DOCUMENT OF COMPLEMENTARY DATA

Nutrigenetics

NUTRIGENETIC MOLECULAR STUDY

PATIENT DATA

LAST NAME _____

FIRST NAME _____ ID N° _____

CLINICAL DATA

HEIGHT: _____ CURRENT WEIGHT: _____

IDENTIFICATION LABEL
(by GENYCA)

BMI (Body Mass Index): _____

BEGINNING OF OVERWEIGHT:

☐ CHILDHOOD ☐ ADOLESCENCE ☐ ADULTHOOD ☐ PREGNANCY/BREASTFEEDING ☐ MENOPAUSE

MAXIMUM WEIGHT REACHED: _____ AGE AT MAXIMUM WEIGHT: _____

PREVIOUS DIETS:

Nº DIETS LONGER THAN 3 MONTHS: _____ MAXIMUM WEIGHT LOST: _____

KINDS OF PREVIOUS DIETS: _____

PHARMACOLOGICAL SUPPORT TREATMENT: _____

CURRENT DIET:_____

KIND OF DIET: _____

PHARMACOLOGICAL SUPPORT TREATMENT: _____

FAMILY MEDICAL HISTORY:

OVERWEIGHT: _____ EATING DISORDERS: _____

OTHER CLINICAL DATA OF INTEREST (specify):

_____

_____

MOLECULAR GENETIC ANALYSIS REQUESTED

☐ NUTRIGENETICS INITIAL STUDY

☐ NUTRIGENETICS COMPLETE STUDY

☐ DIABESITY STUDY

INFORMATION ABOUT THE TEST

Civilization, technological developments and sedentary lifestyles have changed our daily energy requirements, and the easy access to high-calorie food has made it easier to gain fat. Because of these environmental changes, the genotype responsible for energy-saving tendencies through the more efficient maintenance of adipose tissue, which was once considered a beneficial survival adaptation, has now become a disadvantageous marker of disease. People within the scientific community have proposed a thrifty gene hypothesis, which speculates that differential resistance to insulin is a result from the modification and mutation of several “thrifty genes” during the natural selection process. Mutations in other genes, particularly in those that control eating behavior, basal metabolic rate, lipid metabolism, stress response, chronic inflammation, etc., have also occurred in order to ensure adequate nutritional intake, the proper use of these nutrients, and reduction of energy expenditure, among other functions. This has led to the prevalence of obesity and Type 2 diabetes.

Interindividual genetic variability is a critical determinant of each person's unique nutritional requirements and unique response to various associated environmental factors. These genetic factors do not only cause extreme obesity, but also affect the whole spectrum

GENYCA INNOVA Análisis y Diagnóstico Genético S.L. / Dom. Soc. Alegría 18, 28220- Madrid /
Ins. Reg. Mer. de Madrid, Tomo 19.199, Sección 8,Hoja M-336110, Folio 210 / C.I.F.B-83751933
FPG-70 – Ed0 – 20/11/2013

C/ Alegría 18, Majadahonda, Madrid 28220 SPAIN
www.genyca.es T: +34 91 634 61 06

of body statistics. Percentage body fat, weight, height, BMI and all the anthropometric variants have a normal (Gaussian) distribution in the general population because of a multifactorial inheritance in which there is a predisposing genotype that interacts with environmental factors. This is true of both common obesity and multifactorial obesity, in which the many involved genes all have a moderate effect.

There are currently about 300 human genes described that are subjects of nutrigenetic studies. Many of these are unproven candidate genes whose mechanism of action is unknown. GENYCA evaluates the genetic susceptibility to obesity in the nutrigenetic study Nutri-G by analyzing the polymorphisms of 18 genes known as "thrifty genes," which save energy in the form of fat. These 18 genes have been validated by the scientific community and supported by verified references. The genes studied are split into five groups according to their primary function, which allows for targeted therapies based on the underlying pathophysiologic mechanisms:

- Group I: thrifty genes involved in the central regulation of energy balance and food intake
- Group II: thrifty genes involved in lipid metabolism and thermogenic regulation
- Group III: genes related to the inflammatory process in adipose tissue in obesity
- Group IV: genes causing insulin resistance and promoting Type 2 diabetes
- Group V: genes that contribute to cardiovascular risk in obesity

It is important to determine an individual's genetic makeup (via a complete study of all the genes outlined above) in order to examine how the interaction between genetic and environmental factors can cause obesity and in order to establish a more accurate prognosis with regards to established therapeautic measures such as diet and exercise. The molecular study not only assists in the etiopathogenic diagnosis of obesity and allows for personalized therapeutic treatments, but also is predictive of not-yet-present metabolic and cardiovascular complications. This makes it possible to detect at-risk people and take preemptive measures to reduce the severity or prevent the onset of these diseases. Note that this study is not a diagnostic test, but a risk estimation analysis.

By signing below, I confirm that I have read and understood the material presented about the nutrigenetics study and verify that all information provided in this document is valid.

PRINTED NAME: _____

If the patient is a minor or requests legal representation for any other reason, detail the RELATIONSHIP TO THE PATIENT (official documentation verifying the signer's personal identity and relationship to the patient required): _____

ID No. _____ DATE _____

SIGNATURE:

REFERENCES

1. Lo et al., 1998. Quantitative analysis of fetal DNA in maternal plasma and serum: implications for noninvasive prenatal diagnosis. Am J Hum Genet 62:768-775
2. Fan et al., 2008. Noninvasive diagnosis of fetal aneuploidy by shotgun sequencing DNA from maternal blood. PNAS 105(42):16266-71
3. ISPD (International Society for Prenatal Diagnosis), Rapid Response Statement 24 October 2011
4. Chiu RW et al., 2011. Non-invasive prenatal assessment of trisomy 21 by multiplexed maternal plasma DNA sequencing: large scale validity study. BMJ 342:c7401
5. Chen EC et al., 2012. Noninvasive prenatal diagnosis of fetal trisomy 18 and trisomy 13 by maternal plasma DNA sequencing. PLoS ONE e21791
6. Jiang et al., 2012. Noninvasive Fetal Trisomy test: an advanced noninvasive prenatal diagnosis methodology for fetal autosomal and sex chromosomal aneuploidies. BCM Medical Genomics 5:57.
7. Dan et al., 2012. Clinical application of massively parallel sequencing-based prenatal noninvasive fetal trisomy test for trisomies 21 and 18 in 11105 pregnancies with mixed risk factors. Prenat Diag 32:1-8
8. Mennuti et al., 2013. Is it time to sound an alarm about false-positive cell-free DNA testing for fetal aneuploidy?. Am J Obstet Gynecol 209(5):415-419

GENYCA INNOVA Análisis y Diagnóstico Genético S.L. / Dom. Soc. Alegría 18, 28220- Madrid / Ins. Reg. Mer. de Madrid, Tomo 19.199, Sección 8,Hoja M-336110, Folio 210 / C.I.F.B-83751933
FPG-70 – Ed0 – 20/11/2013

C/ Alegría 18, Majadahonda, Madrid 28220 SPAIN
www.genyca.es T: +34 91 634 61 06