EHEC sings: pour some sugar on me

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Gastrointestinal (GI) bacteria sense diverse environmental signals, including host hormones and nutrients, as cues for differential gene regulation and niche adaptation. Although the impact of carbon nutrition on the colonization of the gut by the microbiota has been extensively studied, the extent to which carbon sources affect the regulation of virulence factors by invading pathogens has not been fully defined. The enteric pathogen enterohemorrhagic *Escherichia coli* (EHEC) gages sugar sources as an important cue to regulate expression of its virulence genes. Specifically, this sugar dependent regulation fine tunes the expression of the locus of enterocyte effacement (LEE) pathogenicity island, which encodes for a type three secretion system, effectors, and an adhesin necessary for the formation of attaching and effacing (AE) lesions on enterocytes. Glycolytic environments inhibit the expression of the LEE genes. Conversely, growth within a gluconeogenic environment activates expression of these genes. Part of this sugar-dependent regulation is achieved through two transcription factors: KdpE and Cra. Cra and KdpE interact to optimally directly activate expression of the LEE genes in a metabolite dependent fashion. This sugar dependent regulation is key during infection of the mammalian host, given that a *kdpE* mutant is attenuated *in vivo*. Additionally, a novel two component signal transduction system, named FusKR (where FusK is a membrane bound histidine sensor kinase, and FusR a response regulator) that senses fucose, controls expression of the LEE genes. This fucose-sensing system is required for robust EHEC intestinal colonization. During growth in mucus, the glycophagic prominent member of the GI microbiota, *Bacteroides thetaiotaomicron,* supplies fucose to EHEC, modulating its virulence gene expression. Our findings suggest that EHEC uses fucose, a host-derived signal made available by the microbiota, to modulate EHEC virulence and metabolism, and suggest a new layer of complexity in the inter kingdom signaling that underlies EHEC pathogenicity