark for longer than 2 hours.  These abnormal laboratory findings are often accompanied by carbohydrate craving, the need to urinate more often than �normal� ( >> 8+ times a day, a biological coping mechanism in responce to hyperglycemia as the body tries to rid itself of excess blood glucose through increased kidney perfusion), excess weight gain, especially around the midsection and an inability to lose weight easily despite caloric restriction. In extreme cases, episode of gastroparesis occur. Gastroparesis is an autotomic nervous system abnormality (vaso-vagal reflex) in which the stomach suddenly stops normal parastalsis. It stops emptying and begins to slowly fill with undigested food and liquids and gases intil the over-distention mechanically triggers intractable vomiting. If permicious vomiting continues for a long enough time (several hours) the patient faces circulatory collapse from dehydration and IVs will be necessary to prevent unconscienceness. It goes away as suddenly as it starts and may not reoccur for many months.  Current laborary values for a �normal� GTT are too high. They are up to 110 mgs for fasting, and 140 mgs for 2 hrs postprarandial. These upper limits actually represent ICM rather than healthy metabolism. Ideally fasting levels should be at or under 90 and 2 hr PP at or under 120.  It was recently recommended by the American Diabetic Associaiton that fasting levels be lowered to 95 and 2 hour to 130. These are more in line with efficient cargohydrate metabolism but still on the high side.  Reseach done at Stanford Medical School by Dr Reagen:  Dr Reagen�s research project divided test subjects into four categories -- those with normal glucose metabolism, those with significantly impaired glucose metabalism (pre-diabetic, very mild diabetes), diabetics with moderate disease and those with most severe NID diabetes.  Blood was drawn seven times -- fasting, 1, 2, 3, 4, 5 and 6 hours post prarandial. It was measured for both glucose and insulin levels.  Values were then graphed by these two parameters, in conjunction with the times they were drawn.  Conventional wisdom would dictate that normal subjects experience a rise in blood sugars and insulins levels that are concordant with BG rise. For those with evidence of diabetic disease, the insulin levels should fall behind the evidenced need as revealed by BG (insulin defficiency theory), thereby being lower than the nomoglycemic individual. According to this theory, it is the lack of insulin which results in eleavated blood glucose levels. However, Dr Reagen�s research revealed a startling divergency  from these expectations with regard to insulin levels, especially for the normal subjects and those with the mildest level of impared glucose metabolism/diabetes.  The normal subjests stated out with normal fasting blood sugars (90 or less), rose to a high of  only 120 at 2 hrs postprarandial and returned the general range of fasting (under 100) by about 3 hours. The impared glucose/mild diabetic group followed this same general pattern of blood sugar elevations but at a higher level of all values and a longer sloop for recovery. Their fasting blood glucose levels were all slightly higher -- about 100, 2 hour pp was about 130 and it took longer than 3 hours to return to near fasting levels, which for them were still slightly raised as compared to normal subjects (>100).  Based on BG level alone, there appeared to be little difference between these two groups, with the obvious exception that all BG values were higher.  However, a dramatic and unanticipated difference was revealed by the graph representing insulin levels, especialy for these two categories (normal and mildly impared). For the normal group, insulin levels pretty much tracted the blood sugar levels,  meaning they began low, rose to the highest point in the same ratio as the rise in blood sugar, and then returned again to about the same level as fasting.  According the insulin deficency theory, insulin levels in mild diabetics should start out even lower than those of normal subjects (showing deficency), with a very limited (ie. inadequate) rise as the blood sugars went up postpradially (inability to meet the increased need). However, that is not what happened at all. Insulin levels in the ICM/mild diabetic group start at approximately the same �normal� level and then sky-rocket to 4 or 5 times that of normoglycemics, slowly returning again to nearly normal. Graphically represented this is a huge and impressive spike -- like seeing the Transamerica building in the San Francisco skyline.  For the other two categories (moderate & severe diabetics), insulin level also rose but not as dramatically. None the less, moderate diabetics still tracked higher than the normoglycemic level and those of the most severe diabetics matched those of normoglycemic subjects. What this means is that diabetics is NOT a disease of insulin *difficency* but rather one of insulin resistance. At least one expert on impared carbodrate metabolism identifies increased abdominal fat distribution (�spare-tire�) as a marker of insulin resistance.  This is a very different pathogenisis than any of us would have suspected.  In addition to its crucial role in carbohydate metabolism, insulin is also a growth hormone. The unidentified GDM mother whose body produces 4 or 5 times the normal quanity of insulin after every meal obviously has a fetus which is regularly flooded with extremely high levels of growth-inducing insulin.  Furthermore, the women who flunks her 1 hour screening (blood sugar over 140) then passes her 3 hours GTT is very likely to be a woman with ICM in the early, �pre-diabetic� phase. The reason she has normal 3 hours levels is that she still has a massive production of insulin (4 to 5 times normal) which eventually brings it down to the �normal� level. While this normalizes the numbers for her blood glucose (and confuses the issue for her caregiver!), it also regularly exposes her baby to high level of  growth stimulating insulin and most likely, also triggers insulin resistance characteristics in the baby such as increase abdominal girth with a disproportionally large torso and big shoulders -- a setup for SD.  Carbohydrate rich diets (including the so-called �complex� carbohydrates) have been promoted as superior for both normal population and as the ideal diabetic diet. However this dietary regime  triggers greatly increased insulin productions for those with impaired glucose metabloism.  ----  Modern day carbohydrate metabolism and insulin resistance -- the effects of ancestry and genetic norms for those of northern European extraction, which consisted of a diet high in raindeer meat, fish, whale blubber and non-starcher tubers, roots, etc  Carbohydrate metabolism seems to be very specific to the ethnicity of the individual and reflects the genotype of that subgroup. The very idea of �impaired� carbohydrate metabolism is a culturally defined �norm� which reflects a specific cultural bias relative to major changes in modern-day human diets.  If you trace the history of the �normal� human diet back far enough you see various versions of  �hunter-gather� populations whose eating pattern could not be farther from that of the modern day American diet. In this natural world everything eaten was a �whole food� -- freshly killed small animals, fresh caught fish, birds, termites, other bugs, nuts, roots, other tubers and nonstrarchy vegatable, whole fruits (no orange juice!) and berries . It must be noted here that the human genome was originally molded by this wholesome diet which never, ever included any significant amount of refined sugars or starches (so called �complex� carbohydrates).  Simply changing to an agricultural-based diet is a massive change and not everyone�s genone survived this watershed event. It may be assumed that those individuals which encountered sever metabolic problems adjusting to agricultural diets became victims of selective breeding. This would assume that they died before they could reproduce so that those genes which were straighted jacketed to the hunter-gather diet died with these unfortunate folks.  However, there are many places in the world with populations that have survived intact on wholesome food diets. Many native or �indigeous� populations today get the majority of their calories from unrefined sources such as whole grains, non-strachy vegatables, large amount of fish, fish oils, or other animal meats. Likewise they eat virtually NO refined sugars, starches, stimulants or  alcoholic beverages  (potato chips, candy bars, twinkies, french fries, bread, pasta, crusants, crackers, ice cream, StarBucks coffee, beer, wine, hard liqour, etc.).  Interestingly enough,  the list of those ethnicities who are considered to be �at risk� for GDM, refects many of these same these populations who have until very recently enjoyed a �natural� or whole food diet  -- Asian, Australian aboriginy, Afro-American, American Indian, South and Central  American Indian, Hispanic, Pacific Islander, etc.  Specific aspects of the switch to an agricultural society:  There are many different grains which have been grown for the last several thousand years. Corn, rye, barley, wheat, oats, etc. It is from these �cereal� grains that we get breads, baked goods and pastas. The habit of pouring water over a stale loaf of bread so as to soften it for eating resulted in the acidental �discovery� of beer and other alcoholic beverages. The water that ran off each loaf was caught in a the same bucket and over the course of a few days of sitting around in the warm ambit air, the yeast naturally present in it �brewed� itself into an alcoholic drink.  (an intereting historical aside -- the letter �B� in Indo-Europen languages reflects the fact that bread, brew, beer and breasts all reflect some physical & visual aspect of a �bubble� or �bump� . In fact, most of our �B� words reflect that physical or visual quality -- band, beets, burr, barrel, body, back, belt, bend, bar, barf, ball, ballon, bomb, boat, bottle, barque??)  Agriculture gave rise to the mass production of cereal grains which were then ground into flours and used in a variety of breads, crackers and cereals. Refinement of cereal grains dramatically increases their glycemic index. Of all the grains commonally grown in the northern hemisphere, wheat flour is capable of the most refinement. This is why it is used for delicate pasties and why its added expenses denies it to very poor populations. It also has the highest glycemic index of all grains.  There is very little difference, glycemic index wise, between whole wheat flours and breads baked with them and those made with white flour. Both have equally astronomically high glycemic indices.  One way to think about this propensity for a high glycemic index based on how fine the flour is ground. Consider the difference between dipping our hand in water and �sprinking� water about (in rather big drops), versus a shaker bottle used to sprinkle clothes for ironing, versus a spray bottle such as used to spray Windex versus the water vabor truned into steam in our modern irons. Grinding wheat flour produces the dietary equlvilent of  a fine mist (whole wheat) or water vapor as steam (white pastry flour). Obviously the abilllity of the body to extract carbohydrate is maximized many times by this phenomon  which  produces a rapid extraction of glucose.  Glycemic index theory and meaning - the glycemic index of a food is how quickly it is converted to blood glucose. The index uses the conversion of  50 grams IV glucose as the bench mark a glycemic index of 100. Other foods/fluids are measured against that number (only dates exceed glucose, with a number of 106). Those with rates under 50 are considered to be �low� and those over 55 are considered relatively high. Examples are of differing glycemic indices are table sugar at 55, potatoes 93, lentils 30 and dahl (the germ part of lentils with the outer covering removed) are only 8. The higher the glycemic index of foods or beverages, the greater the stimulation of insulin production.  Liquids invaribly have a faster absorption rate than solids, ground grains higher than whole grains.  Glycemic index of an entire meal can be lowered by mixing low GI foods with those that are, by themselves, unacceptably high. For instance, a protein food (meat /fish/ cheese) a salad and non-starchy vegatable eaten with a starchy vegatable like mashed potatoes or white rice will have a moderate glycemic index and therefore is kind to the individual with ICM.  Most American diet experts at present recommend that  we consume about 50-60% complex carbodrates, not more than 30% fats/oils and about 20% proteins. Physician experts on ICM suggest a �balanced diets, in which these three elements are much more closely equal.  carbohydrate - cholesteral connection:  A number of authors have observed a connection between high complex/low fat diets and abnormal cholesterol production. Dr Swartzbein�s has a whole chaper (ch. 6) in her book explaining  the normal mechanism of cholesterol production. Acording to this information, the precursor dietary event to abnormal cholesterol levels are excess dietary carbohydrates. Among her diabetic and prediabetic patients, cholesterol levels fell when they stopped eating high levels of complex carbos (50/30/20) and switched to her recommendations for a �balanced� eating plan. In this instance, the word �balance� refers to an equal division of calories between the three major food types -- 40% carbohydrates, 35% healthy fats and oils and 25% protein.  Insulin resistance -- an imperfect definition of an imperfectly understood phenonem  This is a leading edge discussion as the current understanding of insulin resistance is imperfect and still evolving.  At present it is unable to be �treated� with any pharmiseutical agent which reverses the pathology as such as the way that insulin reverses diabetic hyperglycemia. The classical explaination focuses on the biological activity of insulin which is a hormone used by the body to �unlock� the cells so that glucose can be moved out of the blood stream and into the intracellar storage space to be available for future use.  Insulin resistance appears to reflect a situation in which the carbohrdrate storage is already at maximum capasity. In an effort to overcome this problem the body continues to pump out more and more insulin in a futile attemt to �unlock� this normal carbohydrate storage mechanism. If this continues on long enough, the pancreas seems to wear out. Also, the unconverted blood sugars eventually are converted to fat and stored as periumbilical deposits of adipose tissue.  So it appears that is not the insulin itself that is �resistant� but rather a storage mechanism which is already at capasity. (An alternative theory is the possibility  that in some cases the �lock� cells have become resistant to the biochamical effect of insulin). What is unknown but can be theorecially proposed is a probablity the each individual�s capasity for carbohydrate storage reflects both current dietary habits and their genotype. This may well be a place where genotyping can add valuable information for people in preventing chronic and debilitating diseases.  For people from genomic groups which have, until the last 100 years or so, eaten a �wholesome� diet, it seems likely that they have a carbohydrate storage capasity that is �normal� (ie very small!) for a wholesome diet  which has little or no refined foods, especially grains and sugars. However exposure to high level of refined foods fills up this small capasity and ultimatley we define them to have an �impaired� carbohydrate metabolism (in fact it is the diet and not the person who is faulty!).  This would apply to populations whose ancestors lived in non-agriculturally-based areas of the world (arctic/ antarctic circles), very cold regions such as the scandinavian countries,  who would naturally eat a large proportion of fish, fish oils (whale blubber!), meats, dairy products, etc). Many subgroups from these regions moved into more southern climes (Ireland, Scotland, Wales, England) a few hundred years ago and are in fact the antecedent population for the US. They surplanted a high protein/high fish oils diet for one that was very low protein and depended on potatoes, rye and other grains as the core food. In addition, lots of alcohol (often called �liquid bread�) is often consumed within these groups. It should be not a surprize that pregnant women from these ethnic groups have a much increased risk for babies with neural tube defects. Many in the general populations suffer from alcoholism.  A marker for ICM is a cultural propensity for alcoholism. Biochemically speaking, alcohol is a carbohydrate molecule with an extra atom on the molecular structure which increases its rate of absorption and its calorie count (carbohydrates are 4 per gram whereas alcohol is 7 per gram). Again one sees the association between populations that have relatively recent roots in wholeseome diets (Asian, Australian aboriginy, Afro-American, American Indian, Hispanic, South and Central  American Indian, Pacific Islander, etc) and alcoholism after adopting the typical western european diet of refined foods and easy access to alcohol.  Unfortunately, not all grains are equal in regard to the load they puts on charbodrate metabolism. This occurs because of the extremely high glycemic index of wheat flours.  Wheat flours and starchy vegatables vs simple sugars (candy, etc). Recent recommendations for a �healthy� diet have focused on a theorectical superiority of �complex� carbohydrates and a low fat, low protein diet which exchews free or simple sugars. The rationale for this has been the reduction of heart disease through lowered cholesterol levels. Many of the research finding which this advise was predicated on were arrived at before the distinction between high and low density lipids was made.  As an aside, the place in the world that has the lowest heart attack rate is also the place with the highest per capita rate of olive oil consumption (the island of Crete).  Whatever its origins and perceived benefits, it appears that the idea of a �complex� carbohydrate is a meaningful distinction only in the laboratory and only for chemists. For humans who chew and swallow foods, all of them (complex starches and simple sugars) are equal by the time they hit the stomach  -- that means that potatoes, white rice, pasta, whole wheat bread has as high (actually higher) glycemic index as table sugar. It is not the teaspoon of sugar added to the breakfast cereal that is the issue, it is the cereal itself that is high carbohydrate/high glycemic index. For the same reason, using Sweet and Low instead of real sugar is of no help and many think aspartame use creates problems of its own.  Also, when the total intake of carbohysrates is lowered, one must increase other sources of calories -- protein and oils being a rich source that is in alignment with the �wholesome� eating patterns of our northern European ancestors and indigenous peoples around the world.  Recommendations for community-based midwives:  Pay very close attention to body tpye and ethnicity in your client population. Follow the recommendation to have everyone screened for DGM (except for that tiny fraction of women with absolutely NO risk factors -- under 25 y/o, normal pre-pregnancy weight and normal pregnancy weight gain,  AGA fetus, no direct relative with known to have ICM/diabetes and not of the above listed ethnicities at increased risk - Asian, Austrailian aboriginies, Afro-American, American indian (north, south or central America), Hispanic, and Pacific Islanders).  Pay close attention to the results of GDM screening, using the tighter criteria of under 95 fasting and under 120 at 2 hours. Also note if fasting BG is under 70 as these women are at risk for SGS babies. Downs is also associated with chronically low BG levels.  Dietary teaching for ICM/GDM moms is essential.  Total calories for a pregnant woman of normal weight would be at least 2100/kal per day.  A most useful skill is the habit of reading labels of packaged foods for the quanity of carbohydrates. It is shocking to realize just how many grams are in the typical �complex� carbohydrate (bread - 14 grams, potatoes, pasta, rice, etc). The grams of carbohydrate in junk food such as coke (41 gms), cookies, candy bars) is truly shocking.  It is my personal bias that self-monitoring of BG level is necessary so that people can see for themselves the correlation between unwise consumption of starchy carbohydrates, the feeling of being unwell, high BG levels, (or hypoglycemia) and how long it takes to normalizing these cirsumstances after intemporate eating.  One of the major resistance to doing this is the fear/pain associated with blood drawing. However, new home monitoring systems which use a finger stick devise actually (really, cross my heart!) don�t hurt. Yes, it is a finger prick but not one that rises to the level one would consider �pain�. Bayer Glycometer Elite is the one i have had the best experience with. It uses the least amont of blood and beeps when has siphoned right amount of blood for test.  It  is important to wash one�s hands before doing a finger stick as any carbohydrate on the finger will skew the results (perhaps by double!).  Any reading that is abnormally high or low it should always be repearted. Occassionally an inadequate amount of blood will give a false reading of hypoglycemia. Make NO management determinations on a single high or low reading.  The typical diatary recommendations relative to a diet-related condition usually focus on percentages for each food group, permissable total of calories per day and perhaps, a set quanity of grams of each type of nutrients. I have choosen not to do that for a couple of reason. First, the information that i am communicating is not the end product of a research process. No RCT have ever been done (nor will they in all likelihood), nor are there any other types of level 1 or 2 studies.  Quite the contrary, this is  a �leading edge� in our understanding of ICM and at present, no hard and fast numbers for pregnancy are avaialbe. Equally important, a tight regeime of specific number for each type of food is not the way people think food or the way they eat on a regular and daily basis.   So what i am about to describe is a general theory relative to developing an improved (or normalized) dietary plan and the life-long process of changing one�s eating pattern to be more in alignment with their genotype.  Anyone interested in checking out these theories must do for themselves (or have done) a initial GTT (50 gm load if pregnant, 75 gram if not), followed by a week or 10 days of checking BGL.  Follow your �regular� eating pattern and do BG determinations every third day for morning fasting level, PPs (Xs 3) and one at bedtime. Only after establishing a base line for you and your regular diet can you reasonably access the effects of any changes.  One of my favorite authors is Christianne Northrup, a woman OB who lectures extensively on related dietary topics. Her recommendation is to consume what she describs as colored or  �rainbow� foods -- red, green, yellow and organge vegatables, brown rice, red, white and blue fruits, etc.  She shows a slide of what she calls the �white foods� -- white flour, sugar, rice, potatoes, pasta, etc�. What she says is �Now friends, think of these as condiments�. This is a point well taken. No food (with the exception of alcohol during pregnancy) should be completely eliminated from the diet.  However the relative ratio and quanities of food groups are significantly differnent.  Favorite foods which have high glycemic index or number of grams of carbohydrates can be eaten as a few bites. Early on one learns not to waste carbo calories on true junk foods -- save then for really good chocolates and a small piece of birthday cake.  General Principles:  Current recommendations  Hunger prevention -- perhaps the most important topic of all.    The best way to prevent intemporate eating is neither fasting, rigid regimes (the �Food Nazi� syndrome!) or caloric restrictions. Fasting stimulates glycogen release by liver --  meaning that it does NOT normalize (lower) blood sugars. Eating regular meals, (including planned snacks during  pregnancy), is necessary to help reduce hyperglyceria.  So the so-called �secret� or trick to normalizing the diet is eating lots of the right foods (those with a low to moderate glycemic index) so that one does not suffer the hunger and anxiety of rapidly rising and falling blood sugar levels. Not surprizing, breakfast is the place to start. The plan is to eat a relatively high protein breakfast so that you have a nice slow release of nutrients  and sustained blood glucose level over the next many hours. Then eat the next meal because its meal time. Its a good idea not to wait until one is  revenously hungry!  Protein and oils -- I am including protein and fats/oils in the same section as they are so frequently found together in our foods. And they both have developed a bad reputation (abet for different reasons) in the last few decades.  I have read in many popular publications that we Americans get too much protein. The idea has been promoted that we really don�t need to do anything special, protein wise, as virtually all foods have some small percentage of protein. We have been encouraged to eliminate any concern about eating a specific quanity of protein itself. The possible exception to this advise was for vegatarians, who have been urged to combine grains (rice and beans for instnace) in oder to get all 8 essential amino acids in the same meal. Perhaps this innate sufficiently of protein in the diet is true for some or even many folks. However, i know of several people who only appreciated how protein deficient their diet had been after trying this different approach.  This eating plan is designed to meet normal nutritional needs (no food group is eliminated) without unduly stimulating insulin production (ie keeping blood glucose levels nearly or at normal levels) and over time, diminishing insulin resistence.  Blood glucose lower is an immediate effect. However it requires a extended time of normalized blood glucose levels to positively impact on insulin resistance. Over-stuffed carbohydrate storage mechanisms must be unloaded by gradually utilizing those stored calories while not adding any more. So some effects are immediate while others are cumulative.  This is not accomplished counting calories but rather by  focusing at each meal on a more balanced intake of all three major food types -- protein, carbohydrates (with a strong showing of non-starchy vegatables) and fats or oils in the whole form (olive oil, butter, cream, cheese). Rather than giving a specific number of grams for each food type, i suggest that people simply think in terms of reducing complex carbo and simple sugars, consider non-starchy carbo as a �free� food (free of damaging side effects of starch carbos), and increasing the level of proteins and good fats/oils until people find themselves to be appropriately satiated most of the time. This means (perhaps for the first time), that you aren�t hungry again until the next meal time and you aren�t thinking of food (or nibbling) between times, except for a planned snack if the time between meals is unduly long.  Substances to restrict to small or occasional use:  Caffeine, tobacco, alcohol (eliminate if pregnant), aspertame, partially hydrinated oils (used by fast food resturants to deep fry foods such as french fries).  These very different ideas on diet produces a very different relationship with carbohydrate, especially those with no nurtitional content (vitamins, minerals, etc). A useful way to think about such things as candy and soft drinks are to see them in the same light a second-hand cigarette smoke -- extremely toxic and totally without socially redeaming value. This is not to say that we do not as individuals have an occassional bite or two (even three!) of candy, cake or a glass of wine on a special occassions. But it does balance off  the innate attractiveness of non-nutrient foods with the sobering reality of their biological  cost.  coke/pepsi 41 grams carbohydrate  Good fats/oils as precursors to healthy hormone production:  Most people know that the role of dietary protein is to repair and maintain body structures. That means rebuilding cells (for example muscle mass). It also is a crucial link in the production of neurotransmitters such as seratonin and other substances that help us feel healthy and happy.  Fats in particular are precuror nutrient for the production of the hormones that regulate every aspect of body function, from sexuality to mental function.  Inadaquate calories puts the body in a starvation mode. That includes a rise in blood sugars due to glycogen release by the liver. It also initially stimulates seratonin release which unfortunately is followed by a �crash� -- a high or rush followed by an intense dificiency. |
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