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New Biotechnological Opportunities to Assess the Influence of Lifestyle Factors in Obesity

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***Abstract*— Background. Obesity results from a malfunction of the body's weight-control mechanisms, which may be influenced by environmental changes. Essentially, the obesity risk relies on two significant interdependent factors: genetic variations (single-nucleotide polymorphisms, haplotypes) and environmental risk exposure. Due to new biotechnologies over 127 potential genes for obesity have been identified, and evidence supports the function of 22 genes in at least five distinct groups. Gene and environment interactions mean that the synergy between genotype and environment is neither additive or multiplicative. The application of innovative methods for both genotype and lifestyle variables should be emphasized.**

Aim of study: Investigate variable data of lifestyle factors in obese people with genetic predisposition and without in order to figure out the trigger risks which transform the predisposition into obesity.

Material and methods: This is a descriptive study. A questionnaire was elaborated. It was developed based on the data of new biotechnological analysis of metabolic changes in obese humans. 142 individuals were included. 82 obese individuals, 42 with genetic predisposition and 40 without, and 60 healthy probands were interviewed. Further followed a comparative statistical analysis. Results: Obese probands were found with higher levels of disability compare those without, cardiovascular events higher compared with healthy probnads, disability level and smoking habits had significantly correlation in obese with genetic predisposition. On the other hand, health probands were found in higher level of anxiety compared obese people with genetic predisposition. Conclusions: All the lifestyle aspects which lead to an increased central nervous overactivity disturb significantly the metabolism and are critical risk factors for people with genetic predisposition relate to the pathogenesis of obesity. that might lead to high disability level associated comorbid states and high risk of cardiovascular events.

Keywords*— *obesity, genetic predispositions, genetic variants, lifestyle, metabolism, risk factors, biotechnology.

I. INTRODUCTION

Obesity results from a combination of environmental and genetic factors. Twin and adoption studies provide the most compelling evidence of a genetic component to obesity [1]. In studies that measured body fat content (either as body mass index [BMI] or skinfold thickness), the comparison of obesity in monozygotic twins with obesity in dizygotic twins revealed heritability quotients ranging from 0.40 to 0.98 (where 0 = no inheritance and 1.0 = complete inheritance of the trait). Although the environments shared by monozygotic twins are more comparable than those shared by dizygotic twins, there is no difference in the heritability of BMI between identical twins raised together and those raised apart. Adoption studies have also shown that obesity has a hereditary component. These findings imply that the genetic transmission of obesity is about equivalent to the nongenetic transmission [2]. Genetic segregation investigations in extended families indicate that 30% to 50% of the obesity phenotype is inherited, and there is evidence for a significant recessive gene or genes with an allele frequency of 0.30. A number of candidate genes for obesity have been found, and the significance of some of these genes has been verified in mice created with human DNA[3]. In the last 20 years, several methodologies have been used to identify genetic determinants of obesity, including severe obesity studies, genome-wide linkage studies, candidate gene analysis, and genome-wide association studies (GWAS). Since 2005, GWAS has helped us understand the genetic causes of obesity. The National Human Genome Research Institute GWAS collection lists 50 locus. The FTO gene has the highest influence on obesity risk to date; each extra risk allele in FTO is linked with a 1- to 1.5-kg rise in body weight and a 20% to 30% increase in obesity risk [4]. The hereditary causes of obesity may be categorized into three categories. First, genes encoding proteins that regulate food intake at

the level of the hypothalamus, such as centrally produced molecules, such as proopiomelanocortin [POMC]-derived alpha-melanocyte-stimulating hormone, which signals via the melanocortin-4 receptor [MC4R], or peripheral molecules, such as leptin, ghrelin and peptide YY [PYY]3–36, appear to influence obesity [5]. In obesity phenotypes linked with relative hyperphagia, defects at this level are likely to prevail. These people may have rapid weight loss in response to calorie restriction and may benefit most from appetite-suppressing drugs. Genetic variation in several genes that regulate preadipocyte differentiation (e.g., peroxisome proliferator-activated receptor-gamma [PPAR]), triglyceride synthesis (e.g., diacylglycerol acyltransferase [DGAT]-1) and lipolytic potential (e.g., beta-adrenergic receptors and perilipin) have been associated with a predisposition to obesity [6]. genes that affect mitochondrial biogenesis and/or adaptive thermogenesis may influence the tendency to acquire or lose weight and may serve as therapeutic targets in obese individuals who are resistant to weight reduction [7].

II. MATERIAL AND METHODS

In order to collect data regarding the studied topics, questioners were collected from subjects by me and my colleagues in a convenient form of sampling. The questioners contained 25 items. Item number one asked about age. Number two asked about gender. Number three asked about smoking habits in terms of pack years. Item number 4 asked about height and weight from which BMI score calculation could be obtained. Item number 5 asked about level of disability from a scale of 1 to 10 in which 1 stands for no disability and 10 for severe disability. Item number 6 asked weather suffering from diagnosed chronic illness. Item 7 asked whether there is a medication which is prescribed under regular base. Item number 8 asked about balanced diet or weather some of the main group such as proteins, carbohydrates, lipids or vitamins are missing. Item number 9 asked about the possibility to eat a decent warm meal during work or not. Item number 10 asked about hours of physical exercise each week. Item number 11 asked about two or more relative who is suffering from the condition, when 2 or more relative suffering from the condition, were assumed to be genetically predisposed. Item 12 asked about level of satisfaction from health from a scale of 1 to 5 where 1 stand for low level satisfaction, and 5 for high level of satisfaction. Item number 13 asked whether a subject is going for a walk after dinner. Item 14 asked whether subject is watching tv after dinner or not. Item 15 asked whether there is a stressful event that led to weight gain. Item number 16 asked about number of cardiovascular events that subjects has been through. Item number 17 asked whether your work is causing you stress or not. Item number 18 was an open question and asked about ways and practices taken by the subject in order to relief stress. Item number 19 asked about alcohol consumption in doses per week. Item number 20 asked weather there is an endocranial disorder, and if so, which hormone does it involve. Item number 21 asked about number of meals eaten each day. Item number 22 asked about quantity of water per glass that the subject drink every day. Item number 23 asked about number of sweet drinks that subject drink each day. Item 24 asked for number or glasses of coffee and with or without sugar. Item number 25 asked about number of steps that the subject is walking each day.

Group Statistics

	obesity	N	Mean	Std. Deviation	Std. Error Mean
number_of_cardiovascular_events	healthy_probands	60	.3833	1.12131	.14476
	obese	82	1.9756	2.90158	.32043

III. RESULTS AND DISCUSSION

On independed t test, obese people were found with a higher number of cardiovascular events than healthy probands ($p<0.01$). the mean of cardiovascular events in healthy probands was 0.3 and was 1.9 on obese subjects (Fig.1)

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
						Significance			Std. Error Difference	95% Confidence Interval of the Difference
		F	Sig.	t	df	One-Sided p	Two-Sided p	Mean Difference		Lower	Upper
number_of_cardiovascular_events	Equal variances assumed	12.948	<.001	-4.033	140	<.001	<.001	-1.59128	.39461	-2.37285	-.80970
	Equal variances not assumed			-4.539	131.806	<.001	<.001	-1.59128	.35063	-2.28491	-.89765

Figure 1. Obese people compared to healthy probands in cardiovascular events count.

Obese individual was also found with higher level of disability ($p<0.01$) than healthy probands (Fig.2).

Group Statistics

	obesity	N	Mean	Std. Deviation	Std. Error Mean
Disability_Level	healthy_probands	60	2.0500	1.58836	.20506
	obese	82	8.0000	2.84583	.31427

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
						Significance			Std. Error Difference	95% Confidence Interval of the Difference
		F	Sig.	t	df	One-Sided p	Two-Sided p	Mean Difference		Lower	Upper
Disability_Level	Equal variances assumed	13.982	<.001	-14.667	140	<.001	<.001	-5.95000	.40574	-6.75333	-5.14667
	Equal variances not assumed			-15.856	131.843	<.001	<.001	-5.95000	.37525	-6.69239	-5.20761

Figure 2. Obese individuals compared to healthy probands in level of disability

Among obese individials that were with genetic predespostion a negative correlation($r=-0.3$) was found between level of physical activity and level of disablilty. That corrolation was not found in obese subjects without genetic predisposition (Fig.3)

Correlations

genetic_predesposition			Disability_Level	PHYSICAL_ACTIVITY
without	Disability_Level	Pearson Correlation	1	-.152
		Sig. (2-tailed)		.574
		N	16	16
	PHYSICAL_ACTIVITY	Pearson Correlation	-.152	1
		Sig. (2-tailed)	.574	
		N	16	16
with	Disability_Level	Pearson Correlation	1	-.357**
		Sig. (2-tailed)		.003
		N	66	66
	PHYSICAL_ACTIVITY	Pearson Correlation	-.357**	1
		Sig. (2-tailed)	.003	
		N	66	66

**. Correlation is significant at the 0.01 level (2-tailed).

Fig.3 correlation between physical activity and level of disability in obese subjects with genetic predisposition and without.

In obese subjects with genetic predisposition a significance correlation ($p<0.01$) with a positive coefficient($r=0.4$). this correlation was not found among obese subjects without genetic predisposition (Fig.4)

Correlations

genetic_predesposition			number_of_cardiovascular_events	ALCOHOL_CONSUMPTION
without	number_of_cardiovascular_events	Pearson Correlation	1	-.473
		Sig. (2-tailed)		.064
		N	16	16
	ALCOHOL_CONSUMPTION	Pearson Correlation	-.473	1
		Sig. (2-tailed)	.064	
		N	16	16
with	number_of_cardiovascular_events	Pearson Correlation	1	.400**
		Sig. (2-tailed)		<.001
		N	66	66
	ALCOHOL_CONSUMPTION	Pearson Correlation	.400**	1
		Sig. (2-tailed)	<.001	
		N	66	66

**. Correlation is significant at the 0.01 level (2-tailed).

Fig.4 The correlation between alcohol consumption and number of cardiovascular events. In obese subjects without genetic predisposition, and individual with genetic predisposition.

Pack year number was also found significantly($p<0.01$) correlated with Disability level among obese subject with genetic predisposition($r=0.4$). this correlation was not found in obese subjects without genetic predisposition (Fig.5)

Correlations

genetic_predesposition			Pack_years	Disability_level
without	Pack_years	Pearson Correlation	1	-.271
		Sig. (2-tailed)		.311
		N	16	16
	Disability_level	Pearson Correlation	-.271	1
		Sig. (2-tailed)	.311	
		N	16	16
with	Pack_years	Pearson Correlation	1	.316**
		Sig. (2-tailed)		.010
		N	66	66
	Disability_level	Pearson Correlation	.316**	1
		Sig. (2-tailed)	.010	
		N	66	66

**. Correlation is significant at the 0.01 level (2-tailed).

Fig.5 the correlation between pack year number and level of disability among obese individual with genetic predisposition and those without.

The results consistent with the based knowledge, and shows that obese individuals are found with higher level of disability and higher number of cardiovascular events. In obese people with genetic predisposition, a correlation was found between smoking habits and level of disability, suggesting these factors have even worse outcomes on this group. Physical activity level however, was found correlated with level of disability with a negative coefficient of correlation, which indicates that physical activity is even more beneficial in this group. It is important to mention that this is a correlation study and according to which we cannot conclude causality. In order to study the causality of the observed variable, a cohort study can be made in order to study the nature of the risk factors and beneficial factors in the studied population.

IV. CONCLUSIONS

1. All lifestyle aspects which had central nervous overactivity as pathophysiological background are triggers of realizing genetic predisposition into obesity.
2. New biotechnologies make possible a better understanding of genetic causality of metabolic changes.
3. Hypodynamia is a critical trigger in obesity with genetical predisposition, comparing with non-genetic obesity.
4. Physical disability and cardiovascular events were consequences met more often in those obese with genetic predisposition.

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