Under the current PSoup algebra, sucrose and genotype modifiers combine only multiplicatively and do not gate each other. As a result, sucrose acts as a uniform gain across genotypes. This guarantees linear scaling of branching output across sucrose environments and mathematically prevents genotype × sucrose (G×E) interactions. So when QTL effects are wired into the modifiers it will not capture the GxE if we assume that the E is coming from the sucrose.

**1. Biology says: sucrose is not just food, it’s also a signal that talks to hormones**

People used to think sucrose = carbon = growth fuel. That’s true, but incomplete. There’s now a big body of work saying sucrose is also an *active signal* that can trigger bud outgrowth and shoot branching, partly by promoting cytokinin and by suppressing strigolactone repression of buds. Sucrose can induce axillary bud release extremely fast, on the scale of hours, even before bulk nitrogen changes, and even before you see strong changes in auxin transport. This is now considered a bona fide regulatory role of sugar in branching (Barbier et al., 2019)

So sucrose interacts with hormones like:

* strigolactones (SL),
* cytokinins (CK),
* ABA,
* auxin transport / export from buds.

That interaction is the whole argument that sucrose supply (an “environmental-ish” thing, because it depends on photosynthesis, shading, stress, etc.) should produce genotype-specific branching behaviour. Review papers in the last ~1 year basically frame sucrose as a “systemic regulator of branching,” not just a nutrient (Xiong et al., 2024)

2. Strigolactone pathway mutants should change sucrose responsiveness

MAX / D14 / D27 / MAX2 / SMXL6,7,8 mutants (the canonical strigolactone signalling pathway)

In Arabidopsis and other species, mutants that block SL synthesis (max3, max4, d27) or SL perception/signaling (max2, d14, smxl6 smxl7 smxl8, etc.) are hyperbranched because they release buds from apical dominance. These mutants essentially remove the “don’t branch” brake. (Wang et al., 2015)

There’s increasing evidence that sucrose *feeds into that exact brake*. Recent work argues sucrose availability suppresses SL signalling, e.g. sucrose reduces SL repression of axillary buds, and sucrose promotes outgrowth partly by weakening SL action and/or boosting cytokinin. In other words, the sucrose signal and the SL brake are not independent — sucrose modulates SL pathway activity. Scott A. Finlayson

A wild type is SL-repressed at low sucrose, but as sucrose goes up, sugar can partially overcome the SL brake.  
A strong SL-insensitive mutant (like max2 or smxl6 smxl7 smxl8 triple) already has that brake disabled. So giving sucrose again shouldn’t change branching as much, because the pathway sucrose normally antagonises is already gone.

That is *literally* a genotype × sucrose interaction:

* WT: branching is low at low sucrose, higher at high sucrose.
* SL-insensitive mutant: branching is already high even at low sucrose, and the slope versus sucrose is flatter because there’s nothing left for sucrose to release.
* This concept (sucrose affecting SL signalling → bud release) is described as sucrose “integrating” with SL to modulate bud outgrowth and shoot architecture. [mdpi.com+1](https://www.mdpi.com/2311-7524/10/12/1348?utm_source=chatgpt.com)  
  It’s also been described as sucrose promoting branching through cytokinin biosynthesis/signaling and transport, which again is hormonally mediated and thus genetically targettable. [OUP Academic](https://academic.oup.com/plphys/article/185/4/1708/6094651?utm_source=chatgpt.com)
* So, yes: in reality, a single pathway mutant like max2 can absolutely change sucrose sensitivity, because sucrose partly acts through that same pathway.
* This is exactly the biology people now talk about: sugars tune SL and CK, SL mutants break that channel, so sugar responsiveness changes.

In real plants:

* **Sucrose (SUC)** provides *carbon* and signals nutrient availability.
* **Strigolactones (SL)** act as a *brake* on branching.
* **CK (cytokinin)** promotes branching (growth).
* **MAX2/SMXL mutants** are *insensitive* to SL — so they behave as if the brake is already off.
*  In **WT (intact SL pathway)**, sucrose increases growth both because it provides energy and because it **relieves** SL repression.
*  In **SL-pathway mutants**, sucrose still provides energy but can’t “relieve” SL inhibition (because the SL signal is already gone) — meaning sucrose should have less effect.