Biologists at CECs publish a comprehensive study that examines the information contained in DNA, energy expenditure and how this is related to obesity.

To improve our understanding of the relationship between our DNA and how much we eat, we need to know which cells are important in the regulation of energy homeostasis. Our brain possesses neurons that are in charge of regulating the amount of food we eat and it is interesting to note that even artificial activation of these neurons pushes us to eat more than we need to.

These neurons driving us to ingest food are known as **AgRP neurons**. Other neurons, **POMC neurons**
, are in charge of limiting the quantity of food we eat. A fine balance between the activity and functioning of these two populations of neurons permits the correct energy balance to be maintained. But alterations in this fine balance lead to pathologies such as obesity. These neuron types are localized in the hypothalamus, a brain region located at the base of our brain, which provides access to the peripheral signals that are to be found circulating in our blood. Our fatty adipose tissue produces and secretes a hormone, called leptin. This hormone regulates the activity of AgRP and POMC neurons in an inverse manner. So, leptin, acting as a sign that energy reserves exist, silences the AgRP neurons and switches on the POMC neurons, having the resultant effect of lessening the amount of food we ingest and increasing the amount of energy we expend.

Now, how is our DNA linked to our desire to eat? DNA contains the information necessary to build the proteins that make up our cells, but it also contains the information needed by our genes in order to respond to changes in the environment. Bredford Kerr and doctorate student, Rodrigo Torres have

published their understanding of the function of DNA in relation to our desire to eat in the journal Exper imental Physiology . Their studies use mice that lack a protein called MeCP2. This protein can bond with methylated DNA.

DNA methylation is a chemical signal for DNA that is capable of altering the interpretation of the information contained within DNA but without modifying the DNA sequence. If the bases of the genetic code were letters of the alphabet that form words, these chemical modifications would be accents that can change the meaning of the message. “Hence the importance -indicates Ro drigo Torres - as we must be able to respond to environmental changes despite the fact that our genetic information remains constant over time. What actually happens is that there is a change in the way the information is interpreted, with the expression of some genes favoured and the expression of others diminished. Thus, in relation to energy homeostasis, we saw that wild type mice responded to leptin as expected, with increased POMC levels and lower amounts of food being eaten. But animals lacking the MeCP2 protein are incapable of responding to leptin at both levels, highlighting the role of MeCP2 in the regulation of POMC". (See Figure 1).