

< FURTHER DEVELOPMENT 12.2>

Separating and guiding with cadherins and calcium

The second force is differential cell cohesion. During gastrulation, the genes encoding the adhesion proteins **paraxial protocadherin** and **axial protocadherin** become expressed specifically in the paraxial (somite-forming; see **Chapter 19**) mesoderm and the notochord, respectively. An experimental dominant-negative form of axial protocadherin prevents the presumptive notochord cells from sorting out from the paraxial mesoderm and blocks normal axis formation. A dominant-negative paraxial protocadherin (which is secreted instead of being bound to the cell membrane) prevents convergent extension (Kim et al. 1998; Kuroda et al. 2002).¹ Moreover, the expression domain of paraxial protocadherin characterizes the trunk mesodermal cells, which undergo convergent extension, distinguishing them from the head mesodermal cells, which do not undergo convergent extension.

A third factor regulating convergent extension is calcium flux. Wallingford and colleagues (2001) found that dramatic waves of calcium ions (Ca^{2+}) surge across the dorsal tissues undergoing convergent extension, causing waves of contraction within the tissue. Ca^{2+} is released from intracellular stores and is required for convergent extension. If Ca^{2+} release is blocked, normal cell specification still occurs, but the dorsal mesoderm neither converges nor extends. These findings support a model for convergent extension wherein regulatory proteins cause changes in the outer surface of the tissue and generate mechanical traction forces that either prevent or encourage cell migration (Beloussov et al. 2006; Davidson et al. 2008; Kornikova et al. 2009)

All the material on this website is protected by copyright. It may not be reproduced in any form without permission from the copyright holder.

© 2023 Oxford University Press |

¹ Dominant-negative proteins are mutated forms of the wild-type protein that interfere with the normal functioning of the wild-type protein. Thus, a dominant-negative protein will have an effect similar to a loss-of-function mutation in the gene that encodes the protein.

