Addition, Retraction, Correction

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Correction to Inhibiting GPI Anchor Biosynthesis in Fungi Stresses the Endoplasmic Reticulum and Enhances Immunogenicity

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Upon resequencing the plasmids encoding *Saccharomyces cerevisiae GWT1*, which were used for this manuscript an unexpected mutation was revealed. This mutation does not change any of the analysis or conclusions reached in the original paper, however. The mutation is an extra adenine added to a string of 10 adenines at bases 28−37 in the wild type nucleotide sequence. This would be expected to cause an early premature translation termination. However, as shown in the paper, and reconfirmed with the resequenced plasmid, it does rescue the lethal phenotype of a *gwt1* deletion. Moreover, when mutant Gwt1 is C-terminally tagged with EGFP, a fusion protein of the expected molecular size is expressed. Using the GPD promoter, as we did in our publication, the level of Gwt1-GFP is similar to that of a strain from the published GFP collection in which *GWT1* (under control of its native promoter elements) is C-terminally tagged by GFP at its endogenous chromosomal locus (Yeast GFP Clone Collection, Invitrogen). We tested a wild type *GWT1* sequence under the control of the same *GPD* promoter used in our studies and found that it drove accumulation of much higher than normal levels of Gwt1 in cells. We conclude that cells have a mechanism for correcting the frame shift (not uncommon in yeast cells). Presumably, the high level of transcription from the GPD promoter provides sufficient levels of expression even with the mutant plasmid to mimic expression from the wild-type gene. Hence, all conclusions in the original paper remain valid.

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