Original Resear	Volume-10 Issue-2 February - 2020 PRINT ISSN No. 2249 - 555X DOI : 10.36106/ijar
Stal Of Applice Elizable # 42102	Medicine PREVALENCE OF DIABETIC RISK FACTORS IN MEDICAL STUDENTS
Dr SP Miital*	Assoct Professor Medicine Department of Medicine. MM Medical College and Hospital Kumarhatti (Solan) - Himachal Pardesh (India) *Corresponding Author
Dr Kiranjeet Kaur	Professor of Biochemistry Department of Medicine. MM Medical College and Hospital Kumarhatti (Solan) - Himachal Pardesh (India)
	ound: India prosperity associated with rise in altered lifestyle and dietary habits, so also the metabolic diseases. mpted us to search risk factors in medical students.

Material and Method: 450 medical students were studied.

Results: 421 students (182 male and 239 female) had completed the study. Mean age of male (M) and female (F) students was 19 ± 1.128 . Diabetes mellitus (DM) was comparatively common in relatives of female students. 32.78% students had obese relatives, especially paternal diabetic, and both parental and maternal diabetic relatives, Hypertension (HTN) [M=13.74% & F=18.41%] and coronary artery disease (CAD). Underweight students were 48 [M=19 & F=29] and overweight 191 [M=91 & F=100]. 31.61% male students had waist \geq 90 cm and 32.42% female \geq 80 cm, and Waist – Hip Ratio (WHR) \geq 0.90 in 38.06% male and \geq 0.80 in 36.53% female. Low birthweight students were 26 (M=7 & F=19) and high birthweight 9 (M=5 & F=4). Systolic HTN was present in 31 (M=26 & F=5) and diastolic HTN in 32 (M=18 & F=14). One male was diabetic and 3 female had Glucose Intolerance and 4 had PCOD.

Conclusion: Students, particularly female and "normal-weight metabolically obese", were at risk of developing DM, obesity, HTN and CAD. Parental DM determined high birth weight. Most of female students were underweight, suggesting DM in lean subjects may shift from male to female. "Obesity paradox" present in students because of "metabolically unhealthy normal-weight".

KEYWORDS : Obesity, physical inactivity, "obesity paradox"

INTRODUCTION:

India is prospering, so associated increase burden of altered dietary and sedentary lifestyle affecting population with metabolic diseases. These appear now in young age. Other contributory factors to these diseases are: genetic propensity, "normal-weight lean mass", physical inactivity, dietary habits, poor nutrition in pregnancy, childhood obesity, westernized lifestyle etc. In medical students, problem is further compounded by stressful academic life. This study is to find out the risk factors for DM and other metabolic morbidities. We planned appropriate suggestions for the vulnerable students.

MATERIAL & METHOD:

This study was conducted in a territory Medical College. 450 medical students were enrolled.

Duration of Study: About One Year Inclusion criteria: All students. Exclusion criteria: nil.

Study design:

RESULTS:

Students were informed about the study. Those who consented were included and evaluated clinically. History of: symptoms of metabolic diseases, drug therapy (influencing metabolism), F/H/O chronic metabolic diseases. Clinical examination included Weight measurement (Kg), Height (cm), BMI, Waist (W) and Hip (H) circumference, W/H ratio. BMI for overweight was considered as 23Kg/m^2 (1), abdominal adiposity if waist circumference as >90 cm in male and >80 cm in female (2). Relevant biochemical tests were done. Bone mineral density (BMD) was calculated as an average of two readings each recorded for one minute, and classified (3): Normal: BMD score > -1, Osteopenia: BMD score < -1 and up to -2.5, Osteoporosis: BMD score <-2.5.

Ethical Issue: Institution Ethical Committee approved the study and conducted accordingly.

421 students (M=182 & F=239) completed the study. Mean age of either sex was 19 ± 1.128 years.

Family history (Table – 1): 191 (45.37%) students, especially female, had diabetic relatives [Female 122 (50.21%)] & Male 69 (37.91%)]. F/H/O obesity was present in 35.71% & 30.54%, HTN 13.74% & 18.41%, CAD 2.2% & 0.84% respectively in relatives of male and female students, and dyslipidemia (parental relative of 1 female).

Table - 1(Depiction of Disabilities in Family)

Family	Pate	rnal	Mat	ernal	Paternal &		Total
History	М	F	М	F	Maternal		
Diseases					М	F	
Diabetes mellitus	27	58	17	29	25	35	191 (45.37%) M= 69 (37.91%) F= 122 (50.21%)
Obesity	31	25	13	24	21	24	138 (32.78%) M=65 (35.71%) F=73 (30.54%)
Hypertension	19	35	1	5	5	4	69 (16.39%) M= 25 (13.74%) F= 44 (18.41%)
CAD	4	1	-	1	-	-	$\begin{array}{ccc} 6 & (1.43\%) \\ M = 4 & (2.2\%) \\ F = 2 & (0.84\%) \end{array}$
Dyslipidemia	-	1	-	-	-	-	F=1 (0.42%)

(N.B. M=male students, F = female students, CAD= coronary artery disease; Paternal family history = Father and paternal relatives of students; Maternal Family history = Mother and maternal relatives of students.)

Association of DM (Paternal) with obesity (Table–2): 85 students had parental DM relatives. Obesity was present more in paternal DM relatives.

Table - 2 (Association of Diabetes mellitus with Obesity, HTN and CAD in family)

N N N N N N N N N N N N N N N N N N N		•	• •			
Family history of DM and other co	Obesity in Paternal relatives		Obesity in Maternal relatives		Obesity in Paternal & Maternal relatives	
morbidities	М	F	M	F	M	F
Diabetes mellitus						
DM (Pat)						
N=85 (M=27, F=58)	7	15	4	5	6	7
DM (Mat)						
N=46 (M= 17, F=29)	3	5	1	5	6	5
DM (Pat & Matl						
N=60 (M= 25, F=35)	4	6	3	6	5	3

32 INDIAN JOURNAL OF APPLIED RESEARCH

Volume-10 | Issue-2 | February - 2020 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

volume-to issue-2 Pebruary - 2020 PKINI ISSIN No. 2249 - 5555A DOI: 10.5						
DM, Obesity & HTN						
	М	F	M	F	М	F
DM (Pat) with Obesity &						
HTN						
Obesity (Pat)= 22	1	5	0	0	0	0
Obesity (Mat)= 9	1	1	0	0	0	0
Obesity (Pat & Mat)= 13	1	1	0	0	1	0
DM (Mat) with Obesity &						
HTN						
Obesity (Pat)= 8	1	1	0	1	0	0
Obesity (Mat)= 6	0	0	0	1	0	0
Obesity (Pat & Mat)= 11	3	1	0	0	1	1
DM (Pat & Mat) with						
Obesity & HTN						
Obesity (Pat)= 10	2	2	0	0	0	0
Obesity (Mat)= 9	1	1	0	0	0	2
Obesity (Pat & Mat)= 8	0	0	0	0	3	3
CAD Associated with	Pat relatives		Mat relatives		Pat & Mat relatives	
	М	F	М	F	М	F
DM, Obesity & HTN	2	1	0	0	0	0
DM & HTN	1	0	0	0	0	0
DM & Obesity	2	1	0	0	0	0
DM	0	0	0	ĩ	0	0
Dyslipidemia Associated with	Pat relatives		Mat relatives		Pat & Mat relatives	
_ , F	M	F	M F		M F	
DM, Obesity & HTN	0	0	0	2	0	0
DM, HTN & CAD	0	0	1	0	0	0
Obesity & HTN	0	0		0	0	0
	5	0	1	0	5	0

(**N.B.** Analysed data depicts number of students (male and female) having F/H/O. Pat = Paternal family & close relatives and Mat = Maternal family & close relatives)

Association of DM (Maternal) with obesity (Table - 2): 46 students had maternal DM. Obesity was present in paternal, and parental and maternal relatives.

Association of DM (both Paternal & Maternal) with obesity (Table -2): Students 60 [F=35 (14.64) & M=25 (13.74%)] had DM in both relatives and almost all were obese.

Association of DM, Obesity and HTN (Table – 2): Out of 85 Paternal DM families HTN was present in relatives of 11 (M=4 & F=7) students, especially in diabetic parental & maternal relatives with or without obesity. Prevalence was more in *parental* obese relatives i.e. parental (N=35) & maternal (N=22) respectively.

Association of CAD with DM, Obesity and HTN (Table – 2): 8 students had F/H/O CAD, all relatives were diabetic, and 6 obese and 4 hypertensive. All morbidities were reported in 1.

Association of Dyslipidemia with CAD, DM, Obesity and HTN (Table – 2): Dyslipidemia was present in maternal relatives of 4 students. Relatives of 2 female and 2 male students had multiple metabolic morbidities. Father of 3 students (F=2 & M=1) had premature death.

Anthropometry and Relevant parameters of Students:

(a) **Body Mass Index** (Table-3): Underweight 48 (11.4%) [M= 19 & F= 29], normal weight 182 (43.23%) [M= 72 & F= 110], and overweight students were 191 (45.37%) [M=91 & F=100].

Table-3 (BMI of Students Sex-wise)

BMI (Kg/m ²)	S	Total	
	Male	Female	
\leq 18.5 (under-weight)	19 (10.44%)	29 (12.13%)	48 (11.4%)
18.5 – 22.9 (Normal	72 (39.56%)	110 (46.03%)	182 (43.23%)
weight)			
23 – 24.9 (Over weight)	35 (19.23%)	38 (15.9%)	73 (17.34%)
25 – 29.9 (Pre obese)	39 (21.43%)	41 (17.15%)	80 (19%)
30 & above	17 (9.34%)	21 (8.79%)	38 (9.03%)

(b) Waist measurement (WC): Students (M=27 & F=20) declined waist/hip measurement. In 155 male: normal WC was present in 106 and \ge 90 cm in 49, and in 219 female: normal WC in 148 and \ge 80 cm in 71.

(c) Waist - Hip Ratio: WHR in 155 male students: normal (up to 0.90)

in 96 and ≥ 0.90 in 59, and 219 female: normal (up to 0.85) in 139 (58.16%) and ≥ 0.85 in 80.

(d) **Birth Weight**: 279 students (M= 132 & F= 147) were unaware of birth weight. Low birth weight (<2.5 Kg) students were 26 (M= 7 & F= 19), normal birth weight 107 (M=38 & F=69) and high birth weight (> 4 kg) 9 (M=5 & F=4).Presently, of the 9 high birth weight students, 3 (M=2 & F=1) are obese, 5 with normal weight (M=3 & F=2) and 1 low weight (F=1).

Five (71.43%) low birth weight *male* students had F/H/O DM (2 paternal, 1 maternal, and 2 parental & maternal) and obesity in 3 (1 maternal, and 2 paternal & maternal). Presently, 2 students are underweight, 2 normal and 1 overweight. Similarly, low birth weight 10 *female* students had F/H/O DM (5 paternal, 2 maternal, and 3 parental & maternal) and obesity (3 paternal, and 3 parental & maternal). Presently, 2 students are underweight, 5 normal and 3 overweight.

(d) **Disabilities**: *Hypertension*: Systolic HTN was detected in 31 (M=26 & F=5) and diastolic HTN in 32 (M=18 & F=14) student. *Diabetes mellitus*: One male student was T1DM. 3 females had Glucose Intolerance.

Other disabilities: PCOD in 4 students.

(e) **Physical activities**: This Institution is located in hill. Most of the students walk over 750 feet distance to and from college. Walking is main exercise by most of them. Some students use other mode of exercise. Mild exercise was performed by 388 students (M=161 & F=236), moderate exercise by 22 (M=12 & F=10), and severe exercise by 11 (M=9 & F=2).

(f) **Bone Mass density**: Students (M=20 & F=15) declined BMD measurement. Mean BMD in remaining students (M=162 & F=224) was -0.129 \pm 0.843 and 0.645 \pm 0.842 respectively. 336 (87.05%) students [M=143 (88.27%) & F=193 (86.16%)] had normal BMD, Osteopenia in 50 [M=19 (11.73%) & F= 31 (13.84%)] and Osteoperosis in none.

DISCUSSION:

Dynamics of DM epidemic are changing rapidly (4). Once a disease of affluent, now DM is increasingly reported in low socio-economical class, obese children and in "lean mass obese" people. India prosperity parallels with increase in sedentary lifestyle and altered food habits which are risk to metabolic diseases, especially the DM. China has surpassed India in prevalence of DM (5, 6) because of fast economy growth. Many risk factors are attributed to the development of DM: genetic propensity, childhood obesity, sedentary lifestyle, poor

INDIAN JOURNAL OF APPLIED RESEARCH 33

nutrition *in utero* and altered food habits. Now DM is diagnosed in young even with lower BMI (6,7) or with normal BMI "normal-weight metabolically obese". Indians develop DM faster with gain in weight than western counterparts. Children born to Indian women with gestational diabetes (GDM) are more prone to T2 DM.

Almost all metabolic diseases have genetic influence and transmitted with variable penetration and expression due to interplay of genetic and environmental factors. As polygenetic transmission and influence of environmental factors are variable, it is difficult to find the individual's risk of developing disease.

We accepted family history and important clinical & biochemical parameters tool to assign student to a low, medium, or high risk group, because students in each risk group share certain characteristics that correlate with probability of developing a disease.

DM has clustering in the families due to strong genetic components, 65 genetic loci has been deciphered (8 - 9). 45.37% students [M=69 & F= 122] had F/H/O DM [In other studies: 72.9% (Qatar), 53.9% (South India) & 70% (Pakistan)] (8,33-34)]. A child's risk of DM is about 50% when both parents are T2DM (8). Our, 60 (14.25%) students [M=25 & F=35] had F/H/O parental and maternal diabetics. Presently, these students are non-diabetic, we can assume that about 30 students (M 12 & F=17) may develop DM later on. Female with paternal or maternal diabetics are more prone to DM and this risk independent of a genetic score, but in men no such association exist (9). It means most of the 122 female students are at risk of developing DM at later age (10), but not all the male. Some studies found DM is substantially more related to F/H/O DM in mothers than in fathers (35 - 36), but in our study, students had predominant parental DM.

WC and WHR (indicators of central obesity) are associated with insulin resistance (IR) – a risk for DM. WC showed a stronger independent association with DM in all areas (11). 49 male and 71 female had increased WC, whereas increased WHR was present in 59 (38.06%) male and 80 female (36.53%) students. Since female are prone to DM development (8-9), increase WC & WHR further contribute to the development of IR / DM subsequently. Male students with abdominal adiposity are also vulnerable to "normal weight metabolic obese" related diseases.

48 under-weight students [M= 19 & F= 29] all were non diabetic, except one. Though, difficult to predict the future development of DM in them, yet we speculate by extrapolating results of our lean students (11.4%), with female predominance lean OPD population with 3.5% -10% T2DM (BMI $\leq 18.5 \text{ kg/m}^2$) (12-13). Major difference is that we have evaluated non-diabetic underweight students and above studies evaluated T2DM underweight patients. Secondly, our students are affluent, whereas above studies screened lean diabetics from general population irrespective of financial status. Many studies have also found the significant prevalence of underweight DM in poor socioeconomic class (12, 14). It is well documented that under birthweight children, exposed to plenty of food, have propensity to develop IR / DM. It is difficult to predict how many low weight students will develop DM.

Obese medical students were 9.03% [M=17 (9.34%) & F= 21 (8.79%)], our results differing from a study (conducted on affluent students) where incident of obesity was 3.4% (15). Pre-obese medical students were 19% [M=39 & F=41]. Overweight and Obesity are risk factors for DM – it is difficult to predict conversion of obese and pre-obese into IR, because of variation in gene penetration and phenotypic expression controlled by exogenous factors.

279 students were unaware of birthweight. In rest of the students low birth weight was in 26 and high birth weight 9. Low and high birthweights are known risks for T2DM (16-19). Some studies have demonstrated a 'U-shaped curve' relationship between them (18-19, 32), and other observed that low birth weight increased risk for T2DM and the high birthweight (20), and other found negative linear association (21). Exact mechanism unknown - how low birthweight increase T2DM risk. Various mechanisms are suggested: (i) compensatory adaptation to an adverse intrauterine environment, where development of important organs leads to IR and abnormal islet development (22), (ii) lack of important nutrients influencing fetal growth / metabolism (23-24), (iii) decrease oxidation ability of postprandial glucose and glycolysis in low birthweight (25-26), (iv) '*Fetal programming hypothesis*': lack of intrauterine nutrients causes a permanent metabolic shift towards IR to support brain glucose supply. After birth, the nutrient supply increases - resulting in obesity and IR (27-28), (v) 'Fetal Insulin Hypothesis': genetic variants decrease insulin secretion and cause low birthweight (29) and (vi) leptin administration to rats during late pregnancy and lactation makes offspring less susceptible to high-fat-diet-induced weight gain and IR (30). Therefore, low birthweight may be a clinical marker of poor intrauterine environment and a risk for T2DM. It is seen that mostly male are lean diabetics with history of childhood malnutrition, poor socioeconomic status and onset of early DM, and increased risk of, cardiovascular and non-cardiovascular mortality when compared to obese diabetic patients (31). Secondly, as most of underweight students were female, the trend may shift lean diabetes from male to female predominance. Thirdly, though our low birthweight students have accessibility to food, only 3 female were overweight, 7 (M=3 and F=4) remained underweight and rest normal weight. Could it be a leptin effect, which offers resistance to T2 DM development (30). A high birth weight increases the risk of T2DM in male, whereas obesity in both sexes (36). Contrary to observation that high weight baby is born to diabetic mother, most of high birth weight female students had parental DM.

In normal weight category M=67 (36.81%) & F= 104 (57.14%) perform mild exercises daily. Is it conceivable that these normal weight students, performing mild exertion, with lean body mass metabolically healthy? High mortality has been documented in certain normal weight DM patients as compare to their obese counterparts. They are "metabolically unhealthy normal-weight". This phenomenon is called the "obesity paradox" (31, 37). It is akin to sarcopenic obesity (high body fat, with reduced lean mass). Most of our students being sedentary may have reduced lean mass and cardiopulmonary fitness, which makes them prone for premature death and higher mortality (31, 37 - 38). In these so called normal weight obese students F/H/O metabolic diseases was widely prevalent. Interestingly, F/H/O DM and HTN were more prevalent in parental relatives, whereas obesity in maternal relatives. These "metabolically unhealthy normal-weight" students, particularly female, are prone to metabolic syndrome related complications as compare to obese counterpart, (39).

Other risk factors for DM in students were:

- HTN and prehypertension were mostly prevalent in male students. HTN and IR are intricately related. Thus hypertensive students are prone to IR.
- Glucose intolerance in 3 female and PCOD in 4 female students.

Recommended preventive measures:

(i) Dietary factors:

- Avoid canned and junk food, soft beverages, alcohol and refined carbohydrates.
- Daily fresh fruits and vegetables: for men: 2 cups of fruit and 2½ to 3 cups of vegetables and for women: 1½ to 2 cups of fruit and 2 to 2½ cups of vegetables.
- Daily intake of 30 g nuts (20 almonds / 15 cashew nuts).
- Add animal products for proteins. For vegetarians soya bean and lentils can compensate.
- (ii) Maintain body weight by healthy dietary habits, and appropriate exercises.
- (iii) Physical activities: 30 min daily moderate intensity exercise, at least 5 days a week, or walking for 10,000 steps / day with fast pace. Climbing gradients, gardening, dancing, jogging etc. are other alternatives.
- (iv) To avoid stress: Sound sleep for at least 6-8 hours, Yoga and meditation.

CONCLUSION:

Our participants lead sedentary lifestyle. Identifiable risk factors in these students, particularly in female, were DM, obesity, HTN, CAD and "normal-weight metabolically obese". Parental DM is important for high birthweight than maternal DM. Female students were mostly underweight. Future lean diabetes may tilt from 'male to female'. "Obesity paradox" exists in our study, this subgroup is equally vulnerable to metabolic diseases and called "metabolically unhealthy normal-weight".

Limitations of the study:

(i) family history and birthweight were only statement based. (ii) some biochemical parameters like lipid profile, glucose tolerance test and

34

INDIAN JOURNAL OF APPLIED RESEARCH

Pract 1999;44:49-58

leptin were not performed. (iii) visceral fat evaluation not done.

Acknowledgment:

Authors are thankful to all participants. We also acknowledge the participation of concerned department of Medical College in this study.

Conflict of Interest: Nothing to disclose.

Authors' contribution: study design, collection and collating relevant medical literature, preparation of manuscript and editing and final revision.

REFERENCES:

- Crozan Barba, Anura Kurpad, K Srinath Reddy, et al. Appropriate body-mass index for 1. Asian population and its implications for policy and intervention strategies (WHO expert consultation). The Lancet 2004; 363: 157-63.
- FrankB, Hu. Globalization of Diabetes. Diabetes Care 2011 Jun; 34(6):1249-1257. WHO Scientific Group on the Prevention and Management of Osteoporosis (2000: 3. Geneva, Switzerland) (2003). "Prevention and Management of Osteoporosis: report of
- WHO scientific group". Retrieved 2007-05-31. Chan JC, Malik V, Jia W, et al. Diabetes in Asia epidemiology, risk factors and 4. pathophysiology. JAMA 2009;301:2129-2140. Yang W, Lu J, Weng J, et al. China National Diabetes and Metabolic Disorders Study
- 5 Group: Prevalence of diabetes among men and women in China. N Eng J Med 2010; 361:1090-1101.
- 6. Ramachandran A. Mary S, Yamuna A, et al. High prevalence of diabetes and cardiovascular risk factors associated with urbanisation in India. Diabetes Care 2008;31:893-898
- 7 Yoon KH, Lee JH, Kim JW, et al. Epidemic obesity and type 2 diabetes in Asia. Lancet 2006;368:1681-1688.
- Abdulbari Bener, Mohammad T Yousafzai, Abdulla OAA Al-Hamaq, et al. Parental transmission of type 2 diabetes mellitus in a highly endogamous population. World J 8.
- Balkau, R. Roussel, S. Wagner, et al. Transmission of Type 2 diabetes to sons and daughters: the D.E.S.I.R. cohort. Diabetic Medicine 2017 (Research: Epidemiology); 9. Volume 34, Issue 11.
- Yang W, Lu J, Weng J, et al. Prevalence of diabetes among men and women in China. N 10 Engl J Med 2010 Mar 25;362(12):1090-101. Ramachandran A, Snehalatha C, Kapur A, et al. Diabetes Epidemiology Study Group in
- 11. Ramachandran A, Shenalatha C, Kapur A, et al. Diabetes Epidemiology study Group in India (DESI): High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. Diabetologia 2001; 44:1094–1101.
 Mohan V, Vijayaprabha R, Rema M, et al.. Clinical profile of lean NIDDM in South India. Diabetes Res Clin Pract 1997;38:101–108.
 S Krishnamoorthy, J Gridhar Muthu, S R Ramakrishna, et al. Clinical and Biochemical Diabetes and State and St
- 12
- 13. Profile of Lean Body Weight Type 2 Diabetics, Normal Weight and Obese Diabetes. Journal of Evolution of Medical and Dental Sciences 2015; Vol 4(71):12397-12413.
- Sidharth Das. Low Bodyweight Type 2 Diabetes Mellitus. Journal of Nutritional & Environmental Medicine 1999; Vo19(3):229-239. 14
- T Aggarwal, RC Bhatia, D Singh, et al. Prevalence of Obesity and Overweight in 15 Affluent Adolescents from Ludhiana, Punjab (Short Communication). J Indian Paediatrics 2008;45:500-502.
- Thomas Harder, Elke Rodekamp, Karen Schellong, et al. Weight and Subsequent Risk 16 of Type 2 Diabetes: A Meta-Analysis. American Journal of Epidemiology 2007; Vol 165(8): 849-857.
- Barker DJ, Hales CN, Fall CH, et al. Type 2 (non-insulin-dependent) diabetes mellitus, 17. hypertension and hyperlipidaemia (syndrome X): relation to reduced fetal growth. Diabetologia 1993; Vol. 36: 62-67.
- McCance DR, Pettitt DJ, Hanson RL, et al. Birth weight and non-insulin dependent 18 diabetes: thrifty genotype, thrifty phenotype, or surviving small baby genotype? BMJ 1994: Vol. 308:942-945
- Wei JN, Sung FC, Li CY, et al. Low birth weight and high birth weight-infants are both 19 at an increased risk to have type 2 diabetes among schoolchildren in Taiwan. Diabetes Care 2003; Vol.26: 343-348.
- Donghua Mi, Hongjuan Fang, Yaqun Zhao, et al. Birth weight and type 2 diabetes: A meta-analysis. Exp Ther Med 2017; Vol14(6):5313-5320. 20
- Lithell HO, McKeigue PM, Berglund L, et al. Relation of size at birth to non-insulin 21 dependent diabetes and insulin concentrations in men aged 50-60 years. BMJ (Clinical research ed) 1996;312:406-410.
- Phillips DI, Hirst S, Clark PM, et al. Fetal growth and insulin secretion in adult life. 22 Diabetologia 1994;37:592-596. Mooorthi MMS, Nadesan B, Ramalingam E, et al. A study of maternal factors
- 23
- Information Weyl States and B. Kantanigan, L. et al. Study of matchina factors influencing very low birth weight babies. Int J Contemp Pediatr 2017;4:1173–1178. Mortensen B, Hingst JR, Frederiksen N, et al. Effect of birth weight and 12 weeks of exercise training on exercise-induced AMPK signaling in human skeletal muscle. Am J Physiol Endocrinol Metab 2013;304:E1379–E1390. 24
- von Bonsdorff MB, Muller M, Aspelund T, et al. Persistence of the effect of birth size on 25 dysglycaemia and type 2 diabetes in old age: AGES-Reykjavik study. Age (Dordr) 2013;35:1401-1409.
- 2015;35:1401–1409. Taylor DJ, Thompson CH, Kemp GJ, et al. Arelationship between impaired fetal growth and reduced muscle glycolysis revealed by 31P magnetic resonance spectroscopy. Diabetologia 1995;38:1205–1212. Vejirazkova D, Lukasova P, Vankova M, et al. Gestational diabetes-metabolic risks of adult women with respect to birth weight. Physical Res 2015;64(Supp12):5135–5145. 26
- 27
- and obesity in childhood: Prospective cohort study. BMJ (Clinical research ed) 2000;320:967–971. 28
- Fowden AL, Forhead AJ. Endocrine interactions in the control of fetal growth. Nestle Nutr Inst Workshop Ser 2013;74:91–102. 29
- 30 Stocker C, O'Dowd J, Morton NM, et al. Modulation of susceptibility to weight gain and insulin resistance in low birth weight rats by treatment of their mothers with leptin during pregnancy and lactation. Int J Obes Relat Metab Disord 2004;28:129-136.
- Amrutha Mary, George Amith, George Jacob, et al. Lean diabetes mellitus: An emerging entity in the era of obesity. World J Diabetes 2015 May 15; 6(4): 613–620. 31.
- Jung-Nan Wei, Fung-Chang Sung, Chung-Yi Li, et al. Low Birth Weight and High Birth Weight Infants Are Both at an Increases Risk to Have Type 2 Diabetes Among School children in Taiwan. Diabetes Care 2003; 26(2):343-348. 32
- Viswanathan M, McCarthy MI, Snehalatha C, et al. Familial aggregation of type 2 (non-insulin-dependent) diabetes mellitus in South India; absence of excess maternal 33. transmission. Diabet Med 1996;13:232–237. Shera AS, Rafique G, Khawaja IA, et al. Pakistan National Diabetes Survey: prevalence
- 34

Karter AJ, Rowell SE, Ackerson LM, et al. Excess maternal transmission of type 2 diabetes: the Northern California Kaiser Permanente Diabetes Registry. Diabetes Care 35 1999: 22:938-943.

of glucose intolerance and associated factors in Baluchistan province. Diabetes Res Clin

- Johnsson IW, Haglund B, Ahlsson F, et al. A high birth weight is associated with 36.
- increased risk of type 2 diabetes and obesity. Pediatr (Obes 2015;10(2);77-83. Carnethon MR, De Chavez PJ, Biggs ML, Lewis CE, Pankow JS, Bertoni AG, et al. Association of weight status with mortality in adults with incident diabetes. JAMA 37 2012-308-581_590
- NathalieEckel, KristinMühlenbruch, KarinaMeidtner, et al. Characterization of 38. metabolically unhealthy normal-weight individuals: Risk factors and their associations with type 2 diabetes. Metabolism 2015;64(8):862-871.
- Hittight Datactions Metaodism 2017;01(0):02-01. Patrick T. Bradshaw, Keri L. Monda, and June Stevens. METABOLIC SYNDROME IN HEALTHY OBESE, OVERWEIGHT AND NORMAL WEIGHT INDIVIDUALS: THE ATHEROSCLEROSIS RISK IN COMMUNITIES STUDY. Obesity (Silver 39 Spring) 2013; 21(1): 203-209.