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Virus yellows disease in sugar beet – a resistance mechanism for the future.

The virus yellows disease (VY) in sugar beet is caused by several aphid-transmissible viruses. Namely, beet yellows virus (BYV, *Closterovirus*), beet mild yellowing virus (BMYV) and beet chlorosis virus (BChV) (both *Polerovirus*). Due to a similar transmission mechanism and synergistic effects, beet mosaic virus (BtMV, *Potyvirus*) is often considered YV associated. Apart from BYV, all aforementioned viruses carry a protein called viral protein genome linked (VPg) that is covalently bound to their genomes 5' end. The VPg is critical for translation initiation and thus viral protein biosynthesis within its host. Previous studies show that potyviral VPgs often interact with plant susceptibility factors the eukaryotic translation initiation factors (eIFs). Furthermore, initial studies in *Arabidopsis thaliana* support that this concept might be used to control paleoviruses as well. If the VPg-eIF interaction is disturbed e.g., by mutations, the virus is unable to replicate within the plant. Due to its heredity, this mechanism is called recessive resistance. We used molecular biological methods such as yeast two hybrid and bimolecular fluorescence complementation to identify different sugar beet eIF isoforms as protein-interaction partners for the respective VY-VPgs. Subsequently, T_0 knockout sugar beets of the corresponding candidate genes were generated by genome editing and subjected to a resistance test. Knockout of *Bv-eIF(iso)4E* resulted in resistance to BChV, which significantly reduced the virus titer as well as infection rates compared to unedited plants. By this making *Bv-eIF(iso)4E* the first known resistance mechanism to a member of VY in sugar beet and the first recessive resistance to poleroviruses in a food crop. Furthermore, this work shows for the first time the suitability of genome editing in sugar beet by focusing on an agronomical important trait. Meanwhile, resistance to BMYV and BtMV could not be obtained. An explanation for this could be simultaneously detected interactions of BMYV-VPg with Bv-eIF(iso)4E and Bv-eIF4E, therefore potentially requiring a double knockout of the respective eIF4Es, which could not be obtained due to lethality of the mutation. Still, after showing that recessive resistance to poleroviruses can be implemented in the sugar beet host system, this knowledge can be used in the future for breeding purposes to identify natural resistance genes or further editing targets. Rollwage, L; van Houtte, H; Hossain, R; Wynant, N; Willems, G. and Varrelmann, M. (2024) Recessive resistance against beet chlorosis virus is conferred by the eukaryotic translation initiation factor (iso)4E in *Beta vulgaris*. *Plant biotechnology journal*, 22, 2129–2141.