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# Genetics, Epigenetics and their relationship with eating disorders

**Gina Llado**[1], **Maria Jose Blanco**[2], and **Mireia Munoz**[3]
[1]Universidad Isabel I, Spain
[2]Universidad Europea de Madrid, Spain
[3]Universitat de Barcelona, Spain

**Introduction:** The biological predisposing factors for the appearance of Eating Disorders (ED) are exposure to hormones during pregnancy, genetic factors, and epigenetic factors. Currently, there is a high interest in the action of genetics and epigenetics and their relationship with ED.

**Objective:** To determine, reviewing studies conducted by other authors, the genetic influence of Anorexia Nervosa (AN) and Bulimia Nervosa (BN).

**Methods:** A review in PubMed was performed with the terms "anorexia nervosa" and "bulimia nervosa", "eating disorders", "genetics", "epigenetics", "family heritage". Articles published in the last 11 years were used.

**Results and Discussion:** (1) Family heritability: Interactions between genes and the environment, modify genes expression and the development of ED. Gene expression can be modified in the uterus of the mother (due to maternal stress or poor nutrition). The nutritional status of the mother provokes epigenetic changes and systematic changes in human metastable epialleles. These are related to the development of ED. If these changes are fixed and transmitted to descendants, it affects heritability. In family studies (with "cases" and "controls" patients), the genetic influence is clear, especially in first-degree relatives. Family "cases" of patients with AN are 11.3 times more likely to suffer from AN than in relatives of "controls". An increased incidence of BN has also been found in relatives of patients with BN. (2) Gender: Procopio and Marriot (2007) indicate the existence of a substance produced by female fetuses that increases the risk of suffering from AN in women. Males are less likely to suffer from the disease but if they coexist in the uterus with female fetuses, their risk of AN increases. In AN, there is a 70% concordance rate in identical twins. It is known that AN is 10 times more frequent in women than in men. These statistics are related to intrauterine hormonal exposure. (3) Twin studies: In monozygotic twins, genetics explains 28% to 88% of predisposition to AN. In the BN, heritability is estimated between 58% and 83%. (4) Adolescence: This heritability is associated with the sexual development that occurs at puberty. The activation of ovarian hormones increases the risk of ED through the differential activation of the transcription of risk genes. As indicated by Culbert et al. (2008), the hormonal changes that occur during puberty influence the development of ED. Genes, in adolescence, are responsible for up to 85% of the risk factors for ED (mainly the concern about weight and body shape). The influence of environmental factors would only be 15%.

## Biography

Gina Lladó Jordan has completed his PhD at the age of 27 years in Universidad Europea de Madrid, Spain. She works as an investigator and teacher in Universidad Isabel I, Burgos, Spain. She has completed 5 masters related to various aspects of health sciences. She has directed 20 Final Degree Projects and participated in multiple studies and research groups. Her main line of research is Eating Disorders and Nutrition.

ginalladojordan@gmail.com

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