**Supplementary methods: Statistical analysis**

Statistical analyses were performed with R version 3.6, scripts are available as supplementary material. Significance for the statistical tests was coded in the figures based on the p-values: \*\*\*: 0 < p < 0.001;  \*\*: 0.001 < p < 0.01; \*: 0.01 < p < 0.05. P-values were corrected for multiple testing by a Holm-Bonferroni method (Holm 1079). Clone sizes were analyzed with a mixed-effect linear model on the logarithm of cell area, considering the treatment (Genotype and Sucrose conditions) as a fixed effects and Series/Larva as random effects (Figures 1, 5, and 7, Table S1). The reported effects (and the corresponding P-values) were obtained from the difference between the (log) area of marked clonal cells and that of control surrounding cells from the same treatment, by setting the appropriate contrast with the “multcomp” package (Hothorn *et al.* 2008), according to the pattern: EA,B = log(MA) – log(WA) – [log(MB) – log(WB)], where EA,B is the difference between treatments (genotype and sucrose levels) A and B, MA and MB standing for the area of marked cells, and WA, WB for the area of control cells in those treatments. This is equivalent to testing whether marked/control cell area ratios differ between treatments. PS6+ clone frequencies were treated as binomial measurements in a mixed-effect generalized linear model “lme4” package (Bates *et al.* 2015), featuring Genotype as a fixed effect, and Series/Larva as random effects. Both datasets of pupal weights were analyzed independently with linear models including Sex, Genotype, and Sucrose level effects and all their interaction terms (Figure 3A-B and Table S3 for PTEN knockdown and Rheb overexpression; Figure 6B and Table S4 for *FASN1-2* mutants). TAG, Protein, Glycogen, and Trehalose concentrations were also analyzed with linear models involving Genotype, Sucrose level, and their interactions as fixed effects (Figure 3 and Table S3). Survival data (Figure 4 and Table S5) were analyzed with the “survival” package (Therneau and Grambsch 2000), assuming a Weibull distribution for mortality; Replicates (vials) being considered as clusters (correlated observations). Pupariation data (Figure 6A and Table S6) were analyzed with a binomial (puparate/not puparate at day 15)generalized mixed-effect model, with Genotype and Sugar as fixed effects and replicate as a random effect. A three-parameter logistic function A/([ 1 + exp(- (t - x))/s ] was fitted to the temporal dynamics of emergence frequencies (A: asymptotic frequency, x: median emergence time, s: scale paramter). The non-linear, mixed-effect model (package nlme) considered Genotype and Sugar as fixed effects for all three logistic parameters, and Replicate (vial) as a random effect (only for A and x due to convergence issues).

**Supplementary references:**

Bates, D., M. Mächler, B. M. Bolker and S. Walker, 2015 Fitting Linear Mixed-Effects Models Using lme4. Journal of Statistical Software 67**:** 1-48.

Holm, S., 1079 A simple sequentially rejective multiple test procedure. Scand J Statist 6.

Hothorn, T., F. Bretz and P. Westfall, 2008 Simultaneous inference in general parametric models. Biom J 50**:** 346-363.

Therneau, T. M., and P. M. Grambsch, 2000 *Modeling survival data : extending the Cox model*. Springer, New York.