Cauliflower mosaic virus uses the plant host cell to sense the aphid vector and optimise Its own transmission

Alexandre Martinière, Stéphane Blanc, Martin Drucker

CAULIFLOWER MOSAIC VIRUS USES THE PLANT HOST CELL TO SENSE THE APHID VECTOR AND OPTIMISE ITS OWN TRANSMISSION

Alexandre Martinière[1,2]; Stéphane Blanc[1]; Martin Drucker[1].

[1]Equipe CaGeTE. UMR BGPI. TA A54K Campus International de Baillarguet. 34398 Montpellier cedex 5. France; drucker@supagro.inra.fr
[2]Plant Endomembrane Research Group, School of Life Sciences, Oxford Brookes University, Oxford OX3 0BP, UK.

Transmission of Cauliflower mosaic virus (CaMV) by aphids depends on the presence of viral inclusions, the Transmission Bodies (TB), in infected plant cells. TB contain the aphid transmission factor, the viral protein P2, and the viral protein P3. When TB do not form, no transmission occurs even when infected cells contain functional P2 (Khelifa et al. 2007, J. Gen. Virol., 88: 2872). Thus, TB are structures specialised for transmission, hence our interest to study their formation and function (Martinière et al. 2009, Plant Journal, 58:135). We detected that stress induces import of apparently soluble tubulin into TB. FRAP experiments indicated a high turnover rate of TB-contained tubulin. In aphid transmission experiments, we found that aphids fed on stressed infected leaves transmitted CaMV better than aphids fed on control leaves, that there was a positive correlation between tubulin entry in TB and transmission efficiency, and that aphid punctures themselves might induce rapid (within seconds) tubulin influx into TB. The Ca^{2+} ionophor A23187 induced tubulin influx into TB; the Ca^{2+} channel blocker La^{3+} completely inhibited transmission. The microtubule depolymeriser oryzalin inhibited transmission indicating involvement of microtubules in CaMV transmission. Finally, incubation of infected protoplasts with NaN_3 induced disintegration of TB and relocalisation of P2 and virions on microtubules, concomitant with drastically increased CaMV transmission. Preliminary data indicate that also ROS might induce TB disintegration. Taken together, our results indicate that a Ca^{2+} signalling cascade, which might be triggered as an early plant defence response to exploratory intracellular stylet punctures of the aphid vector, "activates" the otherwise "dormant" TB for transmission by causing massive entry of tubulin in TB, possibly followed in a second step by redistribution of P2 and virions on microtubules all over the cell. Thus it seems that CaMV deflects host perception and signalling pathways to perceive the presence of the aphid vector and to actively prepare its own acquisition.