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Press release

Obesity: Does our second brain work too well?

Scientists from Inserm have just demonstrated how a high fat and sugar diet prevents the natural destruction of neurons from the enteric nervous system in mice. It seems that, by slowing down natural ageing of the "second brain", this particular diet contributes to the development of obesity. This is the surprising conclusion of a joint French/German research project coordinated by Michel Neunlist, Director of Research at Inserm, and Raphaël Moriez from Inserm unit 913 in Nantes, in their paper "Neuropathies of the enteric nervous system and digestive pathologies: involvement of enteric glial cells". The result is that these neutrons over-proliferate, overwork and make for accelerated gastric emptying. This effect could contribute to the development of obesity by reducing the satiety signals and so increasing the food intake. These works are published in *The Journal of Physiology*.

In addition to our brain that controls all our physiological functions, we also possess a second brain that regulates the digestive functions. This other brain, known as the enteric nervous system (ENS), runs the length of the digestive tube. It is made up of over 100 million neurons, which makes the digestive tube the second most important neurological organ in our body. The ENS plays a central part in controlling numerous functions, ranging from regulating digestive motility (gastric emptying, colic transit), through intestinal barrier functions that protect from external pathogenic agents, to the absorption of nutrients.

Researchers have been finding out about the key role of the ENS over recent years. It plays a major part in numerous pathologies, not only digestive (functional digestive disorders, chronic intestinal inflammatory disorders), but also extra-intestinal, such as Parkinson's disease. Surprisingly, despite that fact that obesity is an increasing problem that is posing a stiff challenge to Public Health, very little is known about the involvement of the ENS in this pathology. All the more surprising because the ENS also plays a part in controlling the key functions that help absorb nutrients and regulate the intake of food.

In order to find out more details on this subject, the researchers[1] studied the impact of a high fat and sugar diet on the ENS and its effect on gastric emptying and intestinal transit.

[1] From Inserm unit U913 of the University of Nantes working with German researchers (University of Munich) and from the Inserm UMR U773

It appears that, by preventing maturation of the second brain, a high fat and sugar diet contributes to the development of obesity.

These works unexpectedly showed up that when this diet is administered to young mice, it inhibited the loss of neurons that is normally observed in the reference population over time.

"We think that by inhibiting the natural development of the enteric nervous system over time, a high fat and sugar diet prevents the digestive tube from adapting to an adult diet by maintaining the young phenotype corresponding to a phase of life where the food intake is at its maximum", says Raphaël Moriez

On a functional level, the neuroprotection induced by the hypercalorific diet eventually modifies the gastric functions. So in animals that are given a high fat and sugar diet, gastric emptying takes place too fast compared to the reference population, and could be directly related to the development of obesity by decreasing the satiety signals and increasing the food intake. This same phenomenon of accelerated gastric emptying has been observed in obsess patients.

From a physiological point of view, this "neuroprotective" effect is associated to an increase in the gastric production of a neuroprotective factor, GDNF, itself induced by leptin, a hormone that is now well know for its role in regulating the feeling of satiety in human beings.

These works have highlighted the ability of nutrients to modulate the operation of the second brain and the part played by this brain in developing obesity, in particular in the young. We believe that we will eventually be able to prevent neurodegenerative disorders or even central nervous system disorders using nutritional approaches.

Sources

Diet-induced obesity has neuroprotective effects in murine gastric enteric nervous system: involvement of leptin and glial cell line-derived neurotrophic factor

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