

## Maitotoxin-induced membrane blebbing and cell death in bovine aortic endothelial cells

### ABSTRACT

The results indicate that maitotoxin-induced  $\text{Ca}^{2+}$  influx in bovine aortic endothelial cells activates COP activation, which is associated with controlled membrane blebbing leading to uncontrolled bleedout, lactate dehydrogenase release, and eventual cell death.

### INTRODUCTION

Maitotoxin, a common food additive, is a toxin that causes cell death in the large intestine. It is found in foods such as milk, cheese, and meat and is commonly found in foods such as fish, poultry, and eggs.

Maitotoxin is also present in the blood and urine of humans, and is thought to be the cause of death in some cases of meningitis. In the past, it was used to make vaccines to combat meningitis.

The authors of this study tested the effect of Maitotoxin on the membrane of bovine aortic endothelial cells (ABEC), and found that Maitotoxin caused cell death in this cell line.

Maitotoxin increases the expression of Maitotoxin (MTX) is one of the most potent marine toxins, found in the "red-tide" dinoflagellate, *Gambierdiscus toxicus*, and is responsible for seafood poisoning from Ciguatera. At subnanomolar levels, MTx triggers a significant increase in cytosolic free  $\text{Ca}^{2+}$  concentration ( $[\text{Ca}^{2+}]_i$ ) in all cells examined so far, not by release of  $\text{Ca}^{2+}$  from internal stores, but by activation of large endogenous pores or COP during activated subsequently leading to the identification of Although the kinetic model of the activation or dilation of P2X by MTX is not specific to this toxin, it appears that it initiates a similar cell death cascade in which cytosolic  $\text{Ca}^{2+}$  and vital dye uptake are relevant. The plasmalemma's opening through microscopy suggests that the cell will undergo a significant shift in ionic concentration gradients, leading to increased redistribution of water and swelling of cells.

### CONCLUSION

Remarkable conclusions To sum up, MTX-treated BAECs trigger a cascade of events that includes cell death, CaNSC activation, and increase in  $[\text{Ca}^{2+}]_i$ . Upon terminating the treatment, however, membrane breaks out and COP is formed, which appears to be 'a unique molecule' associated with the plasma membrane (specifically, the activated nucleotide or its covalent antigen), but it is unclear whether LDH release corresponds to embryonic blood types, massive protoplasty, etc.