The *Lactococcus lactis* maltose ABC transporter complex mediates sensitivity to the circular bacteriocin garvicin ML

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Spontaneously resistant mutants of *Lactococcus lactis* IL1403 to the circular bacteriocin garvicin ML (GarML) were investigated. Preliminary characterization showed that these mutants had lost the ability to metabolize starch and maltose. Growth experiments indicated a link between sensitivity to GarML and sugar metabolism; Growth on non-CCR-inducing sugars maltose and galactose increased sensitivity to the bacteriocin whilst presence of CCR-inducing glucose alleviated this effect. A 13.5 kb chromosomal deletion event, involving 12 ORFs, was shown to cause the mutant phenotype. The genes implicated in this deletion mainly encode functions related to metabolism of starch and its breakdown products, among which *malEFG* encode a maltose CCR-regulated ABC transporter that could potentially function as a target for the bacteriocin. Indeed, complementation with these three genes restored the sensitivity to GarML, and sensitivity was even shown to increase with higher expression of *malEFG* over a 50-fold range. Complementation with the individual genes did not restore sensitivity, indicating that a complex is required for sensitivity to GarML. Furthermore, inactivation of the ATP-binding subunit of the complex did not affect sensitivity, demonstrating that a functional transporter is not essential for sensitivity to GarML. These results together suggest an essential role of the maltose ABC transporter in the antimicrobial activity of GarML, which to our knowledge is the first time a specific protein complex has been demonstrated to confer sensitivity to a circular bacteriocin.