

## LIPID Profile and Growth Indicators among Offspring's of Diabetic Parents in Karachi, Pakistan

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### Abstract

**Background:** Type 2 diabetes mellitus has become a universal problem. Globally 80% of lower and middle income countries are suffering from diabetes. Different studies show that offspring from diabetic parents growth indicators were disturbed the lipid profile and growth indicators. The objective of the study was to determine the levels of lipid profile and growth indicators among offspring of diabetic parents.

**Material and methods:** Cross sectional study was done. Total 180 subjects were recruited from Dow university of Health sciences and classified as both parents were diabetic (BDP), Single Parents Diabetic (SDP) and no parents were diabetic (NDP). The Growth indicators lipid profile was measured. The analyst concentration of Lipids was analyzed by The Roche Hitachi Analyzer 902 Automated Analyzer automatically. Fasting blood sugar levels was determined by glucose oxidase.

**Results:** Those offspring who gave history of diabetes in parents had their growth indicators and Lipid Profile were disturbed or raised when compared with non -diabetic parents. 25.6%, 5.6% and 5.6% of offspring belongs to Both Parents Diabetic (BDP), Single Diabetic Parents (SDP) and Not Diabetic Parents (NDP) respectively have obese. Similar lipid profile indicators also raised as More BDP and SDP subjects had high cholesterol (>200 mg/dL) than NDP (76.9% and 29.6% versus 10% respectively), More BDP and SDP subjects had high cholesterol (>150 mg/dL) than NDP (23.1% and 2.8% versus 0% respectively). More BDP and SDP subjects had high cholesterol (>130 mg/dL) than NDP (76.9% and 29.6% versus 15% respectively).

**Conclusions:** Lipid profiles of offspring were related to diabetic parent's history. Early screening and change in lifestyle modification can be a preventive intervention for the risk of developing diabetes in future.

**Keywords:** Type 2DM; Family history; Lipid profile

### Background

Worldwide, the trend for incidence and prevalence of type 2 diabetes is rapidly increasing and by the year 2030 it is expected that the disease will effect 439 million adults [1-3]. A recent cross sectional study was conducted in Pakistan's rural and urban areas where 5433 individuals were recruited out of which almost 19% of the population showed prevalence of diabetes [4]. Multi system organ damage is seen in subjects with chronically elevated serum glucose levels [4]. New awareness strategies should be developed by the public health sector of the state for an intervention to prevent diabetes in the society. To achieve success in such a strategy, a widely applicable, easily measured index needs to be developed which has a high predictability.

Various risk factors and predictive models have been suggested. Genetic risk factors and inflammatory biomarkers are a new addition to the traditional risk factors, to allow us to predict diabetes in the population. However, these new novel markers have been reported to be of limited benefit [5-8]. By evaluating risk factors and genetic factors we can predict diabetes, but special biomarkers should be designed for specificity. The independent risk factor for developing type 2 diabetes was high fasting plasma glucose levels, even in subjects who were within euglycemic range [9,10]. Thus risk factors such as estimating fasting plasma glucose levels and deranged lipid profile are key indicators for development of diabetes [11].

Studies comparing monozygotic/dizygotic twins and normal population with family history of type 2 diabetes have shown a preference

towards the hereditary basis for insulin resistance and impaired insulin secretion mechanism [11]. Offspring with single parent have a 3-5 time risk of developing DM whereas offspring with both diabetic parents have 6 times the risk for developing DM. Early age for onset of diabetes is noted in offspring with strong family background of diabetes [12]. This study's main objective was to determine the role of diabetic parentage on lipid profile and growth indicators within their offspring.

### Methodology

#### Study area, design and period, sampling technique, sample size

Participants were inducted from different campuses of Dow University of Health Sciences Karachi, (DUHS) eg National Institute of Diabetes and Endocrinology (NIDE), Dow College of Pharmacy and Institute of Nursing.

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Cross-sectional study was conducted and the subjects were classified according to their parent's family history, All participants who had were defined as NDP (no diabetic parents), SDP (Single diabetic parent) BDP (both diabetic parents).

The study was carried from 2010 to 2011. Non probability convenient samplings were used. Sample size is 180, calculated for a confidence interval of 95% and a 5% margin of error. It was estimated by taking the % frequency of diabetes as 13.5% in our population [13]. In this study the inclusion criteria was adults' age between 18-24 yrs with no history of medical problems or any recent or remote diseases. All participants who had NDP (no T2DM parents), SDP (Single T2DM parent) BDP (both diabetic parents).Exclusion criteria was subjects with H/O diabetes or any known endocrinopathies. BMI and WHR were calculated by Asian –Pacific cutoffs [14] .The criterion for fasting plasma glucose was <100 mg/dl and impaired fasting glucose was above 125 mg/dl or (5.6-6.9 mmol/l) according to DDRL {Dow Diagnostic Research laboratories}..

### Instrument and data collection

Participants were provided with a questionnaire and get their consent as well as the information needed; age, gender, history of diabetes in either or both parents, medical history and personal habits. The participants were instructed to come in fasting condition (12 hrs) to Dow Diagnostic Research Laboratories (DDRL) Ojha Campus. Batches of 20 subjects were inducted each time for anthropometric measurements (height, Wt, Waist-Hip circumference) and blood collection (12 ml). Metabolic and Biochemical parameters were assessed. The Weight was recorded by the Stadiometer. Participant stood on center of platform to distribute their weight otherwise weight data is affected. Height was measured by Stadiometer's head piece. Waist circumference (cm) was measured at a level between the lower rib margin and iliac crest with the tape all around the body in horizontal position. Measurer should stand at side of participant. Hip measurement was done at fullest point at buttocks in (cm). Blood pressure was recorded.

For estimation of glucose and lipid profile the blood was centrifuged in HERMLE 2323 centrifuge for 10 minutes and shifted to ROCHE HITACHI 902 AUTOMATED ANALYZER. The working Principle of Hitachi 902 (Photometer) analyzer uses the photometric technique of glucose estimation.

### Data management and analysis

Data were entered and analyzed using SPSS16.0, Means, Standard deviation were calculated and means were compared across all three groups using one way anova (ANOVA) after findings of significance values, a multiple comparison between pair of means were made using, Scheffe method of pair wise comparison, a p-value <0.05 was considered as significant as differences in means of two groups. Correlations were measured by Pearson correlation method.

### Ethical Consideration

The study was approved by ethical committee of Dow University of Health Sciences Karachi Pakistan.

### Results

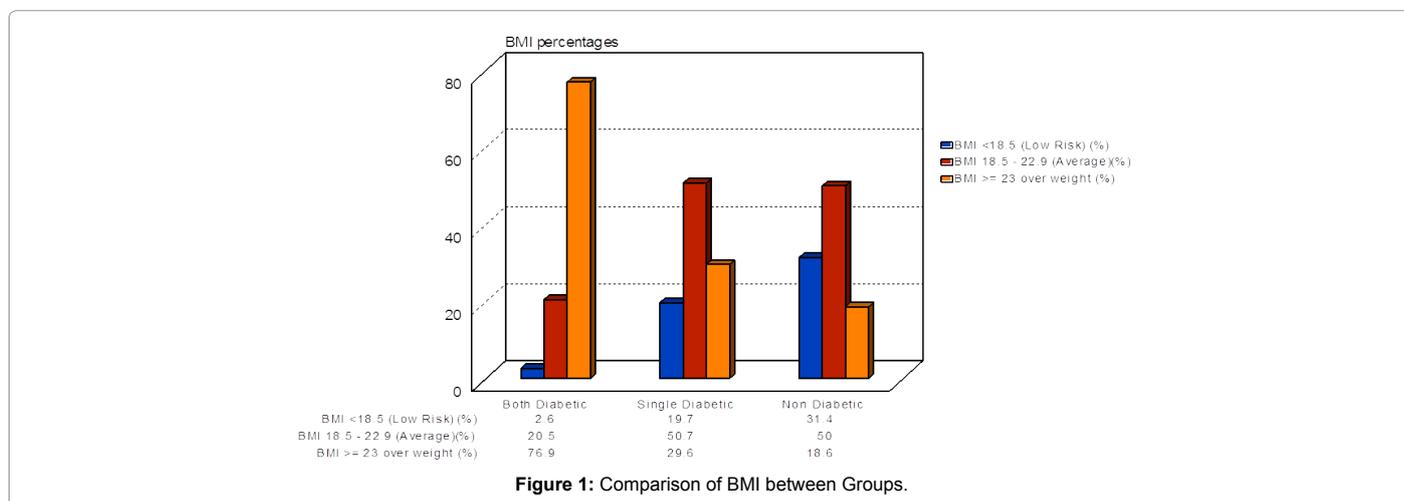
The BMI for BDP and SDP groups were significantly greater than the NDP group (p<0.05, Table 1).

In Figure 1 the frequency of BMI in offspring of BDP, SDP and NDP in underweight (BMI<18.5) was 2.6%, 19.7% and 31.4%, in normal weight (BMI, 18.5-22.9) it was 20.5%, 50.7% and 50% and in overweight (BMI ≥ 23) it was 76.9%, 29.6% and 18.6% respectively .BDP offspring showed high WHR for males i.e.14.35%, 37.50% for females, and SDP offspring had 23.1% in males and 30.4% in females, whereas offspring of NDP had a normal WHR(Waist hip ratio) (Table 1). The diastolic BP (mm Hg) was significantly higher among BDP (80.25 ± 7.4; p<0.05) as compared to both SDP (74.08 ± 5.9) and NDP (73.85 ± 6.4), whereas the

Parameters	BDP n=39 mean(SD)		SDP n=71 mean(SD)		NDP n=70 mean(SD)	
	Male	Female	Male	Female	Male	Female
Body Weight (Kg)	70.38 ± 10.78		57.27 ± 14.33		57.03 ± 10.17	
BMI (Kg/m <sup>2</sup> )	25.58 ± 5.15		22.26 ± 6.80		21.02 ± 6.19	
Systolic Blood Pressure (mmHg)	116.53 ± 5.51		113.52 ± 7.57		113.00 ± 7.29	
Diastolic Blood Pressure (mmHg)	80.25 ± 7.42		74.08 ± 5.99		73.85 ± 6.43	
Waist /Hip Ratio At High Risk	14.3 %	37.5%	23.1%	30.4%	0.0%	0.0%

BMI: Body Mass Index

**Table 1:** Physical characteristics of offspring of Both Diabetic (BDP), Single Diabetic(SDP) and Non Diabetic Parents(NDP).



**Figure 1:** Comparison of BMI between Groups.

systolic BP was significantly higher among BDP only when compared to NDP (p<0.05) and not with SDP (Table 4).

Fasting Plasma Glucose (FPG) were in normal range in all three groups but the mean FPG level was higher in BDP as compared to SDP and NDP (p- value 0.05) (Table 2).

Parameters	BDP n=39 Mean (SD)	SDP n=71 Mean (SD)	NDP n=70 Mean (SD)
Fasting Plasma Glucose	4.73 ± 0.75	4.65 ± 0.47	3.61 ± 0.24
Serum Cholesterol	146.91 ± 24.51	19.04 ± 14.57	15.55 ± 13.49
Serum Triglycerides	138.84 ± 109.75	77.35 ± 29.87	66.33 ± 29.58
LDL mg/dl	111.28 ± 32.73	103.14 ± 24.17	95.83 ± 29.32
HDL mg/dl	44.48 ± 10.52	46.89 ± 20.48	48.98 ± 8.43
Chol:HDL	4.07 ± 1.35	3.24 ± 0.68	3.45 ± 1.07

LDL: Low Density Cholesterol; HDL: High Density Cholesterol

**Table 2:** Biochemical parameters of offspring of Both Diabetic (BDP), Single Diabetic (SDP) and Non Diabetic Parents (NDP).

Parameters	NDP VS SDP* p-value	NDP VS BDP* p-value	SDP VS BDP* p-value
Body Weight (Kg)	0.994	0.00	0.00
BMI (Kg/m <sup>2</sup> )	0.537	0.005	0.056
FPG (mmol/l)	0.701	0.451	0.874
Serum Cholesterol mg/dl	0.05	0.00	0.125
Serum Triglycerides mg/dl	0.978	0.000	0.000
LDL	0.13	0.356	0.025
Chol :HDL	0.495	0.000	0.000

\*vs. NDP, p<0.05, ? vs. SDP, p<0.05

**Table 3:** Sheffe' multiple pair wise comparison of physical and biochemical parameters.

Groups	NDP		SDP		BDP	
	r	P value	r	P value	r	P value
BMI-HDL Ratio	0.099	0.437	0.37	.002<0.05	0.11	0.952
Chol-HDL Ratio	0.193	0.110	0.685	.00<0.05	0.488	0.002
TG-HDL Ratio	0.292	.014<0.05	0.57	.00<0.05	0.635	0.00

**Table 4:** Intragroup correlation (Pearson) (r and P values) of physical and biochemical parameters.

Using the criterion established by DDRL, 5.1% and 4.2% of BDP and SDP offsprings, respectively, had impaired fasting glucose (Figure 2). According to Table 1 the WHR (Low Risk) for males is ≤ 0.95, and for females it is ≤ 0.80 .WHR (High Risk) is 1.0 in males and 0.85 in females.

In Table 1 the mean values for Serum Cholesterol was 171.61 ± 32.37, 159.38 ± 33.24 and 146.91 ± 24.51 in BDP, SDP and NDP. The mean values for Triglycerides (mg/dl) was in BPD (138.84 ± 109.75) as compared to SDP (75.33 ± 29.58) and NDP (77.35 ± 29.87) showing highly significant difference between values of NDP and BDP (p<0.05) as well as between those of SDP and BDP (p<0.05) but not with NDP and SDP (Table 3).

Table 2 shows that mean values for the LDL were significantly raised in BDP and SDP as compared to NDP.

In Table 3 the serum cholesterol p-value was <0.05 in NDP versus BDP and it was also <0.05 in SDP versus BDP. HDL was lower in BDP as compared to SDP and NDP. Serum cholesterol-HDL's P- value had a statistical significant difference<0.05 in BPD and SDP.

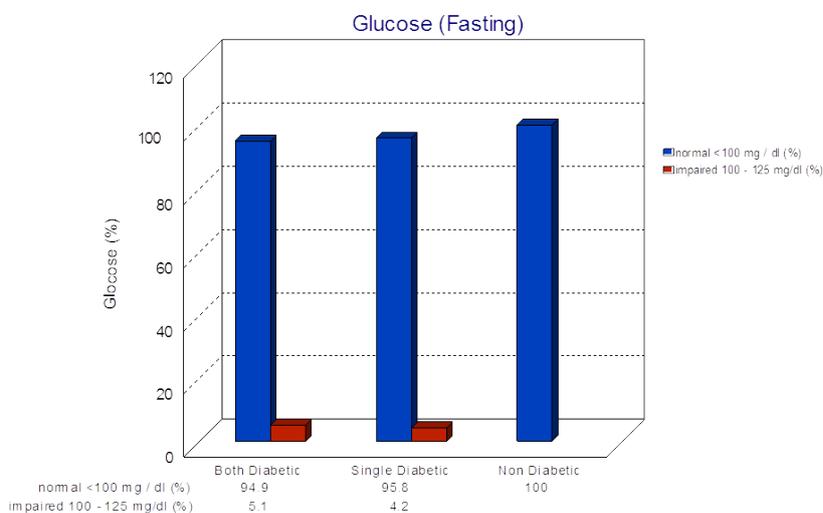
In Table 4 shows intra group relationship of physical and biochemical parameters. Cholestrol HDI ratio and TG and HDL ratio were significantly differ between SDP, BDP versus NDP.

Serum Cholesterol level was 25.65 % in BDP while in SDP it is 5.6% (Figure 3). Serum Triglycerides>150mg/dl was found to be 23 % in BDP and 2.8% in SDP (Figure 3). LDL>130 mg/dl was found in 23.1%in BDP and 8.5% in SDP (Figure 5).

## Discussion

The study revealed that offspring of diabetic parents with raised disturbed growth indicators, Biochemical parameters such as Serum Cholesterol, Triglycerides also showed raised levels thus suggesting that they are the predicting factors for the onset of diabetes.

In a recent study in 2013 also evaluated the these predicting factors and they also found the levels raised in all the subjects lipid profile [15]. In this present study the waist and hip circumference and its ratio in offspring of both diabetes was also increased suggesting that there is metabolic disorders. WHR in addition to BMI has also been shown



**Figure 2:** Comparison of Fasting Glucose between Groups.

as the measure of obesity. It is however, increasingly known that for a given BMI, central rather than lower body fat distribution, leads to greater risk of metabolic and cardiovascular complications of obesity [16].

Schmidt et al. [17] also analyzed the ratio between waist and hip and considered it very crucial. Cassano et al. [18] also proved that abdominal fat was important in predicting onset of diabetes risk. Wei et al. [19] also confirmed importance of abdominal fat and waist circumference. The offsprings in my present study with presented with raised anthropometric parameters. Further, insulin resistance which may be a genetically inherited trait [20] is also known to enhance lipolytic activity thereby increasing fatty acid levels thus bringing about these altered changes in lipid profile and can also cause dyslipidemia in individuals with normal glucose tolerance [21].

In the present study mean values were markedly lower for HDL and higher for LDL in the groups (BDP and SDP) having family history of diabetes when compared to other group (NDP). It was also assessed that those with positive family history of diabetes showed significant p-value for serum cholesterol and LDL as compared to those who do not have diabetes in their families. Subjects with T2DM are refractory to insulin-stimulated glucose uptake by the target cells [22,23] and also prone to dyslipidemia especially increased triglyceride and decreased high-density lipoprotein (HDL) levels, hypertension and ischemic heart disease [24,25] (Figure 4). Body fat mass supply and deposition are due to multiple environmental and genetic factors. Obesity is linked with insulin resistance, hyperinsulinemia, and incident T2DM [26,27]. Furthermore insulin has an anabolic special effect on fat metabolism leading to fat deposition and obesity [28].

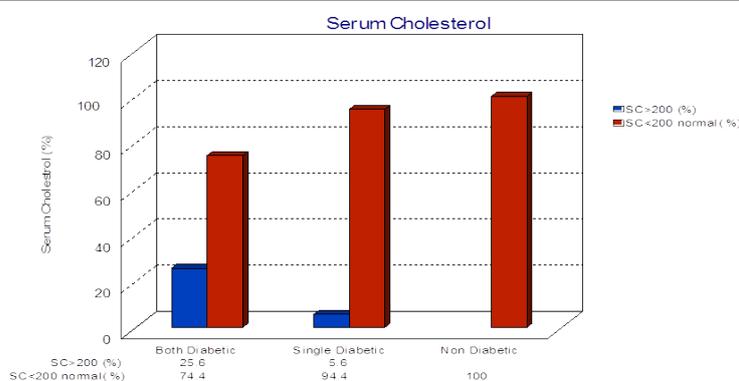


Figure 3: Comparison of Cholesterol between Groups.

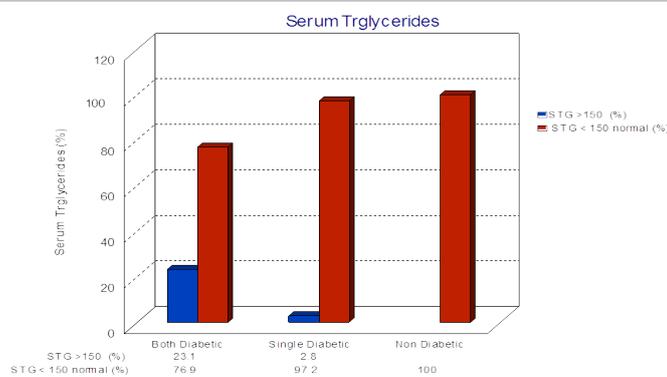


Figure 4: Comparison of Triglyceride between Groups.

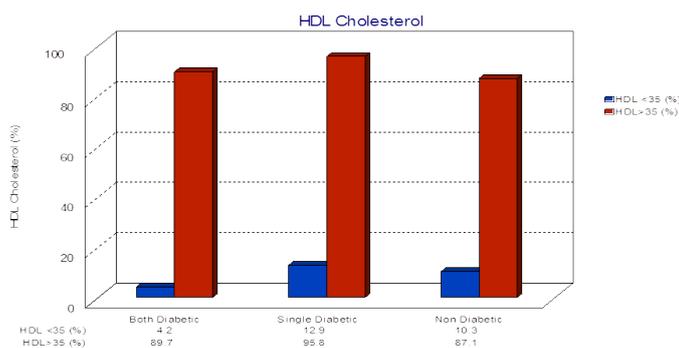


Figure 5: Comparisons for HDL between Groups.

Impaired Glucose Tolerance (IGT) was recognized in children, in the group with positive history of diabetes in their families because obesity in childhood has noteworthy increased in recent years [29,30] and it is strongly related with insulin resistance, the main public health policies are centered on screening obese children and youths. Weijnen et al. [31] researched that levels of insulin & lipid profile were more in those who gave a family history of diabetes & later development of obesity [31].

## Conclusions

Hyperlipidemia and anthropometric measurements was found to be raised in those who give history of diabetes in their parents suggesting that these parameters may be the factors for development of diabetes in future.

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## References

- Shaw JE, Sicree RA, Zimmet PZ (2010) Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* 87: 4-14.
- Yoon KH, Lee JH, Kim JW, Cho JH, Choi YH, et al. (2006) Epidemic obesity and type 2 diabetes in Asia. *Lancet* 368: 1681-1688.
- Task Force Team for Basic Statistical Study of Korean Diabetes Mellitus of Korean Diabetes Association, Parkle B, Kim J, Kim DJ, Chung CH, et al. (2013) Diabetes epidemics in Korea: reappraise nationwide survey of diabetes "diabetes in Korea 2007". *Diabetes Metab J* 37: 233-239.
- Shera AS, Jawad F, Maqsood A (2007) Prevalence of diabetes in Pakistan. *Diabetes Res Clin Pract* 76: 219-222.
- Dallmeier D, Larson MG, Wang N, Fontes JD, Benjamin EJ, et al. (2012) Addition of inflammatory biomarkers did not improve diabetes prediction in the community: the framingham heart study. *J Am Heart Assoc* 1: e000869.
- Kashima S, Inoue K, Matsumoto M, Akimoto K (2013) Do non-glycaemic markers add value to plasma glucose and hemoglobin a1c in predicting diabetes? Yuport health checkup center study. *PLoS One* 8: e66899.
- Echouffo-Tcheugui JB, Dieffenbach SD, Kengne AP (2013) Added value of novel circulating and genetic biomarkers in type 2 diabetes prediction: a systematic review. *Diabetes Res Clin Pract* 101: 255-269.
- Schulze MB, Weikert C, Pischon T, Bergmann MM, Al-Hasani H, et al. (2009) Use of multiple metabolic and genetic markers to improve the prediction of type 2 diabetes: the EPIC-Potsdam Study. *Diabetes Care* 32: 2116-2119.
- Tirosh A, Shai I, Tekes-Manova D, Israeli E, Pereg D, et al. (2005) Normal fasting plasma glucose levels and type 2 diabetes in young men. *N Engl J Med* 353: 1454-1462.
- Rhee SY, Woo JT (2011) The prediabetic period: review of clinical aspects. *Diabetes Metab J* 35: 107-116.
- Radha V, Mohan V (2007) Genetic predisposition to type 2 diabetes among Asian Indians. *Indian J Med Res* 125: 259-274.
- Mohan V (2004) Why are Indians more prone to diabetes? *J Assoc Physicians India* 52: 468-474.
- Shera AS, Rafique G, Khwaja IA, Ara J, Baqai S, et al. (1995) Pakistan national diabetes survey: prevalence of glucose intolerance and associated factors in Shikarpur, Sindh Province. *Diabet Med* 12: 1116-1121.
- Weisell RC (2002) Body mass index as an indicator of obesity. *Asia Pac J Clin Nutr* 11 Suppl 8: S681-684.
- Shobha MV, Ravindra PN, Deepali A (2013) Changes In Anthropometric And Lipid Profile In Healthy Young Offsprings Of Diabetics Are Not Temporally Linked. *Int J Med Health Sci* 2-1.
- Kissebah AH, Krakower GR (1994) Regional adiposity and morbidity. *Physiol Rev* 74: 761-811.
- Schmidt MI, Duncan BB, Canani LH, Karohl C, Chambless L (1992) Association of waist-hip ratio with diabetes mellitus. Strength and possible modifiers. *Diabetes Care* 15: 912-914.
- Cassano PA, Rosner B, Vokonas PS, Weiss ST (1992) Obesity and body fat distribution in relation to the incidence of non-insulin-dependent diabetes mellitus. A prospective cohort study of men in the normative aging study. *Am J Epidemiol* 136: 1474-1486.
- Wei M, Gaskill SP, Haffner SM, Stern MP (1997) Waist circumference as the best predictor of noninsulin dependent diabetes (NIDDM) compared to body mass index, waist / hip ratio and other anthropometric measurements in Mexican Americans 7-year prospective study. *Obes Res* 5: 16-23.
- Gerich JE (1998) The genetic basis of type 2 diabetes mellitus: impaired insulin secretion versus impaired insulin sensitivity. *Endocr Rev* 19: 491-503.
- Steinberger J, Daniels SR; American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee (Council on Cardiovascular Disease in the Young); American Heart Association Diabetes Committee (Council on Nutrition, Physical Activity, et al. (2003) Obesity, insulin resistance, diabetes, and cardiovascular risk in children: an American Heart Association scientific statement from the Atherosclerosis, Hypertension, and Obesity in the Young Committee (Council on Cardiovascular Disease in the Young) and the Diabetes Committee (Council on Nutrition, Physical Activity, and Metabolism). *Circulation* 107: 1448-1453.
- Reaven GM (1983) Insulin resistance in noninsulin-dependent diabetes mellitus. Does it exist and can it be measured? *Am J Med* 74: 3-17.
- DeFronzo RA1, Bonadonna RC, Ferrannini E (1992) Pathogenesis of NIDDM. A balanced overview. *Diabetes Care* 15: 318-368.
- Pyorala K, Savolainen E, Kaukola S, Haapakoski J (1985) Plasma insulin as coronary heart disease risk factor: relationship to other risk factors and predictive value during 9 1/2 year follow-up of the Helsinki Policeman population. *Acta Med Scand* 701: 38-52.
- Ducimetiere P, Eschwege E, Papoz L, Richard JL, Claude JR, et al. (1980) Relationship of plasma insulin levels to the incidence of myocardial infarction and coronary heart disease mortality in a middle-aged population. *Diabetologia* 19: 205-210.
- Ford ES, Williamson DF, Liu S (1997) Weight change and diabetes incidence: findings from a national cohort of US adults. *Am J Epidemiol* 146: 214-222.
- Resnick HE, Valsania P, Halter JB, Lin X (2000) Relation of weight gain and weight loss on subsequent diabetes risk in overweight adults. *J Epidemiol Community Health* 54: 596-602.
- Unger RH (2003) Lipid overload and overflow: metabolic trauma and the metabolic syndrome. *Trends Endocrinol Metab* 14: 398-403.
- Daniels SR, Arnett DK, Eckel RH, Gidding SS, Hayman LL, et al. (2005) Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. *Circulation* 111: 1999-2012.
- Slyper AH (2004) The pediatric obesity epidemic: causes and controversies. *J Clin Endocrinol Metab* 89: 2540-2547.
- Weijnen CF, Rich SS, Meigs JB, Krolewski AS, Warram JH (2002) Risk of diabetes in siblings of index cases with Type 2 diabetes: implications for genetic studies. *Diabet Med* 19: 41-50.

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