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Oct 16, 2015 01:31 PM EDT

Zebrafish Study Shows New Insights On Human Heart Defects

Researchers now have new insights about the causes of congenital heart defects, a rare developmental disorder, by experimenting with zebra fish, according to a news release.

Researchers at New Zealand's University of Otaga have been working with zebra fish, which has allowed them to have a better understanding of the structural abnormalities of the heart, which is common with zebra fish.

The heart disorder known as Cornelia de Lange Syndrome (CdLS) occurs in about 1 in 10,000 births worldwide. CdLS results in a range of developmental abnormalities, which are both physical and cognitive. About 70 percent of people with CdLS have congenital heart defects, according to the researchers.

CdLS occurs from mutations in subunits or regulators of cohesin, which are a group of linked proteins necessary for cell division and other cell processes. It is one of a spectrum of disorders known as cohesinopathies, the researchers revealed.

"Now, through our new study, we show that lowering levels of a particular cohesin protein called Rad21 in embryonic zebra fish produces similar types of heart defects to those found in people with CdLS," Julia Horsfield, researcher and professor at the Otago Department of Pathology.

Horsfield claimed that to this point the mechanisms leading to heart defects in CdLS have been poorly understood.

The researchers found that an essential downstream effect of depleting the protein was that particular cells failed to migrate to the heart, this is where they facilitate in developing heart structures, according to the news release. When they experimented with the zebra fish, they found that zebra fish embryos partially depleted of Rad21 developed almost normally, except for the presence of the structural abnormalities of the heart, according to Horsfield.

"Our findings suggest that heart development is exquisitely sensitive to the available amount of Rad21 and cohesion, it can be compromised even when there is sufficient cohesin present to support relatively normal growth," Horsfield said. "This also raises the possibility that mild mutations in cohesin genes may be the cause of some fraction of congenital heart defects in the general population."

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