THE MONOGAMY GENE COULD ALSO ACT IN HUMANS

AVPR1A COULD BE PROMOTING PAIR-BONDING IN HUMANS SLA

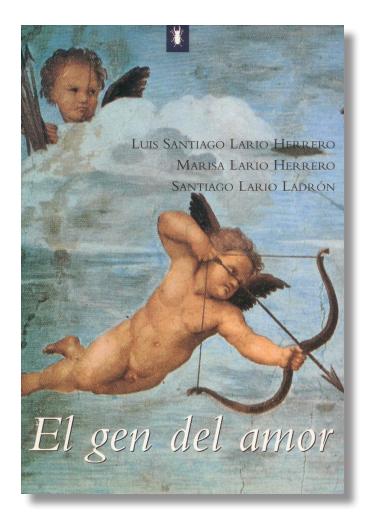
Research has revealed that genetic variations in the human gene AVPR1A affect the disposition and aptitude of individuals to live in a relationship. Thus the activity of this gene could influence the quality of marital relationships and very likely our emotional inclinations.

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INTRODUCTION

Given the crucial role that the gene AVPR1A seems to play in the disposition towards monogamy in species such at the prairie vole (Microtus ochrogaster), a group of Swedish researchers at the Karolinska Institute decided to study its activity in humans. This gene codes for some of the AVP (arginine-vasopressin neuropeptide) receptors.

These researchers studied the quality of marital relationships in 552 pairs of twins who had been living with their partners for over five years and the possible connections between the quality of their relationships and the variations found in this gene.

The results of this study are a modest but obvious correlation between some genetic variants found in the 5' region of this gene and certain differences in male aptitude towards marital life (this correlation has not been confirmed in women).

Carriers of allele 334, which is present in 40% of the study subjects, were more hesitant to marry, more likely to break off their marriage, more prone to infidelity and their relationships tended to be less satisfactory for their partners.

The effects were even more pronounced in the 3.45% of the study population with two copies of this allele. This group had twice the percentage of unmarried couples cohabiting or couples experiencing marital problems during the past year.

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THE INFIDELITY GENE...?

Some media have become fixated on the behaviors caused by allele 334 to the point of baptizing it as "the infidelity gene."

However, we can't forget that throughout the animal kingdom sexuality has a universal inclination towards promiscuity. This is so to such an extent that monogamous species have most assuredly gained some gene (or group of genes) in their genetic inheritance that constrains their sexual behavior.

Therefore we cannot interpret the results of this work as demonstrating the existence of an infidelity gene. An infidelity gene is not necessary as sexuality without the influence of this gene is by nature promiscuous. On the contrary this study suggests the existence of a gene that encourages pair-bonding, but is less active when present in the 334 allele form.

In the publication of their article the authors acknowledge: "These results suggest an association between a single gene and pair-bonding behavior in humans, and indicate that the well characterized influence of AVP on pair-bonding in voles may be of relevance also for humans."

A second result of their study is that variations in the DNA sequence of this gene reduce (or perhaps completely eliminate) its normal functionality resulting in the carrier failing to express an impulse to pair-bond or, at the very least, expressing this impulse to a lesser degree.

According to their research, the effects exerted by these genetic variations, especially when they affect only one copy of the gene, are very modest and do not lend themselves to clear predictions of future behavior.

This is to be expected, given the fact that biological dictates are never as determinant in humans as they are in other species. Our brain gives us a greater deal of freedom over our instincts, and if that were not enough, culture and the social environment in which we operate also influence our behaviors.

... MORE LIKELY IT'S THE LOVE GENE.

According to this research, humans could be endowed with a gene

that predisposes our species to life in partnership (although the data are not very conclusive and we must wait for the results of similar studies to confirm or reject this conclusion). Thus this gene has been called the monogamy, pair-bonding or fidelity gene.

However, everything suggests that this gene's effects are not limited to increasing stability within established partnerships, but in some way or another it actually encourages pair-bonding itself. In other words, it has a similar role in humans (although its effects are much less obvious) as it does in voles.

In order to carry out its function, this gene must be able to encourage and maintain, at least for a certain period of time, an attraction towards a particular person. Originating in our instincts, this attraction would develop in our consciousness as a special feeling. It would become an inclination, affection, or fondness, which are only a few of the words we include in the definition of the word love. Thus, it is not too absurd at all to call it "the love gene."

PREVIOUS PREDICTIONS

That was exactly what we called this gene two years ago when we published on these issues (L. S. Lario, M. Lario and S. Lario, El gen del amor (http://www.unilibro.es/find_buy_es/libro/ediciones_del_bronce/el_gen_del_amor.asp?sku=599186&idaff=0) , Barcelona, Ediciones del Bronce, 1996). We chose this name because we find truly revolutionary the possible existence of a human gene capable of interfering with, and in many cases actually guiding, our emotional bonds.

This would open before us unthought-of horizons that to date are unsuspected. Given the effect emotional ups and downs tend to exert on our happiness, we should dedicate more time and attention to understanding this gene in the future.

Before closing, I must at least briefly mention that the two conclusions supported by the Swedish study were previously described in our book (cited above): The first one is that a gene could be implicated in our preference to live in partnerships, and the second is that the variation with which humans engage in pair-bonding could be determined by genetic differences (see chapters "El gen del amor" [The Love Gene] and "Posibles desigualdades en el patrimonio genético" [Possible Disparity in our Genetic Inheritance]).

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Edited by Solomon H. Snyder, Johns Hopkins University School of Medicine, Baltimore, MD, and approved July 14, 2008
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