French fries and skinny genes: Are we programmed to be obese?

When a British Medical Journal study found an interaction between how much fried food people eat, their genes and their weight, it was big news.

But several stories seem to miss the point, and some were just plain wrong.

Adjua Fisher at Philadelphia Magazine, for example, wrote that people who fall into the low genetic risk category could eat whatever they wanted.

> "However, if you are at low genetic risk for obesity, you can have at those fries without packing on the pounds. Totally unfair, right?"

But, in fact, the study showed no matter what your genetic risk was, if you ate fried foods a lot (four or more times a week in this study) you had higher BMI (by about one point) than your friends who stayed away from the breading.

The novel part of the study showed that if you have a genetic risk towards obesity then the effect fried food consumption has on your BMI doubles. The researchers calculated genetic risk as the number of risky alleles in your genome out of a total 32 known risk loci.

Only Michaeleen Doucleff at NPR pointed out that all the effects were pretty small, in fact, maxing out at about 2 BMI points.

> "The effect isn't huge. But for some people, the extra pounds could be the difference between being "normal" weight and overweight."

People with a BMI of 25 or less are considered to be at a healthy weight; BMI of 25 to 30 is termed overweight; and people with a BMI above 30 are categorized as obese.

The authors themselves noted that the mutations linked to increased body mass in humans don't explain weight variation very well at all. They wrote:

> In addition, the BMI associated loci identified to date account for only a small amount of variation (about 1.5%) in BMI.

The *Philadelphia Magazine* article also exaggerated the impacts this finding would have on patient care:

> “Now that we know genetics are tied to the effects of fried foods, genetic testing could easilybe used to help reveal who is at higher genetic risk for obesity so that they can take the proper preventative measures.”

But realistically, given the size of the effect and the expense of doing analyses with even this handful of known risk loci, physicians are a long way from using a battery of tests in a clinical setting.

And as Christopher Ochner an assistant professor of pediatrics at Icahn School of Medicine at Mount Sinai, New York said in USA Today:

> Most of us “don’t need fancy genetic testing,” to figure out if we are in that group “Just look at your mom and dad.”

No matter how people with this susceptibility are identified, getting to them early with targeted and specific information might help them make the behavioral changes they, in particular, need to keep to a healthy weight.

But this study also shows the limitations of our understanding of complex diseases like obesity. Scientists will keep exploring their genetic underpinnings, but it will likely be a long time before we have enough understanding to explain why some people suffer and others don’t.

Additional Resources:

- Whole-genome sequencing in your doctor’s office? A reality check, but sooner than later, Genetic Literacy Project
- ‘Fat gene’ stands falsely accused, offers lesson in gene-gene interactions, Genetic Literacy Project
- Genes & obesity, Genetic Literacy Project