

Numerous researchers have verified a robust correlation between cholera and the human blood group O phenotype since the linkage was first identified by Barua and Paguio in 1977. Blood type O significantly influences the severity of the sickness, even though it has no effect on the likelihood of contracting *Vibrio cholerae*. For instance, those with blood type O were eight times more likely to require hospitalization for severe cholera when the disease first appeared in Peru in 1991. Thus, blood type can literally mean the difference between life and death for those afflicted with *V. cholerae*.

Furthermore, there's a good chance that the link between blood type O and severe cholera has influenced our evolutionary past. The Ganges delta, where cholera has probably been endemic for ages, has the lowest proportion of the O blood group phenotypic worldwide. This implies that cholera has left a genetic mark in historically endemic places and indicates that the number of people with severe types of cholera will increase as cholera expands to locations where blood type O is more common.

This article from F. Matthew Kuhlmann and colleagues at Washington University advances our understanding of the mechanism underlying the correlation between blood type and cholera severity. It appears in the *American Journal of Tropical Medicine and Hygiene*. The enteroids, a cell culture model created from ileal and colonic stem cells that develop into tissue that closely resembles the intestinal surface in vitro, were the subject of the study by the authors. The enteroids were exposed to cholera toxin. The researchers used this method to compare the reactions of enteroids treated with cholera toxin obtained from blood type O donors to those of enteroids stimulated with the same agent obtained from blood group A donors.

Researchers found that enteroids made from blood type O stem cells had a noticeably higher cyclic adenosine monophosphate response to cholera toxin. The researchers next used clustered regularly interspaced short palindromic repeats technology to change the gene that regulates glycosylation of the ABO blood group glycans, resulting in the conversion of a blood type A-derived cell line to a blood type O phenotypic cell and the demonstration of a similar

impact.

When combined, these findings offer strong proof that cells carrying the blood type O-associated glycan are more sensitive to the effects of cholera toxin. This is consistent with the epidemiologic finding that blood type O does not affect infection risk; it just affects the severity of the disease. The exact mechanism by which cholera toxin causes a greater response in type O cells is still unknown, despite this work. Remarkably, the study's conclusion that blood group O-derived cells were more susceptible to the effects of cholera toxin was unrelated to the level of toxin binding to cells, which was comparable in type A- and type O-derived cells.

The Kuhlmann research aligns with the intriguing findings of Heggelund et al.'s recent publication, which revealed the first high-resolution crystal structure of the cholera toxin attached to the A and O blood group glycans. Heggelund et al. did not assess the cellular reaction to cholera toxin; however, they did show that the toxin attaches to the blood type O determinant more strongly and in a variety of orientations than it does to the blood group A determinants.

The story is all the more fascinating when one realizes that the main receptor for cholera toxin is not the ABO blood group determinant. As an alternative, the cholera toxin's high-affinity attachment to the appropriate GM1 ganglioside receptor is what causes the cellular reaction to it. As such, the effect of binding affinity and orientation to ABO-related blood antigens on the cellular response to cholera toxin remains unclear. Comprehending the effects of differences in ABO blood group glycans on the interaction between cholera toxin and GM1 ganglioside may be the last step towards solving this at a molecular level.

For instance, this might depend on the binding of toxins, but it might also depend on particular interactions between GM1 ganglioside and the glycans of the ABO blood group on the cell surface. It appears likely that there is yet more to discover as a result. It is hoped that a deeper comprehension of the molecular mechanisms underlying this long-noted genetic susceptibility to this old disease may result in novel strategies for battling this significant

worldwide pathogen.

Source

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