**Ideas for Stats EOY Data Analysis Project**

**Their hypothesis: Not clearly stated??**

**Methods/Results:**

1. **IPT expression levels (of IPT1, IPT3, IPT5, IPT7) were tested (reference gene was UBC21) following decapitation to see which ones are responsive to decapitation.**

**8-fold increase in IPT3. No significant changes in the other three.**

1. **Effect of mutated (non-functioning) IPT on branching was tested.**

Single **IPT** **mutants** formed less than one branch on average compared to WT (Col); Triple **IPT** **mutants** formed similarly less than one branch; Quadruple **IPT mutants** formed no branches. \*\*Findings significant with role of CK in promoting branching\*\*

1. **Grafting was done in the TRIPLE IPT MUTANTS to test where in the plant IPT is required to promote branching.**

Grafted TRIPLE **IPT mutants** significantly reduced branching compared to WT(Col). **GRAFTED WT shoot OR root** resulted in WT levels of branching. Therefore, CK produced in WHOLE plant rather than shoot alone contribute to branching in intact plants.

1. **Tested whether the target IPT genes (1, 3, 5, & 7) are required for auxin-mediated apical dominance. Done by decapitating mutant plants.**

SINGLE IPT mutants responded to decapitation by producing more branches. \*\*TRIPLE AND QUADRUPLE IPT MUTANTS DID NOT\*\*

TRIPLE MUTANTS did not even initiate buds, therefore could not activate buds. **IPT3** showed more empty axils than wild-type. **IPT5** and **IPT7** had similar numbers of empty axils than wildtype. Reduced branching in these is most likely due to lack of bud development.

1. **Explored the role of SL in CK mutants (arr3,4,5,6,7,15) and compared to wild-type as well as CK mutants with auxin alone.**

**SL applied with auxin inhibited buds more than auxin alone did. Determined a consistency with the canalization-based hypothesis.**

1. **Compared CK mutants to wild-type in high nitrate and low nitrate conditions and found that CK may be important to enhance branching under high nitrate.**

**In a nutshell:** CKs play little part in auxin-mediated but repression and release from apical dominance, but rather they provide a mechanism for buds to escape apical dominance and activate even in the presence of auxin

**Back ground:** In plants, apical dominance occurs when auxin (a hormone that acidifies cell walls by causing cells to pump hydrogen atoms into the cell wall) travels rootward down the stalk, inhibiting axillary branching from occurring. This is an indirect influence via secondary messengers. Auxin downregulates cytokinin (CK) and upregulates strigolactone (SL), hormones that increase and inhibit bud growth, respectively. When a plant is decapitated, the auxin source is removed and bud growth and expression of Isopentenyl transferase (IPT) - involved in prenylation of adenines as cytokinins - are more likely to increase. A more in-depth description of the relationship between and among these hormones, enzymes, and the involved gene groups of interest is shown in Figure 1.



**Figure 1.**

**ABA (gene group)**

**SL – Inhibits bud growth**

Upregulation

**Auxin (hormone)**

**IPTs(Enzyme)**

**CK – Increases bud growth**

Downregulations

**ARR (gene group)**

**Questions:**

1. **What is causing the lack of bud development in the triple mutants and IPT3?**

**Our Hypotheses:**