Not Everything is Caused by Epigenetics

By Lila Abassi — May 16, 2016

Lately it seems that you cannot have a discussion about epigenetics without realizing that you have stepped on a proverbial land mine. Case in point is the recent controversial article by Siddhartha Mukherjee in the New Yorker magazine and his arguably overly simplistic explanation of epigenetics.

Epigenetics, in short, is the “study of heritable traits that don’t depend on the primary sequence of DNA.” That means that there are ways in which genes are turned on, or off, in individuals by environmental influences. The most popular example used to explain epigenetics is that it's what accounts for observed differences in pairs of identical twins. Two of the better known mechanisms by which this is accomplished is DNA methylation and histone acetylation. Methylation silences a gene and acetylation allows gene to be activated.

In a recent study, published in the American Journal of Stem Cells, researchers are claiming that a father contributes far more to the health and well-being of his offspring that was previously thought. The conventional meme is that the maternal role was the be all and end all of fetal health.

According to this study, this burden falls not only on the mother but on the father as well.

“We know the nutritional, hormonal and psychological environment provided by the mother permanently alters organ structure, cellular response and gene expression in her offspring,” said Joanna Kitlinska, PhD, associate professor of biochemistry, and molecular and cellular biology at Georgetown University Medical Center. “But our study shows the same thing to be true with fathers – his lifestyle, and how old he is, can be reflected in molecules that control gene function. In this way, a father can affect not only his immediate offspring, but future generations as well.”
In their paper, the researchers examined the effects of paternal age, environmental factors, and alcohol consumption. Advanced paternal age is a known risk factor for congenital abnormalities in the offspring. These have been validated through epidemiological studies and experimental animal models. “While the data does not establish a causative relationship, it does suggest that the age-associated methylation of male gamete DNA could contribute to the increased incidence of congenital disorders in progeny.”

Similarly, environmental influences such as starvation could markedly impact epigenetic variability in offspring. High stress levels in male mice these investigators observed resulted in low serum glucose in male and female offspring. Up to 75 percent of children with fetal alcohol spectrum disorders (FASDs) which include low birth weight babies, impaired cognitive function, visual-spatial abnormalities, have biological fathers who are alcoholics, suggesting paternal alcohol consumption has a detrimental impact on offspring. This can occur in the absence of maternal alcohol consumption. Again, similar results were obtained in animal studies.

It seems, however, like many epidemiological data, that the “link” between exposure X and the outcome Y is just that -- there is no causality. Given the limited involvement of the father in the reproductive and gestation process, it seems a real stretch to say paternal alcoholism causes FASD. The more likely scenario is getting an honest answer out of the mother regarding her alcohol consumption.

Additionally, in the realm of regulatory mechanisms involved in gene transcription, we are inappropriately assigning great importance to epigenetic modifications that we can’t prove. In the hierarchy of gene transcription, transcription factors trump epigenetic modification [4]. Placing so much emphasis on the role of epigenetics to explain that which we cannot know is dangerously simplistic.

This area of research is exciting and hotly contested. One has to beware of the publish or perish phenomenon in academia before we accept these “links” or associations as true causation.

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