**Establishment**

As previously mentioned, PAR polarity doesn’t occur spontaneously, requiring a trigger so that polarity happens at the correct time and with the correct orientation. Prior to the establishment of asymmetry, aPARs are uniformly enriched on the cortex, and exclude pPARs through antagonistic interactions. Polarity is then triggered by signals from the MTOC which forms near the site of sperm entry, via two redundant pathways:

**Anterior-directed cortical flows**

A first mechanism involves a local inhibition of RhoA activity in the posterior of the cell, which downregulates actomyosin contractility. This sets up a spatial gradient of contractility, which leads to anterior-directed cortical flows. The aPARs PAR-3, PAR-6 and PKC-3 are able to tap into these flows, which causes them to segregate to the anterior. <more here on local RhoA inhibition, par-3 clustering>

The resulting depletion of PKC-3 concentration in the posterior relieves antagonism of pPARs, allowing them to bind in the posterior and form a nascent domain. This domain is then amplified via a series of antagonistic feedback reactions, which are described in another section. This mechanism of advective triggering followed by self-organisation is sufficient to capture the core features and dynamics of polarity in computer models of the PAR network (Goehring).

Further work has shown that the PAR proteins themselves are able to feed back onto the actomyosin cortex, amplifying contractility asymmetries as polarity progresses (Gross) <More here>. PAR-2, and possibly LGL-1, can also feed back onto the cortex, reducing contractility in the posterior (ref), although this is inessential for proper contractility at establishment phase (Gross) and probably most important during maintenance phase, as discussed later.

**The microtubule pathway**

In the absence of cortical flows, symmetry breaking still occurs, albeit later, indicating the existence of multiple symmetry breaking mechanisms. A second triggering mechanism involves an interaction between PAR-2 and microtubules emanating from the sperm-donated centrosome. In no flow regimes, PAR-2 symmetry breaking occurs late, and correlates spatially and temporally with the site of MTOC-cortex contact (Motegi). Treatments that disrupt microtubules, or mutants at the binding interface on PAR-2 prevent symmetry breaking in no-flow conditions.

Mechanistically, this is carried out by an interaction between PAR-2 and microtubules, which is thought to shield the phosphorylation sites on PAR-2, and has been shown to reduce phosphorylation by PKC-3 in vitro. This creates a zone of local protection in the posterior of the cell, allowing PAR-2, and thus PAR-1 to load, kicking of self-organisation.

**Additional pathways**

Additional mechanisms may underlie triggering in some circumstances. <Evidence of still breaking symmetry when other cues are lost>. Microfabrication studies show that PAR-2 has a preference for curved membranes. This may lead to preferential binding of PAR-2 at the poles of cells, which has been suggested to act as a symmetry breaking cue in cases where normal symmetry breaking is misregulated, (Klinkert). The mechanistic basis for this proposed curvature sensitivity is unclear.