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Fructose uptake in the soil bacterium Pseudomonas putida occurs through a canonical phosphoenolpyruvate (PEP)-dependent sugar transport system (PTSFru). The logic of the genetic circuit that rules its functioning is puzzling: the transcription of the fruBKA operon, encoding all the components of PTSFru, can escape the repression exerted by the catabolite repressor/activator protein Cra solely in the presence of intracellular fructose-1-P, an agonist formed only when fructose has been already transported. To study this apparently incongruous regulatory architecture, the changes in the transcriptome brought about by a seamless Δcra deletion in P. putida strain KT2440 were inspected under different culture conditions. The few genes found to be upregulated in the cra mutant unexpectedly included PP_3443, encoding a bona fide glyceraldehyde-3-P dehydrogenase. An in silico model was developed to explore emergent properties that could result from such connections between sugar uptake with Cra and PEP. Simulation of fructose transport revealed that sugar uptake called for an extra supply of PEP (obtained through the activity of PP_3443) that was kept (i.e., memorized) even when the carbohydrate disappeared from the medium. This feature was traced to the action of two sequential inverters that connect the availability of exogenous fructose to intracellular PEP levels via Cra/PP_3443. The loss of such memory caused a much longer lag phase in cells shifted from one growth condition to another. The term "metabolic widget" is proposed to describe a merged biochemical and regulatory patch that tailors a given node of the cell molecular network to suit species-specific physiological needs. IMPORTANCE The regulatory nodes that govern metabolic traffic in bacteria often show connectivities that could be deemed unnecessarily complex at a first glance. Being a soil dweller and plant colonizer, Pseudomonas putida frequently encounters fructose in the niches that it inhabits. As is the case with many other sugars, fructose is internalized by a dedicated phosphoenolpyruvate (PEP)-dependent transport system (PTSFru), the expression of which is repressed by the fructose-1-P-responding Cra regulatory protein. However, Cra also controls a glyceraldehyde-3-P dehydrogenase that fosters accumulation of PEP (i.e., the metabolic fuel for PTSFru). A simple model representing this metabolic and regulatory device revealed that such an unexpected connectivity allows cells to shift smoothly between fructose-rich and fructose-poor conditions. Therefore, although the metabolic networks that handle sugar (i.e., fructose) consumption look very similar in most eubacteria, the way in which their components are intertwined endows given microorganisms with emergent properties for meeting species-specific and niche-specific needs.