What role does fast food play in the world’s obesity epidemic, and how can we moderate its effect? This week’s journal offers several new insights into the complex interplay between dietary intake, environmental exposure, and genetic risk. We know that eating fried and other energy dense foods increases the risk of obesity, even if the role of certain types of fat in raising cardiovascular risk is uncertain (doi:10.1136/bmj.g2238). We also know that some people are at increased genetic risk of obesity. Qibin Qi and colleagues have now found an interaction between high intake of fried food and high genetic risk, as measured by pooling the effects of common genetic variants (doi:10.1136/bmj.g1610). In an analysis of data from 37 000 men and women they found that the risk of obesity was highest in those who ate a lot of fried foods and also had a high genetic risk score.

As Alexandra Blakemore and Jessica Buxton say in their linked editorial (doi:10.1136/bmj.g1900), this work provides formal proof of interaction between genetic risk and environment, but it won’t have a direct effect on personal healthcare. This is because, although genetic risk scores are robust at the population level, they have poor predictive value for an individual. But they call for the genetics of obesity to be taken more seriously in efforts to tackle the current alarming global rise in the prevalence of obesity. They are particularly keen that doctors should take into account the increasing number of “Mendelian” forms of obesity, with new ones being reported almost monthly. They charge us with taking a more sophisticated approach to treatment where such genetic causes are likely, because lifestyle change and medical approaches are unlikely to help people with the severest forms of obesity. For such people, drugs and even surgery will be needed. Elsewhere in the journal this week Jane Blazeby and colleagues explore the uncertainty around which surgical procedure is best for people with severe and complex obesity (doi:10.1136/bmj.g1763).

Back to the environment, researchers have long tried to prove that having ready access to fast foods increases the amounts people eat and their risk of weight gain. But the results have been mixed. Published this week, a study by Thomas Burgoine and colleagues takes a new approach. Instead of looking at the proximity of fast food outlets to people’s homes, they looked at food outlets near people’s work and along their commuter routes. After adjusting for many confounding factors, they found that people who were more exposed to fast food outlets had a higher body mass index and were more likely to be obese (doi:10.1136/bmj.g1464).

The authors suggest that their study supports calls for more restricted access to fast food outlets. Perhaps first to go should be the Burger King restaurant in the main concourse at Addenbrooke’s Hospital in Cambridgeshire, where the study was based, a move that is reportedly being considered (http://bit.ly/1ifbhoS). But in her linked editorial Kathryn Neckerman says that such an approach was likely to have little effect (doi:10.1136/bmj.g1817). In what she calls “a kind of nutritional ‘whack-a-mole,’” closing takeaway outlets might lead other retailers to expand their offerings of unhealthy food. Rather than restricting takeaway food we should, she says, seek to transform it, by making healthy food as visible, tasty, and cheap as unhealthy food. Change the menu, not the venue.

Cite this as: BMJ 2014;348:g2252
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