**Analysis of APA sites and m6A in WT and oxt6 mutant (CPSF30 knock-out)**

**Hypothesis:**

CPSF30 regulates alternative polyadenylation (APA) through interaction with m6A via its YTH domain. Or put differently: m6A is involved in alternative polyadenylation regulated by CPSF30.

Use motif-guided m6A sites from Wan et al. (2015) and analyse spatial enrichment with PACs in

* WT only
* mutant only
* both (common PACs)

Common PACs is our background (null hypothesis), because these sites are independent of the presence/absence of CPSF30.

**If our hypothesis is true, we expect:**

* Stronger enrichment of m6A sites adjacent to WT only PACs compared to common PACs
* Depletion of m6A sites adjacent to mutant only PACs compared to common PACs

This would support that CPSF30 is regulating APA through interacting with m6A, likely via its YTH domain.

**If our hypothesis is false, we expect no difference in the aforementioned analyses.**

* No difference in enrichment of m6A sites and WT only PACs compared to common PACs
* No difference in enrichment of m6A sites and mutant only PACs compared to common PACs

This could mean two things:

Firstly, the regulation of APA in the case of CPSF30 might be independent of m6A, and CPSF30 has additional functions (unrelated to APA) which are mediated by its interaction with m6A. Secondly, there might be other enzymes that are functionally redundant to CPSF30 and take over its role of m6A reader in APA. This would not be surprising since there are 14 largely uncharacterized YTH domain proteins in Arabidopsis (of which we picked up 11 in the interactome), while there are only three in humans.