An architect gene is involved in the assimilation of breast milk

The presence of a mutated Hoxd3 gene could explain certain types of intestinal insufficiency in newborns, according to researchers from the University of Geneva.

A growth retardation due to a single gene

His team has produced several lines of mice carrying precise and targeted mutations in the HoxD gene cluster. “All of the pups carrying a Hoxd3 gene coding for a dysfunctional protein were severely stunted during the suckling period and displayed an abnormal development of the small intestine”, explains Jozsef Zakany, researcher at the Department of Genetics and Evolution and first author of the study. This gene therefore has a crucial role in the developing gut of suckling mice.

Unlike adults, mammalian babies “eat” milk, incorporating it as undigested droplets. “The Hoxd3 gene appears to promote the maintenance of the intestinal cells responsible for absorbing this nutrient throughout the suckling period. Growth has indeed systematically resumed in surviving mutated animals, reflecting changes in the intestinal wall following weaning”, notes Jozsef Zakany.
From mice to humans

The human and murine genomes are similar, including the genes of the HoxD complex. The same applies to their physiology of nursing. According to the researchers, the presence of a Hoxd3 gene displaying this kind of mutation probably contributes to certain types of intestinal insufficiency in premature infants, such as necrotizing enterocolitis of the newborn. This affliction, which varies from 4 to 22% in babies with a very low birth weight, requires a rapid diagnosis and urgent medical care. “In less than a week, it is possible to test the DNA of infants suspected of having such intestinal insufficiency, to detect a possible mutation of the Hoxd3 gene. This is a path to be explored at the clinical level, as the causes of this disease are still unknown”, concludes Denis Duboule.