

# Effect of cod trypsin on rhinovirus 1A infectivity

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## Introduction

The common cold is an upper respiratory tract infection (URI) caused by several viruses including the Human Rhinovirus (HRV). HRV is a positive-sense single-stranded-RNA virus. The HRV viral genome produces 11 proteins, four of which are capsid proteins VP1, VP2, VP3 and VP4. These proteins have numerous cleavage sites for proteases. Cod trypsin (Ct) is a cold adapted serine-protease that cleaves polypeptide chains next to arginine and lysine residues. Studies have demonstrated that formulations containing Ct have anti-pathogenic properties.

The aim of the project is to analyse the efficacy of cod trypsin in a specific formulation against HRV. An additional goal is to find natural compounds that increase the anti-pathogenic effect of the Ct formulation.

## Method and materials

RD cells were grown in 96-well microtiter plates and incubated at 34 °C and 5% CO<sub>2</sub> for 3 days or until 90% confluency. Rhinovirus-1A was diluted to 10<sup>-4</sup> from a concentrated stock solution (1.3x10<sup>-5</sup> TCID<sub>50</sub>) and treated with 16 or 32 U/mL of Ct followed by incubation for 60 min. Benzamidine was added subsequently to inhibit the trypsin activity before placing the solution on the cells. Positive and negative controls were used for comparison.

## Results

Ct at a concentration of 32 U/mL delayed Rhinovirus-1A infection of RD cells about 2-3 days compared to positive control whereas Ct at 16 U/mL delayed infection by 1-2 days.

## Conclusion

The results demonstrate that Ct can delay Rhinovirus-1A infection of RD cells by several days depending on cod trypsin concentration.