Media again mangles epigenetics: Shutting off ‘love hormone’ unlikely to make us less social

If you are feeling less social today it may be because your body has ‘shut off your love hormone’. At least that’s the story being told by media outlets like the *Los Angeles Times,* which asked its readers in an article last month: “Could wear and tear on the ‘love hormone’ gene make us less social?”

It’s a bizarre question to ask if you understand how epigenetics works, but it’s a classic example of how the media regularly mangles genetics issues.

The so called ‘love hormone’ is oxytocin, a molecule produced in response to a number of normal processes, in particular in women during and right after childbirth. It is also injected medicinally to help induce or speed up labor and is important in the initiation of lactation in new mothers and in wound healing for both sexes.

Its role as the ‘love hormone‘—it goes by several other spectacular names such as the ‘moral molecule’ and the ‘most amazing molecule in the world‘—comes from findings that its levels surge after certain activities such as hugging, cuddling, playing with your dog, tweeting, orgasming and many other social bonding experiences. High levels of the hormone have been linked to trust, cooperation and sociability.

The *Times* piece reported on a study published in the *Proceedings of the National Academy of Sciences*. Researchers collected genetic data from saliva samples in a relatively small population— 120 people. They then gauged the extent to which the oxytocin gene was methylated, and compared that to each person’s sociability. Methylation is one of the most commonly studied epigenetic processes and is found throughout our genome. All our cells have a copy of our entire genome, but every cell doesn’t need to use every gene—your neurons don’t need to know how to make hemoglobin, and your skin cells don’t need to know how to make insulin. So cells have a number of ways to pack and tag regions of DNA that aren’t needed. They do the same with regions of the genome they want to access easily.

That’s epigenetics.

Methylation, the process by which a methyl group is added directly to a gene, is one epigenetic process that allows cells to shut-off or turn down the activity of a gene. If a gene has lots of methyl marks on it, a cell’s machinery can’t access the instructions to make the hormone. There’s a growing highly controversial belief that environmental events—stress, diet, exposure to certain chemicals—can influence (or drive) the addition/subtraction of these ‘off signals’ of certain genes.

In this study, the researchers found that people who had lower methylation of their oxytocin gene (actually the region just before the gene that ‘promotes’ the gene’s expression) were more socially engaging; they felt more secure about relationships and had a greater ability to recognize emotional facial expressions. And those people who had more methylation of their oxytocin gene were “less sociable” at least according to personal surveys and brain activity of regions linked to social behavior. In short: more methylation on oxytocin equalled less social and vice-versa.

Sarina Saturn, a psychology professor at the University of Portland, commented to GLP sister site Genetic Expert News Service (GENeS) that she believed the study could raise awareness of the link between alterations to the oxytocin gene and social disorders like autism and behavioral effects from things like abuse. However, she also noted the controversy over the hormone, indicating we should be cautious about making grand statements about oxytocin or its activity:

> Oxytocin is definitely mischaracterized and overgeneralized by both the scientific community and the press. For example, exogenous (administered) oxytocin can boost prosocial emotions (trust, love, generosity, closeness), 'anti-social' emotions (greed, envy, outgroup hate, ethnocentrism), or nothing at all (many null results have been generated, but these are difficult to publish).

The concerns about how the study has been interpreted go beyond our lack of concrete understanding of oxytocin. The cells studied (saliva normally contains epithelial cells and white blood cells) don't normally produce oxytocin. So it's not surprising to find epigenetic changes that shutdown or silence the oxytocin gene in these cell types, it would be much more appropriate (although admittedly far more difficult) to measure the methylation of this gene in the cells that are expected to produce oxytocin. Combining that with the fact the researchers did not measure any of the subject's oxytocin levels and the results become even more questionable. Harvard geneticist, Steven McCarroll, who was not involved with the study, cautioned against too much excitement:

> There's a lot of good evidence that oxytocin levels fluctuate in response to experience… But whether that relates to methylation is not known, and we wouldn't want to accept it — or dismiss it — casually….you'd want to measure that in the cells that actually make the oxytocin and that shape your mood and actions.

Even the study's lead author Brian W. Haas of the University of Georgia, said, "it's a leap" to claim more methylation means less oxytocin. Many scientists urge that studies (especially their own) be considered within the context of the field and not be characterized by the media as revolutionary breakthroughs. But this is what is happening far too often when a study in the field of epigenetics produces sensational results: the media and activists take far too many leaps.

Haas and his colleagues did not investigate what could have caused the differences in methylation levels between the social and the less social subjects. But considering the current state of popular excitement about epigenetics, many people read about this study and jumped to the unsupportable conclusion that environmental factors explain why 'social butterflies' have less methylation on their oxytocin. This is very evident from the *Times* story in how they prefaced the study (emphasis mine):

> If only we understood the machinery by which **nature and nurture interact** to produce the **social creatures** we are and will become. We might gain new appreciation for our individual differences. We might **know better how to prevent the emergence of despair, anxiety and hate**. In adulthood, we might make choices — in **diet and exercise**, in friends, in pastimes —

that promote the development of our better social selves.

Environment may play a significant role here but it might not. Epigenetic marks are often very transient and we still aren't sure what influences the behavior of these molecules. Personal genetics may also play a significant role in driving these changes, as might other molecules and proteins called transcription factors. But insinuating that diet and exercise can prevent the emergence of hate through epigenetic modifications to genes is an exaggerated and even dangerous leap, one that can lead to even more pseudoscience infiltrating a field that is already overrun by it.

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