Does Cauliflower mosaic virus “sense” the presence of its aphid vector?

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Equipe CaGeTE, UMR BGPI, CIRAD-INRA-SupAgro, Campus International de Baillarguet, 34398 Montpellier Cedex 5, France

Transmission of Cauliflower mosaic virus (CaMV) by aphids depends on the presence of viral electron-lucent inclusion bodies (EL) in infected plant cells. EL contain the aphid transmission factor, the viral protein P2, and the viral protein P3. When EL do not form, no transmission occurs even when infected cells contain functional P2 (Khelifa *et al.*, 2007, *J Gen Virol* 88, 2872-2880). Thus, EL are structures specialised in transmission, hence our interest to study their formation and functions.

We detected that stress induces import of apparently soluble tubulin into EL. FRAP experiments indicated a high turnover rate of EL-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 EL and transmission efficiency, and that aphid punctures themselves might induce tubulin influx into EL. The Ca^{2+} ionophore A23187 induced tubulin influx into EL ; the Ca^{2+} channel blocker La^{3+} completely inhibited transmission. Finally, incubation of infected protoplasts with NaN_3 induced disintegration of EL and relocalisation of P2 and virions on microtubules, concomitant with drastically increased CaMV transmission.

Taken together, our results indicate that a Ca^{2+} signalling cascade, which might be triggered as an early plant defence response by exploratory intracellular stylet punctures of the aphid vector, "activates" the otherwise "dormant" EL for transmission by causing massive entry of tubulin in EL, possibly followed by redistribution of P2 and virions on microtubules all over the cell. Thus it seems that CaMV might deflect a host defence pathway to perceive the presence of the aphid vector and to prepare its acquisition.