Conclusion:

The axial system of yeast cells is set during cytokinesis as the mitotic spindle is assembled, together with an orthogonal contractile ring. Cell division in multicellular organisms is dependent on similar cytoskeletal remodelling mechanisms, with cortical actin fibrils anchored to the extracellular matrix. Thus, the cytoplasmic membrane interface has a critical function during the transmission of mechanical stress between cells. In general, the first determinative embryonic divisions are synchronous, with asymmetric partitioning of morphogenetic factors. However, during later developmental stages most divisions are asynchronous with uncoupled, free-cycling cells. In this context, the first syncytial divisions of Drosophila are atypical, with determinative cleavages delayed until the mid-blastoderm transition. Thereafter, the gap- and segmentation-gene cascade sets the body-plan as a harmonic standing wave, constrained by the surface topography of the egg. As in other bilateral organisms, a longitudinal AMS divides the short (D/V, L/ R) body axis. An additional, orthogonal, set of parasegmental AMSs forms along the long (A/P) embryonic axis. During gastrulation and germ-band extension, cells may be rotated at epithelial infoldings, around one (or more) of their Cartesian axes. In consequence, morphogenetic twin-fields are generated around axes of mirror symmetry, aligned with the embryonic long and short axes. The A > P parasegmental progression deploys incrementally modified developmental pathways, with alternative, orthosegmental (D/V) programmes. Proliferative growth is regulated from AMSs and twin-field boundaries via the TGF-β and Wnt signalling pathways. However, individual cell fates remain labile until terminal PCP signalling.

During larval growth, polarised morphogen flux transmits predominantly scalar information, with asynchronous divisions. By contrast, during terminal differentiation the cortical actin cytoskeleton is remodelled following metachronal waves of actomyosin contraction. This temporal progression may provide a second scalar morphogenetic component. Short-range lateral inhibition mechanisms regulate the initiation of rosette cell clusters, which may rotate with respect to their surrounding interstitial cells. During these processes, the tendency of collagen fibrils to assemble into parallel or orthogonal arrays may function in coupling cellular coordinates across the extracellular matrix (and facilitating 90^{o} axial rotations). Thus, fine-scale patterning is generated by asymmetric partitioning, with recursive, morphogenetic interactions. Vectorial information is generated along advancing metachronal wavefronts, without a pre-existing global positional information matrix. As individual cells adopt differentiated fates, their nucleosome phasing patterns may be re-set, with a restricted range of metabolic functions. Thus, complex tissues may be assembled from 2D, epithelial templates, with morphogenetic mechanisms coupled to the cell-cycle oscillation.

Acknowledgements:

To FlyBase, without whom we would all be lost.

To the fly, mouse and medical communities, for support and criticism over many years:

John Manak and Jean-Marc Reichhart.
M.A. Ashburner, R. Barrio, W. Chia, S. Collier, D. Crowther, T. Dafforn, A. Garcia-Bellido, C.F. Graham, I. Guerrero, D. Huen, K. Moses, J. Roote, P. Ripoll, R. Karp, P. Lasko, E.B. Lewis, D. Lomas, G. Micklem, A.C. Neville, B. Reed, S.E. Reynolds, M.P. Scott, J. Sutherland, J. Tamkun and A. Wilkins.