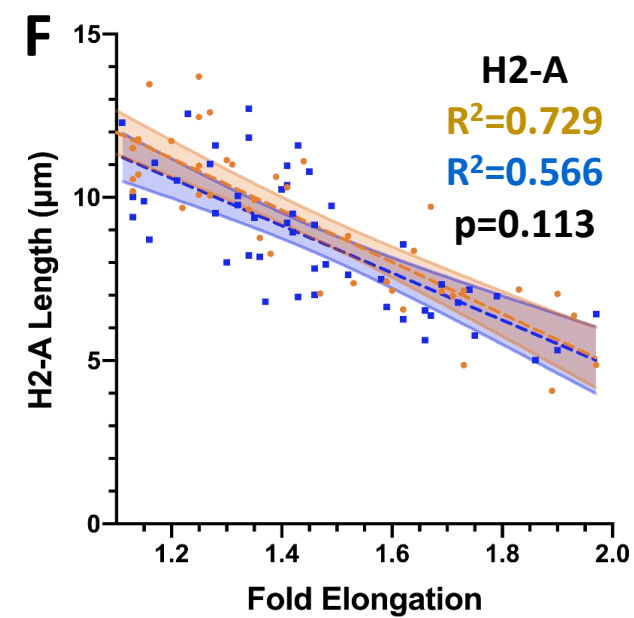
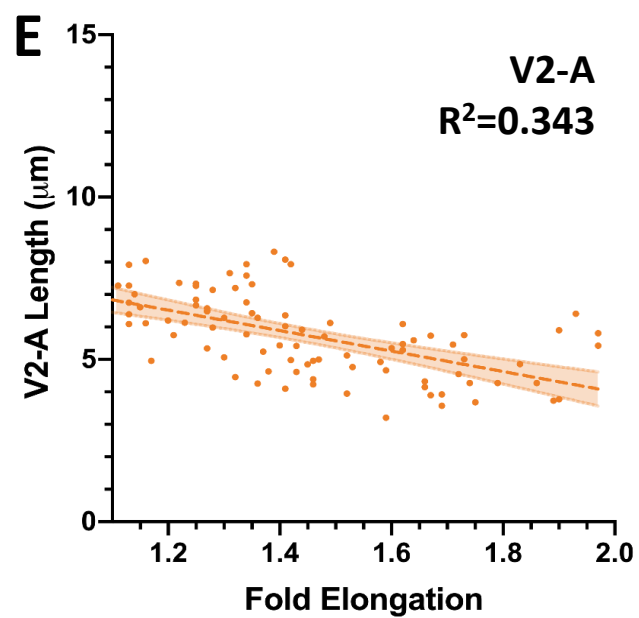
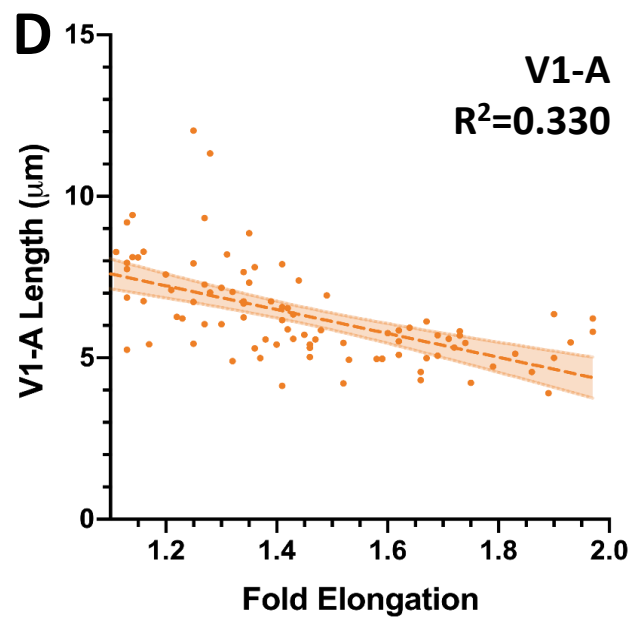
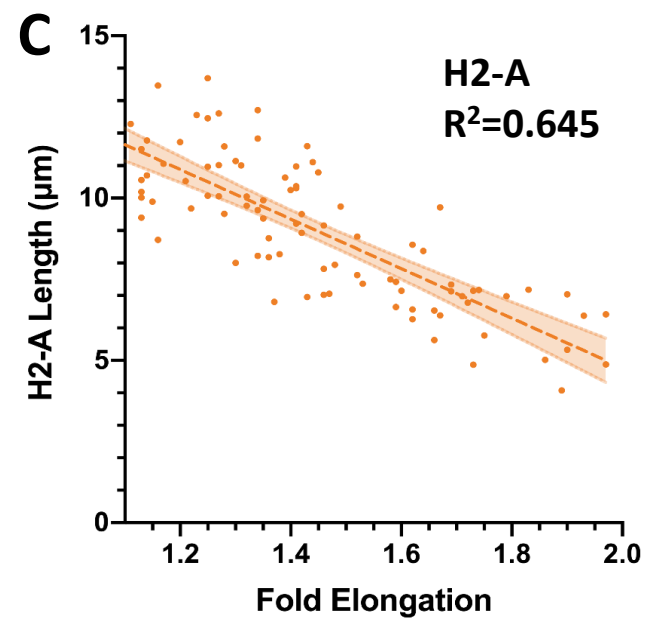
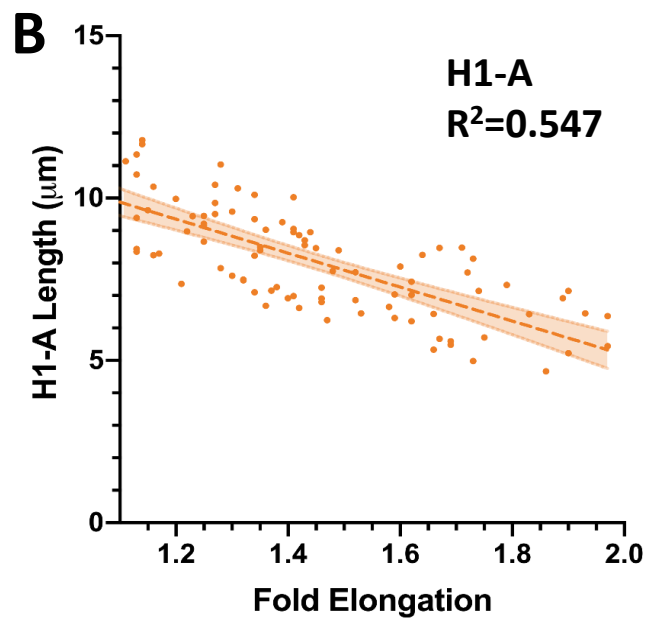
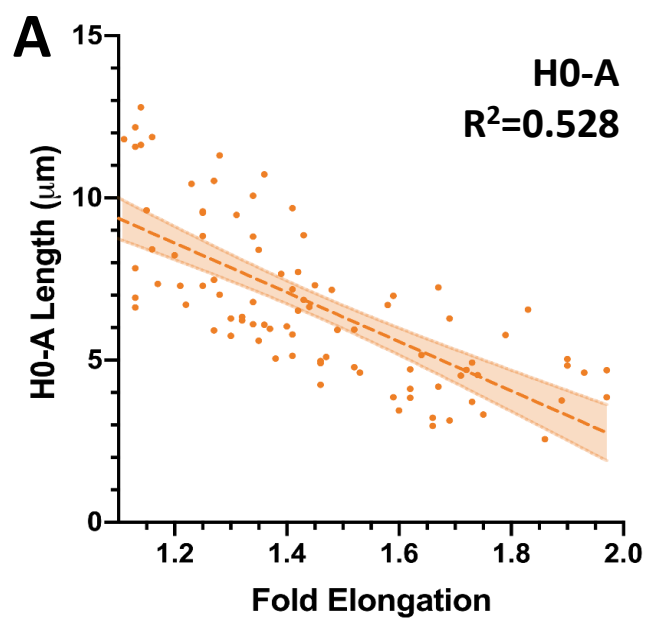


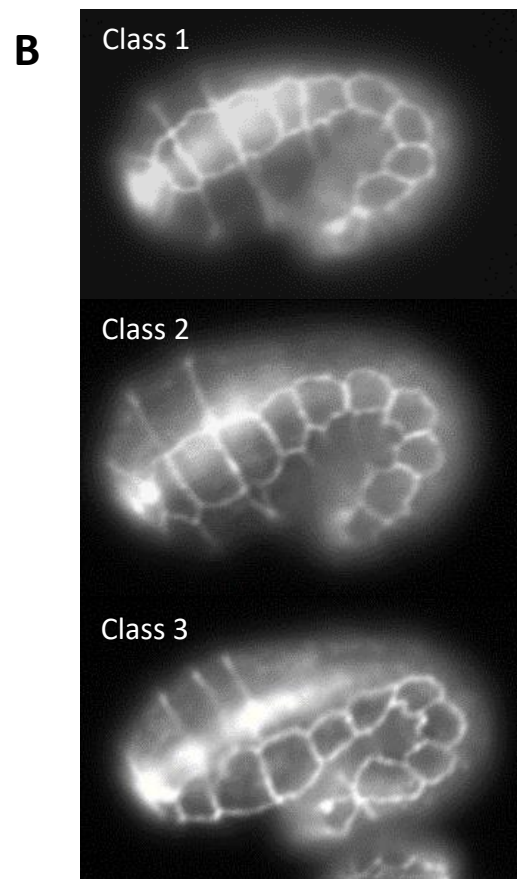
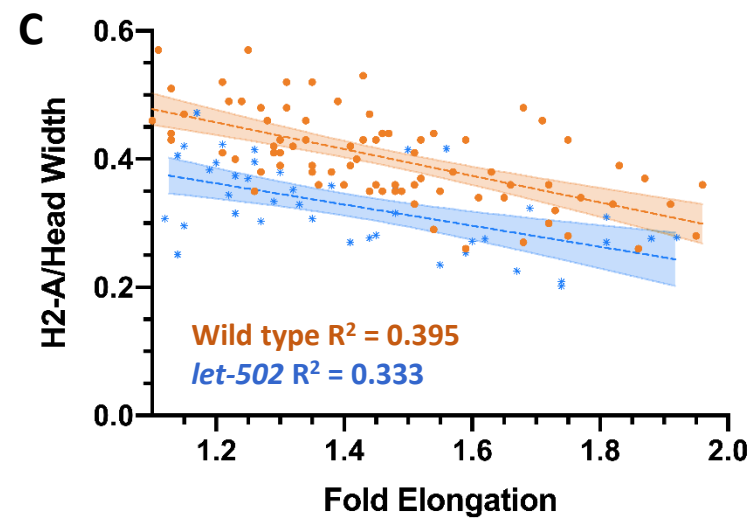
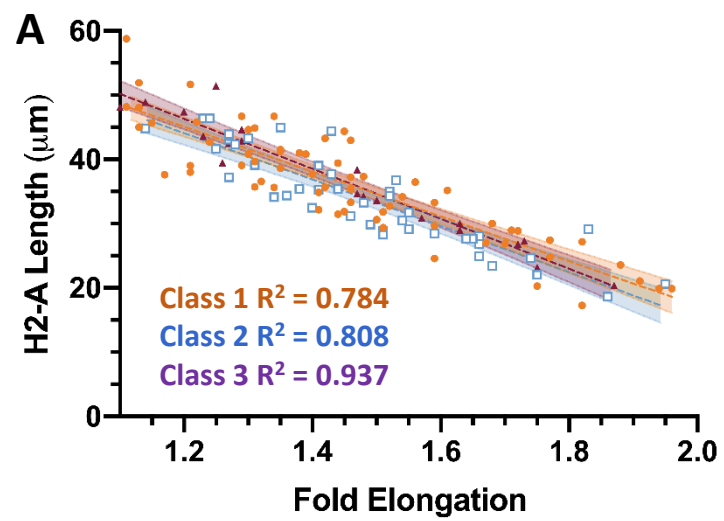
Supplemental Figure 1

**Supplemental Figure 1** Model of elongation proposing that genes act primarily in either lateral or dorsoventral epidermal cells. *let-502*, *rhgf-2* and *fhod-1* were thought to provide lateral force, *pak-1*, *pix-1* and *fem-2* dorsoventral force with *mel-11* and *rga-2* inhibiting force in dorsoventral regions. The first three rows indicate strains where genes mediating lateral force, dorsoventral force or inhibiting dorsoventral force are wild type or mutant (colors correspond to those in the pathway shown in Figure 1B). The next three rows indicate predicted phenotypes in dorsal, lateral or ventral cells. Column 2 indicates all genes are wild type, resulting in passive dorsal and ventral cells but contractile lateral cells. The size of the red arrows in cartoons below indicates the relative contractile forces with the lateral cells contracting with greater force than dorsoventral cells in wild type. Column 3 indicates loss of lateral force in *let-502*, *rhgf-2* or *fhod-1* resulting in more passive lateral cells and hence wider lateral cells at a given degree of embryo lengthening as dorsoventral forces now contribute proportionally more to elongation. Column 4 shows mutants for *pak-1*, *pix-1* or *fem-2* where the tension in dorsoventral cells is diminished such that the lateral cells can contract more, and must do so, to bring about the same degree of elongation. *mel-11* or *rga-2* are mutant in column 5, resulting in strong forces in all epidermal cells. With added dorsoventral contraction, the embryos elongate with less lateral contraction. Column 6 shows the prediction of pairwise double mutants of *let-502* or *rhgf-2* with *mel-11* or *rga-2*. Here embryos have contractile dorsoventral cells and passive lateral cells and so elongate due to dorsoventral force alone. Embryos will thus have wider lateral cells at any given stage of elongation.



Supplemental Figure 2

**Supplemental Figure 2.** Comparisons of the relationship of the length of different anterior lateral cells to fold elongation. The anterior membrane of each cell was measured as described in the Figure 5 legend, and the numbering of the cells is indicated in Figure 5A. (A) H0, (B) H1, (C) H2, (D) V1, (E) V2. The highest correlation for the linear regression of the slope ( $R^2$ ) was found for the anterior H2 membrane (referred to as H2-A in the text) and this was chosen for our analysis. (F) The H2-A membrane of wild type was examined in two separate experiments (blue vs. orange) and was found to be reproducible. Shadings indicate 95% confidence interval for the slope of the regression lines.



Supplemental Figure 3

**Supplemental Figure 3.** (A) Wild-type embryos were scored separately depending upon the orientation of the lateral cells with the plane of the slide. In Class 1 (orange circles), the lateral cells were parallel to the plane of the slide such that they were on the uppermost surface of the embryo. This was also indicated by the gut autofluorescence being directly below the lateral cells. In Class 2 embryos (blue squares), the lateral cells were tipped to one side while in Class 3 (purple triangles) the lateral cells were on the side of the embryo and gut autofluorescence was entirely outside the bounds of those cells. The correlation of H2-A with fold elongation is similar between the three classes. Shading indicates 95% confidence limits for the slopes of the regression lines and two-tailed p values compare the slopes. (B) Examples of each class are shown. (C) The relative proportion of head width taken up by the H2-A membrane as a function of fold elongation in wild type (orange) is compared to *let-502(ts)* (blue). Similar to comparing absolute H2-A length vs. fold (Figure 5B), many mutant cells begin elongation with proportionally narrow widths.