CODI
CONTROVERSIES IN OBESITY & DIABETES

OBESITY AND DIABETES MELLITUS: NATURE OR NURTURE

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INTRODUCTION

Neighborhood Socio-Economic Status (nSES), is reported as the cause of obesity and diabetes in developed and developing countries. Due to the large, varied, and often conflicting data reported on Overweight, Obese, and Diabetes, these polymorphisms have been weakly associated with NALFD-HBV-Liver Ca, but NAFLD[14] cause by nSES has been reported this last decade.[4] Lp(a)-microalbuminuria[23] camouflage dyslipidemia and Subscapular Skinfold Thickness (SsST) as early obesity and diabetes mellitus detection.[21,22]

METHOD

Review articles with my library, and academic search engine, with keywords: UCP1, UCP3, UCP2-Obesity and Diabetes

RESULT

Table 1. The differences of Healthy Normal F1 Non-Family History Syndrome-X vs. FHSX in UCP1 (Subscapular Skinfold Thickness/SsST) and Lp(a) on the Insulin Plasma level.

Variable	NFHSX (n= 34)	FHSX (n=44)	p
Age (y)	23.53±5.7	24.25±5.9	>0.05
BMI	20.70±2.7	22.65±5.2	=0.000
WHR (%)	0.79±0.05	0.79±0.08	>0.05
SsST (mm)	17.75±8.7	24.51±13.10	=0.000
Insulin plasma µU/mL)	5.67±2.59	10.62±9.01	=0.000
Lp(a) mg/dL	15.24±29.49	24.95±29.80	=0.028

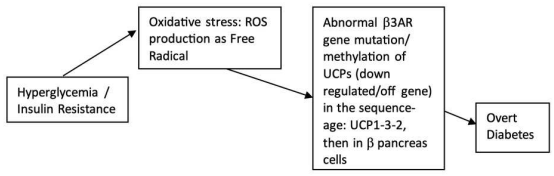

Figure 1. Course of Insulin Resistance -methylation of UCPs 132 to Overt Diabetes

1. Genetic (nature)[12,13,18]
2. Epigenetic – RNAi (nurture)[2,4,5,6,9,10, 14,15,16,17]
3. nSES incl. Pima Indian and geography wet and warm Brazil, Bangladesh, other wet and warm countries climate area (AFB1 exposure environment)[4]
4. Lifestyle sugar, salt, SAFA, red-meat poultry, and AFB1 exposure[4] 5. Mesomorph and Endomorph somatotype with broadening width chest.[8,11]

DISCUSSION

Genetic Polymorphism in glucose metabolism and insulin secretion, epigenetic changes in diabetes (DNA methylation analysis), p53 suppressor gene mutation and fat cell proliferation,[19,20] food in Industry 4.0 without Society 5.0, Uncoupling protein UCP1,3,2 monitoring.[6,7,18]

CONCLUSION

Nurture outcome needs lifestyle and avoid AFB1 exposure from overeating red meats. Change to tempeh and tofu eating habits, no sugar, no salt. Early detection SsST: Normal 18-22 mm, for CKDs and Metabolic Syndrome-Overweight-Obesity, then overt DM.

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CONTROVERSIES IN OBESITY & DIABETES

FOUR CHOLESTEROL PROFILES IN DYSLIPIDEMIA PATIENTS: GEOGRAPHICAL OR HABITUAL VETERINARIAN PROTEIN OVER EATING

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INTRODUCTION

Stroke and atherosclerosis prevalence have been increasing since the pandemic of COVID-19 era (inflammation reaction). The prevalence of hypercholesterolemia, hypertriglyceridemia, high LDL, and low HDL plasma levels are also in high prevalence in the population.[1,2,3]

METHOD

Correlation and differentiated test by Pearson and Spearman of 4 lipid profiles in 30 dyslipidemia patients, measured by Advia 1800 Chemistry System.

RESULT

Each 3 lipid profiles has no correlation with hypercholesterolemia in 30 dyslipidemia patients (p>0,005).

DISCUSSION

1. **Oval fat bodies, microalbuminuria-high Lp(a): no high cholesterol total due to the glomerular albumin/ fat excretion;**
2. **Correlation of each lipid profile with total cholesterol**
3. **Each cholesterol profile Regression with lifestyle in food eating and physical activity.**

Table 1. Significant correlation between Oval Fat Bodies (OFB)-each 3 lipid profiles

	Variable	X± SD (n= 66 DL)	Min	Max	rOFB-x1,2,3	p
1	Hyperlipidemia: OFB n=15 (75%)				0.82	0.01
2	Hypercholesterolemia: OFB n=26 (72.2%)	230.6±36.43mg/dL	150	323	0.84	0.05
3	Hypertriglyceridemia: OFB n= 7 (70%)	210.85±131.74 mg/dL	76.5	1025	0.76	0.05

CONCLUSION

There is no correlation between the 4-item lipid profile due to glomerular leak of fat in CKD, which is neglected, has not become aware, although a higher prevalence in the population, which is with inflammation reaction induced atherosclerosis, CVD, and stroke.

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Keywords: Triglyceride, LDL-cholesterol, HDL-cholesterol, dyslipidemia, microalbuminuria, CKDs

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