A viral effector suppresses cell-to-cell spread of silencing by targeting two plasmodesmal receptor-like kinases

**Abstract**

RNA interference (RNAi) in plants can move from cell to cell, allowing for systemic spread of an anti-viral immune response. How this cell-to-cell spread of silencing is regulated is currently unknown. Here, we describe that the C4 protein from *Tomato yellow leaf curl virus* can inhibit the intercellular spread of RNAi. Using this viral protein as a probe, we have identified the receptor-like kinase (RLK) BARELY ANY MERISTEM 1 (BAM1) as a positive regulator of the cell-to-cell movement of RNAi, and determined that BAM1 and its closest homologue, BAM2, play a redundant role in this process. C4 interacts with the intracellular domain of BAM1 and BAM2 at the plasma membrane and plasmodesmata, the cytoplasmic connections between plant cells, interfering with the function of these RLKs in the cell-to-cell spread of RNAi. Our results identify BAM1 as an element required for the cell-to-cell spread of RNAi and highlight that signalling components have been co-opted to play multiple functions in plants.

**C4 from TYLCV is a plasma membrane/plasmodesmal protein and suppresses cell-to-cell spread of RNAi**

**C4 from TYLCV interacts with the receptor kinase BAM1**

**BAM1 promotes the cell-to-cell spread of RNAi**

**BAM1 has two homologues**

**Conclusions**

- BAM1 promotes the cell-to-cell spread of RNAi.
- BAM2 and BAM1 act redundantly.
- C4 targets BAM1 and suppresses their activity.

This is the first description of an element (BAM1/BAM2) required for the cell-to-cell spread of RNAi.

Our work identifies a novel function of the RLKs BAM1/BAM2 in the cell-to-cell spread of RNAi; however, the underlying molecular mechanism is still elusive.